Chapter VII: Benefit-Cost Analysis

This chapter reports EPA's analysis of the economic benefits of the final HD Engine/Diesel Fuel rule. EPA is required by Executive Order 12866 to estimate the benefits 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 air quality resulting from reductions in nitrogen oxides (NOx), sulfur dioxide (SO_2), non-methane hydrocarbons (NMHC), carbon monoxide (CO) and direct diesel particulate matter (PM) emissions?; 2) how much are the changes in air quality worth to U.S. citizens as a whole in monetary terms?; and 3) how do the benefits compare to the costs? It constitutes one part of EPA's thorough examination of the relative merits of this regulation.

The benefit-cost analysis that we performed for our final rule can be thought of as having four parts, each of which will be discussed separately in the Sections that follow. These four steps are:

1. Calculation of the impact that our standards will have on the nationwide inventories for NOx, non-methane hydrocarbons (NMHC), SO_2, and PM emissions;

2. Air quality modeling to determine the changes in ambient concentrations of ozone and PM that will result from the changes in nationwide inventories of precursor pollutants;

3. A benefits analysis to determine the changes in human health and welfare, both in terms of physical effects and monetary value, that result from the changes in ambient concentrations of various pollutants; and

4. Comparison of the costs of the standards to the monetized benefits.

It is important to note that there are significant categories of benefits which can not be monetized (or in many cases even quantified), resulting in a significant limitation to this analysis. Also, EPA currently does not have appropriate tools for modeling changes in ambient concentrations of CO or air toxics for input into a national benefits analysis. They have been linked to numerous health effects; however, we are unable to quantify the CO- or air toxics-related health or welfare benefits of the HD Engine/Diesel Fuel rule at this time.

EPA has used the best available information and tools of analysis to quantify the expected changes in public health, environmental and economic benefits of the final HD Engine/Diesel Fuel rule, given the constraints on time and resources available for the analysis. In general, we

follow the same general methodology used in the benefits analysis of the Tier 2/Gasoline Sulfur rulemaking. However, we have updated some aspects of the analysis in response to public comment and to reflect advances in modeling and the literature for economics and health effects. EPA also relies heavily on the advice of its independent Science Advisory Board (SAB) in determining the health and welfare effects considered in the benefits analysis and in establishing the most scientifically valid measurement and valuation techniques. Since the publication of the final Tier 2/Gasoline Sulfur RIA, we have updated some of the assumptions and methods used in our analysis to reflect SAB recommendations. Changes to the methodology include the following:

- Using Regulatory Model System for Aerosols and Deposition (REMSAD) to model baseline and post-control ambient particulate matter;
- Updating concentration-response (C-R) functions for PM-related premature mortality;
- Updating C-R functions for PM-related hospital admissions;
- Presenting chronic asthma as an alternative calculation;
- Reporting asthma attacks as a separate endpoint and adjusting minor restricted activity days to remove the possibility of double-counting of asthma attacks;
- Relying only on the value of statistical life method to value reductions in the risk of premature mortality in the primary estimate; and
- Adjusting benefits to reflect the expected growth in willingness-to-pay (WTP) for health and environmental benefits as real income grows over time.

All of these changes are expected to improve the quality of the benefits estimation. These changes reflect the latest peer-reviewed scientific literature and most of the improvements have been approved by the SAB in its review of EPA methods in other analyses. A detailed discussion of each change is included in the body of this chapter.

We have attempted to be as clear as possible in presenting our assumptions, sources of data, and sources of potential uncertainty in the analysis. We urge the reader to pay particular attention to the fact that not all the benefits of the rule can be estimated with sufficient reliability to be quantified and included in monetary terms. Some welfare endpoints, for instance, are quantified to some extent but no dollar value can be reliably assigned. The omission of these items from the total of monetary benefits reflects our inability to measure them. It does not

indicate their lack of importance in the consideration of the benefits of this rulemaking. When it is possible to qualitatively characterize a benefits category, we provide a discussion, although the benefit is not included in the estimate of total benefits.

We use the term *benefits* to refer to any and all positive effects of emissions changes on social welfare that we expect to result from the final rule. We use the term environmental costs (also commonly referred to as "disbenefits") to refer to any and all negative effects of emissions changes on social welfare that result from the final rule. We include both benefits and environmental costs in this analysis. Where it is possible to quantify benefits and environmental costs, our measures are those associated with economic surplus in accepted applications of welfare economics. They measure the value of changes in air quality by estimating (primarily through benefits transfer) the willingness of the affected population to pay for changes in environmental quality and associated health and welfare effects.

This analysis presents estimates of the potential benefits from the HD Engine/Diesel Fuel rule occurring in 2030. The predicted emissions reductions that will result from the rule have yet to occur, and therefore the actual changes in human health and welfare outcomes to which economic values are ascribed are predictions. These predictions are based on the best available scientific evidence and judgment, but there is unavoidable uncertainty associated with each step in the complex process between regulation and specific health and welfare outcomes. The ways in which we deal with these uncertainties are discussed in Section C.

Figure VII-1 illustrates the steps necessary to link the HD Engine/Diesel Fuel rule with economic measures of benefits. The first two steps involve the specification and implementation of the regulation. First, the specific standards for reducing air pollution from heavy duty vehicles and fuels are established. Next, the necessary changes in vehicle technology and fuels are determined (see Chapters IV and V). The changes in pollutant emissions resulting from the required vehicle and fuel changes are then calculated, along with predictions of emissions for other industrial sectors in the baseline. The predicted emissions described in Chapter III are then used as inputs to air quality models that predict ambient concentrations of pollutants over time and space. These concentrations depend on climatic conditions and complex chemical interactions. We have used the best available air quality models to estimate the changes in ambient concentrations (from baseline levels) used as the basis for this benefits analysis.

Changes in ambient concentrations will lead to new levels of environmental quality in the U.S., reflected both in human health and in non-health welfare effects. Thus, the predicted changes in ambient air quality serve as inputs into functions that predict changes in health and welfare outcomes. We use the term "endpoints" to refer to specific effects that can be associated with changes in air quality. Table VII-1 lists the human health and welfare effects identified for

changes in air quality as they related to ozone, PM, CO, and NMHC.[a] This list includes both those effects quantified (and/or monetized) in this analysis and those for which we are unable to provide quantified estimates. All of the effects related to changes in CO and NMHC are not directly quantified for this analysis due to a lack of appropriate air quality models for these pollutants. For changes in risks to human health from changes in ozone and PM, quantified endpoints include changes in mortality and in a number of pollution-related non-fatal health effects. To estimate these endpoints, EPA combines changes in ambient air quality levels with epidemiological evidence about population health response to pollution exposure. For welfare effects, the endpoints are defined in terms of levels of physical damage (for materials damage), economic output for (agriculture and forestry), light transmission (for visibility), and increases in terrestrial and estuarine nutrient loading (for ecological effects).

As with emissions and air quality estimates, EPA's estimates of the effect of ambient pollution levels on all of these endpoints represent the best science available. The majority of the analytical assumptions used to develop our estimates have been reviewed and approved by the SAB. However, like all estimates, they also contain unavoidable uncertainty, as does any prediction of the future. In Section C and its subsections on health and welfare endpoints, this uncertainty is discussed and characterized.

This chapter proceeds as follows: in Sections A and B, we summarize emissions and air quality results and discuss the way emissions and air quality changes are used as inputs to the benefits analysis. In Section C, we introduce the categories of benefits that are estimated, present the techniques that are used, and provide a discussion of how we incorporate uncertainty into our analysis. In Section D, we describe individual health effects and report the results of the analysis for human health effects. In Section E, we describe welfare effects and report the results of the analysis for welfare effects. In Section F, we report our estimates of total monetized benefits and alternative calculations of benefits. Finally, in Section G, we summarize annual cost results and in Section H, we present a comparison of monetized benefits and costs.

[a] The NMHC listed in Table VII-1 are also listed as hazardous air pollutants in the Clean Air Act. We are not able to quantify their direct effects. To the extent that they are precursors to ozone or PM, they are included in our quantitative results.

Figure VII-1. Steps in the Heavy Duty Engine/Diesel Fuel Benefits Analysis

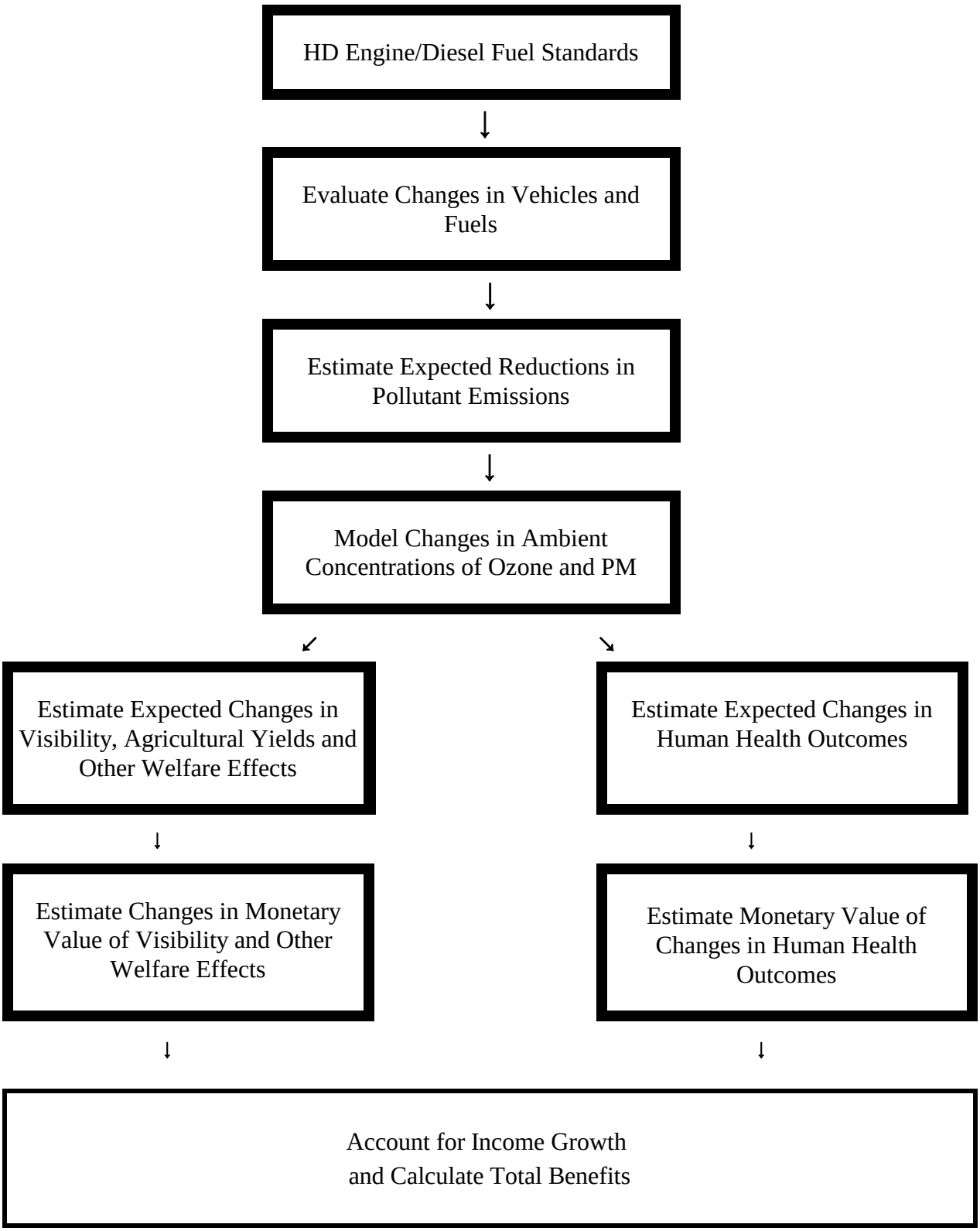

Table VII-1. Human Health and Welfare Effects of Pollutants Affected by the HD Engine/Diesel Fuel Rule

Pollutant/Effect	Primary Quantified and Monetized Effects[A]	Alternative Quantified and/or Monetized Effects[B]	Unquantified Effects
Ozone/Health	Minor restricted activity days Hospital admissions - respiratory and cardiovascular Emergency room visits for asthma Asthma attacks	Chronic Asthma[C]	Premature mortality[D] Increased airway responsiveness to stimuli Inflammation in the lung Chronic respiratory damage Premature aging of the lungs Acute inflammation and respiratory cell damage Increased susceptibility to respiratory infection Non-asthma respiratory emergency room visits
Ozone/Welfare	Decreased worker productivity Decreased yields for commercial crops (selected species) Decreased Eastern commercial forest productivity (selected species)		Decreased Western commercial forest productivity Decreased Eastern commercial forest productivity (other species) Decreased yields for fruits and vegetables Decreased yields for other commercial and non-commercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Damage to ecosystem functions
PM/Health	Premature mortality Bronchitis - chronic and acute Hospital admissions - respiratory and cardiovascular Emergency room visits for asthma Asthma attacks Lower and upper respiratory illness Minor restricted activity days Work loss days		Infant mortality Low birth weight Changes in pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Cancer Non-asthma respiratory emergency room visits

Pollutant/Effect	Primary Quantified and Monetized Effects[A]	Alternative Quantified and/or Monetized Effects[B]	Unquantified Effects
PM/Welfare	Visibility in California, Southwestern, and Southeastern Class I areas	Visibility in Northeastern, Northwestern, and Midwestern Class I areas Visibility in residential and non-Class I areas Household soiling	
Nitrogen and Sulfate Deposition/ Welfare		Costs of nitrogen controls to reduce eutrophication in selected eastern estuaries	Impacts of acidic sulfate and nitrate deposition on commercial forests Impacts of acidic deposition on commercial freshwater fishing Impacts of acidic deposition on recreation in terrestrial ecosystems Impacts of nitrogen deposition on commercial fishing, agriculture, and forests Impacts of nitrogen deposition on recreation in estuarine ecosystems Reduced existence values for currently healthy ecosystems
SO_2/Health			Hospital admissions for respiratory and cardiac diseases Respiratory symptoms in asthmatics
NOx/Health			Lung irritation Lowered resistance to respiratory infection Hospital Admissions for respiratory and cardiac diseases

Pollutant/Effect	Primary Quantified and Monetized Effects[A]	Alternative Quantified and/or Monetized Effects[B]	Unquantified Effects
CO/Health			Premature mortality[B] Behavioral effects Hospital admissions - respiratory, cardiovascular, and other Other cardiovascular effects Developmental effects Decreased time to onset of angina Non-asthma respiratory ER visits
NMHCs [E] Health			Cancer (diesel PM, benzene, 1,3-butadiene, formaldehyde, acetaldehyde) Anemia (benzene) Disruption of production of blood components (benzene) Reduction in the number of blood platelets (benzene) Excessive bone marrow formation (benzene) Depression of lymphocyte counts (benzene) Reproductive and developmental effects (1,3-butadiene) Irritation of eyes and mucous membranes (formaldehyde) Respiratory and respiratory tract Asthma attacks in asthmatics (formaldehyde) Asthma-like symptoms in non-asthmatics (formaldehyde) Irritation of the eyes, skin, and respiratory tract (acetaldehyde) Upper respiratory tract irritation & congestion (acrolein)

Pollutant/Effect	Primary Quantified and Monetized Effects[A]	Alternative Quantified and/or Monetized Effects[B]	Unquantified Effects
NMHCs [E] **Welfare**			Direct toxic effects to animals Bioaccumlation in the food chain

[A] Primary quantified and monetized effects are those included when determining the primary estimate of total monetized benefits of the HD Engine/Diesel Fuel rule. See Section C-2 for a more complete discussion of presentation of benefits estimates.

[B] Alternative quantified and/or monetized effects are those presented as alternatives to the primary estimates or in addition to the primary estimates, but not included in the primary estimate of total monetized benefits.

[C] While no causal mechanism has been identified linking new incidences of chronic asthma to ozone exposure, an epidemiological study shows a statistical association between long-term exposure to ozone and incidences of chronic asthma in some non-smoking men (McDonnell, et al., 1999).

[D] Premature mortality associated with ozone is not separately included in this analysis. It is assumed that the American Cancer Society (ACS)/ Krewski, et al., 2000 C-R function we use for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants (ACS/ Krewski, et al., 2000).

[E] All non-methane hydrocarbons (NMHCs) listed in the table are also hazardous air pollutants listed in the Clean Air Act.

A. Emissions Inventory Implications

This section explains why 2030 emission inventories were developed and what the possible implications are for this benefit-cost analysis of uncertainties associated with the inventories. The national inventories for NOx, NMHC, SO_2, and PM have already been presented and discussed in Chapter III and in the supporting documents referenced in that chapter. Interested readers desiring more information about the inventory methodologies or results should consult that chapter for details.

The HD Engine/Diesel Fuel program has various cost and emission related components, as described earlier in this RIA. These components would begin at various times and in some cases would phase in over time. This means that during the early years of the program there would not be a consistent match between cost and benefits. This is especially true for the vehicle control portions and initial fuel changes required by the program, where the full vehicle cost would be incurred at the time of vehicle purchase, while the fuel cost along with the emission reductions and benefits resulting from all these costs would occur throughout the lifetime of the vehicle. Because of this inconsistency and our desire to more appropriately match the costs and emission reductions of our program, our analysis uses a future year when the fleet is nearly fully turned over (2030). Consequently, we developed emission inventories for 2030 baseline conditions and a 2030 HD Engine/Diesel Fuel control scenario.

In the years before 2030, the benefits from the HD Engine/Diesel Fuel program will be less than those estimated here, because the compliant heavy-duty fleet will not be fully phased in. Moreover, to the extent that a lower ratio of benefits to costs early in the program is the result of the mismatch of costs and benefits in time, a simple analysis of an individual year would be misleading. A more appropriate means of capturing the impacts of timing differences in benefits and costs would be to produce a net present value comparison of the costs and benefits over some period of years. Unfortunately, while this is relatively straight-forward for the costs, it is currently not feasible to do a multi-year analysis of the benefits as this would require a significant amount of air quality modeling to capture each year. We did not have the resources for such an extensive analysis. Instead, for the purpose of the benefit calculations, we assume that 2030 is a representative year for the fully implemented rule to consider in comparison with the costs. The resulting analysis represents a snapshot of benefits and costs in a future year in which the heavy duty fleet consists almost entirely of vehicles and fuels meeting the HD Engine/Diesel Fuel standards.

In addition, there is uncertainty in any prediction, and the emissions inventory growth factors can add uncertainty because they are applied for a 30-year period and propagate through the entire analysis. This uncertainty may be more important for welfare effects such as ozone-related crop damage where the predicted 2030 baseline may be an important factor. These

exposure metrics for crop damage and forestry impacts are a cumulative measure above a certain level (i.e., 0.06 ppm). Thus, the accuracy of the emissions inventory growth rates can affect the magnitude of the benefits (For discussion see Section E). This is less of an issue for exposure metrics that rely on changes in air quality (e.g., the health endpoints). Nevertheless, the inventory is a crucial building block on which the analysis rests.

B. Air Quality Impacts

This section summarizes the methods for and results of estimating air quality for the 2030 base case and HD Engine/Diesel Fuel control scenario for the purposes of benefit-cost analyses. EPA has focused on the health, welfare, and ecological effects that have been linked to air quality changes. These air quality changes include the following:

- Ambient ozone–as estimated using a regional-scale version of the Urban Airshed Model-Variable Grid (UAM-V);
- Ambient particulate matter (PM_{10} and $PM_{2.5}$)–as estimated using a national-scale version of the Regulatory Modeling System for Aerosols and Deposition (REMSAD);
- Visibility degradation (i.e., regional haze), as developed using empirical estimates of light extinction coefficients and efficiencies in combination with REMSAD modeled reductions in pollutant concentrations; and
- Airborne nitrogen deposition to estuaries–as predicted using local and regional coefficients of nitrogen deposition for selected estuaries from the Regional Acid Deposition Model (RADM) in combination with modeled reductions in NOx emissions.

The air quality estimates in this section are based on the emission changes discussed in Chapter III. These air quality results are in turn associated with human populations and ecosystems to estimate changes in health and welfare effects.

In Section B-1, we describe the estimation of ozone air quality using UAM-V, and in Section B-2, we cover the estimation of PM air quality using REMSAD. In Section B-3, we discuss the estimation of visibility degradation. Lastly, in Section B-4 we describe the estimation of nitrogen deposition.

1. Ozone Air Quality Estimates

We use the emissions inputs described in Section A with a regional-scale version of UAM-V to estimate ozone air quality in the Eastern U.S. UAM-V is an Eulerian three-dimensional grid photochemical air quality model designed to calculate the concentrations of both inert and chemically reactive pollutants by simulating the physical and chemical processes in the atmosphere that affect ozone formation. Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, the UAM-V is useful for evaluating the impacts of the HD Engine/Diesel Fuel rule on U.S. ozone concentrations. As described fully in the air quality technical support document, the model performance in the Western U.S. was not acceptable for including those results as inputs to the benefits analysis (US EPA, 2000). Comparisons of base year model output data against ambient observations in the Western U.S. indicated that the model was significantly underestimating (by 30-50 percent) the observed levels of ozone in most areas of the West. Given that model performance was degraded to the extent that the directional response of the model to controls may be questionable, it was determined that this application of the model should not be used in assessing the impacts of the emissions control strategy in the Western U.S.

Thus, our analysis applies the modeling system to the Eastern U.S. for a base-year of 1996 and for two future-year scenarios: a 2030 base case and a 2030 HD Engine/Diesel Fuel control scenario. As discussed in the technical support document, we use the two separate years because the relative model predictions are used with ambient air quality observations from 1996 to determine the expected change in 2030 ozone concentrations due to the rule (Abt Associates, 2000).

The UAM-V modeling system requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, day-specific emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. As applied to the Eastern region of the continental U.S., the model segments the area within the region into square blocks called grids (roughly equal in size to counties), each of which has several layers of air conditions that are considered in the analysis. Using this data, the UAM-V model generates predictions of hourly ozone concentrations for every grid. We then calibrate the results of this process to develop 2030 ozone profiles at monitor sites by normalizing the observations to the actual 1996 ozone data at each monitor site. For areas (grids) without ozone monitoring data, we interpolated ozone values using data from monitors surrounding the area. After completing this process, we calculated daily and seasonal ozone metrics to be used as inputs to the health and welfare C-R functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

a. Modeling Domain

The modeling domain representing the Eastern U.S. is the same as that used in EPA's "Regulatory Impact Analysis for the NO_x SIP Call, FIP, and Section 126 Petitions" (US EPA, 1998b). As shown in Figure VII-2, this domain encompasses most of the Eastern U.S. from the East coast to mid-Texas and consists of two grids with differing resolutions. The shaded area of Figure VII-2 uses a relatively fine grid of 12 km consisting of nine vertical layers. The unshaded area of Figure VII-2 has less horizontal resolution, as it uses a 36 km grid with the same nine vertical layers. The vertical height of the modeling domain is 4,000 meters above ground level, for both the shaded and unshaded regions. The split between Eastern and Western counties is made at the 100th degree longitude (which runs through North and South Dakota, Nebraska, Kansas, Oklahoma, and Texas).

b. Simulation Periods

For use in this benefits analysis, the simulation periods modeled by UAM-V included several multi-day periods when ambient measurements recorded high ozone concentrations. A simulation period, or episode, consists of meteorological data characterized over a block of days

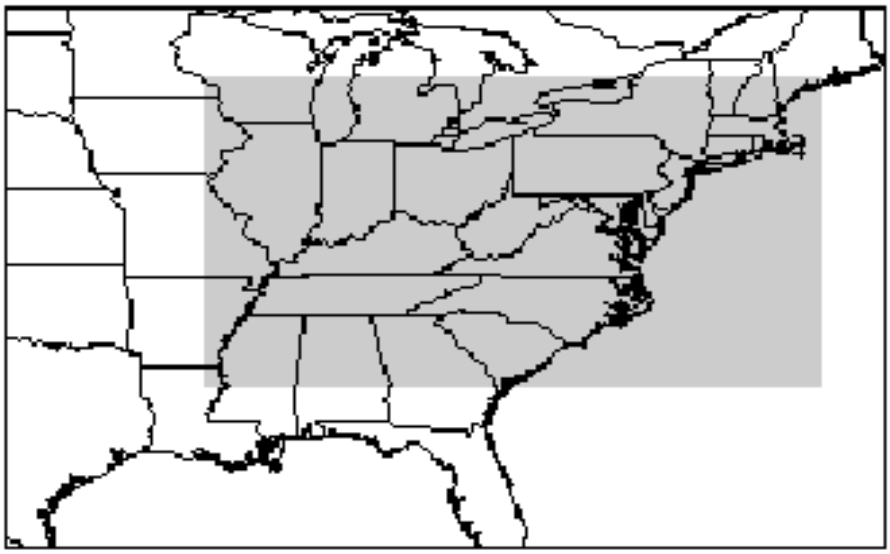

Figure VII-2. UAM-V Modeling Domain for Eastern U.S.

Note: The shaded section represents fine grid modeling (12 km) and the other portions represent coarse grid modeling (36 km).

that are used as inputs to the air quality model. A simulation period is selected to characterize a variety of ozone conditions including some days with high ozone concentrations in one or more portions of the U.S. and observed exceedances of the 1-hour NAAQS for ozone being recorded at monitors. We focused on the summer of 1995 for selecting the episodes to model in the East because 1995 is a recent time period for which we had model-ready meteorological inputs and this timeframe contained several periods of elevated ozone over the Eastern U.S. As detailed in the technical support document for this modeling, this analysis used three multi-day simulation periods to prepare the future-year ozone profiles for the Eastern U.S. ozone analysis: June 12-24, July 5-15, and August 7-21, 1995 (US EPA, 2000). These episodes include a three day "ramp-up" period to initialize the model, but the results for these days are not used in this analysis.

c. Converting UAM-V Outputs to Full-Season Profiles for Benefits Analysis

This study extracted hourly, surface-layer ozone concentrations for each grid-cell from the standard UAM-V output file containing hourly average ozone values. These model predictions are used in conjunction with the observed concentrations obtained from the Aerometric Information Retrieval System (AIRS) to generate ozone concentrations for the entire ozone season.[b,c] The predicted changes in ozone concentrations from the 2030 base case to 2030 HD Engine/Diesel Fuel control scenario serve as inputs to the health and welfare C-R functions of the benefits analysis, i.e., the Criteria Air Pollutant Modeling System (CAPMS).

In order to estimate ozone-related health and welfare effects for the contiguous U.S., full-season ozone data are required for every CAPMS grid-cell. Given available ozone monitoring data, we generated full-season ozone profiles for each location in the contiguous 48 States in two steps: (1) we combine monitored observations and modeled ozone predictions to interpolate hourly ozone concentrations to a grid of 8 km by 8 km population grid-cells, and (2) we converted these full-season hourly ozone profiles to an ozone measure of interest, such as the daily average. [d e] For the analysis of ozone impacts on agriculture and commercial forestry, we use a similar approach except air quality is interpolated to county centroids as opposed to

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[b] The ozone season for this analysis is defined as the 5-month period from May to September; however, to estimate certain crop yield benefits, the modeling results were extended to include months outside the 5-month ozone season.

[c] Based on AIRS, there were 949 ozone monitors with sufficient data, i.e., at least 9 hourly observations per day (8 am to 8 pm) in a given season.

[d] The 8 km grid squares contain the population data used in the health benefits analysis model, CAPMS. See Section C of this chapter for a discussion of this model.

