Free Executive Summary

Estimating Mortality Risk Reduction and Economic Benefits from Controlling Ozone Air Pollution

Committee on Estimating Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure, National Research Council

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Summary

Increased concentrations of ozone in the lower atmosphere are formed from pollutants emitted by such human activities as the combustion of fossil fuels. Natural sources of emissions, such as vegetation, also contribute to ozone formation. Because human exposure to ozone in the lower atmosphere at the increased concentrations that result from precursor emissions can cause respiratory problems and other health effects, ozone is one of the six criteria pollutants regulated by the U.S. Environmental Protection Agency (EPA) under the Clean Air Act.[1]

Studies published since 1990 have yielded mixed evidence of a relationship between short-term exposure to ozone and premature death. According to analyses of evidence published in recent years, the risk of death in the population increases slightly but consistently as exposure to ozone increases. However, at the same time, interpretation of this evidence by EPA and scientists outside the agency has been complicated by ozone's occurrence in mixtures with other pollutants that have similar effects and by uncertainties including those which result from using outdoor-ozone measurements to estimate exposures of people who spend most of their time indoors.

The Clean Air Act requires EPA to review periodically the National Ambient Air Quality Standards (NAAQS) for the criteria pollutants.[2] Each time NAAQS are reviewed, the EPA administrator must weigh the most recent evidence and current uncertainties and make a public-health policy judgment about whether the existing standards are adequate to protect the public health with an adequate margin of safety or should be lowered or raised.

After the NAAQS are determined, EPA must address any mitigation measures needed to reduce emissions. When deciding on mitigation actions expected to cost more than $100 million per year, EPA, like other federal agencies, is required to carry out a cost-benefit analysis of alternative regulatory strategies, such as those to attain the ozone NAAQS. However, EPA is not allowed to consider monetary costs when setting NAAQS.

To assess the benefits portion of its cost-benefit analysis of the ozone NAAQS, EPA uses results of epidemiologic studies to estimate the number of premature deaths avoided by the expected reduction in ozone concentration for the population at risk (that is, the number of deaths postponed to some future year and generally with a different cause of death). It then assigns a monetary value to the avoided deaths by using a concept known as the value of a statistical life. That value is derived from studies of adults (mostly of working age) who indicate or reveal, through choices in the labor market or in other ways, the amount that they would be willing to pay to change their risk of death in a given period by a small amount. EPA applies the same value to all premature deaths avoided regardless of the age or health status of the population experiencing the potential change in risk of death. However, the willingness to pay for a reduction in the risk of death hypothetically depends on the characteristics (for example, life expectancy or health status) of the individuals affected or on the nature of the risk (for example, accident vs illness).

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[1]Most ozone in the lower atmosphere is formed by a complex series of photochemical reactions in the presence of sunlight involving nitrogen oxides and volatile organic compounds. *Ozone* is used here to refer to the broad array of photochemical oxidants in ambient air, of which ozone is the primary component.

[2]As this report was being prepared, EPA was reviewing the NAAQS for ozone. EPA's final decision on the ozone NAAQS based on its review was announced in March 2008.

In light of recent evidence on the relationship of ozone to mortality and questions about its implications for benefit analysis, EPA asked the National Research Council to establish a committee of experts to evaluate independently the contributions of recent epidemiologic studies to understanding the size of the ozone-mortality effect in the context of benefit analysis. The committee was also asked to assess methods for estimating how much a reduction in short-term exposure to ozone would reduce premature deaths, to assess methods for estimating associated increases in life expectancy, and to assess methods for estimating the monetary value of the reduced risk of premature death and increased life expectancy in the context of health-benefits analysis. The charge to this National Research Council committee focused on benefit analysis; it did not include considering how evidence is used to set the ozone NAAQS.[3]

OVERALL CONCLUSIONS AND RECOMMENDATIONS

The committee concludes from its review of the health-based evidence that short-term exposure to ambient ozone is likely to contribute to premature deaths. Despite some continuing questions about the interpretation of the evidence, the committee concluded that the evidence is strong enough to be used in the estimation of the expected mortality-reduction benefits of a decrease in exposure to ozone. Human chamber and toxicologic studies have yielded strong evidence that short-term exposure to ozone can exacerbate lung conditions, causing illness and hospitalization, and can potentially lead to death. The available evidence on ozone exposure and exacerbation of heart conditions, which is less abundant, points to another concern. Epidemiologic studies have also found that exposure to ozone is associated with adverse lung and heart effects.

