Estimating the Public Health Benefits of Proposed Air Pollution Regulations

Committee on Estimating the Health-Risk-Reduction Benefits of Proposed Air Pollution Regulations, National Research Council

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Proposed Air Pollution Regulations

Board on Environmental Studies and Toxicology

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Preface

The U.S. Environmental Protection Agency (EPA) estimates that thousands of premature deaths and numerous cases of illness, such as chronic bronchitis and asthma attacks, could be prevented by reducing exposure to air pollution. These estimates are derived from health benefits analyses, which attempt to quantify changes in the expected cases of mortality and illness that are likely to result from proposed regulations. These estimates are often controversial and the methods used to produce them are often questioned. Because of the importance of these estimates in decision-making, the U.S. Senate directed EPA to request that the National Research Council (NRC) evaluate methods used to derive the health benefits estimates and make recommendations on best practices for these types of analyses.

In this report, the NRC's Committee on Estimating the Health-Risk-Reduction Benefits of Proposed Air Pollution Regulations reviews recent EPA analyses and provides recommendations for improvement of the methods used. Specifically, the committee addressed issues concerned with the structure of the analysis, such as the regulatory options to evaluate, the time frame to use, and the assumptions to make about conditions with and without the regulation. The committee also considered issues regarding the exposure assessment, the selection of health outcomes and the concentration-response function, the analysis of uncertainty, and the presentation of the methods and results.

This report has been reviewed in draft form by individuals chosen for their diverse perspectives and technical expertise according to the procedures approved by the NRC's Report Review Committee. The purpose of this independent review is to provide candid and critical comments that will assist the institution in making its published report as sound as possible and to ensure that the report meets institutional standards for objectivity, evidence, and responsiveness to the study charge. The review comments and draft manuscript remain confidential to protect the integrity of the deliberative process. We wish to thank the following individuals for their review of this report: Aaron J. Cohen, Health Effects Institute, Boston, Massachusetts; Douglas J. Crawford-Brown, University of North Carolina, Chapel Hill, North Carolina; Edmund A.C. Crouch, Cambridge Environmental Inc., Cambridge, Massachusetts; Daniel Krewski, University of Ottawa, Ottawa, Ontario; Alan J. Krupnick, Resources for the Future, Washington, DC; Michal Krzyzanowski, European Centre for Environment and Health, Bonn, Germany; Jonathan I. Levy, Harvard School of Public Health, Boston, Massachusetts; Thomas A. Louis, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland; Robert L. Maynard, U.K. Department of Health, London; Roger O. McClellan (emeritus), Chemical Industry Institute of Toxicology, Albuquerque, New Mexico; Michael H. Scheible, Air Resources Board, Sacramento, California; George D. Thurston, New York University School of Medicine, Tuxedo, New York.

Although the reviewers listed above have provided many constructive comments and suggestions, they were not asked to endorse the conclusions or recommendations, nor did they see the final draft of the report before its release. The review of this report was overseen by Donald R. Mattison, National Institute of Child Health and Human Development, Bethesda, Maryland; and Maureen M. Henderson, (emeritus) University of Washington, Seattle, Washington. Appointed by the NRC, they were responsible for making certain that an independent examination of this report was conducted according to institutional procedures and that all review comments were carefully considered. Responsibility for the final content of this report rests entirely with the authoring committee and the institution.

The committee gratefully acknowledges the following individuals for making presentations to the committee: Robert Brenner and Bryan Hubbell, EPA; Andrew Wheeler, U.S. Senate Subcommittee on Clean Air, Wetlands, Private Property, and Nuclear Safety; Robert O'Keefe, Health

Effects Institute; John Graham, Office of Management and Budget; and Alan Krupnick, Resources for the Future. In addition, the committee especially thanks Armistead Russell, Georgia Institute of Technology, who provided background information and further analysis on air-quality modeling to the committee.

The committee is also grateful for the assistance of the NRC staff in preparing this report. Staff members who contributed to this effort are Ellen Mantus, project director; Roberta Wedge, program director for risk analysis; Eileen Abt, program officer; Ruth E. Crossgrove, editor, Mirsada Karalic-Loncarevic, research assistant; Jennifer Saunders, research assistant; and Lucy Fusco, senior project assistant.

I would especially like to thank all the members of the committee for their efforts throughout the development of this report.

John C. Bailar, III, *Chair*
Committee on Estimating the
Health-Risk-Reduction Benefits
of Proposed Air Pollution Regulations

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Estimating The Public Health Benefits Of Proposed Air Pollution Regulations

Summary

The U.S. Environmental Protection Agency (EPA) has estimated that thousands of premature deaths and numerous cases of illness, such as chronic bronchitis and asthma attacks, could be prevented by reducing exposure to air pollution. These estimates come from regulatory health benefits analyses, which attempt to quantify changes in the expected cases of mortality and illness that are likely to result from proposed air pollution regulations. The estimates are often controversial, and the methods used to prepare them have been questioned.

In 2000, Congress recognized concerns about the methods used by EPA and emphasized the need for "the most scientifically defensible methodology in estimating health benefits." It directed EPA to ask the National Academy of Sciences "to conduct a study of this issue and recommend to the agency a common methodology to be followed in all future analyses."[1]

THE CHARGE TO THE COMMITTEE

In response to EPA's request, the National Research Council (NRC) convened the Committee on Estimating the Health-Risk-Reduction Benefits of Proposed Air Pollution Regulations, which prepared this report. Mem-

[1]U.S. Senate. 2000. Senate Appropriations Report for Fiscal 2001. Report 106-410, 106th Congress, 2d Session.

bers were chosen for their expertise in risk assessment, exposure assessment, toxicology, epidemiology, biostatistics, health economics, and air pollution regulations. The committee was asked to accomplish the following tasks:

1. Consider issues important in estimating the health-risk-reduction benefits of air pollution regulations, including the scientific data, risk-assessment approaches, populations affected, baselines used, assumptions, analysis of uncertainty, and identification of key indicators of exposure and population health status.
2. Critically review methods used for recent estimates of regulatory health benefits.
3. Identify methods used by federal regulatory agencies and others, recommend standard good-practice guidelines and principles for estimating health benefits, and delineate the data-gathering required to better assess health benefits in the future.
4. Identify approaches to estimating regulatory health benefits when relevant information is limited.
5. Where applicable, recommend areas for further research and monitoring.

The committee was not asked to evaluate methods used to estimate other types of benefits, such as improvements in visibility, resulting from air pollution control. The committee also was not asked to review the methods used for economic valuation of health benefits or for regulatory cost analyses.

THE COMMITTEE'S APPROACH

To accomplish its charge, the committee heard, in public session, presentations from representatives of EPA, the U.S. Senate, the Office of Management and Budget (OMB), and other interested parties; reviewed materials submitted by EPA and others; and reviewed current literature relevant to health benefits estimation. The committee selected for detailed review the health benefits analyses contained in the regulatory impact assessments (RIAs) prepared by EPA for the following rule-makings: (1) "Particulate Matter and Ozone National Ambient Air Quality Standards"

(1997), (2) "Tier 2 Motor Vehicle Emissions Standards and Gasoline Sulfur Control Requirements" (1999), and (3) "Heavy Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements" (2000). The committee also reviewed the health benefits analysis completed for EPA's analysis of the benefits and costs of the 1990 Clean Air Act Amendments (CAAA) (1999). All of these analyses are described in Chapter 2 of this report (see Tables 2-1 and 2-5).

Ozone and airborne particulate matter (PM) were the primary focus of the EPA analyses selected by the committee for review. Therefore, the committee spent a considerable amount of time discussing these pollutants, especially PM, and did not address issues associated with the analysis of the hazardous air pollutants (HAPs). However, many of the findings and recommendations of the committee have broad applicability and are not limited to analyses conducted for PM.

THE COMMITTEE'S EVALUATION AND FINDINGS

Despite many inherent uncertainties, the committee concludes that regulatory benefits analysis can be a useful tool for generating information valuable to policy-makers and the public. Properly conducted analyses can help identify the type, magnitude, and relative importance of health benefits, highlight the sensitivity of the benefits estimates to assumptions made in the analysis, and indicate the areas of greatest scientific uncertainty. Information from the analyses can help focus future research efforts to reduce key uncertainties. The committee emphasizes, however, that estimates of health benefits and their economic valuation are only one part of the deliberative and political processes necessary for the development of sound policy.

Estimating the health benefits of a potential reduction in ambient air pollution involves a series of steps. First, the regulatory options to be evaluated must be clearly defined with regard to scope, timing, and implementation. Then, the boundaries of the analysis, such as the time period for which benefits are evaluated, must be established. In addition, the regulatory baseline (the description of conditions without the proposed regulation) must be defined. Once the analysis has been structured, future changes in pollutant emissions and resulting changes in ambient pollutant concentrations and population exposures can be predicted. Changes in health outcomes can then be estimated by applying concentration- or exposure-response

functions (derived from the health literature) to estimated changes in population exposures.

The committee finds that these basic steps provide a reasonable framework for conducting health benefits analysis and that EPA has generally used this basic approach when estimating the expected health benefits of proposed air pollution control regulations. However, on the basis of the analyses reviewed by the committee, EPA's implementation of these steps could be improved. Recommendations for improvements in the process are described in the following pages.

The committee notes that analysis of health benefits for any regulation will require flexible, innovative, and multidisciplinary participation and guidance of scientific experts. Therefore, the committee did not attempt to write a detailed manual for conducting benefits analysis but instead addressed the key methodological issues and their importance in the EPA benefits analyses reviewed by the committee.

Regulatory Options, Boundaries, and Baselines

The health benefits that are estimated to result from reducing air pollution depend on the decisions made at the beginning of the analysis regarding the regulatory options to consider, the health outcomes to evaluate, the time frame over which benefits are estimated, and the assumptions made about conditions with and without implementation of the regulation. In three of the four EPA analyses reviewed by the committee, EPA focused on evaluating a single regulatory option. This approach conflicts with current OMB guidance on benefits analysis, which suggests consideration of a range of regulatory options and a variety of technical and economic interventions.

The committee acknowledges that EPA cannot evaluate every possible regulatory option, given time and resource constraints; however, a realistic range of options guided by expert opinion and technical feasibility should be represented in EPA's benefits analyses. At the beginning of each analysis, EPA should describe this range of options and any preliminary analyses that were conducted to exclude certain options from the formal benefits analysis. This approach would strengthen analyses that might otherwise appear to serve the purpose of justifying EPA's chosen regulatory option.

Once the regulatory options are selected, EPA must determine how

broadly to define the scope of the analysis, including the degree to which secondary or unintended effects of the regulation should be examined. For example, air pollution regulations can change not only ambient air pollution levels but also how fuels are made or how combustion devices are operated. These changes might affect human health through other pathways, such as through water pollution or occupational exposures. An analysis of health benefits that ignores those effects might result in a substantial misrepresentation of the potential impacts of pollution-control measures on society. Although the committee recognizes that assessment of secondary effects may be difficult, the benefits analysis should discuss whether such impacts appear to be important and, if so, should incorporate a plan for assessing them.

Although EPA usually evaluates the *costs* of regulatory options for the time period between introduction and full implementation of the regulation, the *benefits* of the regulation have often been examined for only a single year—typically the year in which the regulation will have been fully implemented. Evaluation of benefits for only a single year has two limitations. First, when the costs of the regulatory action decrease over time and the benefits increase, the comparison of benefits and costs in the distant future could be misleading. Second, choosing an evaluation point in the distant future, such as 2030, is likely to increase the uncertainty associated with estimating both benefits and costs. These limitations can make the analysis misleading. Therefore, benefits should be estimated at reasonable intervals, such as every 5 years, over the regulatory time frame, including both the period of implementation and the expected period of expression of all significant health effects.

To estimate the benefits of a proposed air pollution regulation, EPA makes predictions about conditions expected to occur both with the regulation (control scenario) and without the regulation (baseline scenario). Predictions concerning air emissions and the U.S. population are especially relevant to calculating the health benefits. Two issues regarding emissions predictions particularly concern the committee. First, many important components of an emissions analysis, such as number of vehicles in a class, average miles traveled per vehicle, and emissions per mile, are seldom summarized for the benefits analysis. This lack of information makes it difficult to judge the plausibility of the emissions estimates. Second, current emissions models fail to provide an assessment of uncertainty associated with the emissions predictions for the baseline and control scenarios, which

can be substantial. Comparison of emissions predictions to historical trends could help elucidate discrepancies that should be explained or formally incorporated into an uncertainty analysis and taken into account when estimating health benefits.

Predictions about future populations, such as numbers, age distributions, and baseline health status, are important aspects of EPA's benefits analyses. However, it is difficult to make confident predictions about the characteristics of populations 30 years in the future. EPA should evaluate the uncertainty involved in these predictions and the impacts of these uncertainties on the benefits estimates. Some sense of the uncertainty in these predictions may be obtained by comparing the characteristics, such as age, sex, ethnic mix, disease, and mortality, of the projected future population with those of the populations studied in the epidemiological studies on which the benefits estimates are based.

Exposure Assessment

A critical step in estimating the benefits of proposed air pollution regulations is determining the effect of emissions changes on ambient air quality. This has traditionally been accomplished using air-quality models of varying complexity. EPA's approaches to exposure assessment evolved considerably over the period of the analyses reviewed by the committee as a result of continued improvement in the models and marked increase in available monitoring data for key pollutants. Overall, the methods used in the most recent EPA analysis reviewed by the committee (heavy-duty engine and diesel-fuel analysis) represent an appropriate and reasonably thorough application of the available data and models for exposure assessment.

Several issues, however, deserve to be mentioned regarding the models and the assumptions used in the exposure assessments. First, models are simplifications of reality. Estimating how well a model simulates pollutant concentrations in the ambient air resulting from emissions changes estimated at some future time is difficult and requires a systematic process of model testing and evaluation. Without such a process, it is difficult to know how much confidence to place in the predictions. The methods used to test the models also need to be clearly described in the benefits analysis. Second, many of the models used by EPA are time and resource intensive, thus limiting the modeling that can be conducted. The limitation is problem-

atic because it restricts the number of regulatory options that can be considered and the number of years for which benefits can be estimated.

A tacit or explicit assumption in exposure assessment is that pollutant concentrations in ambient air adequately represent human population exposures. Although ambient concentrations in many cases appear to be reasonable indicators of human exposure, EPA should more rigorously assess the relative contributions of different emissions sources to human exposures. For example, EPA should evaluate whether PM emissions from diesel-fuel vehicles have a greater impact on human exposure than those from stationary sources, because diesel exhaust is emitted closer to people.

Another assumption specific to the analyses reviewed by the committee concerns PM. PM is a heterogeneous mixture that varies in size, composition, and source of origin; therefore, the health effects of PM exposures in one area might be different from those in another area and might vary over time. For example, the health effects of agricultural PM, which are derived primarily from crustal, animal, and plant sources, may differ from the health effects of urban PM, which are derived primarily from combustion sources, such as power plants and automobile and truck traffic. Because scientific information on PM toxicity is incomplete, EPA has typically made the assumption of equivalent potency across particle types. The committee believes that benefits analyses would be strengthened by evaluating a range of alternative assumptions regarding relative particle toxicity in sensitivity or uncertainty analyses.

Health Outcomes

The appropriate selection and definition of adverse health outcomes is integral to any assessment of health benefits. A wide range of health effects, primarily related to the respiratory and cardiovascular systems, is linked to exposure to air pollutants. In the analyses reviewed by the committee, EPA appears to have carefully considered the majority of these effects. However, many health outcomes are not quantified because there are insufficient data or because inclusion of certain health effects in the primary analysis could lead to double-counting.

The committee identified several issues regarding the selection and definition of mortality and morbidity (disease and other adverse health effects) outcomes. Clinically diagnosed illnesses, such as chronic bronchitis

and asthma attacks, are typically evaluated in benefits analyses. A problem with these diagnoses is that they cover a wide range of severity levels and time courses. For example, chronic bronchitis can range from a chronic cough to a severe chronic airway obstruction that requires long-term care. The lack of clear categorization of outcome severity in benefits analyses has implications for quantification and valuation of the outcomes. Although EPA has made some attempt to deal with this issue, it needs to investigate and improve the methods used to reconcile differences between the severity of disease described in air pollution epidemiology and that commonly used to develop estimates of background disease prevalence and incidence.

In each benefits analysis reviewed by the committee, EPA used U.S. studies to provide data to estimate the health benefits. Data for many health outcomes in the U.S. studies are restricted to a specific age group. For example, the data for hospital admissions apply to persons 65 years or older, primarily because the data come from Medicare databases. For the benefits analyses, EPA did not extrapolate those data beyond the age ranges provided in the studies. The committee notes that recent studies conducted outside the United States provide information on certain health outcomes with broader age ranges and on outcomes not currently evaluated by EPA, such as levels of use of the primary-care system. EPA should use such studies when appropriate to extrapolate beyond the age ranges currently considered and to incorporate health outcomes not currently evaluated in the analyses.

Mortality is a well-defined health outcome that was evaluated in each EPA analysis reviewed by the committee. Mortality estimates tend to dominate the overall health benefits estimates when a dollar value is assigned to them. However, the committee notes that data on morbidity is less comprehensive and needs to be improved, especially if the value assigned to mortality decreases and morbidity outcomes begin to play a more dominant role in the benefits analysis.

Another important issue relates to the key assumption that there is a causal association between particular types of air pollution and adverse health outcomes. The EPA benefits analyses reviewed by the committee provided little information concerning this assumption. Although a comprehensive discussion of causality is not necessary for a benefits analysis, the evidence of causality should be summarized to justify the inclusion or exclusion of health outcomes and to assess the uncertainty associated with the assumption of causality. EPA should investigate and, if necessary,

develop methods of evaluating causal uncertainty relating to key outcomes so that this uncertainty can be represented in the final benefits estimates.

Concentration-Response Functions

A primary element of health benefits analysis is the selection of the concentration-response functions, which describe the quantitative association between ambient air pollution levels and the corresponding health effects. Concentration-response functions can be derived from animal studies, human clinical studies, or epidemiological studies. In the analyses reviewed by the committee, EPA relied on epidemiological studies as the basis for estimating concentration-response functions. Because epidemiological studies involve the study of humans in real-world situations and, therefore, are more relevant to the assessment of health benefits than animal toxicity or human clinical studies, the committee supports the use of these studies to estimate concentration-response functions. However, the benefits analyses should reflect the plausibility and uncertainty of the concentration-response function, such as imprecision of exposure and response measures, potential confounding factors, and extrapolation from the study population to the target population in the benefits analysis.

For the analysis of mortality, EPA used cohort studies (epidemiological studies that evaluate health effects in a specific population over a period of years) to derive benefits estimates in each analysis reviewed by the committee. The committee agrees with that approach. Compared with time-series studies (epidemiological studies that provide estimates of health effects due to recent exposure), cohort studies give a more complete assessment because they include long-term, cumulative effects of air pollution. Furthermore, the particular advantage of cohort studies is that they provide data to estimate the number of life-years lost in a population, not just the number of lives lost, thus allowing for several valuation methods to be used.

Overall, the committee found that the epidemiological studies selected by EPA for use in its benefits analyses were generally defensible. However, the criteria and process by which EPA reached its decisions were not articulated in many cases, and at times, the study selection process appeared to be inconsistent. For example, estimates were derived from multiple studies in some cases and from single studies in other cases when

multiple studies were available. This selection process requires judgment on the part of the analyst, and EPA needs to document clearly the rationale for its selection of studies and concentration-response functions.

The committee concluded that EPA's selection of the American Cancer Society (ACS) study[2] for the evaluation of PM-related premature mortality was reasonable, given the size and precision of the study. However, those facts are not necessarily grounds for adoption of this study over others. For example, the Harvard six cities study[3] has some advantages over the ACS study, such as the use of a random population sample and the careful placement of monitors for the study. Because several new studies have since been published, including an extended analysis of the original ACS study, a new U.S. cohort study, and other non-U.S. studies, EPA should review its selection of the most appropriate studies. Furthermore, EPA might want to consider derivation of a weighted-mean estimate from the cohort studies following review of the entire database.

Decision-makers may want to know the effects of a regulation on different subgroups of a population, such as groups with varying health or socioeconomic status. Health effects might vary because the regulation causes different reductions in exposures for different subgroups or because various subgroups may respond differently to a specific exposure reduction. Populations may respond differently because their baseline rates of illness differ or because their concentration-response functions differ. The committee encourages EPA to estimate and report benefits by age, sex, and other demographic factors, when possible. Any assumptions that might explain the differences among subgroups should be clearly stated.

Analysis of Uncertainty

EPA uses a two-part approach to assess uncertainty in its health benefits analyses. The first part of the approach is a primary analysis that

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[2]Pope, C.A. III, M.J. Thun, M.M. Namboodiri, D.W. Dockery, J.S. Evans, F.E. Speizer, and C.W. Heath Jr. 1995. Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am. J. Respir. Crit. Care Med. 151(3 Pt 1):669-674.

[3]Dockery, D.W., C.A. Pope, X. Xu, J.D. Spengler, J.H. Ware, M.E. Fay, B.G. Ferris, and F.E. Speizer. 1993. An association between air pollution and mortality in six U.S. cities. N. Engl. J. Med. 329(24):1753-1759.

produces a probability distribution for each health outcome evaluated. For example, EPA provided a probability distribution for the number of avoided deaths in 2030 in the analysis conducted for the Tier 2 rule-making. Only one source of uncertainty (the random sampling error associated with the estimated concentration-response function) was incorporated into the analysis. EPA typically emphasizes only the mean value of the probability distribution. Because of the lack of consideration of other sources of uncertainty, the results of the primary analysis often appear more certain than they actually are.

The second part of the approach is ancillary uncertainty analyses, which include alternative and supplementary calculations for some uncertainties and sensitivity analyses for others. The ancillary analyses usually examine one source of uncertainty at a time and therefore do not adequately convey the aggregate uncertainty from other sources, nor do they discern the relative degrees of uncertainty in the various components of the health benefits analysis.

EPA should move the assessment of uncertainty from its ancillary analyses into its primary analyses to provide a more realistic depiction of the overall degree of uncertainty. This shift will entail the development of probabilistic, multiple-source uncertainty models based not only on available data but also on expert judgment. EPA should continue to use sensitivity analyses but should attempt to include more than one source of uncertainty at a time. EPA also should strengthen its efforts to identify the uncertainty sources that have the greatest influence on the final results. The committee emphasizes that cost estimates are also subject to great uncertainty, and the same standards should be applied to the assessment of the uncertainties in those estimates.

As more sources of uncertainty are incorporated into the primary analyses, the results inevitably will appear less certain, and the analyses might appear to be less useful to some. However, uncertainty should be described as completely and as realistically as possible for all regulatory options, recognizing that regulatory action might be necessary in the presence of substantial uncertainty. The regulatory decision process will be better informed by a fair assessment of the uncertainty and a realistic evaluation of the likely reductions in that uncertainty attainable through further research.

Accurately characterizing the uncertainties in estimates of health benefits for projected future human populations is difficult. Therefore, EPA should consider conducting preliminary analyses that estimate in current

populations the health benefits resulting from hypothetical changes in current levels of emissions. Such preliminary analyses would help EPA develop an idea of the lower bound on the range of uncertainty. These analyses also would have fewer uncertainties than analyses based on projected future population exposures and health outcomes.

Presentation of Results

A common complaint about EPA's regulatory benefits analyses is that the methods, the rationale behind the decision-making, and the results are not clearly described or presented. After review of the EPA analyses, the committee agrees that the presentations should be improved. The committee is concerned that important factors that drive the results of an analysis are often buried in appendixes or technical-support documents, and the rationales behind key decisions are not clearly discussed. Furthermore, the amount of discussion devoted to some parameters often does not appear to be proportional to their importance to the analysis. For example, in the heavy-duty engine and diesel-fuel analysis, an interpolation method used in the exposure assessment is discussed at length, whereas the exclusion of modeling results for the western United States is acknowledged in only one sentence.

The committee concludes that many of the problems associated with EPA's presentation of such analyses could be solved by inclusion of a detailed summary that presents the key information of the analysis in a straightforward manner. Such information includes the following:

- Regulatory options.
- Analytical boundaries.
- Baselines.
- Emissions changes.
- Changes in ambient air quality.
- Health outcomes evaluated.
- Quantified benefits.
- Uncertainties associated with the estimates.

The summary should highlight all assumptions that have a substantial impact on the results of the analysis.

The results of health benefits analyses are typically used as inputs to cost-benefit or cost-effectiveness analyses. Therefore, EPA should provide benefits estimates in ways that provide useful input to these analyses. For example, benefits estimates should be presented when possible by age group to allow calculation of quality-adjusted life-years, a measure used in cost-effectiveness analysis.

RECOMMENDATIONS

The committee recognizes that some of the following recommendations will be easier for EPA to implement than others. However, with the exception of research needs, these recommendations should not require substantial new resources on the part of EPA, although EPA may need to change its approaches and allocation of resources to accomplish them. The committee acknowledges that some of the research needed is outside EPA's jurisdiction and will require support from other agencies.

- EPA should include in its regulatory benefits analyses comparative estimates of the benefits for several regulatory options that represent a realistic range of choices available to the decision-maker. If regulatory options are eliminated at an early stage, the rationale for the elimination should be provided.
- EPA should examine whether unintended positive or negative impacts on human health or the environment might occur from implementation of the proposed regulation. For example, changes in fuels could result in water pollution, changes in occupational exposures, or reductions in greenhouse gas emissions. If important impacts are identified, a plan to assess them more completely should be included.
- EPA should estimate potential benefits at reasonable intervals, such as every 5 years, over the regulatory time frame, including the period of regulatory implementation and the expected period of occurrence of all significant health effects.
- EPA should present the information on which emissions estimates are based for scenarios with and without the regulation. This information will help readers judge whether the predictions are reasonable and will suggest which components are most important in driving the emissions reductions associated with the regulation.

- EPA should clearly state the projected baseline statistics used in estimating health benefits, including those for air emissions, air quality, and health outcomes.
- EPA should assess the degree to which modeled predictions agree with measured observations that have not been used to derive or calibrate the model. The results of those comparisons should be presented in the benefits analysis and used to help characterize the uncertainties associated with the resulting modeled predictions.
- More emphasis should be given to the assessment, presentation, and communication of changes in morbidity and quality of life. Although often difficult to quantify, these factors may begin to play a more dominant role in benefits analysis if the value assigned to mortality decreases.
- EPA should improve the methods used to account for the spectrum of severity of clinically diagnosed illnesses. When appropriate, EPA should also use data from non-U.S. studies in its benefits analyses to broaden the age ranges to which current estimates apply and to include more types of relevant health outcomes.
- EPA should strive to present the results of its health benefits analyses in ways that avoid conveying an unwarranted degree of certainty, such as by rounding to fewer significant digits, increasing the use of graphs, and placing less emphasis on single numbers and more emphasis on ranges.
- EPA should place the results of its health benefits analyses in context by referring not only to absolute numbers of avoided adverse health outcomes but also to total projected numbers of these outcomes and to population sizes. For example, an estimated number of avoided deaths in a future year should be accompanied by projections of the total number of deaths and the population size in that year.
- EPA should begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses. This shift will require specification of probability distributions for major sources of uncertainty. These distributions should be based on available data and expert judgment.
- To obtain expert judgment needed for its expanded primary uncertainty analyses, EPA should rely on internal expertise, as available, and external experts, as needed. In all cases, the experts whose judgments are used should be identified, and the rationales and empirical bases for their judgments described.
- As EPA incorporates additional sources of uncertainty into its

primary analyses, it should analytically determine which uncertainty sources have the greatest influence on the mean and spread of the probability distributions. The uncertainty sources that have the greatest impact on the spread of the distribution should receive high priority for additional research.

- In presenting the probability distribution for each health benefit estimated in the primary analysis, EPA should more clearly identify the sources of uncertainty that are not evaluated in the primary analysis.
- Although the results of the benefits analyses may appear to be less certain, EPA should describe the uncertainty as completely and realistically as possible, recognizing that regulatory action might be necessary in the presence of substantial uncertainty.
- EPA should consider providing preliminary analyses that estimate in current populations the health benefits resulting from hypothetical changes in current levels of emissions. Such preliminary analyses would help EPA develop an idea of the lower bound on the range of uncertainty. These analyses also would have fewer uncertainties than estimates based on projected future population exposures and health outcomes.
- In all stages of the benefits analysis, EPA should justify and clearly describe the assumptions and methods used to estimate health benefits.
- Each benefits analysis should be accompanied by a brief summary, such as 20 to 30 pages in length, that provides all critical elements of the analysis and the results, so that the reader can approximately estimate the benefits on a national level from the information provided.
- To enhance the quality of future regulatory benefits analyses, a standing, independent, technical review panel should advise EPA in the initial stages of its benefits analysis. This panel should have expertise in regulatory options analysis, emissions and exposure assessment, toxicology, epidemiology, risk analysis, biostatistics, and economics and should be appointed with strict attention to avoiding conflict of interest, balancing biases, and ensuring broad representation. The panel should also be supported by permanent technical staff to ensure consistency of reviews over time. EPA should follow the panel's guidance on the need for peer review.
- In reviewing EPA's health benefits analyses, the committee identified several research needs. Some are relevant to improving the scientific basis for estimating the health benefits of further reductions of PM and other air pollutants. These research recommendations are mentioned in the body of the report. Others have to do with the development of improved methods for health benefits analyses in general. The research recommen-

dations include the need for improvements in the following areas: (1) methods for using expert judgment in support of health benefits analyses, (2) methods for characterizing uncertainty surrounding causal interpretation of epidemiological findings, (3) efficiency and characterization of uncertainty in the atmospheric fate and transport models used in support of health benefits analyses, (4) health surveillance systems to characterize morbidity outcomes, and (5) analysis of mixtures as well as the single pollutant.

1

Introduction

Improving public health is the primary goal of air pollution regulation by the U.S. Environmental Protection Agency (EPA). Accordingly, predictions of the type and size of health improvements likely to result from possible regulatory actions are critical components in making decisions about new regulations. The process of estimating health improvements for various regulatory options is known as health benefits analysis. These analyses, often controversial, attempt to quantify changes in the expected number of mortality and morbidity cases likely to result from the proposed regulation. The estimates obtained typically serve as inputs to other analyses that compare the predicted benefits with the regulatory cost.

Given the potentially high costs of air pollution regulations, the public interest is served by using the best possible methods and data to conduct health benefits analyses. In 2000, Congress recognized concerns about the methods used by EPA and emphasized the need for "the most scientifically defensible methodology in estimating health benefits" (U.S. Senate 2000). EPA was directed by Congress to ask the National Academy of Sciences "to conduct a study of this issue and recommend to the agency a common methodology to be followed in all future analyses." In response to that request, the National Research Council (NRC) convened the Committee on Estimating the Health-Risk-Reduction Benefits of Proposed Air Pollution Regulations, which prepared this report.

THE CHARGE TO THE COMMITTEE

The members of the NRC committee were chosen for their expertise in risk assessment, exposure assessment, toxicology, epidemiology, biostatistics, health economics, and air pollution regulations. The committee was asked to accomplish the following tasks:

1. Consider issues important in estimating the health-risk-reduction benefits of air pollution regulations, including the scientific data, risk assessment approaches, populations affected, baseline used, assumptions, analysis of uncertainty, and identification of key indicators of exposure and population health status.
2. Critically review methods used for recent estimates of regulatory health benefits.
3. Identify methods used by federal regulatory agencies and others, recommend standard good-practice guidelines and principles for estimating health benefits, and delineate the data-gathering required to better assess health benefits in the future.
4. Identify approaches to estimating regulatory health benefits when relevant information is limited.
5. Where applicable, recommend areas for further research and monitoring.

The committee was not asked to evaluate methods used to estimate other types of benefits, such as improvements in visibility, resulting from air pollution control. The committee was also not asked to review methods used for economic valuation of health benefits or for regulatory cost analysis, but was asked to consider ways in which health benefits can best be estimated to inform the cost analysis. In addition, the committee was not asked to address whether it is appropriate to compare the benefits analyses of environmental regulation with those of alternative public health and safety measures to determine which regulations should have priority.

THE COMMITTEE'S APPROACH

To accomplish its task, the committee held five meetings from July 2001 to May 2002. Public sessions were held at the first two meetings, during

which the committee heard presentations from representatives of EPA, the U.S. Senate, the Office of Management and Budget (OMB), and other interested parties. The committee reviewed materials submitted by EPA and others, and it reviewed relevant literature on the estimation of health benefits. The committee reviewed in detail EPA's health benefits analyses contained in the regulatory impact assessments (RIAs) of the "Particulate and Ozone National Ambient Air Quality Standards" (EPA 1997a), the "Tier 2 Motor Vehicle Emissions Standards and Gasoline Sulfur Control Requirements" (EPA 1999a), and the "Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements" (EPA 2000a).[1] The committee also reviewed methods used in EPA's prospective analysis of the benefits and costs of the Clean Air Act from 1990 to 2010 (EPA 1999b).

The focus of the EPA analyses reviewed by the committee were the criteria pollutants, particularly ozone and airborne particulate matter (PM). Therefore, the committee spent a considerable amount of time discussing these pollutants, especially PM, and did not address the many issues associated with the analysis of the hazardous air pollutants. However, many of the findings and recommendations of the committee have broad applicability and are not limited to analyses conducted for PM.

REGULATORY CONTEXT

Benefits analysis as a component of cost-benefit analysis (CBA) has played a role in the regulatory process in at least a rudimentary form since the 1930s (NRC 1990).[2] The role of CBA increased substantially in the 1970s when President Ford issued an executive order that required federal agencies to prepare economic analyses of regulations that were predicted to have substantial economic impact. These analyses came to be known as economic impact statements and were submitted to OMB for review. In

[1]RIAs are broader analyses that examine the feasibility and costs of implementing the proposed regulation, as well as the benefits that might be achieved from implementation. A comparison of the costs to an economic valuation of the benefits is also typically included in these analyses.

[2]CBAs compare the economic value of the benefits estimates with the costs of the regulation to determine the net economic benefit of a regulation.

1978, President Carter issued an executive order that established the Regulatory Analysis Review Group, which reviewed, discussed, and provided comments on regulatory analyses for which federal agencies were directed to consider economic consequences of regulatory options, "to solicit public participation, to choose the least burdensome alternative, and to justify the choice" (NRC 1990).

In 1981, President Reagan formally established CBA as an integral part of the evaluation of proposed regulations with the issuance of Executive Order 12291. This executive order required agencies to assess the costs and benefits of proposed "major" regulations and established OMB as the review agency for these analyses. The executive order also indicated that benefits of the regulation must outweigh the costs and that the preferred option must maximize net benefits and incur the least cost.

