Health-Based Cost-Effectiveness of Reductions in Ambient O_3 and $PM_{2.5}$ Associated with Illustrative O_3 NAAQS Attainment Strategies

Draft Report

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March 11, 2008

Appendix Chapter 7b: Health-Based Cost-Effectiveness of Reductions in Ambient O_3 and $PM_{2.5}$ Associated with Illustrative O_3 NAAQS Attainment Strategies

7b.1 Summary

Health-based cost-effectiveness analysis (CEA) and cost-utility analysis (CUA) have been used to analyze numerous health interventions but have not been widely adopted as tools to analyze environmental policies. Analyses of environmental regulations have typically used benefit-cost analysis to characterize impacts on social welfare. Benefit-cost analyses allow for aggregation of the benefits of reducing mortality risks with other monetized benefits of reducing air pollution, including reduced risk of acute and chronic morbidity, and non-health benefits. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. However, alternative paradigms such as CEA and CUA analyses may also provide useful insights. CEA involves estimation of the costs per unit of benefit (e.g., lives or life years saved). CUA is a special type of CEA using preference-based measures of effectiveness, such as quality-adjusted life years (QALYs). QALYs were developed to evaluate the effectiveness of individual medical treatments, and EPA is still evaluating the appropriate methods for CEA for environmental regulations.

In this CEA, we estimated statistical lives saved, statistical life years saved, and QALYs gained. In addition, where relevant, we used an alternative aggregate effectiveness metric, Morbidity Inclusive Life Years (MILYs), to address some of the concerns about aggregation of life extension and quality-of-life impacts. MILYs represent the sum of life years gained due to reductions in premature mortality and the QALYs gained due to reductions in chronic morbidity. This measure may be preferred to existing QALY aggregation approaches because it does not devalue life extensions in individuals with preexisting illnesses that reduce quality of life. However, the MILY measure is still based on life years and thus still inherently gives more weight to interventions that reduce mortality and morbidity impacts for younger populations with higher remaining life expectancy.

Following the methodology used in the CEA for the PM NAAQS RIA, we did not assign QALY weights to the life years saved – i.e., we calculated life years saved, rather than QALYs gained from mortality avoided. Put another way, we assumed weights of 1.0 for all life years saved. Life years saved in the future, however, were discounted to reflect people's time preference (i.e., a benefit received now is worth more than the same benefit received in the future). We used discount rates of 3 percent and 7 percent.

For each illustrative O_3 NAAQS attainment strategy, we present several metrics: lives saved, life years saved, and cost of the regulation per life saved and per life year saved. Where possible, benefits that could not be quantified in the denominator of our cost-effectiveness ratios were monetized and subtracted from the cost of the regulation in the numerator.

Although there are indirect $PM_{2.5}$-related co-benefits associated with all the illustrative O_3 NAAQS attainment strategies considered, we were able to model the changes in $PM_{2.5}$ occurring

as a result of only one illustrative O_3 NAAQS attainment strategy[1]. Therefore $PM_{2.5}$-related co-benefits are included in the cost effectiveness metrics presented only for that one strategy. The cost effectiveness metrics presented for all of the other illustrative O_3 NAAQS attainment strategies omit the $PM_{2.5}$-related co-benefits and are therefore likely to understate the cost effectiveness of those strategies.

For the illustrative O_3 NAAQS attainment strategy for which we were able to include both direct O_3-related health benefits and indirect $PM_{2.5}$-related co-benefits, in addition to the cost effectiveness metrics listed above we also calculated MILYs and the cost of the regulation (net of the monetized benefits not included in the denominator) per MILY gained.

The results of the analysis are summarized as follows:

- Estimates of O_3-related lives saved were substantially affected by the underlying O_3-mortality study used and, to a greater extent, by the attainment scenario considered. Because all O_3-related mortality was assumed to occur in 2020, we did not discount O_3-related lives saved. Non-zero estimates of O_3-related lives saved based on Bell et al. (2004) ranged from 36 (95% CI: 12 – 60), under full attainment of an alternative standard of 0.079 ppm, to 520 (95% CI: 170 – 880), under full attainment of an alternative standard of 0.065 ppm. Estimates of O_3-related lives saved based on Levy et al. (2005) ranged from 160 (95% CI: 110 – 210) to 2,400 (95% CI: 1,600 – 3,100), under full attainment of the 0.079 ppm and 0.065 ppm alternative standards, respectively.

- Non-zero estimates of O_3-related life years saved also depended substantially on the underlying mortality study used and the attainment scenario considered. In addition, we hypothesized several alternative possible sets of life expectancies associated with age-specific O_3-related deaths avoided, and the choice of life expectancies had a large impact on the estimates of O_3-related life years saved. Using a 3 percent discount rate, the smallest non-zero estimate of O_3-related life years saved was 160 (95% CI: 54 – 270), under full attainment of the alternative standard of 0.079 ppm, based on Bell et al. (2004), and assuming that O_3-related mortality occurs only in the subpopulation with severe preexisting conditions (and thus the shortest life expectancies). The largest estimate of O_3-related life years saved was 26,000 (95% CI: 18,000 – 34,000), under full attainment of the alternative standard of 0.065 ppm, based on Levy et al. (2004), and assuming that O_3-related mortality occurs in the general population.

- Using a 7 percent discount rate, the smallest non-zero estimate of O_3-related life years saved was 140 (95% CI: 46 – 230), under full attainment of the alternative standard of 0.079 ppm, based on Bell et al. (2004), and assuming that O_3-related mortality occurs only in the subpopulation with severe preexisting conditions (and thus the shortest life expectancies). The largest estimate of O_3-related life years saved was 19,000 (95% CI: 13,000 – 25,000), under full attainment of the alternative standard of 0.065 ppm, based

[1] This illustrative attainment strategy has a baseline of partial attainment of the current standard of 0.084 ppm and a control scenario of partial attainment of an alternative standard of 0.070 ppm.

on Levy et al. (2004), and assuming that O_3-related mortality occurs in the general population.

- The estimate of $PM_{2.5}$-related lives saved under the single illustrative attainment strategy for which we were able to model the indirect changes in $PM_{2.5}$ concentrations and thus include $PM_{2.5}$ co-benefits, was 440 (95% CI: 170 – 700), based on Pope et al. (2002), and 2,400 (95% CI: 540 – 1,400), based on Laden et al. (2006). Unlike O_3-related mortality, $PM_{2.5}$-related mortality was not all assumed to occur in the year of exposure. Estimates of $PM_{2.5}$-related life years saved were thus discounted twice – first life years saved were discounted back to the year of avoided death, and then were further discounted back to 2020. Using a 3 percent discount rate, $PM_{2.5}$-related life years saved was estimated to be 4,400 (95% CI: 1,700 – 7000), based on Pope et al. (2002), and 9,900 (95% CI: 5,400 – 14,000), based on Laden et al. (2006). Using a 7 percent discount rate, the corresponding estimates using Pope et al. (2002) and Laden et al. (2006) were 3,000 (95% CI: 1,200 – 4,800) and 6,700 (95% CI: 3,700 – 9,800), respectively.

- Under the single scenario for which we were able to model the indirect changes in $PM_{2.5}$ concentrations and thus include $PM_{2.5}$ co-benefits, we estimated $PM_{2.5}$-related reductions in chronic bronchitis (CB) and non-fatal acute myocardial infarction (AMI) and the corresponding improvements in quality of life as QALYs gained. QALYs gained from $PM_{2.5}$-related reductions in CB were estimated to be 1,970 (95% CI: 270 – 4,700), using a 3 percent discount rate, and 1,300 (95% CI: 180 – 3,000) using a 7 percent discount rate. QALYs gained from $PM_{2.5}$-related reductions in AMI were estimated to be 870 (95% CI: 220 – 1,800) and 680 (95% CI: 180 – 1,400), using 3 percent and 7 percent discount rates, respectively.

- Because both costs (in the numerator) and benefits (in the denominator) increased with the stringency of the alternative regulations considered, the cost effectiveness ratios would not necessarily be expected to show a monotonic pattern across the regulations. Net cost per O_3-related life saved (in 2006 $) (in those illustrative attainment strategies for which we incorporated only O_3-related benefits) were greatest in the illustrative attainment strategy of full attainment of a 0.075 ppm standard. Even under this one strategy, however, cost effectiveness estimates varied substantially, depending on the underlying mortality study used and the discount rate (for cost) assumed – from a low estimate of $18 million per life saved (95% CI: $13 million – $25 million), based on Levy et al. (2005) and using a lower bound estimate of the 7 percent discounted cost, to a high estimate of $110 million (95% CI: $55 million – $280 million), based on Bell et al. (2004) and using an upper bound estimate of the 7 percent discounted cost. Note, however, that all of the cost effectiveness ratios for illustrative attainment strategies for which we incorporated only O_3-related benefits would tend to overstate the cost per life saved – i.e., understate cost effectiveness – because $PM_{2.5}$ co-benefits were not included in the denominator.

- Net cost per life saved tended to be substantially lower for the single scenario for which both O_3-related and $PM_{2.5}$-related lives saved were included, ranging from $1.8 million (95% CI: $1.3 million – $2.6 million), using Levy et al. (2005) and Laden et al. (2006),

to $5.4 million (95% CI: $3.2 million – $9.9 million), using Bell et al. (2004) and Pope et al. (2002).

- The pattern seen for cost per life year saved was similar to that seen for cost per life saved. Net costs per O_3-related life year saved were greatest in the illustrative attainment strategy of full attainment of a 0.075 ppm standard. However, there was substantial variability in cost effectiveness estimates across these illustrative attainment strategies. The lowest cost per life year saved was estimated to be $1.6 million (95% CI: $1.2 million – $2.3 million), under full attainment of a 0.079 ppm standard, using Levy et al. (2005) and a 3 percent discount rate, and assuming life expectancies of the general population. The highest cost per life year saved was estimated to be $29 million (95% CI: $15 million – $75 million), under full attainment of a 0.075 ppm standard, using Bell et al. (2004) and a 7 percent discount rate, and assuming life expectancies of a subpopulation with severe preexisting conditions.

- Net costs per life year saved in the single illustrative strategy for which we included both O_3-related and $PM_{2.5}$-related benefits were substantially smaller than for the other scenarios. This is not surprising, since the cost effectiveness of those other scenarios was understated – and thus the cost per life year saved was overstated – because of the omission of $PM_{2.5}$-related live years saved. The lowest estimate of net cost per life year saved for this illustrative strategy was $0.14 million (95% CI: $0.1 million – $0.2 million), based on Levy et al. (2005) and Laden et al. (2006), and, for O_3-related mortality avoided, assuming life expectancies of the general population, and using a 3 percent discount rate. The highest estimate was $0.79 million (95% CI: $0.44 million – $1.6 million), based on Bell et al. (2004) and Pope et al. (2002), and, for O_3-related mortality avoided, assuming life expectancies of a subpopulation with severe preexisting conditions, and using a 7 percent discount rate.

- Finally, under the single illustrative strategy for which we included both O_3-related and $PM_{2.5}$-related benefits, the lowest estimate of net costs per MILY gained, using a 3 percent discount rate, was $0.12 million (95% CI: $0.09 million – $0.17 million), based on Levy et al. (2005) and Laden et al. (2006) and, for O_3-related mortality avoided, assuming life expectancies of the general population; the highest estimate was $0.30 million (95% CI: $0.19 million – $0.53 million), based on Bell et al. (2004) and Pope et al. (2002) and, for O_3-related mortality avoided, assuming life expectancies of a subpopulation with severe preexisting conditions.

- Using a 7 percent discount rate, the lowest estimate of net costs per MILY gained was $0.18 million (95% CI: $0.14 million – $0.26 million), based on Levy et al. (2005) and Laden et al. (2006) and, for O_3-related mortality avoided, assuming life expectancies of the general population; the highest estimate was $0.48 million (95% CI: $0.29 million – $0.86 million), based on Bell et al. (2004) and Pope et al. (2002) and, for O_3-related mortality avoided, assuming life expectancies of a subpopulation with severe preexisting conditions.

7b.2 Introduction

Health-based cost-effectiveness analysis (CEA) and cost-utility analysis (CUA) have been used to analyze numerous health interventions but have not been widely adopted as tools to analyze environmental policies. Analyses of environmental regulations have typically used benefit-cost analysis to characterize impacts on social welfare. Benefit-cost analyses allow for aggregation of the benefits of reducing mortality risks with other monetized benefits of reducing air pollution, including reduced risk of acute and chronic morbidity, and non-health benefits. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. However, alternative paradigms such as CEA and CUA analyses may also provide useful insights. CEA involves estimation of the costs per unit of benefit (e.g., lives or life years saved). CUA is a special type of CEA using preference-based measures of effectiveness, such as quality-adjusted life years (QALYs).

QALYs were developed to evaluate the effectiveness of individual medical treatments, and EPA is still evaluating the appropriate methods for CEA for environmental regulations. Agency concerns with the standard QALY methodology include the treatment of people with fewer years to live (the elderly); fairness to people with preexisting conditions that may lead to reduced life expectancy and reduced quality of life; and how the analysis should best account for non-health benefits.

The Office of Management and Budget (OMB) recently issued Circular A-4 guidance on regulatory analyses, requiring federal agencies to "prepare a CEA for all major rulemakings for which the primary benefits are improved public health and safety to the extent that a valid effectiveness measure can be developed to represent expected health and safety outcomes." Environmental quality improvements may have multiple health and ecological benefits, however, making application of CEA more difficult and less straightforward.

The Institute of Medicine (a member institution of the National Academies of Science) established the Committee to Evaluate Measures of Health Benefits for Environmental, Health, and Safety Regulation to assess the scientific validity, ethical implications, and practical utility of a wide range of effectiveness measures used or proposed in CEA. This committee prepared a report titled "Valuing Health for Regulatory Cost-Effectiveness Analysis" which concluded that CEA is a useful tool for assessing regulatory interventions to promote human health and safety, although not sufficient for informed regulatory decisions (Miller, Robinson, and Lawrence, 2006). They emphasized the need for additional data and methodological improvements for CEA analyses, and urged greater consistency in the reporting of assumptions, data elements, and analytic methods. They also provided a number of recommendations for the conduct of regulatory CEA analyses. EPA is evaluating these recommendations and will determine a response for upcoming analyses.

CEA and CUA are most useful for comparing programs that have similar goals, for example, alternative medical interventions or treatments that can save a life or cure a disease. They are less readily applicable to programs with multiple categories of benefits, such as those reducing ambient air pollution, because the cost-effectiveness calculation is based on the quantity of a single benefit category. In other words, we cannot readily convert non-health benefits, such as

visibility improvements associated with reductions in $PM_{2.5}$ or increases in worker productivity associated with reductions in O_3, to a health metric such as life years saved. For these reasons, environmental economists prefer to present results in terms of monetary benefits and net benefits.

However, QALY-based CUA has been widely adopted within the health economics literature (Neumann, 2003; Gold et al., 1996) and in the analysis of public health interventions (US FDA, 2004). QALY-based analyses have not been as accepted in the environmental economics literature because of concerns about the theoretical consistency of QALYs with individual preferences (Hammitt, 2002), treatment of nonhuman health benefits, and a number of other factors (Freeman, Hammitt, and De Civita, 2002). For environmental regulations, benefit-cost analysis has been the preferred method of choosing among regulatory alternatives in terms of economic efficiency. Recently several academic analyses have proposed the use of life years-based benefit-cost or CEAs of air pollution regulations (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003; Rabl, 2003; Carrothers, Evans, and Graham, 2002). In addition, the World Health Organization has adopted the use of disability-adjusted life years, a variant on QALYs, to assess the global burden of disease due to different causes, including environmental pollution (Murray et al., 2002; de Hollander et al., 1999).

One of the ongoing controversies in health impact assessment regards whether reductions in mortality risk should be reported and valued in terms of statistical lives saved or in terms of statistical life years saved. Life years saved measures differentiate among premature mortalities based on the remaining life expectancy of affected individuals. In general, under the life years approach, older individuals will gain fewer life years than younger individuals for the same reduction in mortality risk during a given time period, making interventions that benefit older individuals seem less beneficial relative to similar interventions benefiting younger individuals. A further complication in the debate is whether to apply quality adjustments to life years lost. Under this approach, individuals with preexisting health conditions would have fewer QALYs lost relative to healthy individuals for the same loss in life expectancy, making interventions that primarily benefit individuals with poor health seem less beneficial than similar interventions affecting primarily healthy individuals.

In this CEA, we calculated both life years saved and statistical lives saved. Following the methodology used in the CEA for the PM NAAQS RIA, we did not assign QALY weights to the life years saved – i.e., we calculated life years saved, rather than QALYs gained from mortality avoided. Put another way, we assumed weights of 1.0 for all life years saved. Life years saved in the future, however, were discounted to reflect people's time preference (i.e., a benefit received now is worth more than the same benefit received in the future). We used discount rates of 3 percent and 7 percent.

Where possible, benefits that could not be quantified in the denominator of our cost-effectiveness ratios were monetized and subtracted from the cost of the regulation in the numerator. For example, developing QALYs for acute health effects is problematic (Bala and Zarkin, 2000). Therefore, rather than try to derive QALYs for the acute morbidity endpoints, we instead applied valuation estimates and subtracted the total monetized value of all avoided acute morbidity effects from the cost of the regulation, in the numerator of the cost-effectiveness ratios. The

monetized benefits of non-health improvements, where they were estimated, were similarly subtracted from the cost of the regulation. Finally, although QALY estimates were derived for the ($PM_{2.5}$-related) chronic morbidity endpoints, the medical and opportunity costs associated with these chronic illnesses were also subtracted from the cost of the regulation.

Although there are indirect $PM_{2.5}$-related co-benefits associated with all the illustrative O_3 NAAQS attainment strategies, we were able to model the changes in $PM_{2.5}$ occurring as a result of only one illustrative O_3 NAAQS attainment strategy (see Chapter 7 for a full discussion of this issue). Therefore $PM_{2.5}$-related co-benefits are included in the cost effectiveness metrics presented only for that one strategy. The cost effectiveness metrics presented for all of the other illustrative O_3 NAAQS attainment strategies omit the $PM_{2.5}$-related co-benefits and are therefore likely to understate the cost effectiveness of those strategies.

The indirect $PM_{2.5}$-related co-benefits derive not only from avoided cases of premature mortality and acute morbidity, but from avoided cases of chronic morbidity (chronic bronchitis and non-fatal myocardial infarction) as well. In the CEA for the PM NAAQS RIA, EPA derived QALYs for these two chronic morbidity endpoints (see Appendix G of the PM NAAQS RIA, http://www.epa.gov/ttn/ecas/regdata/RIAs/Appendix%20G--Health%20Based%20Cost%20Effectiveness%20Analysis.pdf) and used an alternative aggregate effectiveness metric, Morbidity Inclusive Life Years (MILYs), to address some of the concerns about aggregation of life extension and quality-of-life impacts. MILYs represent the sum of life years gained due to reductions in premature mortality and the QALYs gained due to reductions in chronic morbidity. This measure may be preferred to existing QALY aggregation approaches because it does not devalue life extensions in individuals with preexisting illnesses that reduce quality of life. However, the MILY measure is still based on life years and thus still inherently gives more weight to interventions that reduce mortality and morbidity impacts for younger populations with higher remaining life expectancy.

For each illustrative O_3 NAAQS attainment strategy, we present several metrics: lives saved, life years saved, and cost of the regulation (net of the monetized benefits not included in the denominator) per life saved and per life year saved.

For the illustrative O_3 NAAQS attainment strategy for which we were able to include both direct O_3-related health benefits and indirect $PM_{2.5}$-related co-benefits, in addition to the cost effectiveness metrics listed above we also calculated MILYs and the cost of the regulation (net of the monetized benefits not included in the denominator) per MILY gained.

Note that, like future life years saved, future QALYs gained from avoided cases of chronic bronchitis and myocardial infarction are discounted. All costs and monetized benefits are in 2006 dollars.

Monte Carlo simulation methods as implemented in the Crystal Ball™ software program were used to propagate uncertainty in several of the model parameters throughout the analysis. In particular, we incorporated uncertainty surrounding the coefficients in the concentration-response (C-R) functions, the unit values for the various morbidity endpoints included in the

analysis, and the quality of life weights for the two chronic morbidity endpoints for which we developed QALYs.