[e] This approach is a generalization of planar interpolation that is technically referred to as enhanced Voronoi Neighbor Averaging (EVNA) spatial interpolation (See Abt Associates (2000) for a more detailed description).

population grid-cells. We report ozone concentrations as a cumulative index called the SUM06. The SUM06 is the sum of the ozone concentrations for every hour that exceeds 0.06 parts per million (ppm) within a 12-hour period from 8 am to 8 pm in the months of May to September. These methods are described in detail in the technical support document to the RIA (Abt Associates, 2000).

d. Ozone Air Quality Results

This section provides a summary the predicted ambient ozone concentrations from the UAM-V model for the 2030 base case and changes associated with the HD Engine control scenario. In Table VII-2, we provide those ozone metrics for grid-cells in the Eastern U.S. that enter the concentration response functions for health benefits endpoints. In addition to the standard frequency statistics (e.g., minimum, maximum, average, median), Table VII-2 provides the population-weighted average which better reflects the baseline levels and predicted changes for more populated areas of the nation. This measure, therefore, will better reflect the potential benefits of these predicted changes through exposure changes to these populations. As shown, the rule results in reductions between 3 and 5 percent, or between 0.8 to 1.7 ppb, in the daily and seasonal average ozone concentrations across Eastern U.S. population grid-cells. A similar relative decline is predicted for the population-weighted average, which indicates rather uniform reductions in these concentrations across urban and rural areas. Additionally, the daily maximum ozone concentrations are predicted to decline between 3.5 and 5 percent, or in the neighborhood of 1.5 ppb.

In Table VII-3, we provide the seasonal SUM06 ozone metric for counties in the Eastern U.S. that enters the concentration response function for agriculture benefit end-points. This metric is a cumulative threshold measure so that the increase in baseline NOx emissions from Tier 2 post-control to this rulemaking have resulted in a larger number of rural counties exceeding the hourly 0.06 ppm threshold. As a result, changes in ozone concentrations for these counties are contributing to greater impacts of the HD Engine/Diesel Fuel rule on the seasonal SUM06 ozone metric. Table VII-3 indicates that the average across all Eastern U.S. counties declined by 78 percent, or almost 17 ppb. Similarly high percentage reductions are observed across the other points on the distribution with the maximum declining by almost 30 ppb, or 55 percent, and the median declining by almost 20 ppb, or 83 percent.

An important factor to consider when interpreting the ozone air quality results presented here is the omission of changes in the Western U.S. Over 22 percent of national NOx emission reductions occur in the Western U.S., with over 10 percent of total NOx emissions occurring in California alone. This suggests that ozone changes in the West may be substantial, and that our estimate of Eastern ozone changes may underestimate populations across the nation that will experience ozone-related benefits of the HD Engine/Diesel Fuel NOx reductions.

Table VII-2. Summary of UAM-V Derived Ozone Air Quality Metrics Due to HD Engine/Diesel Fuel Standards for Health Benefits EndPoints: Eastern U.S.

Statistic [A]	*2030 Base Case*	*Change* [B]	*Percent Change* [B]
Seasonal Average 8-Hour Concentration (ppb)			
Minimum [C]	17.60	-1.20	-6.82%
Maximum [C]	81.80	-3.20	-3.91%
Average	34.93	-1.64	-4.65%
Median	34.90	-1.67	-4.78%
Population-Weighted Average [D]	37.76	-1.43	-3.88%
Daily 1-Hour Maximum Concentration (ppb)			
Minimum [C]	22.11	-1.37	-6.20%
Maximum [C]	108.27	-3.66	-3.38%
Average	44.15	-1.68	-3.81%
Median	43.94	-1.57	-3.57%
Population-Weighted Average [D]	49.69	-1.71	-3.44%
Daily 5-Hour Maximum Concentration (ppb)			
Minimum [C]	18.21	-1.32	-7.25%
Maximum [C]	84.43	-3.27	-3.87%
Average	34.96	-1.64	-4.69%
Median	34.98	-2.13	-6.09%
Population-Weighted Average [D]	37.69	-1.43	-3.79%
Daily 24-Hour Average Concentration (ppb)			
Minimum [C]	11.43	-0.59	-5.16%
Maximum [C]	47.71	-1.60	-3.35%
Average	28.30	-0.82	-2.90%
Median	28.40	-0.86	-3.03%
Population-Weighted Average [D]	28.76	-0.72	-2.50%
Daily 12-Hour Average Concentration (ppb)			
Minimum [C]	16.49	-1.10	-6.67%
Maximum [C]	75.90	-2.89	-3.81%
Average	34.46	-1.53	-4.44%
Median	34.52	-1.13	-3.27%
Population-Weighted Average [D]	36.97	-1.35	-3.65%

[A] These ozone metrics are calculated at the CAPMS grid-cell level for use in health effects estimates based on the results of enhanced spatial interpolation. Except for the daily 24-hour average, these ozone metrics are calculated over relevant time periods during the daylight hours (7 am to 7 pm) of the "ozone season," i.e., May through September. For the 8-hour average, the relevant time period is 9 am to 5 pm, and, for the 5-hour maximum, it is 10 am to 3 pm.

[B] The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "2030 Base Case," and then multiplied by 100 to convert the value to a percentage.

[C] The base case minimum (maximum) is the value for the CAPMS grid cell with the lowest (highest) value.

[D] Calculated by summing the product of the projected 2030 CAPMS grid-cell population and the estimated 2030 CAPMS grid-cell seasonal ozone concentration, and then dividing by the total population. The resulting value is then multiplied by 100 to convert the value to a percentage.

Table VII-3. Summary of UAM-V Derived Ozone Air Quality Metrics Due to HD Engine/Diesel Fuel Standards for Welfare Benefits Endpoints: Eastern U.S.

Statistic [A]	*2030 Base Case*	*Change* [B]	*Percent Change* [B]
Sum06 (ppb)			
Minimum [C]	0.00	0.00	0.00%
Maximum [C]	53.36	-29.10	-54.54%
Average	21.66	-16.91	-78.05%
Median	23.44	-19.50	-83.19%
Population-Weighted Average [D]	23.19	-11.19	-48.26%

[A] SUM06 is defined as the cumulative sum of hourly ozone concentrations over 0.06 ppm (or 60 ppb) that occur during daylight hours (from 8am to 8pm) in the months of May through September. It is calculated at the county level for use in agricultural benefits based on the results of enhanced spatial interpolation.

[B] The change is defined as the control case value minus the base case value. The percent change is the "Change" divided by the "2030 Base Case," which is then multiplied by 100 to convert the value to a percentage.

[C] The base case minimum (maximum) is the value for the county level observation with the lowest (highest) concentration.

[D] Calculated by summing the product of the projected 2030 county population and the estimated 2030 county level ozone concentration, and then dividing by the total population. The resulting value is then multiplied by 100 to convert the value to a percentage.

2. PM Air Quality Estimates

We use the previously described emissions inputs with a national-scale version of the Regulatory Model System for Aerosols and Deposition (REMSAD) to estimate PM air quality in the contiguous U.S. REMSAD was developed as an extension of the episodic UAM-V regional model. Like UAM-V, REMSAD is a three-dimensional grid-based Eulerian air quality model designed to estimate annual particulate concentrations and deposition over large spatial scales (e.g., over the contiguous U.S.). Consideration of the different processes that affect primary (directly emitted) and secondary (formed by atmospheric processes) PM at the regional scale in different locations is fundamental to understanding and assessing the effects of proposed pollution control measures that affect ozone, PM and deposition of pollutants to the surface.[f] Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is useful for evaluating the impacts of the HD Engine/Diesel Fuel rule on U.S. PM concentrations. Our analysis applies the modeling system to the entire U.S. for a base-year of 1996 and for two future-year scenarios: a 2030 base case and a 2030 HD Engine/Diesel Fuel control scenario.

[f] Given the potential impact of the HD Engine/Diesel Fuel rule on secondarily formed particles it is important to employ a Eulerian model such as REMSAD. The impact of secondarily formed pollutants typically involves primary precursor emissions from a multitude of widely dispersed sources, and chemical and physical processes of pollutants that are best addressed using an air quality model that employs an Eulerian grid model design.

REMSAD was peer-reviewed in 1999 for EPA as reported in *"Scientific Peer-Review of the Regulatory Modeling System for Aerosols and Deposition.*" Earlier versions of REMSAD have been employed for the EPA's Prospective 812 Report to Congress and for EPA's Analysis of the Acid Deposition and Ozone Control Act (Senate Bill 172). Version 4.1 of REMSAD was employed for this analysis and is fully described in the air quality technical support documents (US EPA, 2000).

REMSAD simulates every hour of every day of the year and, thus, requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, 3-hour average emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. As applied to the contiguous U.S., the model segments the area within the region into square blocks called grids (roughly equal in size to counties), each of which has several layers of air conditions. Using this data, REMSAD generates predictions of 3-hour average PM concentrations for every grid. We then calculated daily and seasonal PM air quality metrics as inputs to the health and welfare C-R functions of the benefits analysis. The following sections provide a more detailed discussion of each of the steps in this evaluation and a summary of the results.

a. Modeling Domain

As shown in Figure VII-3, the modeling domain encompasses the contiguous 48 States. The domain extends from 126 degrees west longitude to 66 degrees west longitude, and from 24 degrees north latitude to 52 degrees north latitude. The model contains horizontal grid-cells across the model domain of roughly 36 km by 36 km. There are 8 vertical layers of atmospheric conditions with the top of the modeling domain at roughly 16,000 meters. The 36 by 36 km horizontal grid results in a 120 by 92 grid (or 10,080 grid-cells) for each vertical layer. Figure VII-4 illustrates the horizontal grid-cells for Maryland and surrounding areas.

b. Simulation Periods

For use in this benefits analysis, the simulation periods modeled by REMSAD included separate full-year application for 2030 base case and control scenarios with emissions inventories described in Chapter III.

c. Model Inputs

REMSAD requires a variety of input files that contain information pertaining to the modeling domain and simulation period. These include gridded, 3-hour average emissions estimates and meteorological fields, initial and boundary conditions, and land-use information. Separate emissions inventories were prepared for the 1996 base-year and each of the 2030 future-year base case and control scenarios. All other inputs were specified for the 1996 base-year model application and remained unchanged for each future-year modeling scenario.

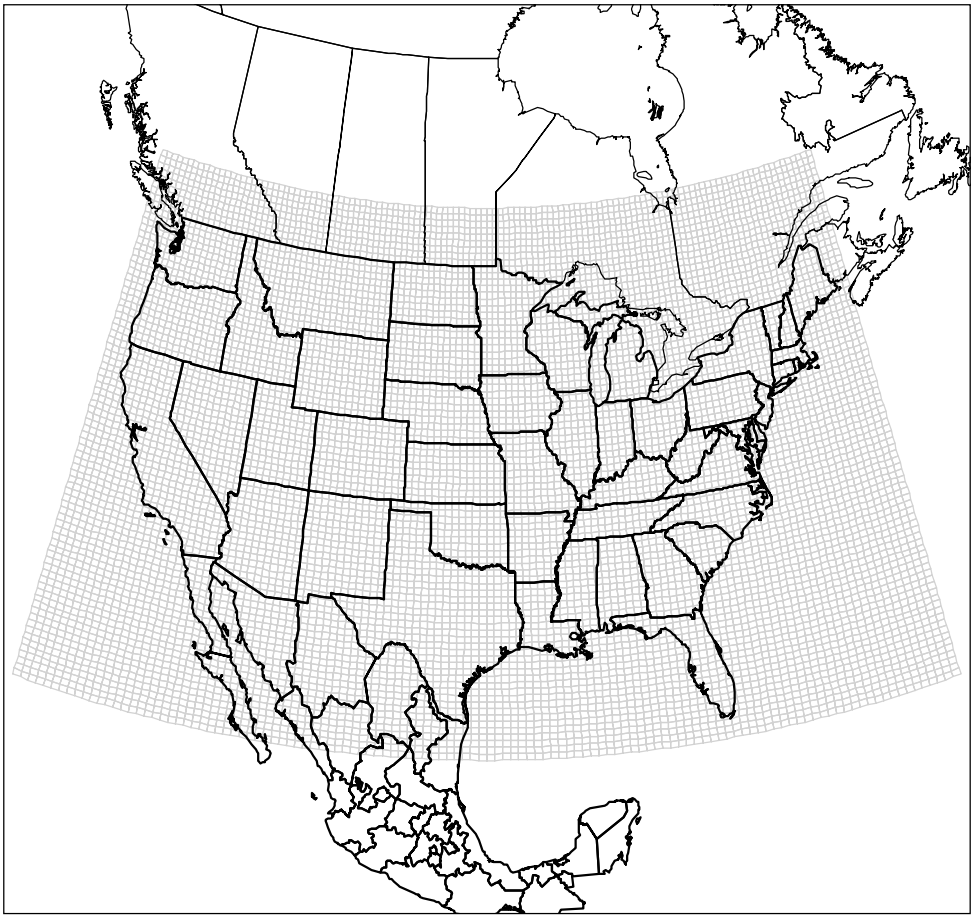

Figure VII-3. REMSAD Modeling Domain for Continental U.S.
Note: Gray markings define individual grid-cells in the REMSAD model.

Similar to UAM-V, REMSAD requires detailed emissions inventories containing

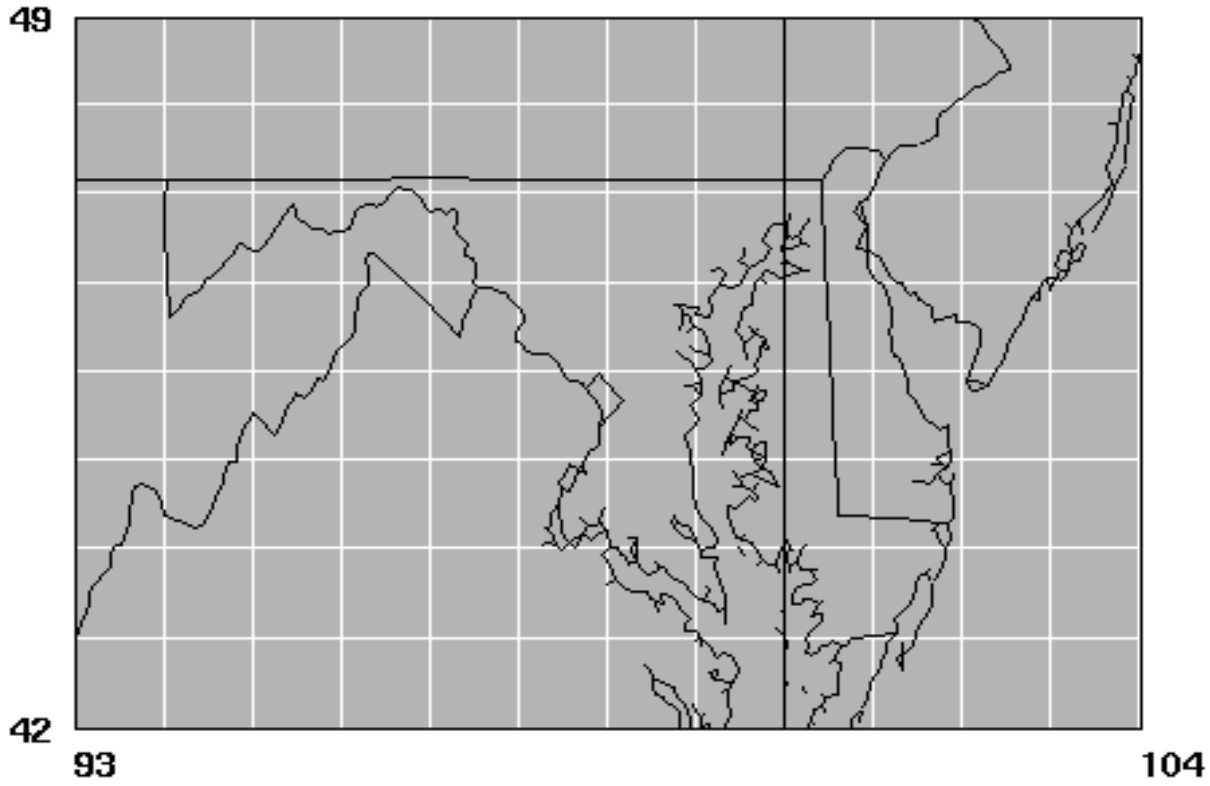

Figure VII-4. Example of REMSAD 36 x 36km Grid-cells for Maryland Area
Note: Gray markings define individual grid-cells in the REMSAD model.

temporally allocated emissions for each grid-cell in the modeling domain for each species being simulated. The previously described annual emission inventories reflecting 2030 base case and control scenarios were preprocessed into model-ready inputs through the Emissions Preprocessing System, Version 2.5 (EPS2.5). The core of EPS2.5 is a series of FORTRAN modules that incorporate spatial, temporal, and chemical resolution into an emissions inventory for use in a photochemical model. Meteorological inputs reflecting 1996 conditions across the contiguous U.S. were derived from Version 5 of the Mesoscale Model (MM5). These inputs included horizontal wind components (i.e., speed and direction), temperature, moisture, vertical diffusion rates, and rainfall rates for each grid cell in each vertical layer. Details of the annual 1996 MM5 modeling are provided in MCNC (2000).

Initial species concentrations and lateral boundary conditions were specified to approximate background concentrations of the species; for the lateral boundaries the concentrations varied (decreased parabolically) with height. These background concentrations are provided in the air quality modeling TSD (US EPA, 2000a). Land use information was obtained from the U.S. Geological Survey database at 10 km resolution.

d. Converting REMSAD Outputs to Benefits Inputs and Model Performance

REMSAD generates predictions of hourly PM concentrations for every grid. The particulate matter species modeled by REMSAD include a primary coarse fraction (corresponding to PM in the 2.5 to 10 micron size range), a primary fine fraction (corresponding to PM less than 2.5 microns in diameter), and several secondary particles (e.g., sulfates, nitrates, and organics). $PM_{2.5}$ is calculated as the sum of the primary fine fraction and all of the secondarily-formed particles. These hourly predictions form the basis for direct calculation of daily and annual PM air quality metrics (i.e., annual mean PM concentration) as inputs to the health and welfare C-R functions of the benefits analysis. In addition, the speciated predictions from REMSAD are employed as inputs to a post-processing module that estimates atmospheric visibility, as discussed later in Section B-3 (US EPA, 2000a).

We modeled 1996 and 2030 base and HD Engine/Diesel Fuel control scenarios. The 2030 modeling is used in this benefits assessment. The goal of the 1996 base year modeling was to reproduce the atmospheric processes resulting in formation and dispersion of $PM_{2.5}$ across the U.S. and to evaluate operational model performance for $PM_{2.5}$ and its related speciated components (e.g., sulfate, nitrate, elemental carbon) in order to estimate the ability of the modeling system to replicate base year concentrations.

This evaluation is comprised principally of statistical assessments of model versus observed pairs. The robustness of any evaluation is directly proportional to the amount and quality of the ambient data available for comparison. Unfortunately, there are few $PM_{2.5}$ monitoring networks with available data for evaluation of the HD Engine/Diesel Fuel PM modeling. Critical limitations of the existing databases are a lack of urban monitoring sites with speciated measurements and poor geographic representation of ambient concentration in the East. The largest available ambient database for 1996 comes from the **I**nteragency **M**onitoring of **PRO**tected **V**isual **E**nvironments (IMPROVE) network. IMPROVE is a cooperative visibility monitoring effort between EPA, federal land management agencies, and state air agencies. Data is collected at Class I areas across the United States mostly at National Parks, National Wilderness Areas, and other protected pristine areas (IMPROVE 2000). There were approximately 60 IMPROVE sites across the nation that had complete annual data in 1996. Forty two of these sites were in the Western U.S. and 18 sites were in the Eastern U.S.

A comparison of predicted versus observed annual average $PM_{2.5}$ concentrations at the IMPROVE sites indicates that $PM_{2.5}$ is underpredicted by about 25% on a nationwide aggregated basis. Most of the underprediction occurs at the Western sites where the overall underprediction is about 35%. However, in the East, ambient $PM_{2.5}$ is overpredicted by about 10%. In addition, model performance was examined for the five component species of $PM_{2.5}$ (sulfate, nitrate, elemental carbon, organic carbon, and other (crustal) fine PM. The results indicate that the performance for both sulfate and elemental carbon was similar to that of $PM_{2.5}$. That is, sulfate and elemental carbon were slightly overpredicted in the East and slightly underpredicted in the West. The performance for nitrate, crustal PM, and organic aerosols was not as good as the performance for the other species. Specifically, nitrate and crustal PM were overpredicted in the East, and organic carbon was underpredicted domainwide.

It should be noted that $PM_{2.5}$ modeling is an evolving science. There have been few regional or national scale model applications for primary and secondary PM. In fact, this is one of the first nationwide applications of a full chemistry Eulerian grid model for the purpose of estimating annual average concentrations of $PM_{2.5}$ and its component species. Also, unlike ozone modeling, there is essentially no database of past performance statistics against which to measure the performance of the HD Engine/Diesel Fuel PM modeling. Given the state of the science relative to PM modeling, it is inappropriate to judge PM model performance using criteria derived for other pollutants, like ozone. Still, the performance of the HD Engine/Diesel Fuel PM modeling is very encouraging, especially considering that the results may be limited by our current knowledge of PM science and chemistry, and by the emissions inventories for primary PM and secondary PM precursor pollutants. Further details of the model performance for PM can be found in the air quality modeling Technical Support Document (US EPA 2000).

e. PM Air Quality Results

Table VII-4 provides a summary of the predicted ambient PM_{10} and $PM_{2.5}$ concentrations from REMSAD for the 2030 base case and changes associated with HD Engine/Diesel Fuel control scenario. The REMSAD results indicate that the predicted change in PM concentrations is composed almost entirely of reductions in fine particulates ($PM_{2.5}$) with little or no reduction in coarse particles (PM_{10} less $PM_{2.5}$). Therefore, the observed changes in PM_{10} are composed primarily of changes in $PM_{2.5}$. In addition to the standard frequency statistics (e.g., minimum, maximum, average, median), Table VII-4 provides the population-weighted average which better reflects the baseline levels and predicted changes for more populated areas of the nation. This measure, therefore, will better reflect the potential benefits of these predicted changes through exposure changes to these populations. As shown, the average annual mean concentrations of $PM_{2.5}$ across all U.S. grid-cells declines by roughly 3.1 percent, or 0.27 $\mu g/m^3$. The population-weighted average mean concentration declined by 4.4 percent, or 0.65 $\mu g/m^3$, which is much larger in absolute terms than the spatial average. This indicates the HD Engine/Diesel Fuel rule generates greater absolute air quality improvements in more populated, urban areas.

Table VII-5 provides information on the 2030 populations that will experience improved PM air quality. There are significant populations that live in areas with meaningful reductions in annual mean $PM_{2.5}$ concentrations resulting from the HD Engine/Diesel Fuel rule. As shown, just over 15 percent of the 2030 U.S. population are predicted to experience reductions of greater than 1 $\mu g/m^3$. Furthermore, almost 33 percent of the 2030 U.S. population will benefit from reductions in annual mean $PM_{2.5}$ concentrations of greater than 0.75 $\mu g/m^3$ and slightly over 60 percent will live in areas with reductions of greater than 0.5 $\mu g/m^3$. This information indicates how widespread the improvements in PM air quality are expected to be and the large populations that will benefit from these improvements.

Table VII-4. Summary of 2030 Base Case PM Air Quality and Changes Due to HD Engine/Diesel Fuel Standards

Statistic	*2030 Base Case*	*Change*[A]	*Percent Change*
PM_{10}			
Minimum Annual Mean ($\mu g/m^3$) [B]	1.52	-0.03	-2.0%
Maximum Annual Mean ($\mu g/m^3$) [B]	65.68	-1.39	-2.1%
Average Annual Mean ($\mu g/m^3$)	10.31	-0.28	-2.4%
Median Annual Mean ($\mu g/m^3$)	8.15	-0.18	-2.3%
Population-Weighted Average Annual Mean ($\mu g/m^3$) [C]	21.70	-0.66	-3.1%
$PM_{2.5}$			
Minimum Annual Mean ($\mu g/m^3$) [B]	1.19	-0.03	-2.4%
Maximum Annual Mean ($\mu g/m^3$) [B]	39.55	-1.35	-3.4%
Average Annual Mean ($\mu g/m^3$)	7.87	-0.27	-3.1%
Median Annual Mean ($\mu g/m^3$)	5.96	-0.17	-3.0%
Population-Weighted Average Annual Mean ($\mu g/m^3$) [C]	14.85	-0.65	-4.4%

[A] The change is defined as the control case value minus the base case value.
[B] The base case minimum (maximum) is the value for the populated grid-cell with the lowest (highest) annual average. The change relative to the base case is the observed change for the populated grid-cell with the lowest (highest) annual average in the base case.
[C] Calculated by summing the product of the projected 2030 grid-cell population and the estimated 2030 PM concentration, for that grid-cell and then dividing by the total population in the 48 contiguous States.

Table VII-5. Distribution of $PM_{2.5}$ Air Quality Improvements Over 2030 Population Due to HD Engine/Diesel Fuel Standards

Change in Annual Mean $PM_{2.5}$ Concentrations ($\mu g/m^3$)	*2030 Population*	
	Number (millions)	*Percent (%)*
$0 > \Delta PM_{2.5}$ *Conc* ≤ 0.25	43.0	11.2%
$0.25 > \Delta PM_{2.5}$ *Conc* ≤ 0.5	95.0	27.5%
$0.5 > \Delta PM_{2.5}$ *Conc* ≤ 0.75	94.9	27.5%
$0.75 > \Delta PM_{2.5}$ *Conc* ≤ 1.0	60.5	17.5%
$1.0 > \Delta PM_{2.5}$ *Conc* ≤ 1.25	23.4	6.8%
$1.25 > \Delta PM_{2.5}$ *Conc* ≤ 1.5	20.9	6.1%
$1.5 > \Delta PM_{2.5}$ *Conc* ≤ 1.75	2.9	0.9%
$\Delta PM_{2.5}$ *Conc* > 1.75	5.2	1.5%

[A] The change is defined as the control case value minus the base case value.

Table VII-6 provides additional insights on the changes in PM air quality resulting from the HD Engine/Diesel Fuel standards. The information presented previously in Table VII-4 illustrated the absolute and relative changes for different points along the distribution of baseline 2030 PM concentration levels, e.g., the change reflects the lowering of the minimum predicted baseline concentration rather than the minimum predicted change for 2030. The latter is the focus of Table VII-6 as it presents the distribution of predicted changes in both absolute terms (i.e., $\mu g/m^3$) and relative terms (i.e., percent) across individual grid-cells. As shown, the absolute reduction in annual mean PM_{10} concentration ranged from a low of 0.02 $\mu g/m^3$ to a high of 2.18 $\mu g/m^3$, while the relative reduction ranged from a low of 0.2 percent to a high of 9.9 percent. Alternatively, for mean $PM_{2.5}$, the absolute reduction ranged from 0.02 to 2.13 $\mu g/m^3$, while the relative reduction ranged from 0.4 to 13.1 percent.

Table VII-6. Summary of Absolute and Relative Changes in PM Air Quality Due to HD Engine/Diesel Fuel Standards

Statistic	*PM_{10} Annual Mean*	*$PM_{2.5}$ Annual Mean*
Absolute Change from 2030 Base Case ($\mu g/m^3$)[A]		
Minimum	-0.02	-0.02
Maximum	-2.18	-2.13
Average	-0.28	-0.27
Median	-0.18	-0.17
Population-Weighted Average [C]	-0.66	-0.65
Relative Change from 2030 Base Case (%)[B]		
Minimum	-0.17%	-0.38%
Maximum	-9.87%	-13.14%
Average	-2.35%	-3.08%
Median	-2.29%	-2.97%
Population-Weighted Average [C]	-3.08%	-4.36%

[A] The absolute change is defined as the control case value minus the base case value for each county.

[B] The relative change is defined as the absolute change divided by the base case value, or the percentage change, for each gridcell. The information reported in this section does not necessarily reflect the same gridcell as is portrayed in the absolute change section.

[C] Calculated by summing the product of the projected 2030 gridcell population and the estimated 2030 gridcell PM absolute/relative measure of change, and then dividing by the total population in the 48 contiguous states.

