Air Quality Modeling

Chapter 4

Air quality modeling links changes in emissions to changes in the atmospheric concentrations of pollutants that may affect human health and the environment. A crucial analytical step, air quality modeling is one of the more complex and resource-intensive components of the prospective analysis. This chapter outlines how we estimated future-year pollutant concentrations under both the Pre- and Post-CAAA scenarios using air quality modeling results and ambient monitor data. The first section of the chapter begins with a discussion of some of the challenges faced by air quality modelers and a brief description of the models we used in this analysis. The following section provides an overview of the general methodology we used to estimate future-year ambient concentrations. This methodology section includes a description of how we used modeling results to adjust monitor concentration data and estimate ambient concentrations for the years 2000 and 2010. The third section of this chapter summarizes the results of the air quality modeling and presents the expected effects of the CAAA on future-year pollutant concentrations. A discussion of the key uncertainties associated with air quality modeling concludes the chapter.

Overview of Air Quality Models

Air quality modelers face two key challenges in attempting to translate emission inventories into pollutant concentrations. First, they must model the dispersion and transport of pollutants through the atmosphere. Second, they must model pertinent atmospheric chemistry and other pollutant transformation processes. These challenges are particularly acute for those pollutants that are not emitted directly, but instead form through secondary processes. Ozone is the best example; it forms in the atmosphere through a series of complex, non-linear chemical interactions of precursor pollutants, particularly certain classes of volatile organic compounds (VOCs) and nitrogen oxides (NO_x). We faced similar challenges when estimating PM concentrations. Atmospheric transformation of gaseous sulfur dioxide and nitrogen oxides to particulate sulfates and nitrates, respectively, contributes significantly to ambient concentrations of fine particulate matter. In addition to recognizing the complex atmospheric chemistry relevant for some pollutants, air quality modelers also must deal with uncertainties associated with variable meteorology and the spatial and temporal distribution of emissions.

Air quality modelers and researchers have responded to the need for scientifically valid and reliable estimates of air quality changes by developing a number of sophisticated atmospheric dispersion and transformation models. Some of these models have been employed in support of the development of federal clean air programs, national assessment studies, State Implementation Plans (SIPs), and individual air toxic source risk assessments. In this analysis, we used several of these well-established models to develop a picture of future changes in air quality resulting from the implementation of the 1990 CAAA.

We focused our air quality modeling efforts on estimating the impact of Pre- and Post-CAAA emissions on future-year ambient concentrations of ozone, PM_{10}, $PM_{2.5}$, SO_2, NO_x, and CO and on future-year acid deposition and visibility. The ideal model for this analysis would be a single integrated air quality model capable of estimating ambient concentrations for all criteria pollutants throughout the U.S. Although EPA is working to develop such a model, at the time of this analysis the model was not sufficiently developed and tested. In the absence of a single integrated model, we employed the Urban Airshed Model (UAM) in our analysis of ozone and used both the Regional Acid Deposition Model/Regional Particulate Model (RADM/RPM) and the Regulatory Modeling System for Aerosols and Acid

Deposition (REMSAD) model to assess PM_{10}, $PM_{2.5}$, acid deposition and visibility. All three of these models are three-dimensional grid models which require emissions and meteorological data as input. Each of these models calculate pollutant concentrations by simulating the physical and chemical pollution formation processes that occur in the atmosphere.

We conducted separate UAM, RADM/RPM, and REMSAD model runs for the 1990 base-year and each future-year projection scenario. The primary model input used for each run consisted of emissions estimates corresponding to the year and scenario being modeled (as described in Chapter 2 and Appendix A) and historical meteorological data corresponding to a past time period, referred to as a simulation period. We selected previous ozone episodes, i.e., multi-day events characterized by weather conditions conducive to ozone formation and transport (and as a result, characterized by multi-day spans with higher than average ozone concentrations), to serve as the simulation periods for UAM model runs. Although ozone concentrations during these simulation periods exceed the seasonal average, because the simulation periods for both the eastern and western U.S. cover roughly a two week span, ozone concentration peaks are largely offset by the surrounding lows. Overall, the selected simulation periods reasonably represent summertime ozone forming meteorological conditions and ozone concentrations. RADM/RPM simulation periods used to model PM, acid deposition, and visibility were chosen using a random selection process, while separate simulation periods at the beginning of each of the four seasons were chosen for REMSAD.

Table 4-1 provides an overview of the air quality models used in this analysis. We modeled concentrations of all pollutants across the 48 contiguous states; however due to the lack of an integrated model, separate air quality models were used to estimate ozone and PM for the eastern and western U.S. Table 4-1 shows the domain for each model and the simulation periods selected for use with each model and provides an overview of the spatial resolution of the models used as part of this analysis. The finer the resolution (i.e., the smaller the grid cells) the better the model can capture the effects of localized changes in emissions and weather conditions on ambient air quality. Recognizing the relationship between grid cell resolution and the certainty of results, we endeavored to estimate pollutant concentrations in more populated areas using higher resolution models. For this reason, we used the fine grid UAM-IV, an urban-scale model, to estimate ambient ozone levels in selected western cities. Similarly, we used an intermediate resolution grid (12 km x 12 km) to model ozone in "inner OTAG" states where population density is high and ozone transport is a major problem.[1]

Using the three-dimensional grid cell models, UAM, RADM/RPM, and REMSAD, we estimated grid-cell specific, hourly ozone and daily PM_{10}, and $PM_{2.5}$ concentrations for each day of the relevant simulation periods. We conducted separate model runs for the 1990 base-year and 2000 and 2010 future-year Pre- and Post-CAAA scenarios. Using these results, we ultimately projected the impact of the CAAA on ozone and PM ambient levels.

We relied on the same models used to predict PM concentrations to estimate changes in future-year acid deposition and visibility. For each model grid-cell we predicted daily acid deposition levels and visibility. Estimates for each day of the simulation period were generated for the base-year and both projection years under the Pre- and Post-CAAA scenarios.

We estimated future-year Pre- and Post-CAAA ambient SO_2, NO, NO_2, and CO concentrations by adjusting 1990 concentrations using future-year to base-year emissions ratios. This technique assumes a linear relationship between changes in emissions in an area and changes in that area's ambient concentration of the emitted pollutant.[2] Although this technique does not take into account pollutant transport or atmospheric chemistry, we believe linear scaling generates reasonable approximations of ambient concentrations of gaseous pollutants such as SO_2, NO_x, and CO.

[1] The Ozone Transport Assessment Group (OTAG) consists of the 37 easternmost states and the District of Columbia. The "inner OTAG" region is comprised of the more eastern (and more populated) states within the OTAG domain.

[2] It is important to emphasize that the correlation expected is between changes in emissions and changes in air quality. Direct correlations between the absolute emissions estimates and empirical air quality measurements used in the present analysis may not be strong due to expected inconsistencies between the geographically local, monitor proximate emissions densities affecting air quality data.

Table 4-1
Overview of Air Quality Models

Air Quality Measure	Region	Model	Spatial Resolution	Simulation Period
Ozone	Eastern U.S.	UAM-V	a) 12 km x 12 km grid for "Inner OTAG Region" b) 36 km x 36 km grid for remainder of 37-state OTAG region	July 20-30, 1993 and July 7-18, 1995
Ozone	Western U.S.	UAM-V	56 km x 56 km grid (regional scale) covering the 11 westernmost states (states west of North and South Dakota, including western Texas)	July 1-10, 1990
Ozone	San Francisco Bay Area	UAM-IV	4 km x 4 km (urban scale) grid covering the San Francisco Bay Area, the Monterrey Bay Area, Sacramento, and a portion of the San Joaquin Valley	Aug. 3-6, 1990
Ozone	Los Angeles Area	UAM-IV	5 km x 5 km grid covering the South Coast Air Basin from Los Angeles to beyond Riverside and including part of the Mojave Desert	June 23-25, 1987 and Aug. 26-28, 1987
Ozone	Maricopa County (Phoenix) Area	UAM-IV	4 km x 4 km grid covering urbanized portion of Maricopa County	Aug. 9-10, 1992 and June 13-14, 1993
Particulate Matter	Eastern U.S.	RADM/RPM	80 km x 80 km grid (coarse resolution) covering eastern North America from the Rocky Mountains eastward to Newfoundland, Canada and the Florida Keys (see Fig. C-14 in Appendix C)	30 randomly selected 5-day periods spanning a four-year period
Particulate Matter	Western U.S.	REMSAD	56 km x 56 km grid covering the 11 westernmost states	ten-day period for each of four seasons: May 1-10, July 1-10, Oct. 1-10, and Dec. 1-10
Visibility	Eastern U.S.	RADM/RPM	(same as PM)	(same as PM)
Visibility	Western U.S.	REMSAD	(same as PM)	(same as PM)
Acid Deposition	Eastern U.S.	RADM	(same as RADM/RPM)	(same as RADM/RPM)
Sulfur Dioxide	U.S.	linear scaling	56 km x 56 km REMSAD grid covering 48 contiguous states	not applicable
Oxides of Nitrogen	U.S.	linear scaling	56 km x 56 km REMSAD grid covering 48 contiguous states	not applicable
Carbon Monoxide	U.S.	linear scaling	56 km x 56 km REMSAD grid covering 48 contiguous states	not applicable