We characterized overall uncertainty in the results with 95 percent credible or confidence intervals based on the Monte Carlo simulations. In addition, we examined the impacts on the cost effectiveness metrics of changing key parameters and/or assumptions, including

- the discount rate (for the cost of the regulation in the numerator and future lives or life years saved and QALYs gained in the denominator);
- the C-R functions for O_3-related and $PM_{2.5}$-related mortality ; and
- the life expectancies (and therefore years of potential life lost) of individuals who die as a result of exposure to O_3 (as explained in Section 7b.5 below).

The methodology presented in this appendix is not intended to stand as precedent either for future air pollution regulations or for other EPA regulations where it may be inappropriate. It is intended solely to demonstrate one particular approach to estimating the cost-effectiveness of direct reductions in ambient O_3 (and indirect reductions in $PM_{2.5}$, where possible) in achieving improvements in public health. Reductions in ambient O_3 and $PM_{2.5}$ are estimated to have other health and environmental benefits that will not be reflected in this CEA. Other EPA regulations affecting other aspects of environmental quality and public health may require additional data and models that may preclude the development of similar health-based CEAs. A number of additional methodological issues must be considered when conducting CEAs for environmental policies, including treatment of non-health effects, aggregation of acute and long-term health impacts, and aggregation of life extensions and quality-of-life improvements in different populations. The appropriateness of health-based CEA should be evaluated on a case-by-case basis subject to the availability of appropriate data and models, among other factors.

The remainder of this appendix provides an overview of the methods used to derive the cost effectiveness metrics developed for this CEA and presents the resulting metrics. Section 7b.3 provides an overview of effectiveness measures. Section 7b.4 discusses general issues in constructing cost-effectiveness ratios. Section 7b.5 presents the methods and results for those illustrative O_3 NAAQS attainment strategies for which we were able to incorporate only the O_3-related benefits; and Section 7b.6 presents the methods and results for the single illustrative O_3 NAAQS attainment strategy for which we were able to include both the O_3-related benefits and $PM_{2.5}$-related co-benefits. Finally, Section 7b.7 presents concluding remarks.

7b.3 Effectiveness Measures

For the purposes of CEA, we focus the effectiveness measures on the quantifiable health impacts of the reductions in O_3 and, where possible, $PM_{2.5}$, estimated to result from each illustrative O_3 NAAQS attainment strategy considered. If the main impact of interest is reductions in mortality risk from air pollution, the effectiveness measures are relatively straightforward to develop. Mortality impacts can be characterized similar to the benefits analysis, by counting the number of premature deaths avoided, or can be characterized in terms of increases in life expectancy or

life years.[2] Estimates of premature mortality have the benefit of being relatively simple to calculate, are consistent with the benefit-cost analysis, and do not impose additional assumptions on the degree of life shortening. However, some have argued that counts of premature deaths avoided are problematic because a gain in life of only a few months would be considered equivalent to a gain of many life years, and the true effectiveness of an intervention is the gain in life expectancy or life years (Rabl, 2003; Miller and Hurley, 2003).

Calculations of changes in life years and life expectancy can be accomplished using standard life table methods (Miller and Hurley, 2003). However, the calculations require assumptions about the baseline mortality risks for each age cohort affected by air pollution. A general assumption may be that air pollution mortality risks affect the general mortality risk of the population in a proportional manner. However, some concerns have been raised that air pollution affects mainly those individuals with preexisting cardiovascular and respiratory disease, who may have reduced life expectancy relative to the general population. This issue is explored in more detail below.

Air pollution is also associated with a number of significant chronic and acute morbidity endpoints. Failure to consider these morbidity effects may understate the cost-effectiveness of air pollution regulations or give too little weight to reductions in particular pollutants that have large morbidity impacts but no effect on life expectancy. The QALY approach explicitly incorporates morbidity impacts into measures of life years gained and is often used in health economics to assess the cost-effectiveness of medical spending programs (Gold et al., 1996). Using a QALY rating system, health quality ranges from 0 to 1, where 1 may represent full health, 0 death, and some number in between (e.g., 0.8) an impaired condition. QALYs thus measure morbidity as a reduction in quality of life over a period of life. QALYs assume that duration and quality of life are equivalent, so that 1 year spent in perfect health is equivalent to 2 years spent with quality of life half that of perfect health. QALYs can be used to evaluate environmental rules under certain circumstances, although some very strong assumptions (detailed below) are associated with QALYs. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommended using QALYs when evaluating medical and public health programs that primarily reduce both mortality and morbidity (Gold et al., 1996). Although there are significant non-health benefits associated with air pollution regulations, over 90 percent of quantifiable monetized benefits are health-related. Thus, it can be argued that QALYs are more applicable for these types of regulations than for other environmental policies. However, the value of non-health benefits should not be ignored. As discussed below, we have chosen to subtract the value of non-health benefits from the costs in the numerator of the cost-effectiveness ratio.

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[2] Life expectancy is an *ex ante* concept, indicating the impact on an entire population's expectation of the number of life years they have remaining, before knowing which individuals will be affected. Life expectancy thus incorporates both the probability of an effect and the impact of the effect if realized. Life years is an *ex post* concept, indicating the impact on individuals who actually die from exposure to air pollution. Changes in population life expectancy will always be substantially smaller than changes in life years per premature mortality avoided, although the total life years gained in the population will be the same. This is because life expectancy gains average expected life years gained over the entire population, while life years gained measures life years gained only for those experiencing the life extension.

The use of QALYs is predicated on the assumptions embedded in the QALY analytical framework. As noted in the QALY literature, QALYs are consistent with the utility theory that underlies most of economics only if one imposes several restrictive assumptions, including independence between longevity and quality of life in the utility function, risk neutrality with respect to years of life (which implies that the utility function is linear), and constant proportionality in trade-offs between quality and quantity of life (Pliskin, Shepard, and Weinstein, 1980; Bleichrodt, Wakker, and Johannesson, 1996). To the extent that these assumptions do not represent actual preferences, the QALY approach will not provide results that are consistent with a benefit-cost analysis based on the Kaldor-Hicks criterion.[3] Even if the assumptions are reasonably consistent with reality, because QALYs represent an average valuation of health states rather than the sum of societal WTP, there are no guarantees that the option with the highest QALY per dollar of cost will satisfy the Kaldor-Hicks criterion (i.e., generate a potential Pareto improvement [Garber and Phelps, 1997]).

Benefit-cost analysis based on WTP is not without potentially troubling underlying structures as well, incorporating ability to pay (and thus the potential for equity concerns) and the notion of consumer sovereignty (which emphasizes wealth effects). Table 7b-1 compares the two approaches across a number of parameters. For the most part, WTP allows parameters to be determined empirically, while the QALY approach imposes some conditions *a priori*.

Table 7b-1. Comparison of QALY and WTP Approaches

Parameter	*QALY*	*WTP*
Risk aversion	Risk neutral	Empirically determined
Relation of duration and quality	Independent	Empirically determined
Proportionality of duration/ quality trade-off	Constant	Variable
Treatment of time/age in utility function	Utility linear in time	Empirically determined
Preferences	Community/Individual	Individual
Source of preference data	Stated	Revealed and stated
Treatment of income and prices	Not explicitly considered	Constrains choices

7b.4 Construction of Cost-Effectiveness Ratios: General Issues

7b.4.1 Dealing with Morbidity Health Effects and Non-health Effects

Health effects from exposure to O_3 and $PM_{2.5}$ air pollution encompass a wide array of chronic and acute conditions in addition to premature mortality. EPA's Ozone and PM Criteria Documents outline numerous health effects known or suspected to be linked to exposure to ambient ozone and PM (US EPA, 2006; US EPA, 2005; Anderson et al., 2004). Although chronic conditions and premature mortality generally account for the majority of monetized

[3] The Kaldor-Hicks efficiency criterion requires that the "winners" in a particular case be potentially able to compensate the "losers" such that total societal welfare improves. In this case, it is sufficient that total benefits exceed total costs of the regulation. This is also known as a potential Pareto improvement, because gains could be allocated such that at least one person in society would be better off while no one would be worse off.

benefits, acute symptoms can affect a broad population or sensitive populations (e.g., asthma-related emergency room visits among asthmatics). In addition, reductions in air pollution may result in a broad set of non-health environmental benefits, including improved worker productivity, improved visibility in national parks, increased agricultural and forestry yields, reduced acid damage to buildings, and a host of other impacts. Lives saved, life years saved, and QALYs gained address only health impacts, and the OMB guidance notes that "where regulation may yield several different beneficial outcomes, a cost-effectiveness comparison becomes more difficult to interpret because there is more than one measure of effectiveness to incorporate in the analysis."

With regard to acute health impacts, Bala and Zarkin (2000) suggest that QALYs are not appropriate for valuing acute symptoms, because of problems with both measuring utility for acute health states and applying QALYs in a linear fashion to very short duration health states. Johnson and Lievense (2000) suggest using conjoint analysis to get healthy-utility time equivalences that can be compared across acute effects, but it is not clear how these can be combined with QALYs for chronic effects and loss of life expectancy. There is also a class of effects that EPA has traditionally treated as acute, such as hospital admissions, which may also result in a loss of quality of life for a period of time following the effect. For example, life after asthma hospitalization has been estimated with a utility weight of 0.93 (Bell et al., 2001; Kerridge, Glasziou, and Hillman, 1995).

How should these effects be combined with QALYs for chronic and mortality effects? One method would be to convert the acute effects to QALYs; however, as noted above, there are problems with the linearity assumption (i.e., if a year with asthma symptoms is equivalent to 0.7 year without asthma symptoms, then 1 day without asthma symptoms is equivalent to 0.0019 QALY gained). This is troubling from both a conceptual basis and a presentation basis. An alternative approach is simply to treat acute health effects like non-health benefits and subtract the dollar value (based on WTP or COI) from compliance costs in the CEA.

To address the issues of incorporating acute morbidity and non-health benefits, OMB suggests that agencies "subtract the monetary estimate of the ancillary benefits from the gross cost estimate to yield an estimated net cost." As with benefit-cost analysis, any unquantified benefits and/or costs should be noted and an indication of how they might affect the cost-effectiveness ratio should be described. We followed this recommended "net cost" approach, specifically in netting out the benefits of health improvements other than reduced mortality and improved quality of life from avoided chronic illness – in particular, the monetized benefits of acute morbidity avoided, the medical and opportunity costs ("cost of illness") of avoided chronic illness, and the benefits of non-health improvements, including increases in worker productivity associated with reductions in O_3 and visibility improvements at national parks associated with reductions in $PM_{2.5}$ (see Chapter 7 for more details on these benefit categories).

7b.4.2 Should Life Years Gained Be Adjusted for Initial Health Status?

The methods outlined below in Sections 7b.5 and 7b.6 provide estimates of the total number of life years gained in a population, regardless of the quality of those life years, or equivalently, assuming that all life years gained are in perfect health. In some CEAs (Cohen, Hammitt, and Levy, 2003; Coyle et al., 2003), analysts have adjusted the number of life years gained to reflect

the fact that 1) the general public is not in perfect health and thus "healthy" life years are less than total life years gained and 2) those affected by air pollution may be in a worse health state than the general population and therefore will not gain as many "healthy" life years adjusted for quality, from an air pollution reduction. This adjustment, which converts life years gained into QALYs, raises a number of serious ethical issues. Proponents of QALYs have promoted the nondiscriminatory nature of QALYs in evaluating improvements in quality of life (e.g., an improvement from a score of 0.2 to 0.4 is equivalent to an improvement from 0.8 to 1.0), so the starting health status does not affect the evaluation of interventions that improve quality of life. However, for life-extending interventions, the gains in QALYs will be directly proportional to the baseline health state (e.g., an individual with a 30-year life expectancy and a starting health status of 0.5 will gain exactly half the QALYs of an individual with the same life expectancy and a starting health status of 1.0 for a similar life-extending intervention). This is troubling because it imposes an additional penalty for those already suffering from disabling conditions. Brock (2002) notes that "the problem of disability discrimination represents a deep and unresolved problem for resource prioritization."

OMB (2003) has recognized this issue in their Circular A-4 guidance, which includes the following statement:

> *When CEA is performed in specific rulemaking contexts, you should be prepared to make appropriate adjustments to ensure fair treatment of all segments of the population. Fairness is important in the choice and execution of effectiveness measures. For example, if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves the lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimated number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts. More generally, when numeric adjustments are made for life expectancy or quality of life, analysts should prefer use of population averages rather than information derived from subgroups dominated by a particular demographic or income group. (p. 13)*

This suggests two adjustments to the standard QALY methodology: one adjusting the relevant life expectancy of the affected population, and the other affecting the baseline quality of life for the affected population.

In addition to the issue of fairness, potential measurement issues are specific to the air pollution context that might argue for caution in applying quality-of-life adjustments to life years gained due to air pollution reductions. A number of epidemiological and toxicological studies link exposure to air pollution with chronic diseases, such as CB and atherosclerosis (Abbey et al., 1995; Schwartz, 1993; Suwa et al., 2002). If these same individuals with chronic disease caused by exposure to air pollution are then at increased risk of premature death from air pollution, there is an important dimension of "double jeopardy" involved in determining the correct baseline for assessing QALYs lost to air pollution (see Singer et al. [1995] for a broader discussion of the double-jeopardy argument).

Analyses estimating mortality from acute exposures that ignore the effects of long-term exposure on morbidity may understate the health impacts of reducing air pollution. Individuals exposed to chronically elevated levels of air pollution may realize an increased risk of death and chronic disease throughout life. If at some age they contract heart (or some other chronic) disease as a result of the exposure to air pollution, they will from that point forward have both reduced life expectancy and reduced quality of life. The benefit to that individual from reducing lifetime exposure to air pollution would be the increase in life expectancy plus the increase in quality of life over the full period of increased life expectancy. If the QALY loss is determined based on the underlying chronic condition and life expectancy without regard to the fact that the person would never have been in that state without long-term exposure to elevated air pollution, then the person is placed in double jeopardy. In other words, air pollution has placed more people in the susceptible pool, but then we penalize those people in evaluating policies by treating their subsequent deaths as less valuable, adding insult to injury, and potentially downplaying the importance of life expectancy losses due to air pollution. If the risk of chronic disease and risk of death are considered together, then there is no conceptual problem with measuring QALYs, but this has not been the case in recent applications of QALYs to air pollution (Carrothers, Evans, and Graham, 2002; Coyle et al., 2003). The use of QALYs thus highlights the need for a better understanding of the relationship between chronic disease and long-term exposure and suggests that analyses need to consider morbidity and mortality jointly, rather than treating each as a separate endpoint (this is an issue for current benefit-cost approaches as well).

Because of the fairness and measurement concerns discussed above, for the purposes of this analysis, we do not reduce the number of life years gained to reflect any differences in underlying health status that might reduce quality of life in remaining years. Thus, we maintain the assumption that all direct gains in life years resulting from mortality risk reductions will be assigned a weight of 1.0. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" (Gold et al., 1996). However, for the purposes of this analysis, we propose an alternative to the traditional aggregate QALY metric that keeps separate quality adjustments to life expectancy and gains in life expectancy. As such, we do not make any adjustments to life years gained to reflect the less than perfect health of the general population. Gains in quality of life will be addressed as they accrue because of reductions in the incidence of chronic diseases. This is an explicit equity choice in the treatment of issues associated with quality-of-life adjustments for increases in life expectancy that still capitalizes on the ability of QALYs to capture both morbidity and mortality impacts in a single effectiveness measure.

7b.4.3 Constructing Cost-Effectiveness Ratios

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. The estimate of costs in the numerator should include both the direct costs of the controls necessary to achieve the reduction in ambient concentrations of the air pollutant and the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). In general, because reductions in air pollution do not require direct actions by the affected populations, there are no specific costs to affected individuals (aside from the overall increases in prices that might be expected to occur as control costs are passed on by affected

industries). Likewise, because individuals do not engage in any specific actions to realize the health benefit of the pollution reduction, there are no decreases in utility (as might occur from a medical intervention) that need to be adjusted for in the denominator. Thus, the elements of the numerator are direct costs of controls minus the avoided costs of illness (COI) associated with chronic illnesses. In addition, as noted above, to account for the value of reductions in acute health impacts and non-health benefits, we netted out the monetized value of these benefits from the numerator to yield a "net cost" estimate.

The denominators of the cost-effectiveness ratios we calculated are either lives saved, life years saved, or, for the single scenario in which we were able to include both O_3-related and $PM_{2.5}$-related benefits, MILYs gained. For the MILY aggregate effectiveness measure, the denominator is simply the sum of life years gained from increased life expectancy and QALYs gained from the reductions in incidence of chronic illnesses associated with $PM_{2.5}$ – chronic bronchitis (CB) and nonfatal acute myocardial infarction (AMI).

7b.5 Cost Effectiveness Metrics Incorporating Only O_3-Related Benefits

In this section we describe the development of cost effectiveness metrics for those illustrative O_3 NAAQS attainment strategies for which we were able to incorporate only O_3-related benefits. This includes the scenarios in which the baseline is full attainment of the current O_3 standard of 0.084 ppm and the control scenarios are full attainment of the following four alternative standards: 0.079 ppm, 0.075 ppm, 0.070 ppm, and 0.065 ppm.

To generate health outcomes, we used the same framework as for the benefit-cost analysis described in Chapter 8. For convenience, we summarize the basic methodologies here. For more details, see Chapter 8 and the Environmental Benefits Mapping and Analysis Program (BenMAP) user's manual (http://www.epa.gov/ttn/ecas/benmodels.html).

BenMAP uses health impact functions to generate changes in the incidence of health effects. Health impact functions are derived from the C-R functions reported in the epidemiology literature. A standard health impact function has four components: an effect estimate from a particular epidemiological study, a baseline incidence rate for the health effect (obtained from either the epidemiology study or a source of public health statistics, such as CDC), the affected population, and the estimated change in the relevant pollutant summary measure.

A typical health impact function might look like this:

$$\Delta y = y_0 \cdot (e^{\beta \cdot \Delta x} - 1),$$

where y_0 is the baseline incidence, equal to the baseline incidence rate times the potentially affected population; β is the effect estimate; Δx is the estimated change in the pollutant (e.g., O_3 or $PM_{2.5}$) and Δy is the estimated change in incidence of the health effect (e.g., the number of deaths avoided) associated with the change in the pollutant, Δx. There are other functional forms, but the basic elements remain the same.

7b.5.1 Reductions in O_3-Related Premature Deaths

To calculate O_3-related life years saved under a given illustrative O_3 NAAQS attainment strategy, we first calculated the numbers of O_3-related statistical lives saved within 5-year age groups, using BenMAP. (For more details on the calculation of statistical lives saved using BenMAP, see Chapter 8 or the BenMAP user's manual (http://www.epa.gov/ttn/ecas/benmodels.html). We used two studies used in the benefit analysis for the O_3 NAAQS RIA – Bell et al. (2004) and Levy et al. (2005). Both studies report estimated C-R functions of the association between premature mortality and short-term exposures to ambient O_3. Bell et al. (2004) is a multi-city study of 95 cities, and as such may avoid the potential for publication bias that may be inherent in single-city studies or meta-analyses of single-city studies. This study provides the lowest estimate of O_3-related premature deaths among the mortality studies included in the O_3 NAAQS RIA benefit analysis. An upper bound estimate of O_3-related premature deaths in the O_3 NAAQS RIA benefit analysis was provided by Levy et al. (2005). More extensive discussions of these studies are given in Chapter 8.

We checked to confirm that, for each O_3 NAAQS attainment strategy, the total number of O_3-related statistical lives saved, summed across all age groups, equals the corresponding number calculated in the benefit analysis. Age group-specific O_3-related premature deaths avoided under the illustrative O_3 NAAQS attainment strategies for which we considered only O_3-related benefits are given in Table 7b-2.