3. Visibility Degradation Estimates

Visibility degradation is often directly proportional to decreases in light transmittal in the atmosphere. Scattering and absorption by both gases and particles decrease light transmittance. To quantify changes in visibility, our analysis computes a light-extinction coefficient, based on the work of Sisler (1996), which shows the total fraction of light that is decreased per unit distance. This coefficient accounts for the scattering and absorption of light by both particles and gases, and accounts for the higher extinction efficiency of fine particles compared to coarse particles. Fine particles with significant light-extinction efficiencies include sulfates, nitrates, organic carbon, elemental carbon (soot), and soil (Sisler, 1996).

Based upon the light-extinction coefficient, we also calculated a unitless visibility index, called a "deciview," which is used in the valuation of visibility. The deciview metric provides a linear scale for perceived visual changes over the entire range of conditions, from clear to hazy. Under many scenic conditions, the average person can generally perceive a change of one deciview. The higher the deciview value, the worse the visibility. Thus, an improvement in visibility is a decrease in deciview value.

Table VII-7 provides the distribution of visibility improvements across 2030 population resulting from the HD Engine/Diesel Fuel rule. The majority of the 2030 U.S. population live in areas with predicted improvement in annual average visibility of between 0.4 to 0.6 deciviews resulting from the HD Engine/Diesel Fuel rule. As shown, almost 20 percent of the 2030 U.S. population are predicted to experience improved annual average visibility of greater than 0.6 deciviews. Furthermore, roughly 70 percent of the 2030 U.S. population will benefit from reductions in annual average visibility of greater than 0.4 deciviews. The information provided in Table VII-7 indicates how widespread the improvements in visibility are expected to be and the share of populations that will benefit from these improvements.

Because the visibility benefits analysis distinguishes between general regional visibility degradation and that particular to Federally-designated Class I areas (i.e., national parks, forests, recreation areas, wilderness areas, etc.), we separated estimates of visibility degradation into "residential" and "recreational" categories. The estimates of visibility degradation for the "recreational" category apply to Federally-designated Class I areas, while estimates for the "residential" category apply to non-Class I areas. Deciview estimates are estimated using outputs from REMSAD for the 2030 base case and HD Engine/Diesel Fuel control scenarios.

Table VII-7. Distribution of Populations Experiencing Visibility Improvements in 2030 Due to HD Engine/Diesel Fuel Standards

Improvements in Visibility [A] *(annual average deciviews)*	*2030 Population*	
	Number (millions)	*Percent (%)*
$0 > \Delta$ *Deciview* ≤ 0.2	12.1	3.5%
$0.2 > \Delta$ *Deciview* ≤ 0.4	87.4	25.3%
$0.4 > \Delta$ *Deciview* ≤ 0.6	179.7	51.9%
$0.6 > \Delta$ *Deciview* ≤ 0.8	54.5	15.8%
$0.8 > \Delta$ *Deciview* ≤ 1.0	10.7	3.1%
Δ *Deciview* > 1.0	1.5	0.4%

[A] The change is defined as the control case deciview level minus the base case deciview level.

a. Residential Visibility Improvements

Air quality modeling results predict that the HD Engine/Diesel Fuel rule will create improvements in visibility through the country. In Table VII-8, we summarize residential visibility improvements across the Eastern and Western U.S. in 2030. The baseline annual average visibility for all U.S. counties is 14.8 deciviews. The mean improvement across all U.S. counties is 0.28 deciviews, or almost 2 percent. In urban areas with a population of 250,000 or more (i.e., 1,209 out of 5,147 counties), the mean improvement in annual visibility was 0.39

deciviews and ranged from 0.05 to 1.08 deciviews. In rural areas (i.e., 3,938 counties), the mean improvement in visibility was 0.25 deciviews in 2030 and ranged from 0.02 to 0.94 deciviews.

On average, the Eastern U.S. experienced slightly larger absolute but smaller relative improvements in visibility than the Western U.S. from the HD Engine/Diesel Fuel reductions. In Eastern U.S., the mean improvement was 0.34 deciviews from an average baseline of 19.32 deciviews. Western counties experienced a mean improvement of 0.21 deciviews from an average baseline of 9.75 deciviews projected in 2030. Overall, the data suggest that the HD Engine/Diesel Fuel rule has the potential to provide widespread improvements in visibility for 2030.

Table VII-8. Summary of 2030 Baseline Visibility and Changes by Region: Residential (Annual Average Deciviews)

Regions[A]	*2030 Base Case*	*Change*[B]	*Percent Change*
Eastern U.S.	19.32	-0.34	-1.7%
Urban	20.88	-0.40	-1.9%
Rural	18.70	-0.32	-1.7%
Western U.S.	9.75	-0.21	-2.1%
Urban	10.58	-0.37	-3.5%
Rural	9.57	-0.18	-1.9%
National, all counties	14.77	-0.28	-1.9%
Urban	17.12	-0.39	-2.3%
Rural	14.06	-0.25	-1.8%

[A] Eastern and Western regions are separated by 100 degrees north longitude. Background visibility conditions differ by region.
[B] An improvement in visibility is a decrease in deciview value. The change is defined as the HD Engine/Diesel Fuel control case deciview level minus the basecase deciview level.

b. Recreational Visibility Improvements

In Table VII-9, we summarize recreational visibility improvements by region in 2030 in Federal Class I areas. These recreational visibility regions are shown in Figure VII-5. As shown, the national improvement in visibility for these areas is 2.4 percent, or 0.34 deciviews. Predicted relative visibility improvements are the largest in the Western U.S. as shown for California (4.9%), and the Southwest (2.4%), the Northwest (2.3%), and the Rocky Mountain (1.9%). Although Federal Class I areas in the Southeast region are predicted to have the second largest absolute improvement of 0.42 deciviews, it reflects only a 1.6 percent change from 2030 baseline visibility of 25.44 deciviews. The Northeast/Midwest region was predicted to have the smallest relative visibility improvement at 1.2 percent, or 0.25 deciview decline from a baseline of 21.25 deciviews.

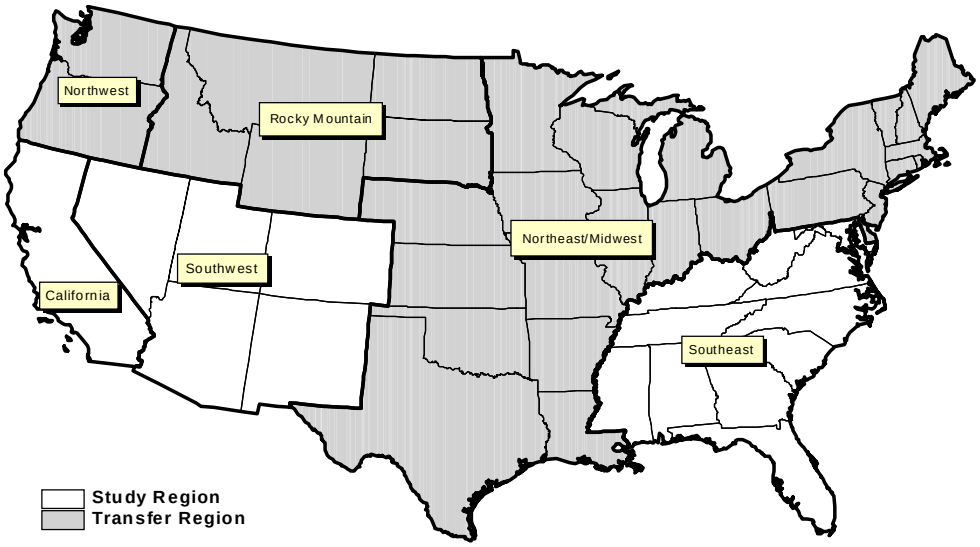

Figure VII-5. Recreational Visibility Regions for Continental U.S.

Table VII-9. Summary of 2030 Baseline Visibility and Changes by Region: Recreational (Annual Average Deciviews)

Class I Visibility Regions[A]	*2030 Base Case*	*Change*[B]	*Percent Change*
Southeast	25.44	-0.42	-1.6%
Southwest	8.90	-0.21	-2.4%
California	12.21	-0.60	-4.9%
Northeast/Midwest	21.25	-0.25	-1.2%
Rocky Mountain	12.54	-0.24	-1.9%
Northwest	15.80	-0.36	-2.3%
National Average (unweighted)	14.38	-0.34	-2.4%

[A] Regions are pictured in Figure VI-5 and are defined in the technical support document (see Abt Associates, 2000).
[B] An improvement in visibility is a decrease in deciview value. The change is defined as the HD Engine/Diesel Fuel control case deciview level minus the basecase deciview level.

Note: Study regions were represented in the Chestnut and Rowe (1990a, 1990b) studies used in evaluating the benefits of visibility improvements, while transfer regions used extrapolated study results.

4. Nitrogen Deposition Estimates

This section presents the methods and results of estimating the potential reductions in airborne nitrogen deposition loadings to estuaries associated with the HD Engine/Diesel Fuel rule. A sampling of 12 estuaries (10 East Coast and 2 Gulf Coast estuaries) were used for this analysis because of the availability of necessary data and their potential representativeness. For each estuary, we completed the following steps as part of this analysis:

n. Baseline loadings of atmospherically supplied nitrogen were obtained from data provided in Valigura, et al. (1996) and from local offices of the Chesapeake Bay Program and the National Estuary Program;

o. Deposition from atmospheric emissions were divided into local and regional areas that contribute to airborne nitrogen deposition;

p. Deposition coefficients, which relate NOx emission changes from a source region to nitrogen deposition changes at a receptor region, were derived for local and regional contributors; and

q. Changes in nitrogen deposition loadings were estimated by multiplying NOx emission changes for the local and regional contributing areas by the appropriate deposition coefficients.

For five of the twelve estuaries, estimates of both direct deposition to the tidal waters and indirect deposition to the entire watershed were available from the literature. For the remaining seven estuaries, only direct deposition estimates were available. Therefore, to obtain indirect deposition estimates where missing, we used RADM-derived nitrogen flux for the watershed (Dennis, 1997). This analysis assumes that 10 percent of nitrogen deposited onto the watershed is delivered via export (pass-through) to the estuary.[g] This calculated indirect deposition value is then added to the direct deposition value obtained from the literature to arrive at the total load from atmospheric deposition.

As stated in Step D above, the nitrogen deposition results are heavily dependent upon the deposition coefficients that estimate the impact of NOx emission changes on nitrogen deposition loadings. For this analysis, two deposition coefficients, an *alpha* and a *beta*, were developed for each estuary. The alpha coefficient relates local emissions to deposition and the beta coefficient relates regional emissions to deposition. These coefficients are calculated for each estuary using deposition outputs from RADM as employed for the final NOx SIP Call (US EPA, 1998b). More detail on this approach and results may be found in Pechan-Avanti (2000).

[g] This assumption is consistent with reported case studies such as Valiela et al., 1997. These authors report that 89 percent of atmospherically deposited nitrogen was retained by the watershed of Waquoit Bay, suggesting an 11 percent pass through factor.

Table VII-10 provides a summary of the baseline deposition and change in nitrogen deposition estimates for the selected estuaries as a result of the HD Engine/Diesel Fuel rule. As shown, implementation results in roughly a 21 percent reduction in the average annual deposition across these estuaries. These predicted reductions range from a low of 17.2 percent for Delaware Inland Bay to highs of 21.6 percent for Long Island Sound and 24 percent for Tampa Bay.

Table VII-10. Summary of 2030 Nitrogen Deposition in Selected Estuaries and Changes Due to HD Engine/Diesel Fuel Rule (million kg/year)

Estuary	*2030 Base Case*	*Change*[A]	*Percent Change*
Albemarle/Pamlico Sound	7.66	-1.64	-21.4%
Cape Cod Bay	2.98	-0.61	-20.4%
Chesapeake Bay	12.04	-2.46	-20.5%
Delaware Bay	2.56	-0.49	-19.4%
Delaware Inland Bays	0.32	-0.05	-17.2%
Gardiners Bay	0.90	-0.19	-20.8%
Hudson River/Raritan Bay	3.07	-0.61	-19.9%
Long Island Sound	4.51	-0.97	-21.6%
Massachusetts Bay	1.03	-0.21	-20.3%
Narragansett Bay	0.89	-0.18	-20.5%
Sarasota Bay	0.24	-0.05	-20.6%
Tampa Bay	1.46	-0.35	-24.0%
All Selected Estuaries	37.64	-7.82	-20.8%

[A] Change is defined here as the emissions level after implementing the HD Engine/Diesel Fuel rule minus the base case emissions.

C. Benefit Analysis

1. Methods for Estimating Benefits from Air Quality Improvements

Environmental and health economists have a number of methods for estimating the economic value of improvements in (or deterioration of) environmental quality. The method used in any given situation depends on the nature of the effect and the kinds of data, time, and resources that are available for investigation and analysis. This section provides an overview of the methods we selected to monetize the benefits included in this HD Engine/Diesel Fuel RIA.

We note at the outset that EPA rarely has the time or resources to perform extensive new research to measure economic benefits for individual rulemakings. As a result, our estimates are based on the best available methods of benefits transfer. Benefits transfer is the science and art of adapting primary benefits research from similar contexts to obtain the most accurate measure of benefits for the environmental quality change under analysis. Where appropriate, adjustments are made for the level of environmental quality change, the sociodemographic and economic characteristics of the affected population, and other factors in order to improve the accuracy and robustness of benefits estimates.

In general, economists tend to view an individual's willingness-to-pay (WTP) for a improvement in environmental quality as the appropriate measure of the value of a risk reduction. An individual's willingness-to-accept (WTA) compensation for not receiving the improvement is also a valid measure. However, WTP is generally considered to be a more readily available and conservative measure of benefits. Adoption of WTP as the measure of value implies that the value of environmental quality improvements is dependent on the individual preferences of the affected population and that the existing distribution of income (ability to pay) is appropriate.

For many goods, WTP can be observed by examining actual market transactions. For example, if a gallon of bottled drinking water sells for one dollar, it can be observed that at least some persons are willing to pay one dollar for such water. For goods not exchanged in the market, such as most environmental "goods," valuation is not as straightforward. Nevertheless, a value may be inferred from observed behavior, such as sales and prices of products that result in similar effects or risk reductions, (e.g., non-toxic cleaners or bike helmets). Alternatively, surveys may be used in an attempt to directly elicit WTP for an environmental improvement.

One distinction in environmental benefits estimation is between use values and non-use values. Although no general agreement exists among economists on a precise distinction between the two (see Freeman, 1993), the general nature of the difference is clear. Use values are those aspects of environmental quality that affect an individual's welfare more or less directly. These effects include changes in product prices, quality, and availability, changes in the quality of outdoor recreation and outdoor aesthetics, changes in health or life expectancy, and the costs of actions taken to avoid negative effects of environmental quality changes.

Non-use values are those for which an individual is willing to pay for reasons that do not relate to the direct use or enjoyment of any environmental benefit, but might relate to existence values and bequest values. Non-use values are not traded, directly or indirectly, in markets. For this reason, the measurement of non-use values has proved to be significantly more difficult than the measurement of use values. The air quality changes produced by the final HD Engine/Diesel Fuel rule cause changes in both use and non-use values, but the monetary benefit estimates are almost exclusively for use values.

More frequently than not, the economic benefits from environmental quality changes are not traded in markets, so direct measurement techniques can not be used. Avoided cost methods are ways to estimate the costs of pollution by using the expenditures made necessary by pollution

damage. For example, if buildings must be cleaned or painted more frequently as levels of PM increase, then the appropriately calculated increment of these costs is a reasonable lower bound estimate (under most conditions) of true economic benefits when PM levels are reduced. A variation on the avoided cost method is used to provide an alternative estimate of the benefits of reductions in nitrogen deposition to estuaries (see Sections C.4 and F). Avoided costs methods are also used to estimate some of the health-related benefits related to morbidity, such as hospital admissions (see section D).

Indirect market methods can also be used to infer the benefits of pollution reduction. The most important application of this technique for our analysis is the calculation of the value of a statistical life for use in the estimate of benefits from mortality reductions. There exists no market where changes in the probability of death are directly exchanged. However, people make decisions about occupation, precautionary behavior, and other activities associated with changes in the risk of death. By examining these risk changes and the other characteristics of people's choices, it is possible to infer information about the monetary values associated with changes in mortality risk (see Section D). For measurement of health benefits, this analysis captures the WTP for most use and non-use values, with the exception of the value of avoided hospital admissions, which only captures the avoided cost of illness because no WTP values were available in the published literature.

The most direct way to measure the economic value of air quality changes is in cases where the endpoints have market prices. For the final rule, this can only be done for effects on commercial agriculture and forestry. Well-established economic modeling approaches are used to predict price changes that result from predicted changes in agricultural and forestry outputs. Consumer and producer surplus measures can then be developed to give reliable indications of the benefits of changes in ambient air quality for these categories (see Section E).

Estimating benefits for visibility and ecosystem services is a more difficult and less precise exercise because the endpoints are not directly or indirectly valued in markets. For example, the loss of a species of animal or plant from a particular habitat does not have a well-defined price. The contingent valuation (CV) method has been employed in the economics literature to value endpoint changes for both visibility and ecosystem functions (Chestnut and Dennis, 1997). The CV method values endpoints by using carefully structured surveys to ask a sample of people what amount of compensation is equivalent to a given change in environmental quality. There is an extensive scientific literature and body of practice on both the theory and technique of CV. EPA believes that well-designed and well-executed CV studies are valid for estimating the benefits of air quality regulation.[h]

[h]Concerns about the reliability of value estimates from CV studies arose because research has shown that bias can be introduced easily into these studies if they are not carefully conducted. Accurately measuring WTP for avoided health and welfare losses depends on the reliability and validity of the data collected. There are several issues to consider when evaluating study quality, including but not limited to 1) whether the sample estimates of WTP are representative of the population WTP; 2) whether the good to be valued is comprehended and accepted by the respondent; 3) whether the WTP elicitation format is designed to minimize strategic responses; 4) whether WTP is sensitive to respondent familiarity with the good, to the size of the change in the good, and to income; 5) whether

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. There is substantial empirical evidence that the income elasticity[i] of WTP for health risk reductions is positive, although there is uncertainty about its exact value. Thus, as real income increases the WTP for environmental improvements also increases. While many analyses assume that the income elasticity of WTP is unit elastic (i.e., ten percent higher real income level implies a ten percent higher WTP to reduce risk changes), empirical evidence suggests that income elasticity is substantially less than one and thus relatively inelastic. As real income rises, the WTP value also rises but at a slower rate than real income.

The effects of real income changes on WTP estimates can influence benefit estimates in two different ways: (1) through real income growth between the year a WTP study was conducted and the year for which benefits are estimated, and (2) through differences in income between study populations and the affected populations at a particular time. Empirical evidence of the effect of real income on WTP gathered to date is based on studies examining the former. The Environmental Economics Advisory Committee (EEAC) of the SAB advised EPA to adjust WTP for increases in real income over time, but not to adjust WTP to account for cross-sectional income differences "because of the sensitivity of making such distinctions, and because of insufficient evidence available at present" (EPA-SAB-EEAC-00-013).

Based on a review of the available income elasticity literature, we adjust the valuation of human health benefits upward to account for projected growth in real U.S. income. Faced with a dearth of estimates of income elasticities derived from time-series studies, we applied estimates derived from cross-sectional studies in our analysis. Details of the procedure can be found in Kleckner and Neumann (1999). An abbreviated description of the procedure we used to account for WTP for real income growth between 1990 and 2030 is presented below.

Reported income elasticities suggest that the severity of a health effect is a primary determinant of the strength of the relationship between changes in real income and WTP. As such, we use different elasticity estimates to adjust the WTP for minor health effects, severe and chronic health effects, and premature mortality. We also expect that the WTP for improved visibility in Class I areas would increase with growth in real income. The elasticity values used to adjust the primary estimate of benefits are presented in Table VII-11. In addition to the primary estimate, we also present the impacts of using different assumed elasticities in Table VII-25.

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the estimates of WTP are broadly consistent with other estimates of WTP for similar goods; and 6) the extent to which WTP responses are consistent with established economic principles.

[i]Income elasticity is a common economic measure equal to the percentage change in WTP for a one percent change in income.

Table VII-11. Elasticity Values Used to Account for Projected Real Income Growth[A]

Benefit Category	Lower Sensitivity Bound	Primary	Upper Sensitivity Bound
Minor Health Effect	0.04	0.14	0.30
Severe and Chronic Health Effects	0.25	0.45	0.60
Premature Mortality	0.08	0.40	1.00
Visibility[B]	--	0.90	--

[A] Derivation of these ranges can be found in Kleckner and Neumann (1999) and Chestnut (1997). Cost of Illness (COI) estimates are assigned an adjustment factor of 1.0.
[B] No range was applied for visibility because no ranges were available in the current published literature.

Accounting for real income growth over time requires projections of both real gross domestic product (GDP) and populations. For consistency with the emissions and benefits modeling, we use population estimates for the years 2015, 2020, and 2030 as described in Davidson (1999). These population estimates are based on 1990 U.S. Census data and Bureau of Economic Analysis growth projections.[j] For the years between 1990 and 2010, we use population estimates provided in Kleckner and Neumann (1999), which were obtained from the US Bureau of Census.[k] We use projections of real GDP provided in Kleckner and Neumann (1999) for the years 1990 to 2010.[l] We use projections of real GDP (in chained 1996 dollars) provided by Standard and Poor's for the years 2010 to 2024.[m] The Standard and Poor's database only provides estimates of real GDP between 1990 and 2024. We were unable to find reliable projections of GDP beyond 2024. As such, we assume that per capita GDP remains constant between 2024 and 2030. This assumption will lead us to under-predict benefits because at least some level of income growth would be projected to occur between the years 2024 and 2030.

Using the method outlined in Kleckner and Neumann (1999), and the population and income data described above, we calculate income growth factors for each of the elasticity

[j] US Bureau of Census. Annual Projections of the Total Resident Population, Middle Series, 1999-2010. (Available on the internet at http://www.census.gov/population/projections/nation/summary/np-t1.txt)

[k] US Bureau of Census. Historic National Population Estimates. (Available on the internet at http://www.census.gov/population/estimates/nation/poplockest.txt) and US Bureau of Census. Resident Population Projections of the U.S.; Middle Series. (Available on the internet at http://www.census.gov/population/estimates/nation/npaltsrs.txt)

[l] US Bureau of Economic Analysis, Table 2A (1992$). (Available on the internet at http://www.bea.doc.gov/bea/dn/0897nip2/tab2a.htm) and US Bureau of Economic Analysis, Economics and Budget Outlook. Note that projections for 2007 to 2010 are based on average GDP growth rates between 1999 and 2007.

[m] Standard and Poor's. 2000. "The U.S. Economy: The 25 Year Focus." Winter 2000.

estimates listed in Table VII-11. Benefits for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility) will be adjusted by multiplying the unadjusted benefits by the appropriate adjustment factor. In Table VII-12 we list the estimated factors. Note that for premature mortality, we apply the income adjustment factor *ex post* to the present discounted value of the stream of avoided mortalities occurring over the lag period. Also note that no adjustments will be made to benefits based on the cost-of-illness approach or to work loss days and worker productivity. This assumption will also lead us to under predict benefits since it is likely that increases in real U.S. income would also result in increased cost-of-illness (due, for example, to increases in wages paid to medical workers) and increased cost of work loss days and lost worker productivity (reflecting that if worker incomes are higher, the losses resulting from reduced worker production would also be higher). No adjustments are needed for agricultural and commercial forestry benefits, as these models are based on projections of supply and demand in future years and should already incorporate future changes in real income. The results are presented in section F.

Table VII-12. Adjustment Factors Used to Account for Projected Real Income Growth[A]

Benefit Category	Lower Sensitivity Bound	Primary	Upper Sensitivity Bound
Minor Health Effect	1.026	1.095	1.214
Severe and Chronic Health Effects	1.176	1.341	1.482
Premature Mortality	1.053	1.297	1.956
Visibility[B]	--	1.821	--

[A] Based on elasticity values reported in Table VII-11, US Census population projections, and projections of real gross domestic product per capita.
[B] No range was applied for visibility because no ranges were available in the current published literature.

2. 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.[n] This analysis is no exception. As outlined both in this and preceding chapters, there are many inputs used to derive the final estimate of benefits, including emission inventories, air quality models (with their associated parameters and inputs), epidemiological estimates of concentration-response (C-R) functions, estimates of values (both from WTP and cost-of-illness studies), population estimates, income estimates, and

[n] It should be recognized that in addition to uncertainty, the annual benefit estimates for the final HD Engine/Diesel Fuel rule presented in this analysis are also inherently variable, due to the truly random processes that govern pollutant emissions and ambient air quality in a given year. Factors such as electricity demand and weather display constant variability regardless of our ability to accurately measure them. As such, the estimates of annual benefits should be viewed as representative of the types of benefits that will be realized, rather than the actual benefits that would occur every year.

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 their location in the benefits analysis, may have a disproportionately large impact on final 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. When compounded with uncertainty in later stages, small uncertainties in emission levels can lead to much larger impacts on total benefits. A more thorough discussion of uncertainty can be found in the benefits technical support document (TSD) (Abt Associates, 2000).

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

- Gaps in scientific data and inquiry;
- Variability in estimated relationships, such as C-R functions, introduced through differences in study design and statistical modeling;
- Errors in measurement and projection for variables such as population growth rates;
- Errors due to misspecification of model structures, including the use of surrogate variables, such as using PM_{10} when $PM_{2.5}$ is not available, excluded variables, and simplification of complex functions; and
- Biases due to omissions or other research limitations.

Some of the key uncertainties in the benefits analysis are presented in Table VII-13. Given the wide variety of sources for uncertainty and the potentially large degree of uncertainty about any primary estimate, it is necessary for us to address this issue in several ways. These include qualitative discussions, probabilistic assessments, alternative calculations, and bounding exercises. For some parameters or inputs it may be possible to provide a statistical representation of the underlying uncertainty distribution. For other parameters or inputs, the information necessary to estimate an uncertainty distribution is not available. Even for individual endpoints, there is usually more than one source of uncertainty. This makes it difficult to provide a quantified uncertainty estimate. For example, the C-R function used to estimate avoided premature mortality has an associated standard error which represents the sampling error around the pollution coefficient in the estimated C-R function. It would be possible to report a confidence interval around the estimated incidences of avoided premature mortality based on this standard error. However, this would omit the contribution of air quality changes, baseline population incidences, projected populations exposed, and transferability of the C-R function to diverse locations to uncertainty about premature mortality. Thus, a confidence interval based on the standard error would provide a misleading picture about the overall uncertainty in the estimates. Information on the uncertainty surrounding particular C-R and valuation functions is provided in the benefits TSD for this RIA (Abt Associates, 2000). But, this information should be interpreted within the context of the larger uncertainty surrounding the entire analysis.

Our approach to characterizing model uncertainty is to present a primary estimate of the benefits, based on the best available scientific literature and methods, and to then provide alternative calculations to illustrate the effects of uncertainty about key analytical assumptions. We do not attempt to assign probabilities to these alternative calculations, as we believe this

would only add to the uncertainty of the analysis or present a false picture about the precision of the results. Instead, the reader is invited to examine the impact of applying the different assumptions on the estimate of total benefits. While it is possible to combine all of the alternative calculations with a positive impact on benefits to form a "high" estimate or all of the alternative calculations with a negative impact on benefits to form a "low" estimate, this would not be appropriate because the probability of all of these alternative assumptions occurring simultaneously is extremely low.[o] Instead, the alternative calculations are intended to demonstrate the sensitivity of our benefits results to key parameters which may be uncertain. Alternative calculations are presented in Table VII-25.

Many benefits categories, while known to exist, do not have enough information available to provide a quantified or monetized estimate. The uncertainty regarding these endpoints is such that we could determine neither a primary estimate nor a plausible range of values.

Our estimated range of total benefits should be viewed as an approximate result because of the sources of uncertainty discussed above (see Table VII-13). The total benefits estimate may understate or overstate actual benefits of the rule.