General Methodology

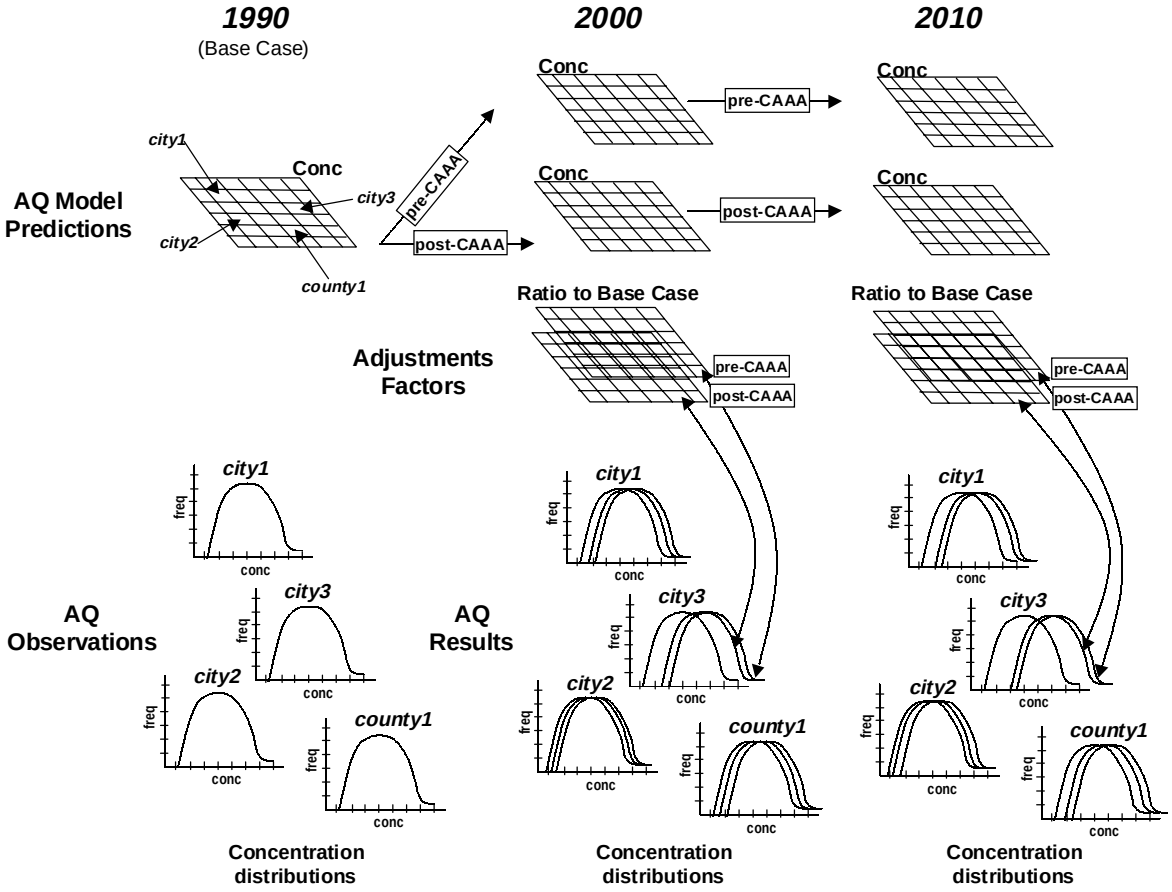
The air quality modeling component of the 812 prospective analysis involved the application of a variety of complex, sophisticated air quality modeling tools and techniques. Overall, however, the method we used to estimate the impact of changes in emissions on air quality was relatively straight forward. We began by gathering 1990 air quality monitor data for the six criteria pollutants analyzed in this study. These observational data served as the air quality baseline for both the Pre- and Post-CAAA scenarios. We then estimated 2000 and 2010 concentrations of each pollutant under each emissions scenario by applying adjustment factors to the 1990 monitor data. The adjustment factors for each future-year projection scenario were based on the relative change in pollutant concentration between 1990 and the desired future-year, as predicted by air quality simulation modeling. This section presents an overview of the methodology we used to estimate future-year ambient concentrations. For a more detailed description, please refer to Appendix C.

The diagram in Figure 4-1 illustrates the methodology used to estimate ozone and PM concentrations. First, we compiled distributions of observed pollutant concentrations recorded at each air quality monitor in 1990. We obtained these data from EPA's Aerometric Information Retrieval System (AIRS), a publicly accessible database of air quality information. Separately, we then developed distributions of estimated concentrations for each pollutant in 1990 using 1990 emissions data and the appropriate air quality model. Unlike the 1990 observed concentrations that were measured at monitoring sites, the 1990 estimated concentrations were calculated at the center of each cell of a grid covering the domain of the applicable air quality model. Using future-year emission inventory estimates for the Pre-CAAA and Post-CAAA scenarios (developed as described in Chapter 2 and Appendix A) and the appropriate air quality models, we next developed distributions of model-estimated concentrations at each grid cell for each of four future-year projection scenarios: 2000 Pre-CAAA, 2010 Pre-CAAA, 2000 Post-CAAA, and 2010 Post-CAAA. These results were used to derive adjustment factors for each air quality monitor, based on the simulation results for the grid cell in which the monitor is located. The future-year/scenario adjustment factor for each pollutant represents the ratio of the simulated future-year/scenario concentration to the 1990 model-estimated concentration. These factors were calculated by matching future-year and 1990 concentrations at regular intervals in each distribution. Finally, four sets of model-derived adjustment factors were applied to the distribution of observed 1990 concentrations at each monitor to forecast distributions of concentrations for each of the four future-year projection scenarios. It is these concentrations that serve as inputs into the CAAA benefits modeling.

An illustrative example follows. Assume the median observed concentration of Pollutant A at Monitor X in 1990 was 0.24 ppm. Air quality modeling for the grid cell in which Monitor X is located predicts a median Pollutant A concentration of 0.30 ppm in 1990 and 0.15 ppm in 2010 under the post-CAAA scenario. The 2010 Post-CAAA adjustment factor for the median Pollutant A concentration would be 0.5, and the predicted 2010 Post-CAAA median concentration at Monitor X would be 0.5 (=0.15/0.30) times the 1990 monitor value of 0.24 ppm, or 0.12 ppm.

Our approach for forecasting concentrations of SO_2, NO_x, and CO involved the same basic approach described above. However, instead of applying model-derived adjustment factors to the 1990 observed distribution of concentrations, we adjusted the 1990 distribution using the ratio of future-year emissions to 1990 emissions in the vicinity of the monitor for each of the four future-year projection scenarios. For more information about this approach, please refer to Appendix C.

Figure 4-1
Schematic diagram of the future-year concentration estimation methodology



NOTE: Figure illustrates how model results and observations are used to produce the air quality profiles (concentration distributions) for the benefits analysis. The figure shows model runs at the top; four sets of "ratios" of model results in space in the middle; and frequency distributions of pollutant monitor concentrations and the space-dependent scaling of these by the ratios of the model predictions on the bottom.

Air Quality Model Results

This section presents a summary representation of the air quality modeling results. We discuss the model-simulated concentration estimates and the adjusted future-year concentration predictions with a focus on the change in air quality resulting from the implementation of the 1990 CAAA.

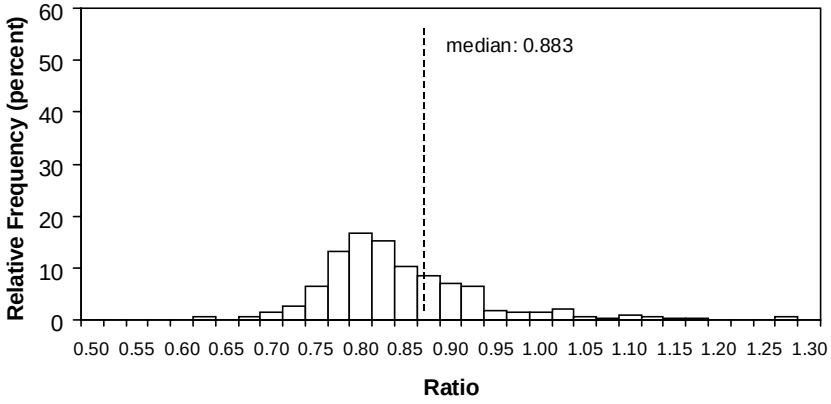
Ozone

We modeled ozone concentrations separately for the eastern U.S., western U.S., San Francisco Bay area, Los Angeles area, and Maricopa County (Phoenix, AZ) area. Examination of base-year and future-year model concentration estimates shows expected increases in Pre-CAAA ozone concentrations and expected decreases in Post-CAAA ozone concentrations in the eastern U.S. In this part of the country, UAM-V predicts Pre-CAAA ozone concentration increases will occur primarily over the states of Virginia, North Carolina, Kentucky, Tennessee, Georgia, and Alabama; while Post-CAAA decreases will be more widespread. Comparison of Pre- and Post-CAAA model estimates shows that, with the exception of a few isolated areas, ambient ozone levels throughout the East will be reduced in the year 2010 as a result of the CAAA. These lower levels are largely due to significant reductions in area source and motor vehicle VOC emissions and utility, point source, and motor vehicle NO_x emissions.

Regional-scale model results for the western U.S. indicate that ozone concentrations in this portion of the country, just as in the eastern U.S., will generally increase from the 1990 base-year under the Pre-CAAA scenario and decrease from 1990 levels under the Post-CAAA scenario. In the West, we anticipate widespread changes under both scenarios; however, we project that the increases in Pre-CAAA ozone concentrations and decreases in Post-CAAA model concentrations will be smaller than the pre-

dicted changes in ambient ozone levels in the eastern U.S Furthermore, comparison of 2010 Pre- and Post-CAAA model estimates shows that future-year western ozone concentrations will be lower as a result of the 1990 Amendments, but UAM-V results indicate that the reductions in the West will likely be about half the size of the reductions in the eastern portion of the country. The difference between the change in western ozone concentrations and the change in eastern ozone concentrations is largely due to the more aggressive NO_x controls expected in the East. Specifically, the Post-CAAA scenario incorporates the effects of a NO_x cap-and-trade system for the eastern U.S. (OTAG region). Another reason for the difference between the modeled change in eastern and western ozone concentrations is that we estimated ozone levels in the East and West using different model grid resolutions. The coarser the resolution, the less responsive the model concentration estimates are to localized changes in emissions. Thus, the smaller estimated change in western ozone concentrations than in eastern ozone concentrations may, in part, be attributable to the fact that UAM-V grid-cells covering the western U.S. are larger than those covering the eastern U.S.