Table 7b-2. Estimated Reduction in Incidence of O_3-Related Premature Mortality Associated with Illustrative O_3 NAAQS Attainment Strategies in 2020

	Reduction in O_3-Related Premature Mortality (95% CI)*							
	Bell et al. (2004)				Levy et al. (2005)			
Age Interval	Baseline of Full Attainment of Current (0.084 ppm) Standard to Control Scenario of Full Attainment of:							
	0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm	0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm
0 - 4	0 (0 - 0)	0 (0 - 1)	1 (0 - 2)	2 (1 - 3)	1 (1 - 1)	2 (2 - 3)	7 (5 - 9)	12 (8 - 16)
5 - 9	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)	1 (0 - 1)	0 (0 - 1)	1 (1 - 1)	3 (2 - 4)	5 (4 - 7)
10 - 14	0 (0 - 0)	0 (0 - 0)	0 (0 - 0)	1 (0 - 1)	0 (0 - 1)	1 (1 - 1)	3 (2 - 4)	5 (4 - 7)
15 - 19	0 (0 - 0)	0 (0 - 0)	0 (0 - 1)	1 (0 - 1)	1 (1 - 1)	2 (1 - 2)	5 (4 - 7)	9 (6 - 12)
20 - 24	0 (0 - 0)	0 (0 - 0)	0 (0 - 1)	1 (0 - 1)	1 (1 - 2)	3 (2 - 4)	9 (6 - 11)	15 (10 - 20)
25 - 29	0 (0 - 0)	0 (0 - 1)	1 (0 - 2)	3 (1 - 4)	2 (1 - 2)	4 (3 - 6)	12 (9 - 16)	22 (15 - 29)
30 - 34	0 (0 - 0)	0 (0 - 1)	1 (0 - 2)	2 (1 - 4)	2 (1 - 2)	4 (3 - 5)	12 (8 - 15)	21 (14 - 27)
35 - 39	0 (0 - 1)	1 (0 - 2)	3 (1 - 5)	5 (2 - 9)	3 (2 - 3)	6 (4 - 8)	18 (12 - 24)	32 (22 - 42)
40 - 44	0 (0 - 1)	1 (0 - 2)	3 (1 - 5)	5 (2 - 8)	2 (2 - 3)	6 (4 - 8)	17 (11 - 22)	29 (20 - 38)
45 - 49	1 (0 - 2)	2 (1 - 4)	7 (2 - 12)	12 (4 - 20)	5 (3 - 6)	12 (8 - 15)	34 (23 - 45)	60 (41 - 79)
50 - 54	1 (0 - 2)	2 (1 - 4)	7 (2 - 12)	13 (4 - 21)	5 (3 - 6)	12 (8 - 15)	35 (24 - 47)	62 (43 - 82)
55 - 59	3 (1 - 4)	7 (2 - 11)	20 (6 - 34)	35 (12 - 59)	12 (8 - 15)	30 (21 - 39)	91 (63 - 120)	160 (110 - 210)
60 - 64	2 (1 - 4)	6 (2 - 10)	19 (6 - 32)	33 (11 - 55)	11 (7 - 14)	27 (19 - 36)	85 (58 - 110)	150 (100 - 190)
65 - 69	4 (1 - 7)	11 (4 - 19)	36 (11 - 61)	63 (21 - 110)	19 (13 - 25)	50 (34 - 65)	160 (110 - 210)	280 (190 - 360)
70 - 74	3 (1 - 6)	9 (3 - 15)	28 (9 - 47)	49 (16 - 83)	15 (10 - 19)	38 (27 - 50)	120 (84 - 160)	220 (150 - 290)
75 - 79	5 (2 - 9)	14 (5 - 23)	45 (14 - 75)	80 (26 - 130)	23 (16 - 30)	61 (42 - 80)	200 (130 - 260)	350 (240 - 460)
80 - 84	3 (1 - 6)	9 (3 - 15)	29 (9 - 49)	51 (17 - 85)	15 (10 - 19)	39 (27 - 51)	130 (86 - 170)	220 (150 - 290)
85+	11 (4 - 19)	30 (10 - 50)	94 (30 - 160)	170 (54 - 280)	49 (34 - 64)	130 (90 - 170)	140 (280 - 540)	240 (500 - 960)
Total:	36 (12 - 60)	94 (31 - 160)	300 (93 - 500)	520 (170 - 880)	160 (110 - 210)	430 (300 - 560)	1,300 (920 - 1,800)	2,400 (1,600 - 3,100)

*95 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

7b.5.2 Life Years Saved as a Result of Reductions in O_3-Related Mortality Risk

The number of life years saved depends not only on the number of statistical lives saved, but also on the life expectancies associated with those statistical lives. As was pointed out in the CEA for the PM NAAQS RIA, age-specific life expectancies for the general population are calculated from mortality rates for the general population, and these reflect the prevalence of chronic disease, which shortens life expectancies. The only reason one might use lower life expectancies than those for the general population in the CEA for the O_3 NAAQS RIA is if the population at risk from exposure to O_3 was limited solely or disproportionately to individuals with preexisting chronic illness, whose life expectancies were, on average, shorter than those of the general population (unless all of those individuals had preexisting chronic illness because of long-term exposure to O_3).

It is reasonable to assume that someone who dies from exposure to an air pollutant is already in a compromised state. However, there are both acute and chronic compromised states. If an individual has an acute illness (e.g., pneumonia) that puts him at risk of mortality when exposed to a high concentration of an air pollutant, then in the absence of that high concentration he could be expected to recover from the illness and go on to live the expected number of years for someone his age – i.e., he would have the age-specific life expectancy of the general population.

If an individual has a chronic illness that makes him vulnerable to a high concentration of an air pollutant, then an important question is whether or not he would have had that chronic illness if he had not been exposed over the long term to high levels of the air pollutant.

We can categorize individuals who are at risk of dying because of exposure to an air pollutant into three groups:

- those who are vulnerable because of a preexisting acute condition;
- those who are vulnerable because of a preexisting chronic condition that they would *not* have had, had they not been exposed over the long term to high levels of the air pollutant; and
- those who are vulnerable because of a preexisting chronic condition that they would have had even in the absence of long term exposure to high levels of the air pollutant.

The age-specific life expectancies of the general population should apply to the first two groups, and the age-specific life expectancies of the subpopulation with the relevant chronic condition(s) should apply to the third group. If we knew the proportions of people who die from exposure to O_3 who are in each group, and the life expectancies of people in the third group, we could calculate the number of life years saved as follows:

$$\textit{Total life years saved} = \sum_i M_i * (p_{1i} * LE_i + p_{2i} * LE_i + p_{3i} * LE_i^*)$$

where

M_i denotes the number of O_3-related deaths of individuals age i,

LE_i denotes the general population life expectancy for age i,

LE_i^* denotes the life expectancy for age i of the subpopulation with the relevant chronic condition(s) – i.e., the third group;

p_{1i} denotes the proportion of the M_i O_3-related deaths that are in the first group;

p_{2i} denotes the proportion of the M_i O_3-related deaths that are in the second group; and

p_{3i} denotes the proportion of the M_i O_3-related deaths that are in the third group.

Unlike for $PM_{2.5}$ (discussed below in Section 7b.6), we currently lack information that would allow us to estimate the relevant proportions necessary to estimate the set of life expectancies that would be appropriate to apply to O_3-related deaths. Although there is substantial evidence linking premature mortality to short-term exposures to O_3, there is currently not similar evidence for long-term exposures. We therefore do not know if the second group above is relevant in the case of O_3-related mortality. Nor do we know what proportion of O_3-related deaths can be attributed to preexisting acute conditions (the first group) versus preexisting chronic conditions that these individuals would have had even in the absence of long term exposure to O_3 (the third group).

Because we currently lack the necessary information to determine the appropriate set of life expectancies to use in calculating life years saved associated with O_3-related premature mortality avoided, we calculated life years saved based on four different underlying assumptions:

- A lower bound assumption of zero life years saved, based on the hypothesis that the observed statistical association between premature mortality and short-term exposures to O_3 is not actually a causal relationship;
- An upper bound assumption that an O_3-related premature death of an individual of a given age will result in a loss of life years equal to the life expectancy in the general population of that age;
- Two intermediate assumptions: That the proportions of O_3-related premature deaths in the three groups delineated above (p_{1i}, p_{2i}, and p_{3i}) are such that, on average, the age-specific life expectancies among people who die O_3-related premature deaths are those of
    - people with severe preexisting chronic conditions, whose life expectancies are substantially shorter than those of the general population; and
    - people with preexisting chronic conditions of a range of severities, whose life expectancies are somewhat shorter than those of the general population.

Life years saved based on the upper bound assumption were calculated from age-specific mortality probabilities for the general population taken from the Centers for Disease Control (CDC) National Vital Statistics Reports, Vol. 56, No. 9, December 28, 2007, Table 1. Life table for the total population: United States, 2004.[4] We used a simplified method of calculating life expectancies from these age-specific mortality probabilities that yielded life expectancies that were close to the life expectancies derived using the more complicated method employed by the

[4] http://www.cdc.gov/nchs/data/nvsr/nvsr56/nvsr56_09.pdf

CDC.[5] In particular, starting with a cohort of size 1,000,000 at birth, we calculated the life-years lived between ages x and (x+1), for x = 0, 1, 2, …, 99, using the age-specific mortality probabilities taken from the CDC Vital Statistics Report (see above) and assuming that all deaths that occurred between ages x and (x+1) occurred midway through the year (i.e., we assigned 0.5 life-year to each year of death). The life expectancy at age n was then calculated as the sum of the life-years lived from age n through age 100 divided by the cohort size at age n. The life expectancy at age n is the number of life years lost due to an O_3-related premature mortality of an individual age n.

To estimate life years saved under the two intermediate assumptions about the life years lost as a result of O_3-related premature mortality, we turned to the epidemiological evidence of a statistically significant association between short-term exposures to O_3 and respiratory hospital admissions. This evidence suggests that these short-term exposures may exacerbate respiratory conditions that were preexisting. It is reasonable to suppose that some of these hospitalizations for respiratory illnesses on days of relatively high O_3 concentrations might result in death. It may also be the case that some individuals who did not go to the hospital might also die. We therefore looked for information on life expectancies of people with chronic respiratory conditions.

While there is information readily available in vital statistics sources on rates of death *from* chronic respiratory diseases, there is not similarly available information on rates of death *among that subpopulation who suffer from those diseases*. It is the latter rate – the rate of death among that subpopulation who suffers from those diseases – that is of interest.

A recent study of people with and without chronic obstructive pulmonary disease (COPD) provided data from which we were able to construct estimates of the mortality rates of interest. Mannino et al. (2006) followed a cohort of 15,440 subjects ages 43 to 66 for up to 11 years. The cohort subjects were selected from the larger cohort of the Atherosclerosis Risk in Communities (ARIC) study, which selected its subjects from the population of four U.S. communities by probability sampling.[6] The subjects in the Mannino study were limited to the ARIC participants who provided baseline information on respiratory symptoms and diagnoses, who underwent pulmonary function testing, and for whom follow-up data were available.

Using a modification of the criteria developed by the Global Initiative on Obstructive Lung Disease (GOLD), Mannino et al. (2006) classified the study subjects into COPD severity groups (or stages), with GOLD stage 0 (presence of respiratory symptoms in the absence of any lung function abnormality) being the least severe COPD group, and GOLD stages 3 and 4 being the most severe. The unadjusted death rates of the study participants (taken from Table 1 of Mannino et al., 2006), ratios of (unadjusted) death rates, and hazard ratios, based on Cox

[5] We calculated life expectancies from the mortality probabilities rather than using the life expectancies given in the CDC table because we were going to also calculate life expectancies for the subpopulations with severe COPD and with "average" COPD by adjusting the age-specific mortality probabilities and then calculating life expectancies using these adjusted probabilities.

[6] In one of the four communities probability sampling was used to select African-Americans only.

proportional hazard regressions, which took into account several covariates (including, among others, age, sex, race, smoking status, and education level) are shown in the table below. In addition, the right-most column of the table below shows the proportion of COPD subjects in the study in each GOLD category.

Table 7b-3. Death Rates and Hazard Ratios for Subjects with Varying Degrees of Severity of COPD (from Mannino et al., 2006)

GOLD* Category	N	Deaths	(%)	Person-Years	Death Rate per 1,000 Person-Years	Ratio of Death Rate to Death Rate for Normal Population	Hazard Ratio**	Proportion of COPD Subjects in GOLD Category
GOLD 3 or 4	271	92	33.9%	2,143	42.9	7.97	5.7	4.77%
GOLD 2	1,484	232	15.6%	12,852	18.1	3.35	2.4	26.14%
GOLD 1	1,679	137	8.2%	15,031	9.1	1.69	1.4	29.57%
GOLD 0	2,244	204	9.1%	20,191	10.1	1.88	1.5	39.52%
Restricted	1,101	150	13.6%	9,644	15.6	2.89	2.3	
Normal	8,661	427	4.9%	79,317	5.4	1.00	1.0	
Total	15,440	1,242	8.0%	139,178	8.9			

*Global Inititative on Obstructive Lung Disease (GOLD) guidelines for the staging of COPD severity.
**See Mannino et al. (2006), p. 117.

The ratios of unadjusted death rates are somewhat larger than the corresponding hazard ratios because these ratios were not adjusted for age. COPD is a progressive disease, so it would be expected that the proportion of older individuals would increase as the stages (and severity) increased, and this was indeed the case in the Mannino study. The hazard ratios, being based on regressions that took age into account, avoid this problem. We therefore used the hazard ratios to derive age-specific mortality rates for individuals with (1) severe COPD and (2) COPD of "average" severity. In particular, to derive age-specific mortality probabilities for the subpopulation with severe COPD, we multiplied each age-specific mortality probability for the general population by 5.7 (the hazard ratio for GOLD 3 or 4); to derive age-specific mortality probabilities for the subpopulation with "average" COPD, we multiplied each age-specific mortality probability for the general population by a weighted average of the GOLD category-specific hazard ratios, where the weight for a GOLD category was the proportion of COPD subjects in that GOLD category (given in the right-most column of Table 1 above). The weighted average hazard ratio was 1.906. Age-specific life expectancies were then derived for the severe COPD and "average" COPD subpopulations using these adjusted mortality probabilities and the method for calculating life expectancies described above.

Once an appropriate set of life expectancies has been determined (e.g., life expectancies for the general population or life expectancies for a subpopulation with severe COPD), these then provide the number of life years lost for an individual who dies at a given age. This information can then be combined with the estimated number of O_3-related premature deaths at each age calculated with BenMAP (see previous subsection). Because BenMAP calculates numbers of premature deaths avoided within age intervals, we can either allocate the premature deaths avoided within an age interval uniformly to the ages within the interval or, alternatively, we can calculate average life expectancies for the age intervals. We illustrate the first approach in

calculating O_3-related life years saved and the second approach in calculating $PM_{2.5}$-related life years saved (see Section 7b.6).

Total O_3-related life years gained was calculated as the sum of life years gained at each age:

$$Total\ life\ years\ gained = = \sum_{i=0}^{N} LE_i \times M_i$$

where LE_i is the remaining life expectancy for age i, M_i is the number of premature deaths avoided among individuals age i, and N is the oldest age considered.

For the purposes of determining cost effectiveness, it is also necessary to consider the time-dependent nature of the gains in life years. Standard economic theory suggests that benefits occurring in future years should be discounted relative to benefits occurring in the present. OMB and EPA guidance suggest discount rates of three and seven percent. Selection of a 3 percent discount rate is also consistent with recommendations from the U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine (Gold et al., 1996).

Discounted total life years gained is calculated as follows:

$$Discounted\ LY = \int_0^{LE} e^{-rt} dt,$$

where r is the discount rate, t indicates time, and LE is the life expectancy at the time when the premature death would have occurred. Because O_3-related premature mortality is associated only with short-term exposures, all O_3-related premature deaths are assumed to occur in the year of exposure. We therefore did not discount O_3-related premature deaths avoided.

Undiscounted age-specific life expectancies, and age-specific life expectancies using discount rates of 3 percent and 7 percent are given for the general population, the subpopulation of individuals with severe COPD, and the subpopulation of individuals with COPD of average severity in Tables 7b-4, 7b-5, and 7b-6, respectively. The O_3-related (discounted) life years saved, based on each of the two O_3-mortality studies and each of the assumptions about relevant life expectancies, are given, using 3 percent and 7 percent discount rates, in Tables 7b-7 and 7b-8, respectively. The O_3-related (discounted) life years saved, under the first assumption – that the observed statistical association between premature mortality and short-term exposures to O_3 is not actually a causal relationship – is zero in all cases (i.e., regardless of the mortality study used and the scenario considered), and is therefore not shown in these Tables.

Table 7b-4. Undiscounted and Discounted Age-Specific Life Expectancies for the General Population

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
0	0.006799	1,000,000	6,799	996,600	77.8	30.9	15.2
1	0.000483	993,201	480	992,961	77.3	30.8	15.2
2	0.000297	992,721	295	992,574	76.4	30.7	15.2
3	0.000224	992,427	222	992,315	75.4	30.6	15.2
4	0.000188	992,204	187	992,111	74.4	30.5	15.2
5	0.000171	992,017	170	991,932	73.4	30.4	15.2
6	0.000161	991,847	159	991,768	72.4	30.3	15.2
7	0.000151	991,688	149	991,613	71.4	30.2	15.2
8	0.000136	991,538	135	991,471	70.4	30.1	15.2
9	0.000119	991,403	118	991,345	69.5	29.9	15.1
10	0.000106	991,286	105	991,233	68.5	29.8	15.1
11	0.000112	991,180	111	991,125	67.5	29.7	15.1
12	0.000149	991,070	148	990,996	66.5	29.5	15.1
13	0.000227	990,922	225	990,809	65.5	29.4	15.1
14	0.000337	990,697	333	990,530	64.5	29.2	15.1
15	0.000460	990,363	456	990,135	63.5	29.1	15.1
16	0.000579	989,907	573	989,621	62.5	28.9	15.1
17	0.000684	989,334	677	988,996	61.6	28.8	15.0
18	0.000763	988,657	755	988,280	60.6	28.6	15.0
19	0.000819	987,902	809	987,498	59.7	28.4	15.0
20	0.000873	987,093	862	986,662	58.7	28.3	15.0
21	0.000926	986,231	913	985,775	57.8	28.1	15.0
22	0.000960	985,318	946	984,845	56.8	27.9	15.0
23	0.000972	984,372	957	983,893	55.9	27.8	14.9
24	0.000969	983,415	953	982,939	54.9	27.6	14.9
25	0.000960	982,462	943	981,991	54.0	27.4	14.9
26	0.000954	981,519	936	981,051	53.0	27.2	14.9
27	0.000952	980,583	933	980,117	52.1	27.0	14.8
28	0.000958	979,650	939	979,181	51.1	26.8	14.8
29	0.000973	978,712	952	978,235	50.2	26.5	14.8
30	0.000994	977,759	972	977,273	49.2	26.3	14.7
31	0.001023	976,787	999	976,287	48.3	26.1	14.7
32	0.001063	975,788	1,038	975,269	47.3	25.9	14.7
33	0.001119	974,750	1,091	974,205	46.4	25.6	14.6
34	0.001192	973,659	1,160	973,079	45.4	25.4	14.6
35	0.001275	972,499	1,240	971,879	44.5	25.1	14.5
36	0.001373	971,259	1,334	970,592	43.5	24.9	14.5
37	0.001493	969,925	1,448	969,201	42.6	24.6	14.4
38	0.001634	968,477	1,582	967,686	41.7	24.3	14.4
39	0.001788	966,895	1,729	966,031	40.7	24.0	14.3
40	0.001945	965,166	1,877	964,228	39.8	23.7	14.3
41	0.002107	963,290	2,029	962,275	38.9	23.5	14.2
42	0.002287	961,260	2,198	960,161	38.0	23.2	14.1
43	0.002494	959,062	2,392	957,866	37.0	22.8	14.0
44	0.002727	956,670	2,609	955,366	36.1	22.5	14.0
45	0.002982	954,061	2,845	952,639	35.2	22.2	13.9
46	0.003246	951,216	3,088	949,672	34.3	21.9	13.8

Table 7b-4. Undiscounted and Discounted Age-Specific Life Expectancies for the General Population (cont'd)