President Clinton reaffirmed the importance of conducting CBAs of proposed regulatory actions with the issuance of Executive Order 12866 in 1993. Specifically, this executive order included the following statements:

> In deciding whether and how to regulate, agencies should assess all costs and benefits of available regulatory alternatives, including the alternative of not regulating. Costs and benefits shall be understood to include both quantifiable measures . . . and qualitative measures of costs and benefits that are difficult to quantify, but nevertheless essential to consider.

Executive Order 12866 further required agencies to conduct CBAs for all "significant" regulations. With the change in terminology from "major" to "significant," Executive Order 12866 expanded the scope of regulations subject to OMB review and effectively broadened OMB's power "to review and request revisions to all regulatory proposals to ensure their consistency with the regulatory principles contained in the Order" (EPA1999c). An important difference between Executive Orders 12291 and 12866 is that Executive Order 12866 requires the benefits of the regulations to "justify" rather than "outweigh" the costs of the regulatory action.

The administration of President George W. Bush has not issued an executive order that amends or revokes Executive Order 12866, which therefore remains the effective requirement for regulatory planning and review. However, OMB under President Bush has issued a memorandum that "describes the general principles and procedures that will be applied by

OMB in the implementation of E.O. 12866 and related statutory and executive authority" (OMB 2001). The memorandum re-asserts that benefits and costs must be assessed in RIAs prepared for economically significant rulemakings and emphasizes the importance of using scientific data that meet a basic quality standard and of conducting rigorous peer review of RIAs and supporting technical documents.

In addition to the overarching requirements mandated in executive orders, Congress has imposed statutory and administrative requirements to conduct CBAs under various acts. The Clean Air Act Amendments of 1990 require EPA to assess periodically the costs and benefits of the Clean Air Act. A retrospective analysis from 1970 to 1990 was published in 1997 (EPA 1997b), and a prospective analysis from 1990 to 2010 was published in 1999 (EPA 1999b).

The analysis of costs and benefits is not necessarily the direct basis for regulatory decisions. The basis for decision-making is specified in individual mandates. For example, under the Clean Air Act, costs are not to be used directly to determine standards for primary air pollutants, although costs can be used to determine the nature and timing of implementing measures needed to attain the standards. CBAs, or more generally RIAs, are intended to inform the government and private parties about the nature and extent of changes in health and the environment and the associated costs that are expected to result from specific regulatory actions. The methods used to predict the changes in health resulting from a regulatory action are the topic of this report. The cost component is not addressed further.

CRITICAL STEPS OF A HEALTH BENEFITS ANALYSIS

According to current guidelines and practices, a health benefits analysis should define conditions with and without the proposed policy implemented and ultimately estimate the differences in health outcomes between those two conditions (OMB 2000). Health benefits analysis can be characterized generally by the following steps (see Chapter 6, Figure 6-1):

- ***Defining the proposed regulation***. Benefits analysis evaluates an air pollution regulation proposed to correct or reduce a perceived environmental problem. For each regulation considered, the scope, timing, and implementation must be defined. Specifically, the regulation must define to

whom it will apply, when it will begin, how long it will last, and what the schedule of compliance will be. For the analysis to be most useful and to meet OMB guidelines, several regulatory options (for example, different levels of stringency requirements or different compliance schedules) should be considered.

- ***Establishing the boundaries of the analysis***. The boundaries of the analysis must be clearly defined. For example, the analyst must specify the period over which the regulation will be evaluated, the intervals at which the benefits will be evaluated, the pollutants that will be the focus of the analysis, and the spatial resolution of the model.
- ***Defining the regulatory baseline***. Conditions without the regulation—the regulatory baseline—must be described. This process requires a description of other air pollution regulations that are assumed to be in force and the extent to which industry and consumers are expected to comply with the regulations. Assumptions about economic activity, especially in highly polluting sectors, such as transportation and electric power generation, must be articulated. In addition, assumptions about baseline health of the population must be described when relative risk models are used to estimate health benefits.
- ***Estimating changes in pollutant emissions***. Once the analysis has been structured, the change in pollutant emissions that is anticipated to result from the regulation is predicted over time and space. The change is measured from the regulatory baseline.
- ***Estimating changes in ambient air pollutant concentrations***.[3] To allow calculation of the health benefits, the changes in pollutant emissions must be translated into changes in ambient air concentrations and should take account of factors that might affect exposure, when possible.
- ***Estimating changes in human health outcomes***. Two steps are typically involved in estimating changes in morbidity and mortality. First, the health outcomes and the appropriate concentration-response functions are determined from epidemiological studies, human clinical studies, or animal toxicity studies. Then, the concentration-response function is applied to the

[3]The committee acknowledges that changes in exposure may be estimated, which would then require use of exposure-response functions to estimate health outcomes in the next step.

relevant populations using the baseline health assumptions and the changes in ambient concentrations calculated in the previous step. This second step provides estimates of changes in health outcomes.

EPA followed this basic approach; however, as a final step in EPA's health benefits analyses, the changes in health outcomes were typically translated into monetary values for comparison with regulatory costs. As noted previously, the committee was not asked to assess methods used for economic valuation of health outcomes and, therefore, does not go further in its assessments than the quantification of the changes in health outcomes.

Integral to each step described above is the assessment of uncertainty. The uncertainty of each component should be carried through the analysis, and an assessment of overall uncertainty should be provided with the final benefits estimates.

GUIDANCE

Few guidance documents are available that specifically address the conduct of a health benefits analysis. Documents that contain some guidance on aspects of these analyses typically focus on broader regulatory analyses, such as RIAs or economic analyses. Relevant documents issued by EPA, OMB, the World Health Organization (WHO), and the NRC are cited, and pertinent information from these documents is summarized in the following sections. Although other agencies clearly conduct regulatory analyses that include benefits analyses, their guidelines (if written) do not appear to be available in the publicly accessible literature.

U.S. Environmental Protection Agency

EPA's Office of Air Quality Planning and Standards (OAQPS) in the Office of Air and Radiation has issued guidance on conducting economic analyses, which include assessing health benefits (EPA 1999c). The document provides guidance on three aspects of benefits analysis relevant to the committee's task—defining the time period of analysis, establishing the baseline for analysis, and analyzing uncertainty. Regarding the time period

for the analysis, OAQPS notes that there is often no obvious basis for establishing the time period for the analysis but continues that the analyst must "capture any specific identified changes expected to occur over time."

Regarding the baseline for the analysis, OAQPS provides several overarching recommendations, including the selection of realistic assumptions regarding future conditions; the use of a consistent baseline throughout the analysis, although alternative baselines may be considered; and the clear identification of all assumptions made in selecting, specifying, and measuring the baseline. OAQPS identifies several specific components of developing a baseline and provides the following advice:

- ***Forecasting baseline economic activity***. Three approaches are presented, which include (1) projection of production changes over time, (2) estimation of current production and application to future years, and (3) estimation of production for a representative year and application to future years.
- ***Assessing compliance with existing regulations***. Most analyses should assume full compliance with existing regulations. Exceptions include analyses conducted for regulations intended to solve problems with compliance. Those analyses should assume the actual compliance estimates for the baseline scenario.
- ***Anticipating future regulatory actions***. Analyses should assume that other regulations that have been or will be promulgated by the effective date of the regulation being considered are in full effect for purposes of the given analysis.
- ***Anticipating nonregulatory factors***. Although nonregulatory factors are important (for example, changes in industrial behavior that affect pollutant emissions but are unrelated to regulatory actions), they are generally not included in the baseline because of time and resource limitations. However, those that might affect the baseline should at least be mentioned qualitatively.
- ***Establishing a starting date for the baseline***. The starting date for the baseline is determined partly by the actions taken by the regulated community. If actions taken by the regulated community prior to promulgation would continue with or without promulgation of the regulation, the baseline would start on the date of promulgation or implementation (that is, the actions taken would be included in the baseline because they would be unrelated to the rule-making). However, if actions taken by the regulated

community would cease if the regulation were not promulgated, the baseline would start on the date of public notification (that is, the actions taken would be pre-emptive and would not be included in the baseline).

Regarding the analysis of uncertainty, OAQPS states that there are three sources of uncertainty—input, model, and estimation—and that the full range of uncertainty should be made transparent in the analysis. Although five methods to analyze uncertainty are listed (scenario analysis, Delphi methods, sensitivity analysis, meta-analysis, and Monte Carlo and probabilistic models), OAQPS states that "for analyses in which benefits unambiguously exceed costs, a sensitivity analysis should be adequate." The committee notes that determining the likelihood of benefits unambiguously exceeding costs requires an uncertainty analysis and not simply sensitivity analyses.

The OAQPS guidelines also emphasize clear communication of the results of the analysis. They suggest using clear and transparent language, identifying data sources and assumptions, describing the modeling and the uncertainty, presenting alternatives in comparable metrics, and clearly identifying nonmonetized and unquantified effects.

More recently, EPA published guidelines for economic analyses prepared by the Economic Consistency Workshop under the direction of the Regulatory Policy Council (EPA 2000b). Because these guidelines were developed for use by all offices within EPA, they tend to provide general guidance and do not address specific aspects of analyses of air pollution regulations. For example, benefits analysis is framed as a three-step approach: (1) identifying types of benefits that might be affected by the regulation, (2) quantifying the benefits, and (3) valuing the benefits. The guidance given for each step is extremely general. For step 1, the guidelines suggest investigating policy options and pollutant effects; given various policy options, evaluating changes in pollutant effects; and identifying those effects most likely to have the most substantial impact on the benefits analysis. For step 2, the guidelines recommend collaborating with experts from different fields, using outcomes from risk assessment that are amenable to economic valuation, and describing qualitatively unquantifiable effects. For step 3, the guidelines suggest using different methods to value benefits and identifying sources of valuation estimates and confidence in the sources.

The critical importance of the selection of the baseline is emphasized, and the information presented on defining a baseline is consistent with that

of OAQPS discussed above. General principles highlighted in the guidelines include focusing on key issues (those affecting policy decisions), considering changes in behavior that might result from changes in environmental quality, avoiding double-counting of benefits, and clearly describing uncertainty and nonmonetized effects.

Throughout the document, the analysis of uncertainty is emphasized. The guidelines state that an analysis should "present outcomes or conclusions based on expected or most plausible values; provide descriptions of all known key assumptions, biases, and omissions; perform sensitivity analysis on key assumptions; and justify the assumptions used in the sensitivity analysis." Furthermore, on the basis of the sensitivity analyses of key parameters, those points at which net benefits switch from positive to negative (switch points) should be clearly identified. Other considerations include presentation of plausible upper- and lower-bound estimates of net benefits and identification of the most likely estimate.

Office of Management and Budget

OMB released a memorandum in 2000 that provided "Guidelines to Standardize Measures of Costs and Benefits and the Format of Accounting Statements" (OMB 2000). These guidelines were based on an OMB document released in 1996 that described "best practices" for conducting economic analyses required under Executive Order 12866 (OMB 1996). Similar to the EPA guidelines, the OMB guidelines provide general information on conducting an economic analysis. The 2000 OMB guidelines state that an analyst "cannot write a good regulatory analysis according to a formula. The preparation of high-quality analysis requires competent professional judgment."

The 2000 OMB guidelines contain some general considerations relevant to this committee's charge. The guidelines note that the problem must be clearly articulated and the need for regulatory action justified. Several reasonable regulatory options should be evaluated. Specifically, the analysis should critically evaluate not only the preferred option but also more and less stringent options—one that would yield more benefits presumably at a higher cost and one that would yield fewer benefits presumably at a lower cost. One exception to the inclusion of a more stringent option would be when the preferred option exceeds the limits of technical feasibility.

The OMB guidelines state that the baseline should be selected so that it is the "best assessment of the way the world would look absent the proposed regulation." Although alternative baselines may be used, the benefits and costs should always be evaluated against the same baseline. Factors to consider when defining the baseline include market changes, regulations or regulatory changes that might affect benefit and cost estimates, and compliance rates with regulations. The guidelines recommend that the analyst assume full compliance unless otherwise indicated.

According to the OMB guidelines, the presentation of the results of the benefits analysis should include a list of monetized benefits that indicates type and timing of benefits expressed in constant, undiscounted dollars, a list of nonmonetized but quantified benefits that indicates timing, and a list of nonmonetized, unquantified benefits that is ranked by expected magnitude. The data or studies on which the estimates are based should be identified. Where applicable, the analysis should explain why certain benefits cannot be monetized or quantified. Furthermore, the benefits estimates should be presented in a way that clearly reflects the degree of uncertainty in the estimates. Probability distributions should be presented with upper- and lower-percentiles and central-tendency values. If probability distributions cannot be generated, sensitivity analyses of plausible alternative assumptions should be conducted. Any analysis that indicates a change in the preferred option or a substantial change in the net benefits should be critically evaluated.

Other considerations noted in the OMB guidelines include avoiding double-counting when estimating benefits, analyzing effects on different groups, identifying any negative effects of regulatory options, and evaluating the sensitivity of estimates to assumptions. The guidelines provide information on valuing benefits, comparing costs and benefits, choosing discount rates, and conducting the cost analysis; however, because these issues are not relevant to the committee's task, they are not discussed further.

World Health Organization

WHO recently released a report that summarizes the findings of a working group that evaluated various aspects of health-impact assessments of air pollution (WHO 2001). The overall objective of the group was "to review the available methods for health impact assessment of air pollution

and to agree upon common approaches." The group focused primarily on the selection of health outcomes and the use of epidemiological data in the assessments. Many of the issues that the WHO working group considered were similar or identical to those considered by this committee. Significant findings relevant to this committee's work are highlighted in this section.

The WHO working group acknowledged that many acute and chronic health effects, including death, have been associated with exposure to air pollution. Regarding mortality, the group debated the use of time-series studies (studies that can provide estimates of premature death due to recent exposure) versus cohort studies (studies that evaluate mortality in a specific population over a period of years) and concluded that cohort studies should be used in air-pollution health-impact assessments because they provide "the most complete estimates of both attributable numbers of deaths and average reductions in life-span attributable to air pollution." However, time-series studies are valuable because they can indicate the adverse health effects of air pollution in specific locations; quantify effects of short-term fluctuations in air pollution; and provide information that can help to identify toxic components of air pollution, support associations between air pollution exposure and chronic health effects, and identify factors that modify the effects of air pollution.

The WHO report stated that the impact of air pollution on all-cause mortality should be assessed, as well as that on cause-specific mortality for the following conditions: cardiovascular disease, chronic nonmalignant respiratory disease, lung cancer, and age-specific deaths, particularly for younger and older populations. The group noted that the effects of air pollution on mortality in sensitive subpopulations should be better estimated and stressed that care must be taken when transferring mortality rates from the study population (the population evaluated in the scientific literature) to the target population (the population characterized in the impact assessment).

Regarding morbidity, the group recommended that all relevant health outcomes be considered in the planning stages of the analysis but not necessarily included in the final analysis and provided a list of potentially relevant health outcomes to consider (see Table 1-1). The group noted that the list might need to be expanded if the impacts of hazardous air pollutants are being evaluated. For example, neurological outcomes should be considered when evaluating lead exposure, outcomes of leukemia and non-Hodgkins lymphoma should be considered when evaluating benzene exposure,

TABLE 1-1 Potentially Relevant Health Outcomes for Air-Pollution Health-Impact Assessment

Acute Outcomes	Chronic Disease Outcomes	Reproductive Outcomes
Daily mortality	Mortality in infants and adults from chronic cardiorespiratory disease	Pregnancy complications, including fetal death
Respiratory hospital admissions	Chronic respiratory disease (asthma, chronic obstructive pulmonary disease, chronic pathological changes) incidence and prevalence	Low birth weight
Cardiovascular hospital admissions	Chronic change in physiological function	Preterm delivery
Emergency room visits for respiratory and cardiac problems	Lung cancer	
Primary-care visits for respiratory and cardiac conditions	Chronic cardiovascular disease	
Use of respiratory and cardiovascular medicines		
Days of restricted activity		
Work absenteeism		
School days missed		
Self-medication		
Avoidance behavior		
Acute symptoms		
Physiological function, such as lung function		

Source: Adapted from WHO 2001.

and the outcome of hematopoietic cancer should be considered when evaluating butadiene exposure. If possible, the impacts on these outcomes should be expressed by age and sex.

The WHO group emphasized the need to evaluate the transferability of the risk estimates of the study population to the target population. Factors that should be considered include the mixture of pollutants to which each population is exposed and each population's baseline health status. Assumptions should be clearly articulated and assessed and justifications provided for transferability of the results from the study population to the target population. If possible, uncertainties should be quantified. The analyst might

need to consider using multisite analyses or meta-analyses rather than one particular study.

When the study and target populations differ, "health impact assessments should strive to characterize exposure in the target population to mirror as closely as possible exposure in the study providing the effect estimate." Care must be exercised when extrapolating beyond the concentration range of the study used to base estimates. Factors that should be considered in the analysis include differences between study and target locations with respect to pollutant sources, pollutant mix, variation in time and space of the pollutant mix, locations of the monitors, and assumptions used to determine population average exposure, such as amount of time spent indoors, work habits, and use of air conditioners.

The group noted that the effects attributed to a specific pollutant in epidemiological studies should be viewed as the effects resulting from exposure to pollutant mixtures emitted by particular sources. Therefore, effect estimates of single pollutants should not be added when derived from single-pollutant statistical models unless they can be confidently shown to act independently on health. Although the current focus is on the health effects of exposure to PM, other pollutants, such as sulfur dioxide, should not be disregarded. The group stated that more research is needed to evaluate the relationship between health impacts and pollutant mixtures.

The group emphasized that uncertainties in the analysis should be explicitly stated and quantitatively evaluated. Rigorous sensitivity analyses should be conducted to determine how the results are affected by deviations in key assumptions (for example, how mortality-impact estimates vary by exposure level). Overall, the group emphasized that the results should be presented with "sufficient detail with regard to various health endpoints, population strata (e.g., age, sex, race, social class), and pollutants to allow policy analysts maximum latitude and flexibility in applying them to regulatory decision-making."

The group provided several recommendations for additional research. Research topics considered to be of primary importance "to improve the scope and reliability of health impact analysis" included (1) quantification of chronic effects of air pollution, (2) identification and evaluation of factors that modify the effects of air pollution and result in the observed variation in response between populations, and (3) quantification of all health effects resulting from exposure to air pollution and better quantification of those that have been identified.

National Research Council

In response to an EPA request in 1986, NRC convened the Steering Committee on Valuing Health Risks, Costs, and Benefits for Environmental Decisions to help EPA identify "some sound scientific basis for approaching the problem of valuing risks" (NRC 1990). The steering committee conducted a conference in 1987 and prepared a conference report that included issue papers prepared for the conference and conclusions and recommendations of the steering committee based on the conference discussions. The conference focused primarily on the legal, political, philosophical, and ethical issues associated with CBA, particularly the valuation techniques, and not on the methodological issues on how to conduct benefits analysis. However, the recommendations made by the steering committee are relevant here. Specifically, the steering committee emphasized that CBA should be considered a "set of information-gathering and organizing tools" rather than a "decision-making mechanism itself," suggested that the appropriate analytical methods and techniques be matched to the given problem (that is, no single analytical technique is suitable to evaluate all regulatory decisions), and encouraged the use of a formal peer-review process for these analyses.

ORGANIZATION OF REPORT

The remainder of this report is divided into five chapters. Chapter 2 contains brief summaries of EPA case studies reviewed by the committee. Chapters 3, 4, and 5 address specific aspects of health benefits analyses. Chapter 3 discusses issues in selecting regulatory options and effects to evaluate, defining the time frame of the analysis, and making assumptions about conditions with and without the regulation implemented. Chapter 4 addresses issues related to exposure estimates, identification of health outcomes, and selection and use of the concentration-response functions that link exposure to health. Chapter 5 presents issues associated with the analysis of uncertainty. Chapter 6 places health benefits analyses in the context of cost-benefit and cost-effectiveness analyses and discusses how the results of benefits analyses should be presented to be compatible with those analyses. Chapter 6 also addresses issues of quality assurance and communication of the methods and results.

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2

Health Benefits Analyses: EPA Case Studies

The committee reviewed the health benefits analyses contained in the regulatory impact assessments (RIAs) prepared for the following EPA rulemakings: (1) "Particulate Matter and Ozone National Ambient Air Quality Standards" (EPA 1997), (2) "Tier 2 Motor Vehicle Emissions Standards and Gasoline Sulfur Control Requirements" (EPA 1999a), and (3) "Heavy Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements" (EPA 2000a). The committee also reviewed the health benefits analysis completed for the EPA prospective analysis of the benefits and costs of the 1990 Clean Air Act Amendments (CAAA) (EPA 1999b), which used methods similar to those used in the other EPA analyses reviewed by the committee. Critical elements of the analyses are summarized in Tables 2-1 and 2-5, and the sections that follow provide a brief summary of the EPA analyses to aid the reader in understanding the critiques in the chapters that follow. Although the analyses provide methods and estimates for welfare benefits (all benefits other than health, such as improvements in visibility), the focus of the following discussion is human health benefits.

PARTICULATE MATTER AND OZONE NATIONAL AMBIENT AIR QUALITY STANDARDS

EPA is required by the Clean Air Act (CAA) to review National Ambient Air Quality Standards (NAAQS) at least once every 5 years and

to revise standards when necessary to protect the public health and the environment (EPA 1997). By the mid-1990s, scientific evidence suggested that the standards for both particulate matter (PM) and ozone needed revision. Accordingly, EPA proposed new PM and ozone NAAQS and released an RIA evaluating the benefits and costs of the proposed standards (EPA 1997). The proposed PM and ozone standards were evaluated in the same RIA because of the similarities in precursors, sources, atmospheric residence times, and atmospheric chemistry. The RIA also included an assessment of a proposed regional haze rule; however, the committee focused on the health benefits analyses conducted for the PM and ozone standards because they were more closely related to its task.

The proposed standards that were evaluated were (1) an annual mean $PM_{2.5}$ standard of 15 micrograms per cubic meter ($\mu g/m^3$) and a 98th percentile 24-hour (hr) average of 65 $\mu g/m^3$ in conjunction with an annual mean PM_{10} standard of 50 $\mu g/m^3$ and 99th percentile 24-hr average of 150 $\mu g/m^3$, and (2) an 8-hr ozone standard of 0.08 parts per million (ppm) based on the fourth highest average daily maximum.[1] Two alternative standards were also evaluated for $PM_{2.5}$ and ozone. EPA evaluated a partial-attainment scenario that accounted for areas that would not be able to meet the proposed standards or alternatives based on current control technologies and a full-attainment scenario that assumed no residual nonattainment. EPA noted that more uncertainty was associated with the estimates for the full-attainment scenario because attainment was based on development of new technologies. The benefits were estimated in the year 2010 because EPA assumed that the majority of CAA-mandated controls would be achieved by that date.

EPA used a six-step approach for estimating the benefits for the proposed and alternative PM and ozone standards. In the first step, EPA developed an emissions inventory for the year 2010. The inventory included estimates for volatile organic compounds (VOCs), nitrogen oxides (NO_x), sulfur dioxide (SO_2), secondary organic aerosols, $PM_{2.5}$, PM_{10}, and ammonia (NH_3). To construct the 2010 inventory, EPA first generated a 1990 emissions inventory using source-specific emissions factors and activity levels, such as fuel consumed by electric utilities or miles traveled by motor vehicles. The 2010 emissions inventory was then projected using the 1990

[1] PM_{10} refers to PM with an aerodynamic diameter of 10 μm or less, and $PM_{2.5}$ refers to PM with an aerodynamic diameter of 2.5 μm or less.

emissions inventory, sector-specific growth assumptions, and source-specific assumptions regarding future CAA-mandated controls expected to be achieved by 2010.

In the second step, county-level baseline air-quality data for the continental United States were generated. For PM, a source-receptor matrix was first generated using the phase 2 climatological regional dispersion model (CRDM). Because the model was shown to overestimate the contribution of fugitive dust to fine PM, the source-receptor matrix was adjusted, and monitoring data were used to calibrate the matrix. Baseline annual mean PM_{10} and $PM_{2.5}$ estimates for 2010 were then generated using the 2010 emissions data and the source-receptor matrix. PM estimates for nonmonitored counties were generated on the basis of the more complete data sets for the monitored counties. Peak-to-mean ratios were used to generate 24-hr averages. For ozone, a regional oxidant modeling (ROM) extrapolation method was used to generate county-level baseline air-quality data for ozone. Ozone air-quality monitoring data from 1990 and ROM air-quality modeling results for 2007 were used to generate ozone air-quality data for 2007. The data for 2007 were then extrapolated using 2010 emissions data and ozone modeling and monitoring data to give 2010 baseline ozone air-quality data. Data for nonmonitored counties were generated by interpolating data from surrounding monitored counties, assuming that the entire county population experienced the air pollution concentration estimated at the geographic center (or centroid) of the county.

In the third step, EPA used the PM and ozone baseline air-quality data to identify counties that would exceed the proposed or alternative standards. In the fourth step, EPA selected control strategies to implement in the nonattainment counties and then estimated the potential costs and economic impacts of the proposed and alternative standards.

In the fifth step, EPA estimated the post-control air-quality data on the basis of the control strategies selected in step four. For the partial-attainment scenario, EPA used the source-receptor matrix to estimate PM air-quality data and a quadratic rollback procedure to estimate ozone air-quality data. For the full-attainment scenario, a proportional and a quadratic rollback procedure were used to estimate PM and ozone air quality, respectively.[2]

[2]Rollback procedures scale an exposure estimate by the changes modeled for the emissions estimates. Therefore, proportional rollback assumes that concentra-

In the sixth step, EPA estimated the human health benefits resulting from implementation of the proposed or alternative standards for each county in the continental United States and then summed across counties to give the national estimates. EPA estimated the reductions in the incidences of a number of human health effects (see Table 2-1). Although EPA indicated that a few additional health effects were quantified, the results were not included in the analysis. Human health effects that could not be quantified but were associated with exposure to the pollutants were also listed. The human health benefits were estimated on the basis of the differences in pre- and post-control air-quality data and quantitative concentration-response functions derived from the epidemiological literature. The Pope et al. (1995) study was used to determine mortality reductions resulting from PM reductions. For ozone, a meta-analysis of nine epidemiological studies was used to determine mortality reductions resulting from ozone decreases. Clinical studies were used to support data for effects of ozone exposure. One important assumption made in this analysis was that the health benefits were realized in the year in which the exposure reductions occurred. The benefits were monetized to derive a total benefits estimate that could be compared with the cost estimate.

The analytical uncertainty was partially reflected by providing a plausible range of benefits estimates.[3] For the high-end estimates, an effects threshold of 12 $\mu g/m^3$ was assumed for $PM_{2.5}$-related long-term mortality, mortality benefits (deaths avoided) were estimated for reductions in ozone concentration using a meta-analysis of nine epidemiological studies, ancillary PM benefits were included in the ozone benefits estimates,[4] and an approach based on the value of a statistical life (VSL) was used to monetize the mortality benefits. For the low-end estimate, an effects threshold of 15 $\mu g/m^3$ was assumed for all $PM_{2.5}$-related health outcomes, no mortality benefits were estimated for reductions in ozone concentration, no ancillary PM benefits were included in the ozone benefits analysis, and an approach based on the value of a statistical life year (VSLY) was used to value the

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tions and emissions are proportionally related, and a quadratic rollback assumes a quadratic relationship between emissions and concentrations.

[3]EPA noted that the plausible ranges provided were not equivalent to upper and lower statistical confidence bounds.

[4]Reduction in precursors resulting from measures to control ozone formation will also result in reduction of PM. The benefits derived from the reduction in PM in this case are referred to as ancillary PM benefits.

TABLE 2-1 Elements of Selected EPA Benefits Analyses

Parameters	PM and Ozone NAAQS (EPA 1997)[a]	Tier 2 Emissions and Gasoline Sulfur Standards (EPA 1999a)	Heavy-Duty Engine and Diesel-Fuel Standards (EPA 2000a)
Year in which benefits evaluated (justification)	2010 (anticipated date when standards will be implemented)	2030 (anticipated date when fleet will be fully turned over)	2030 (anticipated date when fleet will be fully turned over)
Scenarios	Evaluated partial and full attainment of standards for three PM and ozone alternatives	Evaluated conditions with and without the standards being proposed	Evaluated conditions with and without the standards being proposed
Pollutants modeled and methods used for air-quality modeling for benefits analysis	Ozone – quadratic air-quality rollback procedures (partial and full attainment scenarios) based primarily on regional oxidant model and monitoring data PM – source-receptor matrix based on climatological regional dispersion model (partial-attainment scenario); proportional rollback procedure (full-attainment scenario)	Ozone – regional-scale version of the urban airshed model PM – source-receptor matrix based on the climatological regional dispersion model	Ozone – regional-scale version of the urban airshed model-variable grid (note: modeling results for western U.S. not used in benefits analysis) PM – national-scale version of the regulatory modeling system for aerosols and deposition
Geographic scale of models used to estimate air quality	Ozone – 18-km grid squares or county level (size varies) PM – county level (size varies)	Ozone – 12 or 36-km grid squares for eastern U.S. and 56-km grid squares for western U.S. PM – county level (size varies)	Ozone – 12 or 36-km grid squares for eastern U.S. (note: western U.S. not included in analysis) PM – 36-km grid squares

Health outcomes quantified and monetized[b]	Ozone – mortality; hospital admissions (all respiratory, pneumonia, COPD, and emergency dept. visits for asthma); acute respiratory symptoms (asthma attacks and minor restricted-activity days); mortality from air toxics PM – mortality (short- and long-term); bronchitis (chronic and acute); hospital admissions (all respiratory [all respiratory, pneumonia, and COPD], congestive heart failure, and ischemic heart disease), lower and upper (shortness of breath, asthma attacks) respiratory symptoms; work-loss days; minor restricted-activity days	Ozone – chronic asthma; minor restricted-activity days and acute respiratory symptoms; hospital admissions (respiratory and cardiovascular); emergency room visits for asthma PM – premature mortality; bronchitis (chronic and acute); hospital admissions (respiratory and cardiovascular); emergency room visits for asthma; lower and upper respiratory illness; shortness of breath; minor restricted-activity days and acute respiratory symptoms; work-loss days	Ozone – minor restricted-activity days; hospital admissions (respiratory and cardiovascular); emergency room visits for asthma; asthma attacks PM – premature mortality; bronchitis (acute and chronic); hospital admissions (respiratory and cardiovascular); emergency room visits for asthma; asthma attacks; lower and upper respiratory illness; minor restricted-activity days; work-loss days
Concentration-response function used for primary estimates of mortality benefits	Pope et al. (1995)	Pope et al. (1995)	Krewski et al. (2000), a re-analysis of the Pope et al. (1995) study
Threshold assumptions	High-end estimate of benefits assumed 12 $\mu g/m^3$ mean threshold for $PM_{2.5}$-related long-term mortality; low-end estimate of benefits assumed 15 $\mu g/m^3$ threshold for all PM-related health outcomes	No thresholds above background concentrations assumed for modeled health effects	No thresholds above background concentrations assumed for modeled health effects

(Continued)

TABLE 2-1 *Continued*

Parameters	PM and Ozone NAAQS (EPA 1997)[a]	Tier 2 Emissions and Gasoline Sulfur Standards (EPA 1999a)	Heavy-Duty Engine and Diesel-Fuel Standards (EPA 2000a)
Lag-time assumptions	No lag times assumed; benefits assumed to occur in the year that exposure is reduced	5-year lag structure assumed for PM-related premature deaths with 25% in years 1 and 2 and 16.7% in years 3, 4, and 5	5-year lag structure assumed for PM-related premature deaths with 25% in years 1 and 2 and 16.7% in years 3, 4, and 5
Quantification of uncertainty	1. Presented plausible range of benefits estimates calculated by varying key assumptions in analysis; estimates are not upper and lower statistical bounds 2. Conducted several sensitivity analyses, such as one to evaluate using a proportional rollback procedure to estimate ozone air quality	1. Provided alternative calculations of primary benefit estimates by varying key assumptions 2. Conducted sensitivity analysis for alternative lag structures and PM threshold assumptions	1. Provided alternative calculations of primary benefit estimates by varying key assumptions 2. Conducted sensitivity analysis for alternative lag structures and PM threshold assumptions
Study populations evaluated for health outcomes	Majority of benefits appear to be estimated for adult population; however, PM- and ozone-related "all respiratory" hospital admissions were estimated for elderly adults (over 65 yr)	Majority of benefits estimated for adult populations; PM-related acute bronchitis estimated for children aged 8 to12, lower respiratory symptoms estimated for children aged 7 to 14, upper respiratory symptoms estimated for children aged 9 to 11, and shortness of breath estimated for African American children aged 7 to 12; ozone-related chronic asthma estimated for adult males	Majority of benefits estimated for adult populations; PM-related cardiovascular, pneumonia, and COPD hospital admissions estimated for elderly population (over 64 yr) and PM-related acute bronchitis and upper and lower respiratory symptoms estimated for children (ages between 7 and 14 yr) with upper respiratory symptoms estimated specifically for asthmatic children

[a]This regulatory impact assessment (RIA) also included an evaluation of a proposed regional haze rule; however, because the PM and ozone analyses were more closely related to the committee's task, the committee focused on these analyses.
[b]Many health effects were listed as unquantified for ozone and PM in all three RIAs. Unquantified health effects were also listed for carbon monoxide (CO) and hazardous air pollutants in Tier 2 rule-making, and for sulfur dioxide, nitrogen oxides, CO, and nonmethane hydrocarbons in heavy-duty engine and diesel-fuel rule-making.
Abbreviations: PM, particulate matter; NAAQS, National Ambient Air Quality Standards; COPD, chronic obstructive pulmonary disease.

mortality benefits. EPA also indicated that several sensitivity analyses of key assumptions were conducted. One such analysis investigated alternative rollback procedures to estimate post-control ozone air quality. EPA also qualitatively discussed uncertainties relevant to various phases of the analyses and provided an opinion on whether the uncertainty would lead to an overestimate (positive bias) or an underestimate (negative bias) of results.

Annual benefits (avoided cases of morbidity and mortality) of the proposed ozone and $PM_{2.5}$ standards are shown in Table 2-2 for the partial-attainment scenario in 2010. Annual benefits of the proposed ozone standard are incremental to the current ozone standard, and those of the proposed $PM_{2.5}$ standard are incremental to the current ozone and PM_{10} standards. Monetized values are also provided.

