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# Draft Regulatory Impact Analysis: Control of Hazardous Air Pollutants from Mobile Sources

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Assessment and Standards Division
Office of Transportation and Air Quality
U.S. Environmental Protection Agency

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# Chapter 12: Cost-Benefit Analysis

## 12.1 Overview

This chapter reports EPA's analysis of a subset of the public health and welfare impacts and associated monetized benefits to society associated with the proposed standards. In terms of emission benefits, we expect to see significant reductions in mobile source air toxics (MSATs) from the proposed vehicle, fuel and gas can standards, reductions in VOCs (an ozone precursor) from the proposed cold temperature vehicle and gas can standards, and reductions in direct $PM_{2.5}$ from the proposed cold temperature vehicle standards. When translating emission benefits to health effects and monetized values, however, we only quantify the PM-related benefits associated with the proposed cold temperature vehicle standards.

We demonstrate that the proposed standards would reduce cancer and noncancer risk from reduced exposure to MSATs (as described in Chapter 3). However, we do not translate this risk reduction into benefits. We also do not quantify the benefits related to ambient reductions in ozone due to the VOC emission reductions expected to occur as a result of the proposed standards. We describe in more detail below why these benefits are not quantified.

EPA is required by Executive Order (E.O.) 12866 to estimate the benefits and costs of major new pollution control regulations. Accordingly, the analysis presented here attempts to answer three questions: (1) what are the physical health and welfare effects of changes in ambient particulate matter (PM) resulting from direct PM emission reductions related to the proposed cold temperature standards? (2) what is the monetary value of the changes in effects attributable to the proposed rule? and (3) how do the monetized benefits compare to the costs? It constitutes one part of EPA's thorough examination of the relative merits of this regulation.

The analysis presented in this chapter uses a methodology generally consistent with benefits analyses performed for the recent analysis of the Clean Air Interstate Rule (CAIR) standards and the Clean Air Nonroad Diesel Rule (CAND).[1,2] For this reason, the current chapter avoids repeating this information and refers to the appropriate sections of each RIA. The benefits analysis relies on two major components:

1) Calculation of the impact of the proposed cold temperature vehicle standards on the national direct PM emissions inventory for two future years (2020 and 2030).[A]
2) A benefits analysis to determine the changes in human health, both in terms of physical effects and monetary value, based on a PM benefits transfer approach that scales CAND results (see Section 12.2.).

A wide range of human health and welfare effects are linked to the emissions of direct PM and its resulting impact on ambient concentrations of $PM_{2.5}$. Potential human health effects

[A] We consider two future years for analysis (2020 and 2030). Gas can, vehicle, and fuels controls will be fully implemented by 2020. However, for vehicles, the in-use fleet will not be fully turned over to vehicles meeting the new standards by 2020. Therefore, we have analyzed 2030 to represent a more fully turned over fleet.

associated with $PM_{2.5}$ range from premature mortality to morbidity effects linked to long-term (chronic) and shorter-term (acute) exposures (e.g., respiratory and cardiovascular symptoms resulting in hospital admissions, asthma exacerbations, and acute and chronic bronchitis [CB]). Welfare effects potentially linked to PM include materials damage and visibility impacts.

Other standards we are currently proposing, such as the cold temperature vehicle and gas can standards, would also reduce the national emissions inventory of precursors to ozone, such as VOCs. Exposure to ozone has been linked to a variety of respiratory effects including hospital admissions and illnesses resulting in school absences. In addition, recent analyses suggest ozone may have an effect on daily premature mortality rates independent of exposure to PM. Ozone can also adversely affect the agricultural and forestry sectors by decreasing yields of crops and forests. Although ozone benefits are typically quantified in regulatory impact analyses, we do not evaluate them for this analysis.

We estimate that there will be demonstrable VOC reductions as a result of the cold temperature vehicle standards. However, we assume that these emissions would not have a measurable impact on ozone formation since the standards seek to reduce VOC emissions at cold ambient temperatures and ozone formation is primarily a warm ambient temperature issue. There would, however, likely be benefits associated with VOC emission reductions associated with the proposed gas can standards. In Chapter 3, we discuss that the ozone modeling conducted for the proposed gas can standards results in a net reduction in the average population weighted ozone design value metric measured within the modeled domain (37 Eastern states and the District of Columbia). The net improvement is very small, however, and would likely lead to negligible monetized benefits. We therefore do not estimate ozone benefits for the gas can standards due to the magnitude of this change and the uncertainty present in the modeling. Instead, we acknowledge that this analysis may underestimate the benefits associated with reductions in ozone precursor emissions achieved by the various proposed standards and will discuss them qualitatively within this chapter.

Table 12.1-1 summarizes the annual monetized health and welfare benefits associated with the proposed cold temperature standards for two years, 2020 and 2030, assuming a background PM threshold of 3 $\mu g/m^3$ in the calculation of PM mortality. EPA's consistent approach has been to model premature mortality associated with PM exposure as a nonthreshold effect; that is, with harmful effects to exposed populations modeled regardless of the absolute level of ambient PM concentrations (down to background). This approach has been supported by advice from EPA's technical peer review panel, the Science Advisory Board's Health Effects Subcommittee (SAB-HES). However, EPA's most recent $PM_{2.5}$ Criteria Document concludes that "the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies." We consider the impact of a threshold in the PM-mortality concentration response function in Section 12.6.1.1 of the RIA. Table 12.1-2 lists the full complement of human health and welfare effects associated with PM, ozone and air toxics, and identifies those effects that are quantified for the primary estimate and those that remain unquantified because of current limitations in methods or available data.

**Table 12.1-1. Estimated Monetized PM-Related Health Benefits of the Proposed Mobile Source Air Toxics Standards: Cold Temperature Controls**

| | Total Benefits[a, b, c] (billions 2003$) | |
|---|---|---|
| | 2020 | 2030 |
| Using a 3% discount rate | $3.4 + B | $6.5 + B |
| Using a 7% discount rate | $3.1 + B | $5.9 + B |

[a] Benefits include avoided cases of mortality, chronic illness, and other morbidity health endpoints. PM-related mortality benefits estimated using an assumed PM threshold at background levels (3 $\mu g/m^3$). There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate. For a more detailed discussion of this issue, please refer to Section 12.6.1.1 of the RIA.

[b] For notational purposes, unquantified benefits are indicated with a "B" to represent the sum of additional monetary benefits and disbenefits. A detailed listing of unquantified health and welfare effects is provided in Table 13-2 of the RIA.

[c] Results reflect the use of two different discount rates: 3 and 7 percent, which are recommended by EPA's *Guidelines for Preparing Economic Analyses*[3] and OMB Circular A-4. Results are rounded to three significant digits for ease of presentation and computation.

This chapter specifically assesses the PM-related benefits of the proposed cold temperature vehicle standards. However, we note that there would be significant reduction in emissions of air toxics (including benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, naphthalene, and other air toxic pollutants) with the proposed standards in place. While there will be substantial benefits associated with air toxic pollutant reductions, notably with regard to reductions in exposure and risk (see Chapter 3), we do not attempt to extrapolate this risk reduction to monetize those benefits. This is primarily because available tools and methods to assess air toxics risk from mobile sources at the national scale are not adequate for extrapolation to benefits assessment.

The best suite of tools and methods currently available for assessment at the national scale are those used in the National Scale Air Toxics Assessment (NATA; these tools are discussed in Chapter 3). The EPA Science Advisory Board specifically commented in their review of the 1996 National Air Toxics Assessment (NATA) that these tools were not yet ready for use in a national-scale benefits analysis, because they did not consider the full distribution of exposure and risk, or address sub-chronic health effects.[4] While EPA has since improved the tools, there remain critical limitations for estimating incidence and assessing monetized benefits of reducing mobile source air toxics.

In addition to inherent limitations in the tools for national-scale modeling of air quality and exposure, there is a lack of epidemiology data for air toxics in the general population. Therefore, we must rely on health endpoints estimated from occupational or animal exposure studies. For benzene, the cancer unit risk estimate is based on only one endpoint, acute nonlymphocytic leukemia; however, as discussed in Chapter 1, there is a causal relationship between benzene and other leukemias. There are additional limitations in our ability to quantify and value changes in incidence of health effects. For the MSATs of greatest concern, we are

currently unable to estimate cessation lag, which is the time between reduction in exposure and decline in risk to "steady state level." We have not resolved the analytical challenges associated with quantifying partial lifetime probabilities of cancer for different age groups or estimating changes in survival rates over time. In addition, we are currently unable to estimate the premium people are willing to pay to avoid cancer. There is also no data on the cost of treating leukemia cases and little data on how to valuate non-fatal leukemias. Given all the limitations in our ability to develop incidence estimates and to monetize willingness to pay or treatment costs, a quantitative benefits analysis for benzene would not be meaningful or informative. We continue to work to address these limitations, and we are exploring the feasibility of a quantitative benefits assessment for air toxics as part of a case study being done for benzene as part of the ongoing update to the Section 812 retrospective and prospective studies.[B]

**Table 12.1-2. Human Health and Welfare Effects of Pollutants Affected by the Proposed Standards**

| Pollutant/Effect | Quantified and Monetized in Base Estimates[a] | Unquantified Effects - Changes in: |
|---|---|---|
| PM/Health[b] | Premature mortality based on cohort study estimates[c]<br>Bronchitis: chronic and acute<br>Hospital admissions: respiratory and cardiovascular<br>Emergency room visits for asthma<br>Nonfatal heart attacks (myocardial infarction)<br>Lower and upper respiratory illness<br>Minor restricted-activity days<br>Work loss days<br>Asthma exacerbations (asthmatic population)<br>Respiratory symptoms (asthmatic population)<br>Infant mortality | Premature mortality: short term exposures[d]<br>Subchronic bronchitis cases<br>Low birth weight<br>Pulmonary function<br>Chronic respiratory diseases other than chronic bronchitis<br>Nonasthma respiratory emergency room visits<br>UVb exposure (+/-)[e] |
| PM/Welfare | | Visibility in Southeastern Class I areas<br>Visibility in northeastern and Midwestern Class I areas<br>Household soiling<br>Visibility in western U.S. Class I areas<br>Visibility in residential and non-Class I areas<br>UVb exposure (+/-)[e] |

[B] The analytic blueprint for the Section 812 benzene case study can be found at http://www.epa.gov/air/sect812/appendixi51203.pdf.

| Pollutant/Effect | Quantified and Monetized in Base Estimates[a] | Unquantified Effects - Changes in: |
|---|---|---|
| Ozone/Health[f] | | Premature mortality: short term exposures[g]<br>Hospital admissions: respiratory<br>Emergency room visits for asthma<br>Minor restricted-activity days<br>School loss days<br>Asthma attacks<br>Cardiovascular emergency room visits<br>Acute respiratory symptoms<br>Chronic respiratory damage<br>Premature aging of the lungs<br>Nonasthma respiratory emergency room visits<br>UVb exposure (+/-)[e] |
| Ozone/Welfare | | Decreased outdoor worker productivity<br>Yields for:<br>- Commercial forests<br>- Fruits and vegetables, and<br>- Other commercial and noncommercial crops<br>Damage to urban ornamental plants<br>Recreational demand from damaged forest aesthetics<br>Ecosystem functions<br>UVb exposure (+/-)[e] |
| MSAT Health | | Cancer (benzene, 1,3-butadiene, formaldehyde, acetaldehyde, naphthalene)<br>Anemia (benzene)<br>Disruption of production of blood components (benzene)<br>Reduction in the number of blood platelets (benzene)<br>Excessive bone marrow formation (benzene)<br>Depression of lymphocyte counts (benzene)<br>Reproductive and developmental effects (1,3-butadiene)<br>Irritation of eyes and mucus membranes (formaldehyde)<br>Respiratory irritation (formaldehyde)<br>Asthma attacks in asthmatics (formaldehyde)<br>Asthma-like symptoms in non-asthmatics (formaldehyde)<br>Irritation of the eyes, skin, and respiratory tract (acetaldehyde)<br>Upper respiratory tract irritation and congestion (acrolein) |
| MSAT Welfare | | Direct toxic effects to animals<br>Bioaccumulation in the food chain<br>Damage to ecosystem function<br>Odor |

[a] Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of the proposed standards.

[b] In addition to primary economic endpoints, there are a number of biological responses that have been associated with PM health effects including morphological changes and altered host defense mechanisms. The public health impact of these biological responses may be partly represented by our quantified endpoints.