Figure 4-2
Distribution of Monitor Level Ratios for 95th Percentile Ozone Concentrations: 2010 Post-CAAA/Pre-CAAA



Western urban-area modeling results differ from the regional scale results described above. Examination of Pre- and Post-CAAA modeling estimates shows that, in some portions of the urban centers of San Francisco and Los Angeles, future-year Post-CAAA ozone concentrations are expected to be higher than Pre-CAAA estimates. This ozone "disbenefit" is the result of inhibiting a complex chemical reaction termed "NO_x scavenging," during which a reduction in NO_x, an ozone precursor, leads to an increase in ozone production instead of the typical decrease.[3] In the area immediately surrounding the two cities, however, and in Maricopa County, model results show that scavenging is not expected to be influential, if it occurs at all, and future-year Post-CAAA ozone concentration estimates are predicted to be lower than Pre-CAAA estimates.

As described above, we used the UAM-V model results to calculate adjustment factors for each of the four future-year projection scenarios. We estimated future-year monitor-level ozone concentrations by applying these factors to 1990 observed concentrations. Examination of the distribution of adjusted monitor concentration ratios for 95th percentile ozone concentrations is one means of analyzing the impact of the CAAA on air pollution. The distribution of ratios of 2010 Pre-CAAA to 1990 base-year ozone concentrations reveals that the majority of future year Pre-CAAA ozone concentration estimates are between zero and 10 percent greater than 1990 levels, with most concentrations falling in the middle of this range. The distribution of ratios of 2010 Post-CAAA to 1990 base-year shows that in nearly all areas of the U.S. ozone concentrations will be lower in 2010 than in the base-year; in the majority of the country, future-year concentrations will be five to 20 percent lower than in the base-year.[4] The histogram in Figure 4-2 depicts the distribution of ratios of 2010 Post-CAAA ozone estimates to 2010 Pre-CAAA ozone estimates. Most of the ratios in the distribution are less than one, with a median of 0.883. This indicates that the 95th percentile level Post-CAAA concentrations, with few exceptions, are lower than the corresponding Pre-CAAA values. The smaller the ratio, the greater the difference between future-year scenarios.

[3] Scavenging occurs in areas, typically cities, with limited VOC and abundant NO_x. In VOC-limited areas where there is a relatively high NO_x concentration (regions where the concentration of VOC, not NO_x, dictates the amount of ozone that can be formed), these two ozone precursors (VOC and NO_x) compete to react with a particular gaseous compound. To produce ozone, this compound must combine with VOC. As a result, if the compound joins with NO_x, ozone production is impeded; thus, a decrease in NO_x leads to an increase in ozone concentrations.

[4] See Appendix C for histograms illustrating the change in ozone concentrations from the base-year.

Particulate Matter

To model Pre- and Post-CAAA particulate matter (PM_{10} and $PM_{2.5}$) concentrations, we used RADM/RPM for the eastern U.S. and REMSAD for the western U.S. Results from both models show PM concentrations are expected to be lower under the Post-CAAA scenario than under the Pre-CAAA scenario. This projected improvement in air quality is widespread throughout the eastern U.S., with 2010 Post-CAAA PM estimates in some parts of the East up to 15 to 30 percent lower than 2010 Pre-CAAA estimates. In the West, projected reductions in future-year PM concentrations (Pre-CAAA minus Post-CAAA) are largely restricted to urban areas.[5] The broad scale improvement in eastern PM concentrations is driven largely by reductions in utility source sulfur dioxide emissions throughout this portion of the country.[6] In the West, however, sulfur dioxide emissions have a much smaller impact on overall PM concentrations. Western PM concentrations are more significantly influenced by area, motor vehicle, and nonroad source emissions of nitrogen oxides and directly emitted PM. These sources are more concentrated in urban areas. As a result, the impact of the CAAA on PM concentrations in the West is primarily restricted to urban areas.

Examination of the distribution of adjusted monitor-level concentration ratios for annual average PM concentrations reveals that 2010 Pre-CAAA PM_{10} and $PM_{2.5}$ estimates are both higher than 1990 base-year estimates in almost all areas of the country. Pre-CAAA 2010 PM_{10} and $PM_{2.5}$ estimates are generally zero to 10 percent greater than 1990 base-year estimates. The average estimated increase in $PM_{2.5}$ concentrations, however, is slightly larger than the average estimated increase in PM_{10}.[7] The estimated change in PM concentrations from the base-year to 2010 under the Post-CAAA scenarios is less uniform. While the majority of areas experience a reduction in annual average PM_{10} and $PM_{2.5}$ concentrations, in a number of areas ambient PM levels, more frequently $PM_{2.5}$, increase from the base-year under the Post-CAAA scenario. On average, however, 2010 Post-CAAA PM_{10} and $PM_{2.5}$ concentrations are between zero and five percent and zero and 10 percent, respectively, lower than 1990 base-year concentrations.[8]

As shown in Figures 4-3 and 4-4, the percentage reduction in $PM_{2.5}$ concentrations across the U.S. between the Pre- and Post-CAAA scenarios vary more widely than the percentage reduction in PM_{10}. In the emissions analysis we focus on the impact of the CAAA on anthropogenic emissions and, so, hold natural source PM emissions constant at 1990 levels. Natural source emissions make up a much larger portion of PM_{10} concentrations than $PM_{2.5}$ concentrations and dampen the influence of changes in anthropogenic emissions on ambient PM_{10} concentrations.

Comparison of the two distributions in Figures 4-3 and 4-4 shows that, despite the greater variation of $PM_{2.5}$ reductions, the percentage reduction in $PM_{2.5}$ concentrations are larger on average than the percentage reduction in PM_{10} concentrations. The reason for this difference is two fold. First, as described above, $PM_{2.5}$ concentrations are more susceptible to the influence of changes in anthropogenic emissions, which are regulated by the CAAA. Second, the CAAA provisions that influence PM emissions (regulations that focus on secondary PM precursors such as NO_x, and SO_2, and primary PM sources such as diesel engine exhaust standards) affect the fine particulate ($PM_{2.5}$) subset of PM_{10} to a much greater extent than the coarser fraction that makes up the rest of PM_{10}. As a result of these two factors, the projected difference in ambient concentrations between the Pre-CAAA and Post-CAAA scenarios reflect a larger percentage reduction in $PM_{2.5}$ than PM_{10}.

[5] Outside the larger urban areas in the West, REMSAD results show little or no change in PM concentrations between Pre- and Post-CAAA estimates.

[6] Sulfur dioxide is a secondary PM precursor.

[7] In some of the figures in this chapter the Pre-CAAA and Post-CAAA scenarios are referred to as Pre-CAAA90 and Post-CAAA90, respectfully.

[8] See Appendix C for histograms illustrating the change in PM concentrations from the 1990 base-year to each of the Pre-CAAA and Post-CAAA future year scenarios.

Figure 4-3
Distribution of Combined RADM/RPM and REMSAD Derived Monitor Level Ratios for Annual Average PM_{10} Concentrations: 2010 Post-CAAA/Pre-CAAA

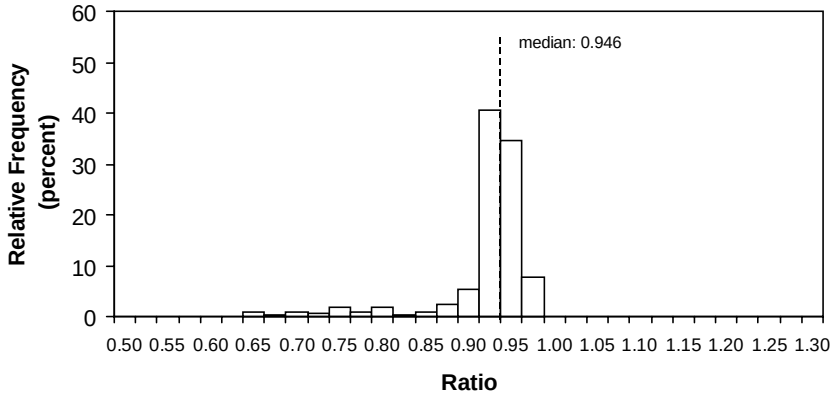
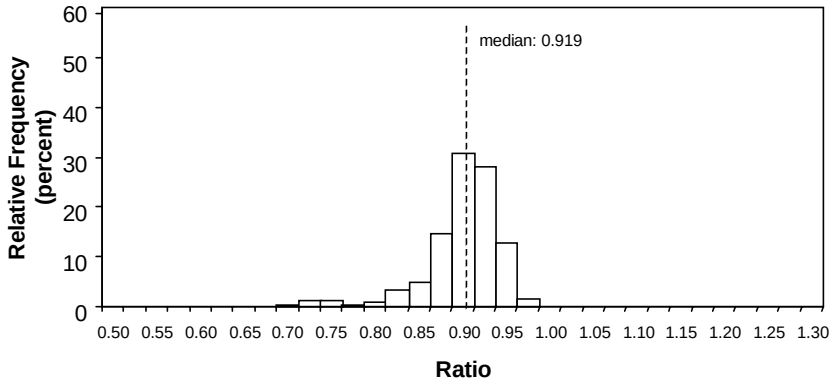


Figure 4-4
Distribution of Combined RADM/RPM and REMSAD Derived Monitor Level Ratios for Annual Average $PM_{2.5}$ Concentrations: 2010 Post-CAAA/Pre-CAAA



Visibility

We also relied on RADM/RPM and REMSAD to estimate the impact of the CAAA on future-year visibility. Tables 4-2 and 4-3 compare the mean annual visibility (expressed in deciviews)[9] in selected eastern urban areas and National Parks, respectively, as estimated by RADM/RPM under the 1990 base-year and 2010 Pre- and Post-CAAA scenarios. Comparison of these values reveals that, in the eastern U.S., we anticipate that future-year visibility in both urban and rural areas is projected to improve under the Post-CAAA scenario. RADM/RPM predicts that Post-CAAA visibility in 2010 will not only be better than Pre-CAAA visibility, but also, in many areas, it will be better than the visibility in the 1990 base-year. This improvement in visibility is attributable to reductions in the concentration of gaseous and suspended particles, such as PM, that scatter and absorb light, and thus influence visibility.