In considering the monetized benefits estimates, the reader should remain aware of the many limitations of conducting these analyses mentioned throughout this RIA. One significant limitation of both the health and welfare benefits analyses is the inability to quantify many of the serious effects listed in Table VII-1. For many health and welfare effects, such as PM-related materials damage, reliable C-R functions and/or valuation functions are not currently available. In general, if it were possible to monetize these benefits categories, the benefits estimates presented in this analysis would increase. Unquantified benefits are qualitatively discussed in the health and welfare effects sections. In addition to unquantified benefits, there may also be environmental costs that we are unable to quantify. Several of these environmental cost categories are related to nitrogen deposition, while one category is related to the issue of ultraviolet light. These endpoints are qualitatively discussed in the health and welfare effects sections 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.

[o] Some recent benefit-cost analyses in Canada and Europe (Holland et al., 1999; Lang et al., 1995) have estimated ranges of benefits by assigning *ad hoc* probabilities to ranges of parameter values for different endpoints. Although this does generate a quantitative estimate of an uncertainty range, the estimated points on these distributions are themselves highly uncertain and very sensitive to the subjective judgements of the analyst. To avoid these subjective judgements, we choose to allow the reader to determine the weights they would assign to alternative estimates.

Table VII-13. Primary Sources of Uncertainty in the Benefit Analysis

1. Uncertainties Associated With Concentration-Response Functions
- The value of the ozone- or PM-coefficient in each C-R function. - Application of a single C-R function to pollutant changes and populations in all locations. - Similarity of future year C-R relationships to current C-R relationships. - Correct functional form of each C-R relationship. - Extrapolation of C-R relationships beyond the range of ozone or PM concentrations observed in the study. - Application of C-R relationships only to those subpopulations matching the original study population.
2. Uncertainties Associated With Ozone and PM Concentrations
- Responsiveness of the models to changes in precursor emissions resulting from the control policy. - Projections of future levels of precursor emissions, especially ammonia and crustal materials. - Model chemistry for the formation of ambient nitrate concentrations. - Lack of ozone monitors in rural areas requires extrapolation of observed ozone data from urban to rural areas. - Use of separate air quality models for ozone and PM does not allow for a fully integrated analysis of pollutants and their interactions. - Full ozone season air quality distributions are extrapolated from a limited number of simulation days. VI. Comparison of model predictions of particulate nitrate with observed rural monitored nitrate levels indicates that REMSAD overpredicts nitrate in some parts of the Eastern US and underpredicts nitrate in parts of the Western US.
3. Uncertainties Associated with PM Mortality Risk
- No scientific literature supporting a direct biological mechanism for observed epidemiological evidence. vii. Direct causal agents within the complex mixture of PM have not been identified. - The extent to which adverse health effects are associated with low level exposures that occur many times in the year versus peak exposures. ii The extent to which effects reported in the long-term exposure studies are associated with historically higher levels of PM rather than the levels occurring during the period of study. - Reliability of the limited ambient $PM_{2.5}$ monitoring data in reflecting actual $PM_{2.5}$ exposures.
4. Uncertainties Associated With Possible Lagged Effects
- The portion of the PM-related long-term exposure mortality effects associated with changes in annual PM levels would occur in a single year is uncertain as well as the portion that might occur in subsequent years.
5. Uncertainties Associated With Baseline Incidence Rates
9. Some baseline incidence rates are not location-specific (e.g., those taken from studies) and may therefore not accurately represent the actual location-specific rates. - Current baseline incidence rates may not approximate well baseline incidence rates in 2030. j. Projected population and demographics may not represent well future-year population and demographics.
6. Uncertainties Associated With Economic Valuation
- Unit dollar values associated with health and welfare endpoints are only estimates of mean WTP and therefore have uncertainty surrounding them. xi. Mean WTP (in constant dollars) for each type of risk reduction may differ from current estimates due to differences in income or other factors. - Future markets for agricultural and forestry products are uncertain.
7. Uncertainties Associated With Aggregation of Monetized Benefits
ii Health and welfare benefits estimates are limited to the available C-R functions. Thus, unquantified or unmonetized benefits are not included.

D. Assessment of Human Health Benefits

The most significant monetized benefits of reducing ambient concentrations of PM and ozone are attributable to reductions in health risks associated with air pollution. EPA's Criteria Documents for ozone and PM list numerous health effects known to be linked to ambient concentrations of these pollutants (US EPA, 1996a and 1996b). This section describes individual effects and the methods used to quantify and monetize changes in the expected number of incidences of various health effects.

In Section 1, we discuss how we have determined the baseline incidences for the health effects impacted by changes in PM and ozone. In Section 2, we explain how we address the issue of health effects thresholds. In Section 3, we describe how we quantify and value changes in individual health effects. Finally, in Section 4 we present quantified estimates of the reductions in health effects resulting from the HD Engine/Diesel Fuel rule and their associated monetary values.

1. Estimating Baseline Incidences for Health Effects

The epidemiological studies of the association between pollution levels and adverse health effects generally provide a direct estimate of the relationship of air quality changes to the relative risk of a health effect, rather than an estimate of the absolute number of avoided cases. For example, a typical result might be that a 10 $\mu g/m^3$ decrease in daily $PM_{2.5}$ levels might decrease hospital admissions by three percent. The baseline incidence of the health effect is necessary to convert this relative change into a number of cases.

The baseline incidence used in our analyses needs to match the specific population studied. For example, because some mortality studies considered only non-accidental mortality, we adjusted county-specific baseline total mortality rates used in the estimation of PM-related premature mortality to provide a better estimate of county-specific non-accidental mortality. We multiplied each county-specific mortality rate by the ratio of national non-accidental mortality to national total mortality (0.93) (US Centers for Disease Control, 1999a). An additional adjustment was necessary to provide baseline incidences for adults 30 and older for use in the Krewski, et al. (2000) and Pope, et al. (1995) PM mortality C-R functions. We estimated county-specific baseline mortality incidences for this population by applying national age-specific death rates to county-specific age distributions, and adjusting the resulting estimated age-specific incidences so that the estimated total incidences (including all ages) equals the actual county-specific total incidences. We applied this same procedure to develop baseline incidences for adults 25 and older for use in alternative premature mortality estimates based on Harvard Six-City/Krewski, et al. (2000).

County-level incidence rates are not available for other endpoints. We used national incidence rates whenever possible, because these data are most applicable to a national assessment of benefits. However, for some studies, the only available incidence information

comes from the studies themselves; in these cases, incidence in the study population is assumed to represent typical incidence at the national level.

2. Accounting for Potential Health Effect Thresholds

When conducting clinical (chamber) and epidemiological studies, C-R functions may be estimated with or without explicit thresholds. Air pollution levels below the threshold are assumed to have no associated adverse health effects. When a threshold is not assumed, as is often the case in epidemiological studies, any exposure level is assumed to pose a non-zero risk of response to at least one segment of the population.

The possible existence of an effect threshold is a very important scientific question and issue for policy analyses such as this one. In the benefits analyses for some recent RIAs (see the PM NAAQS RIA, the Regional Haze RIA, and the NOx SIP Call RIA), the low-end estimate of benefits assumed a threshold in PM health effects at 15 $\mu g/m^3$. However, the SAB subsequently advised EPA that there is currently no scientific basis for selecting a threshold of 15 $\mu g/m^3$ or any other specific threshold for the PM-related health effects considered in this analysis (EPA-SAB-Council-ADV-99-012, 1999). Therefore, for our benefits analysis, we assume there are no thresholds for modeling health effects. It is not appropriate to adopt a threshold for use in either the primary analysis or any alternative calculations because no adequate scientific evidence exists to support such a calculation. Although not included in the primary analysis, the potential impact of a health effects threshold on avoided incidences of PM-related premature mortality is explored as a key sensitivity analysis and is presented in Appendix VII-A.

3. Quantifying and Valuing Individual Health Endpoints

Quantifiable health benefits of the final HD Engine/Diesel Fuel rule may be related to ozone only, PM only, or both pollutants. Decreased worker productivity is the only health endpoint related to ozone but not PM.[p] PM-only health effects include premature mortality, chronic bronchitis, acute bronchitis, upper and lower respiratory symptoms, and work loss days.[q]

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[p] In the benefits analysis for the recent Tier 2/Gasoline Sulfur rule, based on our interpretation of the advice from the SAB (EPA-SAB-COUNCIL-ADV-00-001), we included avoided incidences of chronic asthma in adult males as a primary health endpoint associated with ozone. Recent advice from asthma experts both within and outside the Agency has led us to conclude that while the McDonnell, et al. (1999) study raises concerns about the possibility of a connection, the scientific evidence supporting the relationship between ozone and new incidences of asthma is not sufficient to support its inclusion in our primary analysis. We do, however, include this important endpoint as an alternative calculation in Table VII-25.

[q] Some evidence has been found linking both PM and ozone exposures with premature mortality. The SAB has raised concerns that mortality-related benefits of air pollution reductions may be overstated if separate pollutant-specific estimates, some of which may have been obtained from models excluding the other pollutants, are aggregated. In addition, there may be important interactions between pollutants and their effect on mortality (EPA-SAB-Council-ADV-99-012, 1999).

Because of concern about overstating of benefits and because the evidence associating mortality with exposure to PM is currently stronger than for ozone, only the benefits related to the long-term exposure study

Health effects related to both PM and ozone include hospital admissions, asthma attacks, and minor restricted activity days.

For this analysis, we rely on C-R functions estimated in published epidemiological studies relating serious health effects to ambient air quality. The specific studies from which C-R functions are drawn are included in Table VII-14. A complete discussion of the C-R functions used for this analysis and information about each endpoint are contained in the benefits TSD for this RIA (Abt Associates, 2000).

While a broad range of serious health effects have been associated with exposure to elevated ozone and PM levels (as noted for example in Table VII-1 and described more fully in the ozone and PM Criteria Documents (US EPA, 1996a, 1996b), we include only a subset of health effects in this quantified benefit analysis. Health effects are excluded from this analysis for three reasons: (i) the possibility of double counting (such as hospital admissions for specific respiratory diseases); (ii) uncertainties in applying effect relationships based on clinical studies to the affected population; or (iii) a lack of an established C-R relationship.

When a single published study is selected as the basis of the C-R relationship between a pollutant and a given health effect, or "endpoint," applying the C-R function is straightforward. This is the case for most of the health endpoints selected for inclusion in the benefits analysis. A single C-R function may be chosen over other potential functions because the underlying epidemiological study used superior methods, data or techniques, or because the C-R function is more generalized and comprehensive.

(ACS/Krewkski, et al, 2000) of mortality are included in the total primary benefits estimate. The benefits associated with ozone reductions are presented as a sensitivity analysis in Appendix VII-A but are not included in the estimate of total benefits.

Table VII-14. Endpoints and Studies Included in the Primary Analysis

Endpoint	Pollutant	Study	Study Population
Premature Mortality			
Long-term exposure	$PM_{2.5}$	Krewski, et al. (2000)[A]	Adults, 30 and older
Chronic Illness			
Chronic Bronchitis (pooled estimate)	$PM_{2.5}$ PM_{10}	Abbey, et al. (1995) Schwartz, et al. (1993)	> 26 years > 29 years
Hospital Admissions			
All Respiratory	Ozone	Pooled estimate (8 studies)	All ages
COPD	PM	Samet, et al. (2000)	> 64 years
Pneumonia	PM	Samet, et al. (2000)	> 64 years
Asthma	PM	Sheppard, et al. (1999)	< 65 years
Total Cardiovascular	PM	Samet, et al. (2000)	> 64 years
Cardiac Dysrythmias	Ozone	Burnett, et al. (1999)	All ages
Asthma-Related ER Visits	Ozone	Pooled estimate (3 studies)	All ages
Asthma-Related ER Visits	PM	Schwartz, et al. (1993)	All ages
Other Illness			
Asthma Attacks	PM, Ozone	Whittemore and Korn (1980)	Asthmatics, all ages
Acute Bronchitis	PM	Dockery et al. (1996)	Children, 8-12 years
Upper Respiratory Symptoms	PM	Pope et al. (1991)	Asthmatic children, 9-11
Lower Respiratory Symptoms	PM	Schwartz et al. (1994)	Children, 7-14 years
Work Loss Days	PM	Ostro (1987)	Adults, 18-65 years
Minor Restricted Activity Days (minus asthma attacks)	PM, Ozone	Ostro and Rothschild (1989)	Adults, 18-65 years

[A] Estimate derived from Table 31, PM2.5(DC), All Causes Model (Relative Risk =1.12 for a 24.5 $\mu g/m^3$ increase in mean $PM_{2.5}$).

When several estimated C-R relationships between a pollutant and a given health endpoint have been selected, they are combined or pooled to derive a single estimate of the relationship. The benefits TSD provides details of the procedures used to combine multiple C-R functions (Abt Associates, 2000). For example, pooled C-R functions are used to estimate incidences of chronic bronchitis related to PM exposure and to estimate hospital admissions for all respiratory causes and asthma-related emergency room visits related to ozone exposure.

Whether the C-R relationship between a pollutant and a given health endpoint is estimated by a single function from a single study or by a pooled function of C-R functions from several studies, we apply that same C-R relationship to all locations in the U.S. Although the C-R relationship may in fact vary somewhat from one location to another (for example, due to differences in population susceptibilities or differences in the composition of PM), location-

specific C-R functions are generally not available. A single function applied everywhere may result in overestimates of incidence changes in some locations and underestimates in other locations, but these location-specific biases will, to some extent, cancel each other out when the total incidence change is calculated. It is not possible to know the extent or direction of the bias in the total incidence change based on the general application of a single C-R function everywhere.

The appropriate economic value of a change in a health effect depends on whether the health effect is viewed ex ante (before the effect has occurred) or ex post (after the effect has occurred). Reductions in ambient concentrations of air pollution generally lower the risk of future adverse health affects by a fairly small amount for a large population. The appropriate economic measure is therefore ex ante WTP for changes in risk. However, epidemiological studies generally provide estimates of the relative risks of a particular health effect avoided due to a reduction in air pollution. A convenient way to use this data in a consistent framework is to convert probabilities to units of avoided statistical incidences. This measure is calculated by dividing individual WTP for a risk reduction by the related observed change in risk. For example, suppose a measure is able to reduce the risk of premature mortality from 2 in 10,000 to 1 in 10,000 (a reduction of 1 in 10,000). If individual WTP for this risk reduction is $100, then the WTP for an avoided statistical premature mortality amounts to $1 million ($100/0.0001 change in risk). Using this approach, the size of the affected population is automatically taken into account by the number of incidences predicted by epidemiological studies applied to the relevant population. The same type of calculation can produce values for statistical incidences of other health endpoints.

For some health effects, such as hospital admissions, WTP estimates are generally not available. In these cases, we use the cost of treating or mitigating the effect as a primary estimate. For example, for the valuation of hospital admissions we use the avoided medical costs as an estimate of the value of avoiding the health effects causing the admission. These costs of illness (COI) estimates generally understate the true value of reductions in risk of a health effect. They tend to reflect the direct expenditures related to treatment but not the value of avoided pain and suffering from the health effect. Table VII-15 summarizes the value estimates per health effect that we used in this analysis. Note that the unit values for hospital admissions are the weighted averages of the ICD-9 code-specific values for the group of ICD-9 codes included in the hospital admission categories. Details of the derivation of values for hospital admissions can be found in the benefits TSD for this RIA (Abt Associates, 2000).

In the following sections, we describe individual health endpoints and the C-R functions we have selected to provide quantified estimates of the avoided health effects associated with the final HD Engine/Diesel Fuel rule. In addition, we discuss how these changes in health effects should be valued and indicate the value functions selected to provide monetized estimates of the value of changes in health effects.

Table VII-15. Unit Values Used for Economic Valuation of Health Endpoints

Health or Welfare Endpoint	Estimated Value Per Incidence (1999$) Central Estimate	Derivation of Estimates
Premature Mortality	$6 million per statistical life	Value is the mean of value-of-statistical-life estimates from 26 studies (5 contingent valuation and 21 labor market studies) reviewed for the Section 812 Costs and Benefits of the Clean Air Act, 1990-2010 (US EPA, 1999).
Chronic Bronchitis (CB)	$331,000	Value 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) 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.
Hospital Admissions		
Chronic Obstructive Pulmonary Disease (COPD) (ICD codes 490-492, 494-496)	$12,378	The COI estimates 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 Elixhauser (1993).
Pneumonia (ICD codes 480-487)	$14,693	The COI estimates 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 Elixhauser (1993).
Asthma admissions	$6,634	The COI estimates 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 Elixhauser (1993).
All Cardiovascular (ICD codes 390-429)	$18,387	The COI estimates 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 illnesses) reported in Elixhauser (1993).
Emergency room visits for asthma	$299	COI estimate based on data reported by Smith, et al. (1997).

Table VII-15. Unit Values Used for Economic Valuation of Health Endpoints

Health or Welfare Endpoint	Estimated Value Per Incidence (1999$) Central Estimate	Derivation of Estimates
Respiratory Ailments Not Requiring Hospitalization		
Upper Respiratory Symptoms (URS)	$24	Combinations of the 3 symptoms for which WTP estimates are available that closely match those listed by Pope, et al. result in 7 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) 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 7 different types of URS.
Lower Respiratory Symptoms (LRS)	$15	Combinations of the 4 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.
Acute Bronchitis	$57	Average of low and high values recommended for use in Section 812 analysis (Neumann, et al. 1994)
Restricted Activity and Work Loss Days		
Work Loss Days (WLDs)	Variable	Regionally adjusted median weekly wage for 1990 divided by 5 (adjusted to 1999$) (US Bureau of the Census, 1992).
Minor Restricted Activity Days (MRADs)	$48	Median WTP estimate to avoid one MRAD from Tolley, et al. (1986) .

a. Premature Mortality: Quantification

Both acute and chronic exposures to ambient levels of air pollution have been associated with increased risk of premature mortality. Because of the extreme nature of this endpoint and the high monetary value associated with risks to life, reductions in the risk of premature mortality are the most important health endpoints quantified in this analysis. Although these endpoints account for over 90 percent of the total monetized benefits, considerable uncertainty exists, both among economists and policymakers, as to the appropriate way to value reductions in mortality risks. Because of these factors, we include a more detailed discussion for premature mortality than for other health effects.

Health researchers have consistently linked air pollution, especially PM, with increases in premature mortality. A substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates. Much of this literature is

summarized in the 1996 PM Criteria Document (US EPA, 1996a). There is much about this relationship that is still uncertain. As stated in preamble to the 1997 PM National Ambient Air Quality Standards (40 CFR 50, 1997), "the consistency of the results of the epidemiological studies from a large number of different locations and the coherent nature of the observed effects are suggestive of a likely causal role of ambient PM in contributing to the reported effects," which include premature mortality. The National Academy of Sciences, in their report on research priorities for PM (NAS, 1998), indicates that "there is a great deal of uncertainty about the implications of the findings [of an association between PM and premature mortality] for risk management, due to the limited scientific information about the specific types of particles that might cause adverse health effects, the contributions of particles of outdoor origin to actual human exposures, the toxicological mechanisms by which the particles might cause adverse health effects, and other important questions." EPA acknowledges these uncertainties; however, for this analysis, we assume a causal relationship between exposure to elevated PM and premature mortality, based on the consistent evidence of a correlation between PM and mortality reported in the scientific literature (US EPA, 1996a).

In addition, it is currently unknown whether there is a time lag (a delay between changes in PM exposures and changes in mortality rates) in the chronic PM/premature mortality relationship. The existence of such a lag is important for the valuation of premature mortality incidences because economic theory suggests that benefits occurring in the future should be discounted. Although there is no specific scientific evidence of the existence or structure of a PM effects lag, current scientific literature on adverse health effects, such as those associated with PM (e.g., smoking-related disease) and the difference in the effect size between chronic exposure studies and daily mortality studies suggest that all incidences of premature mortality reduction associated with a given incremental change in PM exposure probably would not occur in the same year as the exposure reduction. This same smoking-related literature implies that lags of up to a few years are plausible. Adopting the lag structure used in the Tier 2/Gasoline Sulfur RIA and endorsed by the SAB (EPA-SAB-COUNCIL-ADV-00-001, 1999), we assume a five-year lag structure, with 25 percent of premature deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years. To explore the uncertainty surrounding this lag structure, Appendix VII-A contains a sensitivity analysis showing how different lag structures affect the estimated value of reductions in premature mortality.

Two types of exposure studies (short-term and long-term exposure) have been used to estimate a PM/premature mortality relationship. Short-term exposure studies attempt to relate short-term (often day-to-day) changes in PM concentrations and changes in daily mortality rates up to several days after a period of elevated PM concentrations. Long-term exposure studies examine the potential relationship between longer-term (e.g., one or more years) exposure to PM and annual mortality rates. Researchers have found significant associations using both types of studies (US EPA, 1996a); however, for this analysis, we follow SAB advice (EPA-SAB-COUNCIL-ADV-99-005, 1999), and we rely exclusively on long-term exposure studies to quantify mortality effects.

Following advice from the SAB (EPA-SAB-COUNCIL-ADV-99-005, 1999), we prefer to use long-term exposure studies that employ a prospective cohort design over those that use an ecologic or population-level design. Prospective cohort studies follow individuals forward in time for a specified period, periodically evaluating each individual's exposure and health status. While the long-term exposure study design is preferred, they are expensive to conduct and consequently there are relatively few well designed long-term exposure studies. For PM, there have been only a few, and the SAB has explicitly recommended use of only one — the American Cancer Society (ACS) Study, as reported in Pope, et al. (1995) (EPA-SAB-COUNCIL-ADV-99-005, 1999). The data from this study were reanalyzed and we used a C-R function from the HEI reanalysis (ACS/Krewski et al., 2000).

The ACS/Pope, et al. study used a prospective cohort design to estimate the risk of premature mortality from long-term exposures to ambient PM concentrations. The ACS/Pope, et al. study is recommended in preference to other available long-term studies because it uses better statistical methods, has a much larger sample size and uses the longer exposure intervals, and more locations (50 cities) in the U.S. than other studies. Recently, the Health Effects Institute (HEI), a non-profit, independent research organization commissioned an extensive reanalysis of the data used in the ACS/Pope, et al. (1995) study.[r]

The HEI reanalysis, as reported in Krewski, et al. (2000) and mentioned above, confirmed the general findings of the ACS/Pope, et al (1995) study. In addition, the reanalysis tested a number of alternative model specifications, some of which may be preferred to the original ACS/Pope, et al. (1995) specification. One important alternative specification examines the relationship between relative risk of premature mortality and mean $PM_{2.5}$ levels rather than median levels used in the Pope, et al. (1995) analysis (Table 31, "$PM_{2.5}$(DC)" model). For policy analysis purposes, functions based on the mean air quality levels may be preferable to functions based on the median air quality levels because changes in the mean more accurately reflect changes in peak values than do changes in the median. Policies which affect peak PM days more than average PM days will result in a larger change in the mean than in the median. In these cases, all else being equal, C-R functions based on median $PM_{2.5}$ will lead to lower estimates of avoided incidences of premature mortality than C-R functions based on mean $PM_{2.5}$. In addition to specifying a preference for the ACS study based on the larger set of cities examined, the SAB has also noted a preference for applying mean $PM_{2.5}$ in premature mortality functions (US EPA-SAB, 1999). For these reasons, we have selected the C-R function based on the relative risk of 1.12 from the "PM2.5(DC), All Causes" model reported in Table 31 of the HEI report.[s]

[r]Additional information on the Health Effects Institute and the reanalysis of the Harvard Six Cities and American Cancer Society Studies can be obtained at http://www.healtheffects.org.

[s] Note that in several recent RIAs, we erroneously applied the ACS/Pope et al. C-R function to a baseline of non-accidental mortality. The correct baseline, matching the mortality measured in the ACS/Pope et al. and Krewski et al. studies is all-cause mortality. This correction results in a slight increase in the estimated mortality reductions resulting from a reduction in $PM_{2.5}$.

Although we use the Krewski, et al. (2000) mean-based ("PM2.5(DC), All Causes") model exclusively to derive our primary estimates of avoided premature mortality, we also examine the impacts of selecting alternative C-R functions for premature mortality. There are several candidates for alternative C-R functions, some from the Krewski, et al. study, and others from the original ACS study by Pope, et al. or from the "Harvard Six-City Study" by Dockery, et al. (1993).

Commentary by an independent review panel noted that "a major contribution of the [HEI] Reanalysis Project is the recognition that both pollutant variables and mortality appear to be spatially correlated in the ACS data set. If not identified and modeled correctly, spatial correlation could cause substantial errors in both the regression coefficients and their standard errors (HEI, 2000)." The HEI reanalysis provides results for several models which control for spatial correlations in the data. These models are based on the original ACS air quality dataset, which contained only median $PM_{2.5}$ concentrations. Ideally, our primary C-R function for premature mortality would be both based on the mean and adjusted for regional variability. Unfortunately, Krewski, et al. do not provide such an estimate. As such, we have chosen to use the mean-based relative risk in our primary analysis and to use the median-based regionally adjusted relative risks to provide alternative estimates exploring the impact of adjustments for spatial correlations (see Table VII-16).

Krewski, et al. (2000) also reanalyzed the data from another prospective cohort study (the Harvard "Six Cities Study") authored by Dockery, et al. (1993). The Dockery, et al.(1993) study used a smaller sample of individuals from fewer cities than the study by Pope, et al.; however, it features improved exposure estimates, a slightly broader study population (adults aged 25 and older), and a follow-up period nearly twice as long as that of Pope, et al. 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). The Dockery, et al. (1993) study finds a larger effect of PM on premature mortality relative to the Pope, et al. (1995) study. To provide a more complete picture of the range of possible premature mortality risks that may be associated with long-term exposures to fine particles, we also present alternative estimates based on the Krewski, et al. (2000) reanalysis of the Dockery, et al. (1993) data and the original study estimates. The HEI commentary notes that "the inherent limitations of using only six cities, understood by the original investigators, should be taken into account when interpreting the results of the Six Cities Study." We emphasize, that based on our understanding of the relative merits of the two datasets, the Krewski, et al. (2000) ACS model based on mean $PM_{2.5}$ levels in 63 cities is the most appropriate model for analyzing the premature mortality impacts of the HD Engine/Diesel Fuel rule. It is thus used for our primary estimate of this important health effect**.**

Table VII-16 summarizes the alternative C-R functions for PM-related premature mortality. Note that the right most column provides a standardized estimate of the incidences of premature mortality that would be reduced by a one microgram reduction in $PM_{2.5}$ applied to a population of one million. Note that the relative magnitude of the values will not necessarily correlate with the estimates of avoided incidences that will result from application of the HD Engine/Diesel Fuel reductions in $PM_{2.5}$ to 2030 national populations. This is because some of the functions are based on changes in mean $PM_{2.5}$ concentrations while others are based on median

$PM_{2.5}$ concentrations. Estimated reductions in premature mortality will depend on both the size of the C-R coefficient and the change in the relevant $PM_{2.5}$ metric (mean or median).