[c] Cohort estimates are designed to examine the effects of long term exposures to ambient pollution, but relative risk estimates may also incorporate some effects due to shorter term exposures (see Kunzli, 2001 for a discussion of this issue).[5]

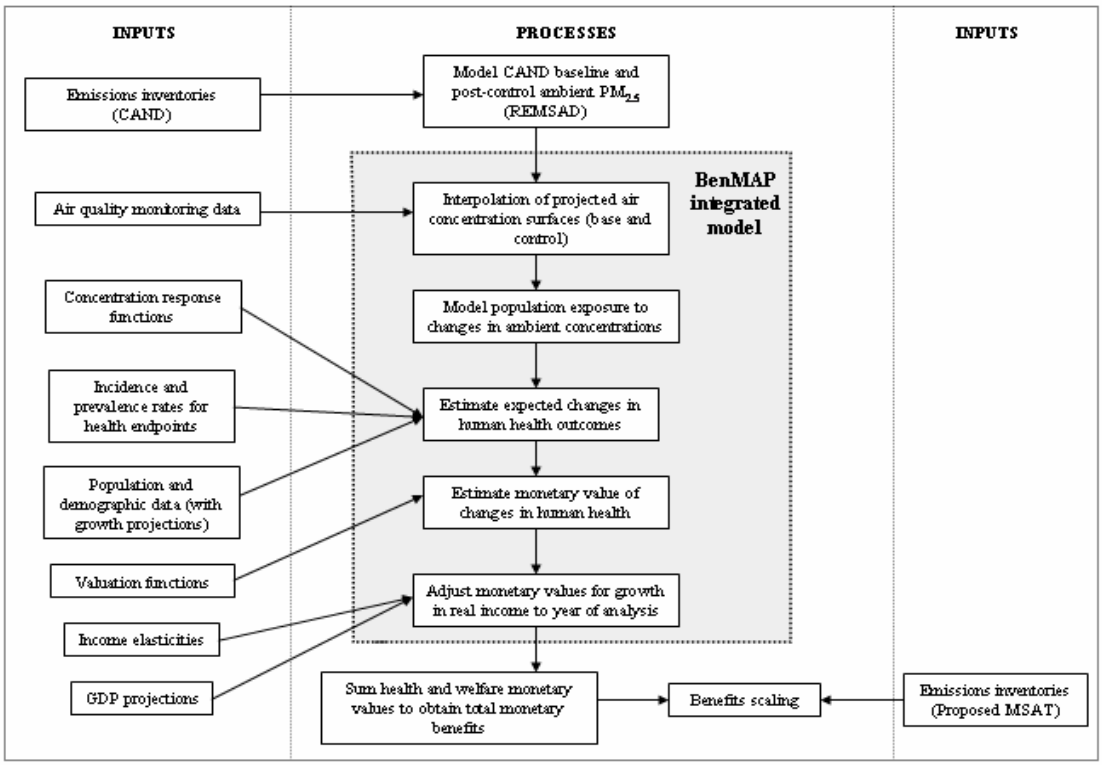
[d] While some of the effects of short term exposure are likely to be captured by the cohort estimates, there may be additional premature mortality from short term PM exposure not captured in the cohort estimates included in the primary analysis.
[e] May result in benefits or disbenefits. See Section 12.5.3. for more details.
[f] In addition to primary economic endpoints, there are a number of biological responses that have been associated with ozone health including increased airway responsiveness to stimuli, inflammation in the lung, acute inflammation and respiratory cell damage, and increased susceptibility to respiratory infection. The public health impact of these biological responses may be partly represented by our quantified endpoints.
[g] EPA sponsored a series of meta-analyses of the ozone mortality epidemiology literature, published in the July 2005 volume of the journal Epidemiology, which found that short-term exposures to ozone may have a significant effect on daily mortality rates, independent of exposure to PM. EPA is currently considering how to include an estimate of ozone mortality in its primary benefits analyses.

Figure 12.1-1 illustrates the major steps in this PM benefits analysis. Given the change in direct PM emissions modeled for the proposed cold temperature vehicle standards, we use a benefits transfer approach to scale PM benefits estimated for the CAND analysis (see Section 12.2 for a description of the scaling approach). For the CAND analysis, EPA ran a sophisticated photochemical air quality model to estimate baseline and post-control ambient concentrations of PM for each future year (2020 and 2030). The estimated changes in ambient concentrations were then combined with population projections to estimate population-level potential exposures to changes in ambient concentrations for use in estimating health effects. Changes in population exposure to ambient air pollution were then input to impact functions[C] to generate changes in the incidence of health effects. The resulting effects changes were then assigned monetary values, taking into account adjustments to values for growth in real income out to the year of analysis (values for health and welfare effects are in general positively related to real income levels). Values for individual health and welfare effects were summed to obtain an estimate of the total monetary value of the changes in emissions. Finally, we scale the CAND results to reflect the magnitude of the direct PM emissions changes we estimate would occur as a result of the proposed cold temperature standards.

Benefits estimates calculated for the CAND analysis, and scaled for the proposed standards, were generated using the Environmental Benefits Mapping and Analysis Program (BenMAP). BenMAP is a computer program developed by EPA that integrates a number of the modeling elements used in previous RIA's (e.g., interpolation functions, population projections, health impact functions, valuation functions, analysis and pooling methods) to translate modeled air concentration estimates into health effect incidence estimates and monetized benefit estimates. Interested parties may wish to consult the webpage http://www.epa.gov/ttn/ecas/benmodels.html for more information.

[C] The term "impact function" as used here refers to the combination of a) an effect estimate obtained from the epidemiological literature, b) the baseline incidence estimate for the health effect of interest in the modeled population, c) the size of that modeled population, and d) the change in the ambient air pollution metric of interest. These elements are combined in the impact function to generate estimates of changes in incidence of the health effect. The impact function is distinct from the C-R function, which strictly refers to the estimated equation from the epidemiological study relating incidence of the health effect and ambient pollution. We refer to the specific value of the relative risk or estimated coefficients in the epidemiological study as the "effect estimate." In referencing the functions used to generate changes in incidence of health effects for this RIA, we use the term "impact function" rather than C-R function because "impact function" includes all key input parameters used in the incidence calculation.

**Figure 12.1-1. Key Steps in Air Quality Modeling Based Benefits Analysis**




All of the benefit estimates for the proposed control options in this analysis are based on an analytical structure and sequence similar to that used in the benefits analyses for the CAND final rule, the CAIR rule, and in the "section 812 studies."[D] By adopting the major design elements, models, and assumptions developed for the CAIR rule and other recent RIAs (such as the CAND rule), we rely on methods that have already received extensive review by the independent Science Advisory Board (SAB), by the public, and by other federal agencies. In addition, we will be working through the next section 812 prospective study to enhance our methods.[E]

These methods incorporate guidance from the National Academy of Sciences (NAS) (2002) report on its review of the Agency's methodology for analyzing the health benefits of measures taken to reduce air pollution. EPA has been updating its methods to address the NAS

[D] The section 812 studies include: (1) U.S. EPA, Report to Congress: The Benefits and Costs of the Clean Air Act, 1970 to 1990, October 1997 (also known as the ``Section 812 Retrospective Report''); and (2) the first in the ongoing series of prospective studies estimating the total costs and benefits of the Clean Air Act (see EPA report number: EPA-410-R-99-001, November 1999). See Docket A-99-06, Document II-A-21.

[E] Interested parties may want to consult the webpage: http://www.epa.gov/science1 regarding components of the 812 prospective analytical blueprint.

comments; the analysis in the final CAIR rule included our most recent updates.[F] Our analysis of the proposed rule incorporates this most recent work when it is analytically feasible.

This chapter is organized as follows. In Section 12.2, we provide an overview of the air quality impacts modeled for the proposed standards that are used as inputs to the benefits analysis. In Section 12.3, we document key differences between this benefits analysis and the benefits analysis completed for the final CAIR and CAND rules. This section also presents and discusses the key inputs and methods used in the benefits analysis. In Section 12.4, we report the results of the analysis for human health and welfare effects. Section 12.5 qualitatively describes benefits categories that are omitted from this analysis, due either to inadequate methods or resources. Section 12.6 discusses how we incorporate uncertainty into our analysis. Section 12.7 presents the health-based cost-effectiveness analysis for the proposed standards. Finally, in Section 12.8, we present a comparison of the costs and benefits associated with the proposed standards.

## 12.2 Air Quality Impacts

This section summarizes the methods for and results of estimating air quality for the 2020 and 2030 base case and proposed control scenario for the purposes of the benefits analysis. EPA has focused on the health, welfare, and ecological effects that have been linked to ambient changes in $PM_{2.5}$ related to direct PM emission reductions estimated to occur due to the proposed cold temperature vehicle standards. We do this by scaling the modeled relationship between emissions and ambient PM concentrations observed for the CAND analysis.[6]

### 12.2.1 PM Air Quality Impact Estimation

To estimate $PM_{2.5}$ benefits from the proposed cold temperature vehicle standards, we rely on a benefits transfer technique. The benefits transfer approach uses as its foundation the relationship between emission reductions and ambient $PM_{2.5}$ concentrations modeled for the Clean Air Nonroad Diesel (CAND) proposal.[G] For a given future year, we first calculate the ratio between CAND direct $PM_{2.5}$ emission reductions and direct $PM_{2.5}$ emission reductions associated with the proposed standards (proposed emission reductions/CAND emission reductions, displayed in Table 12.2-1). We multiply this ratio by the percent that direct $PM_{2.5}$ contributes towards population-weighted reductions in total $PM_{2.5}$ due to the CAND standards (displayed in Table 12.2-2). This calculation results in a "benefits apportionment factor" for the relationship between direct PM emissions and primary $PM_{2.5}$ (displayed in Table 12.2-3), which is then applied to the BenMAP-based incidence and monetized benefits from the CAND proposal. In this way, we apportion the results of the proposed CAND analysis to its underlying direct PM emission reductions and scale the apportioned benefits to reflect differences in

[F] See Chapter 4 of the Final Clean Air Interstate Rule RIA (www.epa.gov/cair) for a discussion of EPA's ongoing efforts to address the NAS recommendations in its regulatory analyses.
[G] See 68 FR 28327, May 23, 2003.

emission reductions between the modeled CAND control option and the proposed standards.[H] This benefits transfer method is consistent with the approach used in other recent mobile and stationary source rules.[I] We refer the reader to the final CAND RIA for more details on this benefits transfer approach.[7]

**Table 12.2-1. Comparison of 48-state Cold Temperature Emission Reductions in 2020 and 2030 Between the CAND and Proposed Cold Temperature Standards**

| Emissions Species | Reduction from Baseline (tons) | | Ratio of Reductions (MSAT/ CAND) |
|---|---|---|---|
| | CAND Modeling Inputs[a] | Cold Temperature Emissions Changes[b] | |
| **2020** | | | |
| Direct $PM_{2.5}$ | 98,121 | 11,803 | 0.120 |
| **2030** | | | |
| Direct $PM_{2.5}$ | 138,208 | 20,096 | 0.145 |

[a] Includes all affected nonroad sources: land-based, recreational marine, commercial marine, and locomotives. See the CAND RIA for more information regarding the CAND emission inventories.
[b] Includes changes to the light duty onroad vehicles inventory.

**Table 12.2-2. Apportionment of Modeled CAND Preliminary Control Option Population-weighted Change in Ambient $PM_{2.5}$ to Nitrate, Sulfate, and Primary Particles**

| | 2020 | | 2030 | |
|---|---|---|---|---|
| | Population-weighted Change (μg/m3) | Percent of Total Change | Population-weighted Change (μg/m3) | Percent of Total Change |
| Total $PM_{2.5}$ | 0.316 | -- | 0.438 | -- |
| Sulfate | 0.071 | 22.5% | 0.090 | 20.5% |
| Nitrate | 0.041 | 13.1% | 0.073 | 16.8% |
| Primary PM | 0.203 | 64.4% | 0.274 | 62.7% |

Source: CAND RIA, Chapter 9.

[H] Note that while the proposed regulations also control VOCs, which contribute to PM formation, the benefits transfer scaling approach only scales benefits based on NOx, SO2, and direct PM emission reductions. PM benefits will likely be underestimated as a result, though we are unable to estimate the magnitude of the underestimation.
[I] See: Clean Air Nonroad Diesel final rule (69 FR 38958, June 29, 2004); Nonroad Large Spark-Ignition Engines and Recreational Engines standards (67 FR 68241, November 8, 2002); Final Industrial Boilers and Process Heaters NESHAP (69 FR 55217, September 13, 2004); Final Reciprocating Internal Combustion Engines NESHAP (69 FR 33473, June 15, 2004); Final Clean Air Visibility Rule (EPA-452/R-05-004, June 15, 2005); Ozone Implementation Rule (documentation forthcoming).

**Table 12.2-3. Calculation of $PM_{2.5}$ Benefits Apportionment Factor for Proposed Cold Temperature-Related Direct PM Emission Reductions**

| | 2020 | | | 2030 | | |
|---|---|---|---|---|---|---|
| | Ratio of Emission Reductions[a] (1) | % of Total Ambient Change[b] (2) | Benefits Apportionment Factor (1*2) | Ratio of Emission Reductions[a] (3) | % of Total Ambient Change[b] (4) | Benefits Apportionment Factor (3*4) |
| Direct PM Emissions | 0.120 | 0.644 | 0.077 | 0.145 | 0.627 | 0.091 |

[a] Calculated by dividing cold temperature vehicle emission reductions by CAND emission reductions. See Table 12.2-1.
[b] See Table 12.2-2.