Visibility in the West is also significantly better under the Post-CAAA scenario than under the Pre-CAAA scenario (see Tables 4-4 and 4-5). Base-year model runs show that visibility in the western U.S. is the poorest in larger metropolitan areas such as Los Angeles, CA; San Francisco, CA; Denver, CO; and Phoenix, AZ. Under the 2010 Pre-CAAA scenario, REMSAD estimates that, throughout much of the West, visibility will remain relatively unchanged from the base-year, and in some cases will even improve. In the metropolitan areas, however, the model predicts visibility degradation. Under the Post-CAAA scenario, however, REMSAD estimates widespread improvement in future-year visibility in the West. In both metropolitan and non-urban areas, deciview levels estimated for 2010 are lower under the Post-CAAA scenario than under the Pre-CAAA scenario. The model suggests Los Angeles and Las Vegas will experience the greatest improvement.

[9] The deciview is a measure of visibility which captures the relationship between air pollution and human perception of visibility. When air is free of the particles that cause visibility degradation, the DeciView Haze Index is zero. The higher the deciview level, the poorer the visibility; a one to two deciview change translates to a just noticeable change in visibility for most individuals.

Table 4-2
Comparison of Visibility in Selected Eastern Urban Areas

		Mean Annual Deciview*		
Area Name	**State**	**1990 Base-Year**	**2010 Pre-CAAA**	**2010 Post-CAAA**
Atlanta Metro Area	GA	20.9	22.8	20.0
Boston Metro Area	MA	13.2	14.0	11.9
Chicago Metro Area	IL	17.5	19.1	17.0
Columbus	OH	16.5	17.7	15.1
Detroit Metro Area	MI	16.0	18.5	15.3
Indianapolis	IN	20.1	21.1	19.0
Little Rock	AR	15.0	17.2	15.1
Milwaukee Metro Area	WI	15.6	18.4	15.3
Minn.-St. Paul Metro Area	MN	10.1	12.4	10.3
Nashville	TN	20.4	21.5	19.0
New York City Metro Area	NY/NJ	15.2	18.0	13.9
Pittsburgh Metro Area	PA	15.8	16.9	14.2
St. Louis Metro Area	MO	16.5	17.8	16.0
Syracuse	NY	12.4	13.2	11.5
Washington, DC Metro Area	DC/VA/MD	17.5	19.2	16.3

*For cities or metropolitan areas not contained by a single RADM/RPM grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

Table 4-3
Comparison of Visibility in Selected Eastern National Parks

		Mean Annual Deciview*		
Area Name	**State**	**1990 Base-Year**	**2010 Pre-CAAA**	**2010 Post-CAAA**
Acadia NP	ME	11.1	12.0	10.4
Everglades NP	FL	7.6	9.2	6.9
Great Smoky Mtns. NP	TN	20.4	22.3	19.6
Shenandoah NP	VA	16.5	17.8	15.2

*For national parks not contained by a single RADM/RPM grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

Table 4-4
Comparison of Visibility in Selected Western Urban Areas

Area Name	State	Mean Annual Deciview*		
		1990 Base-Year	2010 Pre-CAAA	2010 Post-CAAA
Denver	CO	19.4	22.6	21.0
Las Vegas	NV	14.6	17.9	15.2
Los Angeles	CA	22.7	24.6	22.0
Phoenix	AZ	15.4	17.1	15.3
Salt Lake City	UT	12.5	14.8	13.4
San Francisco	CA	24.4	26.1	24.6
Seattle	WA	20.5	22.2	21.0

*For cities not contained by a single REMSAD grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

Table 4-5
Comparison of Visibility in Selected Western National Parks

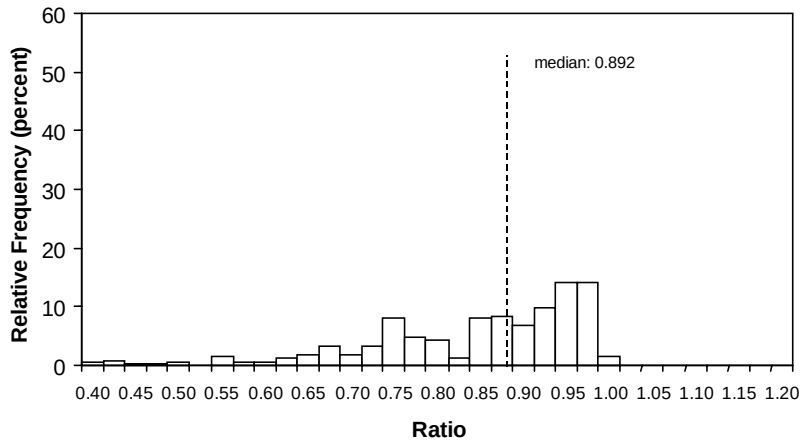
Area Name	State	Mean Annual Deciview*		
		1990 Base-Year	2010 Pre-CAAA	2010 Post-CAAA
Glacier NP	MT	11.2	11.9	11.5
Grand Canyon NP	AZ	8.3	8.8	8.3
Olympic NP	WA	11.1	11.8	11.7
Yellowstone NP	WY	9.0	9.7	9.5
Yosemite NP	CA	11.5	13.2	12.2
Zion NP	UT	8.0	9.0	8.4

*For national parks not contained by a single REMSAD grid cell, the visibility measure presented in this table is a weighted average of the mean annual deciview level from each of the grid cells that together completely contain the selected area. Weighting is based upon the spatial distribution of an area over the various grid cells.

Acid Deposition

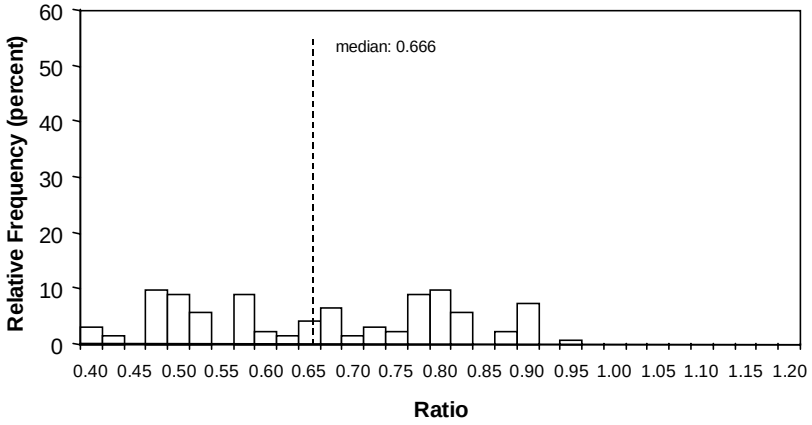
We estimated nitrogen and sulfur deposition for the 1990 base-year and each of the future-year emissions scenarios. Using RADM, we focused on acid deposition in the eastern U.S. where the acidification problem is the most acute. Under the Pre-CAAA scenario, model results show an increase in both nitrogen and sulfur deposition between 1990 and 2010. However, under the Post-CAAA scenario, 2010 deposition projections are not only lower than 2010 Pre-CAAA projections, but also below 1990 base-year levels as well. Average annual acid deposition is expected to decrease as a result of the CAAA. Motor vehicle tailpipe emissions standards and Title IV Acid Rain provisions are expected to significantly reduce both NO_x and SO_2 emissions thus contributing to significant reductions in downwind deposition of acidic nitrogen and sulfur compounds. The differences between the Pre-CAAA and Post-CAAA projections, however, imply that the 1990 Amendments will have a larger impact on the percentage reduction in nitrogen deposition than on the percentage reduction in sulfur deposition. One reason for the greater change in nitrogen deposition is the region-wide NO_x emissions cap-and-trade program that is part of the Post-CAAA scenario.

Figure 4-5
Distribution of Monitor - Level Ratios of SO_2 Emissions



Note: 2.4 percent of the distribution of ratios is less than 0.40.

Figure 4-6
Distribution of Monitor - Level Ratios of NO Emissions



Note: 3.3 percent of the distribution of ratios is less than 0.40.

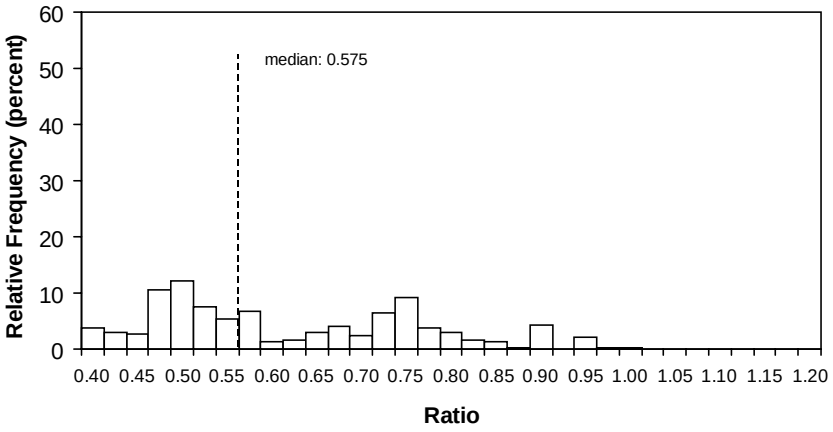
SO_2, NO, NO_2, and CO

To estimate future-year SO_2, NO, NO_2, and CO concentrations we relied on linear emissions scaling, adjusting 1990 base-year concentrations using ratios of future-year to base-year emissions. Ratios greater than one indicate an increase in ambient concentrations relative to the base-year, while ratios less than one indicate a decrease.[10]

Our results indicate that compared to the base-year, future-year concentrations of SO_2, NO, NO_2, and CO tend to increase under the Pre-CAAA scenario, while Post-CAAA concentrations for all four pollutants except SO_2 tend to decrease. For example, the median 2010 Pre-CAAA emission-based ratio for SO_2 is roughly 1.35, indicating an increase in median 2010 Pre-CAAA SO_2 concentration of approximately 35 percent from the 1990 base-year. The median ratios for NO, NO_2, and CO are roughly 1.13, 1.17, and 1.05 respectively. Under the Post-CAAA scenario we estimate that in 2010 NO, NO_2, and CO concentrations will tend to be approximately 25 and 30 percent below base-year levels. The median 2010 Post-CAAA emission-based ratios for these three pollutants are roughly 0.74, 0.70, and 0.76 respectively.