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
47	0.003520	948,129	3,337	946,460	33.5	21.6	13.7
48	0.003799	944,792	3,589	942,997	32.6	21.2	13.6
49	0.004088	941,203	3,848	939,279	31.7	20.9	13.5
50	0.004404	937,355	4,128	935,291	30.8	20.5	13.4
51	0.004750	933,227	4,433	931,010	30.0	20.2	13.3
52	0.005113	928,794	4,749	926,419	29.1	19.8	13.2
53	0.005488	924,045	5,071	921,510	28.2	19.4	13.0
54	0.005879	918,974	5,403	916,273	27.4	19.1	12.9
55	0.006295	913,571	5,751	910,696	26.6	18.7	12.7
56	0.006754	907,820	6,131	904,755	25.7	18.3	12.6
57	0.007280	901,689	6,564	898,407	24.9	17.9	12.4
58	0.007903	895,125	7,074	891,588	24.1	17.5	12.3
59	0.008633	888,051	7,667	884,217	23.3	17.1	12.1
60	0.009493	880,384	8,357	876,205	22.5	16.7	11.9
61	0.010449	872,027	9,112	867,471	21.7	16.2	11.8
62	0.011447	862,915	9,878	857,976	20.9	15.8	11.6
63	0.012428	853,037	10,601	847,736	20.1	15.4	11.4
64	0.013408	842,435	11,295	836,788	19.4	15.0	11.2
65	0.014473	831,140	12,029	825,126	18.6	14.5	11.0
66	0.015703	819,111	12,863	812,680	17.9	14.1	10.7
67	0.017081	806,249	13,771	799,363	17.2	13.7	10.5
68	0.018623	792,477	14,758	785,098	16.5	13.2	10.3
69	0.020322	777,719	15,805	769,817	15.8	12.8	10.0
70	0.022104	761,915	16,841	753,494	15.1	12.3	9.8
71	0.024023	745,073	17,899	736,124	14.4	11.9	9.5
72	0.026216	727,174	19,064	717,642	13.7	11.5	9.3
73	0.028745	708,110	20,355	697,933	13.1	11.0	9.0
74	0.031561	687,756	21,706	676,903	12.5	10.6	8.7
75	0.034427	666,050	22,930	654,585	11.9	10.2	8.4
76	0.037379	643,120	24,039	631,100	11.3	9.7	8.2
77	0.040756	619,080	25,231	606,465	10.7	9.3	7.9
78	0.044764	593,849	26,583	580,558	10.1	8.9	7.6
79	0.049395	567,266	28,020	553,256	9.6	8.5	7.3
80	0.054471	539,246	29,373	524,560	9.0	8.1	7.0
81	0.059772	509,873	30,476	494,635	8.5	7.7	6.7
82	0.065438	479,397	31,371	463,712	8.1	7.3	6.4
83	0.071598	448,026	32,078	431,987	7.6	6.9	6.1
84	0.078516	415,949	32,659	399,619	7.1	6.5	5.8
85	0.085898	383,290	32,924	366,828	6.7	6.2	5.6
86	0.093895	350,366	32,897	333,917	6.3	5.8	5.3
87	0.102542	317,468	32,554	301,192	5.9	5.5	5.0
88	0.111875	284,915	31,875	268,977	5.5	5.1	4.7
89	0.121928	253,040	30,853	237,613	5.1	4.8	4.5
90	0.132733	222,187	29,492	207,441	4.8	4.5	4.2
91	0.144318	192,695	27,809	178,791	4.4	4.2	3.9
92	0.156707	164,886	25,839	151,967	4.1	3.9	3.7
93	0.169922	139,047	23,627	127,234	3.7	3.6	3.4
94	0.183975	115,420	21,234	104,803	3.4	3.3	3.1
95	0.198875	94,186	18,731	84,820	3.0	3.0	2.8
96	0.214620	75,454	16,194	67,357	2.7	2.6	2.5
97	0.231201	59,260	13,701	52,410	2.3	2.2	2.2
98	0.248600	45,559	11,326	39,896	1.8	1.8	1.8
99	0.266786	34,233	9,133	29,667	1.2	1.2	1.2
100	1.000000	25,100	25,100	12,550	0.5	0.5	0.5

*Mortality probabilities for the general population taken from Table 1. Life table for the total population: United States, 2004. CDC National Vital Statistics Reports, Vol. 56, No. 9, December 28, 2007 http://www.cdc.gov/nchs/data/nvsr/nvsr56/nvsr56_09.pdf

Table 7b-5. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with Severe COPD

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
0	0.038755	1,000,000	38,755	980,622	54.5	27.5	14.9
1	0.002752	961,245	2,646	959,922	55.7	27.7	14.9
2	0.001692	958,599	1,622	957,788	54.9	27.5	14.9
3	0.001277	956,977	1,222	956,366	53.9	27.4	14.9
4	0.001074	955,755	1,026	955,242	53.0	27.2	14.9
5	0.000978	954,729	933	954,263	52.1	27.0	14.8
6	0.000916	953,796	873	953,359	51.1	26.8	14.8
7	0.000859	952,923	819	952,513	50.2	26.5	14.8
8	0.000777	952,104	739	951,734	49.2	26.3	14.7
9	0.000677	951,365	644	951,043	48.2	26.1	14.7
10	0.000606	950,721	576	950,433	47.3	25.8	14.7
11	0.000636	950,145	605	949,842	46.3	25.6	14.6
12	0.000850	949,540	807	949,137	45.3	25.3	14.6
13	0.001295	948,733	1,229	948,119	44.4	25.1	14.5
14	0.001918	947,505	1,818	946,596	43.4	24.8	14.5
15	0.002625	945,687	2,482	944,446	42.5	24.6	14.4
16	0.003301	943,205	3,113	941,648	41.6	24.3	14.4
17	0.003901	940,092	3,667	938,258	40.8	24.0	14.3
18	0.004351	936,424	4,075	934,387	39.9	23.8	14.3
19	0.004671	932,350	4,355	930,172	39.1	23.5	14.2
20	0.004976	927,995	4,618	925,686	38.3	23.3	14.1
21	0.005278	923,377	4,873	920,941	37.5	23.0	14.1
22	0.005472	918,504	5,026	915,991	36.7	22.7	14.0
23	0.005542	913,478	5,063	910,947	35.9	22.4	13.9
24	0.005522	908,415	5,016	905,907	35.1	22.2	13.9
25	0.005470	903,399	4,942	900,928	34.2	21.9	13.8
26	0.005436	898,458	4,884	896,016	33.4	21.6	13.7
27	0.005425	893,573	4,847	891,150	32.6	21.2	13.6
28	0.005461	888,726	4,853	886,300	31.8	20.9	13.5
29	0.005547	883,873	4,903	881,422	31.0	20.6	13.4
30	0.005668	878,970	4,982	876,479	30.1	20.2	13.3
31	0.005830	873,988	5,095	871,440	29.3	19.9	13.2
32	0.006061	868,893	5,266	866,260	28.5	19.5	13.1
33	0.006380	863,626	5,510	860,872	27.6	19.2	12.9
34	0.006792	858,117	5,828	855,203	26.8	18.8	12.8
35	0.007269	852,289	6,195	849,191	26.0	18.4	12.7
36	0.007827	846,094	6,622	842,783	25.2	18.0	12.5
37	0.008510	839,472	7,144	835,900	24.4	17.6	12.3
38	0.009312	832,328	7,750	828,452	23.6	17.2	12.2
39	0.010191	824,577	8,403	820,376	22.8	16.8	12.0
40	0.011084	816,174	9,047	811,651	22.0	16.4	11.8
41	0.012008	807,128	9,692	802,282	21.3	16.0	11.7
42	0.013035	797,436	10,395	792,238	20.5	15.6	11.5
43	0.014215	787,041	11,187	781,447	19.8	15.2	11.3
44	0.015546	775,854	12,061	769,823	19.1	14.8	11.1
45	0.016996	763,792	12,981	757,301	18.4	14.4	10.9
46	0.018503	750,811	13,892	743,865	17.7	14.0	10.7

Table 7b-5. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with Severe COPD (cont'd)

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
47	0.020061	736,919	14,784	729,527	17.0	13.6	10.4
48	0.021652	722,135	15,636	714,317	16.3	13.1	10.2
49	0.023303	706,500	16,464	698,268	15.7	12.7	10.0
50	0.025103	690,036	17,322	681,375	15.0	12.3	9.8
51	0.027075	672,714	18,214	663,607	14.4	11.9	9.5
52	0.029144	654,500	19,075	644,963	13.8	11.5	9.3
53	0.031280	635,425	19,876	625,487	13.2	11.1	9.0
54	0.033512	615,549	20,628	605,235	12.6	10.7	8.8
55	0.035880	594,921	21,346	584,248	12.0	10.3	8.5
56	0.038497	573,575	22,081	562,535	11.5	9.9	8.2
57	0.041497	551,494	22,885	540,052	10.9	9.5	8.0
58	0.045046	528,609	23,812	516,703	10.3	9.0	7.7
59	0.049211	504,797	24,842	492,376	9.8	8.6	7.4
60	0.054108	479,956	25,969	466,971	9.3	8.2	7.1
61	0.059560	453,986	27,040	440,467	8.8	7.9	6.9
62	0.065249	426,947	27,858	413,018	8.3	7.5	6.6
63	0.070839	399,089	28,271	384,953	7.9	7.1	6.3
64	0.076425	370,818	28,340	356,648	7.4	6.8	6.0
65	0.082495	342,478	28,253	328,352	7.0	6.4	5.8
66	0.089507	314,225	28,125	300,163	6.6	6.1	5.5
67	0.097361	286,100	27,855	272,173	6.2	5.7	5.2
68	0.106149	258,245	27,413	244,539	5.8	5.4	5.0
69	0.115833	230,833	26,738	217,463	5.4	5.1	4.7
70	0.125993	204,094	25,714	191,237	5.1	4.8	4.4
71	0.136933	178,380	24,426	166,167	4.7	4.5	4.2
72	0.149433	153,954	23,006	142,451	4.4	4.2	3.9
73	0.163847	130,948	21,455	120,220	4.1	3.9	3.7
74	0.179896	109,493	19,697	99,644	3.8	3.6	3.5
75	0.196231	89,795	17,621	80,985	3.5	3.4	3.2
76	0.213062	72,175	15,378	64,486	3.2	3.1	3.0
77	0.232309	56,797	13,194	50,200	3.0	2.9	2.8
78	0.255152	43,603	11,125	38,040	2.7	2.7	2.6
79	0.281552	32,477	9,144	27,905	2.5	2.4	2.4
80	0.310486	23,333	7,245	19,711	2.3	2.2	2.2
81	0.340699	16,089	5,481	13,348	2.1	2.0	2.0
82	0.372994	10,607	3,956	8,629	1.9	1.9	1.8
83	0.408108	6,651	2,714	5,294	1.7	1.7	1.7
84	0.447543	3,937	1,762	3,056	1.5	1.5	1.5
85	0.489619	2,175	1,065	1,642	1.4	1.4	1.4
86	0.535199	1,110	594	813	1.3	1.3	1.2
87	0.584489	516	302	365	1.1	1.1	1.1
88	0.637689	214	137	146	1.0	1.0	1.0
89	0.694992	78	54	51	0.9	0.9	0.9
90	0.756579	24	18	15	0.8	0.8	0.8
91	0.822612	6	5	3	0.6	0.6	0.6
92	0.893232	1	0	0	0.0	0.0	0.0

*Mortality probabilities derived from mortality probabilities for the general population by multiplying by the hazard ratio (5.7) for GOLD 3 or 4, from Mannino et al. (2006).

Table 7b-6. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with COPD of Average Severity

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
0	0.012960	1,000,000	12,960	993,520	69.6	29.9	15.1
1	0.000920	987,040	908	986,586	69.5	29.9	15.1
2	0.000566	986,132	558	985,853	68.6	29.8	15.1
3	0.000427	985,574	421	985,363	67.6	29.7	15.1
4	0.000359	985,153	354	984,976	66.7	29.5	15.1
5	0.000327	984,799	322	984,638	65.7	29.4	15.1
6	0.000306	984,477	301	984,326	64.7	29.3	15.1
7	0.000287	984,176	283	984,034	63.7	29.1	15.1
8	0.000260	983,893	256	983,765	62.7	29.0	15.1
9	0.000226	983,638	223	983,526	61.8	28.8	15.1
10	0.000203	983,415	199	983,315	60.8	28.6	15.0
11	0.000213	983,216	209	983,111	59.8	28.5	15.0
12	0.000284	983,006	279	982,867	58.8	28.3	15.0
13	0.000433	982,727	426	982,514	57.8	28.1	15.0
14	0.000642	982,302	630	981,986	56.8	27.9	15.0
15	0.000878	981,671	862	981,241	55.9	27.8	14.9
16	0.001104	980,810	1,083	980,268	54.9	27.6	14.9
17	0.001304	979,727	1,278	979,088	54.0	27.4	14.9
18	0.001455	978,449	1,424	977,737	53.1	27.2	14.9
19	0.001562	977,025	1,526	976,262	52.1	27.0	14.8
20	0.001664	975,499	1,623	974,688	51.2	26.8	14.8
21	0.001765	973,876	1,719	973,017	50.3	26.6	14.8
22	0.001830	972,157	1,779	971,268	49.4	26.4	14.7
23	0.001853	970,378	1,798	969,479	48.5	26.1	14.7
24	0.001846	968,580	1,788	967,686	47.6	25.9	14.7
25	0.001829	966,792	1,769	965,907	46.7	25.7	14.6
26	0.001818	965,023	1,754	964,146	45.7	25.5	14.6
27	0.001814	963,269	1,747	962,395	44.8	25.2	14.5
28	0.001826	961,521	1,756	960,643	43.9	25.0	14.5
29	0.001855	959,766	1,780	958,875	43.0	24.7	14.5
30	0.001896	957,985	1,816	957,077	42.1	24.4	14.4
31	0.001949	956,169	1,864	955,237	41.1	24.2	14.3
32	0.002027	954,305	1,934	953,338	40.2	23.9	14.3
33	0.002133	952,371	2,032	951,355	39.3	23.6	14.2
34	0.002271	950,339	2,158	949,260	38.4	23.3	14.1
35	0.002431	948,181	2,305	947,028	37.5	23.0	14.1
36	0.002617	945,876	2,476	944,638	36.6	22.7	14.0
37	0.002846	943,400	2,685	942,058	35.7	22.4	13.9
38	0.003114	940,716	2,929	939,251	34.8	22.0	13.8
39	0.003408	937,786	3,196	936,189	33.9	21.7	13.7
40	0.003707	934,591	3,464	932,859	33.0	21.4	13.6
41	0.004016	931,127	3,739	929,257	32.1	21.0	13.5
42	0.004359	927,388	4,042	925,366	31.2	20.7	13.4
43	0.004753	923,345	4,389	921,151	30.4	20.3	13.3
44	0.005199	918,956	4,777	916,567	29.5	20.0	13.2
45	0.005683	914,179	5,196	911,581	28.7	19.6	13.1
46	0.006187	908,983	5,624	906,171	27.8	19.2	13.0

Table 7b-6. Undiscounted and Discounted Age-Specific Life Expectancies for the Subpopulation with COPD of Average Severity (cont'd)

Age at Beginning of Year	Mortality Probability*	Cohort Size	Deaths in Year	Life-Years in Year	Age-Specific Life Expectancy	3% Discounted Remaining Life Expectancy	7% Discounted Remaining Life Expectancy
47	0.006709	903,359	6,060	900,329	27.0	18.9	12.8
48	0.007241	897,298	6,497	894,050	26.2	18.5	12.7
49	0.007793	890,801	6,942	887,331	25.3	18.1	12.5
50	0.008395	883,860	7,420	880,150	24.5	17.7	12.4
51	0.009054	876,440	7,935	872,472	23.7	17.3	12.2
52	0.009746	868,505	8,464	864,273	23.0	16.9	12.1
53	0.010460	860,040	8,996	855,542	22.2	16.5	11.9
54	0.011207	851,044	9,537	846,276	21.4	16.1	11.7
55	0.011999	841,507	10,097	836,458	20.6	15.7	11.5
56	0.012874	831,410	10,703	826,058	19.9	15.3	11.3
57	0.013877	820,707	11,389	815,012	19.1	14.8	11.1
58	0.015064	809,318	12,191	803,222	18.4	14.4	10.9
59	0.016456	797,127	13,118	790,568	17.7	14.0	10.7
60	0.018094	784,009	14,186	776,916	17.0	13.5	10.4
61	0.019917	769,823	15,333	762,157	16.3	13.1	10.2
62	0.021820	754,490	16,463	746,259	15.6	12.7	10.0
63	0.023689	738,028	17,483	729,286	14.9	12.3	9.7
64	0.025557	720,545	18,415	711,337	14.3	11.8	9.5
65	0.027587	702,130	19,370	692,445	13.6	11.4	9.2
66	0.029932	682,760	20,436	672,542	13.0	11.0	8.9
67	0.032558	662,324	21,564	651,542	12.4	10.5	8.7
68	0.035497	640,760	22,745	629,388	11.8	10.1	8.4
69	0.038735	618,015	23,939	606,046	11.2	9.7	8.1
70	0.042133	594,076	25,030	581,561	10.6	9.3	7.8
71	0.045791	569,046	26,057	556,017	10.1	8.9	7.6
72	0.049971	542,989	27,134	529,422	9.6	8.4	7.3
73	0.054791	515,855	28,264	501,723	9.0	8.0	7.0
74	0.060158	487,591	29,333	472,924	8.5	7.6	6.7
75	0.065621	458,258	30,071	443,223	8.0	7.3	6.4
76	0.071249	428,187	30,508	412,933	7.6	6.9	6.1
77	0.077685	397,679	30,894	382,232	7.1	6.5	5.8
78	0.085324	366,785	31,296	351,137	6.7	6.1	5.6
79	0.094152	335,489	31,587	319,696	6.2	5.8	5.3
80	0.103828	303,902	31,554	288,125	5.8	5.4	5.0
81	0.113932	272,349	31,029	256,834	5.5	5.1	4.7
82	0.124731	241,319	30,100	226,269	5.1	4.8	4.5
83	0.136473	211,219	28,826	196,806	4.8	4.5	4.2
84	0.149661	182,394	27,297	168,745	4.4	4.2	4.0
85	0.163731	155,096	25,394	142,399	4.1	3.9	3.7
86	0.178974	129,702	23,213	118,096	3.8	3.7	3.5
87	0.195456	106,489	20,814	96,082	3.5	3.4	3.3
88	0.213247	85,675	18,270	76,540	3.3	3.2	3.1
89	0.232409	67,405	15,666	59,572	3.0	3.0	2.8
90	0.253004	51,740	13,090	45,194	2.8	2.7	2.7
91	0.275086	38,649	10,632	33,333	2.6	2.5	2.5
92	0.298702	28,017	8,369	23,833	2.4	2.4	2.3
93	0.323890	19,649	6,364	16,467	2.2	2.2	2.1
94	0.350677	13,285	4,659	10,955	2.0	2.0	2.0
95	0.379078	8,626	3,270	6,991	1.9	1.8	1.8
96	0.409089	5,356	2,191	4,261	1.7	1.7	1.6
97	0.440695	3,165	1,395	2,468	1.5	1.5	1.5
98	0.473858	1,770	839	1,351	1.3	1.3	1.3
99	0.508523	931	474	695	1.0	1.0	1.0
100	1.000000	458	458	229	0.5	0.5	0.5

*Mortality probabilities derived from mortality probabilities for the general population (see Table 2) by multiplying by the weighted average of hazard ratios for the GOLD severity categories (1.906) from Mannino et al. (2006).