> **Recommendation: The committee recommends that ozone-related mortality be included in future estimates of the health benefits of reducing ozone exposure. The committee further recommends that the greatest emphasis be placed on estimates from new systematic multicity analyses that use national databases of air pollution and mortality, such as in the National Morbidity, Mortality, and Air Pollution Study, without excluding consideration of meta-analyses of previously published studies. Emphasis should also be placed on risk estimation based on analyzing data on multiple days so that delayed acute effects estimates can be included. The health-benefits estimates should be accompanied by a broad array of analyses of uncertainty but should give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure.**

Because older persons appear to be at high risk of health-related effects from ozone pollution, it is appropriate to consider whether the willingness to pay for mortality risk reductions should and could reflect the number of years of life by which life would be extended by reductions in ozone. **The committee concludes that the evidence is insufficient to support a specific adjustment of the aggregate willingness to pay for reduction in annual mortality risk on the basis of differences in remaining life expectancy.**

> **Recommendation: Although there are many concerns about the accuracy of a willingness-to-pay (WTP) value and the corresponding value of a statistical life (VSL) that does not vary with population or risk characteristics, the committee recommends the use of a constant WTP and corresponding VSL as the most scientifically supportable approach to monetary valuation of ozone-related mortality risk given the information available in the epidemiologic and economics literature.**

[3] A full statement of the committee's change is presented in Box 1-1 of Chapter 1.

INTERPRETATION OF RESULTS OF HEALTH STUDIES

The associations between ozone exposure and premature mortality in the recent health studies appear robust, but several factors create considerable uncertainty about them. Those factors can affect estimates of risk of ozone-related mortality in various ways. In some cases, the factors would cause an underestimation of risk; in other cases, an overestimation. On balance, the committee considers the evidence from the studies to be strong enough for use in deriving risk estimates, but the various factors and their potential effects on the estimates should be fully acknowledged.

Short-Term Exposure to Ambient Ozone

Time-series epidemiologic studies of short-term effects of ozone typically characterize human exposure by using ambient concentrations measured at fixed outdoor monitoring sites. Exposure is characterized by applying an averaging period to the daily ambient monitoring data. Changes in the average values are then linked with changes in mortality. When averaged over 24 h, ambient concentrations are at best weakly associated with corresponding personal ozone exposure, although the association is stronger in the summer than in winter. For shorter averaging periods, such as the afternoon (when both personal outdoor activity and ozone concentration can be at their highest), results from one study suggest that hourly or daily peak ambient ozone concentration may be an appropriate proxy for corresponding hourly or peak personal exposure. Whether observations from that study are relevant for people at risk for ozone-related death warrants further examination.

The choice of averaging period to characterize short-term ozone exposure in linking ambient ozone concentration with mortality risk can have a large effect on estimates of benefits of emission-control programs. For example, under some conditions, efforts to lower emissions of oxides of nitrogen could reduce the daily peak ozone concentration but raise daily average concentrations (see Chapter 3). Thus, a cost-benefit analysis of an emission-control program that examines mortality and daily average concentrations of ozone could appear to have a negative effect, whereas an analysis that examines mortality and peak concentrations could appear to show a benefit. It is not known which averaging period reflects personal exposure more accurately or is more closely related to mortality risk.