## 12.3 PM-Related Health Benefits Estimation - Methods and Inputs

The analytical approach used in this benefits analysis is largely the same approach used in the Final CAIR and Final CAND benefits analyses and the reader is referred to each RIA for details on the benefits methods and inputs. This analysis, however, also reflects advances in data and methods in epidemiology, economics, and health impact estimation. Updates to the assumptions and methods used in estimating ozone-related and $PM_{2.5}$-related benefits since the analysis for the CAIR and CAND rules include the following:

- Use of an updated dataset projecting county-level age-specific mortality rates for future scenarios (1997-2050). This approach combines Centers for Disease Control (CDC) county-level mortality rate data for the years 1996-1998 with US Census Bureau mortality projections out to 2050.[8] This approach is different than the fixed 1996-1998 CDC mortality rate data used in the CAND analysis, and the scaled benefits analysis of the proposed standards has been updated accordingly.

- Use of a revised mortality lag assumption. In the Final CAND, we used a five-year segmented lag. Since that analysis, upon which the PM benefits transfer scaling approach is based, the SAB Health Effects Subcommittee (HES) recommended that until additional research has been completed, EPA should assume a segmented lag structure characterized by 30 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 20 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$. The distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. For future analyses, the specific distribution of deaths over time will need to be determined through research on causes of death and progression of diseases associated with air pollution. It is important to keep in mind that changes in the lag assumptions do not change the total number of estimated deaths but rather the timing of those deaths. This approach is different than the 5-year segmented lag used in the CAND analysis, and the scaled benefits analysis of the proposed standards has been

updated accordingly.

For the purposes of this RIA, the health impacts analysis is limited to those health effects that are directly linked to ambient levels of air pollution and specifically to those linked to PM. The specific studies from which effect estimates for the primary analysis are drawn are included in Table 12.3-1. The specific unit values used for economic valuation of health endpoints are included in Table 12.3-2.

## Table 12.3-1. Endpoints and Studies Used to Calculate Total Monetized Health Benefits

| Endpoint | Pollutant | Study | Study Population |
|---|---|---|---|
| Premature Mortality | | | |
| Premature mortality —cohort study, all-cause | $PM_{2.5}$ | Pope et al. (2002)[9] | >29 years |
| Premature mortality — all-cause | $PM_{2.5}$ | Woodruff et al. (1997)[10] | Infant (<1 year) |
| Chronic Illness | | | |
| Chronic bronchitis | $PM_{2.5}$ | Abbey et al. (1995)[11] | >26 years |
| Nonfatal heart attacks | $PM_{2.5}$ | Peters et al. (2001)[12] | Adults |
| Hospital Admissions | | | |
| Respiratory | $PM_{2.5}$ | Pooled estimate:<br>Moolgavkar (2003)[13]—ICD 490-496 (COPD)<br>Ito (2003)[14]—ICD 490-496 (COPD) | >64 years |
| | $PM_{2.5}$ | Moolgavkar (2000)[15]—ICD 490-496 (COPD) | 20–64 years |
| | $PM_{2.5}$ | Ito (2003)—ICD 480-486 (pneumonia) | >64 years |
| | $PM_{2.5}$ | Sheppard (2003)[16]—ICD 493 (asthma) | <65 years |
| Cardiovascular | $PM_{2.5}$ | Pooled estimate:<br>Moolgavkar (2003)—ICD 390-429 (all cardiovascular)<br>Ito (2003)—ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure) | >64 years |
| | $PM_{2.5}$ | Moolgavkar (2000)—ICD 390-429 (all cardiovascular) | 20–64 years |
| Asthma-related ER visits | $PM_{2.5}$ | Norris et al. (1999)[17] | 0–18 years |
| Other Health Endpoints | | | |
| Acute bronchitis | $PM_{2.5}$ | Dockery et al. (1996)[18] | 8–12 years |
| Upper respiratory symptoms | $PM_{2.5}$ | Pope et al. (1991)[19] | Asthmatics, 9–11 years |
| Lower respiratory symptoms | $PM_{2.5}$ | Schwartz and Neas (2000)[20] | 7–14 years |
| Asthma exacerbations | $PM_{2.5}$ | Pooled estimate:<br>Ostro et al. (2001)[21] (cough, wheeze and shortness of breath)<br>Vedal et al. (1998)[22] (cough) | 6–18 years[a] |
| Work loss days | $PM_{2.5}$ | Ostro (1987)[23] | 18–65 years |
| MRADs | $PM_{2.5}$ | Ostro and Rothschild (1989)[24] | 18–65 years |

[a] The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the SAB-HES, we extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group.

## Table 12.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[a]

| Health Endpoint | Central Estimate of Value Per Statistical Incidence | | | Derivation of Estimates |
|---|---|---|---|---|
| | 1990 Income Level | 2020 Income Level[b] | 2030 Income Level[b] | |
| Premature Mortality (Value of a Statistical Life) | $5,500,000 | $6,600,000 | $6,800,000 | Point estimate is the mean of a normal distribution with a 95 percent confidence interval between $1 and $10 million. Confidence interval is based on two meta-analyses of the wage-risk VSL literature: $1 million represents the lower end of the interquartile range from the Mrozek and Taylor (2002)[25] meta-analysis and $10 million represents the upper end of the interquartile range from the Viscusi and Aldy (2003)[26] meta-analysis. The VSL represents the value of a small change in mortality risk aggregated over the affected population. |
| Chronic Bronchitis (CB) | $340,000 | $420,000 | $430,000 | Point estimate is the mean of a generated distribution of WTP to avoid a case of pollution-related CB. WTP to avoid a case of pollution-related CB is derived by adjusting WTP (as described in Viscusi et al., [1991][27]) to avoid a severe case of CB for the difference in severity and taking into account the elasticity of WTP with respect to severity of CB. |
| Nonfatal Myocardial Infarction (heart attack) | | | | Age-specific cost-of-illness values reflect lost earnings and direct medical costs over a 5-year period following a nonfatal MI. Lost earnings estimates are based on Cropper and Krupnick (1990).[28] Direct medical costs are based on simple average of estimates from Russell et al. (1998)[29] and Wittels et al. (1990).[30] |
| 3% discount rate | | | | Lost earnings: |
| Age 0–24 | $66,902 | $66,902 | $66,902 | Cropper and Krupnick (1990). Present discounted value of 5 years of lost earnings: |
| Age 25–44 | $74,676 | $74,676 | $74,676 | |
| Age 45–54 | $78,834 | $78,834 | $78,834 | |
| Age 55–65 | $140,649 | $140,649 | $140,649 | |
| Age 66 and over | $66,902 | $66,902 | $66,902 | |
| 7% discount rate | | | | |
| Age 0–24 | $65,293 | $65,293 | $65,293 | |
| Age 25–44 | $73,149 | $73,149 | $73,149 | |
| Age 45–54 | $76,871 | $76,871 | $76,871 | |
| Age 55–65 | $132,214 | $132,214 | $132,214 | |
| Age 66 and over | $65,293 | $65,293 | $65,293 | |

| age of onset: | at 3% | at 7% |
|---|---|---|
| 25-44 | $8,774 | $7,855 |
| 45-54 | $12,932 | $11,578 |
| 55-65 | $74,746 | $66,920 |

Direct medical expenses: An average of:
1. Wittels et al. (1990) ($102,658—no discounting)
2. Russell et al. (1998), 5-year period ($22,331 at 3% discount rate; $21,113 at 7% discount rate)

(continued)

## Table 12.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[a] (continued)

| Health Endpoint | Central Estimate of Value Per Statistical Incidence | | | Derivation of Estimates |
|---|---|---|---|---|
| | 1990 Income Level | 2020 Income Level[b] | 2030 Income Level[b] | |
| **Hospital Admissions** | | | | |
| Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496) | $12,378 | $12,378 | $12,378 | The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total COPD category illnesses) reported in Agency for Healthcare Research and Quality (2000)[31] (www.ahrq.gov). |
| Pneumonia (ICD codes 480-487) | $14,693 | $14,693 | $14,693 | The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total pneumonia category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov). |
| Asthma Admissions | $6,634 | $6,634 | $6,634 | The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total asthma category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov). |
| All Cardiovascular (ICD codes 390-429) | $18,387 | $18,387 | $18,387 | The COI estimates (lost earnings plus direct medical costs) are based on ICD-9 code-level information (e.g., average hospital care costs, average length of hospital stay, and weighted share of total cardiovascular category illnesses) reported in Agency for Healthcare Research and Quality (2000) (www.ahrq.gov). |
| Emergency Room Visits for Asthma | $286 | $286 | $286 | Simple average of two unit COI values:<br>(1) $311.55, from Smith et al. (1997)[32] and<br>(2) $260.67, from Stanford et al. (1999).[33] |

(continued)

## Table 12.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[a] (continued)

| Health Endpoint | Central Estimate of Value Per Statistical Incidence | | | Derivation of Estimates |
|---|---|---|---|---|
| | 1990 Income Level | 2020 Income Level[b] | 2030 Income Level[b] | |
| **Respiratory Ailments Not Requiring Hospitalization** | | | | |
| Upper Respiratory Symptoms (URS) | $25 | $27 | $27 | Combinations of the three symptoms for which WTP estimates are available that closely match those listed by Pope et al. result in seven different "symptom clusters," each describing a "type" of URS. A dollar value was derived for each type of URS, using mid-range estimates of WTP (IEc, 1994)[34] to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for URS is the average of the dollar values for the seven different types of URS. |
| Lower Respiratory Symptoms (LRS) | $16 | $17 | $17 | Combinations of the four symptoms for which WTP estimates are available that closely match those listed by Schwartz et al. result in 11 different "symptom clusters," each describing a "type" of LRS. A dollar value was derived for each type of LRS, using mid-range estimates of WTP (IEc, 1994) to avoid each symptom in the cluster and assuming additivity of WTPs. The dollar value for LRS is the average of the dollar values for the 11 different types of LRS. |
| Asthma Exacerbations | $42 | $45 | $45 | Asthma exacerbations are valued at $42 per incidence, based on the mean of average WTP estimates for the four severity definitions of a "bad asthma day," described in Rowe and Chestnut (1986).[35] This study surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day," as defined by the subjects. For purposes of valuation, an asthma attack is assumed to be equivalent to a day in which asthma is moderate or worse as reported in the Rowe and Chestnut (1986) study. |
| Acute Bronchitis | $360 | $380 | $390 | Assumes a 6-day episode, with daily value equal to the average of low and high values for related respiratory symptoms recommended in Neumann et al. (1994).[36] |

(continued)

## Table 12.3-2. Unit Values Used for Economic Valuation of Health Endpoints (2000$)[a] (continued)

| Health Endpoint | Central Estimate of Value Per Statistical Incidence | | | Derivation of Estimates |
|---|---|---|---|---|
| | 1990 Income Level | 2020 Income Level[b] | 2030 Income Level[b] | |
| **Restricted Activity and Work/School Loss Days** | | | | |
| Work Loss Days (WLDs) | Variable (national median = ) | | | County-specific median annual wages divided by 50 (assuming 2 weeks of vacation) and then by 5—to get median daily wage. U.S. Year 2000 Census, compiled by Geolytics, Inc. |
| Minor Restricted Activity Days (MRADs) | $51 | $54 | $55 | Median WTP estimate to avoid one MRAD from Tolley et al. (1986).[37] |

[a] Although the unit values presented in this table are in year 2000 dollars, all monetized annual benefit estimates associated with the proposed standards have been inflated to reflect values in year 2003 dollars. We use the Consumer Price Indexes to adjust both WTP- and COI-based benefits estimates to 2003 dollars from 2000 dollars.[38] For WTP-based estimates, we use an inflation factor of 1.07 based on the CPI-U for "all items." For COI-based estimates, we use an inflation factor of 1.14 based on the CPI-U for medical care.

[b] Our analysis accounts for expected growth in real income over time. Economic theory argues that WTP for most goods (such as environmental protection) will increase if real incomes increase. Benefits are therefore adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor to account for income growth over time. For a complete discussion of how these adjustment factors were derived, we refer the reader to Chapter 9 of the CAND regulatory impact analysis (EPA, 2004). Note that similar adjustments do not exist for cost-of-illness-based unit values. For these, we apply the same unit value regardless of the future year of analysis.

EPA typically estimates the welfare impacts of effects such as changes in recreational visibility (related to reductions in ambient PM) and agricultural productivity (related to reductions in ambient ozone) in its RIAs of air quality policy. For the analysis of the proposed standards, however, we are unable to quantitatively characterize these impacts because of limited data availability; we are not quantifying ozone benefits related to the proposed standards and the PM scaling approach does not provide the spatial detail necessary to attribute specific air quality improvements to specific areas of visual interest (Class I areas). Instead, we discuss these welfare effects qualitatively in Section 12.5 of this chapter. We also qualitatively describe the impacts of other environmental and ecological effects for which we do not have an economic value.

Similar to Kunzli et al. (2000)[39] and other recent health impact analyses, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Adjustments are made for the level of environmental quality change, the sociodemographic and economic characteristics of the affected population, and other factors to improve the accuracy and robustness of benefits estimates.