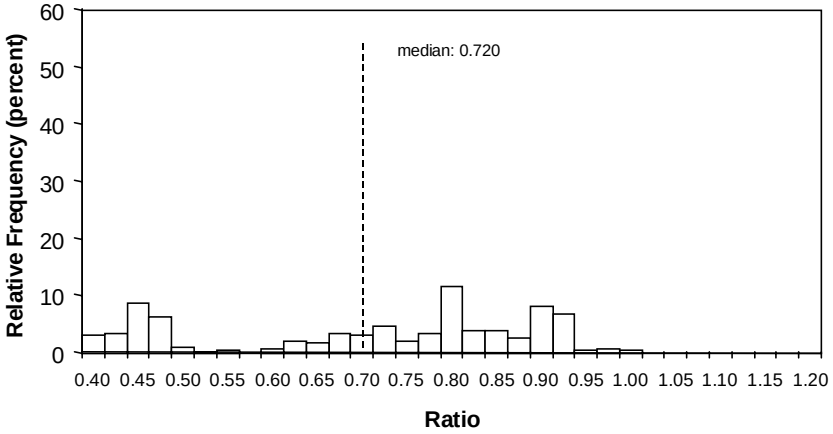
[10] The values in this section represent ratios for actual monitoring site locations. Interpolated data are not included in these figures. We believe, however, that the values presented in this section accurately reflect the impact of the 1990 Amendments on SO_x, NO, NO_2, and CO ambient concentrations.

Figure 4-7
Distribution of Monitor - Level Ratios of NO_2 Emissions



Note: 2.7 percent of the distribution of ratios is less than 0.40.

Figure 4-8
Distribution of Monitor - Level Ratios of CO Emissions



Note: 15.7 percent of the distribution of ratios is less than 0.40.

Comparison of Pre- and Post-CAAA emission-based adjustment factors also helps illustrate the effect of the 1990 Amendments on ambient pollution concentrations. The ratio of 2010 Post-CAAA adjustment factors to 2010 Pre-CAAA adjustment factors shows the impact of the 1990 Amendments on ambient concentrations relative to the baseline scenario. Ratios less than one indicate that we estimate that future-year concentrations of SO_2, NO, NO_2, and CO are lower under the Post-CAAA scenario than under the Pre-CAAA scenario.

Figures 4-5 through 4-8 show the distribution of 2010 Post-CAAA to 2010 Pre-CAAA ratios for summertime SO_2, NO, NO_2, and CO respectively. These figures illustrate the regional variation in the influence of the 1990 Amendments on ambient concentrations of these pollutants. Although we estimate concentrations in some areas will increase under the Post-CAAA scenario relative to Pre-CAAA estimates, the median summertime 2010 Post- to Pre-CAAA ratios for SO_2, NO, NO_2, and CO are 0.90, 0.67, 0.58, and 0.72 respectively. These values, each less than one, indicate that the central tendency for summertime 2010 Post-CAAA concentration estimates of these four pollutants is to be lower than 2010 Pre-CAAA estimates.

Table 4-6 displays the median values of the distribution of Post- to Pre-CAAA ratios for the summer months described above and the remaining three seasons. Just as for the summer; spring, autumn, and winter median values are less than one. Averaged over all four seasons, we estimate a median reduction in SO_2, NO, NO_2, and CO concentrations of approximately 9, 33, 40, and 25 percent respectively. RACT requirements, tailpipe emissions standards, and NO_x emissions trading account for the bulk of the reduc-

Table 4-6
Median Values of the Distribution of Ratios of 2010 Post-CAAA/ Pre-CAAA Adjustment Factors

	SO_2	NO	NO_2	CO
Spring	0.904	0.669	0.598	0.790
Summer	0.892	0.666	0.575	0.720
Autumn	0.916	0.677	0.614	0.756
Winter	0.924	0.686	0.626	0.692

tion in NO and NO_2 concentrations. Title I nonattainment area controls and Title II motor vehicle provisions are responsible for much of the change in CO concentrations, while regulation of utility and motor vehicle emissions account for majority of the decrease in SO_2 concentrations.

Uncertainty in the Air Quality Estimates

Many sources of uncertainty affect the precision and accuracy of the projected changes in air quality presented in this study. These uncertainties arise largely from potential inaccuracies in the emissions inventories used as air quality modeling inputs and potential errors in the structure and parameterization of the air quality models themselves. For example, we estimated changes in PM concentrations in the eastern U.S. based exclusively on changes in the concentrations of sulfate and nitrate particles. By not accounting for changes in organic and primary particulate fractions, we likely underestimate the impact of the CAAA on PM concentrations. Also, by using separate air quality models for individual pollutants and different geographic regions, as opposed to a single integrated model, we were unable to fully capture the interaction among air pollutants or reflect transport of pollutants or precursors across the boundaries of the models covering the western and eastern states. The direction and magnitude of bias these limitations impose on net benefits estimate presented in this analysis can not be determined based on current information.

Some model-related uncertainties, however, may be mitigated because this analysis uses the air quality modeling results in a relative, not absolute, sense. We focus on the change in air quality between the Pre- and Post-CAAA scenarios and not on the ambient concentrations projected by the individual models themselves. Therefore, uncertainties that affect a model's ability to accurately predict the relative change in concentration of a pollutant from one scenario to another are more important in the context of this study than those that affect only the absolute model results.

The relatively coarse grid cells used to model ozone in most areas of the U.S. represents a potential source of uncertainty affecting a model's sensitivity to changes in emissions. Grid size affects chemistry, transport, and diffusion processes that in turn determine the response of pollutant concentrations to changes in emissions. The less accurately a model can predict the impact of changes in emissions on ambient levels, the greater the uncertainty associated with predicted differences between Pre- and Post-CAAA concentration estimates.

Table 4-7 presents the most important specific sources of uncertainty and Appendix C further describes the uncertainties associated with air quality modeling. While the list of potential errors presented in Table 4-7 is not exhaustive, it includes discussion of those factors with the greatest likelihood of contributing to any potential bias in the primary net benefit estimates.

Table 4-7
Key Uncertainties Associated with Air Quality Modeling

Potential Source of Error	Direction of Potential Bias for Net Benefits Estimate	Likely Significance Relative to Key Uncertainties in Net Benefit Estimate*
PM_{10} and $PM_{2.5}$ concentrations in the East (RADM domain) are based exclusively on changes in the concentrations of sulfate and nitrate particles, omitting the effect of anticipated reductions in organic or primary particulate fractions.	Underestimate.	Potentially major. Nitrates and sulfates constitute major components of PM, especially $PM_{2.5}$, in most of the RADM domain and changes in nitrates and sulfates may serve as a reasonable approximation to changes in total PM_{10} and total $PM_{2.5}$. Of the other components, primary crustal particulate emissions are not expected to change between scenarios; primary organic carbon particulate emissions are expected to change, but an important unknown fraction of the organic PM is from biogenic emissions, and biogenic emissions are not expected to change between scenarios. If the underestimation is major, it is likely the result of not capturing reductions in motor vehicle primary elemental carbon and organic carbon particulate emissions.
The number of $PM_{2.5}$ ambient concentration monitors throughout the U.S. is limited. As a result, cross estimation of $PM_{2.5}$ concentrations from PM_{10} (or TSP) data was necessary in order to complete the "monitor-level" observational dataset used in the calculation of air quality profiles.	Unable to determine based on the current information.	Potentially major. $PM_{2.5}$ exposure is linked to mortality, and avoided mortality constitutes a large portion of overall CAAA benefits. Cross estimation of $PM_{2.5}$, however, is based on studies that account for seasonal and geographic variability in size and species composition of particulate matter. Also, results are aggregated to the annual level, improving the accuracy of cross estimation.
Use of separate air quality models for individual pollutants and for different geographic regions does not allow for a fully integrated analysis of pollutants and their interactions.	Unable to determine based on current information.	Potentially major. There are uncertainties introduced by different air quality models operating at different scales for different pollutants. Interaction is expected to be most significant for PM estimates. However, important oxidant interactions are represented in all PM models and the models are being used as designed. The greatest likelihood of error in this case is for the summer period in areas with NOx inhibition of ambient ozone (e.g., Los Angeles).
Future-year adjustment factors for seasonal or annual monitoring data are based on model results for a limited number of simulation days.	Overall, unable to determine based on current information.	Probably minor. RADM/RPM and REMSAD PM modeling simulation periods represent all four seasons and characterize the full seasonal distribution. Potential overestimation of ozone, due to reliance on summertime episodes characterized by high ozone levels and applied to the May-September ozone season, is mitigated by longer simulation periods, which contain both high and low ozone days. Also, underestimation of UAM-V western and UAM-IV Los Angeles ozone concentrations (see below) may help offset the potential bias associated with this uncertainty.