Table 7b-7. Estimated Discounted O_3-Related Life Years Saved Under Alternative Illustrative O_3 NAAQS Attainment Strategies in 2020, Using a 3 Percent Discount Rate

Estimated O_3-Related Life Years Saved* (95% CI)**							
Bell et al. (2004)				Levy et al. (2005)			
Baseline: Full Attainment of Current (0.084 ppm) Standard; Control Scenario: Full Attainment of Alternative Standard of:							
0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm	0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm
Assuming Life Expectancies of the General Population							
380 (130 - 630)	980 (320 - 1,600)	3,000 (960 - 5,100)	5,400 (1,700 - 9,000)	1,800 (1,300 - 2,400)	4,700 (3,300 - 6,200)	15,000 (10,000 - 19,000)	26,000 (18,000 - 34,000)
Assuming Life Expectancies of the Sub-Population with COPD of Average Severity							
290 (97 - 480)	750 (250 - 1,300)	2,300 (740 - 3,900)	4,100 (1,300 - 6,900)	1,400 (1,000 - 1,900)	3,700 (2,500 - 4,800)	11,000 (7,800 - 15,000)	20,000 (14,000 - 26,000)
Assuming Life Expectancies of the Sub-Population with Severe COPD							
160 (54 - 270)	420 (140 - 700)	1,300 (400 - 2,200)	2,300 (730 - 3,800)	840 (580 - 1,100)	2,100 (1,500 - 2,800)	6,500 (4,500 - 8,600)	11,000 (7,900 - 15,000)

*The O_3-related (discounted) life years saved, under the first assumption – that the observed statistical association between premature mortality and short-term exposures to O_3 is not actually a causal relationship – is zero in all cases (i.e., regardless of the mortality study used and the scenario considered, and is therefore not shown.

**95 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

Table 7b-8. Estimated Discounted O_3-Related Life Years Saved Under Alternative Illustrative O_3 NAAQS Attainment Strategies in 2020, Using a 7 Percent Discount Rate

Estimated O_3-Related Life Years Saved* (95% CI)**							
Bell et al. (2004)				Levy et al. (2005)			
Baseline: Full Attainment of Current (0.084 ppm) Standard; Control Scenario: Full Attainment of Alternative Standard of:							
0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm	0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm
Assuming Life Expectancies of the General Population							
290 (96 - 480)	750 (250 - 1,200)	2,300 (740 - 3,900)	4,100 (1,300 - 6,900)	1,400 (940 - 1,800)	3,500 (2,400 - 4,600)	11,000 (7,500 - 14,000)	19,000 (13,000 - 25,000)
Assuming Life Expectancies of the Sub-Population with COPD of Average Severity							
230 (77 - 390)	600 (200 - 1,000)	1,900 (590 - 3,200)	3,300 (1,100 - 5,500)	1,100 (770 - 1,500)	2,900 (2,000 - 3,800)	8,900 (6,100 - 12,000)	16,000 (11,000 - 21,000)
Assuming Life Expectancies of the Sub-Population with Severe COPD							
140 (46 - 230)	350 (120 - 590)	1,100 (340 - 1,800)	1,900 (620 - 3,200)	690 (480 - 900)	1,800 (1,200 - 2,300)	5,400 (3,700 - 7,100)	9,500 (6,500 - 12,000)

*The O_3-related (discounted) life years saved, under the first assumption – that the observed statistical association between premature mortality and short-term exposures to O_3 is not actually a causal relationship – is zero in all cases (i.e., regardless of the mortality study used and the scenario considered, and is therefore not shown.

**95 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

7b.5.3 Cost-Effectiveness Ratios

For each illustrative O_3 NAAQS attainment strategy for which we considered only O_3-related benefits, we calculated one set of cost-effectiveness ratios using total lives saved, based on the Bell study and the Levy study, as the denominator, and another set using total life years saved as the denominator. As discussed above in Section 7b.4, we netted out the monetized benefits of avoided cases of O_3-related acute morbidity (respiratory hospital admissions, asthma-related ER visits, school absence days, and minor restricted activity days) as well as avoided O_3-related worker productivity losses from the direct costs of the controls necessary to achieve the reductions in ambient concentrations of O_3 in the numerator. Incidences of avoided acute morbidity are given in Chapter 8.

We used Monte Carlo procedures to incorporate the uncertainty surrounding the O_3 coefficient in each of the C-R functions (including C-R functions for each of the acute morbidity endpoints as well as the C-R function for mortality) as well as the uncertainty surrounding the unit value (monetized benefit of an avoided case) of each acute morbidity endpoint. This procedure was repeated separately for each of the two mortality C-R functions used, and, for cost-effectiveness ratios using life years saved, for each combination of mortality C-R function and assumption about relevant life expectancies. The results are shown in Table 7b-9 for cost-effectiveness ratios using lives saved. As noted above, O_3-related premature mortality avoided (lives saved) are assumed to be related only to short-term exposures and are not discounted. The cost of the regulation, however, which occurs over a period of time, is discounted (using discount rates of 3 percent and 7 percent). Tables 7b-10 and 7b-11 show cost-effectiveness ratios using life years saved, using discount rates of 3 and 7 percent, respectively. Both the costs of the regulation and the lives saved are discounted.

As noted in Section 1, these cost-effectiveness ratios omit the $PM_{2.5}$-related co-benefits of these illustrative O_3 NAAQS strategies and are therefore likely to understate the cost effectiveness of these strategies. As can be seen in Tables 7b-9 through 7b-11, the direct costs of the controls necessary to achieve the reductions in ambient concentrations of O_3, in the numerators of the cost-effectiveness ratios, increase with the stringency of the alternative standards. The lives and life years saved, in the denominators of the cost-effectiveness ratios, similarly increase with the stringency of the alternative standards. It is therefore not surprising that we do not see a monotonic trend in these ratios across the increasingly more stringent alternative standards.

Table 7b-9. Estimated Net Cost (2006$) per O_3-Related Life Saved Under Alternative Illustrative O_3 NAAQS Attainment Strategies in 2020

Mortality Study	Cost Effectiveness Ratio: Net Cost (in Million $) per Life Saved* (95% CI)**			
	Change From Full Attainment of the Current (0.084 ppm) Std. To Full Attainment of Alternative Std. of:			
	0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm
*Estimated 3% discounted cost of the regulation (in Billion $):***	$2.9	$8.8	$25	$44
Bell et al. (2004)	$93 ($48 - $240)	$110 ($55 - $280)	$98 ($50 - $260)	$96 ($50 - $260)
Levy et al. (2005)	$18 ($13 - $25)	$21 ($15 - $29)	$19 ($14 - $27)	$19 ($14 - $27)
Using lower bound estimate of 7% discounted cost of the regulation (in Billion $):	$2.4	$7.6	$19	$32
Bell et al. (2004)	$76 ($40 - $200)	$92 ($48 - $240)	$74 ($38 - $200)	$70 ($36 - $190)
Levy et al. (2005)	$15 ($11 - $21)	$18 ($13 - $25)	$14 ($11 - $20)	$14 ($10 - $19)
Using upper bound estimate of 7% discounted cost of the regulation (in Billion $):	$2.9	$8.8	$25	$44
Bell et al. (2004)	$93 ($48 - $240)	$110 ($55 - $280)	$98 ($50 - $260)	$96 ($50 - $260)
Levy et al. (2005)	$18 ($13 - $25)	$21 ($15 - $29)	$19 ($14 - $27)	$19 ($14 - $27)

*Because $PM_{2.5}$-related benefits are not incorporated in these cost effectiveness ratios, the cost effectiveness of full attainment of each alternative O_3 standard shown in this table will tend to be understated.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 coefficients in the mortality and morbidity endpoints as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

***Uses the upper bound estimates of the 7% discounted costs of the regulations as proxies for the 3% discounted costs.

Table 7b-10. Estimated Net Cost (2006$) per O_3-Related Life Year Saved Under Alternative Illustrative O_3 NAAQS Attainment Strategies in 2020, Using a 3 Percent Discount Rate

Mortality Study	Life Expectancy Assumption	Cost Effectiveness Ratio: Net Cost (in Million $) per Life Year Saved* (95% CI)**			
		Change From Full Attainment of the Current (0.084 ppm) Std. To Full Attainment of Alternative Std. of:			
		0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm
*Estimated 3% discounted cost of the regulation (in Billion $):***		$2.9	$8.8	$25	$44
Bell et al. (2004)	General Population	$8.7 ($4.6 - $23)	$10 ($5.3 - $27)	$9.5 ($4.8 - $26)	$9.6 ($4.8 - $25)
Bell et al. (2004)	Subpopulation with Average COPD	$11 ($5.9 - $29)	$13 ($6.9 - $35)	$12 ($6.3 - $34)	$12 ($6.3 - $33)
Bell et al. (2004)	Subpopulation with Severe COPD	$20 ($11 - $53)	$24 ($13 - $63)	$22 ($11 - $61)	$22 ($12 - $59)
Levy et al. (2005)	General Population	$1.6 ($1.2 - $2.3)	$1.9 ($1.4 - $2.7)	$1.7 ($1.3 - $2.5)	$1.7 ($1.3 - $2.5)
Levy et al. (2005)	Subpopulation with Average COPD	$2.0 ($1.5 - $2.9)	$2.4 ($1.8 - $3.4)	$2.2 ($1.7 - $3.2)	$2.2 ($1.7 - $3.2)
Levy et al. (2005)	Subpopulation with Severe COPD	$3.5 ($2.6 - $4.9)	$4.2 ($3.1 - $5.9)	$3.9 ($2.9 - $5.5)	$3.9 ($2.9 - $5.5)

*Because $PM_{2.5}$-related benefits are not incorporated in these cost effectiveness ratios, the cost effectiveness of full attainment of each alternative O_3 standard shown in this table will tend to be understated.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

***Uses the upper bound estimates of the 7% discounted costs of the regulations as proxies for the 3% discounted costs.

Table 7b-11. Estimated Net Cost (2006$) per O_3-Related Life Year Saved Under Alternative Illustrative O_3 NAAQS Attainment Strategies in 2020, Using a 7 Percent Discount Rate

Mortality Study	Life Expectancy Assumption	Cost Effectiveness Ratio: Net Cost (in Million $) per Life Year Saved* (95% CI)**			
		Change From Full Attainment of the Current (0.084 ppm) Std. To Full Attainment of Alternative Std. of:			
		0.079 ppm	0.075 ppm	0.070 ppm	0.065 ppm
Using lower bound estimate of 7% discounted cost of the regulation (in Billion $):		$2.4	$7.6	$19	$32
Bell et al. (2004)	General Population	$9.4 ($4.9 - $25)	$12 ($6 - $30)	$9.5 ($4.8 - $25)	$8.8 ($4.6 - $24)
Bell et al. (2004)	Subpopulation with Average COPD	$12 ($6.1 - $31)	$14 ($7.5 - $38)	$12 ($5.9 - $32)	$11 ($5.7 - $29)
Bell et al. (2004)	Subpopulation with Severe COPD	$20 ($10 - $52)	$25 ($13 - $64)	$20 ($10 - $55)	$19 ($9.8 - $50)
Levy et al. (2005)	General Population	$1.8 ($1.3 - $2.5)	$2.2 ($1.6 - $3.1)	$1.7 ($1.3 - $2.5)	$1.7 ($1.2 - $2.4)
Levy et al. (2005)	Subpopulation with Average COPD	$2.2 ($1.6 - $3.1)	$2.7 ($2 - $3.8)	$2.2 ($1.6 - $3.1)	$2.1 ($1.5 - $2.9)
Levy et al. (2005)	Subpopulation with Severe COPD	$3.5 ($2.6 - $5)	$4.4 ($3.3 - $6.2)	$3.6 ($2.6 - $5.1)	$3.4 ($2.5 - $4.8)
Using upper bound estimate of 7% discounted cost of the regulation (in Billion $):		$2.9	$8.8	$25	$44
Bell et al. (2004)	General Population	$11 ($6 - $30)	$14 ($7 - $35)	$13 ($6.3 - $34)	$12 ($6.3 - $32)
Bell et al. (2004)	Subpopulation with Average COPD	$14 ($7.4 - $37)	$17 ($8.7 - $44)	$16 ($7.8 - $42)	$15 ($7.9 - $41)
Bell et al. (2004)	Subpopulation with Severe COPD	$24 ($13 - $63)	$29 ($15 - $75)	$27 ($13 - $72)	$26 ($14 - $70)
Levy et al. (2005)	General Population	$2.2 ($1.6 - $3)	$2.5 ($1.9 - $3.6)	$2.3 ($1.7 - $3.3)	$2.3 ($1.7 - $3.3)
Levy et al. (2005)	Subpopulation with Average COPD	$2.6 ($2 - $3.7)	$3.1 ($2.3 - $4.4)	$2.9 ($2.1 - $4.1)	$2.8 ($2.1 - $4)
Levy et al. (2005)	Subpopulation with Severe COPD	$4.3 ($3.2 - $6)	$5.1 ($3.8 - $7.2)	$4.7 ($3.5 - $6.7)	$4.7 ($3.5 - $6.7)

*Because $PM_{2.5}$-related benefits are not incorporated in these cost effectiveness ratios, the cost effectiveness of full attainment of each alternative O_3 standard shown in this table will tend to be understated.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O3 coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

7b.6 Cost-Effectiveness Metrics Incorporating Both O_3-Related and $PM_{2.5}$-Related Benefits

In this section we describe the development of cost-effectiveness metrics for the single illustrative O_3 NAAQS attainment strategy for which we were able to incorporate both O_3-related benefits and $PM_{2.5}$-related co-benefits, in which the baseline is partial attainment of the current O_3 standard of 0.084 ppm and the control scenario is partial attainment of an alternative standard of 0.070 ppm.

7b.6.1 O_3-related Lives Saved and Life Years Saved

The methods used to calculate O_3-related lives saved and O_3-related life years saved under this scenario are the same as those described above in Section 7b.5. Estimated numbers of O_3-related premature deaths avoided are shown in Table 7b-12. The corresponding O_3-related life years saved, discounted using 3 percent and 7 percent discount rates, are shown in Tables 7b-13 and 7b-14, respectively.

Table 7b-12. Estimated Reduction in Incidence of O_3-Related Premature Mortality Associated with Illustrative O_3 NAAQS Attainment Strategy in 2020: Changing from Partial Attainment of the Current O_3 NAAQS to Partial Attainment of an Alternative O_3 NAAQS of 0.07 ppm

Age Interval	Reduction in O_3-Related Premature Mortality (95% CI)*	
	Baseline of Partial Attainment of Current (0.084 ppm) Standard to Control Scenario of Partial Attainment of 0.07 ppm	
	Bell et al. (2004)	Levy et al. (2005)
0 - 4	0 (0 - 1)	3 (2 - 3)
5 - 9	0 (0 - 0)	1 (1 - 2)
10 - 14	0 (0 - 0)	1 (1 - 1)
15 - 19	0 (0 - 0)	2 (1 - 3)
20 - 24	0 (0 - 0)	3 (2 - 4)
25 - 29	1 (0 - 1)	4 (3 - 6)
30 - 34	0 (0 - 1)	4 (3 - 6)
35 - 39	1 (0 - 2)	7 (4 - 9)
40 - 44	1 (0 - 2)	6 (4 - 8)
45 - 49	3 (1 - 5)	13 (9 - 17)
50 - 54	3 (1 - 5)	14 (9 - 18)
55 - 59	8 (2 - 14)	37 (25 - 50)
60 - 64	8 (2 - 14)	36 (24 - 48)
65 - 69	16 (5 - 27)	70 (47 - 94)
70 - 74	12 (4 - 21)	55 (37 - 73)
75 - 79	20 (6 - 33)	86 (58 - 110)
80 - 84	12 (4 - 21)	55 (37 - 73)
85+	40 (12 - 68)	170 (120 - 230)
Total:	130 (36 - 220)	570 (380 - 760)

*95 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

Table 7b-13. Estimated O_3-Related Life Years Saved Associated with Illustrative O_3 NAAQS Attainment Strategy in 2020: Changing from Partial Attainment of the Current O_3 NAAQS to Partial Attainment of an Alternative O_3 NAAQS of 0.07 ppm, Using a 3 Percent Discount Rate

Estimated O_3-Related Life Years Saved (95% CI)*		
Baseline: Partial Attainment of Current (0.084 ppm) Standard; Control Scenario: Partial Attainment of Alternative Standard of 0.070 ppm		
Mortality Study:	Bell et al (2004)	Levy et al. (2005)
Assuming Life Expectancies of the General Population	1,300 (370 - 2,200)	6,100 (4,100 - 8,100)
Assuming Life Expectancies of the Sub-Population with COPD of Average Severity	980 (280 - 1,700)	4,700 (3,200 - 6,300)
Assuming Life Expectancies of the Sub-Population with Severe COPD	530 (150 - 910)	2,700 (1,800 - 3,500)

*95 percent confidence or credible intervals are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

Table 7b-14. Estimated O_3-Related Life Years Saved Associated with Illustrative O_3 NAAQS Attainment Strategy in 2020: Changing from Partial Attainment of the Current O_3 NAAQS to Partial Attainment of an Alternative O_3 NAAQS of 0.07 ppm, Using a 7 Percent Discount Rate

Estimated O_3-Related Life Years Saved (95% CI)*		
Baseline: Partial Attainment of Current (0.084 ppm) Standard; Control Scenario: Partial Attainment of Alternative Standard of 0.070 ppm		
Mortality Study:	Bell et al (2004)	Levy et al. (2005)
Assuming Life Expectancies of the General Population	990 (280 - 1,700)	4,600 (3,100 - 6,100)
Assuming Life Expectancies of the Sub-Population with COPD of Average Severity	790 (230 - 1,400)	3,700 (2,500 - 4,900)
Assuming Life Expectancies of the Sub-Population with Severe COPD	450 (130 - 780)	2,200 (1,500 - 2,900)

*95 percent confidence or credible intervals are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

7b.6.2 Reductions in $PM_{2.5}$-Related Premature Deaths

To generate $PM_{2.5}$-related health outcomes, we used the same framework as for the benefit-cost analysis described in Chapter 8 and briefly summarized above in the introductory portion of Section 8.4.

As in several recent air pollution health impact assessments (e.g., Kunzli et al., 2000; EPA, 2004), we focused on the prospective cohort long-term exposure studies in deriving the health impact function for the estimate of premature mortality. Cohort analyses are better able to capture the full public health impact of exposure to air pollution over time (Kunzli et al., 2001; NRC, 2002). We selected an effect estimate from the extended analysis of the ACS cohort (Pope et al., 2002) as well as from the Harvard Six City Study (Laden et al., 2006). Given the focus in this analysis on developing a broader expression of uncertainties in the benefits estimates, and the weight that was placed on both the ACS and Harvard Six-city studies by experts participating in the $PM_{2.5}$ mortality expert elicitation, we elected to provide estimates derived from both Pope et al. (2002) and Laden et al. (2006).

This latest re-analysis of the ACS cohort data (Pope et al, 2002) provides additional refinements to the analysis of PM-related mortality by (a) extending the follow-up period for the ACS study subjects to 16 years, which triples the size of the mortality data set; (b) substantially increasing exposure data, including consideration for cohort exposure to $PM_{2.5}$ following implementation of $PM_{2.5}$ standard in 1999; (c) controlling for a variety of personal risk factors including occupational exposure and diet; and (d) using advanced statistical methods to evaluate specific issues that can adversely affect risk estimates, including the possibility of spatial autocorrelation of survival times in communities located near each other. The effect estimate from Pope et al. (2002) quantifies the relationship between annual mean $PM_{2.5}$ levels and all-cause mortality in adults 30 and older. We selected the effect estimate estimated using the measure of PM representing average exposure over the follow-up period, calculated as the average of 1979–1984 and 1999–2000 $PM_{2.5}$ levels. The effect estimate from this study is 0.0058, which is equivalent to a relative risk of 1.06 for a 10 μg change in $PM_{2.5}$.

A recent follow up to the Harvard 6-city study (Laden et al., 2006) both confirmed the effect size from the first study and provided additional confirmation that reductions in $PM_{2.5}$ directly result in reductions in the risk of premature death. This additional evidence stems from the observed reductions in $PM_{2.5}$ in each city during the extended follow-up period. Laden et al. (2006) found that mortality rates consistently went down at a rate proportionate to the observed reductions in $PM_{2.5}$. The effect estimate obtained from Laden et al. (2006) is 0.0148, which is equivalent to a relative risk of 1.16 for a 10 $\mu g/m^3$ change in $PM_{2.5}$.