TIER 2 MOTOR VEHICLE EMISSIONS STANDARDS AND GASOLINE SULFUR CONTROL REQUIREMENTS

The Tier 2 Motor Vehicle Emissions Standards and Gasoline Sulfur Control Requirements Rule (Tier 2 rule) sets new federal motor-vehicle emissions standards and establishes limits on sulfur concentrations in gasoline (EPA 1999a). The emissions standards apply to all passenger cars, light trucks, and medium-duty passenger vehicles, which include sport utility vehicles (SUVs) and passenger vans. The standards are designed to limit emissions, such as NO_x, that contribute to ozone and PM formation and, therefore, will help states meet the ozone and PM NAAQS. Full compliance with the emissions standards should be achieved by 2009, with phase-in periods dependent on vehicle class. Full compliance with the gasoline sulfur limits should be achieved by 2006.

The benefits of the rule were assessed for the year 2030, when full implementation is expected through turnover of the existing vehicle fleet. EPA used a four-step approach for the Tier 2 benefits analysis. First, reductions in motor-vehicle emissions anticipated from the standards were used to estimate the impact on emissions inventories of NO_x, SO_2, nonmethane hydrocarbons (NMHCs), $PM_{2.5}$, PM_{10}, and NH_3 for the continental United States in 2030.[5] Compliance assumptions were not clearly stated in the discussion of the benefits analysis.

[5]The RIA appeared to equate nonmethane hydrocarbons (NMHCs) with VOCs as this class of compounds was later listed instead of NMHCs.

TABLE 2-2 Annual Benefits (Avoided Cases of Morbidity and Mortality and Monetized Value) of the Proposed Ozone and $PM_{2.5}$ Standards for the Partial-Attainment Scenario in 2010

Health Outcome	Avoided Cases (Low- to High-End Estimates)	Monetized Value (1990$ in millions)	Ancillary PM Benefits Included in Ozone High-End Estimate[a]
PM-Related Outcomes			
Mortality	3,300-15,600[b]	1,800-75,100	80 ($400); 250 ($1,210)[c]
Chronic bronchitis	45,000-75,000	11,700-19,400	530 ($140)
Hospital admissions			
All respiratory illnesses (all ages)	3,600-5,700	42-72	90 ($1)
Congestive heart failure	1,200-2,100	30-35	20 ($0)
Ischemic heart disease	1,200-2,400	30-49	20 ($0)
Acute bronchitis	12,000-20,000	1	400 ($0)
Lower respiratory symptoms	179,000-299,000	2-4	4,670 ($0)
Upper respiratory symptoms	36,000-60,000	1	430 ($0)
Work-loss days	1,900,000-3,148,000	156-261	50,440 ($4)
Minor restricted-activity days	15,697,000-26,128,000	600-1,000	420,300 ($16)
Ozone-Related Outcomes			
Mortality	0-80	0-380	—
Hospital admissions			
All respiratory illnesses (all ages)	300[d]	4	—
Acute respiratory symptoms (any of 19)	29,840[d]	1	—
Mortality from air toxics	1[d]	6	—

[a]Ancillary PM benefits are those benefits derived from PM reductions due ozone control measures. Avoided cases are provided with monetary estimates provided in parentheses in millions of 1990 dollars.

[b]Estimates were designated as mortality estimates for short-term exposure; however, the low-end estimate represents short-term exposure and the 15 $\mu g/m^3$ threshold, and the high-end estimate represents long-term exposure and the 12 $\mu g/m^3$ threshold (B. Hubbell, EPA, personal communication, June 4, 2002).

[c]Mortality estimate for short-term exposure; mortality estimate for long-term exposure.

[d]Range not provided.

Source: Data from EPA 1997.

Second, air-quality modeling of ozone and PM was conducted for a base year (1996) and two future scenarios: 2030 with and without the standards implemented. Ambient ozone was modeled using the urban airshed model variable (UAM-V). Monitoring data from 1996 were used to calibrate the model, and data for nonmonitored areas were generated by interpolating values from nearby monitoring sites. The eastern and western United States were modeled separately with finer resolution used in the eastern United States (12- or 36-km grids versus 56-km grids). Two simulation periods (July 12-24, 1995, and July 5-15, 1995, for the eastern United States and July 5-15, 1996, and July 18-31, 1996, for the western United States) were used to generate the ozone data for the benefits analysis. Similar to the analysis for the PM NAAQS, ambient $PM_{2.5}$ and PM_{10} were modeled using a source-receptor matrix based on CRDM. The source-receptor matrix was adjusted for the overestimate of the contribution of fugitive dust to $PM_{2.5}$ and then calibrated using monitoring data.

The criteria air pollutant modeling system (CAPMS) was used to estimate health benefits on the basis of the projected changes in ambient concentrations of ozone and PM and concentration-response functions derived from epidemiological studies. Many health outcomes were quantified (see Table 2-1), and many health outcomes were listed as "unquantified effects" for ozone and PM, as well as for carbon monoxide (CO) and hazardous air pollutants (HAPs). EPA noted that the effects for CO and HAPs were not quantified because no appropriate air-quality models were available. To translate relative risk concentration-response functions into absolute numbers of cases, baseline incidences of each health outcome were estimated within specific age groups. A single concentration-response function for each outcome was applied to the entire country. The Pope et al. (1995) study was used to estimate PM-related premature mortality. No mortality estimates were calculated for ozone because they were assumed to be accounted for in the PM estimates. No thresholds above background concentrations were assumed when modeling the health effects. A 5-year lag structure was assumed for PM-related premature mortality (25% in the first and second years and 16.7% in each of the remaining 3 years).

In the final step, the benefits were monetized for comparison with the cost estimates. EPA used the VSL approach to monetize the premature mortality estimates.

The uncertainty in the analysis was evaluated by identifying key assumptions and presenting alternative calculations. For example, alternative calculations for premature mortality were presented using the Dockery et

al. (1993) study instead of the Pope et al. (1995) study and using a VSLY approach instead of a VSL approach. EPA stated that no probabilities were assigned to the alternative calculations because doing so would make the resulting probabilities seem more precise than they actually were (see Chapter 5). Furthermore, high-end and low-end estimates were not presented because "the probability of all of these alternatives occurring simultaneously is extremely low." However, EPA did present a 5th and 95th percentile estimate, assuming the only source of uncertainty of the benefits estimates was random sampling error in the estimation of the concentration-response coefficients. EPA also conducted several sensitivity analyses; one analysis evaluated various assumptions regarding lag structure for mortality benefits, and another evaluated various assumptions regarding thresholds. EPA also included supplemental calculations for various health outcomes, such as premature mortality resulting from short- term PM or ozone exposure and infant mortality resulting from PM exposure. These supplementary estimates were not considered additive to the primary benefits estimates.

The annual health benefits estimated by EPA for the Tier 2 regulation are summarized in Table 2-3 for the year 2030. The monetized values are also provided. As indicated in the table, mortality benefits dominate the overall estimates when the benefits are monetized.

HEAVY DUTY ENGINE AND VEHICLE STANDARDS AND HIGHWAY DIESEL FUEL SULFUR CONTROL REQUIREMENTS

The Heavy Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements rule (heavy-duty [HD] engine and diesel-fuel rule) establishes new federal emissions standards for heavy-duty vehicles and engines and sets limits on sulfur concentrations in diesel fuel. Emissions standards are established for NO_x, PM, and NMHC. Heavy-duty vehicles must also meet emissions standards for formaldehyde. The standards for both engines and vehicles are to be phased in by 2010, depending on vehicle class or engine type (gasoline or diesel). Full compliance with the sulfur limits for diesel fuel should be achieved by 2006. Similar to the Tier 2 rule, EPA stated that the HD engine and diesel-fuel rule is necessary to help the states meet PM and ozone NAAQS but also noted that some studies have reported health effects below the level of the NAAQS for these two pollutants.

TABLE 2-3 Annual Health Benefits (Avoided Cases of Mortality and Morbidity and Monetized Value) for Tier 2 Regulation in 2030

Health Outcome	Avoided Cases[a]	Monetized Benefit (1997$ in millions)[b]
PM-Related Health Outcomes		
Premature mortality (adults, ages 30 and over)	4,300 (2,700-5,900)	23,380
Chronic bronchitis	2,300 (600-4,100)	730
Hospital admissions		
Respiratory causes	1,200 (400-2,100)	10
Cardiovascular causes	500 (100-1,100)	10
Emergency room visits for asthma	900 (400-1,400)	<1
Acute bronchitis (children, ages 8-12)	7,900 (0-16,300)	<1
Lower respiratory symptoms (children, ages 7-14)	87,100 (39,900-131,100)	<5
Upper respiratory symptoms (children with asthma, ages 9-11)	86,500 (25,500-144,600)	<5
Shortness of breath (African Americans with asthma, ages 7-12)	17,400 (4,700-29,500)	<1
Work-loss days (adults, ages 18-65)	682,900 (597,800-771,800)	70
Minor restricted-activity days and acute respiratory symptoms	3,628,500 (3,034,100-4,177,200)	170
Ozone-Related Health Outcomes		
Chronic asthma (adult males, ages 27 and over)	400 (100-800)	10
Hospital admissions		
Respiratory causes	1,000 (200-1,800)	10
Cardiovascular causes	300 (0-500)	<5
Emergency-room visits for asthma	400 (100-600)	<1
Minor restricted-activity days and acute respiratory symptoms	2,226,500 (1,014,400-3,414,800)	100
Decreased worker productivity (adult working population)	Not reported	140

[a]Mean value provided with 5th and 95th percentile values shown in parentheses rounded to the nearest 100.
[b]Mean value of monetized value provided for reference.
Source: Adapted from EPA 1999a,c.

The benefits of the HD engine and diesel-fuel rule were evaluated using the general procedure used for the Tier 2 rule. However, several aspects differ among the analyses, such as air-quality models used, health outcomes evaluated, concentration-response functions selected, and valuation techniques used. Similarities and differences are highlighted in the following discussion.

Similar to the Tier 2 benefits analysis, a four-step approach was used to estimate benefits for the HD engine and diesel-fuel rule. First, emissions inventories were developed for two scenarios for the year 2030—a baseline scenario in which the rule was not implemented and a control scenario in which the rule was fully implemented. The year 2030 was chosen because it provided "a snapshot of benefits and costs in a future year in which the heavy duty fleet consists almost entirely of vehicles and fuels meeting" the HD engine and diesel-fuel standards. Emissions estimates were developed for NO_x, NMHC, SO_2, and PM. Compliance assumptions were not clearly presented in the discussion of the benefits analysis.

Second, ambient air concentrations of ozone and PM (PM_{10} and $PM_{2.5}$) across the continental United States were modeled for a base-year (1996) and for the baseline and control scenarios in 2030. Both air-quality models used for the analysis simulated the physical and chemical processes in the atmosphere that affect pollution transport and transformation and provided temporal and spatial concentration estimates. Inputs to the models included emissions inventories, meteorological data, and land-use information.

Similar to the Tier 2 analysis, ambient ozone concentrations were estimated using a regional-scale version of the urban airshed model-variable grid (UAM-V). However, for the benefits analysis of the HD engine and diesel-fuel rule, EPA did not include the modeling results for the western United States because of poor model performance in that region (the model significantly underestimated observed concentrations). Hourly ozone concentrations were simulated within 12- or 36-km grid squares covering the eastern United States for three brief periods in the summer (June 12-24, July 5-15, and August 7-21, 1995), which were selected because they represented a recent time period and "contained several periods of elevated ozone over the Eastern U.S." The modeling results were corrected using calibration factors developed from comparison of modeled and monitor data for the base-year of 1996. The modeling results were extrapolated to a 5-month ozone "season" (May-September). Ozone data for nonmonitored areas were obtained by interpolation of data from nearby monitoring sites.

Air-quality estimates for PM were developed using a national-scale

version of the regulatory model system for aerosols and dispersion (REMSAD). This modeling procedure differed from that used for the Tier 2 analysis. Three-hour average PM concentrations were simulated for a full year within 36-km square grids for the continental United States. PM species modeled included primary coarse fraction PM (2.5 to 10 μm diameter range), primary fine particles (under 2.5 μm diameter), and several secondary fine particles, such as sulfates, nitrates, elemental carbon, and organics. All fine-particle components were summed to obtain $PM_{2.5}$ estimates. Because insufficient $PM_{2.5}$ monitoring data were available across the United States, the $PM_{2.5}$ simulations could not be calibrated.

Similar to the Tier 2 analysis, CAPMS was used to estimate health benefits on the basis of differences in ambient air concentrations in the baseline and control scenarios for 2030 and concentration-response functions derived from epidemiological studies. However, there were a few differences in health outcomes evaluated and concentration-response functions selected between the Tier 2 analysis and HD engine and diesel-fuel analysis. For example, chronic asthma and shortness of breath were not evaluated as primary health outcomes for ozone and PM, respectively; however, asthma attacks were evaluated for both ozone and PM. An adjustment was made to the estimates for minor restricted-activity days to avoid double-counting of effects. In addition, the concentration-response function used to estimate PM-related premature mortality was taken from the re-analysis of the Pope et al. (1995) study (Krewski et al. 2000). To translate relative risk concentration-response functions into absolute numbers of cases, baseline incidences of each health outcome were estimated within specific age groups. A single concentration-response function for each outcome was applied to the entire country. No thresholds above background concentrations were assumed, and a 5-year lag structure was assumed for PM-related premature mortality (25% in the first and second years and 16.7% in each of the remaining 3 years).

Finally, benefits were monetized and compared with cost estimates. A VSL approach was used to monetize the mortality benefits. Estimates were not provided using a VSLY approach; however, alternative calculations were provided using an age-adjusted VSL approach. The benefits estimates for this analysis were adjusted to reflect growth in real income.

Uncertainties in this analysis were evaluated using the same approach as that used in the Tier 2 analysis. Alternative calculations were presented for key assumptions and included calculations for avoided cases of prema-

ture mortality using an age-adjusted VSL approach, for avoided cases of chronic asthma for ozone, and for avoided cases of other health outcomes using different concentration-response or valuation functions. Sensitivity analyses were used to evaluate lag structures and threshold assumptions. Supplemental calculations were also presented for several health outcomes, such as premature mortality resulting from short-term PM or ozone exposure and infant mortality resulting from PM exposure. These supplementary estimates were not considered additive to the primary benefits estimates.

The annual health benefits estimated for the HD engine and diesel-fuel regulation are summarized in Table 2-4 for the year 2030. Monetized benefits are also provided. As indicated in the table, the mortality benefits dominate the overall estimate when the benefits are monetized.

PROSPECTIVE ANALYSIS OF THE 1990 CLEAN AIR ACT AMENDMENTS

"The Benefits and Costs of the Clean Air Act, 1990-2010" (EPA 1999b) analyzed the benefits and costs of Titles I-V of the 1990 Clean Air Act Amendments (CAAA). Critical elements of the analysis are summarized in Table 2-5. Each title of the CAAA targets different sources or types of air pollutants. Specifically, Title I, which targets primarily stationary sources, establishes a program for meeting and maintaining the NAAQS; Title II establishes regulations for mobile sources and requirements for reformulated gasoline; Title III regulates hazardous air pollutant (HAP) emissions and defines HAPs to be regulated; Title IV establishes a program for controlling precursors of acid rain (primarily SO_2 emissions from electric utilities); and Title V "requires a new permitting system for primary sources of air pollution." The benefits and costs of Title VI, which limits the emissions of stratospheric ozone-depleting chemicals, are also reported in the study; however, they are based on a previous regulatory impact assessment (RIA), and the methods used to derive them are not discussed further here.

Because each title consists of many individual rules, the analysis is much broader than in most RIAs, including those discussed in this chapter. EPA analyzed two scenarios: a pre-CAAA condition in which all pollution controls are frozen at 1990 levels of stringency and effectiveness and a

TABLE 2-4 Annual Health Benefits (Avoided Cases of Morbidity and Mortality and Monetized Value) for the HD Engine and Diesel-Fuel Regulation for 2030

Health Outcome	Avoided Cases[a]	Monetized Benefit (1995$ in millions)[b]
PM-Related Health Outcomes		
Premature mortality (adults, ages 30 and over)	8,300 (4,800-11,700)	62,580
Chronic bronchitis (adults, ages 26 and over)	5,500 (1,900-9,500)	2,430
Hospital admissions		
Pneumonia (adults, ages 65 and over)	1,100 (600-1,600)	20
COPD (adults, ages 64 and over)	900 (200-1,600)	10
Asthma (ages 65 and younger)	900 (400-1,400)	10
Cardiovascular (adults, ages 65 and over)	2,700 (2,300-3,100)	50
Emergency room visits for asthma (ages 65 and younger)	2,100 (900-3,200)	<5
Asthma attacks (all ages)	175,900 (61,000-291,900)	Not monetized
Acute bronchitis (children, ages 8-12)	17,600 (! 100-35,900)	<5
Lower respiratory symptoms (children, ages 7-14)	192,900 (88,300-295,800)	<5
Upper respiratory symptoms (children with asthma, ages 9-11)	193,400 (65,300-325,400)	10
Work-loss days (adults, ages 18-65)	1,539,400 (1,337,300-1,733,300)	160
Minor restricted-activity days (adults, ages 18-65)	7,990,400 (6,806,700-9,104,800)	430
Ozone-Related Health Outcomes[c]		
Hospital admissions		
Respiratory causes (all ages)	1,200 (200-2,100)	20
Cardiac dysrhymias (all ages)	300 (0-600)	<5
Emergency room visits for asthma (all ages)	300 (100-500)	<1
Asthma attacks (all ages)	185,500 (70,400-305,800)	Not monetized
Minor restricted-activity days (adults, ages 18-65)	1,848,100 (988,600-2,706,600)	100
Decreased worker productivity (adult working population)	Not reported	140

[a]Mean value provided with 5th and 95th percentile values shown in parentheses rounded to the nearest 100.
[b]Mean value of monetized value provided for reference. The estimates have been adjusted for growth in real income.
[c]Estimates provided are for eastern United States only.
Abbreviation: COPD, chronic obstructive pulmonary disease.
Source: Adapted from EPA 2000a,b.

TABLE 2-5 Elements of the Prospective Analysis of the 1990 Clean Air Act Amendments

Parameters	
Benefits evaluation points	2000 and 2010
Scenarios	Evaluated conditions with and without implementation of Titles I-V of the 1990 Clean Air Act Amendments
Pollutants modeled and methods used for air-quality modeling for benefits analysis	Ozone – regional-scale version of the urban airshed model (UAM-V) for eastern and western United States; UAM-IV for Los Angeles, San Francisco, and Phoenix
	PM_{10} and $PM_{2.5}$ – regional acid deposition model/regional particulate model for the eastern United States; regulatory modeling system for aerosols and acid deposition for the western United States
	CO, NO_x, and SO_2 – linear scaling procedure based on percent reduction in emissions
Health outcomes quantified and monetized[a]	Ozone – chronic asthma; minor restricted-activity days and respiratory symptoms; hospital admissions (respiratory and cardiovascular illness); emergency room visits for asthma
	PM – premature mortality; bronchitis (chronic and acute); hospital admissions (respiratory and cardiovascular illness); emergency room visits for asthma; lower and upper respiratory symptoms; shortness of breath; minor restricted-activity days and respiratory symptoms; work-loss days
	CO – hospital admissions (respiratory and cardiovascular illness)
	NO_x – hospital admissions (respiratory and cardiovascular illness); respiratory illness
	SO_2 – hospital admissions (respiratory and cardiovascular illness); chest tightness, shortness of breath, or wheeze
Concentration-response function used to estimate mortality benefits	Pope et al. (1995)
Threshold assumptions	No thresholds above background concentrations assumed for modeled health outcomes

(Continued)

TABLE 2-5 *Continued*

Parameters	
Lag-time assumptions	5-year lag structure assumed for PM-related premature deaths with 25% in years 1 and 2 and 16.7% in years 3, 4, and 5
Quantification of uncertainty	1. Calculated 5th and 95th percentiles that reflected within-study variance and across-study variability in both the health effects estimation and the economic valuation steps; 2. Provided alternative calculations for key assumptions; 3. Conducted sensitivity analyses
Study populations evaluated for health outcomes	Majority of benefits estimated for adult populations. PM mortality estimated for population 30 yr and older. Some hospital admissions studies use entire population; others use the population over 65 yr

[a]Many other health outcomes were listed as unquantified for the listed pollutants. A few health outcomes were quantified but were not monetized because they were included in another benefits category.

post-CAAA condition in which all rules stemming from passage of the 1990 CAAA are implemented. However, the post-CAAA condition does not include the recent regulations described in this chapter (PM and ozone NAAQS, Tier 2 emissions standards, and HD engine and diesel-fuel standards). EPA noted that the recent regulations use the prospective post-CAAA scenario as the baseline; therefore, the benefits estimates in those analyses are considered incremental to those estimated for the prospective analysis (EPA 1999b).

Benefits are analyzed in the aggregate for Titles I-V, and annual estimates of benefits and costs are presented for the years 2000 and 2010. The present value of benefits and costs over the period 1990 to 2010 are also calculated. Categories of benefits estimated include health, visibility, agricultural, and ecological benefits. The process used to calculate the benefits is similar to that used to evaluate benefits for the Tier 2 and the HD engine and diesel-fuel rules.

First, the changes in emissions of PM ($PM_{2.5}$ and PM_{10}), SO_2, NO_x, VOCs, and CO were estimated for the base-year 1990 and for the pre- and post-CAAA scenarios in 2000 and 2010. The changes in emissions are primarily associated with Titles I, II, and IV. The impacts of Title III on HAP emissions were not calculated; consequently, the health benefits

resulting from reductions in HAP emissions were also not calculated. Title V has no direct impact on emissions of the criteria air pollutants.

The emissions estimates were then used to model or calculate changes in ambient air concentrations of ozone, PM, SO_2, NO_x, and CO. Ozone concentrations were modeled using UAM-V for the eastern and the western United States and UAM-IV for three metropolitan areas (Los Angeles, San Francisco, and Phoenix). Spatial resolution of the model was greater for the eastern United States (12- or 36-km square grids) than for the western United States (56-km square grids). Spatial resolution within the cities was still greater (4- or 5-km square grids). One or two simulation periods ranging from 2 to 10 days were used to generate hourly ozone concentrations.

PM concentrations in the western United States were modeled using REMSAD, and PM concentrations in the eastern United States were modeled using the regional acid deposition model (RADM)/regional particulate model (RPM). Spatial resolution of the modeling was greater for the western United States (56-km grid squares) than for the eastern United States (80-km grid squares). Daily $PM_{2.5}$ and PM_{10} concentrations were generated using "30 randomly selected 5-day periods spanning a four-year period" for the eastern United States and using one 10-day period for each season for the western United States.

PM and ozone were modeled for the base-year 1990 and the pre- and post-CAAA scenarios in 2000 and 2010. Ambient concentrations used for the benefits analysis were calculated by adjusting the observed ambient pollutant concentrations in 1990 by a ratio of the predicted concentrations for 2000 or 2010 to the predicted concentrations for 1990. Data were interpolated for the nonmonitored sites in the country.

Ambient concentrations of SO_2, NO_x, and CO were calculated using a linear scaling approach and the assumption that ambient concentrations are reduced by the same percent as the estimated emissions reductions. Accordingly, observed ambient concentrations were multiplied by the ratio of the predicted emissions for 2000 or 2010 to the emissions for 1990.

Differences in ambient air concentrations, population estimates at given locations, and concentration-response functions for given health outcomes were used as inputs into CAPMS to generate benefits estimates for 2000 and 2010. The health benefits that were quantified and monetized in the study are summarized in Table 2-5 and included avoided cases of premature mortality and chronic bronchitis associated with PM, hospital admissions

associated with PM, ozone, CO, NO_x, and SO_2, and minor restricted-activity days associated with PM and ozone. Many other health outcomes were listed, but were not quantified (or were not included in the analysis) because of a lack of data or possibility of double-counting. Estimates of avoided cases of premature mortality were based on the Pope et al. (1995) study. No thresholds above background concentrations were assumed, and a 5-year lag structure was assumed for PM-related premature mortality (25% in the first and second years and 16.7% in each of the remaining 3 years).

Uncertainties in the analysis were addressed by quantitative estimates, qualitative discussions, alternative calculations for key assumptions, and sensitivity analyses. EPA calculated 5th and 95th percentiles that reflected within-study variance and across-study variability in both the health effects estimation and the economic valuation steps. The statistical estimates did not reflect uncertainty in other phases of the analysis (emissions and air-quality modeling). Each stage of the analysis included qualitative discussions about the bias and significance of key uncertainties for that stage of the analysis. Alternative calculations were presented for a few key assumptions. For example, the Dockery et al. (1993) study was used to estimate avoided cases of premature mortality rather than the Pope et al. (1995) study, and a VSLY approach was used to value the premature mortality rather than the VSL approach, which was used for the primary estimate. Several sensitivity analyses were conducted, including one intended to evaluate the influence of the largest source of uncertainty.

The annual mean health benefits for the prospective analysis of the 1990 CAAA are summarized in Table 2-6 for 2010. The monetized values of the health benefits are also provided. As in the other analyses evaluated, the mortality benefits dominate the monetized benefits.

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TABLE 2-6 Annual Mean Health Benefits (Avoided Cases of Morbidity and Mortality and Monetized Values) for the Prospective Analysis of the 1990 CAAA for 2010

Health Outcome	Pollutant	Avoided Cases[a]	Monetized Value (1990$ in millions)
Mortality (ages 30 and older)	PM	23,000 (14,000-32,000)	100,000
Chronic bronchitis	PM	20,000 (5,000-34,000)	5,600
Chronic asthma	Ozone	7,200 (1,800-12,000)	180
Hospitalization			
All respiratory illness	PM, CO, NO_2, SO_2, Ozone	22,000 (13,000-34,000)	130
Total cardiovascular illness	PM, CO, NO_2, SO_2, Ozone	42,000 (10,000-100,000)	390
Emergency room visits for asthma	PM, Ozone	4,800 (430-14,000)	1
Acute bronchitis	PM	47,000 (0-94,000)	2
Upper respiratory symptoms	PM	950,000 (280,000-1,600,000)	19
Lower respiratory symptoms	PM	520,000 (240,000-770,000)	6
Respiratory illness	NO_2	330,000 (76,000-550,000)	6
Moderate or worse asthma[b]	PM	400,000 (80,000-720,000)	13
Asthma attacks[b]	Ozone, PM	1,700,000 (920,000-2,500,000)	55
Chest tightness, shortness of breath, or wheeze	SO_2	110,000 (290-520,000)	0.6
Shortness of breath	PM	91,000 (26,000-150,000)	0.5
Work-loss days	PM	4,100,000 (3,600,000-4,600,000)	340
Minor restricted-activity days and any of 19 respiratory symptoms	Ozone, PM	31,000,000 (25,000,000-37,000,000)	1,200

[a]Mean value provided with 5th and 95th percentile values shown in parentheses.
[b]These results were not included in the total benefits estimate because they overlap with health outcomes included in the category for minor restricted-activity days and any of 19 respiratory symptoms.
Source: Adapted from EPA 1999b.

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3

Framing the Analysis

The estimates of health benefits depend critically on the choices made in framing the analysis (what will and will not be included) at the beginning of the process. The most important of these choices are (1) the regulatory options to consider, (2) the health effects to evaluate, (3) the time frame for the analysis, including the years in which benefits are evaluated, and (4) the assumptions to make about conditions with and without the regulation implemented. The assumptions influence the benefits by determining the size of the emissions reductions attributed to the regulation and by determining the size, income, and health status of the population that will benefit from the air pollution regulation. This chapter discusses how EPA has dealt with each of these sets of decisions and uses examples from the four EPA benefits analyses reviewed by the committee and summarized in Chapter 2 of this report.

REGULATORY OPTIONS EVALUATED

In three of the analyses examined by the committee, EPA focused on evaluating a single set of regulatory options: (1) end-of-tailpipe emissions controls for passenger vehicles and reduction of the sulfur content of gasoline in the Tier 2 emissions standards (EPA 1999a); (2) measures to make heavy-duty engines less polluting and reduction of sulfur content of diesel fuel in the heavy-duty (HD) engine and diesel fuel rule (EPA 2000);

and (3) a set of measures that would achieve the goals of Titles I-V of the 1990 Clean Air Act Amendments (CAAA) in the prospective analysis of the Clean Air Act (EPA 1999b)—hereafter referred to as the prospective analysis. For each of the first two rules, only a single package of phased-in changes in capital equipment and fuel composition was evaluated. Alternative types of controls or different schedules for phasing in the controls were not considered. The prospective analysis estimated the benefits and costs of the first five titles of the 1990 CAAA combined and did not attempt to disaggregate benefits by title.

In the analysis of the particulate matter (PM) and ozone National Ambient Air Quality Standards (NAAQS) (EPA 1997), the agency considered three regulatory alternatives that were combinations of the following annual average and 24-hr standards for $PM_{2.5}$: (1) 16 $\mu g/m^3$ and 65 $\mu g/m^3$, (2) 15 $\mu g/m^3$ and 65 $\mu g/m^3$, and (3) 15 $\mu g/m^3$ and 50 $\mu g/m^3$. Similarly, the maximum number of annual exceedences allowed under the proposed 1-hr ozone standard varied from 3 to 4 to 5. These options were compared, assuming partial attainment of each option.

In general, EPA's approach does not satisfy Office of Management and Budget (OMB 1996, 2000) guidance on benefits analysis. The OMB guidelines include consideration of a range of levels for the standard and different time schedules for compliance, as well as a variety of qualitatively different market interventions, such as information measures, market-based approaches, performance-based standards, and different requirements for different segments of the regulated population. When a regulatory action represents a package of different provisions, such as the various titles of the 1990 CAAA, OMB suggests that the parts of the package be assessed separately to the extent feasible. Specifically, OMB (1996) makes the following statements:

> If the proposed regulation is composed of a number of distinct provisions, it is important to evaluate the benefits and costs of the different provisions separately. The interaction effects between separate provisions (such that the existence of one provision affects the benefits or costs arising from another provision) may complicate the analysis but does not eliminate the need to examine provisions separately. In such a case, the desirability of a specific provision may be appraised by determining the net benefits of the proposed regulation with and without the provision in question. Where the

number of provisions is large and interaction effects are pervasive, it is obviously impractical to analyze all possible combinations of provisions in this way. Some judgment must be used to select the most significant or suspect provisions for such analysis.

For the HD engine and diesel-fuel rule, there are clearly good reasons why some changes should not be considered in isolation from other changes. For example, changes in end-of-pipe pollution-control equipment, such as particle filters and regeneration systems, should not be considered without changes in fuel composition. However, there is no obvious reason why the effects of the fuel changes without the equipment changes or with equipment changes implemented at different periods could not have been evaluated for their effects over time.

In the case of the PM and ozone NAAQS, it would be valuable to know how much benefits and costs increase as the ambient air-quality standard for PM is tightened. In other words, how do benefits and costs change as the $PM_{2.5}$ standard moves from an annual average of 20 $\mu g/m^3$ to 15 $\mu g/m^3$? In the case of the 1990 CAAA, over 80% of the total cost of Titles I-V is associated with Titles I and II alone. Although the costs are reported separately for Titles I and II, it would be useful to know whether the estimated benefits of these two titles exceed their estimated costs.

In agreement with and extending the OMB guidance, the committee believes that EPA should seek to represent a realistic range of regulatory choices guided by expert opinion and technical feasibility. The agency should, at the beginning of each analysis, discuss the range of choices and the preliminary analyses that were conducted to exclude certain options from the formal analysis. This approach would strengthen analyses that currently appear to serve the purpose of justifying the agency's chosen regulatory option without comparing that option with other feasible possibilities.

A related issue concerns assumptions made about compliance with air pollution regulations. As indicated in Chapter 1, current EPA Office of Air Quality, Planning and Standards guidance calls for analysts to assume full compliance with regulatory requirements when estimating the costs and benefits of regulations. The committee believes that this recommended approach should be changed, because decision-makers and the public should be given the likely results of different regulatory choices as accurately as possible. Assuming perfect compliance may often result in overestimation

of the health benefits and costs likely to result from a new regulation. Incorporating alternative assumptions about compliance into a sensitivity or uncertainty analysis would more completely convey the full range of potential benefits.

Furthermore, assuming perfect compliance is likely to result in the agency's neglecting the important issue of the relative cost and effectiveness of alternative implementation and enforcement measures. In the absence of a comparative analysis of implementation, decision-makers will not be able to compare regulatory options that are likely to differ in the ease and reliability of implementation. For example, EPA enforcement of regulatory requirements that change the emissions characteristics of newly marketed engines may be relatively straightforward and inexpensive compared with enforcement of requirements that operators maintain engines at a specific standard.

Notable exceptions to the above criticisms include the compliance assumptions made in the prospective analysis and the HD engine and diesel-fuel analysis. For the prospective analysis, EPA did not assume perfect compliance with proposed regulations but assumed stationary sources would achieve only 80% of target reductions for nitrogen oxides (NO_x) and volatile organic compounds (VOCs). Furthermore, actual emission rates for mobile sources reflected real-world tampering and other noncompliance issues. For the HD engine and diesel-fuel analysis, EPA analyzed the potential impacts on future emissions of tampering with and inadequately maintaining the proposed HD diesel-control technology. The committee endorses EPA's stated goal of expanding its current capability to analyze the potential impacts of incomplete compliance with proposed regulations by developing improved data on actual emissions.

SELECTION OF EFFECTS TO EVALUATE

EPA must determine how broadly to define the scope of each analysis. This task includes determining the categories of benefits to evaluate and the extent of examination of secondary or unintended effects of the regulation. Although the evaluation of the direct effects of the regulation on human health is the primary focus of the analyses reviewed, the committee notes that the regulations may also affect human health indirectly. Air pollution

regulations intended to change air pollution levels may also change how fuels are made or how combustion devices are operated. These changes can then affect human health through other pathways. Although outside the strict boundaries of public exposure to air pollution, an analysis of health benefits that ignores these indirect effects may result in a substantial misrepresentation of the actual impact of pollution control measures on society. Therefore, the analyst should seek expert guidance when appropriate and consider such issues as the following:

- Can the regulation potentially compromise occupational health? For example, a measure to control VOC emissions from an industry may cause an increase in occupational exposures to toxic substances by reducing ventilation in production areas.
- Can the regulation potentially increase pollution in other locations? For example, a policy measure that shifts electricity production toward hydroelectric power plants relative to fossil-fuel power plants might result in substantial increases in cement production and subsequent air pollution consequences in other locations.
- Can the regulation potentially cause cross-media effects? For example, use of methyl-*t*-butyl ether to control air pollution from vehicle emissions resulted in increased water pollution.