> **Recommendation: Future studies of the effects of short-term ozone exposure should determine whether and how much daily peak exposures, such as 1-h or 8-h exposures, and longer-term average exposures, such as over 24 h, are associated with ozone-related mortality. Benefits assessors at EPA and elsewhere should use the results to identify the appropriate exposure averaging periods so that they can estimate how efforts to attain the ozone NAAQS will affect ozone exposure and health. Regulators should take into account the possibility that the effects of ozone-control strategies averaged over 24 h may be quite different from those averaged over shorter periods.**

Potential Confounding by Other Pollutants

Studies have not been sufficient to control fully for potential confounding by or ozone interaction with constituents of airborne particulate matter that has a diameter equal to or less than 2.5 μm ($PM_{2.5}$). Such constituents include sulfates, acids, elemental and organic carbon, and metals. The potential for confounding of ozone health effects by $PM_{2.5}$ constituents differs by region and season. For example, in the eastern United States, confounding is most likely in the summer months, when ozone and $PM_{2.5}$ are strongly correlated in many locations. In the winter, the potential for confounding is likely to be less. It will be difficult to address such confounding with currently available data, however, because data on

$PM_{2.5}$ components at many sites have only recently begun to be routinely collected and because winter ozone concentrations (see next section) are often not measured.

Recommendation: Epidemiologic research on associations between air pollution and health outcomes should investigate regional and seasonal associations between ambient (outdoor) concentrations and human exposures to ozone and $PM_{2.5}$. It should also investigate how data on pollutants can be used to control for confounding and correlations between the various pollutant measures. When possible, researchers should address those issues by focusing on groups of people who are sensitive to ozone and by using data on the chemical and physical components and size distribution of ambient particles.

Recommendation: EPA and the scientific community should account for seasonal and geographic variability in the relationship between ozone and its potential confounders and should increasingly include the growing database on $PM_{2.5}$ constituents in analyses of confounding of ozone associations. The most relevant particle-component data should be collected frequently enough to improve understanding of the potential for confounding.

Ozone-Mortality Relationships During Winter Months

There is a lack of observed association between ozone and mortality during periods when ozone is low, such as winter. Reasons for the lack of association are not well understood in part because of the decrease in monitoring during those periods. Better understanding of ozone-mortality relationships in the winter is important for full exploration of effects at low concentrations. Although ambient ozone is one of the best-characterized pollutants in the United States, ozone monitors are usually operated only during the so-called ozone season—the warmer period of the year, which varies from city to city.

Recommendation: EPA and states should extend operation of ozone monitoring into winter and report the results. The winter program should be sufficient to allow researchers to examine seasonal differences in risk, how these seasonal differences vary spatially between communities with warmer and cooler winters, and ozone-mortality relationships at lower ozone concentrations. Ozone is a regional pollutant, so winter measurements need not be collected at all the summer locations; but if measurements are collected in winter, they should be collected with the same frequency as summer measurements.

Frailty and Ozone Mortality

Benefits assessors seek information on whether the mortality risk associated with acute ozone exposure is attributable to short-term displacement (in this case, advancement) of deaths that would have occurred in a few days or more without acute ozone exposure. If it is so attributable, they can focus their efforts on estimating the value that frail people would place on reducing their ozone-mortality risk.

On the basis of available evidence, the committee concludes that deaths related to exposure to ozone are not restricted to people who are at high risk of death within a few days. For example, a recent study of data collected from several U.S. cities reports that short-term ozone exposure is likely to contribute to shortening life and not only among people who are near death. However, because the evidence comes from only one study, it warrants confirmation by other studies.

Recommendation: EPA and the scientific community should conduct additional studies to investigate whether and how much ozone-related mortality is restricted to people who are already at high risk of death within a few days and how much ozone-related mortality

occurs in people who are not already at risk of death in a few days. The studies should include use of various methods, for example, focus on investigating subjects who have diseases, such as diabetes or heart disease, that are known to be associated with air-pollution-related mortality risk.