Age, cause, and county-specific mortality rates were obtained from CDC for the years 1996 through 1998. CDC maintains an online data repository of health statistics, CDC Wonder, accessible at http://wonder.cdc.gov/. The mortality rates provided are derived from U.S. death records and U.S. Census Bureau postcensal population estimates. Mortality rates were averaged across 3 years (1996 through 1998) to provide more stable estimates. When estimating rates for age groups that differed from the CDC Wonder groupings, we assumed that rates were uniform across all ages in the reported age group. For example, to estimate mortality rates for individuals ages 30 and up, we scaled the 25- to 34-year old death count and population by one-half and then generated a population-weighted mortality rate using data for the older age groups.

The reductions in incidence of $PM_{2.5}$-related premature mortality within each age group associated with the illustrative 0.07 ppm partial attainment strategy in 2020 are summarized in Table 7b-15.

Table 7b-15: Estimated Reduction in Incidence of $PM_{2.5}$-Related All-Cause Premature Mortality Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020

	*Reduction in All-Cause Premature Mortality (95% CI)**	
Age Interval	Pope (2002)	Laden (2006)
30 – 34	4 (1 – 6)	8 (5 – 12)
35 – 44	11 (4 – 18)	25 (13 – 36)
45 – 54	23 (9 – 36)	51 (28 – 75)
55 – 64	56 (22 – 90)	130 (69 – 180)
65 – 74	93 (37 – 150)	210 (120 – 310)
75 – 84	110 (43 – 180)	250 (130 – 360)
85+	140 (56 – 230)	320 (180 – 470)
Total	440 (170 – 700)	990 (540 – 1,400)

*95% confidence intervals are based on the uncertainty surrounding the effect estimate (coefficient) in the mortality C-R function. All estimates rounded to two significant figures.

7b.6.3 Life Years Saved as a Result of Reductions in $PM_{2.5}$-Related Mortality Risk

To calculate life years saved associated with a given change in air pollution, we used a life table approach coupled with age-specific estimates of reductions in premature mortality. We began with the complete unabridged life table for the United States in 2000, obtained from CDC (CDC, 2002). For each 1-year age interval (e.g., zero to one, one to two) the life table provides estimates of the baseline probability of dying during the interval, person years lived in the interval, and remaining life expectancy. From this unabridged life table, we constructed an abridged life table to match the age intervals for which we have predictions of changes in incidence of premature mortality. We used the abridgement method described in CDC (2002). Table 7b-16 presents the abridged life table for 10-year age intervals for adults over 30 (to match the Pope et al. [2002] study population). Note that the abridgement actually includes one 5-year interval, covering adults 30 to 34, with the remaining age intervals covering 10 years each. This is to provide conformity with the age intervals available for mortality rates.

From the abridged life table (Table 7b-16), we obtained the remaining life expectancy for each age cohort, conditional on surviving to that age. This is then the number of life years lost for an individual in the general population dying during that age interval. This information can then be

combined with the estimated number of premature deaths in each age interval calculated with BenMAP (see previous subsection). Total life years gained will then be the sum of life years gained in each age interval:

$$Total Life Years = \sum_{i=1}^{N} LE_i \times M_i ,$$

where LE_i is the remaining life expectancy for age interval i, M_i is the change in incidence of mortality in age interval i, and N is the number of age intervals.

As noted above, for the purposes of determining cost-effectiveness, it is also necessary to consider the time-dependent nature of the gains in life years. Standard economic theory suggests that benefits occurring in future years should be discounted relative to benefits occurring in the present. OMB and EPA guidance suggest discount rates of three and seven percent. Selection of a 3 percent discount rate is also consistent with recommendations from the U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine (Gold et al., 1996).

Table 7b-16. Abridged Life Table for the Total Population, United States, 2000

Age Interval		*Probability of Dying Between Ages x to x+1*	*Number Surviving to Age x*	*Number Dying Between Ages x to x+1*	*Person Years Lived Between Ages x to x+1*	*Total Number of Person Years Lived Above Age x*	*Expectation of Life at Age x*
Start Age	End Age	q_x	l_x	d_x	L_x	T_x	e_x
30	35	0.00577	97,696	564	487,130	4,723,539	48.3
35	45	0.01979	97,132	1,922	962,882	4,236,409	43.6
45	55	0.04303	95,210	4,097	934,026	3,273,527	34.4
55	65	0.09858	91,113	8,982	872,003	2,339,501	25.7
65	75	0.21779	82,131	17,887	740,927	1,467,498	17.9
75	85	0.45584	64,244	29,285	505,278	726,571	11.3
85	95	0.79256	34,959	27,707	196,269	221,293	6.3
95	100	0.75441	7,252	5,471	20,388	25,024	3.5
100+		1.00000	1,781	1,781	4,636	4,636	2.6

Unlike O_3-related premature deaths, $PM_{2.5}$-related premature deaths are associated with long-term exposures. We therefore did not assume that these deaths all occur in 2020. The $PM_{2.5}$-related premature deaths avoided and associated life years saved are thus further discounted to account for the lag between the reduction in ambient $PM_{2.5}$ and the corresponding reduction in mortality risk. We used the same 20-year segmented lag structure that is used in the benefit-cost analysis (see Chapter 8).

The most complete estimate of the impacts of $PM_{2.5}$ on life years is calculated using the Pope et al. (2002) C-R function relating all-cause mortality in adults 30 and over with ambient $PM_{2.5}$ concentrations averaged over the periods 1979–1983 and 1999–2000. Use of all-cause mortality is appropriate if there are no differences in the life expectancy of individuals dying from air pollution-related causes and those dying from other causes. The argument that long-term exposure to $PM_{2.5}$ may affect mainly individuals with serious preexisting illnesses is not supported by current empirical studies. For example, the Krewski et al. (2000) ACS reanalysis suggests that the mortality risk is no greater for those with preexisting illness at time of enrollment in the study. Life expectancy for the general population in fact includes individuals with serious chronic illness. Mortality rates for the general population then reflect prevalence of chronic disease, and as populations age the prevalence of chronic disease increases.

The only reason one might use a lower life expectancy is if the population at risk from air pollution was limited solely to those with preexisting disease. Also, note that the OMB Circular A-4 notes that "if QALYs are used to evaluate a lifesaving rule aimed at a population that happens to experience a high rate of disability (i.e., where the rule is not designed to affect the disability), the number of life years saved should not necessarily be diminished simply because the rule saves lives of people with life-shortening disabilities. Both analytic simplicity and fairness suggest that the estimate number of life years saved for the disabled population should be based on average life expectancy information for the relevant age cohorts." As such, use of a general population life expectancy is preferred over disability-specific life expectancies. Our primary life years calculations are thus consistent with the concept of not penalizing individuals with disabling chronic health conditions by assessing them reduced benefits of mortality risk reductions. $PM_{2.5}$-Related life years saved associated with the illustrative 0.07 ppm partial attainment strategy in 2020 are given in Table 7b-17.

Table 7b-17. Estimated $PM_{2.5}$-Related Life Years Saved Associated with Illustrative O_3 NAAQS Attainment Strategy in 2020: Changing from Partial Attainment of the Current O_3 NAAQS to Partial Attainment of an Alternative O_3 NAAQS of 0.07 ppm

Estimated $PM_{2.5}$-Related Life Years Saved (95% CI)*		
	Pope et al (2002)	Laden et al. (2006)
Discounted back to 2020, using a 3 percent discount rate:	4,400 (1,700 - 7,000)	9,900 (5,400 - 14,000)
Discounted back to 2020, using a 7 percent discount rate:	3,000 (1,200 - 4,800)	6,700 (3,700 - 9,800)

*95 percent confidence or credible intervals (CIs) are based on the uncertainty about the coefficient in the mortality C-R functions. All estimates rounded to two significant figures.

For this analysis, direct impacts on life expectancy are measured only through the estimated change in mortality risk based on the Pope et al. (2002) C-R function. The SAB-HES has advised against including additional gains in life expectancy due to reductions in incidence of chronic disease or nonfatal heart attacks (EPA-SAB-COUNCIL-ADV-04-002). Although reductions in these endpoints are likely to result in increased life expectancy, the HES has suggested that the cohort design and relatively long follow-up period in the Pope et al. study

should capture any life-prolonging impacts associated with those endpoints. Impacts of CB and nonfatal heart attacks on quality of life will be captured separately in the QALY calculation as years lived with improved quality of life. The methods for calculating this benefit are discussed below.

7b.6.4 Calculating Changes in the Quality of Life Years ($PM_{2.5}$-Related Chronic Morbidity)

In addition to directly measuring the quantity of life gained, measured by life years, it may also be informative to measure gains in the quality of life. The indirect reductions in levels of $PM_{2.5}$ also lead to reductions in serious illnesses that affect quality of life. These include chronic bronchitis (CB) and cardiovascular disease, for which we are able to quantify changes in the incidence of nonfatal heart attacks. To capture these important benefits in the measure of effectiveness, they must first be converted into a life-year equivalent so that they can be combined with the direct gains in life expectancy.

For the cost effectiveness analysis for the PM NAAQS RIA, we developed estimates of the QALYs gained from reductions in the incidence of CB and nonfatal heart attacks associated with reductions in ambient $PM_{2.5}$. In general, QALY calculations require four elements:

1. the estimated change in incidence of the health condition,
2. the duration of the health condition,
3. the quality-of-life weight with the health condition, and
4. the quality-of-life weight without the health condition (i.e., the baseline health state).

The first element is derived using the health impact function approach. The second element is based on the medical literature for each health condition. The third and fourth elements are derived from the medical cost-effectiveness and cost-utility literature. In the following two subsections, we discuss the choices of elements for CB and nonfatal heart attacks.

The preferred source of quality-of-life weights are those based on community preferences, rather than patient or clinician ratings (Gold et al., 1996). Several methods are used to estimate quality-of-life weights. These include rating scale, standard gamble, time trade-off, and person trade-off approaches (Gold, Stevenson, and Fryback, 2002). Only the standard gamble approach is completely consistent with utility theory. However, the time trade-off method has also been widely applied in eliciting community preferences (Gold, Stevenson, and Fryback, 2002).

Quality-of-life weights can be directly elicited for individual specific health states or for a more general set of activity restrictions and health states that can then be used to construct QALY weights for specific conditions (Horsman et al., 2003; Kind, 1996). For this analysis, we used weights based on community-based preferences, using time trade-off or standard gamble when available. In some cases, we used patient or clinician ratings when no community preference-based weights were available. Sources for weights are discussed in more detail below. Table 7b-18 summarizes the key inputs for calculating QALYs associated with chronic health endpoints.

Table 7b-18. Summary of Key Parameters Used in QALY Calculations for Chronic Disease Endpoints

Parameter	*Value(s)*	*Source(s)*
Discount rate	0.03 (0.07 sensitivity analysis)	Gold et al. (1996), U.S. EPA (2000), U.S. OMB (2003)
Quality of life preference score for chronic bronchitis	0.5 – 0.7	Triangular distribution centered at 0.7 with upper bound at 0.9 (Vos, 1999a) (slightly better than a mild/moderate case) and a lower bound at 0.5 (average weight for a severe case based on Vos [1999a] and Smith and Peske [1994])
Duration of acute phase of acute myocardial infarction (AMI)	5.5 days – 22 days	Uniform distribution with lower bound based on average length of stay for an AMI (AHRQ, 2000) and upper bound based on Vos (1999b).
Probability of CHF post AMI	0.2	Vos, 1999a (WHO Burden of Disease Study, based on Cowie et al., 1997)
Probability of angina post AMI	0.51	American Heart Association, 2003 (Calculated as the population with angina divided by the total population with heart disease)
Quality-of-life preference score for post-AMI with CHF (no angina)	0.80 – 0.89	Uniform distribution with lower bound at 0.80 (Stinnett et al., 1996) and upper bound at 0.89 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with CHF and angina	0.76 – 0.85	Uniform distribution with lower bound at 0.76 (Stinnett et al., 1996, adjusted for severity) and upper bound at 0.85 (Kuntz et al., 1996). Both studies used the time trade-off elicitation method.
Quality-of-life preference score for post-AMI with angina (no CHF)	0.7 – 0.89	Uniform distribution with lower bound at 0.7, based on the standard gamble elicitation method (Pliskin, Stason, and Weinstein, 1981) and upper bound at 0.89, based on the time trade-off method (Kuntz et al., 1996).
Quality-of-life preference score for post-AMI (no angina, no CHF)	0.93	Only one value available from the literature. Thus, no distribution is specified. Source of value is Kuntz et al. (1996).

7b.6.4.1 Calculating QALYs Associated with Reductions in the Incidence of Chronic Bronchitis

CB is characterized by mucus in the lungs and a persistent wet cough for at least 3 months a year for several years in a row. CB affects an estimated 5 percent of the U.S. population (American Lung Association, 1999). For gains in quality of life resulting from reduced incidences of PM-induced CB, discounted QALYs are calculated as

$$DISCOUNTED\ QALY GAINED = \sum_i \Delta CB_i \times D_i^* \times \left(w_i - w_i^{CB}\right)$$

where ΔCB_i is the number of incidences of CB avoided in age interval i, w_i is the average QALY weight for the *i*th age interval, w_i^{CB} is the QALY weight associated with CB in the *i*th age

interval, and D_i^* is the discounted duration of life with CB for individuals with onset of disease in the *i*th age interval, equal to $\int_0^{D_i} e^{-rt} dt$, where D_i is the duration of life with CB for individuals with onset of disease the *i*th age interval.

A limited number of studies have estimated the impact of air pollution on new incidences of CB. Schwartz (1993) and Abbey et al. (1995) provide evidence that long-term PM exposure gives rise to the development of CB in the United States. Only the Abbey et al. (1995) study was used, because it is the only study focusing on the relationship between $PM_{2.5}$ and new incidences of CB. The number of cases of CB in each age interval was derived by applying the impact function from Abbey et al. (1995) to the population in each age interval with the appropriate baseline incidence rate.[7] The effect estimate from the Abbey et al. (1995) study is 0.0137, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.15 for a 10 μg change in $PM_{2.5}$. Table 7b-19 presents the estimated reduction in new incidences of CB associated with the 0.070 ppm partial attainment strategy.

CB is assumed to persist for the remainder of an affected individual's lifespan. Duration of CB will thus equal life expectancy conditioned on having CB. CDC has estimated that COPD (of which CB is one element) results in an average loss of life years equal to 4.26 per COPD death, relative to a reference life expectancy of 75 years (CDC, 2003). Thus, we subtracted 4.26 from the remaining life expectancy for each age group, up to age 75. For age groups over 75, we applied the ratio of 4.26 to the life expectancy for the 65 to 74 year group (0.237) to the life expectancy for the 75 to 84 and 85 and up age groups to estimate potential life years lost and then subtracted that value from the base life expectancy.

[7] Prevalence rates for CB were obtained from the 1999 National Health Interview Survey (American Lung Association, 2002). Prevalence rates were available for three age groups: 18–44, 45–64, and 65 and older. Prevalence rates per person for these groups were 0.0367 for 18–44, 0.0505 for 45–64, and 0.0587 for 65 and older. The incidence rate for new cases of CB (0.00378 per person) was taken directly from Abbey et al. (1995).

Table 7b-19. Estimated Reduction in Incidence of Chronic Bronchitis Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020

Age Interval	*Reduction in Incidence (95% Confidence Interval)**
25 – 34	75 (14 – 140)
35 – 44	85 (16 – 150)
45 – 54	80 (15 – 150)
55 – 64	85 (16 – 160)
65 – 74	60 (11 – 110)
75 – 84	30 (6 – 54)
85+	13 (2 – 24)
Total	430 (78 – 770)

*95% confidence intervals are based on the uncertainty surrounding the effect estimate (coefficient) in the CB C-R function. All estimates rounded to two significant figures.

Quality of life with chronic lung diseases has been examined in several studies. In an analysis of the impacts of environmental exposures to contaminants, de Hollander et al. (1999) assigned a weight of 0.69 to years lived with CB. This weight was based on physicians' evaluations of health states similar to CB. Salomon and Murray (2003) estimated a pooled weight of 0.77 based on visual analogue scale, time trade-off, standard gamble, and person trade-off techniques applied to a convenience sample of health professionals. The Harvard Center for Risk Analysis catalog of preference scores reports a weight of 0.40 for severe COPD, with a range from 0.2 to 0.8, based on the judgments of the study's authors (Bell et al., 2001). The Victoria Burden of Disease (BoD) study used a weight of 0.47 for severe COPD and 0.83 for mild to moderate COPD, based on an analysis by Stouthard et al. (1997) of chronic diseases in Dutch populations (Vos, 1999a). Based on the recommendations of Gold et al. (1996), quality-of-life weights based on community preferences are preferred for CEA of interventions affecting broad populations. Use of weights based on health professionals is not recommended. It is not clear from the Victoria BoD study whether the weights used for COPD are based on community preferences or judgments of health professionals. The Harvard catalog score is clearly identified as based on author judgment. Given the lack of a clear preferred weight, we selected a triangular distribution centered at 0.7 with an upper bound at 0.9 (slightly better than a mild/moderate case defined by the Victoria BoD study) and a lower bound at 0.5 based on the Victoria BoD study. We will need additional empirical data on quality of life with chronic respiratory diseases based on community preferences to improve our estimates.

Selection of a reference weight for the general population without CB is somewhat uncertain. It is clear that the general population is not in perfect health; however, there is some uncertainty as to whether individuals' ratings of health states are in reference to a perfect health state or to a generally achievable "normal" health state given age and general health status. The U.S. Public Health Service Panel on Cost Effectiveness in Health and Medicine recommends that "since lives saved or extended by an intervention will not be in perfect health, a saved life year will count as less than 1 full QALY" (Gold et al., 1996). Following Carrothers, Evans, and Graham (2002), we assumed that the reference weight for the general population without CB is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0. Note that the reference weight for the general population is used solely to determine the incremental quality-of-life improvement applied to the duration of life that would have been lived with the chronic disease. For example, if CB has a quality-of-life weight of 0.7 relative to a reference quality-of-life weight of 0.9, then the incremental quality-of-life improvement in 0.2. If the reference quality-of-life weight is 0.95, then the incremental quality-of-life improvement is 0.25. As noted above, the population is assumed to have a reference weight of 1.0 for all life years gained due to mortality risk reductions.

We present discounted QALYs over the duration of the lifespan with CB using a 3 percent discount rate. Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALYs gained per incidence of CB for each age interval.[8] Based on the assumptions defined above, the mean 3 percent discounted QALY gained per incidence of CB for each age interval along with the 95 percent confidence interval resulting from the Monte Carlo simulation is presented in Table 7b-20. Table 7b-20 presents both the undiscounted and discounted QALYs gained per incidence, using a 3 percent discount rate.

[8] Monte Carlo simulation uses random sampling from distributions of parameters to characterize the effects of uncertainty on output variables. For more details, see Gentile (1998).

Table 7b-20. QALYs Gained per Avoided Incidence of CB

Age Interval		*QALYs Gained per Incidence*	
Start Age	End Age	Undiscounted	Discounted (3%)
25	34	12.15 (4.40-19.95)	6.52 (2.36-10.71)
35	44	9.91 (3.54-16.10)	5.94 (2.12-9.66)
45	54	7.49 (2.71-12.34)	5.03 (1.82-8.29)
55	64	5.36 (1.95-8.80)	4.03 (1.47-6.61)
65	74	3.40 (1.22-5.64)	2.84 (1.02-4.71)
75	84	2.15 (0.77-3.49)	1.92 (0.69-3.13)
85+		0.79 (0.27-1.29)	0.77 (0.26-1.25)

7b.6.4.2 Calculating QALYs Associated with Reductions in the Incidence of Nonfatal Myocardial Infarctions

Nonfatal heart attacks, or acute myocardial infarctions, require more complicated calculations to derive estimates of QALY impacts. The actual heart attack, which results when an area of the heart muscle dies or is permanently damaged because of oxygen deprivation, and subsequent emergency care are of relatively short duration. Many heart attacks result in sudden death. However, for survivors, the long-term impacts of advanced coronary heart disease (CHD) are potentially of long duration and can result in significant losses in quality of life and life expectancy.