Therefore, a health benefits analysis should examine the potential for important impacts outside the narrow boundaries of population exposures to air pollution. It should also contain a discussion on whether such impacts could be important. If they are, guidance on assessing them more completely should be included. The committee recognizes that time and resource constraints may require trade-offs between the number of scenarios considered and the level of detail for each.

As an aside, the committee notes that the examples provided are unintended negative impacts and that there may be unintended positive impacts of air pollution control regulations outside the boundaries of the analysis. For example, air pollution control in other parts of the world may be accelerated due to a demonstration effect or economic pull of control efforts in the United States. However, these effects are typically difficult to predict in advance or even to assess after the fact.

TIME FRAME FOR THE ANALYSIS

EPA's analysis of the costs of a regulation typically begins in the year the regulation first goes into effect and continues until the regulation is fully implemented. For example, for the HD engine and diesel-fuel rule, costs were computed from 2006 (the year in which proposed engine modifications and other equipment are to be installed in new trucks) to 2030 (the year in which these modifications will be embodied in all trucks in the fleet). Similarly, the prospective analysis computed the costs of implementing the 1990 CAAA from 1990 to 2010 (the years in which selected provisions of the 1990 CAAA are likely to have been fully implemented).

On the other hand, health benefits are typically estimated for only a single year in the future. The analyses for the Tier 2 emissions standards and the HD engine and diesel-fuel rule evaluated benefits only in 2030. The analysis for the PM and ozone NAAQS evaluated health benefits in 2010. In contrast, the prospective analysis evaluated benefits in 2000 and 2010. In the prospective analysis, benefits in intermediate years were interpolated to calculate the present discounted value of benefits from 1990 to 2010.

The years 2030 and 2010 were chosen because the policies under consideration would likely be implemented by these dates. For example, the Tier 2 emissions standards and the HD engine and diesel-fuel rule both involve modifications in new vehicles required before 2010. The policies will not be fully implemented, however, until all vehicles in the fleet contain these modifications.

Evaluating benefits in only a single year in the future has two limitations. First, when the costs of a policy decrease over time and the benefits increase, a comparison of the benefits and costs only in the distant future is highly misleading. The comparison will overstate the benefits achieved in the early years of the policy; however, the committee does not know how great the overstatement would be. This problem arose in the HD engine and diesel-fuel rule in which the costs of the rule are concentrated in the early years of the regulation, in part because of research and development costs. No attempt was made, however, to compute benefits for an intermediate year, such as 2015.

Second, choosing an evaluation point in the distant future, such as 2030, is likely to increase the uncertainty associated with the calculation of benefits and costs. For example, it is highly uncertain what the passenger vehicle fleet will look like in 2030 and how polluting it would be without Tier 2

emissions standards. Unless this uncertainty is accurately reflected in benefit and cost estimates, the analysis will be misleading.

To EPA's credit, the analysis for the HD engine and diesel-fuel rule acknowledged that focusing on 2030 might be misleading but cites the high cost of evaluating benefits in years before 2030—primarily due to the cost of air-quality modeling—as a reason for its decision to use 2030. Specifically, EPA (2000) made the following statements:

> A more appropriate means of capturing the impacts of timing differences in benefits and costs would be to produce a net present value comparison of the costs and benefits over some period of years. Unfortunately, while this is relatively straight-forward for the costs, it is currently not feasible to do a multi-year analysis of the benefits as this would require a significant amount of air quality modeling to capture each year. We did not have the resources for such an extensive analysis.

The high cost of running multiple air-quality scenarios is likewise cited in the following statements by EPA (1999b) as a reason for aggregating Titles I-V in estimating the benefits of the 1990 CAAA:

> The estimates in Table 8-3 reflect the difficulty we encountered in reliably disaggregating benefits by CAAA Title or even by pollutant. . . . These difficulties in separating the effects of individual emissions reductions on the benefits estimates also highlights the need for an integrated air quality modeling system that can more readily analyze multiple scenarios within reasonable time and resource constraints. A tool of this nature could allow us to more reliably and cost-effectively estimate incremental contributions to ambient PM and ozone concentration reductions.

In presentations before the Science Advisory Board (M. Cropper, University of Maryland, personal communication, June 6, 2002), EPA staff also cited the high cost of air-quality modeling as a reason for not quantifying the uncertainty in emissions estimates and carrying this uncertainty forward in estimating avoided cases of morbidity and mortality.

The committee believes, however, that EPA should make every effort to estimate health benefits associated with reductions in air pollution at

reasonably frequent intervals, such as every 5 years, over the regulatory time frame, including the period of implementation and the expected period of expression of all significant health effects. EPA should modify air-quality models used in translating predicted emissions into predicted levels of ambient air quality to reduce the resources required for air-quality modeling. This change is necessary if EPA is to evaluate multiple regulatory alternatives and if it is to evaluate each alternative at reasonable time intervals, such as every 5 years. The ability to evaluate the ambient air quality associated with more emissions scenarios is also essential if the uncertainty inherent in emissions estimates is to be carried through to estimating avoided cases of mortality and morbidity. The committee notes that emissions and ambient air quality with and without the regulation are treated as certain in the EPA analyses reviewed by the committee. EPA also treats costs as certain.

Because some important evaluation methods, particularly net present-value calculations, require annual estimates of benefits (and costs), full benefits estimates should be accompanied by presentations of benefits, using an appropriate and clearly described interpolation method, for intervening years. The committee notes that the additional precision provided by running all the models for intervening years is unlikely to be worth the effort, given the overall uncertainties in benefits estimation.

Finally, the health benefits of reducing emissions in a single year might not occur solely in that year but might occur in subsequent years because of physiological and other lags. The analyses should carefully state and document the lag relationships between pollution reductions and health improvements that have been used (see Chapter 4).

DESCRIPTION OF CONDITIONS WITH AND WITHOUT THE REGULATION

To estimate the benefits of an air pollution regulation, EPA predicts future conditions with and without the regulation enacted. Two sets of predictions are especially relevant to calculating the health benefits of the regulation. The first describes emissions by sector in the absence of the regulation and emissions by sector after the regulation is imposed. The second set of predictions relates to the population affected by the changes in air quality—the number of people (by age, gender, and location) living in

the United States and the disease and death rates in this population. This section addresses how EPA makes and reports these predictions. Chapter 4 discusses how emissions predictions are translated into ambient pollution concentrations and how the change in ambient concentrations, together with population and baseline rates of disease and death, are used to calculate avoided cases of morbidity and mortality.

Emissions Predictions

In all four analyses reviewed by the committee, EPA predicts emissions for all major source categories of the criteria pollutants: industrial point sources, utilities, nonroad engines and vehicles, motor vehicles, and area sources for one or more future years, such as 2010 or 2030. These predictions are made without the regulation analyzed in the study (designated the regulatory baseline) and with the regulation. The complexity of the models used to predict emissions for electric utilities (the integrated planning model [IPM] developed by ICF, Inc.) and for motor vehicles (MOBILE5 and MOBILE6) is such that only the emissions predicted by these models are summarized in the appendixes to the regulatory impact assessments (RIAs). The models are described in other documents (EPA 2002a,b).[1]

Two issues regarding emissions predictions particularly concern the committee. The first issue is how the emissions estimates with and without the regulation are reported. The documents reviewed here fail to give the reader information on what drives the emissions estimates and make it difficult to judge the plausibility of the estimates. In most sectors, emissions are the product of the level of an activity (such as fuel consumed by electric utilities or miles traveled by motor vehicles) multiplied by the amount of

[1]IPM is a linear programming model that describes electricity demand, generation, transmission, and distribution for all plants in the U.S. electric power market. See http://www.epa.gov/capi/ for further details. The MOBILE models use data on the U.S. vehicle fleet to estimate emissions from motor vehicles. The vehicle fleet is characterized by the total number of vehicles in operation within certain categories, their age distribution and fuel type (gasoline or diesel), and their annual mileage rates by age and fuel type. This information, together with estimates of emissions factors, is used to calculate total fleet emissions. See http://www.epa.gov/otaq/mobile.htm for further details.

pollution generated per unit of activity (such as pounds of sulfur dioxide [SO_2] per millions of British thermal units [mmBtus] or grams of NO_x per mile traveled). The assumptions about activity levels and the pollution intensity, both with and without the regulation, can be made explicit even though it is not possible to describe in detail all of the assumptions underlying these numbers. The committee emphasizes that readers might find it easier to judge the plausibility of the estimates if they were expressed as percentages or if they were compared to historical trends. For example, what percent change in vehicle miles traveled is implicit in emissions estimates for 2030 compared with current levels? What is the percent reduction in pollution intensity estimated to be achieved by a regulation?

The second issue concerns the deterministic nature of the models used to predict emissions. Both IPM and EPA's mobile-source emissions models fail to incorporate any uncertainty in their emissions predictions. In general, any variable that is likely to have a substantial impact on mortality and morbidity and to have considerable uncertainty should be a candidate for a formal uncertainty analysis. Predictions of activity levels 20 years in the future, such as percent of light-duty trucks using diesel fuel, fall in this category.

The calculation of emissions predictions, the ways in which the information should be presented, and the relevance of uncertainty to the analysis are discussed in the following sections, using as examples emissions predictions for electric utilities and emissions predictions for motor vehicles.

Emissions Predictions for Electric Utilities

In the prospective analysis, EPA predicts SO_2 emissions for electric utilities in 2010 with and without regulatory action. In each case, total SO_2 emissions are the product of the fuel consumption (measured in mmBtu) and the pollution intensity (the number of pounds of SO_2 per mmBtu produced) for each electricity-generating unit, summed over all units. Equation 1 depicts this calculation.

$$\text{Total } SO_2 \text{ Emissions} = \sum_i (\text{mmBtu})_i \times (SO_2/\text{mmBtu})_i, \qquad (1)$$

where i denotes a generating unit.

The total SO_2 emissions from electric utilities in 2010 are predicted to be 18 million tons without the 1990 CAAA and 9.9 million tons with the 1990 CAAA. However, the analysis gives no information on what accounts for those results. Although this information may be available in technical support documents, additional information about the components of total SO_2 emissions could be presented for the two scenarios in the main text as a table listing the national aggregate fuel consumption by category of power plant and the national average pollution intensity by category of power plant. This breakdown of the components of predicted emissions could also be supplemented with historical information on aggregate fuel consumption and average pollution intensity by class of power plant. This information would allow the reader to compare actual values with agency predictions. This table would indicate the extent to which the predicted reduction in SO_2 emissions attributed to the 1990 CAAA was the result of an average reduction in fuel consumption or pollution intensity. This information should be supplemented with a measure indicating the extent to which emissions reductions are predicted to result from shifting electricity production from dirtier to cleaner units as a result of the 1990 CAAA.

Supplementing aggregate emissions estimates with the information described above would demonstrate how the predicted reduction in emissions is to be achieved and would highlight important factors to consider in an uncertainty analysis. Suppose, for example, that most of the SO_2 reduction is expected to come from a reduction in the average pollution intensity of coal-fired power plants. If this factor drives the results, then it is important to further examine the assumptions underlying pollution intensity with and without the 1990 CAAA. One way to examine the assumptions would be to make the predictions of the IPM model explicit for pollution intensity with and without the 1990 CAAA and to compare those predictions with historical trends in pollution intensity over the period 1980-1995. (The provisions of the 1990 CAAA that affect SO_2 emissions from power plants went into effect in 1995.) If the predictions without the CAAA appear to be inconsistent with historical trends, this discrepancy should be explained and formally incorporated into an uncertainty analysis. Other components of emissions that might be subjected to uncertainty analysis are estimates of electricity demand that underlie the total amount of fuel burned by power plants and, for longer time periods, assumptions about the retirement of old plants and the construction of new plants.

Emissions Predictions for Motor Vehicles

All four of the health benefits analyses examined by the committee make predictions about the effects of air pollution regulations on motor-vehicle emissions. The total emissions of a pollutant, such as NO_x, from motor vehicles can be written as the sum of average annual NO_x emissions for each class of vehicle *i* times the number of vehicles in that class (n_i). Average annual NO_x emissions for vehicles in class *i* are, in turn, the product of NO_x emissions per mile $(NO_x/mile)_i$ times average annual vehicle miles traveled $(VMT)_i$. The overall calculation can be summarized in the following equation:

$$\text{Total } NO_x \text{ Emissions} = \sum n_i \times (NO_x/\text{mile})_i \times VMT_i, \qquad (2)$$

where *i* denotes vehicle class.

EPA's estimates of the benefits of the Tier 2 emissions standards in 2030 require making assumptions about the relevant categories of vehicles to analyze in 2030 as well as assumptions about each of the three components of Equation 2. It is, however, extremely difficult to understand the key assumptions made about these components or the predictions made for each component at the national level with and without the Tier 2 regulations.

For a reasonable number of classes of vehicles, EPA should present a table showing predicted values of the number of vehicles, emissions per mile for each criteria pollutant, and average VMTs for conditions with and without regulatory action in 2030 at the national level. To put those figures in perspective, a similar table should be constructed showing the values of these variables in the recent past.

Presenting those figures is not sufficient explanation of conditions with and without the Tier 2 emissions standards. The figures should be accompanied in the main text by some explanation of the assumptions that drive the results. For example, if analysts predict a rapid increase in the percent of light-duty trucks powered by diesel, this assumption requires an explanation, especially if it accounts for a large percent of the PM_{10} emissions in the regulatory baseline and, thus, a large percent of the particulate reductions attributed to the Tier 2 emissions standards.[2]

[2]In the Tier 2 emissions standards RIA, Table IIIA-13 shows light-duty diesel trucks increasing from 0.1% of light-duty truck sales in the 2001 model year to 24% of sales in the 2015 model year.

As in the case of power-plant emissions, the purpose of describing the various components of total vehicle emissions is to focus attention on the components that have a large impact on emissions with and without regulatory action and on the change in emissions associated with the proposed emissions standards. This information should guide the assessment of uncertainty in the emissions estimates and allow an examination of the possible distribution of values that key components might assume. See Chapter 5 for a description of the procedures for formalizing the uncertainty associated with emissions estimates and other components of the health benefits analysis.

Predictions Regarding Population and Health

The goal of a health benefits analysis associated with a proposed air pollution regulation is to estimate the avoidable risk associated with that regulation—cases of morbidity and mortality that are likely to be avoided if the regulation is implemented. The standard approach to computing avoided cases of morbidity and mortality (assuming a linear concentration-response function) is to multiply the size of the exposed population (Pop) by the baseline incidence of the health effect in question (Y_b) in the year, such as 2030, in which benefits are to be evaluated. This calculation yields the predicted baseline number of cases in 2030. The reduction in cases is estimated by multiplying baseline cases by the slope of a concentration-response function (β) that describes the percent reduction in cases per unit of pollutant and by the reduction in ambient pollution associated with the regulation (ΔC). The overall calculation can be approximated by the following equation:

$$\text{Cases Avoided} = \beta \times \Delta C \times Y_b \times \text{Pop}. \quad (3)$$

Calculation of avoided cases thus requires estimates of population and baseline disease rates (or death rates) for the years in which benefits are to be evaluated. These estimates are required at the level of geographic disaggregation used in modeling the air quality.

EPA is generally clear about how it projects future population and incidence rates. For the HD engine and diesel-fuel rule, EPA clearly stated that population projections come from the U.S. Census Bureau (EPA 1999c). The methods used to interpolate the population projections for the year of the health benefits analysis (2030) are clearly explained, as are the

methods used to associate county-level data with the grid cells used in the air-quality modeling.

The methods used to estimate incidence for various health outcomes are described in Appendixes B and C of the same document. In many cases, incidence in 2030 is assumed to be identical to that in the late 1990s. For example, the annual county mortality rates from 1994 to 1996 are used to estimate nontrauma mortality rates in 2030. For hospital admissions (by International Classification of Disease code), national incidence in 2030 is assumed to be equal to that in 1994, the most recent year available at the time of the study. For health outcomes that lack national incidence data, incidences are assumed equal to those in the epidemiological studies used to compute the number of avoided cases.

Predicting baseline morbidity and mortality rates 30 years into the future is difficult, because there is much evidence that rates can change significantly over such periods. For example, rates of heart disease, one of the major disease categories affected by ambient air pollution, have been remarkably reduced in the past 30 years. Although it is probably not feasible to project baseline rates for all health outcomes considered in health benefits analyses, EPA should incorporate estimates of future trends in mortality and morbidity for major health outcomes, such as those that make up two-thirds of total deaths or lost life-years, that are being considered. At the least, EPA's estimates of avoided cases should reflect the uncertainty in these rates. For some outcomes with available data, this uncertainty can be reduced by disaggregating baseline rates and applying them by age groups, because future shifts in age distribution are less uncertain and are projected routinely by widely accepted sources. This approach should be followed whenever possible.

Another source of uncertainty in estimating avoided cases derives from the distinction between attributable and avoidable risk. The β coefficients in Equation 3 come from studies that relate variation in health impacts to variation in air pollution concentrations based on historical data. The result is a measure of the risk attributable to air pollution in the past. Characteristics of the study population that are not explicitly controlled for in the concentration-response function are implicitly reflected in the β coefficients. The extent to which future populations differ from those in the studies will add to the degree of uncertainty associated with estimating the avoided cases.

To illustrate, all the analyses examined by the committee rely on the American Cancer Society (ACS) study (Pope et al. 1995; Krewski et al.

2000) to estimate the impact of changes in PM concentrations on mortality. The estimate of the impact of fine-particle exposure on the nontrauma death rate (with relative risk assumed the same for all age groups) is used to predict avoided cases of mortality. There are many sources of error in applying this coefficient to populations in the year 2030. One error in applying the ACS study occurs because the impact of PM exposure on nontrauma deaths is actually an average of its impact on various causes of death, such as coronary artery disease and lung cancer. To the extent that the distribution of deaths by cause in the U.S. population in 2030 differs from that in the ACS study population, errors will result. Another source of error occurs because the age distribution of the ACS study population may differ from the age distribution of the population in 2030.

To incorporate these considerations into the computation of avoided cases of morbidity and mortality, the predicted characteristics of the population in 2030 must be compared with the characteristics of the populations in the epidemiological studies used to compute avoided cases. Appropriate adjustments should be made if differences are found.

CONCLUSIONS

- The estimation of health benefits that will result from reducing air pollution depends critically on decisions made at the beginning of the analysis: (1) the regulatory options to consider, (2) the health effects to evaluate, (3) the time frame for the analysis, including the years in which benefits are evaluated, and (4) the assumptions to make about future conditions with and without implementation of the regulation.
- A critical step in the preliminary stages of an RIA is the development of a range of regulatory options to evaluate. Fewer regulatory alternatives than would be needed to follow OMB guidelines are presented or appear to be evaluated in recent EPA analyses. The regulatory options should represent the range of choices available.
- EPA typically evaluates the costs of the regulatory options examined from the time the regulations are first introduced until they have been fully implemented. By contrast, the benefits of the regulations are often examined for only a single year, usually the year in which the policy will be fully implemented. The comparison of benefits and costs focuses on this one future year rather than comparing the benefits and costs over the period of implementation.

- The high cost of air-quality modeling is cited as a major reason for limiting the years in which benefits are evaluated and also as a reason for not calculating the costs and benefits of more regulatory options.
- Predictions about emissions with and without the regulations are treated as certain and are presented in terms of total emissions by sector. The components of emissions, such as number of vehicles in a class, average miles traveled per vehicle, and emissions per mile, are seldom presented, and predicted emissions are seldom compared with historical trends to place them in perspective.
- Predictions about future population trends and the baseline health of the population are more clearly stated than those for emissions; however, these predictions are treated as certain, even when predictions are made far into the future.

RECOMMENDATIONS

- To the extent possible, EPA should estimate the benefits for several regulatory options that represent the full range of choices available to the decision-maker. The regulatory options should include graded levels of stringency requirements and the time schedule for achieving reductions in emissions or exposures. If options are eliminated at an earlier stage, the rationale for doing so should be provided.
- EPA should estimate the benefits over the regulatory time period including both the implementation period and the expression period of all important health effects. Because calculating benefits for every future year is resource-intensive and unlikely to show true increases in precision, calculations can be made, for example, every fifth year with simple interpolation techniques applied to estimate benefits for intervening years.
- EPA should modify the air-quality models used in translating predicted emissions into predicted levels of ambient air quality to reduce resources required for air-quality modeling. This change is necessary if EPA is to evaluate multiple regulatory alternatives and to evaluate each alternative at reasonable time intervals, such as every 5 years. Evaluation of the ambient air quality associated with more emissions scenarios is also essential if the uncertainty inherent in emissions estimates is to be carried through to the estimation of avoided cases of mortality and morbidity.
- The components of emissions estimates (such as number of vehicles in a class, average miles traveled per vehicle, and emissions per mile)

should be presented with and without implementation of the regulation at the national level. This will help readers judge how reasonable these predictions are and will suggest which components of emissions estimates drive the emissions reductions associated with the regulation. Historical trends in these components should also be presented.

- The uncertainty in emissions estimates should be quantified and carried through the health benefits analysis to the calculation of avoided cases of mortality and morbidity.
- EPA should incorporate estimates of future trends in background mortality and morbidity for the major health outcomes, such as those that make up two-thirds of total deaths or lost life-years, that are under consideration.
- EPA should quantify uncertainties with regard to future population distributions and background disease rates. EPA should also summarize what is known about the potential importance of disease interactions and competing risks affecting the health outcomes of primary interest and discuss the possible biases that might be introduced in the final analysis by changes in those factors.
- Because a regulation to improve air quality may affect pathways other than air, EPA should determine whether there are likely to be any important indirect impacts of a regulation on human health and the environment. If any such impacts are identified, EPA should include in the analysis a plan to assess them more completely.

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4

Exposure and Response

This chapter discusses three key components of benefits analyses: exposure assessment, health outcomes, and concentration-response functions. The exposure assessment section begins with an overview of exposure assessment considerations, including issues related to exposure assessments in the epidemiological studies that are frequently used to estimate health benefits of air pollution reductions. A general overview of air-quality modeling and its role in benefits analysis follows. The selection and interpretation of health outcomes are then discussed. Finally, the concentration-response section explores the sources and selection of these functions and issues associated with the existence of thresholds, analysis of population subgroups, and assumptions regarding effects lags (the temporal relationship between changes in exposure and resulting changes in health outcomes).

EXPOSURE ASSESSMENT

Estimating changes in population exposures to air pollutants is an essential component of EPA's benefits analyses, providing the link between anticipated emissions changes and resulting changes in health outcomes. Because it is not possible to observe population exposures to air pollution under different regulatory options, exposure assessment in benefits analysis uses models to simulate air pollution exposures that might occur as a result of

those options. Exposure modeling is a complex process that depends on many assumptions about the future, including pollution emissions reductions resulting from the proposed regulation; changes in emissions due to factors other than the proposed regulation; meteorological conditions; the physical and chemical processes in the atmosphere affecting pollution dispersion, transformations, and deposition; and the nature and degree of pollutant contact with future human populations. As in all other stages of the benefits analysis, the assumptions and methods used in the exposure assessment should be well-justified and clearly described, with careful attention paid to assessing and communicating key sources of uncertainty.

EPA's exposure assessment methods have evolved considerably over time, as is evident in the health benefits analyses reviewed by the committee. This evolution is due to continued improvements in modeling capabilities and to a marked increase in available air-monitoring data for many pollutants. Because the most recent EPA analysis reviewed by the committee (the benefits analysis for the heavy-duty (HD) engine and diesel-fuel rule) uses current data and exposure assessment methods, it serves as an illustrative example throughout this exposure assessment discussion.

The committee considers that the exposure assessment methods used in the analysis for the HD engine and diesel-fuel rule represent an appropriate and reasonably thorough application of available data and models. Although limitations, as noted in following sections, exist, they are primarily due to limitations of available scientific knowledge and, ultimately, the limited time and staff resources available for analysis rather than flawed analytical methods.

Exposure to air pollution has been defined as the intersection in time and space of a concentration of pollution in the air and the presence of a human being (NRC 1991; Ott 1995). For benefits analyses, exposure is typically assessed at the population level by geographically linking estimates of outdoor pollution concentrations with projected population numbers; these together represent the necessary input to population concentration-response functions for calculating health impacts. The use of ambient air concentrations to represent population exposures is justifiable when the health findings underlying the benefits analysis are similarly based on ambient concentration data and when the outdoor concentrations are correlated with personal exposures, as is the case for particulate matter (PM).

Exposure Assessment in Epidemiological Studies

The health benefits analyses reviewed by the committee have depended heavily on the estimated mortality impacts of PM. To better understand the role of and uncertainties in exposure assessment for such benefits analyses, it is important to examine characteristics of the exposure assessments used in the epidemiological studies on which the PM mortality effects were based.

Two classes of study designs have been used to assess mortality effects: time-series and prospective cohort studies (Kinney 1999). The time-series studies examine day-to-day associations between citywide mean daily outdoor PM concentrations and citywide daily death counts. This approach addresses the relationship between acute exposure and health. For example, deaths on a given day are related to PM concentrations on the same day or on a few previous days. In contrast, the prospective cohort studies examine differences between cities in mortality among individuals followed over an extended period and the variations in annual (or longer) mean outdoor PM concentrations. These studies are believed to address the relationship between chronic exposure and mortality. (See the Concentration-Response Function section for a further discussion of time-series and cohort studies.)

Population exposures are assessed in both designs using outdoor citywide average PM concentrations derived from regulatory air-quality monitoring data collected from a small number of sites in each city. Uncertainties may arise in using a citywide average to represent exposures of persons at risk because of spatial variations in ambient concentrations across a city, differences in penetration of ambient air pollution indoors, and the wide range in activity patterns of persons at risk. However, in the single-city time-series studies, central-site fine-particle measurements have been shown to correlate well over time with average population personal exposures (Rojas-Bracho et al. 2000; Sarnat et al. 2000). These findings support the validity of daily ambient PM measurements in capturing variations over time in population exposures to fine particles and strengthen the reliability of benefits estimates of acute health effects that depend on ambient PM concentrations.

Less is known about the reliability of central-site, long-term average, ambient PM concentrations in characterizing variations between cities in

average population exposures. The relationship between population exposures to pollutants of outdoor origin and ambient concentrations measured at central sites may differ across cities because of differences in local sources, indoor penetration efficiency, activity patterns, housing characteristics, and other geographic factors. For example, recent exposure studies highlighted variations across cities in the penetration of ambient PM to indoor environments as a result of weather-related factors, such as the prevalence of air-conditioner use (Rojas-Bracho et al. 2000; Sarnat et al. 2000; Janssen et al. 2002). This result implies geographic differences in the ability of ambient air-monitoring data to characterize population exposures accurately. This uncertainty will affect analyses that estimate benefits in diverse locations and in future years when housing characteristics that affect air-exchange rates may change. As more data become available, EPA should examine how this uncertainty affects benefits estimates and attempt to incorporate this source of uncertainty in an overall uncertainty analysis.

Another important characteristic of the exposure assessments in the epidemiological studies that evaluate PM mortality is their dependence on relatively simple measures of airborne PM, notably PM_{10} (most time-series studies) or $PM_{2.5}$ (most cohort studies). These size classifications incorporate a heterogeneous mixture of particles varying in size, composition, and source of origin. Furthermore, particle characteristics vary to some extent across locations and time. Because of this heterogeneity, the toxicity of different mixtures may vary.

Potential differential toxicity is especially important in a benefits analysis in which PM exposures and resulting health impacts are modeled in diverse locations and at future times, which may result in evaluating particle compositions that differ from those observed in the epidemiological studies used as a basis for analysis. The issue of differential toxicity is an area of active research. Although information is currently inadequate for determining the relative toxicity of different particle types, recent efforts to apportion the relative impacts of different source categories to observed health effects in the epidemiological setting show promise (Laden et al. 2000; Janssen et al. 2002). Lacking information on the relative potencies of different particle types, EPA has made the assumption of constant potency across particle types in its benefits analyses. As data become available, EPA should consider a range of alternative assumptions regarding relative toxicity and incorporate these assumptions in sensitivity or uncertainty analyses.

Regarding the collection of data, most epidemiological studies of air pollution health effects use routinely collected compliance monitoring data on a limited set of criteria pollutants for which toxicity is already well-established. To resolve issues of differential toxicity, EPA will need to expand its air-monitoring network to collect data for species other than the criteria pollutants. An improvement in the air-monitoring network should facilitate generation of more specific effect coefficients, and therefore the estimation of more reliable benefits estimates. Determining the responsible toxic components in the particle mix would also result in more effective regulations, because regulations could be better designed to control the sources responsible for generation of these components.

One exposure-related issue not typically considered explicitly in benefits analyses is that different categories of emissions sources may vary dramatically in their particle intake fractions, which are the fractions of material emitted that are actually inhaled by the population (Smith 1993; Bennett et al. 2002). Differences in intake fractions between sources may be much larger than the relative impacts of the source categories on ambient PM concentrations. For example, a kilogram of primary particle emissions from diesel vehicles may have an order of magnitude or greater impact on actual population exposure than a kilogram from stationary sources, even though they have similar impacts on ambient PM concentrations, because diesel exhaust is typically emitted closer to people (Marshal et al. 2001). EPA should develop standard methods and validation procedures for evaluating intake fractions for major source categories in different locations and conditions for use in benefits estimation. Over time, such information would also help to make effect coefficients derived from epidemiological studies more specific to actual exposures.

When effect coefficients from epidemiological studies are used to derive benefits estimates, they should be applied at the same spatial scales used in the original studies to avoid biased benefits estimates. EPA followed this approach in the benefits analysis for the HD engine and diesel-fuel rule, matching pollution concentrations with population estimates within grid areas similar in scale to metropolitan areas. However, the accuracy and reliability of a central-site monitor in representing human exposures may vary among population subgroups, resulting in differences in exposure misclassification across groups. Furthermore, exposure misclassification is likely to differ by pollutant, because a central-site monitor better represents citywide concentrations for pollutants that exhibit greater spatial homogene-

ity, such as $PM_{2.5}$ and sulfate, than for pollutants that exhibit small-scale spatial variations, such as coarse and ultrafine PM.

In summary, several important uncertainties in the use of exposure assessment in benefits analysis arise from the characteristics and interpretations of exposure assessment in the epidemiological studies. These uncertainties include the assumption that ambient concentrations consistently represent population exposures across locations and at future times, the assumption that sources affect population exposures in the same way that they affect modeled ambient concentrations, and the availability of health information only for aggregate PM measures, such as PM_{10}. Other important uncertainties in exposure assessment for benefits analysis result from methods used to model air quality under alternative regulatory scenarios. Air quality models are discussed in the following section.

Air-Quality Modeling

A critical link in determining the benefits of air pollution controls is to determine how emissions changes impact air quality. This determination is traditionally done using air-quality models of varying complexity. Models can be as simple as ones that assume a direct relationship between emissions and pollutant concentrations such that a 50% reduction in emissions results in a 50% reduction in ambient concentrations. These models are called linear rollback models. Air-quality models can also be considerably more complex, attempting to represent all the processes that have an important influence on ambient pollutant concentrations, including meteorology, emissions, chemistry, and physics across a broad three-dimensional region as a function of time. These models are generally called airshed models and have a wide range of capabilities and complexity. For pollutants that undergo complex nonlinear transformation, such as ozone and many components of PM, airshed models are often used, and EPA used these models in its more recent benefits analyses.

Airshed models solve the mathematical equations governing the physics and chemistry of pollutants in the atmosphere, such as the conservation of chemical species, that characterize the chemical production, chemical destruction, and transport by wind and diffusion. Hundreds of compounds are in the atmosphere; thus, the system of equations to solve could be very large and also nonlinear. Airshed models generally use a subset of all the species

and chemical reactions because not all the compounds are well-characterized.

A difference in models is the complexity of the chemistry reflected in the model. For example, typically 20 to 80 species are used when modeling ozone. The number of species used has grown as computer capabilities have expanded. The actual representation of the chemistry used by a model is called a chemical mechanism. For most regulatory modeling, the mechanism used is carbon bond IV mechanism (CB-IV), which is a relatively more streamlined approach than other modeling mechanisms available (Gery et al. 1989).

Another aspect of models is the spatial resolution or grid size. Most recent models allow the modeler to define the resolution. For example, a model might have a horizontal grid size of 80 kilometer (km) in one application and 36 km in another application. Newer models can also vary resolution in a single application, such as by using nested grids, and some can use grid scales as fine as 1 or 2 km. Finer resolution should improve model results and allow more accurate determination of exposure changes, especially for sources, such as mobile sources, that exhibit strong spatial gradients over fine spatial scales. However, the degree of improvement that can be achieved is limited by the resolution of the input data, such as the emissions inventory data.

EPA has recently used two air-quality models for ozone analyses: the regional oxidant model (ROM) and the urban airshed model variable (UAM-V). The latter model was used in the benefits analysis for the HD engine and diesel-fuel rule. ROM is an older model that uses a nonvariable grid resolution and has relatively little vertical resolution. In addition, ROM uses an early version of CB-IV, which does not have some of the most recent updates. UAM-V has a variable grid that uses nesting and a more recent version of CB-IV and allows for a more comprehensive treatment of meteorology. However, neither ROM nor UAM-V develops the meteorological fields internally; instead, they are provided by an external meteorological model.

To model PM, EPA has recently relied on the Lagrangian particle model (LPM), the climatological regional dispersion model (CRDM), the regional particulate model (RPM), and the regulatory modeling system for aerosols and deposition (REMSAD), which was used in the benefits analysis for the HD engine and diesel-fuel rule. The LPM and CRDM are relatively simple, describing the dispersion of pollutants without chemistry,

whereas the RPM and REMSAD are built on ozone models and include chemistry and some aerosol processes.

Currently, EPA is assessing the use of the community multiscale air-quality model (CMAQ). This model can be considered a state-of-the-science, "one-atmosphere" air-quality model and is to be used in regulatory and research applications. One atmosphere refers to inclusion of all relevant processes that determine the evolution of pollutants and their interactions. The one-atmosphere approach is particularly useful because it allows integrated study of all pollutants that are important to a specific region. One problem with CMAQ is that it requires extensive resources, staff, and computer time.

How well a model works in a specific application is determined by two factors: the fidelity of the model itself and the quality of the model application. The latter is currently the more dominant factor. Thus, the credibility of the model results is determined by the modeling process. A good model application will use and evaluate the most appropriate model inputs, including emissions, meteorology, and topography. EPA relied on the best model inputs that were available at the time in the benefits analysis for the HD engine and diesel-fuel rule.