Susceptibility

Preliminary results indicate that the effect of acute ozone exposure on mortality is likely to be larger than average in persons with pre-existing disease, especially lung and heart diseases. The list of factors that plausibly modify effects is rather long and still insufficiently investigated (see Chapter 4). Although susceptibility factors are important, the distribution of ozone-mortality-effect estimates among the categories of susceptibility is not adequately known. Consequently, the overall (population-weighted average) mortality effect in the total population is the only currently available basis of risk assessments; this approach is a source of an unknown amount of uncertainty in calculating the overall benefit of an ozone reduction in reducing mortality risk.

Recommendation: EPA and the scientific community should identify personal characteristics that are important in understanding ozone-mortality relationships. They should develop a distribution of ozone-mortality effect estimates among the categories of susceptibility; this will enable benefits assessment to include quantitative details of the heterogeneity of effects in the total population.

Presence or Absence of a Threshold for Ozone-Related Mortality

The association between short-term variations in ambient ozone concentrations and fluctuations in mortality rates is usually characterized as linear. Because the association is based on epidemiologic studies that can only approximate exposure on the basis of ambient monitoring data, the assumption of linearity should be viewed with caution. At low ozone concentrations, the question arises whether the association is linear or more accurately characterized as having a "threshold"—a concentration of ozone below which exposure poses no risk of death. Individuals have their own susceptibility, which is characterized by a unique exposure-response association; this association may include a unique threshold value that can vary with susceptibility of the individual at any given moment and with the averaging period used to assess exposure.

On the basis of its review of the evidence, the committee concludes that the association between short-term changes in ozone concentrations and mortality is generally linear throughout most of the concentration range, although uncertainties make it difficult to determine whether there is a threshold for the association at the lower end of the range. If there is a threshold, it is likely to be below the current NAAQS.

Recommendation: EPA and the scientific community should explore further how personal thresholds may vary and the extent to which one's threshold depends on one's frailty at any given moment. Because it is not clear whether there is an association between ozone and mortality in the cooler months, warmer months should be examined separately. The research should involve panel studies of individuals considered to be susceptible to premature death from ozone exposure, such as those with impaired lung or heart function. A sensitivity analysis is needed to assess how different thresholds in exposure-response relationships may affect ozone-mortality risk estimates that are based on results of epidemiologic studies.

Accounting for a Lag in Mortality Response to Ozone

Deaths related to short-term ozone exposure may not occur until several days after the exposure or may be associated with multiple short-term exposures. Many studies of short-term effects investigate a change in death rates for only one or a few days, but distributed-lag models can be used to look further ahead to capture delayed mortality, often referred to as a subacute response. **Distributed-lag analyses appear to capture the overall effects of ozone better than do single-day models, but there have been relatively few such analyses.**

> **Recommendation: EPA and the scientific community should develop appropriate databases and conduct distributed-lag analyses in future epidemiologic investigations to improve understanding of the statistical distribution of time between an increase in the ambient ozone concentration and the occurrence of deaths.**

Chronic Exposures

EPA benefits assessments have not included estimates of mortality risk due to long-term (chronic) exposure to ozone, because evidence does not directly demonstrate a causal relationship when the period between exposure and death is longer than a few days. However, the observed associations between ozone exposure and decreased small-airway lung function during childhood and adolescence suggest that ozone-related mortality is at least partially attributable to exposures that last more than a few days. The general association between poor lung function and shortened life expectancy is strong and well established, so evidence of an effect of ozone exposure on lung function increases the plausibility of mortality from chronic exposure. **The weak current evidence from cohort studies of an association of premature mortality with chronic exposure to ozone suggests that risks may be larger than those observed in acute effects studies alone.**