In this phase of the analysis, we did not independently estimate the gains in life expectancy associated with reductions in nonfatal heart attacks. Based on recommendations from the SAB-HES, we assumed that all gains in life expectancy are captured in the estimates of reduced mortality risk provided by the Pope et al. (2002) analysis. We estimated only the change in quality of life over the period of life affected by the occurrence of a heart attack. This may understate the QALY impacts of nonfatal heart attacks but ensures that the overall QALY impact estimates across endpoints do not double-count potential life-year gains.

Our approach adapts a CHD model developed for the Victoria Burden of Disease study (Vos, 1999b). This model accounts for the lost quality of life during the heart attack and the possible health states following the heart attack. Figure 7b-1 shows the heart attack QALY model in diagrammatic form.

The total gain in QALYs is calculated as:

DISCOUNTED AMI QALY GAINED =

$$\sum_i \Delta AMI_i \times D_i^{*AMI} \times \left(w_i - w_i^{AMI}\right) + \sum_i \sum_{j=1}^{4} \Delta AMI_i \times p_j D_{ij}^{*PostAMI} \times \left(w_i - w_{ij}^{postAMI}\right)$$

where ΔAMI_i is the number of nonfatal acute myocardial infarctions avoided in age interval *i*, w_i^{AMI} is the QALY weight associated with the acute phase of the AMI, p_j is the probability of being in the *j*th post-AMI status, $w_{ij}^{postAMI}$ is the QALY weight associated with post-AMI health status *j*, w_i is the average QALY weight for age interval i, $D_i^{*AMI} = \int_{t=1}^{D_i^{AMI}} e^{-rt} dt$, the discounted value of D_i^{AMI}, the duration of the acute phase of the AMI, and $D_i^{*postAMI} = \int_{t=1}^{D_i^{postAMI}} e^{-rt} dt$, is the discounted value of $D_{ij}^{PostAMI}$, the duration of post-AMI health status *j*.

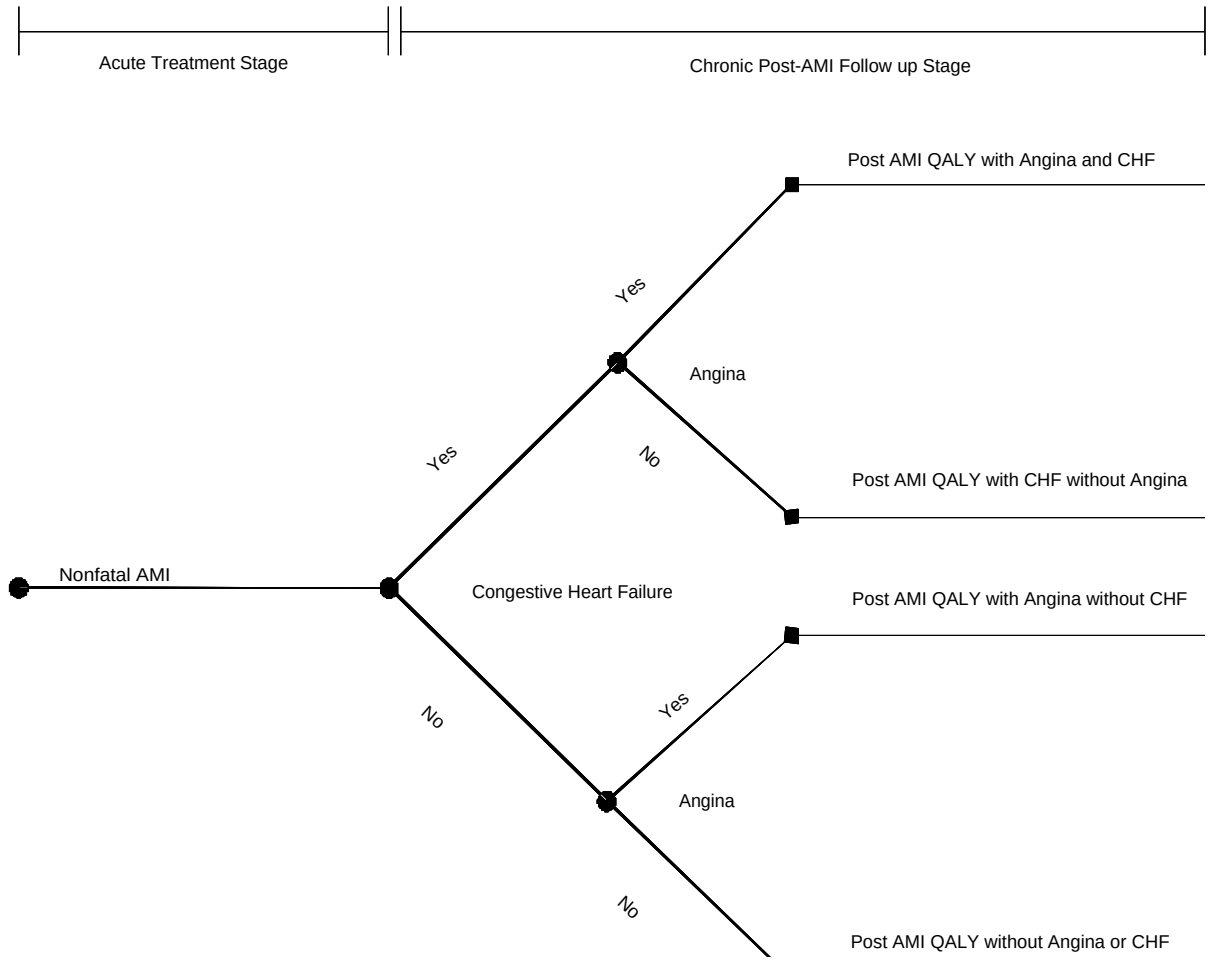

Figure 7b-1. Decision Tree Used in Modeling Gains in QALYs from Reduced Incidence of Nonfatal Acute Myocardial Infarctions

Nonfatal heart attacks have been linked with short-term exposures to $PM_{2.5}$ in the United States (Peters et al., 2001) and other countries (Poloniecki et al., 1997). We used a recent study by Peters et al. (2001) as the basis for the impact function estimating the relationship between $PM_{2.5}$ and nonfatal heart attacks. Peters et al. is the only available U.S. study to provide a specific estimate for heart attacks. Other studies, such as Samet et al. (2000) and Moolgavkar (2000), show a consistent relationship between all cardiovascular hospital admissions, including for nonfatal heart attacks, and PM. Given the lasting impact of a heart attack on longer-term health costs and earnings, we chose to provide a separate estimate for nonfatal heart attacks based on the single available U.S. effect estimate. The finding of a specific impact on heart attacks is consistent with hospital admission and other studies showing relationships between fine particles and cardiovascular effects both within and outside the United States. These studies provide a weight of evidence for this type of effect. Several epidemiologic studies (Liao et al., 1999; Gold et al., 2000; Magari et al., 2001) have shown that heart rate variability (an indicator of how much

the heart is able to speed up or slow down in response to momentary stresses) is negatively related to PM levels. Heart rate variability is a risk factor for heart attacks and other CHDs (Carthenon et al., 2002; Dekker et al., 2000; Liao et al., 1997, Tsuji et al., 1996). As such, significant impacts of PM on heart rate variability are consistent with an increased risk of heart attacks.

The number of avoided nonfatal AMI in each age interval was derived by applying the impact function from Peters et al. (2001) to the population in each age interval with the appropriate baseline incidence rate.[9] The effect estimate from the Peters et al. (2001) study is 0.0241, which, based on the logistic specification of the model, is equivalent to a relative risk of 1.27 for a 10 μg change in $PM_{2.5}$. Table 7b-21 presents the estimated reduction in nonfatal AMI associated with the illustrative Ozone NAAQS attainment strategies.

Table 7b-21. Estimated Reduction in Nonfatal Acute Myocardial Infarctions Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020

Age Interval	*Reduction in Incidence (95% Confidence Interval)**
18 – 24	1 (0 – 1)
25 – 34	5 (2 – 7)
35 – 44	32 (17 – 46)
45 – 54	97 (52 – 140)
55 – 64	240 (130 – 350)
65 – 74	290 (150 – 420)
75 – 84	210 (120 – 310)
85+	130 (71 – 190)
Total	1,000 (540 – 1,500)

*95% confidence intervals are based on the uncertainty surrounding the effect estimate (coefficient) in the AMI C-R function.

[9] Daily nonfatal myocardial infarction incidence rates per person were obtained from the 1999 National Hospital Discharge Survey (assuming all diagnosed nonfatal AMI visit the hospital). Age-specific rates for four regions are used in the analysis. Regional averages for populations 18 and older are 0.0000159 for the Northeast, 0.0000135 for the Midwest, 0.0000111 for the South, and 0.0000100 for the West.

Acute myocardial infarction results in significant loss of quality of life for a relatively short duration. The WHO Global Burden of Disease study, as reported in Vos (1999b), assumes that the acute phase of an acute myocardial infarction lasts for 0.06 years, or around 22 days. An alternative assumption is the acute phase is characterized by the average length of hospital stay for an AMI in the United States, which is 5.5 days, based on data from the Agency for Healthcare Research and Quality's Healthcare Cost and Utilization Project (HCUP).[10] We assumed a distribution of acute phase duration characterized by a uniform distribution between 5.5 and 22 days, noting that due to earlier discharges and in-home therapy available in the United States, duration of reduced quality of life may continue after discharge from the hospital. In the period during and directly following an AMI (the acute phase), we assigned a quality of life weight equal to 0.605, consistent with the weight for the period in treatment during and immediately after an attack (Vos, 1999b).

During the post-AMI period, a number of different health states can determine the loss in quality of life. We chose to classify post-AMI health status into four states defined by the presence or absence of angina and congestive heart failure (CHF). This makes a very explicit assumption that without the occurrence of an AMI, individuals would not experience either angina or CHF. If in fact individuals already have CHF or angina, then the quality of life gained will be overstated. We do not have information about the percentage of the population have been diagnosed with angina or CHF with no occurrence of an AMI. Nor do we have information on what proportion of the heart attacks occurring due to PM exposure are first heart attacks versus repeat attacks. Probabilities for the four post-AMI health states sum to one.

Given the occurrence of a nonfatal AMI, the probability of congestive heart failure is set at 0.2, following the heart disease model developed by Vos (1999b). The probability is based on a study by Cowie et al. (1997), which estimated that 20 percent of those surviving AMI develop heart failure, based on an analysis of the results of the Framingham Heart Study.

The probability of angina is based on the prevalence rate of angina in the U.S. population. Using data from the American Heart Association, we calculated the prevalence rate for angina by dividing the estimated number of people with angina (6.6 million) by the estimated number of people with CHD of all types (12.9 million). We then assumed that the prevalence of angina in the population surviving an AMI is similar to the prevalence of angina in the total population with CHD. The estimated prevalence rate is 51 percent, so the probability of angina is 0.51.

Combining these factors leads to the probabilities for each of the four health states as follows:

I. Post AMI with CHF and angina = 0.102

II. Post AMI with CHF without angina = 0.098

III. Post AMI with angina without CHF = 0.408

IV. Post AMI without angina or CHF = 0.392

[10] Average length of stay estimated from the HCUP data includes all discharges, including those due to death. As such, the 5.5-day average length of stay is likely an underestimate of the average length of stay for AMI admissions where the patient is discharged alive.

Duration of post-AMI health states varies, based in part on assumptions regarding life expectancy with post-AMI complicating health conditions. Based on the model used for established market economies (EME) in the WHO Global Burden of Disease study, as reported in Vos (1999b), we assumed that individuals with CHF have a relatively short remaining life expectancy and thus a relatively short period with reduced quality of life (recall that gains in life expectancy are assumed to be captured by the cohort estimates of reduced mortality risk). Table 7b-22 provides the duration (both discounted and undiscounted) of CHF assumed for post-AMI cases by age interval.

Table 7b-22. Assumed Duration of Congestive Heart Failure

Age Interval		*Duration of Heart Failure (years)*	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	7.11	6.51
25	34	6.98	6.40
35	44	6.49	6.00
45	54	5.31	4.99
55	64	1.96	1.93
65	74	1.71	1.69
75	84	1.52	1.50
85+		1.52	1.50

Duration of health states without CHF is assumed to be equal to the life expectancy of individuals conditional on surviving an AMI. Ganz et al. (2000) note that "Because patients with a history of myocardial infarction have a higher chance of dying of CHD that is unrelated to recurrent myocardial infarction (for example, arrhythmia), this cohort has a higher risk for death from causes other than myocardial infarction or stroke than does an unselected population." They go on to specify a mortality risk ratio of 1.52 for mortality from other causes for the cohort of individuals with a previous (nonfatal) AMI. The risk ratio is relative to all-cause mortality for an age-matched unselected population (i.e., general population). We adopted the same ratios and applied them to each age-specific all-cause mortality rate to derive life expectancies (both discounted and undiscounted) for each age group after an AMI, presented in Table 7b-23. These life expectancies were then used to represent the duration of non-CHF post-AMI health states (III and IV).

Table 7b-23. Assumed Duration of Non-CHF Post-AMI Health States

Age Interval		Post-AMI Years of Life Expectancy (non-CHF)	
Start Age	End Age	Undiscounted	Discounted (3%)
18	24	55.5	27.68
25	34	46.1	25.54
35	44	36.8	22.76
45	54	27.9	19.28
55	64	19.8	15.21
65	74	12.8	10.82
75	84	7.4	6.75
85+		3.6	3.47

For the four post-AMI health states, we used QALY weights based on preferences for the combined conditions characterizing each health state. A number of estimates of QALY weights are available for post-AMI health conditions.

The first two health states are characterized by the presence of CHF, with or without angina. The Harvard Center for Risk Analysis catalog of preference scores provides several specific weights for CHF with and without mild or severe angina and one set specific to post-AMI CHF. Following the Victoria Burden of Disease model, we assumed that most cases of angina will be treated and thus kept at a mild to moderate state. We thus focused our selection on QALY weights for mild to moderate angina. The Harvard database includes two sets of community preference-based scores for CHF (Stinnett et al., 1996; Kuntz et al., 1996). The scores for CHF with angina range from 0.736 to 0.85. The lower of the two scores is based on angina in general with no delineation by severity. Based on the range of the scores for mild to severe cases of angina in the second study, one can infer that an average case of angina has a score around 0.96 of the score for a mild case. Applying this adjustment raises the lower end of the range of preference scores for a mild case of angina to 0.76. We selected a uniform distribution over the range 0.76 to 0.85 for CHF with mild angina, with a midpoint of 0.81. The same two studies in the Harvard catalog also provide weights for CHF without angina. These scores range from 0.801 to 0.89. We selected a uniform distribution over this range, with a midpoint of 0.85.

The third health state is characterized by angina, without the presence of CHF. The Harvard catalog includes five sets of community preference-based scores for angina, one that specifies scores for both mild and severe angina (Kuntz et al., 1996), one that specifies mild angina only (Pliskin, Stason, and Weinstein, 1981), one that specifies severe angina only (Cohen, Breall, and Ho, 1994), and two that specify angina with no severity classification (Salkeld, Phongsavan, and Oldenburg, 1997; Stinnett et al., 1996). With the exception of the Pliskin, Stason, and Weinstein score, all of the angina scores are based on the time trade-off method of elicitation. The Pliskin, Stason, and Weinstein score is based on the standard gamble elicitation method. The scores for the nonspecific severity angina fall within the range of the two scores for mild angina specifically. Thus, we used the range of mild angina scores as the endpoints of a uniform distribution. The range of mild angina scores is from 0.7 to 0.89, with a midpoint of 0.80.

For the fourth health state, characterized by the absence of CHF and/or angina, there is only one relevant community preference score available from the Harvard catalog. This score is 0.93, derived from a time trade-off elicitation (Kuntz et al., 1996). Insufficient information is available to provide a distribution for this weight; therefore, it is treated as a fixed value.

Similar to CB, we assumed that the reference weight for the general population without AMI is 0.95. To allow for uncertainty in this parameter, we assigned a triangular distribution around this weight, bounded by 0.9 and 1.0.

Based on the assumptions defined above, we used Monte Carlo simulation methods as implemented in the Crystal Ball™ software program to develop the distribution of QALYs gained per incidence of nonfatal AMI for each age interval. For the Monte Carlo simulation, all distributions were assumed to be independent. The mean QALYs gained per incidence of nonfatal AMI for each age interval is presented in Table 7b-24, along with the 95 percent confidence interval resulting from the Monte Carlo simulation. Table 7b-24 presents both the undiscounted and discounted QALYs gained per incidence.

Table 7b-24. QALYs Gained per Avoided Nonfatal Myocardial Infarction

Age Interval		QALYs Gained per Incidence[a]	
Start Age	*End Age*	*Undiscounted*	*Discounted (3%)*
18	24	4.18 (1.24-7.09)	2.17 (0.70-3.62)
25	34	3.48 (1.09-5.87)	2.00 (0.68-3.33)
35	44	2.81 (0.88-4.74)	1.79 (0.60-2.99)
45	54	2.14 (0.67-3.61)	1.52 (0.51-2.53)
55	64	1.49 (0.42-2.52)	1.16 (0.34-1.95)
65	74	0.97 (0.30-1.64)	0.83 (0.26-1.39)
75	84	0.59 (0.20-0.97)	0.54 (0.19-0.89)
85+		0.32 (0.13-0.50)	0.31 (0.13-0.49)

[a] Mean of Monte Carlo generated distribution; 95% confidence interval presented in parentheses.

7b.6.5 Aggregating Life Expectancy and Quality-of-Life Gains

Given the estimates of changes in life expectancy and quality of life, the next step is to aggregate life expectancy and quality-of-life gains to form an effectiveness measure that can be compared to costs to develop cost-effectiveness ratios. This section discusses the proper characterization of the combined effectiveness measure for the denominator of the cost-effectiveness ratio.

To develop an integrated measure of changes in health, we simply sum together the gains in life years from reduced mortality risk in each age interval with the gains in QALYs from reductions in incidence of chronic morbidity endpoints (CB and acute myocardial infarctions). The resulting measure of effectiveness then forms the denominator in the cost-effectiveness ratio. This combined measure of effectiveness is not a QALY measure in a strict sense, because we have not adjusted life-expectancy gains for preexisting health status (quality of life). It is however, an effectiveness measure that adds a scaled morbidity equivalent to the standard life years calculation. Thus, we term the aggregate measure morbidity inclusive life years, or MILYs. Alternatively, the combined measure could be considered as QALYs with an assumption that the community preference weight for all life-expectancy gains is 1.0. If one considers that this weight might be considered to be a "fair" treatment of those with preexisting disabilities, the effectiveness measure might be termed "fair QALY" gained. However, this implies that all aspects of fairness have been addressed, and there are clearly other issues with the fairness of QALYs (or other effectiveness measures) that are not addressed in this simple adjustment. The MILY measure violates some of the properties used in deriving QALY weights, such as linear substitution between quality of life and quantity of life. However, in aggregating life expectancy and quality-of-life gains, it merely represents an alternative social weighting that is consistent with the spirit of the recent OMB guidance on CEA. The guidance notes that "fairness is important in the choice and execution of effectiveness measures" (OMB, 2003). The resulting aggregate measure of effectiveness will not be consistent with a strict utility interpretation of QALYs; however, it may still be a useful index of effectiveness.

Applying the life expectancies and distributions of QALYs per incidence for CB and AMI to estimated distributions of incidences yields distributions of life expectancy and QALYs gained under the illustrative attainment strategy with a baseline of partial attainment of the current (0.084 ppm) O_3 NAAQS and a control scenario of partial attainment of an alternative 0.070 ppm O_3 NAAQS. These distributions reflect both the quantified uncertainty in estimates of avoided incidence and the quantified uncertainty in QALYs gained per incidence avoided.

Tables 7b-25 and 7b-26 present the discounted life years, QALYs, and MILYs gained, based on each combination of O_3-mortality study, $PM_{2.5}$-mortality study, and life expectancy assumption for O_3-related life years saved used for the analysis of this attainment strategy, using a 3 percent and 7 percent discount rate, respectively.