Emissions are believed to have the greatest role in air-quality model uncertainty, followed by meteorology. Significant strides have been made to improve our understanding of emissions, and many of the biases in older inventories are believed to have been remedied. At this time, the ammonia emissions inventory is believed to be the most uncertain. Ammonia is important in PM and ozone modeling because it limits the production of secondary ammonium sulfate and ammonium nitrate. Considerable research is being dedicated to this issue and is viewed as an important step in reducing uncertainties associated with these secondary products.

It is difficult to make broad generalizations regarding the accuracy of model predictions. The accuracy will depend on the model used, the pollutant modeled, the quality of the application, the available data, the spatial and temporal resolution used, the averaging times, and the areas of interest. Model accuracy should be determined empirically by comparing model estimates to actual observations in a recent period. For the HD engine and diesel-fuel rule, EPA presented fairly extensive and appropriate data on the agreement between modeled and monitored concentrations of ozone. For example, EPA reported mean normalized biases (the average difference between model predictions and observations normalized by the observa-

tions) for ozone in the eastern United States ranging from -20% to +12%, depending on the region (northeast or southeast) and specific month (June, July, or August 1995) being modeled (EPA 2000, see Table 2A-1). Poor model performance (consistent negative biases of 30-50%) in the western United States led EPA to eliminate the western United States from the benefits analysis (EPA 2000, see p. 7-12). Although extensive evaluation of $PM_{2.5}$ estimates has not been possible to date due to the lack of monitoring data, this limitation may be readily addressed in future analyses with the recent establishment of a nationwide $PM_{2.5}$ monitoring program.

To increase the accuracy of modeling predictions, air-quality models are typically calibrated by comparing current air quality to model predictions for current conditions. Specifically, the model is used to calculate the fractional change in pollutant concentrations between a recent time period for which data exist (the base case) and a hypothetical future time period after emissions are controlled (the control case). The fractional change is then applied to the observed pollutant level for the recent time period to derive predictions of future concentrations when proposed emissions controls have been implemented. For example, if the current observed peak ozone level is 140 parts per billion (ppb), the simulated base case is 120 ppb, and the simulated control case is 90 ppb, the ratio of the modeled quantities (90:120 or 0.75, which is known as the correction factor) is multiplied by the observed ozone level (140 ppb) to yield a predicted future ozone concentration of 105 ppb for the control case. This approach may help reduce the bias introduced by modeling errors and, therefore, may be more accurate than using model results directly (absolute values) to estimate future pollutant levels. The committee recognizes that EPA appropriately used this approach for ozone for the benefits analysis for the HD engine and diesel-fuel rule but did not do so for $PM_{2.5}$, citing the lack of available $PM_{2.5}$ monitoring data.

The above discussion suggests that there are still significant uncertainties in model applications. Although these uncertainties are poorly characterized, they may be decreasing with time. The models that have been used in past benefits analyses noted above are subject to many uncertainties, the older ones more so than the newer ones. Many deficiencies of the older models have been remedied in the newest model, CMAQ, which may yield improved results. However, until tests are conducted that demonstrate the expected improvements in performance, CMAQ results will have to be treated as if they carry similar levels of uncertainty to current models.

One final point regarding models is that resource constraints often prevent simultaneously estimating concentration fields with fine spatial resolution over long periods and broad areas, such as the continental United States. Compromises must be made in one or more of these dimensions (area, time, or spatial resolution). Thus, models tend to be used to estimate concentrations over low-resolution grids, such as 36 × 36 km squares, for a few days or weeks. Such large spatial scales are more appropriate for secondary pollutants (such as ozone), which exhibit relatively smooth spatial variations, than for primary pollutants (such as diesel particles), which show strong spatial gradients. Using large spatial scales limits one's ability to assess differential exposure within urban areas and, therefore, risks to population subgroups. Although an evaluation of differential exposures would be valuable, it can only be accomplished if source emissions or air-monitoring data are available at similar or finer scales and if sufficient resources are allocated to the task.

Resource constraints have also limited the periods of air quality that have been modeled in recent benefits analyses. The temporal resolution of the model outputs in days or weeks is well-suited for modeling of episodic excursions in the standards implementation context, which is the purpose for development of most models, but relatively less useful for benefits analysis, for which longer exposure records would result in more reliable health benefits estimates. For the HD engine and diesel-fuel rule, full benefits analyses were conducted only for the year 2030, although exposure modeling results were also given for two intermediate time periods (2007 and 2020). Given the need for long-term exposure estimates and the national importance of the benefits analyses, the committee recognizes that overcoming the resource constraints is a critical need.

HEALTH OUTCOMES

Air pollution may give rise to health outcomes depending on specific pollutants and their concentration or exposure levels. The appropriate selection and interpretation of health outcomes is integral to any assessment of health benefits. Overall, the health effects of air pollution can be described on three levels. The first level is the way that air pollution adversely affects biochemical, physiological, and pathological mechanisms. The second level concerns the way these mechanisms translate into recognized

health effects (symptoms, impairment of activity, pain, or death). The third level involves translation of health effects into public health terms (incidence, prevalence, and mortality rates).

The pathophysiological effects will, if sufficiently severe, become manifest in individuals as illness (symptoms, impairment, pain, disability, death) and be attributed to certain clinical diagnoses, such as asthma or pneumonia. These effects may be associated with the use of medical services or medications. However, the health effects of many air pollutants, such as PM and ozone, lack specificity. In other words, the manifestations of these air pollutants may have other causes and cannot be understood independently from risk factors with the same outcomes.

The primary health effects resulting from air pollution have been observed in the respiratory and cardiovascular systems (ATS 1996, 2000). There is also growing evidence that air pollution exposure may cause reproductive and developmental effects (Brunekreef 1999). Short-term effects are typically minor and reversible at the levels of air pollution generally encountered in the United States, unless there is a preexisting condition that has already reduced the reserve or adaptability of the individual (ATS 1996, 2000). For example, certain air pollutants may cause a transient mild cough or eye irritation in a healthy person with plenty of functional reserve. However, for an older person who has advanced chronic respiratory disease and who is acutely ill with a respiratory infection, exposure to air pollution might result in death or some other clinically observable outcome, such as hospital admission. If the person would die soon regardless of the exposure to air pollution, the additional effect of the air pollution could be small in terms of life-years lost.[1] On the other hand, if the person would otherwise recover from the respiratory infection, the loss of life-years could be appreciable. Regarding the development of chronic disease, such as chronic obstructive pulmonary disease or asthma, the effects of air pollution are likely to act together with other risk factors, such as exposure to environmental tobacco smoke.

Most of the wide range of health outcomes described by the World Health Organization (WHO 2001) were considered by EPA for its benefits analyses (see Tables 2-1 and 2-5). However, many health outcomes were not quantified (EPA 1999, 2000; see Table 7-1) and included in the primary

[1]This scenario is referred to as short-term mortality displacement or harvesting.

analysis either because of insufficient data or because of possible double-counting of health outcomes. For benefits analysis, the most important distinction is between mortality and morbidity (illness). The use of mortality and specific morbidity outcomes in the context of benefits analysis is addressed in greater detail in the following sections. A discussion on causality follows these discussions. Because the outcomes evaluated in the EPA analyses reviewed by the committee were based on epidemiological studies, the following discussions focus on issues associated with using these studies as the basis for selection of outcomes.

Mortality

Mortality effects tend to dominate health benefits estimates when they are converted to life-years lost or to dollars (monetized). The attributes of mortality—socially important, accurately recorded, and one occurrence per person—make this outcome particularly suitable for health benefits analysis. However, although recorded by a doctor and classified using the International Classification for Diseases (ICD), the medical cause of death is subject to misclassification, especially for diseases of the cardiorespiratory system. Although misclassification may be a problem for epidemiological studies, it is less important for health benefits analyses, because the available estimates for valuation of mortality are relatively similar to those for specific causes of mortality, such as cardiovascular and chronic respiratory disease, considered in health benefits analyses for criteria air pollutants. This situation may change over time with the development of disease-specific cost estimates. For benefits analyses that use the American Cancer Society (ACS) study (Pope et al. 1995), a combined coefficient was used for cardiac and pulmonary mortality, avoiding the problem of cross-coding between these two disease categories.

Evidence is strongest for the effects of air pollution on adult mortality. However, EPA (1999, 2000) noted the emerging evidence linking air pollution and infant and child mortality. EPA did not incorporate this outcome into its primary benefits estimates because the body of evidence is too sparse. As more evidence emerges, EPA should consider incorporating this outcome into its primary benefits estimates.

Morbidity

For the estimation of health benefits, the committee notes that morbidity outcomes may be classified into five categories: pathophysiological, clinical diagnoses, symptoms, use of services, and effects on activity. For pathophysiological outcomes, examples mentioned in EPA analyses include effects on lung function and pulmonary inflammation. Changes in serum fibrinogen and heart-rate variability have also been found to be important physiological outcomes (Gold et al. 2000; Schwartz 2001a). These outcomes are useful for demonstrating toxicity and understanding mechanisms but have not been used for estimating health benefits either because they are unquantifiable or because they are not easy to translate into health effects to which a dollar value can be assigned. Furthermore, they might overlap with clinical outcomes that have been quantified, and including them would result in double-counting in the total benefits estimate. For example, quantification of changes in lung function may be possible because several cohort studies are available that show a relationship between reduced lung function and mortality. However, including benefits for changes in lung function in the total benefits estimate would result in at least some double-counting, because benefits for avoided mortality and acute respiratory morbidity are already directly estimated and included in the total benefits estimate.

The second category of outcomes (clinical diagnoses) includes chronic bronchitis, asthma attacks, and chronic asthma. The problem with the diagnosis of chronic bronchitis is that it covers a wide range of severity and time courses. At the mild end of the spectrum, it can be characterized as a chronic productive cough not associated with disabling symptoms and can even remit if other factors, such as smoking or occupational exposure, are reduced. At the other end of the spectrum, chronic bronchitis can be characterized by severe chronic airways obstruction accompanied by severe disability from shortness of breath and a need for long-term care. The same problems arise with the diagnosis of asthma because of the variation in frequency, duration, and intensity of asthma attacks.

The spectrum of severity in the study population (population evaluated in the epidemiological study) cannot be easily related to the target population (population characterized for the benefits analysis). The lack of clear cate-

gorization of severity also has implications for the application of monetization techniques, such as willingness to pay (see Chapter 6). For example, the severity of an avoided case of chronic bronchitis described in a valuation study should match the severity used to derive the concentration-response function; however, such matching is difficult if severity has not been adequately characterized in the epidemiological study. EPA has recognized the problems associated with the lack of clear categorization. In the primary analysis for the HD engine and diesel-fuel rule, EPA did not include cases of chronic bronchitis that remitted but provided an alternative calculation that included the remitted cases of chronic bronchitis as cases with the lowest severity rating (EPA 2000, see Table 7-25). EPA should continue to investigate and develop methods to account for different levels of severity when estimating health benefits.

The third category of outcomes is symptoms. Benefits for reductions in a variety of respiratory symptoms were estimated in the EPA analyses examined by the committee. EPA acknowledges the difficulties of estimating these benefits. One problem is that symptoms usually occur in clusters, such as cough, shortness of breath, and wheeze. Therefore, although estimating benefits for each symptom separately, EPA correctly cautions against adding them. Another problem lies in the valuation of symptoms, which are subjective and dependent on the severity of the effect.

The fourth category of outcomes relates to medical-care interventions resulting from health problems caused by air pollution. The outcome with most available data is the use of emergency rooms or hospital admissions. Different diagnoses will incur different costs because of variations in the length of hospital stay and the costs of treatments. EPA has recognized that studies of hospital admissions often use different groupings of ICD codes, which can cause overlap and double-counting.

In the United States, most evidence for the fourth category relates to admissions for individuals aged 65 or more, because the most accessible data for epidemiological studies are from Medicare. Many studies from outside North America report admissions across all ages, but these studies have not been used by EPA, presumably because they were considered less applicable to the United States. Use of the primary-care system is another potential outcome for this category that might be important at a population level. At present, only a few studies show an association between this outcome and exposures to air pollution, and these studies were all conducted outside the United States. The committee recommends that EPA consider

data from other studies to expand the age groups for which the outcomes apply and to incorporate the use of other relevant outcomes, such as the use of the primary-care system, in its benefits analyses.

The fifth category of health outcomes relates to the effect of air pollution on general well-being and activity level. EPA has primarily used major and minor restricted-activity days and workdays lost as the indicator for this category. A restricted-activity day is a day in which a person limits his or her normal activities because of illness. As a generalized measure of illness, the use of indicators of restricted activity has considerable potential; however, few studies are available. When restricted-activity days are included in an analysis, double-counting with other specific morbidity outcomes should be avoided. For estimating health benefits, EPA realizes that people may limit their activities for example, by staying indoors or taking more preventive medication, to avoid exposure to air pollution. However, these averting behaviors are probably not included in studies that link restricted-activity days to ambient air pollution. Nevertheless, averting behaviors may represent a substantial cost to society and should be acknowledged as being unmeasured in benefits analyses.

Any health benefits analysis presupposes that the concentration-response function can be applied to a population or to subgroups within the population. Accordingly, baseline measures of outcome prevalence or incidence are required. In the case of mortality, these measures are available. However, a baseline estimate for the benefits analysis that uses the same definition of disease severity or symptoms used in the epidemiological study that provided the concentration-response functions is generally not available for certain morbidity outcomes, such as diagnoses of chronic bronchitis and asthma attacks or symptoms of cough or shortness of breath. For example, no good baseline data are available to describe the incidence or pattern of asthma attacks in the community. That makes it difficult to estimate health benefits using results from a study of subjects who were not selected at random from the population of persons with asthma. The committee concludes that the uncertainties concerning the baseline should be included in any uncertainty analysis.

The goal of health benefits analysis is to consider all relevant health outcomes; exclusion of a health benefit from an analysis should be justified. However, information is insufficient at present to ensure that all relevant pathways and mechanisms of health effects are known. This state of uncertainty supports the use of total mortality as an outcome because it does

not require knowledge of the various pathways by which air pollution led to premature death. Regarding morbidity outcomes, restricted-activity days are also a useful measure because they encapsulate a variety of health outcomes and provide a generalized measure of well-being as stated above. More research should be conducted to provide better effect estimates for restricted-activity days.

Causality

A comprehensive discussion of causality is not necessary for a benefits analysis. This discussion is typically provided in the scientific documentation for the rule-making, such as the criteria document and other related reports, and in guidance provided by EPA's Science Advisory Board. However, a brief review of the evidence for causality is needed in a benefits analysis for two reasons. First, the review should provide justification for inclusion and exclusion of specific health outcomes considered for a given analysis. Second, a causal association between air pollution and health outcomes is a key assumption in a benefits analysis, and the uncertainty associated with this assumption needs to be incorporated into the final benefits estimates (see Chapter 5 on uncertainty).

The analyses reviewed by the committee relied on observational epidemiological studies. Approaches to assessing causality from these studies have often been based on consideration of a number of "viewpoints" described by Hill (1965).[2] The viewpoints included (1) temporal sequence of the associations (cause precedes effect), (2) consistency of the findings in different studies, (3) size of the effect, (4) monotonic exposure-response relationship, (5) coherence of the study results, (6) a plausible biological mechanism, (7) specificity of outcome, (8) analogy with similar exposures, and (9) evidence of change following an intervention.

These viewpoints should not be regarded as criteria because none is sufficient, and only one (the temporal relationship) is necessary for establishing causality. The use of such viewpoints as a checklist for causality was criticized extensively by Rothman (1986) and others (Lanes and Poole

[2]A similar list was put forth by a Surgeon General's Advisory Committee (1964), and a more highly elaborated system of criteria was developed by Susser (1973, 1977, 1988, 1991).

1984). Furthermore, Weed and colleagues documented the arbitrary and capricious use of causal criteria to justify predetermined conclusions (Weed 1994, 1997; Weed and Gorelic 1996; Weed and Hursting 1998; Potischman and Weed 1999). In a randomized trial, Holman et al. (2001) also provided experimental evidence showing little consensus among epidemiologists on use of causal criteria.

Hill's (1965) intention was to provide a framework for scientific reasoning that would allow a judgment to be made on the plausibility of explanations other than causality for associations reported in observational epidemiological studies. Therefore, using these viewpoints as the direct basis for a quantitative scale to express the likelihood of causality is inappropriate. However, reasoned consideration of these and other factors does and should influence the expert judgment about the plausibility of causal interpretation of studies.

Regarding Hill's viewpoints in the context of time-series studies that have evaluated the relationship between PM and health effects, the strongest arguments for causality have been the consistency of effects, the existence of an exposure-response relationship, and coherence of findings (Bates 1992). These arguments are bolstered by clear evidence of health effects in severe air pollution episodes (Ito et al. 1993) and the increasing mechanistic evidence linking particles to health outcomes (Gold et al. 2000; Pope et al. 1999; Peters et al 2000a,b; Peters et al. 2001). However, the toxicological evidence for health effects at low PM concentrations is mixed (Vedal 1997; Gamble 1998; Heyder et al. 1999).

Regarding Hill's viewpoints in the context of the major cohort studies that have evaluated the relationship between PM and health effects, the considerations differ somewhat from those for time-series studies. There are too few cohort studies to satisfy the consideration of consistency, and there is less supporting experimental evidence. However, there is some specificity for cardiopulmonary outcomes and lung cancer, considerable coherence of the study results, and an analogy with similar exposures (environmental tobacco smoke).

To determine the health outcomes that should be included in a benefits analysis or to estimate the uncertainty that is associated with the causal assumptions, several factors should be considered when interpreting epidemiological studies (Hennekens and Buring 1987). One factor to consider is the strength of the association between the pollutant and a health outcome. Appropriate statistical methods are typically applied to determine the

degree to which the observed association can be explained by chance (random variability), and EPA has included the random sampling error of the estimated concentration-response function in its uncertainty assessments (see Chapter 5). Another factor to consider is the possibility that the given association can be explained by confounding. For example, in cohort studies, it is important to control for such factors as education, smoking, environmental tobacco smoke, occupation, and region. In time-series studies, it is important to control for time-varying confounders, such as season, weather variables, and day of the week. Other factors that could influence study results are bias resulting from some aspect of the study design, such as the way in which study participants are selected, and error in the measurement of input data, such as exposure or disease data. A factor that should also be considered is whether the appropriate model has been used to evaluate the data.

Two general issues pertaining to confounding are relevant to air pollutants. First, air pollutants often have a common source and are subject to similar atmospheric dispersion processes. Therefore, their concentrations will tend to be correlated, and their independent effects are often difficult to disentangle using multipollutant models (see Concentration-Response Function section). However, the EPA analyses reviewed by the committee focused on PM and ozone, which tend not to confound one another. PM is a heterogeneous mixture, however, and the component responsible for the observed effects has not been determined. That adds to the uncertainty involved in estimating health benefits. The second issue is the possibility that some unknown factor that has not been controlled for explains the association. For example, variations in air pollution levels may also be associated with short-term (day-to-day) variations in societal activity, such as increased driving, construction, and industrial activity, which may increase the daily risk of health effects (Rietveld et al. 1999; Phillips et al. 2001). Similar arguments could be constructed for behavior related to the weather (Valberg and Watson 1998).

Little information was provided in the EPA analyses to judge the plausibility of the causal relationships assumed. As stated above, the evidence of causality should be summarized to justify the inclusion or exclusion of the health outcomes and to assess the uncertainty associated with the assumption of causality, which should be incorporated into the final benefits estimates, when possible. EPA should investigate and, if necessary, develop methods of evaluating causal uncertainty in relation to key outcomes, so that this uncertainty can be represented in the final benefits estimates.

CONCENTRATION-RESPONSE FUNCTIONS

A key element of benefits analysis is the "risk function" or "response function" that describes the quantitative association between ambient air pollution and the health effect. The term "concentration-response" used by the committee in this report reflects the assumption that measurements of ambient air pollutant concentrations adequately represent population exposures. The term coined from laboratory toxicology is traditionally known as dose-response. However, dose is rarely measured in air pollution epidemiology, and therefore, the committee has adopted the use of the term concentration-response to describe population exposure-response functions.

Benefits analysis assumes that a unit reduction in the concentration will lead to a specific level of reduction in the relevant health effects as reflected by the concentration-response coefficient. Several scientific methods are used to derive the response functions, and the inherent strengths and weaknesses of each approach affect the range of uncertainty of the resulting function. In the following sections, sources of concentration-response functions are described, issues associated with selecting epidemiological studies are discussed, and strengths and weaknesses of short-term and long-term epidemiological studies are addressed. Issues associated with threshold assumptions, analysis of population subgroups, and assumptions regarding effects lags are addressed in the final sections of this chapter. For each topic area, approaches chosen by EPA are discussed.

Sources of Concentration-Response Functions

Several types of scientific studies can be used to provide concentration-response functions. The following sections describe the strengths and weaknesses of using animal studies, human experimental studies, and epidemiological studies as sources for concentration-response functions.

Animal Studies As Sources of Response Functions

Animal toxicological studies typically involve controlled experiments of animals in chambers exposed to specified doses of pollutants. Animal studies have the advantage of applying fully controlled randomized experimental

designs and are able to specify accurately an exposure concentration and duration to a particular pollutant. Histological examination of tissues allows observation of specific biological, genetic, or biochemical changes and may assist in identifying whether a chemical is toxic through functional impairment, tissue damage, inflammation, hyperplasia, carcinogenesis, or mutagenesis. Toxicological studies also help elucidate the biological mechanism of the effect and may allow detection of minor changes, such as alterations in enzyme levels, that might not be symptomatic in humans.

Despite the advantages of animal studies, several substantial disadvantages limit their usefulness or create additional uncertainties in the assessment of human health benefits. First, use of animal studies in a health benefits analysis requires extrapolating from animal species to humans. Second, some effects observed in certain animals may not occur in humans because of differences in organ structure, metabolism, or other factors. Third, animals are typically exposed to high concentrations to elicit a statistically significant response in a study involving only a limited number of animals. Therefore, extrapolation from responses at high concentrations to responses at concentrations similar to ambient concentrations is required. Fourth, there is often substantial uncertainty about the shape of the concentration-response function at the lower concentrations. Fifth, only limited sample sizes and subgroups may be studied with toxicological studies making it difficult to capture the whole spectrum of sensitivities in human populations. Sixth, many important public health outcomes, such as hospitalization and work loss, and exposure conditions (for example, multiple decades at low levels) cannot be studied. Finally, replication of the actual mix of pollutants found in the ambient air has been difficult. Most laboratory studies have isolated one pollutant or a set of pollutants for experimental purposes. The strength of this approach is that it can help identify the most bioactive components of the ambient air pollutant mix. However, interactions among different pollutants as they exist under true conditions cannot be assessed.

In summary, toxicological animal studies may be useful in determining whether a given pollutant is toxic and in helping to elucidate potential biological mechanisms and pathways. However, application of results from animal studies to estimate the health benefits of ambient air pollution control requires several extrapolations, each of which involves considerable uncertainty. Because of these substantial limitations, data from human studies have typically been preferred to those from animal studies.

Human Experimental Studies As Sources of Concentration-Response Functions

Given the state of the science, benefits analyses should ideally rely on human data rather than animal data. Aside from the population-based epidemiological studies discussed in the next section, human data can be obtained from functional assessments made in clinical studies and in studies of occupationally exposed workers. Clinical studies typically involve controlled experiments of human volunteers in exposure chambers. A common example of this technique involves observing changes in lung function or asthma symptoms in subjects exposed to varying levels of ozone over set periods of time. Advantages of this method include the preciseness with which the exposure and the response may be assessed, the lack of a need to extrapolate from animal species to humans, the ability to detect subclinical changes, and the ability to determine whether an exposure has an effect. Disadvantages of using clinical studies for assessing benefits include limitations in sample size, the range of certain subgroups that can be studied (typically excluding children, older persons, and those with relatively severe cardiovascular disease), the use of only acute exposures, and the difficulty in replicating the entire mix of ambient pollutants. As with animal studies, clinical studies cannot examine such outcomes as visits to the doctor or hospitalization.

Health data on occupationally exposed workers can address some of the shortcomings of clinical studies. However, workers have historically been exposed to much higher concentrations of air pollutants than the general population. Therefore, extrapolation to lower exposure concentrations is sometimes necessary. Although the baseline health of the worker population is better than that of the general population, the elevated, long-term exposures experienced by workers can provide insight into the toxicity or lack of toxicity of specific components of ambient air pollution. The epidemiological studies described in the next section allow one to estimate concentration-response functions for the general population exposed to ambient air pollutant concentrations.

Epidemiological Studies As Sources of Concentration-Response Functions

Observational epidemiological studies involve the study of humans in

real situations. Thus, human health effects are observed under a wide range of behaviors and conditions using this method. Specific to air pollution, epidemiological studies have shown that when air pollutant concentrations changed at fixed site monitors, a corresponding change occurred in the observed incidence of many health effects.

The primary advantages to using epidemiological studies are the lack of a need to extrapolate results across species and the ability to study a wide range of health outcomes, including mortality, hospitalization, and respiratory symptoms. Different subgroups also may be examined in detail. For example, the correlation between air pollution and reported health may be examined in individuals with severe asthma or older people with chronic bronchitis. An additional advantage is that researchers can examine a wide range of pollutants, pollutant mixes, and averaging times by considering multiple seasons or locations.

Disadvantages to using epidemiological studies include imprecision in the measurement of exposure and response, potential confounding of the results, and the possibility of spurious findings. Furthermore, it is difficult to determine the underlying mechanism causing the effect or to identify the specific chemical constituent responsible for the observed effect. As noted previously, finding a statistically significant association between a health effect and a specific air pollutant does not prove causality.

Another issue with epidemiological studies is that some degree of extrapolation is required. Because epidemiological studies are time-consuming and expensive, all potential health effects of ambient air pollution in all cities or regions are not investigated. Therefore, using concentration-response functions from epidemiological studies for benefits analyses will require extrapolation from the study populations to the target populations in the benefits analysis. The extrapolation of results from epidemiological studies assumes a fairly similar spatial relationship between pollution monitors and population. Therefore, researchers often assume that a given change in the ambient air concentration of an air pollutant, such as PM_{10}, will result in the same increase in risk in the applied area of the benefits analysis as in the original study area. Given the replication of health effects associated with PM_{10} in many parts of the United States and the world (Holgate et al. 1999), this assumption appears reasonable.

The process of extrapolation, however, involves several uncertainties. First, the underlying socioeconomic or health status of the population for which the benefits analysis is being conducted may differ from that of the

original study. Second, the human susceptibility for effects of ambient air pollution varies within populations. For example, short-term effects of air pollution on mortality are particularly associated with cardiopulmonary death. Age or underlying diseases, such as atherosclerosis or diabetes, may further modify this effect (Zanobetti et al. 2000; Zanobetti and Schwartz 2001). Similarly, some evidence exists that educational attainment itself, or as a marker for socioeconomic status or exposure, may modify the effects of long-term exposure to air pollution (Krewski et al. 2000; Pope et al. 2002). Therefore, the point estimate of the concentration-response function may differ across populations, depending on the distribution of these factors within the target population. One additional factor that may add uncertainty to the extrapolation is the variation in the composition of an air pollutant in different locations. For example, as discussed previously, PM is composed of different chemical constituents and particle sizes and, therefore, may differ in toxicity from one location to another. This uncertainty may diminish for gaseous pollutants, such as ozone.

Sources of Concentration-Response Functions For EPA's Analyses

For the health benefits analyses reviewed by the committee, EPA used concentration-response functions from epidemiological studies. The committee believes that this approach was appropriate because using epidemiological studies avoids many of the problems encountered in the other types of studies. This approach cannot be generalized to all analyses because epidemiology might not be able to provide valid concentration-response functions for some toxic agents. Ultimately, the plausibility of the concentration-response function and the uncertainty surrounding it should be reflected in the benefits analysis. This requires a brief summary of the evidence for causality, including animal toxicity and human clinical studies.

Selecting the Appropriate Epidemiological Studies

A key issue in benefits analysis is selecting the concentration-response estimate from those in several studies. For each epidemiological study, a concentration-response function is derived for a given population, observa-

tion time, and exposure. This function is treated as the best estimate of the underlying true function. A small portion of the inherent uncertainty between the observed best estimate and the true unknown function is usually described with confidence intervals. The uncertainty increases with each level of generalization of the observed results. For example, the uncertainty increases when the results are transferred to nonparticipants of the same population and even more when they are transferred to other ages, ethnicities, disease status, cities, regions, or countries. Therefore, a better estimate might be an average function derived from several studies that evaluate different cities and populations.

Epidemiologists have sometimes given higher credence to concentration-response functions that are averages of a number of independent but valid single studies on the same pollutant and response. However, in air pollution epidemiology, studies conducted in different regions may involve air pollution of different quality and composition and, thus, different health relevance. Difficulties are also encountered, however, when a local study is judged as methodologically less valid than some nonlocal, more sophisticated study, possibly leading to the decision to ignore local concentration-response functions and adopt the nonlocal functions. Given these uncertainties and necessary judgments, it is not surprising that analysts do not have a universally accepted paradigm or set of rules for selecting concentration-response functions. International experts have written guidelines on behalf of the European office of the World Health Organization describing the difficulties and the range of decision options available (WHO 2000, 2001). Different projects have applied different processes to select the set of studies to derive concentration-response functions. Recent studies have tended (1) to include rather than exclude studies conducted in the same region or country for which the analysis has been done; (2) to rely on variance-weighted mean estimates rather then single studies; and (3) to combine North American and western European estimates but not assume quantitative comparability of these data with those from studies conducted in South America or Asia (Ostro et al. 1996; COMEAP 1997; Ostro and Chestnut 1998; Ostro et al. 1999; Künzli et al. 2000).

The committee believes that generally the most appropriate approach is to calculate a weighted mean estimate rather than choose one study from a set of studies conducted on the same health outcome to derive the concentration-response function. This estimate should be based on the available single estimates and a weighting procedure that takes the uncer-

tainty of each point estimate into account (for example, an inverse-variance approach). Additional weighting schemes, such as those giving higher weight to local studies, to more recent studies, or to some measure of central tendency or prior evidence, may be applied. Whatever process is adopted, the inclusion or exclusion of studies and the weighting scheme should be justified and clearly explained. The committee notes that the selection process ultimately should focus only on the validity and transferability of a study and not on whether a result is statistically significant.

Selecting a particular study or a set of studies is not the only challenge. The analyst must also choose among a vast array of models and specifications within the original study or studies. In fact, researchers are usually encouraged to evaluate the sensitivity of their results to alternative models and specifications. For example, in short-term-effect studies, researchers often present results for different lag structures; for different controls for weather, time, and seasonality; and for single- and multipollutant models. Sometimes the results are relatively robust (insensitive) to these alternative model specifications. However, more often, the results vary. If the results vary using different models and specifications, the analyst should attempt to evaluate the study carefully and use the most appropriate risk estimates.

The use of single- versus multipollutant models may have a large influence on risk estimates. Some researchers have attempted to identify one or more pollutants responsible for a given health effect by entering several pollutants as independent variables into an explanatory regression model. The results of multipollutant modeling can be difficult to interpret and do not necessarily yield more reliable results.

If the effect estimate for a pollutant of interest is unchanged by the addition of a second pollutant to the regression model, then the added pollutant is either not a confounder or, because of measurement error or variable misspecification, its confounding effects cannot be detected. If the addition of a second pollutant to a multivariate regression model changes the effect estimate for the pollutant of interest, this observation may be the result of collinearity among the pollutants. Regression estimates can vary widely with the inclusion or exclusion of highly correlated covariates. Including a highly correlated copollutant increases the standard error of the estimate and the associated confidence interval and often results in highly unstable effect estimates for the pollutant of interest. In addition, the relative effect estimates of the two pollutants may be influenced by the relative magnitudes of their exposure measurement errors. Given these potential uncertainties,

a multipollutant model cannot be assumed to yield necessarily the most accurate results. Failure to include a causally linked copollutant, however, can result in both omitted variable bias and imprecision in the estimate of risk associated with exposure to the primary pollutant.

A way to estimate the importance of a given pollutant in the presence of correlated copollutants is to examine the pollutant of interest in alternative cities. For example, it is useful to consider the effects of PM_{10} in a range of cities that have both high and low correlations with other relevant copollutants, such as ozone or sulfur dioxide (Schwartz 2000a). A consistent PM_{10} effect estimate under such different circumstances supports the notion of a causal relationship.

The findings of Sarnat et al. (2001) are also important in assessing the usefulness of multipollutant models. This study demonstrated that over time, ambient concentrations of gaseous pollutants were not associated with personal exposure to these gases but were associated with personal exposure to $PM_{2.5}$. $PM_{2.5}$ personal exposure was in turn associated with ambient concentrations of $PM_{2.5}$. The authors concluded that ambient $PM_{2.5}$ may be a suitable surrogate for personal $PM_{2.5}$ exposure and that ambient gaseous pollutants may also be surrogates, not confounders, for $PM_{2.5}$. These relationships should be examined in future studies to establish whether this important finding can be generalized to other settings.

In summary, statistics alone cannot resolve the question of the relative influence of various pollutants on a given health outcome. Statistical results must be interpreted by experts familiar with the strengths and limitations of various modeling approaches and causal mechanistic information. In many cases, expert judgments may have as large a role as the numerical analysis in interpreting such data for benefits assessment.

EPA's Selection of Epidemiological Studies

Overall, the committee found that the studies selected by EPA for use in its benefits analysis were generally reasonable choices. However, the criteria and process by which EPA reached its decisions are not clearly articulated in many cases. EPA should document clearly the rationale for its selection of studies and concentration-response coefficients, because these choices require judgment on the part of the analyst. For example, weighted averages of coefficients are used in some cases and coefficients

from single studies are used in others, even when multiple studies are available. It would be reasonable in some cases to extend the effect estimates to age groups beyond those used in the original study. For example, estimates for respiratory symptoms are provided only for those ages included in the original studies, such as children aged 7 to 14, when the effects are probably not restricted to this narrow age group.

Although the EPA analyses may rely on different underlying studies, the concentration-response functions for various outcomes tend to be similar across different studies. Thus, inconsistencies in the selection of studies may have little impact on the overall estimates; however, comparability across analyses might be difficult. One advantage of using the same estimate as used in other benefits analyses (rather than deriving new, even more appropriate estimates) is the ability to compare benefits estimates across different areas, times, and studies (Künzli et al. 2000).