Table 4-7
Key Uncertainties Associated with Air Quality Modeling (continued)

Potential Source of Error	Direction of Potential Bias for Net Benefits Estimate	Likely Significance Relative to Key Uncertainties in Net Benefit Estimate*
Comparison of modeled and observed concentrations indicates that ozone concentrations in the western states were somewhat under-predicted by the UAM-V model, and ozone concentrations in the Los Angeles area were underestimated by the UAM-IV model.	Unable to determine based on current information.	Probably minor. Because model results are used in a relative sense (i.e., to develop adjustment factors for monitor data) the tendency for UAM-V or UAM to underestimate absolute ozone concentrations would be unlikely to affect overall results. To the extent that the model is not accurately estimating the relative changes in ozone concentrations across regulatory scenarios, the effect could be greater.
Ozone modeling in the eastern U.S. relies on a relatively coarse 12 km grid, suggesting NO_x inhibition of ambient ozone levels may be under represented in some eastern urban areas. Coarse grid may affect both model performance and response to emissions changes.	Unable to determine based on current information.	Probably minor. Though potentially major for eastern ozone results in those cities with known NO_x inhibition, ozone benefits contribute only minimally to net benefit projections in this study. Grid size affects chemistry, transport, and diffusion processes which in turn determine the response to changes in emissions, and may also affect the relative benefits of low-elevation versus high-stack controls. However, the approach is consistent with current state-of-the-art for regional-scale ozone modeling.
UAM-V modeling of ozone in the western U.S. uses a coarser grid than the eastern UAM-V (OTAG) or UAM-IV models, limiting the resolution of ozone predictions in the West.	Unable to determine based on current information.	Probably minor. Also, probably minor for ozone results. Grid cell-specific adjustment factors for monitors are less precise for the west and may not capture local fluctuations. However, exposure tends to be lower in the predominantly non-urban west, and models with finer grids have been applied to three key population centers with significant ozone concentrations. May result in underestimation of benefits in the large urban areas not specifically modeled (e.g., Denver, Seattle) with finer grid.
Emissions estimated at the county level (e.g., area source and motor vehicle NO_x and VOC emissions) are spatially and temporally allocated based on land use, population, and other surrogate indicators of emissions activity. Uncertainty and error are introduced to the extent that area source emissions are not perfectly spatially or temporally correlated with these indicators.	Unable to determine based on current information.	Probably minor. Potentially major for estimation of ozone, which depends largely on VOC and NO_x emissions; however, ozone benefits contribute only minimally to net benefit projections in this study.
The REMSAD model under-predicted western PM concentrations during fall and winter simulation periods.	Unable to determine based on current information.	Probably minor. Because model results are used in a relative sense (i.e., to develop adjustment factors for monitor data) REMSAD's underestimation of absolute PM concentrations would be unlikely to significantly affect overall results. To the extent that the model is not accurately estimating the relative changes in PM concentrations across regulatory scenarios, or the individual PM components (e.g., sulfates, primary emissions) do not vary uniformly across seasons, the effect could be greater.

Table 4-7
Key Uncertainties Associated with Air Quality Modeling (continued)

Potential Source of Error	Direction of Potential Bias for Net Benefits Estimate	Likely Significance Relative to Key Uncertainties in Net Benefit Estimate*
Lack of model coverage for acid deposition in Western states.	Underestimate	Probably minor. Because acid deposition tends to be a more significant problem in the eastern U.S. and acid deposition reduction contributes only minimally to net monetized benefits, the monetized benefits of reduced acid deposition in the western states would be unlikely to significantly alter the total estimate of monetized benefits.
Uncertainties in biogenic emissions inputs increase uncertainty in the AQM estimates.	Unable to determine based on current information.	Probably minor. Potentially major impacts for ozone outputs, but ozone benefits contribute only minimally to net benefit projections in this study. Uncertainties in biogenics may be as large as a factor of 2 to 3. These biogenic inputs affect the emissions-based VOC/NO_x ratio and, therefore, potentially affect the response of the modeling system to emissions changes.

* The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

Human Health Effects of Criteria Pollutants

Chapter 5

Health benefits resulting from improved air quality constitute a significant portion of the overall benefits of the Clean Air Act Amendments of 1990. As part of the prospective analysis of these amendments, we have identified and, where possible, estimated the magnitude of the health benefits that Americans are likely to enjoy in future years as a result of the CAAA. These health benefits are expressed as avoided cases of air-pollution related health effects such as premature mortality, heart disease, and respiratory illness. This chapter presents an overview of our approach to modeling these changes in adverse health effects, discusses key assumptions associated with this approach, and summarizes modeling results for major health effect categories. Although this chapter focuses predominantly on the human health effects associated with exposure to criteria pollutants, the final section of this chapter presents a discussion of the effects associated with air toxics and stratospheric ozone.

In general, this analysis finds that the CAAA will result in significant reductions in mortality, respiratory illness, heart disease, and other adverse health effects, with much of these reductions resulting from decreases in ambient particulate matter concentrations.

Analytical Approach

We estimate the impact of the CAAA on human health by analyzing the difference in the expected incidence of adverse health effects between the Pre-CAAA and Post-CAAA regulatory scenarios. As described in Chapter 2, the Pre-CAAA scenario assumes no further controls on criteria pollutant emissions besides those already in place in 1990, while the Post-CAAA scenario assumes full implementation of the 1990 CAAA. For each regulatory scenario, we use the Criteria Air Pollutant Modeling System (CAPMS) to estimate the incidence of health effects for 1990 (base-year), 2000, and 2010. Modeling the incidence of adverse health effects resulting from exposure to criteria air pollutants requires three types of inputs: (1) estimates of the changes in air quality for the Pre- and Post-CAAA scenarios in 2000 and 2010; (2) estimates of the number of people exposed to air pollutants at a given location; and (3) concentration-response (C-R) functions that link changes in air pollutant concentrations with changes in adverse health effects. We discuss each of these inputs in greater detail below.

Air Quality

The development of criteria pollutant concentration estimates for use in the CAPMS model consists of two steps. First, air quality modeling and 1990 base-year monitoring data are used to project ambient pollution levels at monitors throughout the 48 contiguous states. Second, because air quality monitors are neither uniformly nor pervasively distributed across the country, concentration data at monitors are extrapolated to non-monitored areas in order to generate a more comprehensive air quality data set covering the 48 contiguous states and the District of Columbia.

The projections of criteria pollutant concentrations at air pollution monitors are developed as summarized in Chapter 4 and described in detail in Appendix C. Briefly, baseline 1990 concentrations at each monitor are adjusted using monitor- and pollutant-specific adjustment factors to produce estimates of concentrations in 2000 and 2010 for each regulatory scenario. Each adjustment factor reflects the relative change in the concentration of a pollutant in a specific geographic area between 1990 and the target year, as predicted by air quality modeling.

To develop pollutant concentration estimates for the entire continental U.S. we extrapolate the 1990 monitor data and the future-year estimates to the eight kilometer by eight kilometer CAPMS grid cells that cover the 48 contiguous states. Within each of these cells, we calculate an estimated pollutant concentration using data from nearby monitors according to a distance-weighted averaging method described in Appendix D. We then use these grid cell pollutant concentration estimates to predict changes in health effects among the population residing within each cell.

Population

Health benefits resulting from the CAAA are related to the change in air pollutant exposure experienced by individuals. Because the expected changes in pollutant concentrations vary from location to location, individuals in different parts of the country may not experience the same level of health benefits. This analysis apportions benefits among individuals by matching the change in air pollutant concentration in a CAPMS grid cell with the size of the population that experiences that change.

As a result, we require an estimate of the distribution of the U.S. population among CAPMS grid cells. The grid-cell-specific population counts for 1990 are derived from U.S. Census Bureau block level population data. Grid cell population estimates for future years are extrapolated from 1990 levels using the ratio of future-year and 1990 state-level population estimates provided by the U.S. Bureau of Economic Analysis.[1]

Concentration-Response Functions

We calculate the benefits attributable to the CAAA as the avoided incidence of adverse health effects. Such benefits can be measured using C-R functions specific to each health effect. C-R functions are equations that relate the change in the number of individuals in a population exhibiting a "response" (in this case an adverse health effect such as respiratory disease) to a change in pollutant concentration experienced by that population. The C-R functions used in CAPMS generate changes in the incidence of an adverse health effect using three values: the grid-cell-specific change in pollutant concentration, the grid-cell-specific population, and an estimate of the change in the number of individuals that suffer an adverse health effect per unit change in air quality.[2] As described in Appendix D, we derive this last factor, as well as the specific form of the C-R equation, from the published scientific literature for each pollutant/health effect relationship of interest.

Using the appropriate C-R functions, CAPMS generates estimates for each grid-cell of the change in incidence of a set of adverse health effects resulting from the incremental change in exposure between the Pre- and Post-CAAA scenarios in 2000 and 2010. For each health effect, CAPMS then generates national health benefits estimates by summing the annual incidence change across all grid cells.

Each criteria pollutant evaluated in the 812 prospective analysis has been associated with multiple adverse health effects. The published scientific literature contains information that supports the estimation of some, but not all, of these effects. Thus, it is not possible currently to estimate all of the human health benefits attributable to the CAAA. In addition, for some of the health effects we do quantify, the current economic literature does not support the estimation of the economic value of these effects. For each of the criteria pollutants we evaluate in this analysis, Table 5-1 presents the health effects that are quantitatively estimated and those that can not currently be quantified. The sixth criteria pollutant, lead (Pb), is not included in this analysis since airborne emissions of lead were virtually eliminated by pre-1990 Clean Air Act programs.

Key Analytical Assumptions

The modeling of health benefits attributable to the CAAA involves numerous judgments and assumptions to address data limitations and other constraints. Each of these analytical assumptions affects both the accuracy and precision with which we can estimate health benefits of the CAAA, but some as-

[1] U.S. Bureau of Economic Analysis. 1995. BEA Regional Projections to 2045: Volume 1, States. U.S. Department of Commerce. Washington, DC. July.

[2] An estimate of the baseline incidence of the adverse health effect may also be required for certain C-R functions.