## 12.4 Benefits Analysis Results for the Proposed Cold Temperature Vehicle Standards

Applying the impact and valuation functions described previously in this chapter to the estimated changes in $PM_{2.5}$ associated with the proposed cold temperature vehicle standards results in estimates of the changes in physical damages (e.g., premature mortalities, cases, admissions) and the associated monetary values for those changes. Estimates of physical health impacts are presented in Table 12.4-1. Monetized values for those health endpoints are presented in Table 12.4-2, along with total aggregate monetized benefits. All of the monetary benefits are in constant-year 2003 dollars.

## Table 12.4-1. Estimated Reduction in Incidence of Adverse Health Effects Related to the Proposed Cold Temperature Standards[a]

| Health Effect | 2020 Incidence Reduction | 2030 Incidence Reduction |
|---|---|---|
| **PM-Related Endpoints** | | |
| Premature Mortality[b,c] | | |
| Adult, age 30+ and Infant, age <1 year | 480 | 910 |
| Chronic bronchitis (adult, age 26 and over) | 330 | 590 |
| Nonfatal myocardial infarction (adults, age 18 and older) | 820 | 1,600 |
| Hospital admissions—respiratory (all ages)[d] | 260 | 540 |
| Hospital admissions—cardiovascular (adults, age >18)[e] | 220 | 400 |
| Emergency room visits for asthma (age 18 years and younger) | 360 | 630 |
| Acute bronchitis (children, age 8–12) | 790 | 1,400 |
| Lower respiratory symptoms (children, age 7–14) | 9,400 | 17,000 |
| Upper respiratory symptoms (asthmatic children, age 9–18) | 7,100 | 13,000 |
| Asthma exacerbation (asthmatic children, age 6–18) | 12,000 | 21,000 |
| Work loss days (adults, age 18–65) | 63,000 | 110,000 |
| Minor restricted-activity days (adults, age 18–65) | 370,000 | 620,000 |

[a] Incidences are rounded to two significant digits. PM estimates represent benefits from the proposed rule nationwide.

[b] PM premature mortality impacts for adults are based on application of the effect estimate derived from the Pope et al (2002) cohort study.[40] Infant premature mortality based upon studies by Woodruff, et al 1997.[41]

[c] PM-related mortality benefits estimated using an assumed PM threshold at background levels (3 $\mu g/m^3$). There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate. For a more detailed discussion of this issue, please refer to Section 12.6.1.1 of the RIA.

[d] Respiratory hospital admissions for PM include admissions for COPD, pneumonia, and asthma.

[e] Cardiovascular hospital admissions for PM include total cardiovascular and subcategories for ischemic heart disease, dysrhythmias, and heart failure.

**Table 12.4-2. Estimated Monetary Value in Reductions in Incidence of Health and Welfare Effects (in millions of 2003$)[a,b]**

| PM-Related Health Effect | 2020 Estimated Value of Reductions | 2030 Estimated Value of Reductions |
|---|---|---|
| Premature mortality[c,d,e] | | |
| Adult, age 30+ and Infant, < 1 year | | |
| 3% discount rate | $3,100 | $6,000 |
| 7% discount rate | $2,800 | $5,400 |
| Chronic bronchitis (adults, 26 and over) | $150 | $270 |
| Non-fatal acute myocardial infarctions | | |
| 3% discount rate | $80 | $150 |
| 7% discount rate | $77 | $150 |
| Hospital admissions for respiratory causes | $4.8 | $10 |
| Hospital admissions for cardiovascular causes | $5.1 | $9.4 |
| Emergency room visits for asthma | $0.12 | $0.21 |
| Acute bronchitis (children, age 8–12) | $0.32 | $0.58 |
| Lower respiratory symptoms (children, 7–14) | $0.17 | $0.30 |
| Upper respiratory symptoms (asthma, 9–11) | $0.20 | $0.37 |
| Asthma exacerbations | $0.57 | $1.0 |
| Work loss days | $9.2 | $14 |
| Minor restricted-activity days (MRADs) | $21 | $36 |
| Monetized Total [f] | | |
| Base Estimate: | | |
| 3% discount rate | $3,400+ B | $6,500+ B |
| 7% discount rate | $3,100+ B | $5,900+ B |

[a] Monetary benefits are rounded to two significant digits for ease of presentation and computation. PM benefits are nationwide.

[b] Monetary benefits adjusted to account for growth in real GDP per capita between 1990 and the analysis year (2020 or 2030)

[c] PM-related mortality benefits estimated using an assumed PM threshold at background levels (3 $\mu g/m^3$). There is uncertainty about which threshold to use and this may impact the magnitude of the total benefits estimate. For a more detailed discussion of this issue, please refer to Section 12.6.1.1 of the RIA.

[d] Valuation assumes discounting over the SAB recommended 20 year segmented lag structure described earlier. Results reflect the use of 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).[42,43]

[e] Adult premature mortality estimates based upon studies by Pope, et al 2002.[44] Infant premature mortality based upon Woodruff et al 1997.[45]

[f] B represents the monetary value of health and welfare benefits and disbenefits not monetized. A detailed listing is provided in Table 12-2.

In addition to omitted benefits categories such as air toxics, ozone, and various welfare effects, not all known PM-related health and welfare effects could be quantified or monetized. The monetized value of all of these unquantified effects is represented by adding an unknown "B" to the aggregate total. The estimate of total monetized health benefits of the proposed MSAT control package is thus equal to the subset of monetized PM-related health benefits plus B, the sum of the nonmonetized health and welfare benefits.

Total monetized benefits are dominated by benefits of mortality risk reductions. The

primary estimate projects that the proposed cold temperature vehicle standards would result in 480 avoided premature deaths annually in 2020 and 910 avoided premature deaths annually in 2030. The increase in annual benefits from 2020 to 2030 reflects additional emission reductions from the proposed cold temperature vehicle standards, as well as increases in total population and the average age (and thus baseline mortality risk) of the population.

Our estimate of total monetized benefits in 2020 for the proposed cold temperature vehicle standards is $3.4 billion using a 3 percent discount rate and $3.1 billion using a 7 percent discount rate. In 2030, the monetized benefits are estimated at $6.5 billion using a 3 percent discount rate and $5.9 billion using a 7 percent discount rate. The monetized benefit associated with reductions in the risk of premature mortality, which accounts for $3.1 billion in 2020 and $6.0 billion in 2030 (assuming a 3 percent discount rate), is over 90 percent of total monetized health benefits. The next largest benefit is for reductions in chronic illness (CB and nonfatal heart attacks), although this value is more than an order of magnitude lower than for premature mortality. Hospital admissions for respiratory and cardiovascular causes, minor restricted activity days, and work loss days account for the majority of the remaining benefits. The remaining categories each account for a small percentage of total benefit; however, they represent a large number of avoided incidences affecting many individuals. A comparison of the incidence table to the monetary benefits table reveals that there is not always a close correspondence between the number of incidences avoided for a given endpoint and the monetary value associated with that endpoint. For example, there are over 100 times more work loss days than premature mortalities, yet work loss days account for only a very small fraction of total monetized benefits. This reflects the fact that many of the less severe health effects, while more common, are valued at a lower level than the more severe health effects. Also, some effects, such as hospital admissions, are valued using a proxy measure of willingness-to-pay (e.g., cost-of-illness). As such, the true value of these effects may be higher than that reported in Table 12-9.

## 12.5 Unquantified Health and Welfare Effects

In considering the monetized benefits estimates, the reader should remain aware of the many limitations of conducting the analyses mentioned throughout this RIA. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the effects listed in Table 12.1-2. For many health and welfare effects, such as changes in health effects due to reductions in air toxics exposure, changes in ecosystem functions and PM-related materials damage, reliable impact functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefit categories, the benefits estimates presented in this analysis would increase, although the magnitude of such an increase is highly uncertain.

Other welfare effects that EPA has monetized in past RIAs, such as recreational visibility, are omitted from the current analysis. Due to time and resource constraints, we did not run the full-scale PM air quality modeling needed to estimate this benefit category. Instead, we relied on the PM scaling benefits transfer approach that provides analytical efficiency but sacrifices the full range of outputs typically generated when models such as the Community

Multiscale Air Quality (CMAQ) model or the Regional Modeling System for Aerosols and Deposition (REMSAD) are run. We will explore how to monetize these welfare effects using the available tools for the analysis of the final standards.

Unquantified benefits are qualitatively discussed in the following health and welfare effects sections. In addition to unquantified benefits, there may also be environmental costs (disbenefits) that we are unable to quantify, which we qualitatively discuss as well. The net effect of excluding benefit and disbenefit categories from the estimate of total benefits depends on the relative magnitude of the effects. Although we are not currently able to estimate the magnitude of these unquantified and unmonetized benefits, specific categories merit further discussion. EPA believes, however, the unquantified benefits associated with health and non-health benefit categories are likely significant.

### 12.5.1 Human Health Impact Assessment

In addition to the PM health effects discussed above, there is emerging evidence that human exposure to PM may be associated a number of health effects not quantified in this analysis (see Table 12.1-2). An improvement in ambient PM concentrations may reduce the number of incidences within each of these unquantified effect categories that the U.S. population would experience. Although these health effects are believed to be PM-induced, effect estimates are not available for quantifying the benefits associated with reducing these effects. The inability to quantify these effects lends a downward bias to the monetized benefits presented in this analysis.

Other standards we are currently proposing, such as the gas can standards, would also reduce the national emissions inventory of precursors to ozone, such as VOCs. Exposure to ozone has been linked to a variety of respiratory effects including hospital admissions, emergency room visits, minor restricted activity days, worker productivity and illnesses resulting in school absences. Emerging evidence has also shown that human exposure to ozone may be associated with a number of other health effects not quantified in this analysis (see Table 12.1-2). Ozone can also adversely affect the agricultural and forestry sectors by decreasing yields of crops and forests. Although ozone benefits are typically quantified in regulatory impact analyses, we do no evaluate them for this analysis because of the magnitude of, and uncertainty associated with, the ambient ozone modeling data. As discussed earlier in this chapter (and in Chapter 3), the ozone modeling conducted for the proposed gas can standards results in a net reduction, when population weighted, in the ozone design value metric measured within the modeled domain (37 Eastern states and the District of Columbia). The net improvement, however, is very small. For the most part, quantifiable ozone benefits do not contribute significantly to the monetized benefits; thus, their omission will not materially affect the conclusions of the benefits analysis.

Over the past several years, EPA has consulted with the Science Advisory Board regarding evidence for an independent ozone mortality effect. Because of new studies and the recommendations from the SAB, EPA sponsored three independent meta-analyses of the ozone-mortality epidemiology literature to inform a determination on including this important health

endpoint. The three meta-analyses were published in the journal Epidemiology in July 2005.[46,47,48] These meta-analyses, as well as another major study in the Journal of the American Medical Association,[49] reported that on average, short-term changes in ozone are significantly associated with premature mortality, and that the significance of the association is robust to adjustment for particulate matter. The JAMA study used the extensive National Morbidity, Mortality, and Air Pollution Study database to examine associations between ozone and premature mortality in 95 U.S. urban communities.

The Agency believes that publication of these studies significantly enhances the scientific defensibility of benefits estimates for ozone that include the benefits of premature mortality reductions. In the future we plan to examine a variety of ozone mortality quantification methods, including approaches that provide information on relative probability of different benefits levels. Using effect estimates similar to those found in these new studies, EPA estimates that the monetary value of the ozone-related premature mortality benefits could be substantial.

### 12.5.2 Welfare Impact Assessment

For many welfare effects, such as changes in ecosystem functions and PM-related materials damage, reliable impact functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefit categories, the benefits estimates presented in this analysis would increase, although the magnitude of such an increase is highly uncertain.

#### 12.5.2.1 Visibility Benefits

Changes in the level of ambient PM caused by the reduction in emissions from the proposed standards would change the level of visibility in much of the United States. Visibility directly affects people's enjoyment of a variety of daily activities. Individuals value visibility both in the places they live and work, in the places they travel to for recreational purposes, and at sites of unique public value, such as the Great Smoky Mountains National Park. Though not quantified in this analysis, the value of improvements in visibility monetized for regulatory analyses such as the final CAIR are significant. We refer the reader to that analysis for a complete description of the methods used to value visibility.[50]

#### 12.5.2.2 Agricultural, Forestry and other Vegetation-Related Benefits

The Ozone Criteria Document notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant" (EPA, 1996, page 5-11).[51] Changes in ground-level ozone would result from the proposed standards are expected to affect crop and forest yields throughout the affected area.

Well-developed techniques exist to provide monetary estimates of these benefits to agricultural producers and to consumers. These techniques use models of planting decisions, yield response functions, and agricultural products' supply and demand. The resulting welfare measures are based on predicted changes in market prices and production costs. Models also

exist to measure benefits to silvicultural producers and consumers. However, these models have not been adapted for use in analyzing ozone-related forest impacts. Because of resource limitations, we are unable to provide agricultural or forestry benefits estimates for the proposed standards.

12.5.2.2.1 Agricultural Benefits

Laboratory and field experiments have shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN), examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the United States."[54] In addition, economic studies have shown a relationship between observed ozone levels and crop yields.[52]

12.5.2.2.2 Forestry Benefits

Ozone also has been shown conclusively to cause discernible injury to forest trees (EPA, 1996; Fox and Mickler, 1996).[54,53] In our previous analysis of the HD Engine/Diesel Fuel rule, we were able to quantify the effects of changes in ozone concentrations on tree growth for a limited set of species. Because of resource limitations, we were not able to quantify such impacts for this analysis.