Specifically, for long-term effects of air pollution on mortality, EPA used results from the large ACS study (Pope et al. 1995) and evaluated use of the Harvard six cities study (Dockery et al. 1993) in the sensitivity analyses. Given the size and precision in the ACS study, this decision appears to have been a reasonable one. However, the large ACS sample size is not necessarily grounds for adoption of this study over the others with smaller samples. For example, the Harvard six cities study has some advantages over the ACS study, such as the use of a random population sample and the careful placement of monitors for the study. Furthermore, the educational attainment of the Harvard six cities study was more representative of the general population than that of the population in the ACS study, indicating that the effect estimate from the ACS study might be low for the general population as educational attainment appears to be an effect modifier.

Given the reanalyses of both studies (Krewski et al. 2000), the recent extended analyses of the ACS study (Pope et al. 2002), the availability of a third U.S. cohort study (Abbey et al. 1999; McDonnell et al. 2000), the Swedish case-control study on lung cancer mortality and air pollution (Nyberg et al. 2000), and the publication of the first European cohort study (Hoek et al. in press), EPA should thoroughly review the selection of the best estimate for long-term effects of air pollution on mortality. EPA may want to consider derivation of a weighted mean estimate from the cohort studies following review of the entire database.

Short-Term Versus Long-Term Studies

Air pollution epidemiological studies have investigated the association of acute and chronic health outcomes with both short- and long-term exposures (Künzli et al. 2001a). For benefits analysis, a clear understanding of the strengths and weaknesses of studies that examine the effects of short- and long-term exposure is crucial because health benefits resulting from improvements in air quality will appear on different time scales. Therefore, key issues regarding these studies are reviewed in this section. Given its dominant role in benefits analysis and the large quantity of available data, mortality is the focus of this discussion. Data on morbidity outcomes is less comprehensive and must be improved, especially if the value assigned to mortality decreases and morbidity outcomes play a more dominant role in the benefits analyses.

Short-term exposures typically have been studied using time-series methods that test the hypothesis that daily changes in air pollution are followed within days or weeks by changes in mortality or morbidity among the exposed population in a specific area. For example, a time-series study could investigate the association between emergency room visits and air pollution each day in a community over several years. Figure 4-1 provides a simplified model of the proposed course of events that time-series studies investigate. An inherent feature of these studies is the assumed length of time periods, which are typically short (days). The duration of exposure (t_a) is usually a 1-day average, often extended to a simple average over a few days. The lag time (t_b) between exposure (t_a) and a change in health (t_c) is usually set at 1 or a few days. The time during which effects of the exposure might occur (t_c) is usually fixed at 1 day.

Time-series studies have the distinct advantage of reducing potentially confounding or omitted variables because population characteristics, such as age, smoking habits, occupational exposure, and health habits, are basically unchanged over the study period. The only factors that are likely to vary with daily mortality and morbidity are environmental and meteorological conditions. Some studies have shown that mortality and morbidity vary for unknown reasons with day of week, calendar date, and certain social factors (Möller et al. 1999; Phillips et al. 1999; Smyth et al. 1999). If these factors vary on a daily basis with air pollution, then they could be confounders. However, time-series studies have typically taken into account certain weather factors, such as temperature and relative humidity, that vary on a

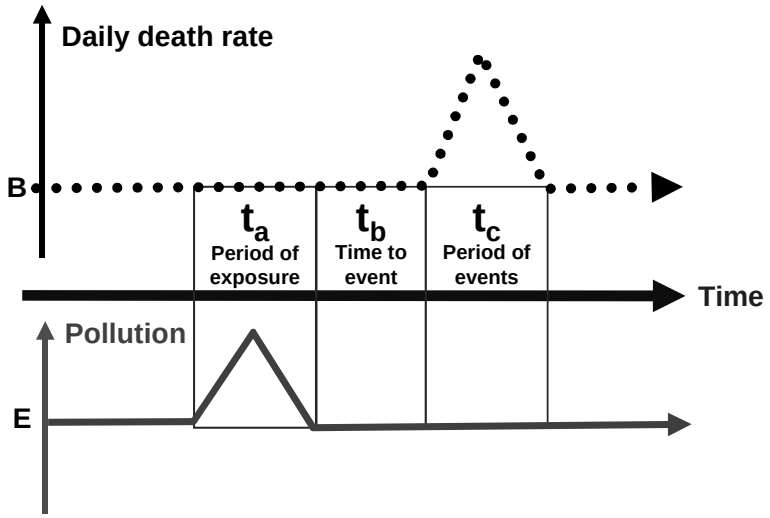

FIGURE 4-1 Simplified model of the time-series analysis, which addresses whether a change in air pollution is followed by changes in mortality (or morbidity). All time periods (period of exposure, lag time to the event, duration of the period with changed outcome rates) relevant to the model are short (a few days or weeks). B is the long-term average death rate in the population. E is the long-term average ambient pollutant concentration.

daily basis with air pollution (Samet et al. 2000; Katsouyanni et al. 2001). Another advantage of the times-series studies is that the large number of these studies offers the unprecedented opportunity to evaluate confounding, effect modification across cities, and consistency of results.

Although time-series studies are attractive for benefits analysis because the concentration-response estimates from these studies may provide strong evidence for the occurrence of an effect, most time-series studies underestimate the short-term impact. Furthermore, they do not include effects that result from long-term exposure (Künzli et al. 2001a,b).[3] Only a few time-

[3]The committee acknowledges that time-series study may capture cases where long-term exposure has moved a person to a state of higher susceptibility to air pollution. However, because exposure history is not a part of the time-series study design, the time-series studies do not distinguish between cases where cumulated exposure has had an impact on terminal susceptibility and cases where past air pollution exposure is irrelevant.

series studies formally consider exposure over several days before the mortality event (Schwartz 2000b; Braga et al. 2001; Zanobetti et al. 2002). The more recent time-series studies that have evaluated longer exposure periods have generated effect estimates two to three times higher than those using a single-day exposure. These findings support the assumption that restricting relevant time periods to 1 or 2 days excludes short-term health effects that take a few more days or even weeks to occur. For example, an exposure may trigger a myocardial infarction, resulting in intensive care unit treatment rather than an immediate death; however, the infarction may lead to death in some cases during the period of convalescence, which may be 2 weeks after the primary event (Künzli et al. 2001a,b; Martuzzi 2001). Therefore, when evaluating the short-term effects of air pollution, analysts should use the results of time-series studies that integrate over several days or weeks the exposure period and the time period to the event (cumulative or distributed lag models) rather than those that restrict these time periods to 1 or 2 days.

Another disadvantage of using time-series studies to assess the impact of air pollution on mortality for a benefits analysis is that they do not provide information about the amount of time lost resulting from the premature deaths, which is critical information for certain valuation techniques that use life-years lost rather than mortality cases (see Chapter 6). There is evidence that the time lost due to short-term exposure is more than just displacement of a few days. Using both frequency- and time-domain methods, Zeger et al. (1999) and Schwartz (2000b) showed that most air-pollution-associated mortality is not due to such displacement. Specifically, the average life-shortening for cardiovascular deaths appears to be greater than 2 to 3 months. However, deaths resulting from chronic obstructive pulmonary disease (COPD), which consists mainly of emphysema and chronic bronchitis, may be consistent with a short-term mortality displacement hypothesis (Schwartz 2000c, 2001b).

The committee notes that the ideal epidemiological study should assess both the cumulated long-term life-time exposure and the more recent exposure patterns, including the exposure period shortly before death. The best approach would be to assess the effect of various degrees of exposure on life expectancy using a randomized intervention study, but this study design is not feasible in the field of ambient air pollution research. Studies of long-term exposure have involved both cross-sectional and prospective cohort study designs. Cross-sectional mortality studies compare baseline mortality

across populations rather than the fluctuation of rates over short time periods. However, these studies lack information on individuals, making proper adjustment for relevant covariates, such as smoking and occupational exposure, difficult, if not impossible. Therefore, cross-sectional comparisons of mortality rates are inferior sources of concentration-response functions for long-term effects.

A better approach to assessing the influence of air pollution on baseline mortality rates is the prospective cohort mortality study. These studies follow large groups of people living under environmentally distinct conditions over time and assess both their exposures and relevant health covariates. For benefits analyses, a more complete assessment of the impacts of air pollution is generated by prospective cohort mortality studies (Dockery et al. 1993; Pope et al. 1995; Abbey et al. 1999; Pope et al. 2002; Hoek et al. in press) than by time-series studies. Whereas time-series studies do not assess the effect of air pollution on baseline mortality rates, cohort studies directly measure the association of long-term exposure on life expectancy. The cohort studies are not restricted to a narrow time period between exposure and health effect but assume that some cumulated exposure experience might result in shorter life expectancy due to, for example, illnesses, such as chronic bronchitis or lung cancer (Abbey et al. 1995; Nyberg et al. 2000; Pope et al. 2002). The assumptions made are comparable to those made regarding the health effects of smoking for which the cumulated pack-years are associated with shortening of life expectancy. In contrast, the time-series approach would address the question of whether the risk of dying might be higher a few days after a day of smoking 20 cigarettes compared with smoking only five cigarettes.

Figure 4-2 illustrates the effect of long-term exposure on mortality evaluated in a prospective cohort study. In this graph, the average frailty or susceptibility of death (probability of dying) is lowest after the neonatal period and then increases over a lifetime leading to death at time T_0. Repeated and cumulated long-term exposures from regular smoking or ambient air pollutants, for example, may shift the frailty level upward. As a result, time of death is shifted to a younger age (T_e), Δt being the life-years lost when life expectancy in the exposure scenario is compared with that in a no-exposure scenario. Therefore, this approach assumes that the shortening of life is due to not only the exposure pattern experienced shortly before death, but also the long-term cumulated exposure experience. The exposure in the days before death might be influential because it could cause

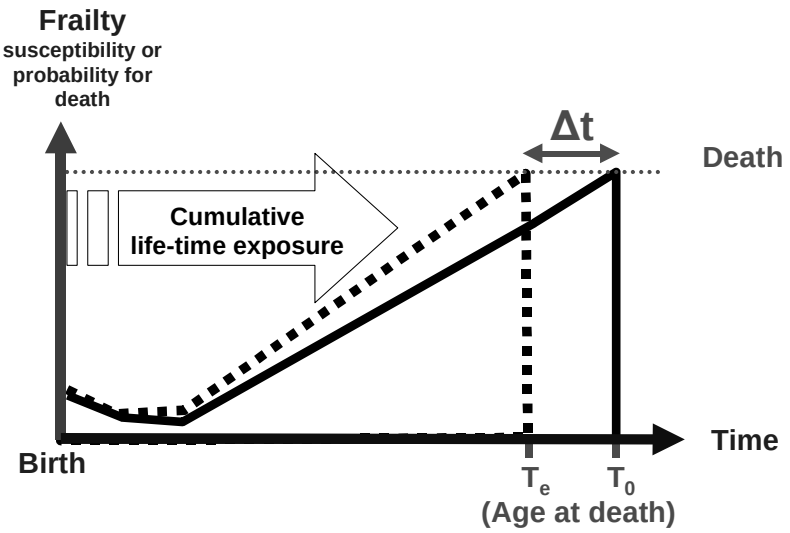

FIGURE 4-2 Simplified model for long-term exposure effects in which the frailty level increases over the lifetime, resulting in death. The basic model without exposure reaches death at age T_0. Under the long-term exposure scenario (lifetime air pollution or long-term smoking), the increase in the frailty level is accelerated, reaching death at an earlier point in life, T_e. Δt reflects the life-years lost due to exposure.

additional life-years to be lost (increase Δt). Thus, there might be cases in which only the past long-term exposure contributed to life-shortening, cases in which only short-term exposure before death contributed to life-shortening, and cases in which both aspects of exposure contributed to life-shortening (Künzli et al. 2001a,b). Prospective cohort studies could include the cases of mortality due to short-term exposure, as well as cases resulting from long-term exposure.

The particular advantage of the cohort studies is the measurement of time (person-years or life expectancy). Accordingly, concentration-response functions from cohort studies theoretically can be used to provide estimates of the number of lives lost due to air pollution each year and the amount of life-years lost in a population. From a public-health perspective, life-years lost might be more relevant than annual number of mortality cases.

A disadvantage of the prospective cohort air pollution study is the ecological assignment of exposure, which is inherently imprecise.[4] Thus, the exposure measures may increase the statistical variability in the data, reducing the ability to observe effects. This source of variation may be particularly great when long time periods are evaluated for increasingly mobile populations. Another key disadvantage is assessing differences in mortality across different populations, in contrast to time-series studies that evaluate differences within a single population. Therefore, cohort studies may have a greater chance of confounding because of some unmeasured population characteristics.

Although cohort studies measure person-time directly, the available cohort studies present only death rates and numbers of deaths, not estimates of life-years lost. The additional number of people dying in 1 year and the life-years lost by these deaths are linked by the age distribution of these individuals at death. However, this distribution has not been published, and assumptions must be made about the age structure of air-pollution-attributable deaths. Several authors have used local data on age- and disease-specific death rates to estimate indirectly the amount of time lost due to air pollution. However, these estimates have larger uncertainties than the estimates for the lives lost (Brunekreef 1997; Pope 2000; Sommer et al. 2000; Miller 2001).

Despite some differences in the central estimates of concentration-response coefficients, the cohort studies from the United States suggest important associations between long-term exposure and time to death and appear to be the most appropriate study design to assess the impacts of air pollution on health. One finding that supports using the cohort study design over the time-series study design is the reported association between lung cancer and air pollution exposure (Nyberg et al. 2000; Pope et al. 2002). Lung cancer involves a process in which exposure to carcinogens results in the development of clinical disease (cancer) many years after exposure. In such cases, death may occur regardless of the ambient air quality during the days before death. Given this fact, the time-series study will not typically capture such cases of death, although air pollution was an underlying long-term contributing factor that led to life-shortening. In other words, the

[4]Ecological assignment of exposure means that all people living in an area are assigned the same exposure concentration based on the monitor in that area.

total life-years lost from short-term exposures is not identical to the total life-years lost observed in the cohort studies. The latter quantity is greater than the quantity that can be captured by the time-series study. Therefore, it is essential to use the cohort studies in benefits analysis to capture all important effects from air pollution exposure.

EPA's Approach to Using Short-Term and Long-Term Studies

For the estimation of mortality benefits, EPA had to choose a concentration-response function from the small number of cohort studies or from the large number of time-series studies. The committee finds that EPA's decision to base mortality estimates primarily on the cohort studies is reasonable. Given the uncertainties in the cohort data and the publication of new studies, EPA should reevaluate the database of studies now available to derive a concentration-response function.

EPA used the time-series studies to derive benefits estimates of selected morbidity outcomes for its primary analyses. For example, in the benefits analysis for the HD engine and diesel-fuel rule, hospital admissions for various illness, such as those for COPD, were estimated using time-series studies. The committee believes that consideration of these studies is appropriate to estimate acute effects from short-term exposure. However, recent investigations have revealed problems for some models, but not all, that use the S-plus statistical software to fit generalized additive models (GAM) to the data (HEI 2002). Therefore, this problem should be thoroughly investigated to determine the effect on the coefficients used in the benefits estimation.

Analysts face the challenge of interpreting the findings from the studies that evaluate short-term and long-term exposure. Theoretically, a cohort study measures the total life-years lost due to long-term exposure to air pollution. However, the available cohort studies use crude measures of cumulative exposure, such as the annual mean value, and the effects of short-term exposures are unlikely to be fully captured in the cohort studies. Thus, the overall effect estimates may be a combination of effects from long-term exposure plus some fraction from short-term exposure. The amount of overlap is unknown (Künzli et al. 2001a,b). Zeger et al. (in press) provides a new analytical framework for addressing the contributions of various exposure time periods to the total life-years lost. The approach

has not been applied to benefits analysis but may provide further insight into the contribution of short-term and long-term exposure to life-years lost and may clarify the amount of overlap. Determining the amount of overlap between the study types is an important research need.

Finally, short- and long-term effects of air pollution on morbidity have been investigated less extensively than mortality. Therefore, the analytical uncertainties may be larger for morbidity than mortality. For long-term exposure, the contribution of PM and other pollutants to development of cardiopulmonary morbidity is an important area of scientific inquiry. EPA could play a leading role in addressing these knowledge gaps.

Linearity and Thresholds

The shape of the concentration-response functions may influence the overall estimate of benefits. The shape is particularly important for lower ambient air pollution concentrations to which a large portion of the population is exposed. For this reason, the impact of the existence of a threshold may be considerable.

In epidemiological studies, air pollution concentrations are usually measured and modeled as continuous variables. Thus, it may be feasible to test linearity and the existence of thresholds, depending on the study design. In time-series studies with the large number of repeated measurements, linearity and thresholds have been formally addressed with reasonable statistical power. For pollutants such as PM_{10} and $PM_{2.5}$, there is no evidence for any departure of linearity in the observed range of exposure, nor any indication of a threshold. For example, examination of the mortality effects of short-term exposure to PM_{10} in 88 cities indicates that the concentration-response functions are not due to the high concentrations and that the slopes of these functions do not appear to increase at higher concentrations (Samet et al. 2000). Many other mortality studies have examined the shape of the concentration-response function and indicated that a linear (nonthreshold) model fit the data well (Pope 2000). Furthermore, studies conducted in cities with very low ambient pollution concentrations have similar effects per unit change in concentration as those studies conducted in cities with higher concentrations. Again, this finding suggests a fairly linear concentration-response function over the observed range of exposures.

Regarding the studies of long-term exposure, Krewski et al. (2000)

found that the assumption of a linear concentration-response function for mortality outcomes was not unreasonable. However, the statistical power to assess the shape of these functions is weakest at the upper and lower end of the observed exposure ranges. Most of the studies examining the effects of long-term exposure on morbidity compare subjects living in a small number of communities (Dockery et al. 1996; Ackermmann-Liebrich 1997; Braun-Fahrländer et al. 1997). Because the number of long-term effects studies are few and the number of communities studied is relatively small (8 to 24), the ability to test formally the absence or existence of a no-effect threshold is not feasible. However, even if thresholds exist, they may not be at the same concentration for all health outcomes.

A review of the time-series and cohort studies may lead to the conclusion that although a threshold is not apparent at commonly observed concentrations, one may exist at lower levels. An important point to acknowledge regarding thresholds is that for health benefits analysis a key threshold is the population threshold (the lowest of the individual thresholds). However, the population threshold would be very difficult to observe empirically through epidemiology, because epidemiology integrates information from very large groups of people (thousands). Air pollution regulations affect even larger groups of people (millions). It is reasonable to assume that among such large groups susceptibility to air pollution health effects varies considerably across individuals and depends on a large set of underlying factors, including genetic makeup, age, exposure measurement error, preexisting disease, and simultaneous exposures from smoking and occupational hazards. This variation in individual susceptibilities and the resulting distribution of individual thresholds underlies the concentration-response function observed in epidemiology. Thus, until biologically based models of the distribution of individual thresholds are developed, it may be productive to assume that the population concentration-response function is continuous and to focus on finding evidence of changes in its slope as one approaches lower concentrations.

EPA's Use of Thresholds

In EPA's benefits analyses, threshold issues were discussed and interpreted. For the PM and ozone National Ambient Air Quality Standards (NAAQS), EPA investigated the effects of a potential threshold or refer-

ence value below which health consequences were assumed to be zero (EPA 1997). Specifically, the high-end benefits estimate assumed a 12-microgram per cubic meter ($\mu g/m^3$) mean threshold for mortality associated with long-term exposure to $PM_{2.5}$. The low-end benefits estimate assumed a 15-$\mu g/m^3$ threshold for all PM-related health effects. The studies, however, included concentrations as low as 7.5 $\mu g/m^3$. For the Tier 2 rule and the HD engine and diesel-fuel rule, no threshold was assumed (EPA 1999, 2000). EPA in these analyses acknowledged that there was no evidence for a threshold for PM.

Several points should be noted regarding the threshold assumptions. If a threshold is assumed where one was not apparent in the original study, then the data should be refit and a new curve generated with the assumption of a zero slope over a segment of the concentration-response function that was originally found to be positively sloped. The assumption of a zero slope over a portion of the curve will force the slope in the remaining segment of the positively sloped concentration-response function to be greater than was indicated in the original study. A new concentration-response function was not generated for EPA's benefits analysis for the PM and ozone NAAQS for which threshold assumptions were made. The generation of the steeper slope in the remaining portion of the concentration-response function may fully offset the effect of assuming a threshold. These aspects of assuming a threshold in a benefits analysis where one was not indicated in the original study should be conveyed to the reader. The committee notes that the treatment of thresholds should be evaluated in a consistent and transparent framework by using different explicit assumptions in the formal uncertainty analyses (see Chapter 5).

Analysis of Population Subgroups

Regulators may want to understand the differential effects of regulations for a variety of reasons, including the question of equity, the desire to achieve the maximum benefit, and regional interests. Differential health effects may occur because the effects of the regulation result in different reductions in population exposures or because subgroups within the population vary in response to a given exposure reduction. The latter effect can occur because baseline rates of health outcomes may vary across subgroups or because the concentration-response function may differ across

subgroups. Differential health effects may be of interest across geographic regions; across demographic categories of sex, age, or race; or across groups with varying health status (such as persons with asthma versus those without asthma), socioeconomic status, or behavioral factors (such as smokers versus nonsmokers).

To investigate the possibility of differential exposure reductions, exposure must be estimated on relevant spatial scales. Exposure is most easily estimated spatially at the level of town or region. However, analysts may need to consider other dimensions. For example, the association between health effects and proximity to heavily traveled roads may need to be investigated. For this study, the exposure could be estimated using geographical information system (GIS) methods, and possible health effects could be evaluated using epidemiological data relating increases in respiratory problems to traffic proximity. It might also be important to model exposure at the small area level, such as inner-city environments. As indicated previously, when estimating health benefits associated with finely mapped exposures, concentration-response functions should be derived from epidemiological studies conducted at similar geographical exposure scales.

Given the assumption that the relative risk of a health outcome is proportional to the level of exposure, the predicted number of cases for a specific health outcome will also be proportional to the baseline rate for that health outcome. Because baseline risks for subgroups can vary by an order of magnitude or more, the additional number of cases in two subgroups of the same size can vary by that amount. Therefore, special attention should be given to the subgroups at the greatest baseline risks, where the attributable risks would be greatest (Künzli 2002; Röösli et al. in press).

As noted above, expected health benefits across subgroups may vary because of differences in the concentration-response function. The differences may arise from variations in vulnerability due to age, preexisting disease, or factors related to socioeconomic status. Alternatively, regional differences in concentration-response functions may reflect differential toxicity of regional PM. Recent studies suggest some differences in concentration-response functions across different subgroups, such as persons from cities with higher traffic-related primary emissions (Katsouyanni et al. 2001; Rijnders et al. 2001) or persons with different educational levels (Krewski et al. 2000; Pope et al. 2002).

The complexity and uncertainty of analyzing subgroups in benefits analysis can be illustrated with the results of the recent extended follow-up of

the ACS cohort (Pope et al. 2002). The study observed different sizes of the concentration-response function for men and women, the concentration-response function being smaller and nonsignificant for women. Furthermore, associations of air pollution with mortality were strong in groups with low educational level but decreased with increased educational level. No association was observed among those with more than a high-school degree. Specifically, relative risks of all-cause mortality were estimated to be 1.2 per 10 $\mu g/m^3$ for the subgroup with less than a high-school education and 1.0 per 10 $\mu g/m^3$ for those with more than a high-school education. The underlying reasons for these differences across subgroups are not known, but several explanations may be possible.

Depending on the interpretation, analysts may choose different strategies for benefits analysis, reaching potentially different results. For example, if a sex difference is explained genetically (only men being susceptible to effects of long-term exposure) only men would be included in the analysis—modeling and deriving exposure, baseline health frequencies, and concentration-response functions for men only. However, differences in exposure to air pollution may cause concentration-response functions to appear dissimilar. For example, in the Pope et al. (2002) study, the disappearance of an effect in the high-education group may be explained partly by errors in the assigned exposure. Specifically, the wealthier individuals typically live in the cleaner parts of cities. Thus, the assigned concentration may overestimate true residential exposure among the wealthy, but underestimate exposure among the economically disadvantaged. Given this scenario, the true exposure may be lower among the high-education group and the variation of exposure for this group across cities may be smaller than the measures used in model estimation. This may reduce the statistical power in the high-education group and bias the concentration-response function. If the subgroup findings are driven by exposure measurement issues, a subgroup benefits analysis may be less appropriate than simply applying the aggregate total risk function for the full population.

EPA's Analysis of Subgroups

EPA analyzed subgroup-specific effects only to the extent that benefits were assessed for the subgroups considered in the original studies (for example, restriction by age for mortality [more than 30 years] or for lower

respiratory symptoms among children [7-14 years]). Other potentially relevant strata were not considered. EPA should explain its decision to extrapolate (or not) to other subgroups. Risk assessors should be aware of the paradox that a sophisticated assessment requiring more complex analytical tools may have more biased findings than a simple aggregated analysis.

As a final note, a hierarchical set of models (models that increase in complexity with each stage of the analysis) may help to distinguish factors that affect the results. For example, in the first stage of a multicity analysis, regressions may be run relating mortality and morbidity to air pollution for each city. In the second stage of the analysis, city-specific factors, such as socioeconomic and demographic factors or copollutants, may be examined to determine whether these factors influence the first-stage city-specific effect estimates. Samet et al. (2000) tested for effect modification of the PM_{10}-mortality association among the 90 cities used in the study. Using citywide statistics, they tested for potential modification using local socioeconomic-related variables, including household income, educational level, public transit use, and unemployment level. None of those factors helped to explain the city-specific pollution effects. However, the variable representing educational level had a moderate association with the regression coefficient for PM_{10}.

Effect Lags and EPA's Assumptions

Understanding long-term disease processes is important for benefits analysis. For example, certain health benefits resulting from a change in air quality may occur only after several years. Although it appears that mortality following short-term exposure to PM occurs within a relatively short time, little is known about the temporal relationship between longer-term exposure and mortality as demonstrated in the prospective cohort studies. For example, the ACS study (Pope et al. 1995) provided little information as to whether the observed geographic differences in mortality risks are due to a 1-year average or some multiyear history of PM exposures preceding mortality. Thus, it is not known which period of exposure is the most important and how quickly benefits from air pollution reductions will appear in the case of long-term disease processes. In the Swedish lung cancer study (Nyberg et al. 2000), effects were strongest for the exposure 20-30 years ago. For other outcomes, other time periods may be relevant.

The time course relating exposure to outcome is an important assumption in benefits analysis, especially when long-term mortality effects dominate the analysis, as occurs in PM analyses. It is important because health benefits that occur far into the future may count less based on the way the benefits are monetized. In EPA's benefits analyses for the Tier 2 rule and the HD engine and diesel-fuel rule, EPA assumed a weighted 5-year time course of benefits in which 25% of the PM-related mortality benefits were assumed to occur in the first and second year, and 16.7% were assumed to occur in each of the remaining 3 years. Although recommended by EPA's Science Advisory Board, the committee found little justification for a 5-year time course and recommends that future benefits analyses more fully account for the uncertainty regarding lags in health effects by incorporating a range of assumptions and probabilities on the temporal relationship.

CONCLUSIONS

- EPA's approaches to exposure assessment have evolved considerably over time because of the continued improvement in the models and the marked increase in available monitoring data for key pollutants. Overall, the methods used in the most recent EPA analysis reviewed by the committee (heavy-duty engine and diesel-fuel analysis) represent an appropriate and reasonably thorough application of the available data and models for exposure assessment.
- Many uncertainties associated with exposure assessment need to be addressed more fully as more data become available. These uncertainties include the assumptions that ambient pollutant concentrations consistently represent population exposures across locations and at future times, that sources affect actual exposures in the same way that they affect ambient concentrations, and that all particle types have a constant potency.
- The appropriate selection and definition of adverse health outcomes is integral to any assessment of health benefits. A wide range of health effects associated with exposure to air pollution has been described and most of them have been carefully considered by EPA. However, many health outcomes are not quantified because of insufficient data or because of the potential for double-counting.
- Data for many health outcomes are restricted to a specific age group, and EPA did not extrapolate those data beyond the age ranges pro-

vided in the studies. However, recent studies conducted outside the United States provide information on certain health outcomes with broader age ranges and on outcomes, such as use of the primary care system, not evaluated by EPA.

- EPA used concentration-response functions from epidemiological studies. The committee supports this approach because using epidemiological studies avoids many of the problems encountered with animal toxicity and human clinical studies.
- The studies selected by EPA for use in its benefits analyses were generally reasonable choices. However, the criteria and the process by which EPA reached its decisions are often not clearly articulated.
- For the analysis of mortality, EPA used cohort studies to derive benefits estimates in the analyses reviewed by the committee. The committee supports this approach. Compared with time-series studies, cohort studies give a more complete assessment of the long-term, cumulative effects of air pollution. Furthermore, the particular advantage of cohort studies is that they provide data to estimate the number of life-years lost in a population, not just the number of lives lost, thus allowing for several valuation methods to be used.

RECOMMENDATIONS

- As in all other stages of the benefits analysis, EPA should justify and clearly describe the assumptions and methods used to assess exposure, choose health outcomes, and select studies and concentration-response functions, paying careful attention to assessing and communicating key sources of uncertainty.
- Because pollution modeling rarely addresses the smaller-scale issue of how local concentrations from specific source categories interact with human time-activity patterns, EPA should examine how different major source categories, for example, mobile versus large stationary sources, affect total exposures per unit emissions.
- EPA has typically made the assumption of equivalent potency across particle types because of insufficient scientific information. As more data become available, EPA should strengthen its benefits analyses by evaluating a range of alternative assumptions regarding relative particle toxicity and incorporate these assumptions in sensitivity or uncertainty analyses.

- The lack of clear categorization of severity of certain health outcomes in benefits analyses has implications for the quantification and the valuation of these outcomes. Although EPA has made some attempt to recognize this issue, it should continue to develop and improve methods used to reconcile differences between the severity of disease described in air pollution epidemiology and that commonly used to develop estimates of background disease prevalence and incidence.
- EPA should consider data from U.S. and non-U.S. studies to extrapolate beyond the age groups evaluated and incorporate other relevant outcomes not evaluated in its current benefits analyses.
- EPA should give more emphasis to the assessment, presentation, and communication of changes in morbidity. Although often difficult to quantify, these factors may begin to play a more dominant role in benefits analysis if the value assigned to mortality decreases.
- EPA provided little information in the benefits analyses reviewed by the committee on causal association between particular types of air pollution and adverse health outcomes. EPA should summarize the evidence for causality to justify the inclusion or exclusion of the health outcomes and to assess the uncertainty associated with the assumption of causality.
- EPA should investigate and, if necessary, develop methods of evaluating causal uncertainty relating to key outcomes so that this uncertainty can be represented in the final benefits estimates.
- Although the committee believes the use of the ACS study to derive premature mortality estimates was reasonable, EPA should thoroughly review its selection of the best estimate for long-term effects of air pollution on mortality. Several new studies have been published since the ACS study, including an extended analysis of the ACS study, a new U.S. cohort study, and other non-U.S. studies. EPA should also consider whether the derivation of a weighted mean estimate from the cohort studies is appropriate following review of the database.
- To evaluate short-term effects of air pollution, EPA should use concentration-response functions from studies that integrate over several days or weeks the exposure period and the time period to the event (cumulative or distributed lag models) rather than those that restrict these time periods to 1 or 2 days.
- Although the assumption of no thresholds in the most recent EPA benefits analyses was appropriate, EPA should evaluate threshold assump-

tions in a consistent and transparent framework using several alternative assumptions in the formal uncertainty analysis.

- The committee found little justification for the 5-year time course of exposure and outcome assumed in the more recent EPA analyses and recommends that EPA more fully account for the uncertainty regarding lags in health effects by incorporating a range of assumptions and probabilities on the temporal relationship.
- EPA is encouraged to estimate and report benefits by age, sex, and other demographic factors. The committee recognizes, however, that evaluating the differences for various subgroups adds complexity and uncertainty to the analysis and that caution must be exercised in the interpretation of such results.

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5

Uncertainty

There are several major barriers to broad acceptance of recent EPA health benefits analyses. One barrier is the large amount of uncertainty inherent in these analyses, and another is the manner in which the agency deals with this uncertainty. A third barrier is that projected health benefits are often reported as absolute numbers of avoided death or adverse health outcomes without a context of population size or total numbers of outcomes. Areas of uncertainty include air-quality modeling, population demographics and heterogeneity, intersubject variability, health and exposure baselines, compliance with control measures, effectiveness of controls in reducing pollutant emissions, validity and precision of concentration-response functions and use of alternative models (linear, nonlinear), estimation of these functions as relative effects (relative risks) or absolute effects (risk differences), relative toxicity of mixture components, and applicability of these functions to target populations of regulatory concern. These uncertainties are rooted in incomplete scientific knowledge. When benefits are estimated for future target populations, the cumulative magnitude of the uncertainties can be formidable. Many of them can be reduced by further research, but on the whole, they are likely to remain high.

Even great uncertainty does not imply that action to promote or protect public health should be delayed. Decisions about whether to act, when to act, and how aggressively to act can only be made with some understanding of the likelihood and consequences of alternative courses of action. The potential for improving decisions through research must be balanced against

the public health costs incurred because of a delay in the implementation of controls. Complete certainty is an unattainable ideal.

Health benefits analyses compare alternative scenarios that would result with and without regulatory action. As a consequence, these analyses are inherently speculative and their results unverifiable. Because only one regulatory option can be chosen by decision-makers, the outcomes of the remaining regulatory options, including the baseline with no action (if not chosen), can never be directly observed.

Analyses of health benefits should represent the uncertainties in the choices facing decision-makers and society at large (Hattis and Anderson 1999). Analyses should attempt to provide insight into the variability of impacts (among persons, places, and other dimensions of interest) and the extent and sources of uncertainties in the results. The representation of uncertainty requires a good faith appraisal of the imperfection in the state of information about these impacts (Hattis and Burmaster 1994). Uncertainty assessment should not overrepresent or underrepresent the quality and completeness of available information.

This chapter discusses EPA's current approach to assessing uncertainty in health benefits analyses for air pollution control regulations. The agency's analysis of the health benefits for the final Tier 2 vehicle emissions standards and gasoline sulfur control rule-making (EPA 1999a) is used for illustration. The chapter outlines a revised approach that would reflect overall uncertainty more realistically, in part by using probabilistic expressions of expert judgment. The chapter also briefly reviews the history of probabilistic uncertainty assessment in EPA health benefits analyses under the Clean Air Act.