Table VII-16. Alternative Concentration-Response Models Relating Premature Mortality and Chronic Exposure to Fine Particulates

Model Description (as listed in the study)	# of Cities	PM Metric	Reported Relative Risk[A] (95% Confidence Interval)	Avoided Incidences of Premature Mortality per Million Population for a 1 µg/m^3 Decrease in $PM_{2.5}$[B]
PM2.5(DC), All Causes Source: Table 31, Krewski, et al. (2000)	63	Mean	1.12 (1.06-1.19)	68
Fine Particles Alone, Random Effects, Regional Adjustment Source: Table 46, Krewski, et al. (2000)	50	Median	1.16 (0.99-1.37)	89
Fine Particles Alone, Random Effects, Independent Cities Source: Table 46, Krewski, et al. (2000)	50	Median	1.29 (1.12-1.48)	152
All Combined, All Cause, Fine Particles Source: Table 3, Pope, et al. (1995)[C]	50	Median	1.17 (1.09-1.26)	90
All Causes, Extended, Age Time Axis: Table 3, Krewski, et al. (2000)	6	Mean	1.27 (1.09-1.48)	173
All Subjects Source: Table 3, Dockery, et al. (1993)	6	Mean	1.26 (1.08-1.47)	153

[A] Reported relative risks for the Pope, et al. (1995) and Dockery, et al. (1993) studies are comparisons of mortality rates between most polluted and least polluted cities. For the Pope et al. study the relative risk is based on a difference in median PM2.5 levels of 24.5 µg/m^3. For the Dockery, et al. study, the relative risk is based on a difference of 18.6 µg/m^3. The Krewski, et al. reanalysis of the Pope, et al. study reports all relative risks based on a 24.5 µg/m^3 difference for comparability with the Pope, et al. (1995) results, rather than comparing the means or medians of the most polluted and least polluted studies. Likewise, the Krewski, et al. reanalysis of the Dockery, et al. Harvard Six Cities study reports all relative risks based on a 18.6 µg/m^3 difference for comparability with the Dockery, et al. (1993) study.

[B] Assumes national all-cause mortality rate of 0.0147 per person for adults aged 30 and older and 0.0131 per person for adults aged 25 and older. (US Centers for Disease Control. 2000 *National Vital Statistics Reports* 48(11): Table 8).

[c] The Pope, et al. estimate of the relative risk of premature mortality from fine particle exposure is the basis for the estimates of premature mortality found in the final Tier 2/Gasoline Sulfur rule.

b. Premature Mortality: Valuation

We estimate the monetary benefit of reducing premature mortality risk using the "value of statistical lives saved" (VSL) approach, even though the actual valuation is of small changes in mortality risk experienced by a large number of people. The VSL approach applies information from several published value-of-life studies to determine a reasonable benefit of preventing premature mortality. The mean value of avoiding one statistical death is estimated to be $6 million in 1999 dollars. This represents an intermediate value from a variety of estimates that appear in the economics literature, and it is a value EPA has frequently used in RIAs for other rules and in the Section 812 Reports to Congress.

This estimate is the mean of a distribution fitted to the estimates from 26 value-of-life studies identified in the Section 812 reports as "applicable to policy analysis." The approach and set of selected studies mirrors that of Viscusi (1992) (with the addition of two studies), and uses the same criteria as Viscusi in his review of value-of-life studies. The $6 million estimate is consistent with Viscusi's conclusion (updated to 1999$) that "most of the reasonable estimates of the value of life are clustered in the $3.7 to $8.6 million range." Five of the 26 studies are contingent valuation (CV) studies, which directly solicit WTP information from subjects; the rest are wage-risk studies, which base WTP estimates on estimates of the additional compensation demanded in the labor market for riskier jobs. As indicated in the previous section on quantification of premature mortality benefits, we assume for this analysis that some of the incidences of premature mortality related to PM exposures occur in a distributed fashion over the five years following exposure. To take this into account in the valuation of reductions in premature mortality, we apply an annual three percent discount rate to the value of premature mortality occurring in future years.[t]

The economics literature concerning the appropriate method for valuing reductions in premature mortality risk is still developing. The adoption of a value for the projected reduction in the risk of premature mortality is the subject of continuing discussion within the economic and public policy analysis community. Regardless of the theoretical economic considerations, EPA prefers not to draw distinctions in the monetary value assigned to the lives saved even if they differ in age, health status, socioeconomic status, gender or other characteristic of the adult population.

Following the advice of the EEAC of the SAB, EPA currently uses the VSL approach in calculating the primary estimate of mortality benefits, because we believe this calculation to provide the most reasonable single estimate of an individual's willingness to trade off money for

[t] The choice of a discount rate, and its associated conceptual basis, is a topic of ongoing discussion within the federal government. EPA adopted a 3 percent discount rate for its primary analysis in this case to reflect reliance on a "social rate of time preference" discounting concept. We have also calculated benefits and costs using a 7 percent rate consistent with an "opportunity cost of capital" concept to reflect the time value of resources directed to meet regulatory requirements. In this case, the benefit and cost estimates were not significantly affected by the choice of discount rate. Further discussion of this topic appears in EPA's *Guidelines for Preparing Economic Analyses* (in press).

reductions in mortality risk (EPA-SAB-EEAC-00-013). While there are several differences between the labor market studies EPA uses to derive a VSL estimate and the particulate matter air pollution context addressed here, those differences in the affected populations and the nature of the risks imply both upward and downward adjustments. Table VII-17 lists some of these differences and the expected effect on the VSL estimate for air pollution-related mortality. For example, adjusting for age differences may imply the need to adjust the $6 million VSL downward, but the involuntary nature of air pollution-related risks and the lower level of risk-aversion of the manual laborers in the labor market studies may imply the need for upward adjustments. In the absence of a comprehensive and balanced set of adjustment factors, EPA believes it is reasonable to continue to use the $6 million value while acknowledging the significant limitations and uncertainties in the available literature.

Some economists emphasize that the value of a statistical life is not a single number relevant for all situations. Indeed, the VSL estimate of $6 million (1999 dollars) is itself the central tendency of a number of estimates of the VSL for some rather narrowly defined populations. When there are significant differences between the population affected by a particular health risk and the populations used in the labor market studies, as is the case here, some economists prefer to adjust the VSL estimate to reflect those differences. Some of the alternative approaches that have been proposed for valuing reductions in mortality risk are discussed in Figure VII-6.

There is general agreement that the value to an individual of a reduction in mortality risk can vary based on several factors, including the age of the individual, the type of risk, the level of control the individual has over the risk, the individual's attitudes towards risk, and the health status of the individual. While the empirical basis for adjusting the $6 million VSL for many of these factors does not yet exist, a thorough discussion of these factors is contained in the benefits TSD for this RIA (Abt Associates, 2000). EPA recognizes the need for investigation by the scientific community to develop additional empirical support for adjustments to VSL for the factors mentioned above.

Table VII-17. Expected Impact on Estimated Benefits of Premature Mortality Reductions of Differences Between Factors Used in Developing Applied VSL and Theoretically Appropriate VSL

Attribute	Expected Direction of Bias
Age	Uncertain, perhaps overestimate
Attitudes toward risk	Underestimate
Income	Uncertain
Voluntary vs. Involuntary	Uncertain, perhaps underestimate
Catastrophic vs. Protracted Death	Uncertain, perhaps underestimate

Figure VII-6. Alternative Approaches for Assessing the Value of Reduced Mortality Risk

Stated preference studies – These studies use survey responses to estimate WTP to avoid risks. *Strengths:* flexible approach allowing for appropriate risk context, good data on WTP for individuals. *Weaknesses:* risk information may not be well-understood by respondents and questions may be unfamiliar.

Consumer market studies – These studies use consumer purchases and risk data (e.g., smoke detectors) to estimate WTP to avoid risks. *Strengths:* uses revealed preferences and is a flexible approach. *Weaknesses:* very difficult to estimate both risk and purchase variables.

Value of statistical life year (VSLY) – Provides an annual equivalent to value of statistical life estimates. *Strengths:* provides financially accurate adjustment for age at death. *Weaknesses:* adjustment may not reflect how individuals consider life-years; assumes equal value for all remaining life-years.

Quality adjusted life year – Applies quality of life adjustment to life-extension data, uses cost-effectiveness data to value. *Strengths:* widely used in public health literature to assess private medical interventions. *Weaknesses:* lack of data on health state indices and life quality adjustments that are applicable to an air pollution context. Similar to VSLY, adjustment may not reflect how individuals consider life-years, and typically assumes an equal value for all remaining life-years despite evidence to the contrary.

WTP for a change in survival curve – Reflects WTP for change in risk, potentially incorporates age-specific nature of risk reduction. *Strengths:* theoretically preferred approach that most accurately reflects risk reductions from air pollution control. *Weaknesses:* almost no empirical literature available; difficulty in obtaining reliable values.

WTP for a change in longevity – Uses stated preference approach to generate WTP for longevity or longer life expectancy. *Strengths:* life expectancy is a familiar term to most individuals. *Weaknesses:* does not incorporate age-specific risk information; problems in adapting to air pollution context.

Cost-effectiveness – Determines the implicit cost of saving a life or life-year. *Strengths:* widely used in public health contexts. *Weaknesses:* health context is for private goods, dollar values do not necessarily reflect individual preferences.

One important factor in Figure VII-6 for which the impact on total benefits can be illustrated is the difference in age distribution between the population affected by air pollution and the population for which most of the VSL estimates were developed. In the recent Tier 2/Gasoline Sulfur benefits analysis, we employed a value of statistical life years (VSLY) approach developed for the Section 812 studies in exploring the impact of age on VSL. Since the VSLY alternative calculation was introduced in the Section 812 studies, the SAB raised new and additional concerns about the merits of the VSLY approach. Specifically, they note in their recent report that "inferring the value of a statistical life year, however, requires assumptions about the discount rate and about the time path of expected utility of consumption" (EPA-SAB-EEAC-00-013). In considering the merits of age-based adjustments, the Committee also notes that "the theoretically appropriate method is to calculate WTP for individuals whose ages correspond to those of the affected population, and that it is preferable to base these calculations on empirical estimates of WTP by age." Several studies conducted by Jones-Lee, et al. (1985, 1989, 1993) found a significant effect of age on the value of mortality risk reductions expressed

by citizens in the United Kingdom. Using the results of the Jones-Lee et al. analysis, U.S. EPA (2000b) calculated ratios of the value of life for different age groups to the mean value of life estimated by Jones-Lee, et al. (1989, 1993). The Jones-Lee-based analysis suggests a U-shaped relationship between age and VSL, peaking around age 40, and declining to between 60 and 90 percent of the mean VSL value for individuals over the age of 70, and declining further as individuals age. This finding has been supported by two recent analyses conducted by Krupnick, et al. (2000a, 2000b), which asked samples of Canadian and U.S. residents their values for reductions in mortality risk. We apply the ratios based on the Jones-Lee, et al. (1989, 1993) studies to the estimated premature mortalities within the appropriate age groups to provide an alternative age-adjusted estimate of the value of avoided premature mortalities. However, we have not attempted in this analysis to provide a consistent treatment of age-dependence between the underlying wage-risk studies and the present calculation. Therefore, the downward adjustment for age relative to our primary benefit estimate may be significantly overestimated, implying a significant underestimation of age-adjusted total benefits.

The SAB-EEAC advised in their recent report that the EPA "continue to use a wage-risk-based VSL as its primary estimate, including appropriate sensitivity analyses to reflect the uncertainty of these estimates," and that "the only risk characteristic for which adjustments to the VSL can be made is the timing of the risk"(EPA-SAB-EEAC-00-013). In developing our primary estimate of the benefits of premature mortality reductions, we have discounted over the lag period between exposure and premature mortality. However, in accordance with the SAB advice, we use the VSL in our primary estimate and present the Jones-Lee calculations in the table of alternative calculations, Table VII-25.

c. Chronic Bronchitis: Quantification

Chronic bronchitis is characterized by mucus in the lungs and a persistent wet cough for at least three months a year for several years in a row. Chronic bronchitis affects an estimated five percent of the U.S. population (American Lung Association, 1999). There are a limited number of studies that have estimated the impact of air pollution on new incidences of chronic bronchitis. Schwartz (1993) and Abbey, et al.(1995) provide evidence that long-term PM exposure gives rise to the development of chronic bronchitis in the U.S. Following the same approaches, the Section 812 Prospective Report (US EPA, 1999a), our analysis pools the estimates from these studies to develop a C-R function linking PM to chronic bronchitis. The Schwartz (1993) study examined the relationship between exposure to PM_{10} and prevalence of chronic bronchitis. The Abbey, et al. (1995) study examined the relationship between $PM_{2.5}$ and new incidences of chronic bronchitis. Both studies have strengths and weaknesses which suggest that pooling the effect estimates from each study may provide a better estimate of the expected change in incidences of chronic bronchitis than using either study alone. However, the HD Engine/Diesel Fuel rule is expected to result in reductions in both the fine and coarse fractions of PM_{10}. As such, reliance on the Abbey, et al. (1995) estimate will result in an underestimate of the change in chronic bronchitis incidences if both the fine and coarse fractions of PM_{10} are associated with chronic bronchitis. To address this problem, we apply the C-R functions from both Schwartz (1993) and Abbey, et al. (1995) to generate the changes in chronic bronchitis incidences associated with the change in $PM_{2.5}$ and then pool the incidence estimates to obtain a

primary estimate of avoided $PM_{2.5}$ related chronic bronchitis incidences. We then apply the Schwartz (1993) C-R function to the change in coarse PM ($PM_{2.5-10}$) to obtain a primary estimate of avoided incidences of chronic bronchitis due to the change in coarse fraction PM. The primary estimate of total avoided incidences is then the sum of the avoided incidences from changes in $PM_{2.5}$ and $PM_{2.5-10.}$[u]

It should be noted that Schwartz used data on the *prevalence* of chronic bronchitis, not its *incidence*. Following the Section 812 Prospective Report, we assume that it is appropriate to estimate the percentage change in the prevalence rate for chronic bronchitis using the estimated coefficient from Schwartz's study in a C-R function, and then to assume this percentage change applies to a baseline incidence rate obtained from another source. For example, if the prevalence declines by 25 percent with a drop in PM, then baseline incidence drops by 25 percent with the same drop in PM.

d. Chronic Bronchitis: Valuation

The best available estimate of WTP to avoid a case of chronic bronchitis (CB) comes from Viscusi, et al. (1991).[v] The Viscusi, et al. study, however, describes a severe case of CB to the survey respondents. We therefore employ an estimate of WTP to avoid a pollution-related case of CB, based on adjusting the Viscusi, et al. (1991) estimate of the WTP to avoid a severe case. This is done to account for the likelihood that an average case of pollution-related CB is not as severe. The adjustment is made by applying the elasticity of WTP with respect to severity reported in the Krupnick and Cropper (1992) study. Details of this adjustment procedure are provided in the benefits TSD for this RIA (Abt Associates, 2000).

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of CB in this analysis. The distribution incorporates uncertainty from three sources: (1) the WTP to avoid a case of severe CB, as described by Viscusi, et al.; (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi, et al.); and (3) the elasticity of WTP with respect to severity of the illness. Based on assumptions about the distributions of each of these three uncertain components, we derive a distribution of WTP to avoid a pollution-related case of CB by statistical uncertainty analysis techniques. The expected value (i.e., mean) of this distribution,

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[u] This assumption implies that the observed relationship between chronic bronchitis and PM_{10} in the Schwartz (1993) study is equally attributable to the fine and coarse fractions of PM_{10}. If the relationship is due primarily to the fine fraction, then the estimate of avoided incidences associated with coarse fraction PM changes will be overstated. However, if this is the case then the estimate of avoided incidences associated with fine fraction will be somewhat understated. The net effect on avoided incidences of chronic bronchitis is ambiguous.

[v] The Viscusi, et al. (1991) study was an experimental study intended to examine new methodologies for eliciting values for morbidity endpoints. Although these studies were not specifically designed for policy analysis, the SAB (EPA-SAB-COUNCIL-ADV-00-002, 1999) has indicated that the severity-adjusted values from this study provide reasonable estimates of the WTP for avoidance of chronic bronchitis. As with other contingent valuation studies, the reliability of the WTP estimates depends on the methods used to obtain the WTP values.

which is about $331,000 (1999$), is taken as the central tendency estimate of WTP to avoid a PM-related case of CB.

e. Hospital and Emergency Room Admissions: Quantification

There is a wealth of epidemiological information on the relationship between air pollution and hospital admissions for various respiratory and cardiovascular diseases; in addition, some studies have examined the relationship between air pollution and emergency room (ER) visits. Because most ER visits do not result in an admission to the hospital (the majority of people going to the ER are treated and return home) we treat hospital admissions and ER visits separately, taking account of the fraction of ER visits that are admitted to the hospital.

Hospital admissions require the patient to be examined by a physician, and on average may represent more serious incidents than ER visits. The two main groups of hospital admissions estimated in this analysis are respiratory admissions and cardiovascular admissions. There is not much evidence linking ozone or PM with other types of hospital admissions. The only type of ER visits that have been linked to ozone and PM in the U.S. are asthma-related visits.

i. PM-related Hospital Admissions

To estimate avoided incidences of hospital admissions associated with PM, we use a study by Samet, et al. (2000) which examined the relationship between PM_{10} and admissions for pneumonia, chronic obstructive pulmonary disease (COPD), and cardiovascular disease in fourteen U.S. cities. In previous analyses, we have pooled estimates from a number of studies in different cities. However, Samet, et al. (2000) represents a comprehensive analysis of the relationship between hospital admissions and air pollution conducted under the auspices of the Health Effects Institute as part of the National Morbidity, Mortality, and Air Pollution study. This extensive analysis by the HEI was intended to provide a consistent, comparable set of effects estimates over a wide range of cities. As such, the pooled estimates of relative risk for pneumonia, COPD, and cardiovascular disease provided by the study (Table 14, "Unconstrained distributed lag, Random effects estimate"), which covers most of the studies included individually in previous benefits analyses, represents the most up-to-date estimate of the relationship between PM air pollution and hospital admissions. One study (Moolgavkar, 1997) found a much lower effect of PM on hospital admissions for pneumonia and COPD. The effect of using Moolgavkar (1997) instead of Samet, et al. (2000) is presented as a alternative calculation in Table VII-25.

The Samet, et al. (2000) HEI analysis estimated separate C-R functions for pneumonia and COPD hospital admissions for people 65 years and older. In addition, Sheppard, et al. (1999) estimated a C-R function for asthma hospital admissions for people under age 65. These three estimates can be combined to calculate total avoided incidences of PM-related respiratory-related hospital admissions.

To estimate the effects of PM air pollution reductions on asthma-related ER visits, we use the C-R function based on a study of Seattle residents by Schwartz, et al. (1993). Because we are

estimating ER visits as well as hospital admissions for asthma, we must avoid counting twice the ER visits for asthma that are subsequently admitted to the hospital. To avoid double-counting, the baseline incidence rate for ER visits is adjusted by subtracting the percentage of patients that are admitted into the hospital. The reported incidence rates suggest that ER visits for asthma occur 2.7 times as frequently as hospital admissions for asthma. The baseline incidence of asthma ER visits is therefore taken to be 2.7 times the baseline incidence of hospital admissions for asthma. To avoid double-counting, however, only 63 percent of the resulting change in asthma ER visits associated with a given change in pollutant concentrations is counted in the ER visit incidence change.

ii. Ozone-related Hospital Admissions

To estimate avoided incidences of hospital admissions associated with ozone, we use a number of studies examining hospital admissions for a range of respiratory illnesses and one study examining hospital admissions for cardiac dysrythmias. Hospital admissions for respiratory diseases studied include admissions for pneumonia, COPD, asthma, and a number of other respiratory illnesses. Hospital admissions for cardiac dysrythmias are estimated using a C-R function derived from Burnett, et al. (1999).

f. Hospital Admissions: Valuation

An individual's WTP to avoid a hospital admission will include, at a minimum, the amount of money he or she pays for medical expenses (i.e., payment towards the hospital charge and the associated physician charge) and the loss in earnings. In addition, an individual is likely to be willing to pay some amount to avoid the pain and suffering associated with the illness itself. Even if they incurred no medical expenses and no loss in earnings, most individuals would still be willing to pay something to avoid the illness.

In the absence of estimates of WTP to avoid hospital admissions for specific illnesses, estimates of total cost-of-illness (COI) are typically used although they underestimate the benefits. These estimates are biased downward because they do not include the value of avoiding the illness itself. Some analyses adjust COI estimates upward by multiplying by an estimate of the ratio of WTP to COI, to better approximate total WTP. Other analyses have avoided making this adjustment because of the possibility of over adjusting -- that is, possibly replacing a known downward bias with an upward bias. Consistent with the advice offered by the SAB, the COI values used in this benefits analysis will not be adjusted (EPA-SAB-COUNCIL-ADV-98-003, 1998).

For the valuation of avoided respiratory and cardiovascular hospital admissions, the current literature provides well-developed and detailed cost estimates of hospitalization by health effect or illness. Using illness-specific estimates of avoided medical costs and avoided costs of lost work-time that Elixhauser (1993) developed, we construct COI estimates specific to the suite of health effects defined by each C-R function. Using the methods developed for the Section 812 reports, ICD-code-specific COI estimates were generated based on estimated hospital charges and the estimated opportunity cost of time spent in the hospital (estimated as the value of the lost

daily wage, regardless of whether or not the individual is in the workforce). The value of an avoided asthma-related ER visit is based on data reported in Smith, et al. (1997). The average cost per ER visit reported in this study (1999$) is $298.62.

g. Asthma Attacks: Quantification

Asthma is the most prevalent chronic disease among children in the U.S., affecting over seven percent of children under 18 years old (US CDC, 1998). Among adults, it currently affects over six percent of the U.S. population (US CDC, 1998). Asthma attacks are a serious health effect for people with asthma. During an attack, muscles around the airways constrict, the airways become inflamed, and less air passes in and out of the lungs. The attack is also called an episode or exacerbation and can include coughing, chest tightness, wheezing, and difficulty breathing (Jack, Boss, and Millington, 2000). The literature supports a direct relationship between air pollution and increased incidence and severity of asthma-related respiratory symptoms. Studies have documented this relationship for both PM (Yu, et al., 2000; McConnell et al., 1999; Delfino et al., 1998; Delfino et al., 1997; US EPA, 1996a; Ostro et al., 1995; Whittemore and Korn, 1980) and ozone (Delfino et al., 1998; Thurston et al., 1997; US EPA, 1996b; Delfino et al., 1996; Ostro et al., 1995; US EPA, 1986; Whittemore and Korn, 1980).

There are a number of these studies showing a relationship between PM and/or ozone levels and asthma-related respiratory symptoms such as wheezing, coughing, acute bronchitis and shortness of breath. However, only one study (Whittemore and Korn, 1980) estimated the relationship between asthma attacks and photochemical air pollutant concentrations. The likely reason for the emphasis of most studies on particular asthma symptoms is the subjective definition of an asthma attack and the subsequent lack of specificity in measuring an asthma attack occurrence. In this analysis, the endpoint "asthma attack" is a better match for the economic valuation studies and avoids potential overprediction (as one attack may involve some combination of symptoms). Accordingly, an asthma attack is an endpoint that summarizes the collection of symptoms, so potential double-counting may occur if individual asthma symptoms estimated from other studies are summed. An asthma attack, as measured by Whittemore and Korn (1980), is based on subjective reporting by study participants and likely consisting of one or more of the respiratory symptoms listed above occurring at varying levels of severity. For example, a subject reporting an asthma attack in the Whittemore and Korn (1980) study may have shortness of breath and wheezing. This is accounted for as one attack, while using individual symptom studies would record this as two separate symptom occurrences. Conversely, a participant may experience symptoms but not consider the symptoms to be "an attack." Thus, the use of "asthma attacks" as an indicator may understate symptoms.

In addition, a limited number of economic studies have been conducted on the value of reduced asthma symptoms. One valuation study by Rowe and Chestnut (1986) calculated the value of reduction in "bad asthma days," which we interpret as equivalent to a day with an asthma attack. By using the Whittemore and Korn (1980) asthma attack C-R function in combination with the Rowe and Chestnut (1986) valuation study, we are able to provide a quantified and monetized estimate of asthma-related symptoms that is representative of the full spectrum of impacts of air pollution reductions on asthma sufferers.

Although the Whittemore and Korn (1980) study had a number of methodological flaws, including omission of some potentially confounding variables and use of proxies for ozone and PM[w], we believe that the more recent literature supports the general magnitude of the relationship. As such, we use Whittemore and Korn in our primary analysis to estimate the effects of air pollution on asthma symptoms, recognizing that the Whittemore and Korn based estimate represents symptoms examined in other studies, though perhaps undercounting the frequency of symptom occurrence. Other analyses of the impacts of air pollution reductions on asthma symptoms have used collections of asthma symptom studies (Kunzli et al., 2000). However, we believe it is more illustrative to provide a single endpoint that represents a combination of symptoms. The Whittemore and Korn study was also previously used to estimate asthma attacks in the Section 812 analysis (although it was not included in the primary estimate of total benefits), which was reviewed and accepted by the EPA SAB. Table VII-18 provides a summary of the more recent studies of air pollution and respiratory symptoms in asthmatics. Also, several asthma-related endpoints are provided as supplementary calculations in Appendix VII-A to this chapter.

Note that the estimated number of avoided asthma attacks is the total change over the full population of asthmatics, potentially including multiple avoided attacks for a single individual. Also, because our estimate of asthma attacks is based on both the incidence of asthma attacks and the prevalence of asthma in the population, to the extent that asthma incidence rates are increasing (or decreasing), the number of asthma attacks avoided will also be increasing (or decreasing). The prevalence of asthma, especially among children, has been increasing over the past two decades (Pew Environmental Health Commission, 2000), suggesting that if current trends continue, the impact on asthma symptoms of reductions in air pollution will be greater than we estimated in this analysis.

h. Asthma Attacks: Valuation

In the primary analysis, we do not present a monetized value. As an alternative, asthma attacks are valued at $41 per incidence (1999$), based on the mean of average WTP estimates for the four severity definitions of a “bad asthma day,” described in Rowe and Chestnut (1986). 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. To the extent that an asthma attack differs from a “bad asthma day” as defined by Rowe and Chestnut (1986), the value of an asthma attack may be over or underestimated. Recent evidence from the United Kingdom (Hoskins et al., 2000) suggests that our value for avoided asthma attacks may understate true benefits by a significant amount. Hoskins et al. used a very specific definition of an asthma attack that is likely to be more severe than at least some of the asthma attacks reported by subjects in the Whittemore and Korn (1980) study. Using this definition, however, they found that asthmatics who suffered at least one asthma attack in a year had increased asthma-related costs of £273, or around $450US (1999$).

[w]Whittemore and Korn used oxidants instead of ozone and TSP instead of PM_{10}.

Table VII-18. Recent Studies on the Effects of Air Pollution on Asthma Symptoms

Study	Location/ Date	Asthmatic Study Population[A]	Symptoms[B]	Pollutants	Main Findings for Ozone and PM Exposures [C]
McConnell, et al. (1999)	Southern CA, 1993	493 asthmatic children, ages 9-15	Bronchitis, Phlegm, Cough	PM_{10}, $PM_{2.5}$, NO_2, Ozone, Acid vapor	Significant effects of PM_{10} on bronchitis (OR=1.4, 95% CI =1.1, 1.4) and phlegm (OR=2.1, 95% CI=1.4, 3.3). Significant effect of $PM_{2.5}$ on phlegm (OR=2.6, 95% CI=1.2, 5.4)
Delfino, et al. (1997)	Southern CA, 1994	22 asthmatics, ages 9-46	Symptom severity, PEFR, inhaler use	pollen, fungi, Ozone, PM_{10}	Significant effect of PM_{10} on inhaler use (0.15 inhaler puffs/10 $\mu g/m^3$, $p<0.02$)
Ostro, et al. (1995)	Los Angeles, 1992	83 African-American asthmatic children, ages 7-12	Shortness of breath	Ozone, PM_{10}, SO_2, NO_2, pollen, fungi	Significant effect of PM_{10} on shortness of breath (OR=1.6, 95% CI=1.1 ,2.4). Significant effect of ozone on shortness of breath (OR=1.4, 95% CI=1.0, 1.8)
Thurston, et al. (1997)	CT, 1991-1993	166 asthmatic children, ages 7-13	Chest symptoms, PEFR, inhaler use	Ozone, SO_4, Hydrogen ion	Significant effect[D] of ozone on chest symptoms (OR=1.4) and inhaler use (OR=1.4)
Delfino, et al. (1998)	Southern CA, 1995	25 asthmatic children, ages 9-17	Asthma symptom score	PM_{10}, ozone, fungi	Significant effect of 24-hr mean PM_{10} on asthma symptoms (OR=1.7, 95% CI=1.0, 2.7)
Delfino, et al. (1996)	San Diego, CA, 1993	12 asthmatic children, ages 9-16	Asthma symptom score, inhaler use	Ozone, $PM_{2.5}$, fungi	Significant effect of ozone on inhaler use (1.1 puffs/100 ppb, $p<0.03$) and symptom scores

[A] Study population is not the only measure of the power of a statistical analysis. For some studies, such as the Delfino, et al. (1996) analysis, the relatively small number of subjects were followed for a period of time. Thus, the number of person-days in these studies is a better indicator of statistical power than the number of study subjects.
[B] PEFR is peak expiratory flow rate, a measure of lung function.
[C] OR is the odds ratios.
[D] No 95% confidence interval was reported for the odds ratios in the Thurston, et al. (1997) study.

i. Other Health Effects: Quantification

As indicated in Table VII-1, in addition to mortality, chronic illness, and hospital admissions, there are a number of acute health effects not requiring hospitalization that are

associated with exposure to ambient levels of ozone and PM. The sources for the C-R functions used to quantify these effects are described below.