12.5.2.2.3 Other Vegetation Effects

An additional welfare benefit expected to accrue as a result of reductions in ambient ozone concentrations in the United States is the economic value the public receives from reduced aesthetic injury to forests. There is sufficient scientific information available to reliably establish that ambient ozone levels cause visible injury to foliage and impair the growth of some sensitive plant species (EPA, 1996).[54] However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

Urban ornamentals (floriculture and nursery crops) represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to affect large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative economic benefits analysis has been conducted. The farm production value of ornamental crops was estimated at over $14 billion in 2003 (USDA, 2004).[54] This is therefore a potentially important welfare effects category. However, information and valuation methods are not available to allow for plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

### 12.5.2.3 Benefits from Reductions in Materials Damage

The proposed standards that we modeled are expected to produce economic benefits in the form of reduced materials damage. There are two important categories of these benefits. Household soiling refers to the accumulation of dirt, dust, and ash on exposed surfaces. $PM_{2.5}$ also has corrosive effects on commercial/industrial buildings and structures of cultural and historical significance. The effects on historic buildings and outdoor works of art are of particular concern because of the uniqueness and irreplaceability of many of these objects.

Previous EPA benefits analyses have been able to provide quantitative estimates of household soiling damage. Consistent with SAB advice, we determined that the existing data (based on consumer expenditures from the early 1970s) are too out of date to provide a reliable estimate of current household soiling damages (EPA-SAB-COUNCIL-ADV-98-003, 1998).[55]

EPA is unable to estimate any benefits to commercial and industrial entities from reduced materials damage. Nor is EPA able to estimate the benefits of reductions in PM-related damage to historic buildings and outdoor works of art. Existing studies of damage to this latter category in Sweden (Grosclaude and Soguel, 1994)[56] indicate that these benefits could be an order of magnitude larger than household soiling benefits.

### 12.5.3 UVb Exposure

In contrast to the unquantified benefits of the proposed standards discussed above, it is also possible that this rule will result in disbenefits in some areas of the United States. The effects of ozone and PM on radiative transfer in the atmosphere can lead to effects of uncertain magnitude and direction on the penetration of ultraviolet light and climate. Ground level ozone makes up a small percentage of total atmospheric ozone (including the stratospheric layer) that attenuates penetration of ultraviolet - b (UVb) radiation to the ground. EPA's past evaluation of the information indicates that potential disbenefits would be small, variable, and with too many uncertainties to attempt quantification of relatively small changes in average ozone levels over the course of a year.[57] EPA's most recent provisional assessment of the currently available information indicates that potential but unquantifiable benefits may also arise from ozone-related attenuation of UVb radiation.[58] EPA believes that we are unable to quantify any net climate-related disbenefit or benefit associated with the combined ozone and PM reductions in this rule.

## 12.6 Methods for Describing Uncertainty

In any complex analysis using estimated parameters and inputs from numerous models, there are likely to be many sources of uncertainty. This analysis is no exception. As outlined both in this and preceding chapters, many inputs were used to derive the proposed benefits estimate, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological health effect estimates, estimates of values (both from WTP and COI studies), population estimates, income estimates, and estimates of the future state of the world (i.e., regulations, technology, and human behavior). Each of these inputs may be uncertain and, depending on its role in the benefits analysis, may have a disproportionately large impact on estimates of total benefits. For example, emissions estimates are used in the first stage of the analysis. As such, any uncertainty in emissions estimates will be propagated through the entire analysis.

Some key sources of uncertainty in each stage of the benefits analysis are the following:

- The exclusion of potentially substantial benefit categories (such as health, odor, and ecological benefits of reduction in air toxics, ozone, and PM);
- Errors in measurement and projection for variables such as population growth;
- Uncertainties in the estimation of future year emissions inventories and air quality, including uncertainties in the estimated reductions in PM emissions resulting from the cold temperature standard for light-duty vehicles;
- Uncertainties associated with the scaling of the PM results of the modeled benefits analysis to the proposed standards, especially regarding the assumption of similarity in geographic distribution between emissions and human populations and years of analysis;
- Uncertainty in the estimated relationships of health and welfare effects to changes in pollutant concentrations including the shape of the C-R function, the size of the effect estimates, and the relative toxicity of the many components of the PM mixture;
- Uncertainties in exposure estimation; and
- Uncertainties associated with the effect of potential future actions to limit emissions.

The NRC report on estimating public health benefits of air pollution regulations recommended that EPA begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses (NRC, 2002).[59] The probability distributions required for these analyses should be based on available data and expert judgment.

As part of EPA's approach to characterizing uncertainties in the benefits assessment, we generate a probabilistic estimate of statistical uncertainty based on standard errors reported in the underlying studies used in the benefits modeling framework, with particular emphasis on the health impact functions. Using a Monte Carlo procedure, the distribution of each health endpoint and its unit dollar value is characterized by the reported mean and standard error derived from the epidemiology and valuation literature. Details on the distributions used for individual health endpoints are provided in the CAIR RIA (Appendix B; EPA, 2005).[60] It is likely that these

distributions do not capture the full range of benefits, and in fact are likely to understate the uncertainty, especially on the high end of the range due to omission of potentially significant benefit categories. We estimate them here as an illustration of EPA's traditional approach towards characterizing uncertainty using probabilistic, statistical error-based distributions. Results and discussion of the Monte Carlo approach can be found in Appendix 12.A.

In addition to the Monte Carlo approach to characterizing statistical sources of uncertainty, we also supplement our primary estimates of benefits with a series of sensitivity calculations that use other sources of health effect estimates and valuation data for key benefits categories. The supplemental estimates examine sensitivity to both valuation issues (e.g., the type of lag structure used for the valuation of PM-related premature mortality) and physical effects issues (e.g., alternative health impact functions for PM-related premature mortality). The results of these supplemental calculations are presented in Appendix 12.B.

### 12.6.1 Uncertainty Related to PM-Mortality

As part of an overall program to improve the Agency's characterization of uncertainties in health benefits analyses, we attempt to address uncertainties associated with the PM mortality health impact function relationship and valuation. Use of the Pope et al., 2002-derived mortality function to support this analysis is associated with uncertainty resulting from: (a) potential of the study to incompletely capture short-term exposure-related mortality effects, (b) potential mis-match between study and analysis populations which introduces various forms of bias into the results, (c) failure to identify all key confounders and effects modifiers, which could result in incorrect effects estimates relating morality to $PM_{2.5}$ exposure, and (d) model uncertainty. EPA is researching methods to characterize all elements of uncertainty in the dose-response function for mortality.

As is discussed in detail in both the CAND RIA and the CAIR RIA, EPA has used two methods to quantify uncertainties in the mortality function, including: the statistical uncertainty derived from the standard errors reported in the Pope et al., 2002 study, and the use of results of a pilot expert elicitation conducted in 2004 to investigate other uncertainties in the mortality estimate. Because this analysis utilizes the PM scaling benefits transfer approach to estimate mortality incidence for the proposed standard, we can not quantify the PM mortality uncertainty to the same extent as was done for the CAIR or CAND analyses. However, in a similar fashion to the analysis conducted for the Clean Air Visibility Rule (CAVR),[61] we can scale the results of the CAND mortality uncertainty analysis to the direct PM emission changes modeled for the proposed cold temperature standards.

In the benefit analysis of the CAND 2030 emission control standards, the statistical uncertainty represented by the standard error of the Pope et al, 2002 study was one and one-half times the mean benefit estimate at the 95$^{th}$ percentile and less than one-half of the mean at the 5$^{th}$ percentile. The expert elicitation provided mean estimates that ranged in value from less than one-third of the mean estimate from the Pope et al, 2002 study-based estimate to nearly one and one-half times the Pope et al., 2002-based estimate. The confidence intervals from the pilot elicitation applied to the CAND 2030 benefit analysis ranged in value from zero at the 5$^{th}$

percentile to a value at the 95th percentile that is approximately three times higher than the Pope et al., 2002-based mean estimate.

These results are highly dependent on the air quality scenarios applied to the concentration-response functions of the Pope et al, 2002 study and the pilot expert elicitation. Thus, the characterization of uncertainty discussed in the CAND RIA could differ greatly from what would be observed for the proposed standards due to differences in population-weighted changes in concentrations of $PM_{2.5}$ (i.e., the location of populations exposure relative to the changes in air quality), and may be especially sensitive to the differences in baseline $PM_{2.5}$ air quality experienced by populations prior to the implementation of the proposed standards.

Table 12.6-1 shows the mean estimate and estimated 5th and 95th percentiles of premature deaths avoided for our 2030 scaled primary estimate based on the Pope et al. (2002) study and based on the scaled responses for each of the 5 experts. This table shows that for the proposed standards, our estimates are higher than those based on the functions provided by four of the experts and lower than that provided by one expert, but falls within the scaled uncertainty bounds of all but one expert. The table shows that for the proposed standards, the average estimated annual number of premature deaths avoided in 2030 ranges from approximately 260 (based on the judgments of Expert C) to 1,200 (based on the judgments of Expert E). The 5th to 95th percentile of all the estimates, including the Pope et al.-based distribution, overlap. Although the distributions for each expert include zero, and some distributions have significant percentiles at zero, all of the distributions have a positive mean estimate. EPA is continuing its research of methods to characterize uncertainty in total benefits estimates, and is conducting a full-scale expert elicitation. The full-scale expert elicitation is scheduled to be completed in 2006.

**Table 12.6-1. Results of Illustrative Application of Pilot Expert Elicitation: Annual Reductions in Premature Mortality in 2030 Associated with the Proposed Cold Temperature Vehicle Standards Scaled from the CAND Analysis**

| Source of Mortality Estimate | 2030 Primary Option | | |
|---|---|---|---|
| | 5th Percentile | Mean | 95th Percentile |
| Pope et al. (2002) | 410 | 910 | 1,400 |
| Expert A | 0 | 750 | 1,400 |
| Expert B | 0 | 410 | 1,800 |
| Expert C | 0 | 260 | 670 |
| Expert D | 0 | 630 | 1,600 |
| Expert E | 0 | 1,200 | 2,500 |

### 12.6.1.1 PM-Mortality Cutpoint Analysis

Another source of uncertainty that has received recent attention from several scientific review panels is the shape of the concentration-response function for PM-related mortality, and specifically whether there exists a threshold below which there would be no benefit to further reductions in $PM_{2.5}$. The consistent advice from EPA's SAB[J] has been to model premature mortality associated with PM exposure as a nonthreshold effect, that is, with harmful effects to exposed populations regardless of the absolute level of ambient PM concentrations. However, EPA's most recent $PM_{2.5}$ Criteria Document concludes that "the available evidence does not either support or refute the existence of thresholds for the effects of PM on mortality across the range of concentrations in the studies".[62] Some researchers have hypothesized the presence of a threshold relationship. That is, the hypothesized relationship includes the possibility that there exists a PM concentration level below which further reductions no longer yield premature mortality reduction benefits.

To consider the impact of a threshold in the response function for the chronic mortality endpoint, the proposed PM NAAQS RIA[63] constructed a sensitivity analysis by assigning different cutpoints below which changes in $PM_{2.5}$ are assumed to have no impact on premature mortality. In applying the cutpoints, the PM NAAQS analysis adjusted the mortality function slopes accordingly.[K] Four cutpoints were included in the sensitivity analysis: (a) 15 $\mu g/m^3$ (based on the current NAAQS); (b) 10 $\mu g/m^3$ (reflects comments from CASAC, 2005)[64]; (c) 7.5 $\mu g/m^3$ (reflects recommendations from SAB-HES (2004)[65] to consider estimating mortality benefits down to the lowest exposure levels considered in the Pope et al. (2002)[66] study used as the basis for modeling chronic mortality); and (d) background or 3 $\mu g/m^3$ (reflects NAS (2002)[67] recommendation to consider effects all the way to background). The results of the sensitivity analysis displayed the change in avoided mortality cases and associated monetary benefits associated with the alternative cutpoints (see the proposed PM NAAQS RIA, Chapter 3, Table 3-8).

A sensitivity analysis such as this can be difficult to interpret, because when a threshold above the lowest observed level of PM2.5 in the underlying epidemiology study (Pope et al., 2002) is assumed, the slope of the concentration-response function above that level must be adjusted upwards to account for the assumed threshold.[L] Depending on the amount of slope adjustment and the proportion of the population exposed above the assumed threshold, the estimated mortality impact can either be lower (if most of the exposures occur below the threshold) or higher (if most of the exposures occur above the threshold). To demonstrate this,

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[J] The advice from the 2004 SAB-HES (EPA-SAB-COUNCIL-ADV-04-002)[69] is characterized by the following: "For the studies of long-term exposure, the HES notes that Krewski et al. (2000) have conducted the most careful work on this issue. They report that the associations between $PM_{2.5}$ and both all-cause and cardiopulmonary mortality were near linear within the relevant ranges, with no apparent threshold. Graphical analyses of these studies (Dockery et al., 1993, Figure 3, and Krewski et al., 2000, page 162) also suggest a continuum of effects down to lower levels. Therefore, it is reasonable for EPA to assume a no threshold model down to, at least, the low end of the concentrations reported in the studies."