Table 7b-25. Estimated Gains in Discounted MILYs, Using a 3 Percent Discount Rate, Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	O3-Related Life Years Gained from Mortality Risk Reductions (95% CI)	PM2.5-Related Life Years Gained from Mortality Risk Reductions (95% CI)	QALYs Gained from Reductions in PM2.5-Related Chronic Bronchitis (95% CI)	QALYs Gained from Reductions in PM2.5-Related Non-Fatal Myocardial Infarction (95% CI)	Total MILYs Gained (95% CI)
Bell et al. (2004)	Pope et al. (2002)	General Population	1,300 (400 - 2,200)	4,400 (1,700 - 7,000)	1,970 (270 - 4,700)	870 (220 - 1,800)	8,500 (4,700 - 12,000)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	1,000 (300 - 1,700)				8,200 (4,500 - 12,000)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	500 (200 - 900)				7,700 (4,100 - 12,000)
Levy et al. (2005)	Pope et al. (2002)	General Population	6,100 (4,100 - 8,100)				13,000 (9,100 - 18,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	4,700 (3,200 - 6,300)				12,000 (7,900 - 16,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	2,700 (1,800 - 3,500)				9,900 (6,200 - 14,000)
Bell et al. (2004)	Laden et al. (2006)	General Population	1,300 (400 - 2,200)	9,900 (5,400 - 14,000)			14,000 (8,500 - 20,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	1,000 (300 - 1,700)				14,000 (8,200 - 19,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	500 (200 - 900)				13,000 (7,800 - 19,000)
Levy et al. (2005)	Laden et al. (2006)	General Population	6,100 (4,100 - 8,100)				19,000 (13,000 - 25,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	4,700 (3,200 - 6,300)				17,000 (12,000 - 23,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	2,700 (1,800 - 3,500)				15,000 (9,900 - 21,000)

*Life years, QALYs, and MILYs are discounted back to 2020. 95% confidence or credible intervals (CIs) around the point estimates are based on the uncertainty surrounding the effect estimates (coefficients) in the C-R functions and, for QALYs and MILYs, the uncertainty surrounding the quality of life weights. All estimates rounded to two significant figures.

Table 7b-26. Estimated Gains in Discounted MILYs, Using a 7 Percent Discount Rate, Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	O3-Related Life Years Gained from Mortality Risk Reductions (95% CI)	PM2.5-Related Life Years Gained from Mortality Risk Reductions (95% CI)	QALYs Gained from Reductions in PM2.5-Related Chronic Bronchitis (95% CI)	QALYs Gained from Reductions in PM2.5-Related Non-Fatal Myocardial Infarction (95% CI)	Total MILYs Gained (95% CI)
Bell et al. (2004)	Pope et al. (2002)	General Population	990 (280 - 1,700)	3,000 (1,200 - 4,800)	1,300 (180 - 3,000)	680 (180 - 1,400)	5,900 (3,300 - 8,700)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	790 (230 - 1,400)				5,700 (3,100 - 8,500)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	450 (130 - 780)				5,400 (2,800 - 8,100)
Levy et al. (2005)	Pope et al. (2002)	General Population	4,600 (3,100 - 6,100)				9,500 (6,600 - 13,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	3,700 (2,500 - 4,900)				8,600 (5,800 - 12,000)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	2,200 (1,500 - 2,900)				7,100 (4,400 - 10,000)
Bell et al. (2004)	Laden et al. (2006)	General Population	990 (280 - 1,700)	6,700 (3,700 - 9,800)			9,700 (5,900 - 13,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	790 (230 - 1,400)				9,500 (5,700 - 13,000)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	450 (130 - 780)				9,200 (5,400 - 13,000)
Levy et al. (2005)	Laden et al. (2006)	General Population	4,600 (3,100 - 6,100)				13,000 (9,400 - 17,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	3,700 (2,500 - 4,900)				12,000 (8,600 - 16,000)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	2,200 (1,500 - 2,900)				11,000 (7,200 - 15,000)

*Life years, QALYs, and MILYs are discounted back to 2020. 95% confidence or credible intervals (CIs) around the point estimates are based on the uncertainty surrounding the effect estimates (coefficients) in the C-R functions and, for QALYs and MILYs, the uncertainty surrounding the quality of life weights. All estimates rounded to two significant figures.

7b.6.6 Estimating the Avoided Costs of Chronic Illness

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. As noted above (see Section 7b.4.1), our estimate of costs in the numerator is net of the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). Among the morbidity costs subtracted from the direct costs of controls in the numerator are the avoided costs of illness (COI) associated with $PM_{2.5}$-related CB and nonfatal AMI.

Avoided costs for CB and nonfatal AMI are based on estimates of lost earnings and medical costs.[11] Using age-specific annual lost earnings and medical costs estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a lifetime present discounted value (in 2006$) due to CB of $185,774 for someone between the ages of 27 and 44; $121,177 for someone between the ages of 45 and 64; and $14,293 for someone over 65. The corresponding age-specific estimates of lifetime present discounted value (in 2006$) using a 7 percent discount rate are $105,974, $89,506, and $11,641, respectively. These estimates assumed that 1) lost earnings continue only until age 65, 2) medical expenditures are incurred until death, and 3) life expectancy is unchanged by CB.

Because the costs associated with a myocardial infarction extend beyond the initial event itself, we consider costs incurred over several years. Using age-specific annual lost earnings estimated by Cropper and Krupnick (1990) and a 3 percent discount rate, we estimated a present discounted value in lost earnings (in 2006$) over 5 years due to a myocardial infarction of $10,758 for someone between the ages of 25 and 44, $15,856 for someone between the ages of 45 and 54, and $91,647 for someone between the ages of 55 and 65. The corresponding age-specific estimates of lost earnings (in 2006$) using a 7 percent discount rate are $9,631, $14,195, and $82,051, respectively. Cropper and Krupnick (1990) do not provide lost earnings estimates for populations under 25 or over 65. Thus, we do not include lost earnings in the cost estimates for these age groups.

Two estimates of the direct medical costs of myocardial infarction are used. The first estimate is from Wittels, Hay, and Gotto (1990), which estimated expected total medical costs of MI over 5 years to be $51,211 (in 1986$) for people who were admitted to the hospital and survived hospitalization (there does not appear to be any discounting used). Using the CPI-U for medical care, the Wittels estimate is $141,124 in year 2006$. This estimated cost is based on a medical cost model, which incorporated therapeutic options, projected outcomes, and prices (using "knowledgeable cardiologists" as consultants). The model used medical data and medical

[11] Gold et al. (1996) recommend not including lost earnings in the cost-of-illness estimates, suggesting that in some cases, they may be already be counted in the effectiveness measures. However, this requires that individuals fully incorporate the value of lost earnings and reduced labor force participation opportunities into their responses to time-tradeoff or standard-gamble questions. For the purposes of this analysis and for consistency with the way costs-of-illness are calculated for the benefit-cost analysis, we have assumed that individuals do not incorporate lost earnings in responses to these questions. This assumption can be relaxed in future analyses with improved understanding of how lost earnings are treated in preference elicitations.

decision algorithms to estimate the probabilities of certain events and/or medical procedures being used. The second estimate is from Russell et al. (1998), which estimated first-year direct medical costs of treating nonfatal myocardial infarction of $15,540 (in 1995$), and $1,051 annually thereafter. Converting to year 2006$, that would be $28,787 for a 5-year period (without discounting).

The two estimates from these studies are substantially different, and we have not adequately resolved the sources of differences in the estimates. Because the wage-related opportunity cost estimates from Cropper and Krupnick (1990) cover a 5-year period, we used estimates for medical costs that similarly cover a 5-year period. We used a simple average of the two 5-year estimates, or $84,956, and add it to the 5-year opportunity cost estimate. The resulting estimates are given in Table 7b-27.

Table 7b-27. Estimated Costs Over a 5-Year Period (in 2006$) of a Nonfatal Myocardial Infarction

Age Group	Opportunity Cost	Medical Cost[a]	Total Cost
0 – 24	$0	$84,956	$84,956
25-44	$10,757b	$84,956	$95,714
45 – 54	$15,856[b]	$84,956	$100,812
55 – 65	$91,647[b]	$84,956	$176,603
>65	$0	$84,956	$84,956

[a] An average of the 5-year costs estimated by Wittels, Hay, and Gotto (1990) and Russell et al. (1998).

[b] From Cropper and Krupnick (1990), using a 3 percent discount rate.

The total avoided COI by age group associated with the reductions in CB and nonfatal acute myocardial infarctions (using a 3 percent discount rate) is provided in Table 7b-28. The total avoided COI associated with this illustrative attainment strategy (using a 3 percent discount rate) is about $172 million. Note that these estimates do not include any direct avoided medical costs associated with premature mortality. Nor do they include any medical costs that occur more than 5 years from the onset of a nonfatal AMI. Therefore, they are likely underestimates of the true avoided COI associated with this illustrative attainment strategy.

Table 7b-28. Avoided Costs of Illness Associated with Reductions in Chronic Bronchitis and Nonfatal Acute Myocardial Infarctions Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020

	Avoided Cost of Illness (in millions of 2006$)	
Age Range	*Chronic Bronchitis*	*Nonfatal Acute Myocardial Infarction*
18-24	—	$0.07
25-34	$17	$0.4
35-44	$19	$3
45-54	$12	$9.8
55-64	$13	$42
65-74	$1.1	$24
75-84	$0.5	$18
85+	$0.2	$11
Total	$63	$110

7b.6.7 Cost-Effectiveness Ratios

Construction of cost-effectiveness ratios requires estimates of effectiveness (in this case measured by lives saved, life years gained, or MILYs gained) in the denominator and estimates of costs in the numerator. As noted above (see Section 7b.4.1), the estimate of costs in the numerator should include both the direct costs of the controls necessary to achieve the reduction in ambient O_3 (and, indirectly, $PM_{2.5}$) and the avoided costs (cost savings) associated with the reductions in morbidity (Gold et al., 1996). In general, because reductions in air pollution do not require direct actions by the affected populations, there are no specific costs to affected individuals (aside from the overall increases in prices that might be expected to occur as control costs are passed on by affected industries). Likewise, because individuals do not engage in any specific actions to realize the health benefit of the pollution reduction, there are no decreases in utility (as might occur from a medical intervention) that need to be adjusted for in the denominator. Thus, the elements of the numerator are direct costs of controls minus the avoided COI associated with CB and nonfatal AMI. In addition, to account for the value of reductions in O_3- and $PM_{2.5}$-related acute health impacts and non-health benefits, we netted out the monetized value of these benefits from the numerator to yield a "net cost" estimate. For the MILY aggregate effectiveness measure, the denominator is simply the sum of (O_3- and $PM_{2.5}$-related) life years gained from increased life expectancy and QALYs gained from the ($PM_{2.5}$-related) reductions in CB and nonfatal AMI. The separate O_3- and $PM_{2.5}$-related inputs to the denominators of the cost-effectiveness ratios are summarized above in Tables 7b-25 through 7b-26. The cost-effectiveness ratios and 95 percent confidence (credible) intervals resulting from all of the sources of uncertainty considered, using Monte Carlo procedures as implemented in the Crystal Ball™ software program and incorporating both the O_3- and $PM_{2.5}$-related benefits are shown in the tables below. Tables 7b-29 and 7b-30 show cost per life saved, using a 3 percent

and 7 percent discount rate, respectively. Tables 7b-31 and 7b-32 show cost per life year saved at the two discount rates; and Tables 7b-33 and 7b-34 show cost per MILY gained.

Table 7b-29. Estimated Net Cost (2006$) per O_3- and $PM_{2.5}$-Related Life Saved Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Cost Effectiveness Ratio: Net Cost (in Million $) per Life Saved* (95% CI)**
Bell et al. (2004)	Pope et al. (2002)	$4.5 ($2.7 - $8.7)
Bell et al. (2004)	Laden et al. (2006)	$2.3 ($1.5 - $3.8)
Levy et al. (2005)	Pope et al. (2002)	$2.3 ($1.7 - $3.4)
Levy et al. (2005)	Laden et al. (2006)	$1.5 ($1.1 - $2.2)

*The 3 percent discounted cost of the regulation is estimated to be $2.6 billion. $PM_{2.5}$-related avoided deaths are discounted back to 2020. O_3-related deaths are assumed to occur in 2020.

**95 percent confidence or credible intervals incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 7b-30. Estimated Net Cost (2006$) per O_3- and $PM_{2.5}$-Related Life Saved Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Cost Effectiveness Ratio: Net Cost (in Million $) per Life Saved* (95% CI)**
Bell et al. (2004)	Pope et al. (2002)	$5.4 ($3.2 - $9.9)
Bell et al. (2004)	Laden et al. (2006)	$2.7 ($1.8 - $4.5)
Levy et al. (2005)	Pope et al. (2002)	$2.6 ($1.9 - $3.8)
Levy et al. (2005)	Laden et al. (2006)	$1.8 ($1.3 - $2.6)

*The 7 percent discounted cost of the regulation is estimated to be $2.8 billion. $PM_{2.5}$-related avoided deaths are discounted back to 2020. O_3-related deaths are assumed to occur in 2020.

**95 percent confidence or credible intervals incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 7b-31. Estimated Net Cost (2006$) per O_3- and $PM_{2.5}$-Related Life Year Saved Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Million $) per Life Year Saved* (95% CI)**
Bell et al. (2004)	Pope et al. (2002)	General Population	$0.42 ($0.25 - $0.81)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$0.45 ($0.26 - $0.89)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.50 ($0.28 - $1)
Levy et al. (2005)	Pope et al. (2002)	General Population	$0.22 ($0.16 - $0.32)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$0.25 ($0.18 - $0.38)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.33 ($0.22 - $0.54)
Bell et al. (2004)	Laden et al. (2006)	General Population	$0.21 ($0.13 - $0.35)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$0.21 ($0.14 - $0.36)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.22 ($0.14 - $0.38)
Levy et al. (2005)	Laden et al. (2006)	General Population	$0.14 ($0.1 - $0.2)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$0.16 ($0.11 - $0.23)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.18 ($0.12 - $0.29)

*The 3 percent discounted cost of the regulation is estimated to be $2.6 billion. All life years are discounted back to the year of death. $PM_{2.5}$-related avoided deaths are discounted back to 2020. O_3-related deaths are assumed to occur in 2020.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 7b-32. Estimated Net Cost (2006$) per O_3- and $PM_{2.5}$-Related Life Year Saved Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Million $) per Life Year Saved* (95% CI)**
Bell et al. (2004)	Pope et al. (2002)	General Population	$0.67 ($0.39 - $1.2)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$0.71 ($0.41 - $1.4)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.79 ($0.44 - $1.6)
Levy et al. (2005)	Pope et al. (2002)	General Population	$0.33 ($0.24 - $0.47)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$0.37 ($0.26 - $0.55)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.49 ($0.33 - $0.78)
Bell et al. (2004)	Laden et al. (2006)	General Population	$0.33 ($0.21 - $0.55)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$0.34 ($0.22 - $0.56)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.35 ($0.23 - $0.6)
Levy et al. (2005)	Laden et al. (2006)	General Population	$0.22 ($0.16 - $0.31)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$0.24 ($0.17 - $0.34)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.28 ($0.19 - $0.42)

*The 7 percent discounted cost of the regulation is estimated to be $2.8 billion. All life years are discounted back to the year of death. $PM_{2.5}$-related avoided deaths are discounted back to 2020. O_3-related deaths are assumed to occur in 2020.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 7b-33. Estimated Net Cost (2006$) per O_3- and $PM_{2.5}$-Related MILY Gained Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020, Using a 3 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Million $) per MILY Gained* (95% CI)**
Bell et al. (2004)	Pope et al. (2002)	General Population	$0.27 ($0.17 - $0.46)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$0.28 ($0.18 - $0.49)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.30 ($0.19 - $0.53)
Levy et al. (2005)	Pope et al. (2002)	General Population	$0.17 ($0.12 - $0.24)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$0.19 ($0.14 - $0.28)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.23 ($0.16 - $0.35)
Bell et al. (2004)	Laden et al. (2006)	General Population	$0.16 ($0.11 - $0.26)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$0.17 ($0.11 - $0.27)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.17 ($0.12 - $0.28)
Levy et al. (2005)	Laden et al. (2006)	General Population	$0.12 ($0.09 - $0.17)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$0.13 ($0.09 - $0.18)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.15 ($0.1 - $0.22)

*The 3 percent discounted cost of the regulation is estimated to be $2.6 billion. All life years are discounted back to the year of death. $PM_{2.5}$-related avoided deaths are discounted back to 2020. All QALYs are discounted back to 2020. O_3-related deaths are assumed to occur in 2020.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

Table 7b-34. Estimated Net Cost (2006$) per O_3- and $PM_{2.5}$-Related MILY Gained Under an Illustrative Strategy of Changing from Partial Attainment of the Current (0.084 ppm) O_3 NAAQS to Partial Attainment of an Alternative 0.070 ppm O_3 NAAQS in 2020, Using a 7 Percent Discount Rate

O_3 Mortality Study	$PM_{2.5}$ Mortality Study	Life Expectancy Assumption for O_3-Related Mortality	Cost Effectiveness Ratio: Net Cost (in Million $) per MILY Gained* (95% CI)**
Bell et al. (2004)	Pope et al. (2002)	General Population	$0.43 ($0.27 - $0.73)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Average COPD	$0.45 ($0.28 - $0.77)
Bell et al. (2004)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.48 ($0.29 - $0.86)
Levy et al. (2005)	Pope et al. (2002)	General Population	$0.26 ($0.19 - $0.37)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Average COPD	$0.29 ($0.2 - $0.41)
Levy et al. (2005)	Pope et al. (2002)	Subpopulation with Severe COPD	$0.35 ($0.24 - $0.54)
Bell et al. (2004)	Laden et al. (2006)	General Population	$0.26 ($0.17 - $0.41)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Average COPD	$0.26 ($0.18 - $0.42)
Bell et al. (2004)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.27 ($0.18 - $0.44)
Levy et al. (2005)	Laden et al. (2006)	General Population	$0.18 ($0.14 - $0.26)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Average COPD	$0.20 ($0.14 - $0.28)
Levy et al. (2005)	Laden et al. (2006)	Subpopulation with Severe COPD	$0.23 ($0.16 - $0.33)

*The 7 percent discounted cost of the regulation is estimated to be $2.8 billion. All life years are discounted back to the year of death. $PM_{2.5}$-related avoided deaths are discounted back to 2020. All QALYs are discounted back to 2020. O_3-related death are assumed to occur in 2020.

**95 percent confidence or credible intervals (CIs) incorporate uncertainty surrounding the O_3 and $PM_{2.5}$ coefficients in the mortality and morbidity C-R functions as well as the uncertainty surrounding unit values of morbidity endpoints. All estimates rounded to two significant figures.

7b.7 Conclusions

We estimated the effectiveness of several illustrative O_3 NAAQS attainment strategies based on reductions in premature deaths and, in the case of the one strategy for which we were able to estimate both direct O_3-related benefits and indirect $PM_{2.5}$-related co-benefits, incidence of chronic disease. We measured effectiveness using several different metrics, including lives saved, life years saved, and QALYs gained (for improvements in quality of life due to reductions in incidence of chronic disease). We suggested a new metric for aggregating life years saved and improvements in quality of life, morbidity inclusive life years (MILY) which assumes that society assigns a weight of one to years of life extended regardless of preexisting disabilities or chronic health conditions. As noted above, however, the cost effectiveness metrics presented for all but one of the illustrative O_3 NAAQS attainment strategies omit the $PM_{2.5}$-related co-benefits and are therefore likely to understate the cost effectiveness of those strategies

CEA of environmental regulations that have substantial public health impacts may be informative in identifying programs that have achieved cost-effective reductions in health impacts and can suggest areas where additional controls may be justified. However, the overall efficiency of a regulatory action can only be judged through a complete benefit-cost analysis that takes into account all benefits and costs, including both health and non-health effects. The benefit-cost analysis for the O_3 NAAQS attainment strategies, provided in Chapter 9, shows that the attainment strategies we modeled have potentially large net benefits, indicating that implementation of the revised O_3 NAAQS will likely result in improvements in overall public welfare.

7b.8 References

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