The standard approach to investigating effects of cumulative ozone exposure on life expectancy is the cohort study, in which large numbers of subjects are followed for several years. After taking into account all other factors that are likely to affect mortality, cohort studies can test the null hypothesis that mortality is the same among populations that have different ozone-exposure histories. However, none of the cohort studies available was designed to investigate chronic effects of ozone, and differences in ozone exposure among subjects in each study tended to be rather small. **If further confirmed, the weak current evidence from cohort studies of an association of premature mortality with longer-term exposure would support the notion that effects seen in time-series studies reflect only a portion of the total effect.**

> **Recommendation: EPA, the National Institutes of Health, and the scientific community should encourage additional studies of the association between long-term ozone exposure and mortality. They should also encourage development of models of long-term ozone exposures that can account for variations in exposure at the individual level and between and within cities. As new cohort evidence of effects of chronic exposures becomes available, EPA should consider including it in its benefits assessments.**

Effect of Ozone Exposure on Life Expectancy

Effects of long-term cumulated exposure are, by design, not addressed in short-term time-series studies. Distributed-lag models integrate the distribution of the time between exposure and death, but they focus on a short window (several days to weeks) after exposure. **It is appropriate to use time-series results to estimate changes in life expectancy due to acute exposures by using cohort life-table**

methods if it is assumed that all members of the population at risk for death from ozone exposure have the same life expectancy as others in the same age and sex cohort (see Chapter 4). However, the committee finds that that assumption is questionable because people at greatest risk for death from short-term ozone exposure are likely to be those who have pre-existing diseases and thus life expectancy lower than average for their age.

> **Recommendation: Additional studies are needed to assess the extent to which differences in susceptibility in a general population affect the variability associated with mortality risk estimates. To the extent that data are not available, models and assumptions can be used for sensitivity analysis to assess how risk estimates might vary with susceptibility.**

Characterizing Mortality Risk by Using Studies of Acute and Chronic Exposure

Ozone-mortality risk is often expressed as the expected number of deaths attributable to ozone air pollution or lives saved by reducing ozone pollution by some amount. However, reductions in ozone exposure are expected to increase life expectancy and decrease age-adjusted annual death rates (for example, number of deaths per 100,000 of population). Thus, the number of older people would increase, the absolute number of deaths at higher ages would increase, and the annual number of deaths would return to normal in future years, although they would occur at higher ages and probably with different causes. Alternative approaches to the expression of ozone-mortality effects, such as death rates, will have similar results if one is concerned only with short-term effects of pollution changes. However, when one includes subacute and chronic effects estimates on mortality, the discrepancies between the results of the different approaches increase, and the conceptual flaws of the "attributable-cases" model become more pronounced.

> **Recommendation: EPA should evaluate alternative approaches for expressing ozone-mortality risk associations and consider the implications of using them in benefits assessments. EPA should consider placing greater emphasis on reporting changes in age-specific death rates in the relevant population and develop models for consistent calculation of changes in life expectancy and changes in numbers of deaths at all ages.**

VALUATION

Willingness-to-Pay Estimates

Estimates of the value of a statistical life (VSL) are derived from estimates of an individual's willingness to pay (WTP) for changing his or her mortality risk by a small amount in a given period (usually annual). The objective of an economic-benefits assessment is to develop an aggregate estimate of the welfare gain for everyone who benefits from a policy or program that is intended to reduce risk. Both economic theory and available empirical evidence are inconclusive about how people's WTP values vary with two important individual characteristics: age, as a proxy for remaining life expectancy, and health status. The evidence is also inconclusive about how WTP varies with cause of death, but there is greater clarity that reducing the risks of latent mortality response should be valued less than reducing risks of immediate death. **The committee concludes that the empirical evidence is insufficient to support a specific quantitative adjustment of WTP estimates to account for differences in remaining life expectancy, but it does not reject the general concept that such adjustments may be appropriate.** It is plausible that people with shorter remaining life expectancy would be willing to devote less of their resources to reducing their mortality risk than those with longer remaining life expectancy. In contrast, if the condition causing the shortened life expectancy can be treated and improved and an acceptable quality

of life can be preserved or restored, people may put a high value on extending life, even if they have other health impairments or are quite elderly.