[K] Note that the PM NAAQS analysis only adjusted the mortality slopes for the 10 $\mu g/m^3$ and 15 $\mu g/m^3$ cutpoints since the 7.5 $\mu g/m^3$ and background cutpoints were at or below the lowest measured exposure levels reported in the Pope et al. (2002) study for the combined exposure dataset.

[L] See NAS (2002)[71] and CASAC (2005)[68] for discussions of this issue.

we present an example from the proposed PM NAAQS RIA. In its examination of the benefits of attaining alternative PM NAAQS in Chicago,[M] the analysis found that, because annual mean levels are generally higher in Chicago, there was a two-part pattern to the relationship between assumed threshold and mortality impacts. As the threshold increased from background to 7.5 $\mu g/m^3$, the mortality impact fell (because there is no slope adjustment). However, at an assumed threshold of 10 $\mu g/m^3$, estimated mortality impacts actually increased, because the populations exposed above 10 $\mu g/m^3$ were assumed to have a larger response to particulate matter reductions (due to the increased slope above the assumed threshold). And finally, mortality impacts again fell to zero if a 15 $\mu g/m^3$ threshold was assumed, because these impacts were measured incremental to attainment of the current standard.

We are unable to do this type of sensitivity analysis for the proposed MSAT rule because of the analytical limitations of the PM benefits scaling procedure. When EPA conducted the CAND analysis (from which the primary estimates of benefits for the proposed cold temperature vehicle standards are based), there were no PM mortality concentration-response functions with the slope adjusted upwards to account for an assumed threshold. Instead, our primary PM benefits estimate for the proposed cold temperature vehicle standards reflects a background threshold assumption of 3 $\mu g/m^3$. For the final MSAT rule analysis, we plan on examining the impact cutpoints have on our primary estimate of PM mortality benefits related to the proposed cold temperature vehicle standards. For now, however, we present in Table 12.6-2 the results of our scaled PM-related mortality benefits in the context of its relationship to other cutpoints. Note that to the extent we are able, we will endeavor to quantify the omissions in this table in the analysis of the final MSAT rule.

[M] See the proposed PM NAAQS RIA (2005),[67] Appendix A, pp. A63-A64.

**Table 12.6-2. PM-Related Mortality Benefits of the Proposed Cold Temperature Vehicle Standards: Cutpoint Sensitivity Analysis[a]**

| *Certainty that Benefits are At Least Specified Value* | *Level of Assumed Threshold* | *Discount Rate* | **PM Mortality Benefits (Billion 2003$)** | |
|---|---|---|---|---|
| | | | 2020 | 2030 |
| More Certain | 15 μg/m$^3$ [c] | 3% | N/A[b] | |
| | | 7% | | |
| | 10 μg/m$^3$ [d] | 3% | N/A | |
| | | 7% | | |
| | 7.5 μg/m$^3$ [e] | 3% | N/A | |
| | | 7% | | |
| | 3 μg/m$^3$ [f] | 3% | $3.1 | $6.0 |
| Less Certain | | 7% | $2.8 | $5.9 |

[a] Note that this table only presents the effects of a cutpoint on PM-related mortality incidence and valuation estimates.
[b] We are unable to provide cutpoint analysis results for the proposed MSAT rule because of the analytical limitations of the PM benefits scaling procedure. To the extent we are able, we will endeavor to quantify the omissions in this table in the analysis of the final MSAT rule.
[c] EPA intends to analyze a cutpoint between 12 μg/m$^3$and 15 μg/m$^3$ for the final RIA.
[d] CASAC (2005)[68]
[e] SAB-HES (2004)[69]
[f] NAS (2002)[71]

## 12.7 Health-Based Cost Effectiveness Analysis

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. The Office of Management and Budget (OMB) 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, making application of CEA more difficult and less straightforward. For the CAIR analysis, the first to incorporate an analysis of this kind, CEA provided a useful framework for evaluation: nonhealth benefits were substantial, but the majority of quantified benefits came from health effects. EPA included in the CAIR RIA a preliminary and experimental application of one type of CEA—a modified quality-adjusted life-years (QALYs) approach. For CAIR, EPA concluded that the direct usefulness of cost-effectiveness analysis is mitigated by the lack of rule alternatives to compare relative effectiveness, but that comparisons could still be made to other benchmarks bearing in mind methodological differences.

QALYs were developed to evaluate the effectiveness of individual medical treatments, and EPA is still evaluating the appropriate methods for CEA of 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 nonhealth benefits, such as improved visibility.

The Institute of Medicine (a member institution of the National Academies of Science) has 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 is expected to produce a report by the beginning of 2006. In the interim, however, agencies are expected to provide CEAs for rules covered by Circular A-4 requirements.

In Appendix G of the RIA for the CAIR,[63] EPA conducted an extensive cost-effectiveness analysis using morbidity inclusive life years (MILY). That analysis concluded that reductions in PM2.5 associated with CAIR were expected to be cost-saving (because the value of expenditures on illnesses and non-health benefits exceeded costs), and that costs of the CAIR could have been significantly higher and still result in cost-effective improvements in public health. Because the current analysis relies on a benefits transfer approach to estimate PM-related benefits, scaling PM benefits from the CAND rule, we do not have the necessary inputs to develop a valid cost-effectiveness measure for the proposed cold temperature standards. Furthermore, the CAND analysis did not include a health-based CEA, the results of which might have been scaled in a similar fashion to the benefits.

For the CAVR rule, EPA was able to draw inferences from the CAIR CEA by scaling the relative magnitude of the costs and health impacts between the two rules.[68] While the CAVR was not expected to be cost-saving like CAIR, EPA expected that CAVR was likely to have a relatively low cost per MILY. For the proposed cold temperature standards, however, it is difficult to draw similar inferences with CAIR because the geographic distribution of emission changes, the distribution of those changes over time, and the age distribution of the mortality and chronic disease reductions are all expected to differ between the two rules. For these reasons, we do not scale the CAIR health-based cost-effectiveness analysis for the proposed cold temperature standards. We will, however, endeavor to conduct a formal health-based cost-effectiveness analysis for the final MSAT rule.

## 12.8 Comparison of Costs and Benefits

This proposed rule provides three separate provisions that reduce air toxics emissions from mobile sources: cold temperature vehicle controls, an emissions control program for gas cans, and a control program limiting benzene in gasoline. A full appreciation of the overall economic consequences of these provisions requires consideration of the benefits and costs expected to result from each standard, not just those that could be expressed here in dollar terms. As noted above, due to limitations in data availability and analytical methods, our benefits analysis only monetizes the $PM_{2.5}$-related benefits from direct PM emission reductions associated with the cold temperature standards. There are a number of health and environmental

effects associated with the proposed standards that we were unable to quantify or monetize (see Table 12.1-2).

Table 12.8-1 contains the estimates of monetized benefits of the proposed cold temperature vehicle standards and estimated social welfare costs for each of the proposed control programs.[N] The annual social welfare costs of all provisions of this proposed rule are described more fully in Chapter 13. It should be noted that the estimated social welfare costs for the vehicle program contained in this table are for 2019. The 2019 vehicle program costs are included for comparison purposes only and are therefore not included in the total 2020 social costs. There are no compliance costs associated with the vehicle program after 2019; as explained in Chapter 13, the vehicle compliance costs are primarily R&D and facilities costs that are expected to be recovered by manufacturers over the first ten years of the program.

The results in Table 12.8-1 suggest that the 2020 monetized benefits of the cold temperature vehicle standards are greater than the expected social welfare costs of that program in 2019. Specifically, the annual benefits of the program would be approximately $3,400 + B million or $3,100 + B million annually in 2020 (using a 3 percent and 7 percent discount rate in the benefits analysis, respectively), compared to estimated social welfare costs of approximately $11 million in the last year of the program (2019). These benefits are expected to increase to $6,500 + B million or $5,900 + B million annually in 2030 (using a 3 percent and 7 percent discount rate in the benefits analysis, respectively), even as the social welfare costs of that program fall to zero. Table 12.8-1 also presents the costs of the other proposed rule provisions: an emissions control program for gas cans and a control program limiting benzene in gasoline. Though we are unable to present the benefits associated with these two programs, we note for informational purposes that the benefits associated with the proposed cold temperature vehicle standards alone exceed the costs of all three proposed rule provisions combined.

[N] Social costs represent the welfare costs of the rule to society. These social costs do not consider transfer payments (such as taxes) that are simply redistributions of wealth.

**Table 12.8-1. Summary of Annual Benefits of the Proposed Cold Temperature Vehicle Standards and Costs of All Provisions of the Proposed Standards[a] (Millions of 2003 dollars)**

| Description | 2020 (Millions of 2003 dollars) | 2030 (Millions of 2003 dollars) |
|---|---|---|
| **Estimated Social Welfare Costs[b]** | | |
| **Proposed Cold Temperature Vehicle Standards** | $11[c] | $0 |
| **Proposed Gasoline Container Standards** | $32 | $39 |
| **Proposed Fuel Standards[d]** | $210 | $250 |
| **Total** | $240 | $290 |
| **Fuel Savings** | -$73 | -$82 |
| **Total Social Welfare Costs** | $170 | $205 |
| **Total $PM_{2.5}$-Related Health Benefits of the Proposed Cold Temperature Vehicle Standards[e]** | | |
| **3 percent discount rate** | $3,400 + B[f] | $6,500 + B[f] |
| **7 percent discount rate** | $3,100 + B[f] | $5,900 + B[f] |

[a] All estimates are rounded to two significant digits and represent annualized benefits and costs anticipated for the years 2020 and 2030, except where noted. Totals may not sum due to rounding.

[b] Note that costs are the annual total costs of reducing all pollutants associated with each provision of the proposed MSAT control package. Also note that while the cost analysis only utilizes a 7 percent discount rate to calculate annual costs, the benefits analysis uses both a 3 percent and 7 percent discount rate to calculate annual benefits. Benefits reflect only direct PM reductions associated with the cold temperature vehicle standards.

[c] These costs are for 2019; the vehicle program compliance costs terminate after 2019 and are included for illustrative purposes. They are not included in the total social welfare cost sum for 2020.

[d] Our modeling for the total costs of the proposed gasoline benzene program included California gasoline, since it was completed before we decided to propose that California gasoline not be covered by the program. California refineries comprise approximately 1 percent of these projected costs. For the final rule, we expect to exclude California refineries from the analysis.

[e] Valuation of premature mortality based on long-term PM exposure assumes discounting over the SAB recommended 20 year segmented lag structure described in the Regulatory Impact Analysis for the Final Clean Air Interstate Rule (March 2005). Annual benefits analysis results reflect the use of a 3 percent and 7 percent discount rate in the valuation of premature mortality and nonfatal myocardial infarctions, consistent with EPA and OMB guidelines for preparing economic analyses (US EPA, 2000 and OMB, 2003). [O]

[f] Not all possible benefits or disbenefits are quantified and monetized in this analysis. B is the sum of all unquantified benefits and disbenefits. Potential benefit categories that have not been quantified and monetized are listed in Table 12.1-2.

[O] U.S. Environmental Protection Agency, 2000. Guidelines for Preparing Economic Analyses. www.yosemite1.epa.gov/ee/epa/eed/hsf/pages/Guideline.html.

Office of Management and Budget, The Executive Office of the President, 2003. Circular A-4. http://www.whitehouse.gov/omb/circulars.

# Appendix 12A: Supplemental Analysis Addressing Uncertainties in the Benefits Analysis

## 12A.1 Introduction

The recent NAS report on estimating public health benefits of air pollution regulations recommended that EPA begin to move the assessment of uncertainties from its ancillary analyses into its primary analyses by conducting probabilistic, multiple-source uncertainty analyses. We present two approaches to generating probabilistic distributions designed to illustrate the potential influence of some aspects of the uncertainty in the C-R function in a PM benefits analysis. The first approach uses the results from a pilot expert elicitation designed to characterize certain aspects of uncertainty in the ambient $PM_{2.5}$/mortality relationship. We present the results of that analysis in the discussion of primary benefits associated with the proposed standards (see Chapter 12). The second approach generates a probabilistic estimate of statistical uncertainty based on standard errors reported in the underlying studies used in the benefit modeling framework, with particular emphasis on the health impact functions. In this appendix, we describe this second approach toward characterizing uncertainties in our economic benefits estimates.

It should be recognized that in addition to uncertainty, the annual benefits estimates for the proposed standards also are inherently variable, due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as hourly rate of emissions and daily weather display constant variability regardless of our ability to accurately measure them. As such, the primary estimates of annual benefits presented in this chapter and the sensitivity analysis estimates presented in this and other appendices should be viewed as representative of the types of benefits that will be realized, rather than the actual benefits that would occur every year. The distributions of the estimate of annual benefits should therefore be viewed as representative of the types of benefits that will be realized, rather than the actual benefits that would occur every year.