Table 5-1
Human Health Effects of Criteria Pollutants

Pollutant	Quantified Health Effects	Unquantified Health Effects†
Ozone	Respiratory symptoms Minor restricted activity days Respiratory restricted activity days Hospital admissions- All Respiratory and All Cardiovascular Emergency room visits for asthma Asthma attacks	Mortality‡ 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
Particulate Matter (PM_{10}, $PM_{2.5}$)	Mortality* Bronchitis - Chronic and Acute New asthma cases Hospital admissions - All Respiratory and All Cardiovascular Emergency room visits for asthma Lower respiratory illness Upper respiratory illness Shortness of breath Respiratory symptoms Minor restricted activity days All restricted activity days Days of work loss Moderate or worse asthma status (asthmatics)	Neonatal mortality‡ Changes in pulmonary function Chronic respiratory diseases other than chronic bronchitis Morphological changes Altered host defense mechanisms Cancer Non-asthma respiratory emergency room visits
Carbon Monoxide	Hospital Admissions - All Respiratory and All Cardiovascular	Behavioral effects Other hospital admissions Other cardiovascular effects Developmental effects Decreased time to onset of angina Non-asthma respiratory emergency room visits
Nitrogen Oxides	Respiratory illness Hospital Admissions - All Respiratory and All Cardiovascular	Increased airway responsiveness to stimuli Chronic respiratory damage / Premature aging of the lungs Inflammation of the lung Increased susceptibility to respiratory infection Acute inflammation and respiratory cell damage Non-asthma respiratory emergency room visits
Sulfur Dioxide	Hospital Admissions - All Respiratory and All Cardiovascular In exercising asthmatics: Chest tightness, Shortness of breath, or Wheezing	Changes in pulmonary function Respiratory symptoms in non-asthmatics Non-asthma respiratory emergency room visits

† Some of the unquantified adverse health effects of air pollution may be associated with adverse health endpoints that we have quantitatively evaluated (e.g., chronic respiratory damage and premature mortality). However, it is likely that the value assigned to the quantified endpoint may not fully capture the value of the associated health effect (e.g., chronic respiratory damage may result in significant pain and suffering prior to mortality). As a result, we include such effects separately in the unquantified health effects column.

‡Appendix D includes detailed discussion of the scientific evidence for these potential health effects and includes illustrative benefit calculations for them. Current uncertainties in our understanding of these effects do not support including these quantitative estimates in the overall CAAA benefits estimate. However, ozone-related mortality may be implicitly quantified in the overall analysis as part of the PM mortality estimate because of the significant correlation between ozone and PM concentrations.

* This analysis estimates avoided mortality using PM as an indicator of the criteria air pollutant mix to which individuals were exposed.

sumptions introduce greater uncertainty into the results than others. This section characterizes these key assumptions and the associated uncertainties to allow the reader to gain a better understanding of the potential for misestimation of avoided health effects. In addition, health benefits are presented as ranges to reflect the aggregate effect of the uncertainty in key variables (see Results section below). This section discusses the most important analytical assumptions of this modeling effort, grouped into the following categories: (1) exposure analysis, (2) selection and application of C-R functions, and (3) estimation of changes in PM-related mortality.

Exposure Analysis

The key analytical assumptions involved in estimating exposure to criteria air pollutants relate to two steps: the extrapolation of air quality data from monitors and the mapping of population data to air quality data.

As discussed above, actual ambient air pollution data are available only for a limited number of monitor sites that are not uniformly distributed across the U.S. Thus, to estimate the impact of air pollution changes on the health of the U.S. population, data from monitors are extrapolated to the cells of a grid that covers the 48 contiguous states and are matched with population data for each grid-cell. Essentially, the extrapolation method uses data from the closest set of monitors surrounding a grid-cell to compute a weighted average concentration for that cell. Monitors closer to the grid cell are assumed to yield a more accurate estimate of air quality in the cell; thus data from these monitors receive more weight than data from more distant monitors when calculating an air quality estimate for the cell.[3] The resulting estimates are uncertain because the geography, weather, land use, and other factors influencing air pollution may differ significantly between a grid cell and the monitor or monitors used to generate estimates of air quality, especially as the monitor-to-grid-cell distance grows.[4] As a result, they may not sufficiently capture local variation in air pollution levels (e.g., hot spots).

However, since the uncertainty in these extrapolated values is inversely proportional to the density of monitors in a given area, and since air quality monitors are more prevalent in high pollution areas than in low pollution areas, this extrapolation method estimates the air quality in high pollution areas (where the potential benefits of the CAAA are greatest) with greater certainty than in low pollution areas. Thus, grid-cell ozone estimates in the eastern U.S., where ozone levels and ozone monitor density are higher, are likely to be more accurate than those in the west, where monitor coverage is more sparse. Also, estimates of concentrations of criteria pollutants, which are measured by a greater number of monitors nationwide (PM, ozone, SO_2), are expected to be less uncertain than estimates for CO and NO_x, which are measured by considerably fewer monitors.

Air pollutant concentration changes are mapped to grid-cell population data derived from U.S. Census bureau data, and extrapolated to future years using population growth estimates from the U.S. Bureau of Economic Analysis. There are two key assumptions associated with this population mapping. First, we assume the population in each grid cell grows at the same rate as the state population as a whole. As a result, exposures (and potential benefits) in individual grid cells may be either under- or over-estimated if population growth varies from the state average during the 1990 to 2010 period. This uncertainty is likely to be more significant in larger states such as California and Texas, which may have more geographic variability in growth patterns. Also, the effect of this assumption may be less significant for large population centers because their growth rate better approximates the growth rate of the state as a whole. Second, we assume in the exposure analysis that the population in the grid cell is similar in terms of its activity patterns and demographic characteristics to the populations in the studies from which the C-R functions are derived. This is a potentially significant uncertainty which is discussed further in the next section and in Appendix D.

[3] Specifically, monitor data are weighted based on the inverse of the distance between the monitor and the grid-cell center. Additional information on the extrapolation method is provided in Appendix D.

[4] In order to address this issue for long-distance extrapolation (i.e., grid cells greater than 50 kilometers from a monitor), the method is modified to also incorporate air quality modeling predictions for the source and target locations. See Appendix D for details.

Selection and Application of C-R Functions

We rely on the most recent available, published scientific literature to ascertain the relationship between air pollution and adverse human health effects. The uncertainties underlying those published studies and our method for selecting studies that could be used to derive C-R functions likely contributes to the uncertainty of the health effects results. For example, the uncertainty associated with the current state of the published scientific literature could potentially have two contradictory influences on the results of this analysis. First, to the extent that the published literature may collectively overstate the effects of pollution, our analysis will overstate the benefits of CAAA-related pollution reduction. This overestimation is possible because scientific journals tend to publish research reporting significant associations between pollution and disease more often than research that fails to find such associations. On the other hand, our analysis may underestimate overall health benefits of the CAAA because, as the state of the science evolves, current pollutant/health effect associations may be found to be stronger than previously thought, and new associations may be identified. For example, in recent years, studies have shown the potential health benefits from reductions in ambient PM to be much greater than previously believed. To the extent that the present analysis does not include health effects whose link to air pollution has not been subject to adequate scientific inquiry, this analysis may understate CAAA-related health benefits.

Our method of identifying appropriate C-R functions for use in the benefits analysis may also introduce uncertainty. We evaluate studies using the nine selection criteria summarized in Table 5-2 and described in detail in Appendix D. These criteria include consideration of whether the study was peer-reviewed, the study design and location, and characteristics of the study population, among others. The selection of C-R functions for the benefits analysis is guided by the goal of achieving a balance between comprehensiveness and scientific defensibility. However, to the extent that this selection process may lead to the exclusion of valid studies, the process introduces uncertainty into the analysis. The overall effect of this uncertainty is expected to be minor, given the emphasis of the selection process on scientific validity. Appendix D lists the studies selected for each category of health effects, and presents the associated C-R functions for each criteria pollutant.

Once the C-R functions have been selected, uncertainty may also enter the analysis due to both within-study and across-study variation in C-R functions for individual health effects. Within-study variation refers to the uncertainty and error that may surround a given study's estimate of a C-R function. Health effects studies provide both "best estimates" of the relationship between air quality changes and health effects and a measure of the statistical uncertainty of the relationship. We use statistical simulation modeling techniques to evaluate the overall uncertainty of the results given the uncertainties associated with individual studies. Across-study variation refers to the fact that different published studies of the same pollutant/health effect relationship typically do not report identical findings; in some instances the differences are substantial. These differences can exist even between equally reputable studies and may result in health effect estimates that vary considerably.

Across-study variation can result from two possible causes. One possibility is that studies report different estimates of the single true relationship between a given pollutant and a health effect due to differences in study design, random chance, or other factors. For example, a hypothetical study conducted in New York and one conducted in Seattle may report different C-R functions for the relationship between PM and mortality in part because of differences between these two study populations (e.g., demographics, activity patterns). Alternatively, study results may differ because they are in fact estimating different relationships; that is, the same reduction in PM in New York and Seattle may result in different reductions in premature mortality. This may result from a number of factors, such as differences in the relative sensitivity of these two populations to PM pollution and differences in the composition of PM in these two locations.[5] In either case, where we identify multiple studies that are appro-

[5] PM is a mix of particles of varying size and chemical properties. The composition of PM can vary considerably from one region to another depending on the sources of particulate emissions in each region.