> **Recommendation: Researchers should continue to explore how WTP for reduction in mortality risk may vary with individual characteristics (such as age and health status), type of risk (such as accident vs illness), and time between changes in air quality and changes in risk. Efforts to obtain better information about the preferences of the older population regarding reductions in mortality risk will probably entail greater use of surveys in which subjects respond to hypothetical situations.**

Value of a Statistical Life, Individual Characteristics, and Risk Contexts

To estimate VSL, EPA mostly uses WTP values that are based on a context (for example, traffic accidents or workplace accidents) and a population that differ from the context and population relevant to the pollution-related risks that EPA is assessing. Two general approaches are available for measuring WTP for changes in mortality risk. The revealed-preference approach analyzes actual human behavior from which WTP for mortality-risk reductions may be estimated, for example, a wage-risk study of people's decisions on tradeoff between income and job-related mortality risk. The stated-preference approach surveys subjects' responses to hypothetical situations designed to reveal their WTP. **EPA's use of average WTP values in different risk contexts and for different population characteristics introduces considerable uncertainty about how these factors affect estimates of benefits. However, the current literature is inconclusive about how and how much the WTP values may vary with those factors. Although it is difficult to say how much the WTP values may differ, it is apparent that wage-risk studies cannot focus on the population and the risk context for ozone mortality.**

> **Recommendation: EPA should ensure that estimates of average WTP selected from the literature reflect results of both revealed-preference studies and stated-preference studies. EPA should consider the strengths and weaknesses of each study approach and consider how closely the available studies match the policy context in population at risk and type of risk. EPA should give less weight to wage-risk studies in selecting estimates of the WTP than in the past.**

The Value of a Statistical Life Year

Given that the committee recommends the development of models for estimating life years saved (in addition to estimating changes in annual death rates and reductions in premature deaths), is it feasible to assign a monetary value directly to changes in life expectancy? Use of a constant value per life year in the valuation of increases in life expectancy assumes that WTP values for mortality-risk reductions are consistently declining with increasing age. **Available empirical evidence does not support that assumption, so it does not support the use of a *constant* value of a statistical life year (VSLY) for benefits assessment. However, the economics literature does not reject the use of a *non-constant* VSLY. There is likely to be good reason to use a non-constant VSLY, but available studies do not provide robust estimates to assign to a non-constant VSLY.**

> **Recommendation: Unless future research produces empirical support for the assumptions that underlie a constant VSLY, EPA should not attempt to make valuation adjustments for changes in remaining life expectancy by estimating life years saved and applying a constant**

VSLY. More research is needed on appropriate ways to measure the values that people attach to changes in life expectancy.

Sensitivity Analyses

Use of the average VSL obtained from the literature may overestimate the WTP to reduce ozone-related risk of premature death. That is because the population of older people appears to have greater mortality risk associated with ozone. Because older people have average remaining life expectancy that is substantially less than that of the whole population, the WTP to reduce the risk of death in the older population might be less than the WTP of the population as a whole. However, the effect of shorter life expectancy on older people's WTP may be offset to some extent by a higher WTP for a reduction in risk because of their poorer health status or their higher baseline risk compared with those of the general population. Results in the empirical literature are not consistent, but several studies suggest that WTP to reduce mortality risk is constant or declines slightly with age. **That implies that a proportional adjustment of the VSL for remaining life expectancy (that is, using a constant VSLY) would result in using too low a value of WTP for reducing ozone-related mortality.**

Recommendation: Given the uncertainty in the accuracy of available estimates of the VSL for ozone-related mortality, EPA should conduct sensitivity analyses that use a range of estimates or assumptions to see how the overall conclusions of the cost-benefit comparison might change. The selection of alternative assumptions for the sensitivity analyses could be based on either theory or evidence. However, when there is less confidence in the alternative assumptions used in sensitivity analyses, their results should not be given equal weight in the presentation of results.