### 12A.1.1 General Approach

For the proposed standards, we did not attempt to assign probabilities to all of the uncertain parameters in the model because of a lack of resources and reliable methods. At this time, we simply generate estimates of the distributions of dollar benefits for PM health effects and for total dollar benefits. For all quantified PM endpoints, we scaled the likelihood distributions of the benefit estimates from the CAND uncertainty analysis,[P] based on the same benefits transfer approach we used to estimate the benefits of the proposed standards presented in Chapter 12. The CAND likelihood distributions were based solely on the statistical uncertainty surrounding the estimated C-R functions and the assumed distributions around the unit values.

[P] U.S. Environmental Protection Agency. May 2004. *Final Regulatory Analysis: Control of Emissions from Nonroad Diesel Engines*. Prepared by: Office of Air and Radiation. Available at http://www.epa.gov/nonroad-diesel/2004fr.htm#documents. Accessed December 15, 2005.

We use the benefits transfer approach to scale those distributions to reflect the predicted direct PM emission reductions of the proposed cold temperature standards. Though the scaling approach adds another element of uncertainty that we cannot characterize in the distributions, we believe the scaled uncertainty is a reasonable approximation of the statistical uncertainty based on standard errors reported in the underlying epidemiological and valuation studies.

Our scaled estimates of the likelihood distributions for health-related PM benefits should be viewed as incomplete because of the wide range of sources of uncertainty that we have not incorporated. The 5$^{th}$ and 95$^{th}$ percentile points of our scaled estimate are based on statistical error, and cross-study variability provides some insight into how uncertain our estimate is with regard to those sources of uncertainty. However, it does not capture other sources of uncertainty regarding the benefits transfer scaling approach or the inputs to the CAND modeling upon which the scaling is based, including emissions, air quality, baseline population incidence, and projected exposures. It also does not account for aspects of the health science not captured in the studies, such as the likelihood that PM is causally related to premature mortality and other serious health effects. Thus, a likelihood description based on the standard error would provide a misleading picture about the overall uncertainty in the estimates.

Both the uncertainty about incidence changes[Q] and uncertainty about unit dollar values can be characterized by *distributions*. Each "likelihood distribution" characterizes our beliefs about what the true value of an unknown variable (e.g., the true change in incidence of a given health effect in relation to PM exposure) is likely to be, based on the available information from relevant studies.[R] Unlike a sampling distribution (which describes the possible values that an *estimator* of an unknown variable might take on), this likelihood distribution describes our beliefs about what values the unknown variable itself might be. Such likelihood distributions can be constructed for each underlying unknown variable (such as a particular pollutant coefficient for a particular location) or for a function of several underlying unknown variables (such as the total dollar benefit of a regulation). In either case, a likelihood distribution is a characterization of our beliefs about what the unknown variable (or the function of unknown variables) is likely to be, based on all the available relevant information. A likelihood description based on such distributions is typically expressed as the interval from the 5$^{th}$ percentile point of the likelihood distribution to the 95$^{th}$ percentile point. If all uncertainty had been included, this range would be the "credible range" within which we believe the true value is likely to lie with 90 percent probability.

## 12A.2 Monte-Carlo Based Uncertainty Analysis

[Q] Because this is a national analysis in which, for each endpoint, a single C-R function is applied everywhere, there are two sources of uncertainty about incidence: statistical uncertainty (due to sampling error) about the true value of the pollutant coefficient in the location where the C-R function was estimated and uncertainty about how well any given pollutant coefficient approximates $\beta^*$.

[R] Although such a "likelihood distribution" is not formally a Bayesian posterior distribution, it is very similar in concept and function (see, for example, the discussion of the Bayesian approach in Kennedy (1990), pp. 168-172).

The uncertainty about the total dollar benefit associated with any single endpoint combines the uncertainties from these two sources (the C-R relationship and the valuation) and is estimated with a Monte Carlo method. In each iteration of the Monte Carlo procedure, a value is randomly drawn from the incidence distribution, another value is randomly drawn from the unit dollar value distribution; the total dollar benefit for that iteration is the product of the two.[S] When this is repeated for many (e.g., thousands of) iterations, the distribution of total dollar benefits associated with the endpoint is generated.

Using this Monte Carlo procedure, a distribution of dollar benefits can be generated for each endpoint. As the number of Monte Carlo draws gets larger and larger, the Monte Carlo-generated distribution becomes a better and better approximation of a joint likelihood distribution (for the considered parameters) making up the total monetary benefits for the endpoint.

After endpoint-specific distributions are generated, the same Monte Carlo procedure can then be used to combine the dollar benefits from different (nonoverlapping) endpoints to generate a distribution of total dollar benefits.

The estimate of total benefits may be thought of as the end result of a sequential process in which, at each step, the estimate of benefits from an additional source is added. Each time an estimate of dollar benefits from a new source (e.g., a new health endpoint) is added to the previous estimate of total dollar benefits, the estimated total dollar benefits increases. However, our bounding or likelihood description of where the true total value lies also increases as we add more sources.

As an example, consider the benefits from reductions in PM-related hospital admissions for cardiovascular disease. Because the actual dollar value is unknown, it may be described using a variable, with a distribution describing the possible values it might have. If this variable is denoted as X1, then the mean of the distribution, E(X1) and the variance of X1, denoted Var(X1), and the 5th and 95th percentile points of the distribution (related to Var(X1)), are ways to describe the likelihood for the true but unknown value for the benefits reduction.

Now suppose the benefits from reductions in PM-related hospital admissions for respiratory diseases are added. Like the benefits from reductions in PM-related hospital admissions for cardiovascular disease, the likelihood distribution for where we expect the true value to be may be considered a variable, with a distribution. Denoting this variable as X2, the benefits from reductions in the incidence of both types of hospital admissions is X1 + X2. This variable has a distribution with mean $E(X1 + X2) = E(X1) + E(X2)$, and a variance of $Var(X1 + X2) = Var(X1) + Var(X2) + 2Cov(X1,X2)$; if X1 and X2 are stochastically independent, then it has a variance of $Var(X1 + X2) = Var(X1) + Var(X2)$, and the covariance term is zero.

[S] This method assumes that the incidence change and the unit dollar value for an endpoint are stochastically independent.

The benefits from reductions in all nonoverlapping PM-related health and welfare endpoints are (Xm+1, ..., Xn) is X = X1 + ... + Xn. The mean of the distribution of total benefits, X, is

$$E(X) = E(X1) + E(X2) + ... + E(Xn)$$

and the variance of the distribution of total benefits—assuming that the components are stochastically independent of each other (i.e., no covariance between variables), is

$$Var(X) = Var(X1) + Var(X2) + ... + Var(Xn)$$

If all the means are positive, then each additional source of benefits increases the point estimate (mean) of total benefits. However, with the addition of each new source of benefits, the variance of the estimate of total benefits also increases. That is,

$$E(X1) < E(X1 + X2) < E(X1 + X2 + X3) < ... < E(X1 + ... + Xn) = E(X)$$

$$Var(X1) < Var(X1 + X2) < Var(X1 + X2 + X3) < ... < Var(X1 + ... + Xn) = Var(X)$$

That is, the addition of each new source of benefits results in a larger mean estimate of total benefits (as more and more sources of benefits are included in the total) about which there is less certainty. This phenomenon occurs whenever estimates of benefits are added.

Calculated with a Monte Carlo procedure, the distribution of X is composed of random draws from the components of X. In the first draw, a value is drawn from each of the distributions, X1, X2, through Xn; these values are summed; and the procedure is repeated again, with the number of repetitions set at a high enough value (e.g., 5,000) to reasonably trace out the distribution of X. The 5th percentile point of the distribution of X will be composed of points pulled from all points along the distributions of the individual components and not simply from the 5th percentile. Although the sum of the 5th percentiles of the components would be represented in the distribution of X generated by the Monte Carlo, it is likely that this value would occur at a significantly lower percentile. For a similar reason, the 95th percentile of X will be less than the sum of the 95th percentiles of the components, and instead the 95th percentile of X will be composed of component values that are significantly lower than the 95th percentiles.

The physical effects estimated in this analysis are assumed to occur independently. It is possible that, for any given pollution level, there is some correlation between the occurrence of physical effects, due to say avoidance behavior or common causal pathways and treatments (e.g., stroke, some kidney disease, and heart attack are related to treatable blood pressure). Estimating accurately any such correlation, however, is beyond the scope of this analysis, and instead it is simply assumed that the physical effects occur independently.

### 12A.2.1 Monte Carlo Analysis Using Classical Statistical Sources of Uncertainty

Based on the Monte Carlo techniques and benefits transfer methods described earlier, we scaled the CAND likelihood distributions for the dollar value of total PM health-related benefits for the proposed standards. For this analysis, the likelihood descriptions for the true value of each of the health endpoint incidence estimates, including premature mortality, were based on classical statistical uncertainty measures. The measures include the mean and standard deviation of the C-R relationships in the epidemiological literature, and assumptions of particular likelihood distribution shapes for the valuation of each health endpoint value based on reported values in the economic literature. The distributions for the value used to represent incidence of a health effect in the total benefits valuation represent both the simple statistical uncertainty surrounding individual effect estimates and, for those health endpoints with multiple effects from different epidemiology studies, interstudy variability. Distributions for unit dollar values are summarized in Chapter 12, Table 12-7.

Results of the scaled Monte Carlo simulations are presented in Table 12A-1. The table provides the scaled means of the distributions and the estimated 5th and 95th percentiles of the distributions. The contribution of mortality to the mean benefits and to both the 5th and 95th percentiles of total benefits is substantial, with mortality accounting for over 90 percent of the mean estimate, and even the 5th percentile of mortality benefits dominating close to the 95th percentile of all other benefit categories. Thus, the choice of value and the shape for likelihood distribution for VSL should be examined closely and is key information to provide to decision makers for any decision involving this variable. The 95th percentile of total benefits is approximately twice the mean, while the 5th percentile is approximately one-fourth of the mean. The overall range from 5th to 95th represents about one order of magnitude.

## Table 12A-1. Distribution of Value of Annual PM-Related Human Health Benefits in 2030 for the Proposed Mobile Source Air Toxics Rule: Cold Temperature Controls [a]

| Endpoint | Monetary Benefits[b, c] (Millions 2003$, Adjusted for Income Growth) | | |
|---|---|---|---|
| | 5th Percentile | Mean | 95th Percentile |
| Premature mortality[c], Long-term exposure | | | |
| Adults, 30+ yrs and Infants, <1yr | | | |
| 3% Discount Rate | $1,400 | $6,000 | $12,000 |
| 7% Discount Rate | $1,300 | $5,400 | $11,000 |
| Chronic bronchitis (adults, 26 and over) | $13 | $270 | $910 |
| Nonfatal myocardial infarctions | | | |
| 3% Discount Rate | $33 | $150 | $340 |
| 7% Discount Rate | $31 | $150 | $340 |
| Hospital admissions from respiratory causes | $3.2 | $10 | $17 |
| Hospital admissions from cardiovascular causes | $5.5 | $9.4 | $14 |
| Emergency room visits for asthma | $0.12 | $0.21 | $0.31 |
| Acute bronchitis (children, aged 8–12) | ($0.017) | $0.58 | $1.4 |
| Lower respiratory symptoms (children, aged 7–14) | $0.12 | $0.30 | $0.56 |
| Upper respiratory symptoms (asthmatic children, aged 9–11) | $0.091 | $0.37 | $0.81 |
| Asthma exacerbations | $0.014 | $1.0 | $2.9 |
| Work loss days (adults, aged 18–65) | $12 | $14 | $16 |
| Minor restricted-activity days (adults, aged 18–65) | $21 | $36 | $52 |
| Monetized Total[d] | | | |
| 3% Discount Rate | $1,500 + B | $6,500 + B | $13,000 + B |
| 7% Discount Rate | $1,400 + B | $5,900 + B | $12,000 + B |

[a] Monetary benefits are rounded to two significant digits.

[b] Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and 2030.

[c] Results show 3 percent and 7 percent discount rates consistent with EPA and OMB guidelines for preparing economic analyses (EPA, 2000; OMB, 2003).

[d] B represents the monetary value of the nonmonetized health and welfare benefits. A detailed listing of unquantified PM-, ozone-, and air toxics-related health effects is provided in Chapter 12, Table 12-2.

# Appendix 12B: Sensitivity Analyses of Key Parameters in the Benefits Analysis

The primary analysis presented in Chapter 12 is based on our current interpretation of the scientific and economic literature. That interpretation requires judgments regarding the best available data, models, and modeling methodologies and the assumptions that are most appropriate to adopt in the face of important uncertainties and resource limitations. The majority of the analytical assumptions used to develop the primary estimates of benefits have been used to support similar rulemakings and approved by EPA's Science Advisory Board (SAB). Both EPA and the SAB recognize that data and modeling limitations as well as simplifying assumptions can introduce significant uncertainty into the benefit results and that alternative choices exist for some inputs to the analysis, such as the mortality C-R functions.