Around five percent of U.S. children between ages five and seventeen experience episodes of acute bronchitis annually (Adams, et al, 1995). Acute bronchitis is characterized by coughing, chest discomfort, and extreme tiredness. Incidences of acute bronchitis in children between the ages of five and seventeen are estimated using a C-R function developed from Dockery, et al. (1996).

Incidences of lower respiratory symptoms (i.e., wheezing, deep cough) in children aged seven to fourteen are estimated using a C-R function developed from Schwartz, et al. (1994).

Because asthmatics have greater sensitivity to stimuli (including air pollution), children with asthma can be more susceptible to a variety of upper respiratory symptoms (i.e., runny or stuffy nose; wet cough; and burning, aching, or red eyes). Research on the effects of air pollution on upper respiratory symptoms have thus focused on effects in asthmatics. Incidences of upper respiratory symptoms in asthmatic children aged nine to eleven are estimated using a C-R function developed from Pope, et al. (1991).

Health effects from air pollution can also result in missed days of work (either from personal symptoms or from caring for a sick family member). Work loss days are estimated using a C-R function developed from Ostro (1987).

The endpoint minor restricted activity days (MRAD) is estimated using a C-R function derived from Ostro and Rothschild (1989). Because MRADs are characterized by many of the same symptoms as those which define an asthma attack and the study population in Ostro and Rothschild did not exclude asthmatics, we reduce the estimated number of avoided MRAD incidences by the estimated number of avoided asthma attacks to prevent double-counting of asthma attacks. This simple subtraction may result in an underestimate of non-asthma attack related MRADs, since asthma attacks are estimated for asthmatics of all ages and MRADs are estimated only for ages 18 to 65. However, without further information on the percent of MRADs that are related to asthma attacks, we have chosen to provide a conservative estimate of MRAD benefits.

In addition to the health effects discussed above, human exposure to PM and ozone is believed to be linked to health effects such as ozone-related premature mortality (Ito and Thurston, 1996; Samet, et al. 1997), PM-related infant mortality (Woodruff, et al., 1997), cancer (US EPA, 1996b), increased emergency room visits for non-asthma respiratory causes (US EPA, 1996a; 1996b), impaired airway responsiveness (US EPA, 1996a), increased susceptibility to respiratory infection (US EPA, 1996a), acute inflammation and respiratory cell damage (US EPA, 1996a), premature aging of the lungs and chronic respiratory damage (US EPA, 1996a; 1996b). An improvement in ambient PM and ozone air quality may reduce the number of incidences within each effect category that the U.S. population would experience. Although these health effects are believed to be PM or ozone-induced, C-R data 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.

Another category of potential effects that may change in response to ozone reduction strategies results from the shielding provided by ozone against the harmful effects of ultraviolet radiation (UV-B) derived from the sun. The great majority of this shielding results from naturally occurring ozone in the stratosphere, but the 10 percent of total "column"ozone present in the troposphere also contributes (NAS, 1991). A variable portion of this tropospheric fraction of UV-B shielding is derived from ground level or "smog" ozone related to anthropogenic air pollution. Therefore, strategies that reduce ground level ozone will, in some small measure, increase exposure to UV-B from the sun.

While it is possible to provide quantitative estimates of benefits associated with globally based strategies to restore the far larger and more spatially uniform stratospheric ozone layer, the changes in UV-B exposures associated with ground level ozone reduction strategies are much more complicated and uncertain. Smog ozone strategies, such as mobile source controls, are focused on decreasing peak ground level ozone concentrations, and it is reasonable to conclude that they produce a far more complex and heterogeneous spatial and temporal pattern of ozone concentration and UV-B exposure changes than do stratospheric ozone protection programs. In addition, the changes in long-term total column ozone concentrations are far smaller from ground-level programs. To properly estimate the change in exposure and impacts, it would be necessary to match the spatial and temporal distribution of the changes in ground-level ozone to the spatial and temporal distribution of exposure to ground level ozone and sunlight. More importantly, it is long-term exposure to UV-B that is associated with effects. Intermittent, short-term, and relatively small changes in ground-level ozone and UV-B are not likely to measurably change long-term risks of these adverse effects.

For all of these reasons, we were unable to provide reliable estimates of the changes in UV-B shielding associated with ground-level ozone changes. This inability lends an upward bias to the net monetized benefits presented in this analysis. It is likely that the adverse health effects associated with increases in UV-B exposure from decreased tropospheric ozone will, however, be relatively small because 1) the expected long-term ozone change resulting from this rule is small relative to total anthropogenic tropospheric ozone, which in turn is small in comparison to total column natural stratospheric and tropospheric ozone; 2) air quality management strategies are focused on decreasing peak ozone concentrations and thus may change exposures over limited areas for limited times; 3) people often receive peak exposures to UV-B in coastal areas where sea or lake breezes reduce ground level pollution concentrations regardless of strategy; and 4) ozone concentration changes are greatest in urban areas and areas immediately downwind of urban areas. In these areas, people are more likely to spend most of their time indoors or in the shade of buildings, trees or vehicles.

j. Other Health Effects: Valuation

The valuation of a specific short-term morbidity endpoint is generally estimated by representing the illness as a cluster of acute symptoms. For each symptom, the WTP is

calculated. These values, in turn, are aggregated to arrive at the WTP to avoid a specific short term condition. For example, the endpoint lower respiratory symptoms (LRS) is represented by two or more of the following symptoms: runny or stuffy nose; coughing; and eye irritation. The WTP to avoid one day of LRS is the sum of values associated with these symptoms. The primary advantage of this approach is that it provides some flexibility in constructing estimates to represent a variety of health effects.

Valuation estimates for individual minor health effects are listed in Table VII-16 Derivation of the individual valuation estimates is provided in the benefits TSD for this RIA. Mean estimates range from $15 for an avoided day of lower respiratory symptoms to $57 for an avoided incidence of acute bronchitis. The value of work loss days varies depending on the location of an affected population. Using the median daily wage, the representative value of a work loss day is $106 (1999$). However, depending on where an affected individual lives, the value of work loss day may be higher or lower than $106.

k. Lost Worker Productivity: Quantification and Valuation

While not technically a health effect, lost worker productivity related to pollution exposure is presumably linked to reductions in the physical capabilities of workers in outdoor jobs. The value of lost worker productivity due to ozone exposure is directly estimated based on a study of California citrus workers (Crocker and Horst, 1981; US EPA, 1994). The study measured productivity impacts as the change in income associated with a change in ozone exposure, given as the elasticity of income with respect to ozone concentration (or the percentage change in income for a one percent change in ambient ozone concentration). The reported elasticity translates a ten percent reduction in ozone to a 1.4 percent increase in income.

l. Estimated Reductions in Incidences of Health Endpoints and Associated Monetary Values

Applying the C-R and valuation functions described above to the estimated changes in ozone and PM yields estimates of the number of avoided incidences (i.e. premature mortalities, cases, admissions, etc.) and the associated monetary values for those avoided incidences. These estimates are presented in Table VII-19. All of the monetary benefits are in constant 1999 dollars.

Not all known PM- and ozone-related health effects could be quantified or monetized. These unmonetized benefits are indicated by place holders, labeled B_1 and B_2. In addition, unmonetized benefits associated with CO and NMHC reductions are indicated by the placeholders B_3 and B_4. Unquantified physical effects are indicated by U_1 through U_4. The estimate of total monetized health benefits is thus equal to the subset of monetized PM- and ozone-related health benefits plus $\mathbf{B_H}$, the sum of the unmonetized health benefits.

An important factor to consider when interpreting the ozone-related benefits in Table VII-19 is the omission of ozone-related benefits in the Western U.S.[x] Over 22 percent of national NOx emission reductions occur in the Western U.S., with over 10 percent of total NOx emissions occurring in California alone. This suggests that ozone benefits in the West may be substantial, and that our estimate of Eastern ozone benefits may significantly underestimate national ozone-related benefits of the HD Engine/Diesel Fuel NOx reductions.

The largest monetized health benefit is associated with reductions in the risk of premature mortality, which accounts for over $60 billion, which is over 90 percent of total monetized health benefits. The next largest benefit is for chronic bronchitis reductions, although this value is more than an order of magnitude lower than for premature mortality. Minor restricted activity days, work loss days, and worker productivity account for the majority of the remaining benefits. The remaining categories account for less than $10 million each, however, they represent a large number of avoided incidences affecting many individuals. Alternative calculations for premature mortality incidences and valuation are presented in Tables VII-24 and VII-25, respectively. An alternative calculation is also provided in Table VII-25 for chronic bronchitis incidences and for chronic asthma incidences.

[x] We define the Western U.S. as west of 100 degrees longitude.

Table VII-19. Primary Estimate of Annual Health Benefits Associated With Air Quality Changes Resulting from the HD Engine/Diesel Fuel Rule in 2030

Endpoint	Avoided Incidence[A] (cases/year)	Monetary Benefits[B] (millions 1999$, not adjusted for growth in real income)	Monetary Benefits[B] (millions 1999$, adjusted for growth in real income)
PM-related Endpoints[C]			
Premature mortality[D] (adults, 30 and over)	8,300	$48,250	$62,580
Chronic bronchitis (adults, 26 and over)	5,500	$1,810	$2,430
Hospital Admissions – Pneumonia (adults, over 64)	1,100	$20	$20
Hospital Admissions – COPD (adults, 64 and over)	900	$10	$10
Hospital Admissions – Asthma (65 and younger)	900	$10	$10
Hospital Admissions – Cardiovascular (adults, over 64)	2,700	$50	$50
Emergency Room Visits for Asthma (65 and younger)	2,100	<$5	<$5
Asthma Attacks (asthmatics, all ages)[E]	175,900	B_a	B_a
Acute bronchitis (children, 8-12)	17,600	<$5	<$5
Lower respiratory symptoms (children, 7-14)	192,900	<$5	<$5
Upper respiratory symptoms (asthmatic children, 9-11)	193,400	$10	$10
Work loss days (adults, 18-65)	1,539,400	$160	$160
Minor restricted activity days (adults, age 18-65)	7,990,400	$390	$430
Other PM-related health effects[E]	U_1	B_1	B_1
Ozone-related Endpoints (Eastern U.S. only)[F]			
Hospital Admissions – Respiratory Causes (all ages)	1,200	$20	$20
Hospital Admissions – Cardiac Dysrhymias (all ages)	300	<$5	<$5
Emergency Room Visits for Asthma (all ages)	300	<$1	<$1
Asthma Attacks (asthmatics, all ages)[E]	185,500	B_a	B_a
Minor restricted activity days (adults, age 18-65)	1,848,100	$100	$100
Decreased worker productivity (adult working population)	—	$140	$140
Other ozone-related health effects[E]	U_2	B_2	B_2
CO and NMHC-related health effects[E]	U_3+U_4	B_{3+} B_4	B_3+B_4
Monetized Total Health-related Benefits[G]	—	$50,980+$\mathbf{B_H}$	$65,970+$\mathbf{B_H}$

[A] Incidences are rounded to the nearest 100.
[B] Dollar values are rounded to the nearest 10 million.
[C] PM-related benefits are based on the assumption that Eastern U.S. nitrate reductions are equal to one-fifth the nitrate reductions predicted by REMSAD (see Chapter II for a discussion of REMSAD and model performance).
[D] Premature mortality associated with ozone is not separately included in this analysis (also note that the estimated value for PM-related premature mortality assumes the 5 year distributed lag structure described in Section D-3.
[E] A detailed listing of unquantified PM, ozone, CO, and NMHC related health effects is provided in Table VII-1. For some endpoints such as asthma attacks, we are able to quantify the reduction in incidence, but we present the monetization as an alternative calculation.
[F] Ozone-related benefits are only calculated for the Eastern U.S. due to unavailability of reliable modeled ozone concentrations in the Western U.S. See Section C-3 for a detailed discussion of the UAM-V ozone model and model performance issues.
[G] $\mathbf{B_H}$ is equal to the sum of all unmonetized categories, i.e. B_a+B_1+B_2+B_3+B_4.

E. Assessment of Human Welfare Benefits

PM and ozone have numerous documented effects on environmental quality that affect human welfare. These welfare effects include direct damages to property, either through impacts on material structures or by soiling of surfaces, direct economic damages in the form of lost productivity of crops and trees, indirect damages through alteration of ecosystem functions, and indirect economic damages through the loss in value of recreational experiences or the existence value of important resources. EPA's Criteria Documents for PM and ozone list numerous physical and ecological effects known to be linked to ambient concentrations of these pollutants (US EPA, 1996a; 1996b). This section describes individual effects and how we quantify and monetize them. These effects include changes in commercial crop and forest yields, visibility, and nitrogen deposition to estuaries.

In section 1, we describe how we quantify and value changes in visibility, both in federal Class I areas (national parks and wilderness areas) and in the areas where people live and work. In section 2, we describe how we value the benefits of increased agricultural and commercial forest yields resulting from decreased levels of ambient ozone. In section 3, we describe the damage to materials caused by particulate matter. In section 4, we discuss the effects of nitrogen deposition on ecosystems (especially estuarine ecosystems) and describe how we quantify changes in nitrogen loadings. Finally, in section 5, we summarize the monetized estimates for welfare effects. A more detailed description of these analyses can be found in the benefits TSD for this RIA (Abt Associates, 2000).

1. Visibility Benefits

Changes in the level of ambient particulate matter caused by the final HD Engine/Diesel Fuel rule will change the level of visibility in much of the U.S. 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 Grand Canyon. This section discusses the measurement of the economic benefits of visibility.

It is difficult to quantitatively define a visibility endpoint that can be used for valuation. Increases in PM concentrations cause increases in light extinction. Light extinction is a measure of how much the components of the atmosphere absorb light. More light absorption means that the clarity of visual images and visual range is reduced, *ceteris paribus.* Light absorption is a variable that can be accurately measured. Sisler (1996) created a unitless measure of visibility based directly on the degree of measured light absorption called the *deciview*. Deciviews are standardized for a reference distance in such a way that one deciview corresponds to a change of about 10 percent in available light. Sisler characterized a change in light extinction of one deciview as "a small but perceptible scenic change under many circumstances." Air quality

models were used to predict the change in visibility, measured in deciviews, of the areas affected by the final HD Engine/Diesel Fuel rule.[y]

EPA considers benefits from two categories of visibility changes: residential visibility and recreational visibility. In both cases economic benefits are believed to consist of both use values and non-use values. Use values include the aesthetic benefits of better visibility, improved road and air safety, and enhanced recreation in activities like hunting and birdwatching. Non-use values are based on people's beliefs that the environment ought to exist free of human-induced haze. Non-use values may be a more important component of value for recreational areas, particularly national parks and monuments.

Residential visibility benefits are those that occur from visibility changes in urban, suburban, and rural areas, and also in recreational areas **not** listed as federal Class I areas.[z] For the purposes of this analysis, recreational visibility improvements are defined as those that occur specifically in federal Class I areas. A key distinction between recreational and residential benefits is that only those people living in residential areas are assumed to receive benefits from residential visibility, while all households in the U.S. are assumed to derive some benefit from improvements in Class I areas. Values are assumed to be higher if the Class I area is located close to their home.[aa]

Only two existing studies provide defensible monetary estimates of the value of visibility changes. One is a study on residential visibility conducted in 1990 (McClelland, et. al., 1993) and the other is a 1988 survey on recreational visibility value (Chestnut and Rowe, 1990a; 1990b). Both utilize the contingent valuation method. There has been a great deal of controversy and significant development of both theoretical and empirical knowledge about how to conduct CV surveys in the past decade. In EPA's judgment, the Chestnut and Rowe study contains many of the elements of a valid CV study and is sufficiently reliable to serve as the basis for monetary estimates of the benefits of visibility changes in recreational areas.[bb] This study serves as an

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[y] A change of less than 10 percent in the light extinction budget represents a measurable improvement in visibility, but may not be perceptible to the eye in many cases. Some of the average regional changes in visibility are less than one deciview (i.e. less than 10 percent of the light extinction budget), and thus less than perceptible. However, this does not mean that these changes are not real or significant. Our assumption is then that individuals can place values on changes in visibility that may not be perceptible. This is quite plausible if individuals are aware that many regulations lead to small improvements in visibility which when considered together amount to perceptible changes in visibility.

[z] The Clean Air Act designates 156 national parks and wilderness areas as Class I areas for visibility protection.

[aa] For details of the visibility estimates discussed in this chapter, please refer to the benefits technical support document for this RIA (Abt Associates 2000).

[bb] An SAB advisory letter indicates that "many members of the Council believe that the Chestnut and Rowe study is the best available." (EPA-SAB-COUNCIL-ADV-00-002, 1999) However, the committee did not formally approve use of these estimates because of concerns about the peer-reviewed status of the study. EPA believes the study has received adequate review and has been cited in numerous peer-reviewed publications

essential input to our estimates of the benefits of recreational visibility improvements in the primary benefits estimates. Consistent with SAB advice, EPA has designated the McClelland, et al. study as significantly less reliable for regulatory benefit-cost analysis, although it does provide useful estimates on the order of magnitude of residential visibility benefits (EPA-SAB-COUNCIL-ADV-00-002, 1999). Residential visibility benefits are therefore only included as an alternative calculation in Table VII-25. The methodology for this alternative calculation, explained below, is similar to the procedure for recreational benefits.

The Chestnut and Rowe study measured the demand for visibility in Class I areas managed by the National Park Service (NPS) in three broad regions of the country: California, the Southwest, and the Southeast. Respondents in five states were asked about their willingness to pay to protect national parks or NPS-managed wilderness areas within a particular region. The survey used photographs reflecting different visibility levels in the specified recreational areas. The visibility levels in these photographs were later converted to deciviews for the current analysis. The survey data collected were used to estimate a WTP equation for improved visibility. In addition to the visibility change variable, the estimating equation also included household income as an explanatory variable.

The Chestnut and Rowe study did not measure values for visibility improvement in Class I areas outside the three regions. Their study covered 86 of the 156 Class I areas in the U.S. We can infer the value of visibility changes in the other Class I areas by transferring values of visibility changes at Class I areas in the study regions. However, these values are not as defensible and are thus presented only as an alternative calculation in Table VII-25. A complete description of the benefits transfer method used to infer values for visibility changes in Class I areas outside the study regions is provided in the benefits TSD for this RIA (Abt Associates, 2000).

The estimated relationship from the Chestnut and Rowe study is only directly applicable to the populations represented by survey respondents. EPA used benefits transfer methodology to extrapolate these results to the population affected by the final HD Engine/Diesel Fuel rule. A general willingness to pay equation for improved visibility (measured in deciviews) was developed as a function of the baseline level of visibility, the magnitude of the visibility improvement, and household income. The behavioral parameters of this equation were taken from analysis of the Chestnut and Rowe data. These parameters were used to calibrate WTP for the visibility changes resulting from the final HD Engine/Diesel Fuel rule. The method for developing calibrated WTP functions is based on the approach developed by Smith, et al. (1999). Available evidence indicates that households are willing to pay more for a given visibility improvement as their income increases (Chestnut, 1997). The benefits estimates here incorporate Chestnut's estimate that a 1 percent increase in income is associated with a 0.9 percent increase in WTP for a given change in visibility.

(Chestnut and Dennis, 1997).

Using the methodology outlined above, EPA estimates that the total WTP for the visibility improvements in California, Southwestern, and Southeastern Class I areas brought about by the final HD Engine/Diesel Fuel rule is $3.3 billion. This value includes the value to households living in the same state as the Class I area as well as values for all households in the U.S. living outside the state containing the Class I area, and the value accounts for growth in real income.

For the alternative calculation for residential visibility, the McClelland, et al. study's results were used to calculate the parameter to measure the effect of deciview changes on WTP. The WTP equation was then run for the population affected by the final HD Engine/Diesel Fuel rule. The results indicate that improvements to residential visibility provide an economic benefit of $ 2.1 billion dollars for the continental U.S.[cc]

One major source of uncertainty for the visibility benefit estimate is the benefits transfer process used. Judgments used to choose the functional form and key parameters of the estimating equation for willingness to pay for the affected population could have significant effects on the size of the estimates. Assumptions about how individuals respond to changes in visibility that are either very small, or outside the range covered in the Chestnut and Rowe study, could also affect the results.

2. Agricultural and Forestry 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" (US EPA, 1996). Reduced levels of ground-level ozone resulting from the final HD Engine/Diesel Fuel rule will have generally beneficial results on agricultural crop yields and commercial forest growth.

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

a. 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

[cc] The McClelland, et al. (1993) study examined visibility changes in two Eastern cities, Chicago and Atlanta. Transferring these values to residential visibility changes in the Western U.S. may introduce greater uncertainty than transferring the values to other Eastern cities. As such, an additional alternate calculation showing the value of residential visibility just for the Eastern U.S. is included in Table VII-25.

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 U.S." (US EPA, 1996). In addition, economic studies have shown a relationship between observed ozone levels and crop yields (Garcia, et al., 1986). The economic value associated with varying levels of yield loss for ozone-sensitive commodity crops is analyzed using the AGSIM© agricultural benefits model (Taylor, et al., 1993). AGSIM© is an econometric-simulation model that is based on a large set of statistically estimated demand and supply equations for agricultural commodities produced in the United States. The model is capable of analyzing the effects of changes in policies (in this case, the implementation of the final HD Engine/Diesel Fuel rule) that affect commodity crop yields or production costs.[dd]

The measure of benefits calculated by the model is the net change in consumer and producer surplus from baseline ozone concentrations to the ozone concentrations resulting from attainment of particular standards. Using the baseline and post-control equilibria, the model calculates the change in net consumer and producer surplus on a crop-by-crop basis.[ee] Dollar values are aggregated across crops for each standard. The total dollar value represents a measure of the change in social welfare associated with the final HD Engine/Diesel Fuel rule.

The model employs biological exposure-response information derived from controlled experiments conducted by the NCLAN (NCLAN, 1996). For the purpose of our analysis, we analyze changes for the six most economically significant crops for which C-R functions are available: corn, cotton, peanuts, sorghum, soybean, and winter wheat.[ff] For some crops there are multiple C-R functions, some more sensitive to ozone and some less. Our primary estimate assumes that crops are evenly mixed between relatively sensitive and relatively insensitive varieties. The primary estimate of the net change in economic surplus resulting from changes in ozone associated with the HD Engine/Diesel Fuel rule is $1.1 billion (1999$).

b. Forestry Benefits

Ozone also has been shown conclusively to cause discernible injury to forest trees (US EPA, 1996; Fox and Mickler, 1996). In this section, we describe methods for benefits we are able to quantify and we present a qualitative description of benefits we are not able to quantify at this time. For commercial forestry impacts, the effects of changes in ozone concentrations on tree growth for a limited set of species are predicted. For future analyses, it would be helpful to use

[dd] AGSIM© is designed to forecast agricultural supply and demand out to 2010. We were not able to adapt the model to forecast out to 2030. Instead, we apply percentage increases in yields from decreased ambient ozone levels in 2030 to 2010 yield levels, and input these into an agricultural sector model held at 2010 levels of demand and supply. It is uncertain what impact this assumption will have on net changes in surplus.

[ee] Agricultural benefits differ from other health and welfare endpoints in the length of the assumed ozone season. For agriculture, the ozone season is assumed to extend from April to September. This assumption is made to ensure proper calculation of the ozone statistic used in the exposure-response functions. The only crop affected by changes in ozone during April is winter wheat.

[ff] The total value for these crops in 1998 was $47 billion.

econometric models of forest product supply and demand to estimate changes in prices, producer profits and consumer surplus. However, for this RIA we were not able to monetize the biological changes we predicted for commercial tree species. For commercial forestry, well-developed techniques are used to estimate biological and market changes. Limitations of the approach presented here include: the lack of underlying forest inventory information which is not available for the Western U.S., and the unavailability of parameterization data for all relevant species nationally. Thus, we must assume that no ozone-related changes occur to forest inventories in the Western U.S. or Canada, although as described earlier, it is likely that across the country, this rulemaking could result in decreases in ozone and improvements in forest health compared to baseline conditions. Therefore, using these assumptions will underestimate the commercial forestry benefits associated with the program.

Similar to the agriculture analysis, assessing the forestry benefits couples air quality modeling results, C-R functions derived from a biological model, forest inventory estimates, and an economic model. Again, we are only able to quantify the physical effect, and further details are contained in the technical support document (Hubbell et al., 2000).

Our analysis used species-specific C-R functions derived from the TREGRO model (Laurence, et al., 2000) . We developed ozone C-R functions for 6 species for which there were parameterization data by climatic region of the Eastern U.S.: black cherry, loblolly pine, red oak, red spruce, sugar maple, and tulip poplar. TREGRO is a model of tree physiological response to environmental stresses (Weinstein and Yanai, 1994). It was developed to simulate the response of sapling and mature trees to ozone and acidic precipitation stress in conjunction with other stressors. The model has been used to evaluate long-term effects of pollutants on resource availability.

The next step would be to use economic model such as the Timber Assessment Market Model (TAMM)/Aggregated Timberland Assessment System (ATLAS). In brief, the approach would be to use the biological inputs to modify the accumulation of inventory within ATLAS, which then shifts the timber supply functions in TAMM. The economic value of yield changes for commercial forests would be estimated using TAMM. This model is a US Forest Service (Adams and Haynes, 1996) spatial model of the solid wood and timber inventory elements of the U.S. forest products sector. The model provides projections of timber markets by geographic region and wood type through the year 2050. Nine regions covering the continental U.S. are included in the analysis; however, the effects of reduced O_3 concentrations were only considered for the Eastern U.S. The TAMM model perturbs timber market (spatial) equilibrium and yields timber price, quantity and welfare effects. However, it is limited to sawtimber and does not capture all relevant forest product markets (e.g., pulp wood). TAMM, in turn, would predict the effect of these reductions on timber markets by changing the annual growth rates of commercial forest growing-stock inventories. The model uses applied welfare economics to value changes in ambient O_3 concentrations. However, we were not able to complete this step for the RIA.

The six species we analyzed account for as much as 73 percent and as little as zero percent of total growing stock volume depending on the region and forest type. The annual change in growth adjustment factors ranged from zero to 0.009841. While the adjustment factor

may seem small on an absolute basis, when compounded over the lifetime of a tree, the effects may be significant. The full set of adjustment factors are presented in the technical support document (Hubbell et al., 2000).

c. Other Effects

An additional welfare benefit expected to accrue as a result of reductions in ambient ozone concentrations in the U.S. 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 (US EPA, 1996c, p. 5-521). However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to impact 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. It is estimated that more than $20 billion (1990 dollars) are spent annually on landscaping using ornamentals (Abt Associates, 1995), both by private property owners/tenants and by governmental units responsible for public areas. 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.