Recommendations for Future Research on Valuation

There is a fundamental need to understand better how age and remaining life expectancy affect WTP for reductions in mortality risk or increased life expectancy. An important next step is to ask that researchers report total age effects (WTP by age cohort) in addition to effects of age alone on WTP for a small reduction in mortality risk in a given period. Given the correlation of age with some of the other factors, there may be less uncertainty in the estimates of a total age effect. However, age-related income differences, sex differences, health differences, and the like would then be embedded in the estimates of WTP, and it might not be appropriate to use different VSLs that have these effects embedded.

Several recent economic studies have attempted to assess the effects of age and other factors on valuation. However, the efforts have been hampered by the lack of availability of the datasets produced for the published studies. EPA should urge researchers whom it funds for WTP studies to make their datasets available for future meta-analyses in addition to providing their published results.

EPA and the scientific community should explore and develop methods for characterizing and valuing changes in mortality risk that reflect the full life cycle. Studies to date have focused on WTP for annual changes in mortality risk, but the risk change of interest in most pollution-control assessments is more comprehensively described as a shift in survival probabilities across a large part of the human life span.

Environmental-benefits assessments rely primarily on estimates of WTP for reductions in risks of accidental death to estimate values for reducing risks of illness-related deaths. It is unknown how risk context (such as illness vs. a work-related accident) affects a valuation estimate. EPA and the scientific community should seek to learn more about how mortality-risk characteristics affect the valuation of reducing risks.

FUTURE REGULATORY-IMPACT ANALYSES INVOLVING OZONE MORTALITY

Because short-term exposure to ambient ozone is likely to contribute to premature deaths, future regulatory-impact analyses (RIAs) concerning ozone- control measures should include the benefits of reduced mortality risk. As in EPA's RIA for the recently finalized ozone NAAQS, emphasis should be on using estimates from new systematic multicity analyses that used national databases of air pollution and mortality, such as in the National Morbidity, Mortality, and Air Pollution Study, without excluding consideration of meta-analysis of previously published studies. Future RIAs should give little or no weight to the assumption that there is no causal association between estimated reductions in premature mortality and reduced ozone exposure. Health-benefits estimates should be accompanied by a broad array of analyses of uncertainty.

Distributed-lag models over several days appear better than single-day models at capturing the acute and subacute mortality effects of ozone exposure and should be part of future benefits assessments to the extent that they are supported in the literature.

Future RIAs should incorporate research results on the mortality effects of chronic ozone exposure and research that addresses key uncertainties related to potential confounding factors, exposure measures, and susceptibility as appropriate.

Despite many concerns about the accuracy of any specific WTP value and a corresponding VSL that does not vary with population or risk characteristics, the committee recommends a single VSL as the most scientifically supportable approach at present for monetary valuation of ozone-related mortality. Before making a substantial change in its approach for valuation of mortality-risk reductions, EPA should have fairly conclusive empirical evidence to support the change. It is the committee's judgment that the available evidence is not now sufficient to support such a change, but sensitivity analyses should explore alternative approaches and further research should be conducted to answer the questions raised about the validity of EPA's current approach. Benefits-assessment methods may need to be revised as new information emerges on characteristics of populations susceptible to an ozone-mortality risk and on variations in WTP for mortality-risk reductions (or increases in life expectancy) based on different population characteristics.

EPA should consider placing greater emphasis on reporting changes in age-specific death rates and changes in life expectancy in the relevant populations than on reporting estimates of lives saved or premature deaths avoided.

In this report, the committee has identified major gaps in knowledge about methods for assessing benefits of ozone-related mortality risk reduction and has recommended research strategies to close those gaps. The committee recognizes that many of the recommended research activities are complex and will be difficult to undertake, and that sufficient resources may not be available to undertake them all in the near term. Therefore, EPA and other agencies that might carry out the recommended research will need to set priorities and develop a strategy for addressing the various information needs.