This chapter is confined to uncertainty in the analysis of health benefits expressed solely in terms of health. Although uncertainties in the monetary valuation of health benefits and in the analysis of regulatory costs are not considered, the committee notes that there are great uncertainties in those analyses as well.

EPA'S APPROACH TO UNCERTAINTY ANALYSIS

EPA uses a two-part approach to assessing uncertainty in health benefits analyses that rely on epidemiological studies as the source of estimated concentration-response functions, although different approaches are some-

times used, especially when epidemiological evidence is lacking (EPA 1997). The first part is a primary analysis, which produces numerical estimates or projections of each health benefit in the form of a probability distribution. This analysis incorporates only one source of uncertainty: the random sampling error in the epidemiological study or studies that provide the estimated concentration-response function. The second part of the uncertainty assessment is an array of ancillary analyses in which many other sources of uncertainty are considered in several disparate ways.

Primary Uncertainty Analysis

The primary uncertainty analysis produces a numerical estimate of each health benefit EPA believes to be plausible for a particular regulatory action. Typically, the benefit is expressed as a number of deaths or cases of an adverse health event that will be avoided in the United States in a future year if some regulatory action is taken. The year chosen is often far into the future to allow for the action to be implemented, for the implementation to result in exposure reductions, and for the reduced exposures to result in health benefits. In the Tier 2 analysis, the chosen year was 2030.

EPA reports each numerical health benefit estimate in the form of a probability distribution and summarizes the distribution by reporting its mean and 5th and 95th percentiles. The distribution assigns a nonzero probability to every possible value including the null hypothesis of no benefit. The mean of the distribution is interpreted as the expected benefit based upon the analysis performed. The 5th and 95th percentiles are defined as a credible range within which the true benefit value will lie with a 90% probability (EPA 1999a, p. 3-26).

The solid line in Figure 5-1 shows the probability distribution from EPA's primary analysis of avoided mortality for the proposed Tier 2 rule for the year 2030. The mean of the distribution (which is also the median and the 50th percentile) is 4,307 avoided deaths among persons 30 years of age and older. The 5th and 95th percentiles are 2,671 and 5,891 avoided deaths, respectively (EPA 1999a, p. 6-3).

The probability models in EPA's primary analyses incorporate only one of the many sources of uncertainty in these analyses: the random sampling error in the estimated concentration-response function derived from either an epidemiological study or a meta-analytic or pooled aggregation of two or

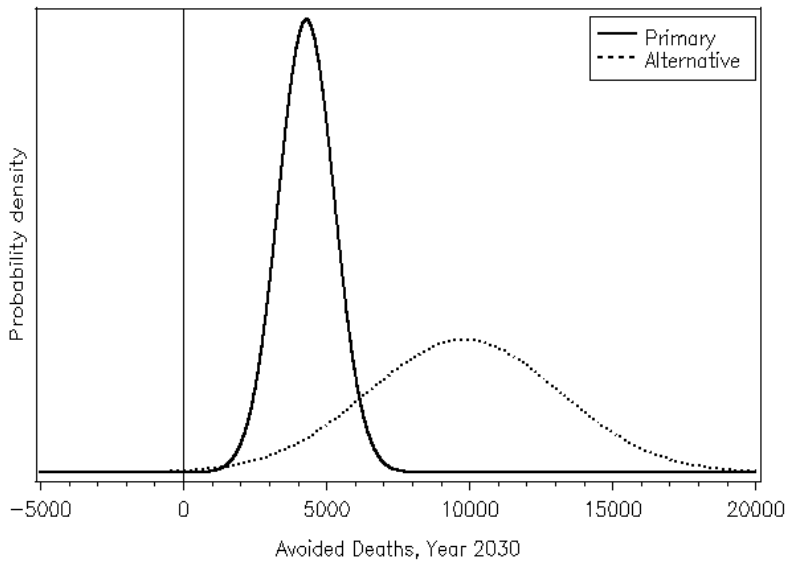

FIGURE 5-1 Probability distributions from primary and alternative analyses of avoided mortality for Tier 2 analysis. Source: Data from EPA 1999a.

more such studies. In a meta or pooled analysis of separate studies, a summary estimate of the concentration-response function is produced by averaging study estimates that may include ones that vary in strength and ones that suggest little or no effect. To estimate avoided mortality for the Tier 2 rule, the agency chose an estimated concentration-response function from a log-linear (Poisson regression) analysis of results from a study by the American Cancer Society (Pope et al. 1995). For a change in concentration from 9 to 33.5 Fg/m^3, the result was an estimated relative risk of 1.17 with a 95% confidence interval of 1.09 to 1.26 (EPA 1999a, p. C-2). The random sampling error represented by this confidence interval is the only source of uncertainty in the agency's probability distribution for avoided mortality. The incorporation of additional sources of uncertainty would widen the distribution.

EPA correctly notes that incorporating only the uncertainty from random sampling error in concentration-response function estimates into its primary health benefits analyses "omits important sources of uncertainty, such as the contribution of air quality changes, baseline population incidences, projected populations exposed, transferability of the concentration-

response function to diverse locations, and uncertainty about premature mortality" and "would provide a misleading picture about the overall uncertainty in the estimates" (EPA 1999a, p. 3-26).

Ancillary Uncertainty Analyses

EPA assesses all other uncertainties in a second part of each health benefits analysis. The agency begins with a list of as many key uncertainties as it can identify. The list compiled for the Tier 2 analysis is given in Table 5-1. Much of this uncertainty results from unavoidable and expected variability or heterogeneity in concentration-response functions estimated by epidemiological studies. Some of it results from baseline statistical variation, as no study has infinite sample size and all study populations differ in their distributions of background causes of health outcomes and in their distributions of susceptibility to toxic agents. Projection of future baselines, such as the death rate to be expected 30 or more years in the future if no action is taken, are particularly uncertain. Important uncertainty is also produced by variation in study design, data collection, and statistical analysis. Although there may be other uncertainties that have not been identified, EPA typically makes no allowance for these unidentified sources of uncertainty.

EPA takes a variety of approaches regarding these identified uncertainties. Some are merely mentioned. Other uncertainties are discussed qualitatively with regard to the direction and, sometimes, the magnitude of the impact that they are likely to exert on the mean value of the probability distribution. For example, in the discussion of the epidemiological study providing the estimated concentration-response function for avoided mortality in the Tier 2 analysis, EPA referred to downward biases from the relatively healthy study population and from intercity migration of study participants, which the agency believed would counteract an upward bias associated with historical air-quality trends (EPA 1999a, p. C-1).

For selected sources of uncertainty, EPA conducted supplemental calculations, alternative calculations, and sensitivity analyses (EPA 1999a, p. 3-19). These terms have specific meanings in EPA health benefits assessments. Supplemental calculations "provide additional information about specific health effects, but are not suitable for inclusion in the primary or

TABLE 5-1 Key Sources of Uncertainty in the Tier 2 Benefits Analysis

1. Uncertainties Associated with Concentration-Response (C-R) Functions
– The value of the ozone- or particulate matter (PM)-coefficient in each C-R function. – Application of a single C-R function to pollutant changes and populations in all locations. – Similarity of future year C-R relationships to current C-R relationships. – Correct functional form of each C-R relationship. – Extrapolation of C-R relationships beyond the range of ozone or PM concentrations observed in the study.
2. Uncertainties Associated with Ozone and PM Concentrations
– Estimating future-year baseline and hourly ozone and daily PM concentrations. – Estimating the change in ozone and PM resulting from the control policy.
3. Uncertainties Associated with PM Mortality Risk
– No scientific literature supports a direct biological mechanism for observed epidemiological evidence. – Direct causal agents within the complex mixture of PM responsible for reported health effects have not been identified. – The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. – Possible confounding in the epidemiological studies of $PM_{2.5}$ effects with other factors (such as other air pollutants, weather, and indoor and outdoor air). – The extent to which effects reported in the long-term studies are associated with historically higher concentrations of PM rather than the concentrations occurring during the period of study. – Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated with Possible Lagged Effects
– What portion of the PM-related long-term exposure mortality associated with changes in annual PM levels would occur in a single year, and what portion might occur in subsequent years.
5. Uncertainties Associated with Baseline Incidence Rates
– Some baseline incidence rates are not location-specific (such as those taken from studies) and might not accurately represent the location-specific rates of interest. – Current baseline incidence rates might not approximate baseline incidence rates in the year 2030. – Projected population and current demographics—used to derive incidences—might not approximate future-year populations and demographics.

(Continued)

TABLE 5-1 *Continued*

6. Uncertainties Associated with Aggregation of Monetized Benefits
– Health and welfare benefit estimates are limited to the available C-R functions. Thus, unquantified benefit categories will cause total benefits to be underestimated.

Source: Adapted from EPA 1999a, Exhibit 3-3, p. 3-20.

alternative estimates due to concerns about double-counting of benefits or the high degree of uncertainty about the estimates" (EPA 1999a, p. 3-21). The supplemental analyses in the Tier 2 report pertained to short-term mortality, infant (postneonatal) mortality, ozone mortality, asthma attacks, restricted-activity days, and ozone-related cardiovascular disease (EPA 1999a, pp. 3-23, 3-24, A-1).

In other contexts, both EPA's alternative calculations and sensitivity analyses would be called sensitivity analyses (Morgan et al. 1990; Greenland 1998). The distinction for EPA lies in its judgment of their plausibility. Alternative calculations "are based on relatively plausible alternatives to the assumptions used in deriving the primary benefit estimates" (EPA 1999a, p. 3-21). Sensitivity analyses "examine the sensitivity of estimated benefits results to less plausible alternatives to the assumptions used in the primary analyses" (EPA 1999a, p. 3-25). For both calculations and analyses, assumptions or sources of uncertain quantities are varied and the mean of the health benefit probability distribution is recomputed.

In all cases, the alternative calculations and sensitivity analyses are conducted for only one source of uncertainty at a time. In addition, they are conducted only to determine the sensitivity of the mean of the probability distribution from the primary analysis to modified assumptions and information sources. With one exception, the spread of the health benefit probability distribution, as gauged by the distance of the interval between its 5th and 95th percentiles, is not affected. EPA's rationale for focusing only on the mean is that an "attempt to assign probabilities to these alternative calculations . . . would only add to the uncertainty of the analysis or present a false picture about the precision of the results" (EPA 1999a, p. 3-21). EPA does not discuss why adding to the uncertainty of the analysis would be inappropriate. Noting that some analyses of health benefits of air pollution reductions (Lang et al. 1995; Holland et al. 1999) have included the assignment of "probabilities to ranges of parameter values for different endpoints,"

EPA argued that "the estimated points on these distributions are themselves highly uncertain and very sensitive to the subjective judgements of the analyst. To avoid these subjective judgements, we choose to allow the reader to determine the weights they would assign to alternative estimates" (EPA 1999a, p. 3-21).

For the Tier 2 analysis, alternative calculations were performed for an alternative source of the estimated concentration-response function and for life-years saved rather than avoided deaths as a measure of health benefit (EPA 1999a, pp. 3-21, 23). Sensitivity analyses were conducted for thresholds and alternative lag structures (EPA 1999a, p. 3-25).

The one exception to the exclusive focus on the mean of the health benefit probability distribution occurs when an alternative calculation involves the use of a different study to provide the estimated concentration-response function, which has its own standard error estimate. The broken line in Figure 5-1 shows the probability distribution when the concentration-response function from an analysis of the Harvard six cities study (Dockery et al. 1993) is used. This study produced a higher point estimate of the relative risk, so the mean of the probability distribution is higher (10,000 avoided deaths). The alternative study was smaller, however, so its estimate had more random sampling error and the distribution is wider. The 5th and 95th percentiles are 5,000 and 15,000 avoided deaths, respectively.

CRITIQUE OF EPA'S CURRENT UNCERTAINTY ASSESSMENTS

Numerical projections appear to be essential in health benefits analyses, and probability distributions can be used to describe the uncertainty in these analyses. Issues arise, however, over which sources of uncertainty the distributions should incorporate, how to incorporate them, and how to present the results. EPA's decision to incorporate only one source of uncertainty, the random sampling error in the estimated concentration-response function, into the probability distributions resulting from its health benefits analyses is worth reconsidering. The committee agrees with the agency's judgment that its current practice produces health benefits probability distributions that give "a misleading picture about the overall uncertainty in the estimates" (EPA 1999a, p. 3-26). In particular, the distributions suggest that there is less uncertainty, perhaps much less, than is actually present.

The committee finds that the mean of the distributions should not be interpreted as "best" estimates, and the intervals between the 5th and 95th percentiles of the distributions should not be interpreted as "90 percent credible intervals," within which "the true benefit lies with 90 percent probability" (EPA 1999a, p. 3-26).

The committee agrees with EPA's statement that it would require expert judgment to specify probability distributions for many of the uncertain components of the health benefits analyses. In these cases, probability would be used not only in its connotation of variability[1] but also in its connotation of subjective uncertainty or lack of complete belief as well (Hacking 1984; Poole 1988; Lindley 2000). EPA is correct that the elicitation of expert opinions in the form of probability distributions is a difficult and uncertain process (Morgan et al. 1990; Cooke 1991; Pate-Cornell 1996). The committee does not agree, however, that these difficulties are sufficient reasons for not trying to obtain such advice. Nor does the committee find any reason to avoid the attempt on the ground that it "would only add to the uncertainty of the analysis or present a false picture about the precision of the results" (EPA 1999a, p. 3-21). On the contrary, by growing wider, the health benefits probability distributions would more accurately depict the uncertainty and lack of precision in the analyses. As difficult and uncertain as these specifications are, they are preferable to EPA's current practice of treating important and highly uncertain model components as though they were certain.

The probability models from which standard errors are estimated for concentration-response-function estimates from observational epidemiological studies are less than certain as well. These models would have a firm theoretical foundation only if study populations were randomly sampled from target populations and exposure concentrations were randomly allocated to study participants (Greenland 1990; Poole 2001). In observational studies such as the American Cancer Society study (Pope et al. 1995) and the

[1]In risk analysis, a distinction is often made between characterization of variability (the true variation in a parameter over time, space, or persons) and uncertainty (ignorance about the true value of the parameter). Variability is characterized primarily to provide information about the true distribution of exposure and risk and to suggest opportunities for control or to provide a sense of equity. Uncertainty is characterized primarily to give a sense of the confidence that can be placed in study results and to help in setting priorities.

Harvard six cities study (Dockery et al. 1993), neither random sampling nor random exposure allocation was used. Nevertheless, analysts use probability models for these design features in analyzing observational data because larger observational studies have less variability than smaller ones and account for the incomplete enumeration of the population of interest.

The applicability of probability models for random variability to observational data is less than perfectly secure, but their use is preferable to assuming that there is no variability related to study size in observational results. The use of probability models for uncertainties involving expert judgment is also preferable to assuming that these uncertainties do not exist.

Many of the key uncertainties in these analyses may be characterized only subjectively by reference to expert judgment. The question is not whether to rely on expert judgment but how best to elicit and summarize the views of experts and how to incorporate them into the analysis. Probability distributions are a legitimate and useful way to express the uncertainties in expert judgments. Incorporation of those uncertainties as probability distributions into the primary analysis would likely change the expected value and widen the resulting probability distribution for each health benefit. The result will include more of the uncertainty in the health benefits assessment.

The alternative calculations and sensitivity analyses conducted by EPA help to describe the uncertainty in the analyses, but they are not sufficient. The major problems with them are that EPA consigns them to an ancillary status and not to the primary analysis, that the various sources of uncertainty are considered one at a time, and that EPA explicitly offers no judgment as to the relative plausibility of the alternative scenarios considered in these analyses. Without a combined, simultaneous assessment of multiple uncertainty sources, it is impossible to gain an appreciation of the overall magnitude of the uncertainty in the analysis. The committee does not agree with the agency's decision to have the reader determine the plausibility and relative weighting of alternative assumptions and data sources and integrate these assessments across uncertainty sources.

In its current analyses, EPA does not systematically or probabilistically address the extension of results beyond a study population's age range. The typical assumption is that the health-outcome-rate ratio is constant across age; however, this assumption is seldom tested and seldom has any strong etiological justification, even when compared with a simple alternative, such as a constant-rate difference. For example, the method of extrapolating to additional age groups can be of crucial importance if the study

population excludes elderly persons who are at especially high baseline risk and the target population includes a sizable proportion of elderly persons. A large portion of the overall health benefit may then be projected for an age range that has not been studied. In such cases, the mixture of model and data would be tilted heavily toward the model.

Two additional illustrative examples are thresholds for adverse effects and lag structures.[2] EPA considers implausible any threshold for mortality in the particulate matter (PM) exposure ranges under consideration (EPA 1999a, p. 3-8). Although the agency conducts sensitivity analyses incorporating thresholds, it provides no judgment as to their relative plausibility. In a probabilistic uncertainty analysis, EPA could assign appropriate weights to various threshold models. For PM-related mortality in the Tier 2 analysis, the committee expects that this approach would have resulted in only a slight widening of the probability distribution for avoided mortality and a slight reduction in the mean of that distribution, thus reflecting EPA's views about the implausibility of thresholds. The committee finds that such formal incorporation of EPA's expert judgments about the plausibility of thresholds into its primary analysis would have been an improvement.

Uncertainty about thresholds is a special aspect of uncertainty about the shape of concentration-response functions. Typically, EPA and authors of epidemiological studies assume that these functions are linear on some scale. Often, the scale is a logarithmic transformation of the risk or rate of the health outcome, but when a rate or risk is low, a linear function on the logarithmic scale is approximately linear on the scale of the rate or risk itself. Increasingly, epidemiological investigators are employing analytic methods that permit the estimation of nonlinear shapes for concentration-response functions (Greenland et al. 1999). As a consequence, EPA will need to be prepared to incorporate nonlinear concentration-response functions from epidemiological studies into the agency's health benefits analyses. Any source of error or bias that can distort an epidemiological association can also distort the shape of an estimated concentration -response function, as can variation in individual susceptibility (Hattis and Burmaster 1994; Hattis et al. 2001).

EPA expressed much less certainty about alternative lag structures than it did about thresholds in the Tier 2 analysis. The lag structure used in the

[2]A lag reflects the time course between pollutant exposure and development of clinical disease. A lag structure reflects the variation among the population in the lags experienced by various individuals.

primary analysis was recommended by the Science Advisory Board (EPA 1999a, pp. 4-6, 4-7), but the agency considered a range of alternative lag structures plausible. Here a probabilistic weighting of alternative lag structures based on expert judgment might have led to a more appreciable widening of the health benefit probability distribution.

Although EPA considered alternative lag structures to vary in plausibility, these variations were not, but could have been, approximately captured by subjective probability distributions. The incorporation of these distributions into the final probability distribution for the primary analysis would have resulted in a more realistic presentation of acknowledged sources of uncertainty.

In principle, many components of the health benefits model need realistic probabilistic models (see Table 5-1 for a listing of such components), in addition to concentration-response thresholds and time lags between exposure and response. For example, additional features of the concentration-response function—such as projection of the results from the study population to the target populations (which may have etiologically relevant characteristics outside the range seen in the study population) and the projection of baseline frequencies of morbidity and mortality into the future—must be characterized probabilistically. Other uncertainties that might affect the probability distributions are the estimations of population exposure (or even concentration) from emissions, estimates of emissions themselves, and the relative toxicity of various classes of particles. Similarly, many aspects of the analysis of the impact of regulation on ambient concentrations and on population exposure involve considerable uncertainty and, therefore, may be beneficially modeled in this way. Depending on the analytic approach used, joint probability distributions will have to be specified to incorporate correlations between model components that are structurally dependent upon each other, or the analysis will have to be conducted in a sequential fashion that follows the model for the data-generating process.

EPA should explore alternative options for incorporating expert judgment into its probabilistic uncertainty analyses. The agency possesses considerable internal expertise, which should be employed as fully as possible. Outside experts should also be consulted as needed, individually or in panels. In all cases, when expert judgment is used in the construction of a model component, the experts should be identified and the rationales and empirical bases for their judgments should be made available.

One other potential limitation of the sensitivity analyses and alternative calculations is that the longer and more detailed the ancillary uncertainty analyses are, the less realistic the results of the primary analyses tend to be. Decision-makers and others may be tempted, however, to reach the opposite conclusion. The length and depth of the ancillary uncertainty analyses might give the impression that the mean and 90% "credible interval" from the primary analysis have been rendered more certain and well supported when the opposite is true. The mean of a health benefit probability distribution is in some sense the best single estimate, but no estimate can be considered best if only one of the large number of uncertainties is included in the analysis producing that estimate. Moreover, variable degrees of uncertainty among analyses dictate that some best estimates are better than others.

EPA should present the results of its health benefits analyses in ways that foster an appropriate appreciation of their inherent uncertainty. The reporting of too many significant digits, as in the Tier 2 estimate of 682,898 work-loss days avoided (EPA 1999a, p. 6-3), lends an unwarranted impression of exactitude. EPA's focus on the mean value of the distribution rather than on the distribution range, such as the interval from the 5th to the 95th percentile of the distribution, also contributes to an impression of undue precision. The absence of graphical displays of the probability distributions, as shown in Figure 5-1, prevents an understanding of how sharply or gradually the probability falls away from its highest values to less plausible health benefits.

EXAMPLES OF UNCERTAINTY ASSESSMENTS

EPA is aware of health benefits analyses for air pollution reduction measures that have used probabilistic uncertainty assessment incorporating expert judgment (Lang et al. 1995; Holland et al. 1999). These assessments use the same methods as those the agency uses to combine health benefits probability distributions with probability distributions for valuation of those benefits as was done for the prospective analysis of the 1990 Clean Air Act Amendments (EPA 1999b). Furthermore, EPA has a limited but promising history of exploring the use of probabilistic uncertainty assessment in air pollution health benefits analysis.

Greenland (2001) illustrated the impact that one additional source of uncertainty can have on an analysis conducted to comply with California

regulatory guidelines. The analysis looked at the relative risk of skin cancer in a cohort of patients with severe psoriasis who received topical coal-tar-derived therapy. One of the sources of uncertainty pertained to the cohort's baseline expectation of skin cancer frequency. Typically, this source of uncertainty would be qualitatively described as a "study limitation." The analysis, which was similar to an EPA health benefits analysis, produced a point estimate of 0.71 and a 95% confidence interval of 0.46 to 1.12. When a probability distribution for this source of uncertainty was incorporated into the analysis, the mean of the probability distribution shifted upward to a relative risk of 0.77, and the 95% probability interval widened to a range of 0.43 to 1.37. Adding further sources of uncertainty to the analysis might cause the mean to rise or fall but would further widen the probability distribution.

In 1994, the National Research Council recommended that EPA conduct formal uncertainty analyses, including probabilistic assessment of uncertainties that "cannot be quantified on the basis of data" (NRC 1994, p. 12) and that therefore require expert judgment to quantify. That committee observed, "Objective probabilities might seem inherently more accurate than subjective probabilities, but this is not always true . . . . There can be no rule that objective probability estimates are always to be preferred to subjective estimates, or vice versa" (NRC 1994).

The EPA Office of Air Quality Planning and Standards (OAQPS) has demonstrated the potential use of formal expert judgment in support of the development of air quality standards (McCurdy and Richmond 1983). An initial effort in 1977 was made to apply judgmental probabilities elicited from health and air-quality experts to evaluate the health risks associated with alternative air-quality standards for ozone (EPA 1978). In 1979, the EPA Science Advisory Board reviewed this work, commended EPA for its initiative, critiqued certain elements of the approach used, and recommended further work in this area (SAB 1979). The major criticism of this initial work was that rather than focusing on point estimates of subjective probabilities, EPA attempted to elicit secondary probabilities (interval estimates of the probabilities).[3] The SAB (1980) endorsed the concept but asked EPA to develop approaches that did not involve secondary probability.

In 1980, OAQPS held a public meeting involving six groups of experts

[3]Discussion. Presented by H.M. Richmond at the American Statistical Association–EPA conference, Washington, DC, October 2, 1986.

from the fields of decision analysis, biostatistics, and behavioral psychology to discuss alternative approaches for the elicitation of expert judgment. Two approaches were selected for further development. OAQPS began work to illustrate how these approaches could be applied to estimate the health risks from carbon monoxide and also began to conduct an illustrative probabilistic risk assessment for lead.

By 1983, the OAQPS effort moved from the developmental stage to initiation of its lead National Ambient Air Quality Standard (NAAQS) risk assessment. Probabilistic dose-response functions for two health outcomes of lead exposure were elicited from 10 nationally recognized experts. This work was reviewed favorably by the EPA's Clean Air Scientific Advisory Committee (CASAC 1986a,b). In 1990-1991, a second effort was made to apply expert judgment to assess the risks of ozone. In view of the lack of adequate human data, EPA developed a risk assessment for chronic lung damage from ozone based on formally elicited expert judgment (Rosenbaum et al. 1995). However, the risk assessment for chronic lung injury was not formally used in support of the 1997 NAAQS revision for ozone. By the time the standard was set, the risk assessment was out of date, and the experts elicited had been told that their judgments would not be used for standard setting.

The committee recommends that the EPA offices responsible for health benefits analysis build on OAQPS' experience. Although the specific methods for selection and elicitation of experts may need to be modified somewhat, the protocols that have been developed and tested by OAQPS provide a solid foundation for future work in this area. EPA may also consider having its approaches reviewed and critiqued by decision analysts, biostatisticians, and psychologists from other fields where expert judgment has been applied (for example, nuclear-power-plant-accident-consequence risk assessment). Much has been learned in this area since EPA's last formal review of methods in the late 1970s.

An approach for the analysis of uncertainty is the 1994 NRC report, which included a case study of a probabilistic uncertainty analysis in an assessment of cancer risk from coal-fired power plant emissions of chromium, arsenic, cadmium, and benzene. The authors identified 49 uncertain parameters, which they reduced to 22 on the basis of a preliminary assessment of their degree of uncertainty and potential influence on the final results. "Evaluation of the probability distributions of the 22 influential parameters of the model was performed on the basis of available statistical data,

literature value ranges, and personal judgment" (Seigneur et al. 1994). This analysis is mentioned not for its substantive implications, but because it illustrates how the propagation of the uncertainties in many individual parameters can be analyzed and how the results of such an analysis can be used to identify the most influential sources of uncertainty.

A recent analysis of estimated short-term mortality of $PM_{2.5}$ provides another example (Dominici et al. 2002). Figure 5-2 shows the effect of adjusting for ozone, carbon monoxide, and sulfur dioxide. The mean of the resulting distribution does not change very much, but the spread of the distribution appropriately widens. Although not the result of a formal probabilistic uncertainty analysis, this widening is an approximate reflection of existing uncertainty that these copollutants might affect mortality and be associated with $PM_{2.5}$, thus confounding the $PM_{2.5}$ effect estimate.

RECOMMENDED APPROACH TO UNCERTAINTY ASSESSMENT

Other NRC reports addressed the issue of uncertainty in risk assessment and benefits estimation (NAE 1972; NRC 1975, 1982, 1983, 1994, 1996; Presidential Commission 1997). Without exception, they found that proper characterization of uncertainty is essential. Almost all expressed concern that most risk assessments and health benefits analyses tend to underestimate uncertainties and leave decision-makers with a false sense of confidence in estimates of risk. In addition, most of the reports suggested that failure to address model uncertainty adequately is often a major issue.

Despite this broad agreement on the importance of honest characterization of uncertainty and the common view that model uncertainty in particular tends to be understated or ignored, the NRC reports reached somewhat different conclusions about how best to resolve the problem. All agreed that sources of uncertainty should be listed and described, and most recommended that parameter uncertainties (and variability) be quantitatively characterized. Furthermore, they recommended that formal approaches for uncertainty, such as Monte Carlo analysis, be applied to understand the cumulative uncertainty and to provide insight into the dominant sources of parameter uncertainty. However, on the question of characterization of model uncertainty, differences in opinion emerged—some discussed and

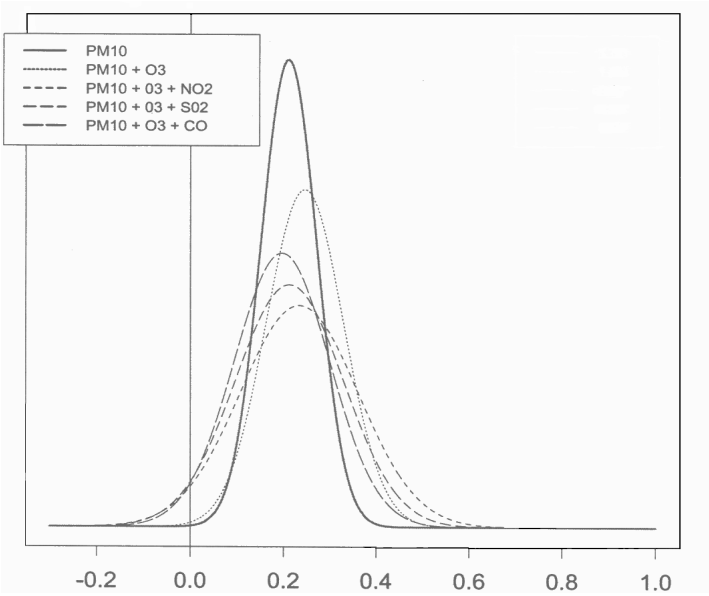

FIGURE 5-2 Posterior probability distributions for estimated short-term mortality effects of PM_{10} with and without adjustment for copollutants. The x-axis is the percent change in daily mortality ([relative risk ! 1] × 100%) for a 10 Fg/m^3 increase in PM_{10} concentration. Thus, 0.2% is a relative risk of 1.002. Source: Adapted from NMMAPS 2002.

recommended the use of expert judgment to characterize epistemic uncertainty, while others recommended that such basic scientific uncertainties should be thoroughly described but not quantified.

The committee shares the view that proper characterization of uncertainty is essential to good decision-making and agrees that uncertainties are often underestimated, leaving decision-makers with a false sense of security. Having reached this conclusion, the committee shares the view of M. Granger Morgan (Morgan et al. 1990):

> When the value of an uncertain quantity is needed in policy analysis, and limits in data or understanding preclude the use of conventional statistical techniques to produce probabilistic estimates, about the only remaining option is to ask experts for their best professional judgment.

In the committee's view then, the question is not whether to use expert judgment but how to use expert judgment. The options are to pick a particular model component from a range of varyingly plausible alternatives and treat that one as though it were absolutely certain or to specify a judgment-based probability model for the alternatives that reflects their varying degrees of plausibility and incorporate those probabilities into the primary analysis. The latter option has many difficulties, but it has the potential to portray the existing uncertainty more realistically than the former option does.

The committee recommends that EPA begin to incorporate additional sources of uncertainty into the probability models it uses in its primary health benefits analyses. Furthermore, the committee recognizes that decision-makers will need to be informed about how and why uncertainty was added to the health benefits analysis and how, in turn, this uncertainty might be communicated to the public. This process will use probability distributions to replace model components that are treated as known fixed values. Of necessity, the probability distributions for the uncertain model components will have to reflect a combination of empirical observations and expert judgment. This will result in a more realistic picture of the overall uncertainty in the analyses. The mean of the health benefit distribution will reflect the expected magnitude of the health benefit more accurately and, as a consequence, will be more defensible. The mean might shift upward, downward, or not at all as each additional source of uncertainty is added to the core analysis. The effect on the spread of the distribution, as reflected by the interval between its 5th and 95th percentiles for example, will be a predictable widening.

There is a large and growing body of literature on the use of expert judgment in risk assessment and quantitative policy analysis (Morgan et al. 1990; Cooke 1991). It has been applied in fields such as climate change (Reilly et al. 2001). There are several applications in health risk assessment (North and Merkhofer 1976; Morgan et al. 1984; Evans et al.1994). In fact, as described above, OAQPS has been a pioneer in the application of these approaches to estimating the health risks due to exposure to air pollutants (Richmond 1981; Feagans and Biller 1981; Whitfield et al. 1991; Rosenbaum et al. 1995). These approaches have also been used in cases of residential radon cancer risks (Krewski et al. 1999) and for stratospheric ozone depletion (NRC 1979a,b).

As it incorporates additional sources of uncertainty into its primary

health benefits analyses, EPA should consider conducting analyses to determine which uncertainty sources have the greatest influence on the final results. Those impacts should be measured not only on the mean but also on the spread of the health benefit probability distribution. The sources that have the greatest influence on the spread of the distribution of a health benefit should be given priority for future research. Value-of-information analysis, a branch of statistical decision analysis, provides a well-structured approach for estimating the decision-making benefits of information that might be expected to flow from various research strategies (Raiffa 1970; Lindley 1985).

EPA should also consider conducting analyses to determine the sensitivity of the final results to the specification of reasonable alternative probability distributions for the uncertainty sources in the primary analyses. The need for sensitivity analyses will be particularly great for distributions that are based solely or largely on expert judgment.

EPA should consider comparing predictions from health benefits analysis models with subsequent observations that were not used in deriving or calibrating the models. Ideally, the subsequent observations and comparisons should be made by researchers who are independent from the authors of the original model and the investigators whose observations were used to derive and calibrate it. The results of the comparisons should be presented in future health benefits analyses and used to assess, quantify, and reduce uncertainties in the resulting estimates.

As it begins in the transition to incorporate additional sources of uncertainty into its primary health benefits analyses, EPA should continue the sensitivity analyses it has traditionally conducted. These analyses should be expanded, however, to include more than one source of uncertainty at a time. For example, if EPA were to include three additional uncertainty sources into its primary analysis of a health benefit, it might also conduct a traditional sensitivity analysis of these three sources jointly. With three illustrative scenarios for each component, for example, this expansion of the traditional sensitivity analysis would produce mean health benefits estimates for all 27 possible combinations of the scenarios. EPA then would be able to refer to the probability assigned to these combinations in the primary analysis to reflect their varying degrees of plausibility.

EPA should consider distinguishing between the uncertainties that arise from difficulties in projecting the future and the uncertainties inherent in estimating health benefits in current populations on the basis of hypothetical

changes in current levels of emissions. By providing a preliminary analysis that estimates in current populations the health benefits resulting from hypothetical changes in current levels of emissions, EPA might develop an idea of the lower bound on the uncertainty in any prediction of consequences projected into the distant future. There would be fewer uncertainties in these preliminary analyses than in analyses of the impacts of proposed regulatory actions on future exposures and health outcomes.

EPA should continue to strive to present the results of its health benefits analyses in ways that avoid conveying an unwarranted degree of certainty. These alternative approaches should include rounding to fewer significant digits. For example, the mean of the Tier 2 distribution for avoided mortality could have been reported as 4,300 or 4,000 avoided deaths rather than 4,307. Another need is to place less emphasis on a single value, such as the mean of a health benefit probability distribution, and more on ranges, such as the interval between the distribution's 5th and 95th percentiles. It would also be helpful to increase the use of graphs to display health benefits probability distributions in their entirety. Graphs will be especially helpful as the incorporation of additional uncertainties results in asymmetrical health benefit probability distributions (Read and Morgan 1998).