The final HD Engine/Diesel Fuel rule, by reducing NO_X emissions, will also reduce nitrogen deposition on agricultural land and forests. There is some evidence that nitrogen deposition may have positive effects on agricultural output through passive fertilization. Holding all other factors constant, farmers' use of purchased fertilizers or manure may increase as deposited nitrogen is reduced. Estimates of the potential value of this possible increase in the use of purchased fertilizers are not available, but it is likely that the overall value is very small relative to other health and welfare effects. The share of nitrogen requirements provided by this deposition is small, and the marginal cost of providing this nitrogen from alternative sources is quite low. In some areas, agricultural lands suffer from nitrogen over-saturation due to an abundance of on-farm nitrogen production, primarily from animal manure. In these areas, reductions in atmospheric deposition of nitrogen from PM represent additional agricultural benefits.

Information on the effects of changes in passive nitrogen deposition on forests and other terrestrial ecosystems is very limited. The multiplicity of factors affecting forests, including other potential stressors such as ozone, and limiting factors such as moisture and other nutrients, confound assessments of marginal changes in any one stressor or nutrient in forest ecosystems. However, reductions in deposition of nitrogen could have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor (US EPA, 1993).

On the other hand, there is evidence that forest ecosystems in some areas of the United States are nitrogen saturated (US EPA, 1993). Once saturation is reached, adverse effects of additional nitrogen begin to occur such as soil acidification which can lead to leaching of nutrients needed for plant growth and mobilization of harmful elements such as aluminum. Increased soil acidification is also linked to higher amounts of acidic runoff to streams and lakes and leaching of harmful elements into aquatic ecosystems.

3. Benefits from Reductions in Materials Damage

The final HD Engine/Diesel Fuel rule is 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. Criteria pollutants also have 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 benefit 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 1970's) are too out of date to provide a reliable enough estimate of current household soiling damages (EPA-SAB-Council-ADV-003, 1998). An estimate is included in the alternative calculations presented in Table VII-25.

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) indicate that these benefits could be an order of magnitude larger than household soiling benefits.

4. Benefits from Reduced Ecosystem Damage

The effects of air pollution on the health and stability of ecosystems are potentially very important, but are at present poorly understood and difficult to measure. The reductions in NO_X caused by the final rule could produce significant benefits. Excess nutrient loads, especially of nitrogen, cause a variety of adverse consequences to the health of estuarine and coastal waters. These effects include toxic and/or noxious algal blooms such as brown and red tides, low (hypoxic) or zero (anoxic) concentrations of dissolved oxygen in bottom waters, the loss of submerged aquatic vegetation due to the light-filtering effect of thick algal mats, and fundamental shifts in phytoplankton community structure (Bricker et al., 1999).

Reductions in nitrogen loadings are estimated for twelve eastern estuaries (including two on the Gulf Coast). These estimated reductions are described earlier in this Chapter. Four of these estuaries have established consensus goals for reductions in annual nitrogen loads, indicating an intention of reaching these goals through implementation of controls on nitrogen sources. These four estuaries and their reduction goals are listed in Table VII-20.

Table VII-20. Reduction Goals and 1998 Nitrogen Loads to Selected Eastern Estuaries (tons per year)

Estuary	Total Nitrogen Loadings	Nitrogen Loadings from Atmospheric Deposition	Overall Reduction Goal
Albemarle/Pamlico Sound	25,300	11,000	7,600
Chesapeake Bay	185,000	49,500	35,600
Long Island Sound	53,700	13,200	31,460
Tampa Bay	3,900	2,100	100

Source: US EPA, 1998.

Estimated reductions in deposition of atmospheric nitrogen to these four estuaries are listed in Table VII-21, along with the percentage of the reduction goal accounted for by these reductions. These figures suggest that the reductions in nitrogen deposition resulting from the final HD Engine/Diesel Fuel rule will provide significant progress towards meeting nitrogen reduction goals in several of these estuaries.

Table VII-21. Estimated Annual Reductions in Nitrogen Loadings in Selected Eastern Estuaries for the Final HD Engine/Diesel Fuel Rule in 2030 (tons per year)

Estuary	Change in Nitrogen Loadings	% of Estuary Nitrogen Reduction Goal
Albemarle/Pamlico Sound	1,804	23.7%
Chesapeake Bay	2,706	7.6%
Long Island Sound	1,067	3.4%
Tampa Bay[A]	385	over 100%

[A] Tampa Bay had a very low nitrogen loadings reduction goal. As such, the HD Engine/Diesel Fuel rule provides more reductions than are necessary to achieve the stated goal.

Direct C-R functions relating changes in nitrogen loadings to changes in estuarine benefits are not available. The preferred WTP based measure of benefits depends on the availability of these C-R functions and on estimates of the value of environmental responses. Because neither appropriate C-R functions nor sufficient information to estimate the marginal value of changes in water quality exist at present, calculation of a WTP measure is not possible. An alternative is to use an avoided cost approach to estimate the welfare effects of PM on estuarine ecosystems. The use of the avoided cost approach to establish the value of a reduction in nitrogen deposition is problematic if there is not a direct link between reductions in air

deposited nitrogen and the abandonment of a costly regulatory program. However, there are currently no readily available alternatives to this approach.

Based on SAB advice, we use the avoided cost approach only to derive an alternative calculation of the value of reductions in atmospheric nitrogen loadings to estuaries (EPA-SAB-COUNCIL-ADV-00-002, 1999). The SAB believes that the avoided cost approach for nitrogen loadings is valid only if the state and local governments have established firm pollution reduction targets, and that displaced costs measured in the study represent measures not taken because of the Clean Air Act (EPA-SAB-COUNCIL-ADV-00-002, 1999). Because the nitrate reduction targets in the studied estuaries are not firm targets, and there is not assurance that planned measures would be undertaken in the absence of the Clean Air Act, we are currently unable to provide a meaningful primary estimate. Thus, the avoided cost estimate is presented as an alternative calculation in Table VII-25.

If better models of ecological effects can be defined, EPA believes that progress can be made in estimating WTP measures for ecosystem functions. These estimates would be superior to avoided cost estimates in placing economic values on the welfare changes associated with air pollution damage to ecosystem health. For example, if nitrogen or sulfate loadings can be linked to measurable and definable changes in fish populations or definable indexes of biodiversity, then CV studies can be designed to elicit individuals' WTP for changes in these effects. This is an important area for further research and analysis, and will require close collaboration among air quality modelers, natural scientists, and economists.

5. Estimated Values for Welfare Endpoints

Applying the valuation methods described above to the estimated changes in ozone and PM in 2030 yields estimates of the value of changes in visibility and agricultural and forestry yields. These estimates are presented in Table VII-22. All of the monetary benefits are in constant 1999 dollars.

We are unable to provide primary monetized estimates of residential visibility, household soiling, materials damage, and nitrogen deposition, in addition to the other welfare effects listed in Table VII-1. These unmonetized benefits are indicated by placeholders, labeled B_5 to B_{12}. The estimate of total monetized welfare benefits is thus equal to the subset of monetized welfare benefits plus $\mathbf{B_W}$, the sum of the unmonetized welfare benefits.

Table VII-22. Primary Estimate of Annual Monetary Values for Welfare Effects Associated With Improved Air Quality Resulting from the HD Engine/Diesel Fuel Rule in 2030

Endpoint	Monetary Benefits (millions 1999$, Unadjusted for growth in real income)[A]	Monetary Benefits (millions 1999$, Adjusted for Growth in Real Income)[A]
PM-related Endpoints		
Recreational Visibility (86 Class I areas in California, the Southeast and the Southwest)	$1,790	$3,260
Residential Visibility	B_5	B_5
Household Soiling	B_6	B_6
Materials Damage	B_7	B_7
Nitrogen Deposition to Estuaries	B_8	B_8
Other PM-related welfare effects[B]	B_9	B_9
Ozone-related Endpoints		
Commercial Agricultural Benefits (6 major crops) (Eastern U.S. only)[C]	$1,120	$1,120
Commercial Forestry Benefits (Eastern U.S. only)[C]	B_9	B_9
Other ozone-related welfare effects[B]	B_{10}	B_{10}
CO-related welfare effects[B]	B_{11}	B_{11}
NMHC-related welfare effects[B]	B_{12}	B_{12}
Total Monetized Welfare-related Benefits[D]	$2,910	$4,380

[A] Rounded to the nearest 10 million and visibility benefits are adjusted to account for growth in real GDP per capita between 1990 and 2030. See Section C. [B] A detailed listing of unquantified PM, ozone, CO, and NMHC related welfare effects is provided in Table VII-1.
[C] Ozone-related benefits are only calculated for the Eastern U.S. due to unavailability of reliable modeled ozone concentrations in the Western U.S. This results in an underestimate of national ozone-related benefits. See Section D-3 for a detailed discussion of the UAM-V ozone model and model performance issues. [D] $\mathbf{B_W}$ is equal to the sum of all unmonetized welfare categories, i.e. $B_5+B_6+...+B_{13}$.

Total monetized welfare-related benefits are around $4.4 billion. Monetized welfare benefits are roughly 1/20th the magnitude of monetized health benefits. However, due to the difficulty in quantifying and monetizing welfare benefits, a higher proportion of welfare benefits are not monetized. It is thus inappropriate to conclude that welfare benefits are unimportant just by comparing the estimates of the monetized benefits. Also, as with health benefits, ozone-related welfare benefits may be significantly underestimated due to the omission of ozone-related benefits in the Western U.S.

Alternative calculations for recreational visibility, residential visibility, household soiling, and nitrogen deposition are presented in Table VII-25 later in this chapter.

F. Total Benefits

We provide our primary estimate of benefits for each health and welfare endpoint as well as the resulting primary estimate of total benefits. To obtain this estimate, we aggregate dollar benefits associated with each of the effects examined, such as hospital admissions, into a total benefits estimate assuming that none of the included health and welfare effects overlap. The primary estimate of the total benefits associated with the health and welfare effects is the sum of the separate effects estimates. Total monetized benefits associated with the final HD Engine/Diesel Fuel rule are listed in Table VII-23, along with a breakdown of benefits by endpoint. Note that the value of endpoints known to be affected by ozone and/or PM that we are not able to monetize are assigned a placeholder value (e.g., B_1, B_2, etc.). Unquantified physical effects are indicated by a U. The estimate of total benefits is thus the sum of the monetized benefits and a constant, B, equal to the sum of the unmonetized benefits, $B_1+B_2+...+B_n$.

A comparison of the incidence column to the monetary benefits column 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 40 times more asthma attacks than premature mortalities, yet these asthma attacks 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 asthma attacks, are valued using a proxy measure of WTP. As such the true value of these effects may be higher than that reported in Table VII-23.

Our primary estimate of total monetized benefits for the final HD Engine/Diesel Fuel rule is $70.4 billion, of which $62.6 billion is the benefits of reduced premature mortality risk from PM exposure. Total monetized benefits are dominated by the benefits of reduced mortality risk. Mortality related benefits account for 89 percent of total monetized benefits followed by recreational visibility (4.6 percent) and chronic bronchitis (3.5 percent). Health benefits account for 94 percent of total benefits.

Table VII-23. Primary Estimate of Annual Monetized Benefits Associated With Improved Air Quality Resulting from the HD Engine/Diesel Fuel Rule in 2030[A,B]

Endpoint	Pollutant	Avoided Incidence[C] (cases/year)	Monetary Benefits[D] (millions 1999$, Adjusted for Income Growth)
Premature mortality[E] (adults, 30 and over)	PM	8,300	$62,580
Chronic bronchitis (adults, 26 and over)	PM	5,500	$2,430
Hospital Admissions from Respiratory Causes	O_3 and PM	4,100	$60
Hospital Admissions from Cardiovascular Causes	O_3 and PM	3,000	$50
Emergency Room Visits for Asthma	O_3 and PM	2,400	<$5
Acute bronchitis (children, 8-12)	PM	17,600	<$5
Lower respiratory symptoms (children, 7-14)	PM	192,900	<$5
Upper resp. symptoms (asthmatic children, 9-11)	PM	193,400	$10
Asthma attacks (asthmatics, all ages)[F]	O_3 and PM	361,400	B_a
Work loss days (adults, 18-65)	PM	1,539,400	$160
Minor restricted activity days (adults, age 18-65)	O_3 and PM	9,838,500	$530
Other health effects[F,G]	O_3, PM, CO, HAPs	$U_1+U_2+U_3+U_4$	$B_1+B_2+B_3+B_4$
Decreased worker productivity	O_3	—	$140
Recreational visibility (86 Class I Areas)	PM	—	$3,260
Residential visibility	PM	—	B_5
Household soiling damage	PM	—	B_6
Materials damage	PM	—	B_7
Nitrogen Deposition to Estuaries	Nitrogen	—	B_8
Agricultural crop damage (6 crops)	O_3	—	$1,120
Commercial forest damage (6 species)	O_3	—	B_9
Other welfare effects[F,G]	O_3, PM, CO, HAPs	—	$B_{10}+B_{11}+B_{12}+B_{13}$
Monetized Total[H]			$70,360+**B**

[A] Monetary benefits are adjusted to account for growth in real GDP per capita between 1990 and 2030. See Section C. [B] Ozone-related benefits are only calculated for the Eastern U.S. due to unavailability of reliable modeled ozone concentrations in the Western U.S. This results in an underestimate of national ozone-related benefits. See Section D-3 for a detailed discussion of the UAM-V ozone model and model performance issues. [C] Incidences are rounded to the nearest 100. [D] Dollar values are rounded to the nearest 10 million. [E] Premature mortality associated with ozone is not separately included in this analysis. It is assumed that the Section D-3 ACS/Krewski et al., 2000 C-R function for premature mortality captures both PM mortality benefits and any mortality benefits associated with other air pollutants. Also note that the valuation assumes the 5 year distributed lag structure described earlier. [F] The U_i are the incidences for the unquantified category i, the B_i are the monetary values for the unquantified endpoint i. For some categories such as asthma attacks, we were able to quantify the reduction in incidence, but we present the monetization as an alternative calculation. [G] A detailed listing of unquantified PM, ozone, CO, and NMHC related health and welfare effects is provided in Table VII-1. [H] **B** is equal to the sum of all unmonetized categories, i.e. $B_a+B_1+B_2+...+B_{13}$.

As discussed in Section C.1, we have adjusted our primary estimate of benefits to reflect the projected growth in real income between base income in 1990 and the 2030 analytical year. We account for real income growth by applying the primary adjustment factors from Table VII-12 to the appropriate health and welfare endpoints in Tables VII-19 and VII-22.

In addition to the primary estimate in Table VII-23, in Tables VII-24 and VII-25 we present alternative calculations representing how the value for individual endpoints or total benefits would change if we were to make a different assumption about an element of the benefits analysis. Specifically, in Table VII-24, we present the impact of different C-R functions for PM-related premature mortality. In Table VII-25, we show the impact of alternative assumptions about other parameters. For example, Table VII-25 can be used to answer questions like "What would total benefits be if we were to value avoided incidences of premature mortality using the VSLY approach rather than the VSL approach?" This table provides alternative calculations both for valuation issues (e.g. the correct value for a statistical life saved) and for physical effects issues (e.g., possible recovery from chronic illnesses). This table is not meant to be comprehensive. Rather, it reflects some of the key issues identified by EPA or commentors as likely to have a significant impact on total benefits. As discussed earlier, individual adjustments in the table should not be added together without addressing potential issues of overlap and low joint probability among the endpoints. Accompanying Table VII-25 is a brief discussion of each of the alternative calculations.

While Tables VII-24 and VII-25 provide alternative calculations for specific alternative assumptions, there are some parameters to which total benefits may be sensitive but for which no or limited credible scientific information exists to determine plausible values. Sensitivity analyses for these parameters are presented in Appendix VII-A. Issues examined in this appendix include alternative specifications for the lag structure of PM related premature mortality and impacts of assumed thresholds on the estimated incidence of avoided premature mortality. This appendix also contains several illustrative endpoint calculations for which the scientific uncertainty is too great to provide a reasonable estimate and if included, might lead to double-counting of benefits. These include premature mortality associated with daily fluctuations in PM, infant mortality associated with PM, and premature mortality associated with daily fluctuations in ozone.

Table VII-24. Alternative Estimates of Premature Mortality Benefits for the HD Engine/Diesel Fuel Rule in 2030

	Model	Avoided Incidences	Value Adjusted for Growth in Real Income (million 1999$)	Impact on Primary Benefits Estimate Adjusted for Growth in Real Income (million 1999$)
1	Fine Particles Alone, Random Effects, Regional Adjustment Source: Table 46, Krewski, et al. (2000) "ACS Study "	9,400	$69,940	+$7,370 (+10.5%)
2	Fine Particles Alone, Random Effects, Independent Cities Source: Table 46, Krewski, et al. (2000) "ACS Study"	16,000	$93,940	+$59,270 (+84.2%)
3	All Combined, All Cause, Fine Particles Source: Table 3, Pope, et al. (1995) "ACS Study"	9,900	$75,360	+12,780 (+18.2%)
4	All Causes, Extended, Age Time Axis: Table 3, Krewski, et al. (2000) "Harvard Six-city Study"	24,200	$181,080	+$118,500 (+168.4%)
5	All Subjects Source: Table 3, Dockery, et al. (1993) "Harvard Six-city Study"	23,100	$173,450	+$110,874 (+157.6%)

[A] Reported relative risks for the Pope, et al. (1995) and Dockery, et al. (1993) studies are comparisons of mortality rates between most polluted and least polluted cities. For the Pope et al. study the relative risk is based on a difference in median PM2.5 levels of 24.5 $\mu g/m^3$. For the Dockery et al. study, the relative risk is based on a difference of 18.6 $\mu g/m^3$. The Krewski et al. reanalysis of the Pope et al. study reports all relative risks based on a 24.5 $\mu g/m^3$ difference for comparability with the Pope, et al. (1995) results, rather than comparing the means or medians of the most polluted and least polluted studies. Likewise, the Krewski et al. reanalysis of the Dockery et al. Harvard Six Cities study reports all relative risks based on a 18.6 $\mu g/m^3$ difference for comparability with the Dockery, et al. (1993) study.
[B] Assumes national all-cause mortality rate of 0.0147 per person for adults aged 30 and older and 0.0131 per person for adults aged 25 and older. (U.S. Centers for Disease Control. 2000 *National Vital Statistics Reports* 48(11): Table 8).

The first alternative C-R function (row 1 of Table VII-24) is based on the relative risk of 1.16 from the "Fine Particles Alone, Regional Adjustment Random Effects" model reported in Table 46 of the HEI report. This C-R function is a reasonable specification to explore the impact of adjustments for broad regional correlations. However, the HEI report noted that the spatial adjustment methods "may have over adjusted the estimated effect for regional pollutants such as fine particles and sulfate compared with the effect estimates for more local pollutants such as sulfur dioxide." Thus, the estimates of avoided incidences of premature mortality based on this C-R function may underestimate the true effect. Note that this C-R function is based on the original air quality dataset used in the ACS study, covering 50 cities, and used the median $PM_{2.5}$ levels rather than mean $PM_{2.5}$ as the indicator of exposure.

Krewski, et al. (2000) also estimated a random effects model which accounts for between city variation but "ignores possible regional patterns in mortality." The estimated avoided incidences of premature mortality is based on the original 50 city air quality dataset used in the ACS study and used the median $PM_{2.5}$ levels rather than mean $PM_{2.5}$ as the indicator of exposure (row 2 of Table VII-24).

For comparison with earlier benefits analyses, we also include estimates of avoided incidences of premature mortality based on the original ACS/Pope et al. (1995) analysis (row 3 of Table VII-24) and the original "Harvard Six Cities" estimate as reported in Dockery, et al. (1993) analysis (row 5 of Table VII-24).

The Krewski, et al. "Harvard Six Cities" estimate of the relationship between PM exposure and premature mortality (row 4 of Table VII-24) is a plausible alternative to the Krewski, et al. "ACS 50 City" primary estimate. 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). However, the Krewski-Harvard Six Cities study had a more limited geographic scope (and a smaller study population) than the Krewski-ACS study. The demographics of the ACS study population, i.e., largely white and middle-class, may also produce a downward bias in the estimated PM mortality coefficient, because short-term studies indicate that the effects of PM tend to be significantly greater among groups of lower socioeconomic status. The Krewski-Harvard Six Cities study also covered a broader age category (25 and older compared to 30 and older in the ACS study) and followed the cohort for a longer period (15 years compared to 8 years in the ACS study). For these reasons, the Krewski-Harvard Six Cities study is considered to be a plausible alternative estimate of the avoided premature mortality incidences associated with the final HD Engine/Diesel Fuel rule.

Table VII-25. Additional Alternative Benefits Calculations for the HD Engine/Diesel Fuel Rule in 2030

Alternative Calculation		Description of Estimate	Impact on Primary Benefit Estimate Adjusted for Growth in Real Income (million 1999$)
1	Value of avoided premature mortality incidences based on age-specific VSL	Calculate the age distribution of avoided incidences of premature mortality and apply age-adjusted VSL to the incidences. Sources of age-adjustment ratios are Jones-Lee (1989) and Jones-Lee et al. (1993)	Jones-Lee (1989) -$28,510 (-40.5%) Jones-Lee (1993) -$6,820 (-10.0%)
2	Chronic Asthma	Avoided incidences of chronic asthma are estimated using the McDonnell, et al. (1999) C-R function. The number of avoided incidences of chronic asthma is 820.	+$40 (+<1%)
3	Reversals in chronic bronchitis treated as lowest severity cases	Instead of omitting cases of chronic bronchitis that reverse after a period of time, they are treated as being cases with the lowest severity rating. The number of avoided chronic bronchitis incidences increases from 5,480 to 10,250 (87%).	+$940 (+1.3%)
4	COPD and pneumonia hospital admissions.	Hospital admissions for Pneumonia and COPD estimated using the Moolgavkar (1997) C-R function instead of the Samet et al. (2000) pooled C-R function. The number of hospital admissions for these two causes decreases from 2,010 to 600 (-70%)	-$20 (-<1%)
5	Value of visibility changes in all Class I areas	Values of visibility changes at Class I areas in California, the Southwest, and the Southeast are transferred to visibility changes in Class I areas in other regions of the country.	+$1,240 (+1.8%)
6	Value of visibility changes in Eastern U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Eastern U.S. based on the reported values for Chicago and Atlanta from McClelland et al. (1990).	+$1,250 (+1.8%)
7	Value of visibility changes in Western U.S. residential areas	Value of visibility changes outside of Class I areas are estimated for the Western U.S. based on the reported values for Chicago and Atlanta from McClelland et al. (1990).	+$910 (+1.3%)
8	Household soiling damage	Value of decreases in expenditures on cleaning are estimated using values derived from Manuel, et al. (1983).	+$260 (+0.4%)
9	Avoided costs of reducing nitrogen loadings in east coast estuaries	Estuarine benefits in 12 East coast estuaries from reduced atmospheric nitrogen deposition are approximated using the avoided costs of removing or preventing loadings from terrestrial sources.	+$150 (+0.3%)
10	Asthma attacks	Avoided incidences of asthma attacks monetized using Rowe and Chestnut (1986).	+$20 (+<1%)

The age-specific VSL alternative calculation (row 1 of Table VII-25) recognizes that individuals who die from air pollution related causes tend to be older than the average age of individuals in the VSL studies used to develop the $6 million value. A complete discussion of this issue can be found in section 3.b of this chapter. For this calculation, the method we use to account for age differences is to adjust the VSL based on ratios of VSL's for specific ages to the VSL for a 40 year old individual (row 2 of Table VII-25). There are several potential sources for these ratios. Estimates from two Jones-Lee studies provide a reasonable low and high end for this type of adjustment. The ratios based on Jones-Lee (1989), as summarized in U.S. EPA (2000), suggest a steep inverted U shape between age and VSL, with the VSL for a 70 year old at 63 percent of that for a 40 year old, and the VSL for an 85 year old at 7 percent of that for 40 year old. The ratios based on Jones-Lee (1993) and summarized in U.S. EPA (2000b), suggest a much flatter inverted U shape, with the VSL for a 70 year old at 92 percent of that for a 40 year old, and the VSL for an 85 year old at 82 percent of that for a 40 year old. The general U shaped relationship is supported by recent analyses conducted in Canada and the U.S. by Krupnick et al. (2000a, 2000b). Their results suggest a curvature somewhere between the two Jones-Lee estimates. The wide range of age-adjustment ratios, especially at older ages demonstrates the difficulty in making these kinds of adjustments. To calculate the age-adjusted VSL, we first calculate the number of avoided premature mortalities in each age category, and then apply the age adjusted VSL to the appropriate incidences in each age category[gg].

The alternative calculation for the development of chronic asthma (row 2 of Table VII-25) is estimated using a recent study by McDonnell, et al. (1999) which found a statistical association between ozone and the development of asthma in adult white, non-Hispanic males. Other studies have not identified an association between air quality and the onset of asthma. Chronic asthma is characterized by repeated incidences of inflammation of the lungs. This causes restriction in the airways and results in shortness of breath, wheezing, and coughing. Asthma is also characterized by airway hyper responsiveness to stimuli. Chronic asthma affects over seven percent of the U.S. population (US Centers for Disease Control and Prevention, 1999b).

The McDonnell, et al. study is a prospective cohort analysis, measuring the association between long-term exposure to ambient concentrations of ozone and development of chronic asthma in adults. The study found a statistically significant effect for adult males, but none for adult females. EPA also believes it to be appropriate to apply the C-R function to all adult males over age 27 because no evidence exists to suggest that non-white adult males have a lower responsiveness to air-pollution. For other health effects such as shortness of breath, where the study population was limited to a specific group potentially more sensitive to air pollution than the general population (Ostro et al., 1995), EPA has applied the C-R function only to the limited population.

[gg] The age categories and lower and upper end estimated age-adjustment ratios are: 30-39 (0.89, 0.98), 40-59 (1.0, 1.0), 60-69 (0.86, 0.97), 70-79 (0.63, 0.92), 80-84 (0.28, 0.85), 85+ (0.07, 0.82).

Some commentors have raised questions about the statistical validity of the associations found in this study and the appropriateness of transferring the estimated C-R function from the study populations (white, non-Hispanic males) to other male populations (i.e. African-American males). Some of these concerns include the following: 1) no significant association was observed for female study participants also exposed to ozone; 2) the estimated C-R function is based on a cross-sectional comparison of ozone levels, rather than incorporating information on ozone levels over time; 3) information on the accuracy of self-reported incidence of chronic asthma was collected but not used in estimating the C-R function; 4) the study may not be representative of the general population because it included only those individuals living 10 years or longer within 5 miles of their residence at the time of the study; and 5) the study had a significant number of study participants drop out, either through death, loss of contact, or failure to provide complete or consistent information. EPA believes that while these issues may result in increased uncertainty about this effect, none can be identified with a specific directional bias in the estimates. In addition, the SAB reviewed the study and deemed it appropriate for quantification of changes in ozone concentrations in benefits analyses (EPA-SAB-COUNCIL-ADV-00-001, 1999). EPA recognizes the need for further investigation by the scientific community to confirm the statistical association identified in the McDonnell, et al. study.

Following SAB advice (EPA-SAB-COUNCIL-ADV-00-001, 1999) and consistent with the Section 812 Prospective Report, we quantify this endpoint for the RIA. However, it should be noted that it is not clear that the intermittent, short-term, and relatively small changes in annual average ozone concentrations resulting from this rule alone are likely to measurably change long-term risks of asthma.

Similar to the valuation of chronic bronchitis, WTP to avoid chronic asthma is presented as the net present value of what would potentially be a stream of costs and lower well-being incurred over a lifetime. Estimates of WTP to avoid asthma are provided in two studies, one by Blumenschein and Johannesson (1998) and one by O'Conor and Blomquist (1997). Both studies use the contingent valuation method to solicit annual WTP estimates from individuals who have been diagnosed as asthmatics. The central estimate of lifetime WTP to avoid a case of chronic asthma among adult males, approximately $25,000, is the average of the present discounted value from the two studies. Details of the derivation of this central estimate from the two studies is provided in the benefits TSD for this RIA (Abt Associates, 2000).