This appendix supplements our primary estimates of benefits with a series of sensitivity calculations that use other sources of health effect estimates and valuation data for key benefits categories. It should be noted, however, that these supplemental estimates have been scaled from results of the Clean Air Nonroad Diesel (CAND) supplemental sensitivity analysis using the same benefits transfer approach we used to estimate the benefits of the proposed standards presented in Chapter 12. Though the scaling approach adds another element of uncertainty that we cannot characterize in the sensitivity analyses, we believe the scaled results of the supplemental estimates presented here are a reasonable approximation of the primary estimates' sensitivity to the assumptions and judgments used in the benefits analysis.

The supplemental estimates examine sensitivity to both valuation issues (e.g., the form of the lag structure for PM-related premature mortality) and for physical effects issues (e.g., the effect of thresholds on PM-related premature mortality). These supplemental estimates are not meant to be comprehensive. Rather, they reflect some of the key issues identified by EPA or commentors as likely to have a significant impact on total benefits. The individual adjustments in the tables should not simply be added together because: 1) there may be overlap among the alternative assumptions; and 2) the joint probability among certain sets of alternative assumptions may be low.

## 12B.1 Premature Mortality - Long-Term Exposure

Reduction in the risk of premature mortality is the most important PM-related health outcome in terms of contribution to dollar benefits in the analysis for this rule. There are at least three important analytical assumptions that may significantly impact the estimates of the number and valuation of avoided premature mortalities. These include selection of the C-R function, structure of the lag between reduced exposure and reduced mortality risk, and effect thresholds. Results of this set of sensitivity analyses are presented in Table 12B-1.

**Table 12B-1. Sensitivity of Benefits of Premature Mortality Reductions to Alternative Assumptions (Relative to Primary Benefits Estimates of the Proposed Standards)**

| Description of Sensitivity Analysis | | Avoided Incidences 2020 | Avoided Incidences 2030 | Value (million 2003$)[b] 2020 | Value (million 2003$)[b] 2030 |
|---|---|---|---|---|---|
| *Alternative Concentration-Response Functions for PM-Related Premature Mortality* | | | | | |
| Pope/ACS Study (2002)[c] | | | | | |
| | Lung Cancer | 70 | 140 | $470 | $910 |
| | Cardiopulmonary | 370 | 720 | $2,400 | $4,800 |
| Krewski/Harvard Six-Cities Study | | 1,050 | 2,000 | $6,700 | $13,000 |
| *Alternative Lag Structures for PM-Related Premature Mortality* | | | | | |
| None | Incidences all occur in the first year | 480 | 910 | $3,400 | $6,600 |
| 8-year | Incidences all occur in the 8th year | | | | |
| | 3% Discount Rate | 480 | 910 | $2,800 | $5,400 |
| | 7% Discount Rate | 480 | 910 | $2,100 | $4,100 |
| 15-year | Incidences all occur in the 15th year | | | | |
| | 3% Discount Rate | 480 | 910 | $2,300 | $4,400 |
| | 7% Discount Rate | 480 | 910 | $1,300 | $2,600 |
| Alternative Segmented | 20 percent of incidences occur in 1st year, 50 percent in years 2 to 5, and 30 percent in years 6 to 20 | | | | |
| | 3% Discount Rate | 480 | 910 | $3,000 | $5,800 |
| | 7% Discount Rate | 480 | 910 | $2,600 | $5,000 |
| 5-Year Distributed | 50 percent of incidences occur in years 1 and 2 and 50 percent in years 2 to 5 | | | | |
| | 3% Discount Rate | 480 | 910 | $3,200 | $6,300 |
| | 7% Discount Rate | 480 | 910 | $3,000 | $5,900 |

[a] Incidences rounded to two significant digits.

[b] Dollar values rounded to two significant digits. Note that dollar values reflect the use of a 3 percent discount rate in the lag adjustment for mortality valuation for the alternative C-R function and alternative threshold analyses. The alternative lag structure analysis presents benefits calculated using both a 3 percent and 7 percent discount rate.

[c] Note that the sum of lung cancer and cardiopulmonary deaths will not be equal to the total all-cause death estimate. Some residual mortality is associated with long-term exposures to $PM_{2.5}$ that is not captured by the cardiopulmonary and lung cancer categories.

### 12B.1.1 Alternative C-R Functions

Following the advice of the most recent EPA SAB-Health Effects Subcommittee (HES), we used the Pope et al. (2002)[69] all-cause mortality model to derive our primary estimate of avoided premature mortality (EPA-SAB-COUNCIL-ADV-04-002, 2004).[70] While the SAB-HES "recommends that the base case rely on the Pope et al. (2002) study and that EPA use total mortality concentration-response functions (C-R), rather than separate cause-specific C-R functions, to calculate total PM mortality cases," (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.2) they also suggested that "the cause-specific estimates can be used to communicate the relative contribution of the main air pollution related causes of death." (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.18) As such, in Table B-1 we provide the scaled estimates of cardiopulmonary and lung cancer deaths based on the Pope et al. (2002) study.

In addition, the SAB-HES noted that the ACS cohort used in Pope et al. (2002) "has some inherent deficiencies, in particular the imprecise exposure data, and the nonrepresentative (albeit very large) population" (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.18). The SAB-HES suggests that while not necessarily a better study, the ACS is a prudent choice for the primary estimate. They go on to note that "the Harvard Six-Cities C-R functions are valid estimates on a more representative, although geographically selected, population, and its updated analysis has not yet been published. The Six-Cities estimates may be used in a sensitivity analysis to demonstrate that, with different but also plausible selection criteria for C-R functions, benefits may be considerably larger than suggested by the ACS study," (EPA-SAB-COUNCIL-ADV-04-002, 2004, p.18).

In previous advice, the SAB has noted that "the [Harvard Six-Cities] study had better monitoring with less measurement error than did most other studies," (EPA-SAB-COUNCIL-ADV-99-012, 1999).[71] The demographics of the ACS study population (i.e., largely white and middle to upper middle-class) may also produce a downward bias in the estimated PM mortality coefficient, because a variety of analyses indicate that the effects of PM tend to be significantly greater among groups of lower socioeconomic status (Krewski et al., 2000),[72] although the cause of this difference has not been identified. The Harvard Six-Cities study also covered a broader age category (25 and older compared to 30 and older in the ACS study). We emphasize that, based on our understanding of the relative merits of the two datasets, the Pope et al. (2002) ACS model based on mean $PM_{2.5}$ levels in 63 cities is the most appropriate model for analyzing premature mortality impacts. Thus it is used for our primary estimate of this important health effect.

### 12B.1.2 Alternative Lag Structures

Over the last ten years, there has been a continuing discussion and evolving advice regarding the timing of changes in health effects following changes in ambient air pollution. It has been hypothesized that some reductions in premature mortality from exposure to ambient $PM_{2.5}$ will occur over short periods of time in individuals with compromised health status, but other effects are likely to occur among individuals who, at baseline, have reasonably good health that will deteriorate because of continued exposure. No animal models have yet been developed to quantify these cumulative effects, nor are there epidemiologic studies bearing on this question.

The SAB-HES has recognized this lack of direct evidence. However, in early advice, they also note that "although there is substantial evidence that a portion of the mortality effect of PM is manifest within a short period of time, i.e., less than one year, it can be argued that, if no lag assumption is made, the entire mortality excess observed in the cohort studies will be analyzed as immediate effects, and this will result in an overestimate of the health benefits of improved air quality. Thus some time lag is appropriate for distributing the cumulative mortality effect of PM in the population," (EPA-SAB-COUNCIL-ADV-00-001, 1999, p. 9).[73] In recent advice, the SAB-HES suggests that appropriate lag structures may be developed based on the distribution of cause-specific deaths within the overall all-cause estimate (EPA-SAB-COUNCIL-ADV-04-002, 2004). They suggest that diseases with longer progressions should be

characterized by longer-term lag structures, while air pollution impacts occurring in populations with existing disease may be characterized by shorter-term lags.

A key question is the distribution of causes of death within the relatively broad categories analyzed in the long-term cohort studies. Although it may be reasonable to assume the cessation lag for lung cancer deaths mirrors the long latency of the disease, it is not at all clear what the appropriate lag structure should be for cardiopulmonary deaths, which include both respiratory and cardiovascular causes. Some respiratory diseases may have a long period of progression, while others, such as pneumonia, have a very short duration. In the case of cardiovascular disease, there is an important question of whether air pollution is causing the disease, which would imply a relatively long cessation lag, or whether air pollution is causing premature death in individuals with preexisting heart disease, which would imply very short cessation lags.

The SAB-HES provides several recommendations for future research that could support the development of defensible lag structures, including using disease-specific lag models and constructing a segmented lag distribution to combine differential lags across causes of death (EPA-SAB-COUNCIL-ADV-04-002, 2004). The SAB-HES indicated support for using "a Weibull distribution or a simpler distributional form made up of several segments to cover the response mechanisms outlined above, given our lack of knowledge on the specific form of the distributions," (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24). However, they noted that "an important question to be resolved is what the relative magnitudes of these segments should be, and how many of the acute effects are assumed to be included in the cohort effect estimate," (EPA-SAB-COUNCIL-ADV-04-002, 2004, p. 24-25). Since the publication of that report in March 2004, EPA has sought additional clarification from this committee. In its follow-up advice provided in December 2004, the SAB suggested that until additional research has been completed, EPA should assume a segmented lag structure characterized by 30 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 20 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$ (EPA-COUNCIL-LTR-05-001, 2004).[74] The distribution of deaths over the latency period is intended to reflect the contribution of short-term exposures in the first year, cardiopulmonary deaths in the 2- to 5-year period, and long-term lung disease and lung cancer in the 6- to 20-year period. Furthermore, in their advisory letter, the SAB-HES recommended that EPA include sensitivity analyses on other possible lag structures. In this appendix, we investigate the sensitivity of premature mortality-reduction related benefits to alternative cessation lag structures, noting that ongoing and future research may result in changes to the lag structure used for the primary analysis.

In previous advice from the SAB-HES, they recommended an analysis of 0-, 8-, and 15-year lags, as well as variations on the proportions of mortality allocated to each segment in the segmented lag structure (EPA-SAB-COUNCIL-ADV-00-001, 1999, (EPA-COUNCIL-LTR-05-001, 2004). The 0-year lag is representative of EPA's assumption in previous RIAs. The 8- and 15-year lags are based on the study periods from the Pope et al.

(1995)[75] and Dockery et al. (1993)[76] studies, respectively.[T] However, neither the Pope et al. nor Dockery et al. studies assumed any lag structure when estimating the relative risks from PM exposure. In fact, the Pope et al. and Dockery et al. analyses do not support or refute the existence of a lag. Therefore, any lag structure applied to the avoided incidences estimated from either of these studies will be an assumed structure. The 8- and 15-year lags implicitly assume that all premature mortalities occur at the end of the study periods (i.e., at 8 and 15 years).

In addition to the simple 8- and 15-year lags, we have added two additional sensitivity analyses examining the impact of assuming different allocations of mortality to the segmented lag of the type suggested by the SAB-HES. The first sensitivity analysis assumes that more of the mortality impact is associated with chronic lung diseases or lung cancer and less with acute cardiopulmonary causes. This illustrative lag structure is characterized by 20 percent of mortality reductions occurring in the first year, 50 percent occurring evenly over years 2 to 5 after the reduction in $PM_{2.5}$, and 30 percent occurring evenly over the years 6 to 20 after the reduction in $PM_{2.5}$. The second sensitivity analysis assumes the 5-year distributed lag structure used in previous analyses, which is equivalent to a three-segment lag structure with 50 percent in the first 2-year segment, 50 percent in the second 3-year segment, and 0 percent in the 6- to 20-year segment.

The estimated impacts (scaled from the CAND analysis) of alternative lag structures on the monetary benefits associated with reductions in PM-related premature mortality (estimated with the Pope et al. ACS impact function) are presented in Table B-1. These estimates are based on the value of statistical lives saved approach (i.e., $5.5 million per incidence) and are presented using both a 3 percent and 7 percent discount rate over the lag period.

## 12B.2 Summary of Results

The results of these scaled sensitivity analyses demonstrate that choice of effect estimate can have a large impact on benefits, potentially doubling benefits if the effect estimate is derived from the HEI reanalysis of the Harvard Six-Cities data ( Krewski et al., 2000). Because of discounting of delayed benefits, the lag structure may also have a large downward impact on monetized benefits if an extreme assumption that no effects occur until after 15 years is applied. However, for most reasonable distributed lag structures, differences in the specific shape of the lag function have relatively small impacts on overall benefits. For example, the overall impact of moving from the previous 5-year distributed lag to the segmented lag recommended by the SAB-HES in 2004 in the 2030 primary estimate is relatively modest, reducing benefits by approximately 5 percent when a 3 percent discount rate is used and approximately 10 percent when a 7 percent discount rate is used. If no lag is assumed, benefits increase by around 10 percent relative to the segmented lag with a 3 percent discount rate and 23 percent with a 7 percent discount rate.

[T]Although these studies were conducted for 8 and 15 years, respectively, the choice of the duration of the study by the authors was not likely due to observations of a lag in effects but is more likely due to the expense of conducting long-term exposure studies or the amount of satisfactory data that could be collected during this time period.

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