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Board on Environmental Studies and Toxicology

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Preface

The U.S. Environmental Protection Agency (EPA) asked for an independent study by the National Research Council to evaluate the scientific and technical bases of approaches used by EPA for estimating ozone-mortality reduction and associated benefits of health-based standards over time. In response, the National Research Council established the Committee on Mortality Risk Reduction Benefits from Decreasing Tropospheric Ozone Exposure. Biographic information on the committee members is presented in Appendix A.

In the course of preparing this report, the committee met four times. At three of the meetings—which were held in Irvine, CA; Washington, DC; and Woods Hole, MA—officials of EPA and academic researchers were invited to meet with the committee and present their views and results of their work. Interested members of the public at large were also given an opportunity to speak on those occasions. The fourth meeting was held in closed session so that the committee could complete drafting its report. Subsequently, the committee held three teleconferences to complete its deliberations.

As this report was being written, EPA was in the process of reviewing the existing National Ambient Air Quality Standards (NAAQS) for ozone. The primary (health-based) ozone NAAQS was set at 0.08 parts ppm for the annual fourth-highest daily maximum 8-h average concentration, averaged over 3 years. The committee's statement of task (see Chapter 1) and its deliberations were not dependent on EPA's decisions provided in its final rule on March 12, 2008 which lowered the level of the 8-h standard to 0.075 ppm.

The committee received oral and written presentations from John Balmes, University of California, San Francisco; Michelle Bell, Yale University; Kiros Berhane, University of Southern California; J.R. DeShazo, University of California, Los Angeles; James Hammitt, Harvard University; Bryan Hubbell, EPA; Al McGartland, EPA; Joel Schwartz, Harvard University; Anne Smith, CRA International; Deborah Shprentz, consultant to the American Lung Association; and Ira Tager, University of California, Berkeley.

Nathalie Simon, of EPA, provided the committee with information from EPA and the published scientific literature.

This report has been reviewed in draft form by persons chosen for their diverse perspectives and technical expertise in accordance with procedures approved by the National Research Council's Report Review Committee. The purpose of the 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 of 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 thank the following for their review of this report: Francesca Dominici, Johns Hopkins University; Mark Frampton, University of Rochester; John Graham, Frederick S. Pardee RAND Graduate School; Jane Hall, California State University; James Hammitt, Harvard University; Fintan Hurley, Institute of Occupational Medicine (in the United Kingdom); Jonathan Levy, Harvard University; Thomas Lumley, University of Washington; Frederick Lurmann, Sonoma Technology, Inc.; Jennifer Peel, Colorado State University; Richard Smith, University of North Carolina; Ira Tager, University of California, Berkeley; and Sverre Vedal, University of Washington.

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 the report was overseen by Gilbert Omenn, University of

Michigan Medical School, and Edwin Clark, II, Earth Policy Institute. Appointed by the National Research Council, they were responsible for making certain that an independent examination of the report was carried out in accordance with institutional procedures and that all review comments were carefully considered. Responsibility for the final content of the report rests entirely with the author committee and the institution. We thank the report review monitor and coordinator.

We also thank Bailus Walker, Jr. for his constructive service on the committee; he resigned from the committee for personal reasons.

The committee's work for this report was assisted by staff of the National Research Council's Board on Environmental Studies and Toxicology (BEST). We thank Raymond Wassel, project director, and James Reisa, director of BEST. Technical information was provided by Mirsada Karalic-Loncarevic. Invaluable logistical support was provided by John Brown. Other staff members, who contributed to this effort, are Radiah Rose (senior editorial assistant) and Heidi Murray-Smith (research associate). The report was edited by Norman Grossblatt.

John C. Bailar III, *Chair*
Committee on Mortality Risk Reduction Benefits
from Decreasing Tropospheric Ozone Exposure

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