Another important issue related to chronic conditions is the possible reversal in chronic bronchitis incidences (row 3 of Table VII-25). Reversals are defined as those cases where an individual reported having chronic bronchitis at the beginning of the study period but reported not having chronic bronchitis in follow-up interviews at a later point in the study period. Since, by definition, chronic diseases are long-lasting or permanent, if the disease goes away it is not chronic. However, we have not captured the benefits of reducing incidences of bronchitis that are somewhere in-between acute and chronic. One way to address this is to treat reversals as cases of chronic bronchitis that are at the lowest severity level. These cases thus get the lowest value for chronic bronchitis.

For this benefits analysis, we have adopted the C-R function for COPD and pneumonia hospital admissions from Samet, et al. (2000). This estimate, while representing the state of the art in epidemiological studies, is a good deal larger than the estimate from Moolgavkar (1997). We explore the impact of using the Moolgavkar (1997) estimate instead of the Samet, et al. (2000) estimate in row 4 of Table VII-25.

The alternative calculation for recreational visibility (row 5 of Table VII-25) is an estimate of the full value of visibility in the entire region affected by the final HD Engine/Diesel Fuel rule. The Chestnut and Rowe study from which the primary valuation estimates are derived only examined WTP for visibility changes in the southeastern portion of the affected region. In order to obtain estimates of WTP for visibility changes in the northeastern and central portion of the affected region, we have to transfer the southeastern WTP values. This introduces additional uncertainty into the estimates. However, we have taken steps to adjust the WTP values to account for the possibility that a visibility improvement in parks in one region, is not necessarily the same environmental quality good as the same visibility improvement at parks in a different region. This may be due to differences in the scenic vistas at different parks, uniqueness of the parks, or other factors, such as public familiarity with the park resource. To take this potential difference into account, we adjusted the WTP being transferred by the ratio of visitor days in the two regions.

The alternative calculations for residential visibility (rows 6 and 7 of Table VII-25) are based on the McClelland, et al. study of WTP for visibility changes in Chicago and Atlanta. As discussed in Section F-1, SAB advised EPA that the residential visibility estimates from the available literature are inadequate for use in a primary estimate in a benefit-cost analysis. However, EPA recognizes that residential visibility is likely to have some value and the McClelland, et al. estimates are the most useful in providing an estimate of the likely magnitude of the benefits of residential visibility improvements.

The alternative calculation for household soiling (row 8 of Table VII-25) is based on the Manuel, et al. study of consumer expenditures on cleaning and household maintenance. This study has been cited as being “the only study that measures welfare benefits in a manner consistent with economic principals (Desvouges et al., 1998). However, the data used to estimate household soiling damages in the Manuel, et al. study are from a 1972 consumer expenditure survey and as such may not accurately represent consumer preferences in 2030. EPA recognizes this limitation, but believes the Manuel, et al. estimates are still useful in providing an estimate of the likely magnitude of the benefits of reduced PM household soiling.

The alternative calculation for the avoided costs of reductions in nitrogen loadings (row 9 of Table VII-25) is constructed by examining the avoided costs to surrounding communities of reduced nitrogen loadings for three case study estuaries (US EPA, 1998). The three case study estuaries are chosen because they have agreed upon nitrogen reduction goals and the necessary nitrogen control cost data. The values of atmospheric nitrogen reductions are determined on the basis of avoided costs associated with agreed upon controls of nonpoint water pollution sources. Benefits are estimated using a weighted-average, locally-based cost for nitrogen removal from water pollution (US EPA, 1998). Valuation reflects water pollution control cost avoidance based

on the weighted average cost per pound of current non-point source water pollution controls for nitrogen in the three case study estuaries. Taking the weighted cost per pound of these available controls assumes States will combine low cost and high cost controls, which could inflate avoided cost estimates. The avoided cost measure is likely to be an underestimate of the value of reduced nitrogen loadings in eastern estuaries because: 1) the 12 estuaries represent only about 50 percent of the total watershed area in the Eastern U.S.; and 2) costs avoided are not good proxies for WTP, understating true WTP under certain conditions.

We monetize the reduction of 361,400 asthma attacks (row 10 of Table VII-25) using Rowe and Chestnut (1986).

G. Comparison of Costs to Benefits

Benefit-cost analysis provides a valuable framework for organizing and evaluating information on the effects of environmental programs. When used properly, benefit-cost analysis helps illuminate important potential effects of alternative policies and helps set priorities for closing information gaps and reducing uncertainty. According to economic theory, the efficient policy alternative maximizes net benefits to society (i.e., social benefits minus social costs). However, not all relevant costs and benefits can be captured in any analysis. Executive Order 12866 clearly indicates that unquantifiable or nonmonetizable categories of both costs and benefits should not be ignored. There are many important unquantified and unmonetized costs and benefits associated with reductions in emissions, including many health and welfare effects. Potential benefit categories that have not been quantified and monetized are listed in Table VII-1 of this chapter.

In addition to categories that cannot be included in the calculated net benefits, there are also practical limitations for the comparison of benefits to costs in this analysis, as discussed throughout this chapter. Several specific limitations deserve to be mentioned again here:

- The state of atmospheric modeling is not sufficiently advanced to provide a workable "one atmosphere" model capable of characterizing ground-level pollutant exposure for all pollutants of interest (e.g., ozone, particulate matter, carbon monoxide, nitrogen deposition, etc). Therefore, the EPA must employ several different pollutant models to characterize the effects of alternative policies on relevant pollutants. Also, not all atmospheric models have been widely validated against actual ambient data. In particular, since the monitoring network for $PM_{2.5}$ has produced only one year of data, atmospheric models designed to capture the effects of alternative policies on $PM_{2.5}$ have not yet been fully validated. Additionally, significant shortcomings exist in the data that are available to perform these analyses. While containing identifiable shortcomings and uncertainties, EPA believes the models and assumptions used in the analysis are reasonable based on the available evidence.

- Another dimension adding to the uncertainty of this analysis is time. In our analysis we are projecting over a 30 year time period, which can introduce significant uncertainty.

Projected growth in factors such as population, income, source-level emissions, and vehicle miles traveled over the 30-year period have a significant effect on the benefits estimates, as will changes in health baselines, technology, and other factors. In addition, there is no clear way to predict future meteorological conditions compared to those used in these analyses. Again, EPA believes that the assumptions used to capture these elements are reasonable based on the available evidence..

- Qualitative and more detailed discussions of the above and other uncertainties and limitations are included in detail in earlier sections. Where information and data exist, quantitative characterizations of these uncertainties are included (in this chapter, the benefits TSD, and Appendix VII-A). However, data limitations prevent an overall quantitative estimate of the uncertainty associated with final estimates. Nevertheless, the reader should keep all of these uncertainties and limitations in mind when reviewing and interpreting the results.

- The primary benefit estimate does not include the monetary value of health benefits from ozone changes in the Western U.S. It also does not include the monetary value of several known ozone and PM-related welfare effects, including residential visibility, recreational visibility in over half of Federal Class I areas, agricultural and forestry benefits in the Western U.S. and for many crops and species, household soiling and materials damage, and deposition of nitrogen to sensitive estuaries.

Nonetheless, if one is mindful of these limitations, the relative magnitude of the benefit-cost comparison presented here can be useful information. Thus, this section summarizes the benefit and cost estimates that are potentially useful for evaluating the efficiency of the final HD Engine/Diesel Fuel rulemaking.

Our estimates of annual costs for this rulemaking are developed in Chapter V. As described in that chapter, at a 7 percent discount rate, the total program cost in 2030 is approximately **$4.3 billion (1999$)**. If a discount rate of 3% is used instead, this cost estimate drops to approximately **$4.2 billion (1999$)**. This latter value is used in our comparison of costs to benefits for calendar year 2030.

The primary estimate of monetized benefits is **$70.4 billion (1999$).** Comparing this with costs of **$4.2 billion (1999$)**, monetized net benefits are approximately **$66.2 billion (1999$).** Therefore, implementation of the HD Engine/Diesel Fuel program will provide society with a net gain in social welfare based on economic efficiency criteria. Table VII-26 summarizes the costs, benefits, and net benefits for the HD Engine/Diesel Fuel rule. Note that the cost and benefit estimates presented in Table VII-26 assume a 3 percent discount rate. Assuming a 7 percent discount rate does not materially alter the outcome. Net benefits are reduced by $3.9 billion to $62.3 billion, a reduction of 6 percent.

Table VII-26. 2030 Annual Monetized Costs, Benefits, and Net Benefits for the Final HD Engine/Diesel Fuel Rule

	Billions of 1999$
Annual compliance costs	$4.2
Monetized PM-related benefits[B,C]	$69.0 + $\mathbf{B_{PM}}$
Monetized Ozone-related benefits[B,D]	$1.4 + $\mathbf{B_{Ozone}}$
NMHC-related benefits	not monetized ($\mathbf{B_{NMHC}}$)
CO-related benefits	not monetized ($\mathbf{B_{CO}}$)
Total annual benefits	$70.4 +$\mathbf{B_{PM}}$ + $\mathbf{B_{Ozone}}$ + $\mathbf{B_{NMHC}}$ + $\mathbf{B_{CO}}$
Monetized net benefits[E]	$66.2 + **B**

[A] For this section, all costs and benefits are rounded to the nearest 100 million. Thus, figures presented in this chapter may not exactly equal benefit and cost numbers presented in earlier sections of the chapter.

[B] Not all possible benefits or disbenefits are quantified and monetized in this analysis. Potential benefit categories that have not been quantified and monetized are listed in Table VII-1. Unmonetized PM- and ozone-related benefits are indicated by B_{PM}. And B_{Ozone}, respectively.

[C]

[D] Ozone-related benefits are only calculated for the Eastern U.S. due to unavailability of reliable modeled ozone concentrations in the Western U.S. This results in an underestimate of national ozone-related benefits. See US EPA (2000a) for a detailed discussion of the UAM-V ozone model and model performance issues.

[E] **B** is equal to the sum of all unmonetized benefits, including those associated with PM, ozone, CO, and NMHC.

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Appendix VII-A: Supplementary Benefit Estimates and Sensitivity Analyses of Key Parameters in the Benefits Analysis

A. Introduction and Overview

In chapter VII, we estimated the benefits of the final HD Engine/Diesel Fuel rule using the most comprehensive set of endpoints available. For some health endpoints, this meant using a concentration-response (C-R) function that linked a larger set of effects to a change in pollution, rather than using C-R functions for individual effects. For example, the minor restricted activity day endpoint covers most of the symptoms used to characterize days of moderate or worse asthma and shortness of breath. For premature mortality, we selected a C-R function that captured reductions in incidences due to both long and short-term exposures to ambient concentrations of particulate matter (PM). In addition, the premature mortality C-R function is expected to capture at least some of the mortality effects associated with exposure to ozone. This ozone effect is described more fully below in section A.2.

In order to provide the reader with a fuller understanding of the health effects associated with reductions in air pollution associated with the final HD Engine/Diesel Fuel rule, this appendix provides estimates for those health effects which, if included in the primary estimate, could result in double-counting of benefits. For some endpoints, such as ozone mortality, additional research is needed to provide separate estimates of the effects for different pollutants, i.e. PM and ozone. These supplemental estimates should not be considered as additive to the primary estimate of benefits, but illustrative of these issues and uncertainties. Supplemental estimates included in this appendix include premature mortality associated with short-term exposures to PM and ozone, acute respiratory symptoms in adults, shortness of breath in asthmatic children, and occurrences of moderate or worse asthma symptoms in asthmatic adults. In addition, an estimate of the avoided incidences of premature mortality in infants is provided. Because the Pope, et al. estimate applies only to adults, avoided incidences of infant mortality are additive to the primary benefits estimate.

Tables VII-24 and VII-25 in Chapter VII reports the results of alternative calculations based on plausible alternatives to the assumptions used in deriving the primary estimate of benefits. In addition to these calculations, four important parameters, the length and structure of the potential lag in mortality effects, thresholds in PM health effects, discount rates, and the income elasticity of WTP have been identified as key to the analysis, and are explored in this appendix through the use of sensitivity analyses.

B. Supplementary Benefit Estimates

In the primary estimate, we use the Krewski, et al. (2000) study to provide the C-R function relating premature mortality to long-term PM exposure. The primary analysis assumes that this mortality occurs over a five year period, with 25 percent of the deaths occurring in the first year, 25 percent in the second year, and 16.7 percent in each of the third, fourth, and fifth years. Studies examining the relationship between short-term exposures and premature mortality can reveal what proportion of premature mortality is due to immediate response to daily variations in PM. There is only one short-term study (presenting results from 6 separate U.S. cities) that uses $PM_{2.5}$ as the metric of PM (Schwartz et al., 1996). As such, the supplemental estimate for premature mortality related to short-term PM exposures is based on the pooled city-specific, short-term $PM_{2.5}$ results from Schwartz, et al.

Based on advice from the SAB (EPA-SAB-Council-ADV-99-012, 1999), we examine ozone-related premature mortality as a supplemental estimate to avoid potential double-counting of benefits captured by the Pope, et al. PM premature mortality endpoint.[hh] There are many studies of the relationship between ambient ozone levels and daily mortality levels. The supplemental estimate is calculated using results from only four U.S. studies (Ito and Thurston, 1996; Kinney et al., 1995; Moolgavkar et al., 1995; and Samet et al., 1997), based on the assumption that demographic and environmental conditions on average would be more similar between these studies and the conditions prevailing when the HD Engine/Diesel Fuel rule is implemented. However, the full body of peer-reviewed ozone mortality studies should be considered when evaluating the weight of evidence regarding the presence of an association between ambient ozone concentrations and premature mortality. We combined these studies using probabilistic sampling methods to estimate the impact of ozone on mortality incidence. The technical support document for this analysis provides additional details of this approach (Abt Associates, 2000). The estimated incidences of short-term premature mortality are valued using the value of statistical lives saved method, as described in Chapter VII.

The estimated effect of PM exposure on premature mortality in infants (post neo-natal) is based on a single U.S. study (Woodruff et al.,1997) which, on SAB advice, was deemed too uncertain to include in the primary analysis. Adding this endpoint to the primary benefits estimate would result in an increase in total benefits.

[hh] While the growing body of epidemiological studies suggests that there may be a positive relationship between ozone and premature mortality, there is still substantial uncertainty about this relationship. Because the evidence linking premature mortality and particulate matter is currently stronger than the evidence linking premature mortality and ozone, it is important that models of the relationship between ozone and mortality include a measure of particulate matter as well. Because of the lack of monitoring data on fine particulates or its components, however, the measure of particulate matter used in most studies was generally either PM_{10} or TSP or, in some cases, Black Smoke. If a component of PM, such as $PM_{2.5}$ or sulfates, is more highly correlated with ozone than with PM or TSP, and if this component is also related to premature mortality, then the apparent ozone effects on mortality could be at least partially spurious. Even if there is a true relationship between ozone and premature mortality, after taking particulate matter into account, there would be a potential problem of double counting in this analysis if the ozone effects on premature mortality were added to the PM effects estimated by Pope et al., 1995, because, as noted above, the Pope, et al. study does not include ozone in its model.

As noted in Chapter VII, asthma affects over seven percent of the U.S. population. One study identifies a statistical association between air pollution and the development of asthma in some non-smoking adult men (McDonell et al., 1998). Other studies identify a relationship between air quality and occurrences of acute asthma attacks or worsening of asthma symptoms. Supplemental estimates are provided for two asthma related endpoints. Occurrence of moderate or worse asthma symptoms in adults is estimated using a C-R function derived from Ostro, et al. (1991). Incidences of shortness of breath (in African American asthmatics[ii]) are estimated using a C-R function derived from Ostro, et al. (1995). The magnitude of these alternative calculations confirms the magnitude of the asthma attack endpoint estimated from the Whittemore and Korn (1980) study.

Occurrence of moderate or worse asthma symptoms are valued at $41 per incidence (updated to 1999 dollars), based on the mean of average WTP estimates for the four severity definitions of a “bad asthma day,” described in Rowe and Chestnut (1986), a study which surveyed asthmatics to estimate WTP for avoidance of a "bad asthma day,” as defined by the subjects. Incidences of shortness of breath are valued at $7 per incidence, based on the mean of the median estimates from three studies of WTP to avoid a day of shortness of breath (Ostro et al., 1995; Dickie et al., 1991; Loehman et al., 1979).

Table VII-A-1 presents estimated incidences and values for the supplemental endpoints listed above. The supplemental estimate of 1,200 avoided incidences of premature mortality from short-term exposures to PM indicates that these incidences are approximately 25 percent of the total premature mortality incidences estimated using the Pope, et al. study (4,300). This lends support for the assumption that 25 percent of the premature deaths predicted to be avoided in the first year using the Pope, et al. study should be assigned to the first year after a reduction in exposure.

The infant mortality estimate indicates that exclusion of this endpoint does not have a large impact, either in terms of incidences (13) or monetary value (approximately $80 million). Estimates of the value for separate asthma endpoints are well under the estimate of the value of all respiratory symptoms. All of these supplemental estimates support the set of endpoints and assumptions chosen as the basis of the primary benefits estimate described in Chapter VII.

[ii]Shortness of breath due to PM exposure is not necessarily limited to African-American asthmatics. However, the Ostro et al. study was based on a sample of African-American children, who may be more sensitive to air pollution than the general population so we chose not to extrapolate the findings to the general population.

Table VII-A-1. Supplemental Benefit Estimates for the Final HD Engine/Diesel Fuel Rule for the 2030 Analysis Year[A]

Endpoint	*Pollutant*	*Avoided Incidence[B] (cases/year)*	*Monetary Benefits[C] (millions 1999$, adjusted for growth in real income)*
Premature mortality (short-term exposures) (all ages)	PM	2,600	$19,230
Premature mortality (short-term exposures) (all ages)	Ozone	500	$3,430
Premature mortality in infant population	PM	30	$260
Any of 19 acute respiratory symptoms	PM and ozone	4,987,600	$790
Shortness-of-breath (African-American asthmatics, 7-12)	PM	39,000	<$1
Moderate or Worse Asthma (adult asthmatics, 18-65)	PM	182,500	$10

[A]Ozone-related benefits estimated only for the Eastern U.S. due to ozone model performance issues (see chapter VII for details).
[B] Incidences are rounded to the nearest 100.
[C] Dollar values are rounded to the nearest 10.

C. Sensitivity Analyses

As discussed in Chapter VII, there are two key parameters of the benefits analysis for which there are no specific values recommended in the scientific literature. These parameters, the lag between changes in exposure to PM and reductions in premature mortality and the threshold in PM-related health effects, are investigated in this section through the use of sensitivity analyses. We perform an analysis of the sensitivity of benefits valuation to the lag structure by considering a range of assumptions about the timing of premature mortality. To examine the threshold parameter, we show how the estimated avoided incidences of PM-related premature mortality are distributed with respect to the level of modeled $PM_{2.5}$.

1. Alternative Lag Structures

As noted by the SAB (EPA-SAB-COUNCIL-ADV-00-001, 1999), "some of the mortality effects of cumulative exposures 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." However, 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 a 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." In the primary analysis, based on SAB advice, we assume that mortality occurs over a five year period,

with 25 percent of the deaths occurring in the first year, 25 percent in the second year, and 16.7 percent in each of the third, fourth, and fifth years. Readers should note that the selection of a 5 year lag is not supported by any scientific literature on PM-related mortality. Rather it is intended to be a best guess at the appropriate distribution of avoided incidences of PM-related mortality.

Although the SAB recommended the five-year distributed lag be used for the primary analysis, the SAB has also recommended that alternative lag structures be explored as a sensitivity analysis (EPA-SAB-COUNCIL-ADV-00-001, 1999). Specifically, they recommended an analysis of 0, 8, and 15 year lags. 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. and Dockery, et al. studies, respectively[jj]. However, neither the Pope, et al. or 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. studies do not contain any data either supporting or refuting 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. We also present two additional lags: a 15 year distributed lag with the distribution skewed towards the early years and a 15 year distributed lag with the distribution skewed towards the later years. This is to demonstrate how sensitive the results are not only to the length of the lag, but also to the shape of the distribution of incidences over the lag period. 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.

The estimated impacts of alternative lag structures on the monetary benefits associated with reductions in PM-related premature mortality (estimated with the Pope, et al. C-R function) are presented in Table VII-A-2. These estimates are based on the value of statistical lives saved approach, i.e. $6 million per incidence, and are presented for both a 3 and 7 percent discount rate over the lag period. The results using the primary 5-year lag are repeated here for comparison. The table reveals that the length of the lag period is not as important as the distribution of incidences within the lag period. A 15-year distributed lag with most of the incidences occurring in the early years reduces monetary benefits less than an 8-year lag with all incidences occurring at the eighth year. Even with an extreme lag assumption of 15 years, benefits are reduced by less than half relative to the no lag and primary (5-year distributed lag) benefit estimates.

[jj]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.

Table VII-A-2. Sensitivity Analysis of Alternative Lag Structures for PM-related Premature Mortality

Lag	Description	Monetary Benefit Adjusted for Growth in Real Income (millions 1999$)		Percent of Primary Estimate	
		3% discount rate	7% discount rate	3% discount rate	7% discount rate
5-year distributed	Primary estimate, incidences are distributed with 25% in the 1st and 2nd years, and 16.7% in the remaining 3 years.	$62,570	$58,770	100%	94%
None	Incidences all occur in the first year	$65,820	$65,820	105%	105%
8-year	Incidences all occur in the 8th year	$53,520	$40,990	86%	66%
15-year	Incidences all occur in the 15th year	$43,510	$25,530	70%	41%
15-year distributed - skewed early	Incidences are distributed with 30% in the 1st year, 25% in the 2nd year, 15% in the 3rd year, 6% in the 4th year, 4% in the 5th year, and the remainder 20% distributed over the last 10 years.	$61,270	$56,530	98%	90%
15-year distributed - skewed late	Incidences are distributed with 4% in the 11th year, 6% in the 12th year, 15% in the 13th year, 25% in the 14th year, and 30% in the 15th year, with the remaining 20 % distributed over the first 10 years.	$47,200	$31,280	75%	53%

2. PM Health Effect Threshold

The SAB advises that there is currently no scientific basis for selecting a threshold of 15 $\mu g/m^3$ or any other specific threshold for the PM related health effects considered in this analysis (EPA-SAB-Council-ADV-99-012, 1999). The most important health endpoint that would be impacted by a PM threshold is premature mortality, as measured by the ACS/Krewski, et al. (2000) C-R function. Krewski, et al. did not explicitly include a threshold in their analysis. However, if the true mortality C-R relationship has a threshold, then Krewski, et al.'s slope coefficient would likely have been underestimated for that portion of the C-R relationship above the threshold. This would likely lead to an underestimate of the incidences of avoided cases above any assumed threshold level. It is difficult to determine the size of the underestimate without data on a likely threshold and without re-analyzing the Krewski, et al. data. Nevertheless, it is illustrative to show at what threshold levels benefits are significantly affected.

Any of the PM-related health effects estimated in the primary analysis could have a threshold; however a threshold for PM-related mortality would have the greatest impact on the overall benefits analysis. Figure A-1 shows the effect of incorporating a range of possible thresholds, using 2030 PM levels and the ACS/Krewski, et al. (2000) study.

The distribution of premature mortality incidences in Figure A-1 indicate that approximately 90 percent of the premature mortality related benefits of the final HD Engine/Diesel Fuel rule are due to changes in PM concentrations occurring above 10 $\mu g/m^3$, and around 80 percent are due to changes above 12 $\mu g/m^3$, the lowest observed level in the ACS/Krewski, et al. study. Over 60 percent of avoided incidences are due to changes occurring above 15 $\mu g/m^3$.

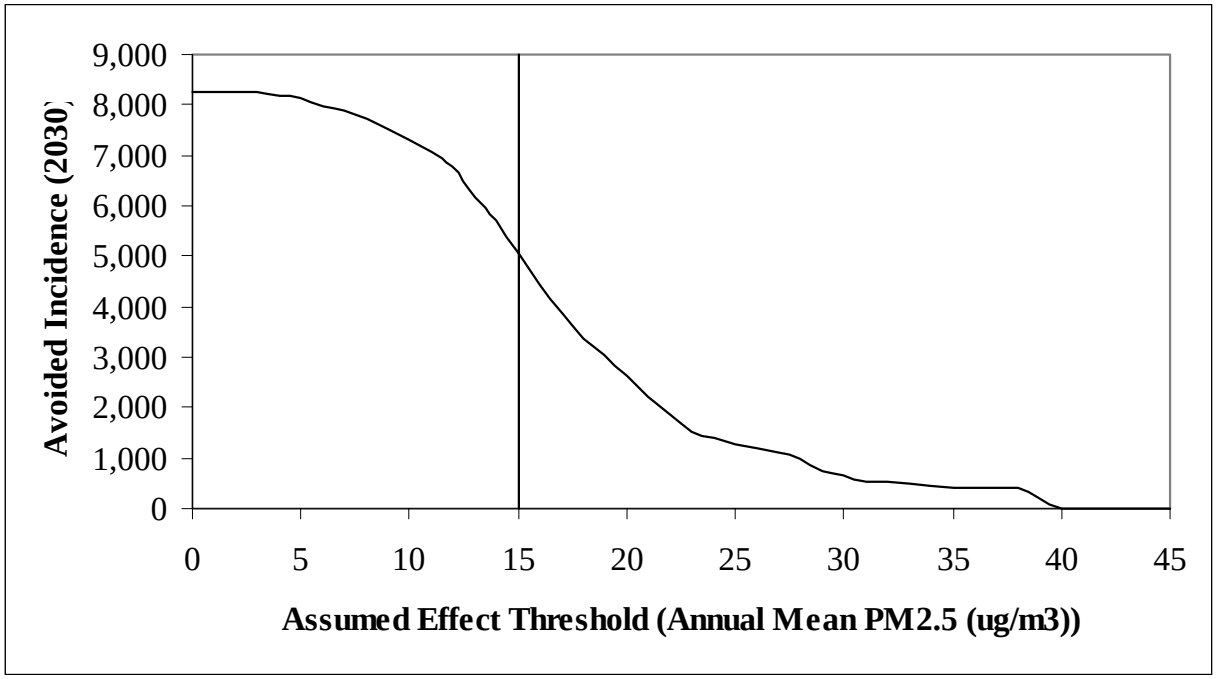

Figure VII-A-1. Impact of PM Health Effects Threshold on Avoided Incidences of Premature Mortality Estimated with the American Cancer Society/Krewski, et al. (2000) C-R Function

3. Income Elasticity of Willingness to Pay

As discussed in section C.1 of Chapter VII, our primary estimate of monetized benefits accounts for growth in real GDP per capita by adjusting the WTP for individual endpoints based on the primary estimate of the adjustment factor for each of the categories (minor health effects, severe and chronic health effects, premature mortality, and visibility). We examine how sensitive the primary estimate of total benefits is to alternative estimates of the income elasticities. The results of this sensitivity analysis are presented in Table VII-A-3. Note that the alternative elasticities and adjustment factors on which this sensitivity analysis is based are presented in Tables VII-11 and VII-12, respectively.

Consistent with the impact of mortality on total benefits, the adjustment factor for mortality has the largest impact on total benefits. The value of mortality ranges from 81 percent to 150 percent of the primary estimate based on the lower and upper sensitivity bounds on the income adjustment factor. The effect on the value of minor and chronic health effects is much less pronounced, ranging from 93 percent to 111 percent of the primary estimate for minor effects and from 88 percent to 110 percent for chronic effects.

Table VII-A-3. Sensitivity Analysis of Alternative Income Elasticities

Benefit Category	Lower Sensitivity Bound	Primary	Upper Sensitivity Bound
Minor Health Effect	$510	$550	$610
Severe and Chronic Health Effects	$2,120	$2,420	$2,670
Premature Mortality	$50,680	$62,580	$94,140
Visibility[A]	—	$3,260	—
Total Benefits	$56,980	$70,360	$97,830

[A] No range was applied for visibility because no ranges were available in the current published literature.

Chapter VII. Appendix A References

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