In presenting a probability distribution for each health benefit produced by a primary analysis, EPA should emphasize even more than it has in the past the sources of uncertainty that remain unaccounted for in the primary analysis. Along with depicting the uncertainty in its primary health benefits analyses more realistically, EPA should foster a discussion in which it rebuts explicitly the misperception that such analyses would not be "useful." That view comes from a mistaken belief that a very high degree of certainty is required before regulatory action can be considered warranted to protect the environment and the public health. The result is needless pressure to make the scientific basis for that regulatory action appear more certain than it is. A more defensible position is that decision-makers can make much better decisions when provided with realistic assessments of the nature and extent of the uncertainty that is present. The correct mix of action and research is a policy decision that can be informed by a full appraisal of the sources, nature, and extent of uncertainty.

The committee recommends that formally elicited expert judgment be used in the characterization of uncertainty in estimates of health benefits, although the committee recognizes that a number of issues must be addressed to use this approach responsibly. However, the committee be-

lieves approaches that explicitly incorporate judgmental probabilities into EPA estimates of health benefits are preferable to ones that fail to characterize the degree and key sources of uncertainty in estimates of health benefits from regulatory action. Furthermore, the committee recommends that EPA formally acknowledge those experts from whom it elicited judgments on uncertainty issues in the health benefits analysis.

CONCLUSIONS

- In its primary analyses of health benefits, EPA reports the uncertainty as a probability distribution. Only one source of uncertainty, the random sampling variability of the estimated concentration-response function, is given with an emphasis on the mean of the probability distribution. The absence of other sources of uncertainty makes the results of the primary analyses appear more certain than they are.
- To address other sources of uncertainty, EPA uses ancillary analyses, such as alternative and supplementary calculations and sensitivity analyses. With the exception of concentration-response function estimates, these ancillary analyses usually examine only one source of uncertainty at a time and only for the impact on the mean value of the probability distribution from the primary analysis. As a consequence, though laudable steps in the right direction, these ancillary analyses do not adequately convey the relative or aggregate degree of uncertainty created by the sources of uncertainty addressed in the analyses, nor, of course, do they depict uncertainty from other sources.

RECOMMENDATIONS

- EPA should begin to move the assessment of uncertainties from its ancillary analyses to its primary analyses. This shift will require the specification of a probability distribution for each uncertainty source that is added to the primary analysis and, as necessary, the specification of joint distributions for the uncertainty sources that are not independent of each other. Expert judgment, as well as data, will be required to specify these distributions. Although the effect on the mean of the resulting probability distribution might increase, decrease, or remain the same, the effect on the spread

of the distribution will be a predictable widening and, therefore, a more realistic depiction of the overall uncertainty in the analysis.

- As it incorporates additional sources of uncertainty into its primary health benefits analyses, EPA should consider conducting analyses to determine which uncertainty sources have the greatest influence on the mean and spread of the probability distribution. The need for these sensitivity analyses will be particularly great for distributions that are based on expert judgment. The uncertainty sources that have the greatest consequences for decision-making, including those that have the greatest impact on the spread of the distribution, should be given high priority for additional research.
- Because the incorporation of expert judgment when data are unavailable will influence the estimates of health benefits as well as the uncertainty analyses, the committee also recommends that EPA clearly distinguish between data-derived estimates of some components—such as the concentration-response function—and expert opinions about other components that are lacking in scientific data—such as the degree of compliance with a particular regulation 30 years into the future. In this way, policymakers will better understand how existing data and expert judgment combine to produce estimates and where new data would be most valuable.
- As EPA begins the transition to incorporate additional sources of uncertainty into its primary health benefits analyses, it should continue the sensitivity analyses it has traditionally conducted. These analyses should be expanded, however, to consider sources of uncertainty jointly rather than singly.
- In presenting the probability distribution for each health benefit produced by a primary analysis, EPA should emphasize even more than it has in the past the sources of uncertainty that remain unaccounted for in the primary analysis. These uncertainties should continue to be described as completely and realistically as possible.
- EPA should consider providing a preliminary analysis that estimates in current populations the health benefits resulting from hypothetical changes in current levels of emissions. These preliminary analyses would help EPA develop an idea of the lower bound on the uncertainty of future consequences and would have fewer uncertainties than analyses of the impacts of proposed regulatory actions on future exposures and health outcomes.
- EPA should continue to strive to present the results of its health benefits analyses in ways that avoid conveying an unwarranted degree of

certainty. Such ways include rounding to fewer significant digits, increasing the use of graphs, presenting projected baseline along with projected health benefits, and placing less emphasis on single numbers (for example, the mean of the probability distribution for a health benefit) and greater emphasis on ranges (for example, the range between 5th and 95th percentiles of the distribution).

- There is a common misperception that a high degree of certainty is required for regulatory actions to take place to protect public health. As a result, primary health benefits analyses that more fully and accurately portray the uncertainties might not be considered useful. It is unrealistic for EPA to defer decisions until it can make them on the basis of perfect science. A careful and deliberate balancing of the benefits and costs is required, and this balancing must be informed by a fair assessment of the current levels of uncertainty and a realistic evaluation of the likely reductions in uncertainty attainable through further research.
- EPA should perform similar detailed analyses of uncertainty in the valuation of health benefits and in the regulatory cost analyses that the committee recommends for the health benefits analyses.

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6

Using, Presenting, and Reviewing Health Benefits Analyses

Estimating the health benefits of a reduction in ambient air pollution concentrations involves the series of steps described in Chapter 1 of this report. To summarize, the regulatory options to be evaluated must be clearly defined with regard to scope, timing, and implementation. Boundaries of the analysis must be established, and the regulatory baselines must be defined. Once the analysis has been structured, changes in pollutant emissions can be estimated and translated into changes in pollutant concentrations or exposures. Changes in health outcomes can then be estimated from the changes in pollutant concentrations or exposures and concentration- or exposure-response functions. As discussed in Chapter 5, uncertainties at each stage of the analysis should be quantified and carried through the entire process.

The results of health benefits analyses are often used in cost-benefit or cost-effectiveness analyses of air pollution regulations. Although the philosophical foundations of cost-benefit analysis and especially the economic valuation of benefits remain controversial, it is important to discuss the implications of cost-benefit analysis for conducting a health benefits analysis. Accordingly, this chapter discusses how health benefits should be computed to be compatible with cost-benefit and cost-effectiveness analyses. Methods of presenting the results of health benefits analyses and issues regarding quality assurance and quality control are also addressed.

COMPATIBILITY OF HEALTH BENEFITS ANALYSES WITH COST-BENEFIT ANALYSES

The goal of a cost-benefit analysis is to compare the monetized benefits of a regulation with its costs. Regulations are often ranked according to the size of their net benefits (benefits minus costs). As a result, health benefits must be calculated and then converted to dollars using a value per avoided case to allow comparison to the costs.

Monetization of Health Benefits

Economists estimate the value of avoided morbidity by determining the amount a person is willing to pay to avoid an illness. The estimate should include the value of avoided pain and suffering, the value of time lost due to illness (both leisure and work time), and the costs of medical treatment. If some of the costs are not borne by the individual and therefore not reflected in the person's "willingness to pay" (WTP), these costs must be added to the estimate to measure the total benefits to society of reduced morbidity. For certain chronic illnesses, such as chronic bronchitis, economists try to measure what a person would be willing to pay to reduce his risk of contracting the illness. The amount is usually expressed in terms of the "value of a statistical case" of a given illness, such as chronic bronchitis, and represents the sum, across different individuals, of WTPs for risk reductions that together equal one statistical case.

Similarly, for premature mortality, economists try to measure what a person would be willing to pay to reduce his risk of dying. The amount is usually expressed in terms of the "value of a statistical life" (VSL) and represents the sum, across different individuals, of WTPs for risk reductions that together equal one statistical life. For example, if 10,000 people are willing to pay $100 each to reduce their risk of dying by 1 in 10,000, together they are willing to pay $1 million for risk reductions that equal one statistical life. The $1 million is the VSL. The VSL includes the lost income associated with dying prematurely but does not reflect the medical costs that might precede death. These costs are assumed to be included in the value of morbidity.

Empirical estimates of the value of avoided morbidity that include all three components (WTP to avoid pain or discomfort, lost earnings, and

medical costs) do not exist for many of the health outcomes associated with air pollution in epidemiological studies. For example, there are few studies of WTP to avoid the discomfort associated with a hospital admission or to reduce the risk of a heart attack or stroke. For these outcomes, the unit values typically used in EPA analyses reflect medical costs and lost earnings.

Implications of Monetization for Estimation of Health Benefits

Does the monetization of health benefits imply that health benefits estimates should be reported in a particular way, such as by income, age, or health status? According to economic theory, WTP to reduce risk of death and WTP to avoid illness should increase with income. For equity reasons, however, unit health values are currently not varied according to the income of the affected population. The relation between WTP and income is used only to adjust unit health values over time to reflect the impact of income growth on the value of avoided morbidity or death. Therefore, for purposes of monetization, health benefits do not need to be reported by income group.

For chronic illnesses and mortality, one might expect WTP to vary with age and possibly health status and conclude that health benefits (avoided cases) should be estimated and reported by these factors. There is no evidence that WTP to reduce risk of death varies with current health status (Alberini et al. 2002; Krupnick et al. 2002). However, statistical lives saved might need to be reported by age group with estimates of remaining life expectancy. The empirical literature suggests that WTP to reduce risk of death and hence the VSL eventually decline with age (Jones-Lee et al. 1985; Krupnick et al. 2002). To allow for the possibility that the VSL varies with age, estimates of statistical lives saved by air pollution control programs should be presented by the age of the beneficiaries.

The remaining life expectancy for each age group must also be reported if mortality reductions are monetized using the "value of a statistical life-year" (VSLY) approach. To illustrate this approach, suppose that the VSL is $5 million and that the average age of people receiving this benefit is 40. If remaining life expectancy at age 40 is 35 years and the interest rate is zero, then the VSLY is approximately $140,000. The value of preventing the death of an 80-year-old, with 8 years of remaining life expectancy, would be 8 times $140,000 or $1.2 million. Thus, applying the VSLY re-

quires an estimate of the remaining life expectancy. The committee notes that this procedure has been criticized because it implicitly assumes that each year of life is equally valuable and that the VSL is strictly proportional to remaining life expectancy. It has been used by EPA (1997, 1999), however, and is commonly used in the European Union (ExternE 1999).

COMPATIBILITY OF HEALTH BENEFITS ANALYSES WITH COST-EFFECTIVENESS ANALYSES

The goals of a cost-effectiveness analysis are to aggregate the health benefits of a program by using a measure that reflects the magnitude and the duration of improvements in health status and to express the cost of the program as a cost per unit of health benefit received. This practice should not be confused with the use of cost-effectiveness to screen pollution control options, which expresses the cost of the measure as cost per ton of emissions reduced. A commonly used measure of health benefits for this analysis is the "quality adjusted life-year" (QALY) (Gold et al. 1996), where a weight of one represents optimal health and a weight of zero represents death.[1] The cost-effectiveness estimate of a program is expressed as the ratio of the program costs (numerator) to the QALYs achieved by a program (denominator).

In a cost-effectiveness analysis for a program, the value of avoided morbidity is the product of the duration of the avoided illness and the difference between the health status index, such as QALY, with and without the program. To illustrate, suppose that an air pollution regulation prevents a person from contracting chronic bronchitis at age 40 and living with the

[1]The World Health Organization, World Bank, and a number of other international and national agencies commonly use the disability-adjusted life-year (DALY), a type of QALY, for health comparisons (World Bank 1993; Murray and Lopez 1996). A distinction of the DALY is that it has been used to develop databases that enumerate the entire pattern of illness by age, sex, and location (city, province, nation, or in WHO's [2002] Global Burden of Disease Database, the world). It varies from the QALY in that lost life-years only vary by age and sex and not health status. If DALYs are to be calculated, a health benefits analysis should use the categories of illness (disease and injury states) available in the appropriate burden of disease database.

disease for 30 years. The regulation would save 30 times the difference between the QALY weight assigned to the individual's current health state and the QALY weight assigned to living with chronic bronchitis ($30 \times [QALY_{\text{current health}} ! QALY_{\text{chronic bronchitis}}]$). See Gold et al. (1996) for a discussion of the different methods used to assign QALY weights and for alternative health-related quality of life scales.

As the example above implies, avoided cases of chronic morbidity and mortality must be reported in terms of age at onset or age at which mortality is prevented and in terms of remaining life expectancy if the benefits estimates are to be used as inputs into a cost-effectiveness analysis. In the case of avoided mortality, it is also useful to have some knowledge of the health status of persons whose deaths are postponed. If reducing air pollution is more likely to prevent the deaths of persons with congestive heart failure or coronary artery disease than persons in the general population, the number of QALYs saved will be fewer than if the deaths were evenly distributed over the population. That is because the QALY weights assigned to coronary artery disease and congestive heart failure are smaller than the average QALY weight assigned to the current health of the general population.

For avoided cases of acute morbidity, the QALY approach also requires an indication of the period of avoided illness. For example, if 10 million fewer restricted-activity days (RADs) are experienced in a year in a population of 20 million then an average of 0.5 fewer RADs per person per year are experienced. The annual QALYs gained per person would be the difference between the QALY weight assigned to having 0.5 fewer RADs and the baseline QALY weight. To calculate the total QALYs gained, the number of QALYs gained per person would be multiplied by the size of the exposed population, which is 20 million in this example.

COMPATIBILITY OF HEALTH BENEFITS ANALYSES WITH COST ANALYSES

In estimating health benefits, assumptions must be made about the size of the population exposed to air pollution for each year of the analysis and about the baseline incidence of each health outcome evaluated. Assumptions must also be made about remaining life expectancy when a VSLY or a QALY approach is used. In monetizing benefits, assumptions are made

about the rate of income growth, and those assumptions are used to estimate how the unit values assigned to each health outcome change over time.

The assumptions made about disease incidence and mortality and especially income and population growth used in the health benefits analysis must be consistent with assumptions made in computing the costs of air pollution control. The choice of years for which to compute health benefits must also correspond in a sensible way to the period for which costs are computed.

COMMUNICATION OF METHODS AND RESULTS OF ANALYSIS

A common complaint about EPA benefits analyses is that the methods, the rationale behind the decision-making, and the results are not clearly presented or described. After review of the four EPA analyses, the committee agrees that the presentation could be improved and is concerned that factors that drive the analysis are buried in appendixes or technical support documents. Furthermore, the committee noted that the lengths of discussion devoted to certain components of the analysis are not based on their importance to the analysis. For example, for the heavy-duty engine and diesel-fuel rule, EPA uses four pages of text to describe Voronoi neighbor averaging to interpolate ambient air pollution concentrations at the mid-point of each spatial grid used in the atmospheric model (EPA 2000). The method of interpolation is a technical issue that is unlikely to be a key determinant of the ultimate prediction of air quality. The fact that simulations of ozone concentrations were done only for the eastern United States and were based on meteorological data for only 30 days in the summer of 1995 is of much greater importance. This limitation, however, is dealt with in one sentence. For further contrast, the entire topic of statistical uncertainty is covered in approximately one page.

Although the documents reviewed by the committee contained executive summaries, the committee believes that the summary should be more detailed, such as 20-30 pages in length, and present the key information summarized in Table 6-1. Subsequent chapters should describe the methods used in each step of the analysis, the validation of models used in the analysis, and the uncertainty associated with the estimates at each stage of the

TABLE 6-1 Items to Be Reported in the Summary of a Benefits Analysis of an Air Pollution Control Regulation

Framing the Analysis	Emissions and Air Quality	Health Benefits
Describe each regulatory option Geographic scope Timing Parties affected Describe the boundaries of the analysis Time period of benefits analysis Intervals at which benefits are calculated Pollutants evaluated Degree of compliance with regulation Describe the regulatory baseline Conditions without regulation, including other regulations in place and assumptions about the economy and population. Highlight any assumptions that have a substantial impact on the results of the analysis	Summarize emissions at the national level by sector with and without the regulation Compare baseline emissions to historical trends Present emissions changes associated with the regulation in absolute and in percentage terms Summarize ambient air quality by region and at the national level with and without the regulation Report as population-weighted annual averages Compare baseline air quality to historical trends Present pollution changes associated with the regulation in absolute and in percentage terms	List health outcomes evaluated and describe each Indicate time path of avoided cases for each health outcome For quantified outcomes at each time period for which results are presented, the following information should be presented Size of exposed population Baseline number of cases (cases/100,000) Coefficient of concentration-response function Number of avoided cases For avoided mortality and chronic morbidity, information indicated above should be presented by age at onset and remaining life expectancy

analysis. EPA should discuss more fully the components that are the important contributors to the benefits estimates and that may have substantial uncertainty associated with them.

The summary should begin with a description of the regulatory options considered, including their timing, scope, and assumptions made about the degree of compliance with each option. This description should be contrasted with the conditions that are assumed to exist without the regulation—that is, the regulations that are in place and the air quality without enactment of the proposed regulation.

It is critical that the implications of a regulation for emissions and ambient air quality be presented clearly. Summary information should be provided describing emissions of the air pollutants with and without enacting the regulation. An excellent example of how this information should be presented is contained in Appendix A of the prospective analysis of the Clean Air Act (EPA 1999). The charts show aggregate emissions of each criteria pollutant (one chart for each pollutant) with and without the 1990 Clean Air Act Amendments (CAAA) over the period of analysis. Historical emission trends for 1980-1997 are shown on each graph so that the reader can judge how reasonable the predictions without CAAA regulations are. The changes in emissions (in absolute and percentage terms) attributable to the regulation should also be presented. Similar statistics should be provided for ambient concentrations and changes in ambient concentrations. In the case of ambient concentrations, it would be useful to describe changes in population-weighted average concentrations both for regions of the country and for the nation as a whole.

The summary should include a description of health outcomes evaluated in the study. For quantified outcomes, the number of avoided cases should be listed for each year of the analysis, both in absolute terms and as a percentage of baseline cases of the outcome (see EPA 1999, Table 5-3). Avoided cases of mortality and chronic morbidity, such as chronic bronchitis, should be broken down by age group with estimates of average remaining life expectancy presented for each age group. The benefits may also need to be presented by various demographic or other subgroups when the expected changes in pollution and thus the health benefits are not uniformly distributed across the population. The presentation of this information would alert and aid decision-makers when issues of equity are concerned. Explanations for not quantifying certain health outcomes should also be provided.

In addition to presenting numbers of avoided cases, enough information should be provided for at least 1 year of the analysis to permit approximation of the estimates of health benefits provided at the national level. For example, the number of avoided cases of chronic bronchitis resulting from

a reduction in air pollution is the product of (1) the size of the exposed population, (2) the baseline incidence of chronic bronchitis (cases per 100,000), (3) the percentage change in incidence per unit of pollution, and (4) the population-weighted change in pollution. The committee acknowledges that this calculation would only approximate a similar calculation performed at the county level and then aggregated across counties; however, summarizing this information for each health outcome would allow the reader to at least approximate the calculations.

As discussed in Chapter 5, the analysis of the uncertainty is an important component of health benefits analysis. The summary chapter should discuss briefly the methods used to address and quantify uncertainty and should highlight sources of uncertainty that could not be adequately assessed. EPA should strive to present the results of the analyses in ways that avoid conveying an unwarranted degree of certainty. Such ways include rounding to few significant digits, increasing the use of graphs, and placing less emphasis on single numbers and greater emphasis on ranges.

To clarify further the methods used in the health benefits process, a detailed flow diagram should be added to the introduction of the summary (see Figure 6-1). This diagram would provide at a glance the regulatory options considered, the time frame of the analysis, and any assumptions that substantially affect the results of the analysis. Pollutants and modeling techniques should be indicated, and the health outcomes and basis for their quantification should be presented in this diagram. The diagram would serve as a reference point for the discussion that follows. Figure 6-1 could be considered an expansion of the diagrams that EPA provides in its analyses that simply indicate generic steps of a benefits analysis (see EPA 2000, Table 7-1).

If the benefits analysis is an integral part of a cost-benefit analysis, then enough information should be provided to allow approximation of the monetized estimates or to produce alternative results where there is disagreement with the assumptions used. The information shown below should be provided in the summary.

- Unit values used to monetize health outcomes should be listed in a table with the year in which dollar estimates apply (see EPA 1999, Table 6-1).
- Each unit value should indicate whether it includes WTP for pain and suffering, medical costs, and lost earnings.

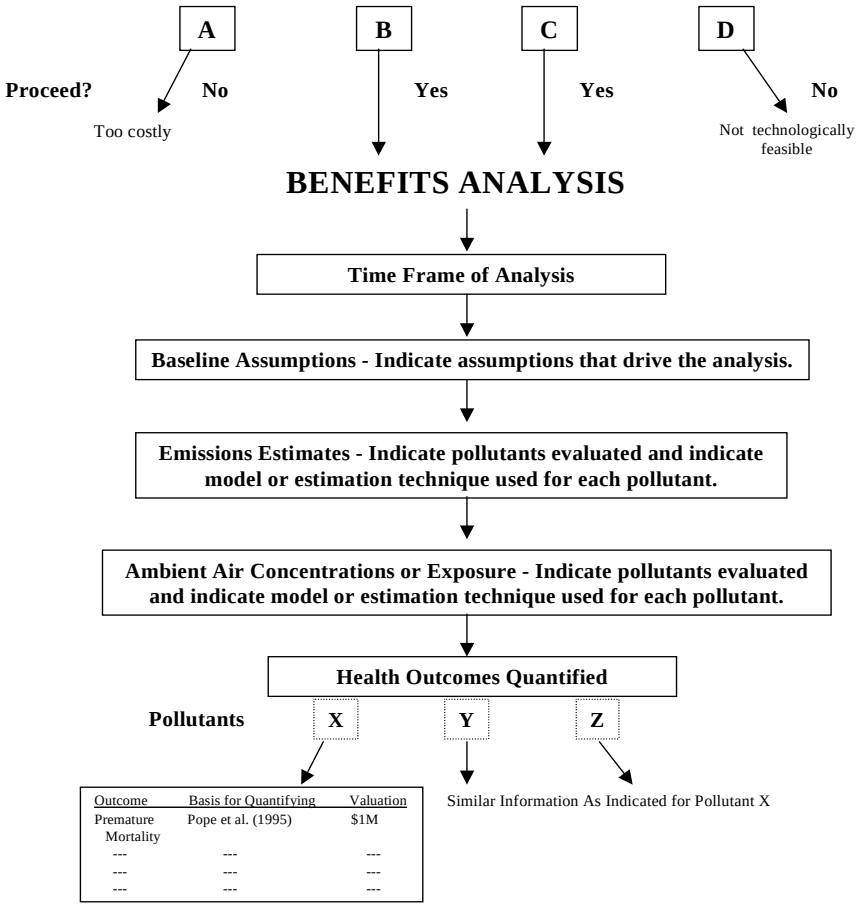

FIGURE 6-1 Flow diagram of a health benefits analysis.

- Information should be provided on how unit values are assumed to change over time.
- Total monetized value of health benefits by year and current discounted value of health benefits should be provided.
- The discount rate used should be clearly stated, and alternative rates should be presented for sensitivity analysis.

If the benefits analysis is a part of a cost-effectiveness analysis, then

additional information should be provided. In this case, the weights used to aggregate the avoided cases of morbidity and mortality and the durations of avoided illnesses should be presented. The analysis should be explicit about how avoided lost earnings and medical costs were incorporated into the analysis.

Presentation of the information discussed in this section would allow a more complete and straightforward assessment of the methods and results of the analysis. With the presentation of the avoided cases of morbidity and mortality and the unit valuations, estimates of the annual dollar value of health benefits achieved or annual QALYs avoided could be checked. This would increase confidence in the estimates generated.

QUALITY ASSURANCE AND QUALITY CONTROL

Reporting the information in Table 6-1 in a summary at the beginning of each report will not guarantee that the study is scientifically sound and satisfies the following criteria:

- The study considers the appropriate regulatory options.
- The study chooses an appropriate time period for analysis.
- The study uses state-of-the-art data and validated models.
- The study uses models and data comparable to those in similar analyses.

The issues of quality control that are specific to a particular study include the regulatory options that are considered relevant, the appropriate time period for the analysis, and the assumptions regarding compliance and the regulatory baseline. Clearly, peer review of these aspects of the study would be most useful at the beginning of the study. Although an expert panel similar to a subcommittee of the EPA's Science Advisory Board could be assigned such a task, few experts have the technical knowledge to evaluate these aspects of study design. Therefore, a standing, independent technical review panel with a professional staff should be established to evaluate analyses at the initial stages and throughout the process. This panel should include members with expertise in regulatory options analysis, emissions and exposure assessment, epidemiology, toxicology, risk analysis, biostatistics, and economics and should be appointed with strict attention to

avoiding conflict of interest, balancing bias, and ensuring broad representativeness. This panel could ensure that there is reasonable consistency among similar types of analyses produced within EPA and across other agencies.

CONCLUSIONS

- The results of health benefits analyses are typically used as inputs to cost-benefit or cost-effectiveness analyses; therefore, the results need to be presented in ways compatible for these analyses.
- The presentation of methods, rationale, and results from health benefits analyses is sometimes inadequate. For example, EPA's analyses do not highlight key assumptions that drive the analysis, do not indicate the rationale behind study selection, and do not present results in ways that allow verification of estimates obtained.
- Benefits analyses are typically not scrutinized at the initial stages of study design, during the process, or at the final stages of the process.

RECOMMENDATIONS

- EPA should provide health benefits estimates in ways that will support multiple kinds of analysis, including various approaches to mortality valuation and aggregation of benefits using quality-adjusted life-years.
- EPA should provide a summary of the analysis containing information as outlined in Table 6-1. This information would allow the reader to evaluate the study design and verify estimates obtained in the analysis.
- Each analysis should provide results according to demographic or other subgroups when the expected changes in pollution and thus the health benefits are not distributed uniformly across the population. This information would aid decision-makers in situations in which equity issues might be involved.
- To enhance the quality of future regulatory benefits analyses, a standing, independent, technical review panel should advise EPA in the initial stages of its benefits analysis. This panel should have expertise in regulatory options analysis, emissions and exposure assessment, toxicology, epidemiology, risk analysis, biostatistics, and economics and should be ap-

pointed with strict attention to avoiding conflict of interest, balancing biases, and ensuring broad representation. This panel should be supported by permanent technical staff to ensure consistency of reviews over time. EPA should follow the panel's guidance on the need for peer review.

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Appendix

BIOGRAPHICAL INFORMATION ON THE COMMITTEE ON ESTIMATING THE HEALTH-RISK-REDUCTION BENEFITS OF PROPOSED AIR POLLUTION REGULATIONS

JOHN C. BAILAR, III, is professor emeritus at the University of Chicago. He is a retired commissioned officer of the U.S. Public Health Service and worked for the National Cancer Institute for 22 years. He has also held academic appointments at Harvard University and McGill University. Dr. Bailar's research interests include assessing health risks from chemical hazards and air pollutants and interpreting statistical evidence in medicine with a special emphasis on cancer. He was editor-in-chief of the *Journal of the National Cancer Institute* for 6 years and a member of the editorial board of the *New England Journal of Medicine* for 7 years. Dr. Bailar was elected to the Institute of Medicine in 1993 and is a member of the International Statistical Institute. He received his M.D. from Yale University and his Ph.D. in statistics from American University.

HUGH ROSS ANDERSON is a professor of epidemiology and public health and the chairman of the Department of Public Health Sciences at St. George's Hospital Medical School in London, England. The focus of his research is on the short- and long-term health effects of air pollution with an emphasis on the relationships between daily variations in air pollution and mortality, hospital admissions, and medical consultations. He has conducted

epidemiological studies to investigate the health effects of an air pollution episode in London in 1991 and to assess the risk factors for asthma deaths. Dr. Anderson is a member of the steering group of the International Study of Asthma and Allergies in Childhood, the U.K. Department of Health's Committee on the Medical Effects of Air Pollution, and the U.K. Department of Environment's Expert Panel on Air Quality Standards. He also served as a member of several World Health Organization working groups on air pollution. Dr. Anderson received his M.D. from Melbourne University in Australia.

MAUREEN L. CROPPER is a professor of economics at the University of Maryland, a lead economist at the World Bank, and a university fellow at Resources for the Future. She is past president of the Association of Environmental and Resource Economists and a former chair of the Advisory Council for Clean Air Act Compliance Analysis, a subcommittee of EPA's Science Advisory Board. Her research has focused on valuing environmental amenities (especially environmental health effects), on the discounting of future health benefits, and on the tradeoffs implicit in environmental regulations. Her recent research focuses on factors affecting deforestation in developing countries and on the externalities associated with motorization. Dr. Cropper received her Ph.D. in economics from Cornell University.

JOHN S. EVANS is a senior lecturer on environmental sciences and the codirector of the Program in Environmental Science and Risk Management at the Harvard School of Public Health. His research focuses on risk assessment, uncertainty analysis, and decision-making in environmental health. Dr. Evans has developed and applied approaches for quantitatively characterizing the uncertainty in health risk assessments and for analyzing the value of information provided by alternative research strategies. He received a Sc.D. in environmental health sciences from Harvard University.

DALE B. HATTIS is a research professor at the George Perkins Marsh Institute at Clark University. His research focuses on the development and application of methods to assess the health impacts of regulatory options. His emphasis is on incorporating interindividual variability data into risk assessments for both cancer and noncancer end points. He has served as a member of several NRC committees (such as the Committee on Neuro-

toxicology and Models for Assessing Risk and the Subcommittee on Methyl Bromide). Dr. Hattis received his Ph.D. in genetics from Stanford University.

ROGENE F. HENDERSON is the deputy director of the National Environmental Respiratory Center at the Lovelace Respiratory Research Institute in Albuquerque, New Mexico. Her research interests include biochemistry of the lung and the pharmacokinetics of inhaled xenobiotics. She has extensively studied the use of biomarkers to predict environmental exposures and health outcomes. She has served on numerous NRC committees (such as the Committee on Epidemiology of Air Pollutants and Committee on Risk Assessment Methodology). She has served as the chair of the Committee on Toxicology and is currently serving on the Board of Environmental Studies and Toxicology. Dr. Henderson received her Ph.D. in chemistry from the University of Texas at Austin and is a diplomate of the American Board of Toxicology.

PATRICK L. KINNEY is an associate professor at the Columbia University School of Public Health. He conducts epidemiological research on the respiratory health impacts of air pollution with an emphasis on characterization of human exposure. His current research includes investigating the relationship between indoor air pollutants and asthma; characterizing outdoor, indoor, and personal concentrations of a variety of toxic air pollutants to which urban residents are exposed; and assessing exposures and health impacts of air pollution at the cellular and molecular level. He served on the NRC Committee on an Assessment of Asthma and Indoor Air Quality. Dr. Kinney received his Sc.D. in environmental science and physiology at the Harvard School of Public Health.

NINO KÜNZLI is an assistant professor (PD) at the Institute of Social and Preventive Medicine in the Department of Environmental Health at the University of Basel, Switzerland. His research focus is environmental epidemiology with an emphasis on air pollution epidemiology. He has completed a European assessment of the public health impact of outdoor and traffic-related air pollution. He is a member of the World Health Organization (WHO) Air Pollution Health Impact Assessment Working Group. Dr. Künzli received his M.D. from the University of Basel and his M.P.H. and Ph.D. from the University of California at Berkeley. In September 2002,

Dr. Künzli became associate professor at University of Southern California, Keck School of Medicine, Division of Occupational and Environmental Health.

BART D. OSTRO is chief of the Air Pollution and Epidemiology Unit, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency, Oakland. His research activities have included developing a method for estimating air pollution health effects for the World Health Organization, evaluating health and economic consequences of air pollution in developing countries for the World Bank, and conducting epidemiological studies of the health effects of air pollution. He was the coauthor of the U.S. Environmental Protection Agency health and economic assessment that resulted in the ban of leaded gasoline, and his work has contributed to the development of state and federal ambient air quality standards. Dr. Ostro received his Ph.D. in health economics from Brown University and a certification in environmental epidemiology from the State of California.

CHARLES POOLE is an associate professor of epidemiology at the School of Public Health of the University of North Carolina at Chapel Hill. His research focus is the development and application of general epidemiological principles and methods. These include problem conceptualization, study design, data collection, analysis, and interpretation. He is a member of the Solvent Panel of the Institute of Medicine's Committee on Gulf War and Health: Review of the Literature on Pesticides and Solvents. Dr. Poole received his M.P.H. in health administration from the University of North Carolina at Chapel Hill and his Sc.D. in epidemiology from the Harvard School of Public Health.

KIRK R. SMITH is a professor and chair of the Division of Environmental Health Sciences at the University of California at Berkeley. Dr. Smith first identified, characterized, and quantified indoor air pollution in poor countries as the major source of air pollution exposure and as one of the most significant environmental health risks. Currently, he is investigating the application of total exposure assessment methods to develop cost-effective strategies for urban and rural pollution control and is involved in on-site air-pollution, greenhouse gas, and health-impacts monitoring in Asia and Latin America. Dr. Smith was elected to the National Academy of Sciences in

1997 and serves on the NRC Board of Environmental Studies and Toxicology as well as the NRC Committee on Collaborative Opportunities with India in Energy and Environment. He received his Ph.D. and M.P.H. from the University of California at Berkeley.

PETER A. VALBERG is a principal at Gradient Corporation in Cambridge, Massachusetts. He specializes in human health risk assessment, human toxicology, and biological modeling of human exposure to environmental chemicals and has particular expertise in health effects of air pollutants. From 1985 to 2000, he was an associate or adjunct professor of physiology at the Harvard School of Public Health and conducted research on human health effects of air toxics, methods to measure lung macrophage function, and lung deposition and clearance of radioactive tracer particles. Dr. Valberg received a Ph.D. in physics from Harvard University and an M.S. in human physiology and inhalation toxicology from the Harvard School of Public Health.

SCOTT L. ZEGER is a professor and chair of the Department of Biostatistics at the Johns Hopkins School of Public Health. His research is on the use of regression analyses for correlated responses. He has extended generalized linear models to situations in which observations occur in clusters, such as in longitudinal, time-series, or genetic studies. Dr. Zeger served on the NRC Committee on Health Effects Associated with Exposure During the Persian Gulf War. He received his Ph.D. from Princeton University.