Table 5-2
Summary of Considerations Used in Selecting C-R Functions

Consideration	Comments
Peer reviewed research	Peer reviewed research is preferred to research that has not undergone the peer review process.
Study type	Among studies that consider chronic exposure (e.g., over a year or longer) prospective cohort studies are preferred over cross-sectional studies (a.k.a. "ecological studies") because they control for important confounding variables that cannot be controlled for in cross-sectional studies. If the chronic effects of a pollutant are considered more important than its acute effects, prospective cohort studies may also be preferable to longitudinal time series studies because the latter type of study is typically designed to detect the effects of short-term (e.g. daily) exposures, rather than chronic exposures.
Study period	Studies examining a relatively longer period of time (and therefore having more data) are preferred, because they have greater statistical power to detect effects. More recent studies are also preferred because of possible changes in pollution mixes, medical care, and life style over time.
Study population	Studies examining a relatively large sample are preferred. Studies of narrow population groups are generally disfavored, although this does not exclude the possibility of studying populations that are potentially more sensitive to pollutants (e.g., asthmatics, children, elderly). However, there are tradeoffs to comprehensiveness of study population. Selecting a C-R function from a study that considered all ages will avoid omitting the benefits associated with any population age category. However, if the age distribution of a study population from an "all population" study is different from the age distribution in the assessment population, and if pollutant effects vary by age, then bias can be introduced into the benefits analysis.
Study location	U.S. studies are more desirable than non-U.S. studies because of potential differences in pollution characteristics, exposure patterns, medical care system, and life style.
Pollutants included in model	Models with more pollutants are generally preferred to models with fewer pollutants, though careful attention must be paid to potential collinearity between pollutants. Because PM has been acknowledged to be an important and pervasive pollutant, models that include some measure of PM are highly preferred to those that do not.
Measure of PM	$PM_{2.5}$ and PM_{10} are preferred to other measures of particulate matter, such as total suspended particulate matter (TSP), coefficient of haze (COH), or black smoke (BS) based on evidence that $PM_{2.5}$ and PM_{10} are more directly correlated with adverse health effects than are these other measures of PM.
Economically valuable health effects	Some health effects, such as forced expiratory volume and other technical measurements of lung function, are difficult to value in monetary terms. These health effects are not quantified in this analysis.
Non-overlapping endpoints	Although the benefits associated with each individual health endpoint may be analyzed separately, care must be exercised in selecting health endpoints to include in the overall benefits analysis because of the possibility of double counting of benefits. Including emergency room visits in a benefits analysis that already considers hospital admissions, for example, will result in double counting of some benefits if the category "hospital admissions" includes emergency room visits.

priate for estimating a given health effect, we use the multiple C-R estimates, applied to the entire U.S., to derive a range of possible results for that health effect.

Whether this analysis estimates the C-R relationship between a pollutant and a given health endpoint using a single function from a single study or using multiple C-R functions from several studies, each C-R relationship is applied throughout the U.S. to generate health benefit estimates. However, to the extent that pollutant/health effect relationships are region-specific, applying a location-specific C-R function at all locations in the U.S. may result in overestimates of health effect changes in some locations and underestimates of health effect changes in other locations. It is not possible, however, to know the extent or direction of the overall effect on health benefit estimates introduced by application of a single C-R function to the entire U.S. This may be a sig-

nificant uncertainty in the analysis, but the current state of the scientific literature does not allow for a region-specific estimation of health benefits.

PM-Related Mortality

This section discusses the estimation of one of the most serious health impacts of air pollution: premature mortality associated with PM exposure. This section consists of three parts. It begins with a discussion of the uncertainties surrounding the PM/mortality relationship. Then, it presents specific factors to consider when selecting a PM mortality C-R function. It ends with a brief discussion of the advantages and disadvantages of the study we selected for the PM mortality analysis: Pope et al., 1995.

Uncertainties in the PM Mortality Relationship

Health researchers have consistently linked air pollution, especially PM, with excess mortality. A substantial body of published scientific literature recognizes a correlation between elevated PM concentrations and increased mortality rates. However, there is much about this relationship that is still uncertain.[6] These uncertainties include:

- **Causality.** For this analysis, we assume a causal relationship between exposure to elevated PM and premature mortality, based on the evidence of a correlation between PM and mortality reported in the scientific literature. This assumption is necessary because the epidemiological studies on which this analysis relies, by design, can not definitively prove causation.

- **Other Pollutants.** PM concentrations are correlated with the concentrations of other criteria pollutants, such as ozone and CO, and it is unclear how much each pollutant may influence elevated mortality rates. Recent studies have explored whether ozone and CO may have mortality effects independent of PM, but we do not view the evidence as sufficient to include such effects in the overall CAAA-related health benefits estimate.[7] As a result, we use the reported PM/mortality relationship as a proxy for the mortality effects of the air pollutant mixture.

- **Shape of the C-R Function.** The shape of the true PM mortality C-R function is uncertain, but this analysis assumes the C-R function to have a log-linear form (as derived from the literature) throughout the relevant range of exposures.[8] If this is not the correct form of the C-R function, or if certain scenarios (e.g., 2010 Pre-CAAA) predict concentrations well above the range of values for which the C-R function was fitted, avoided mortality may be mis-estimated.

- **Regional Differences.** As discussed earlier, significant variability exists in the results of different PM studies. This variability may reflect regionally-specific C-R functions resulting from regional differences in factors such as the physical and chemical composition of PM. If true regional differences exist, applying these C-R functions to regions other than the study location would result in mis-estimation of effects in these regions.

- **Exposure/Mortality Lags.** 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/mortality relationship. The existence of such a lag could be important for the valuation of benefits, if one were to assume that lagged incidences of premature mortality should be discounted over the period between when the fatal increment of exposure is experienced and premature mortality actually occurs. 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) leaves us skeptical that all inci-

[6] The morbidity studies used in this analysis may also be subject to many of the uncertainties listed in this section.

[7] Appendix D discusses the evidence linking both ozone and CO with mortality. It also describes and presents the results of an illustrative analysis estimating CAAA-related reductions in ozone-related mortality using currently available studies.

[8] C-R functions for other health effects may be assumed to be linear or log-linear. See Appendix D for more details.

dences of premature mortality associated with a given incremental change in PM exposure would occur in the same year as the exposure reduction. This same literature implies that lags of up to a few years are plausible, and we chose to assume a five-year lag structure, with 25 percent of deaths occurring in the first year, another 25 percent in the second year, and 16.7 percent in each of the remaining three years.

- **Cumulative Effects.** We attribute the PM/mortality relationship used in this study (Pope et al., 1995) primarily to PM-associated cumulative damage to the cardiopulmonary system, since the short-term mortality estimates reported in time-series studies account for only a minor fraction of total excess mortality. However, the relative roles of exposure duration and exposure level remain unknown at this time.

Selection of a PM Mortality C-R Function

In addition to the study selection criteria listed in Table 5-2, we consider three additional factors when selecting a PM mortality function. The first focuses on the PM indicator (i.e., PM_{10} or $PM_{2.5}$), the second focuses on whether the study measured short-term or long-term PM exposure, and the third focuses on whether the study used a cohort or ecologic design.

Current research suggests that particle size matters when estimating the health impacts of PM. Particulate matter is a heterogeneous mixture that includes particles of varying sizes. Fine PM is generally viewed as having a more harmful impact than coarse PM, especially for coarse particles larger than 10μm in aerodynamic diameter, although it is not clear to what extent this may differ by the type of health effect or the exposed population. While one cannot necessarily assume that coarse PM has no adverse impact on health, we prefer the use of $PM_{2.5}$ as the best currently available measure of the impact of PM on mortality.[9]

Two types of exposure studies (short-term and long-term) have been used to estimate a PM/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., annual) changes in exposure to PM and annual mortality rates. Researchers have found significant correlations using both types of studies; however, for this analysis, we rely exclusively on long-term studies to quantify PM mortality effects, though the short-term studies provide additional scientific evidence supporting the PM/mortality relationship.

Because short-term studies focus only on the acute effects associated with daily peak exposures, they are unable to evaluate the degree to which observed excess mortality is premature,[10] and they may underestimate the C-R coefficient because they do not account for the cumulative mortality effects of long-term exposures (i.e., exposures over many years rather than a few days). Long-term studies, on the other hand, are able to discern changes in mortality rates due to long-term exposure to elevated air pollution concentrations, and are not limited to measuring mortalities that occur within a few days of a high-pollution event (though they may not predict cases of premature mortality that were only hastened by a few days). Consequently, the use of C-R functions derived from long-term studies is likely to result in a more complete assessment of the effect of air pollution on mortality risk. However, to the extent that long-term studies fail to capture acute mortality effects related to peak exposures, the use of long-term mortality studies may underestimate CAAA-related avoided mortality benefits.

Among long-term PM studies, we prefer studies using a prospective cohort design to those using an ecologic or population-level design. Prospective

[9] Due to the relative abundance of studies using PM_{10}, however, and the reasonably good correlation between $PM_{2.5}$ and PM_{10}, the 812 prospective analysis also uses PM_{10} studies to estimate the impact of PM on non-mortality health effects.

[10] This can be important in cost-benefit analysis if benefits are estimated in terms of life-years lost. In short-term studies evaluating peak pollution events, it is likely that many of the "excess mortality" cases represented individuals who were already suffering impaired health, and for whom the high-pollution event represented an exacerbation of an already serious condition. Based on the episodic studies only, however, it is unknown how many of the victims would have otherwise lived only a few more days or weeks, or how many would have recovered to enjoy many years of a healthy life in the absence of the high-pollution event.

cohort studies follow individuals forward in time for a specified period, periodically evaluating each individual's exposure and health status. Population-level ecological studies assess the relationship between population-wide health information (such as counts for daily mortality) and ambient levels of air pollution. Prospective cohort studies are preferred because they are better at controlling a source of uncertainty known as "confounding." Confounding is the mis-estimation of an association that results if a study does not control for factors that are correlated with both the outcome of interest (e.g., mortality) and the exposure of interest (e.g., PM exposure). For example, smoking is associated with mortality. If populations in high PM areas tend to smoke more than populations in low PM areas, and a PM exposure study does not include smoking as a factor in its model, then the mortality effects of smoking may be erroneously attributed to PM, leading to an overestimate of the risk from PM. Prospective cohort studies are better at controlling for confounding than ecologic studies because the former follow a group of individuals forward in time and can gather individual-specific information on important risk factors such as smoking. However, it is always possible, even in well-designed studies, that a relevant risk factor (e.g., climate, the presence of other pollutants) may not have been adequately considered or controlled for. As a result, it is possible that differences in mortality rates ascribed to differences in average PM levels may be due, in part, to some other factor or factors (e.g., differences among communities in diet, exercise, ethnicity, climate, industrial effluents, etc.) that have not been adequately addressed in the exposure models.

The Pope Study

Three recent studies have examined the relationship between mortality and long-term exposure to PM: Pope et al. (1995), Dockery et al. (1993), and Abbey et al. (1991). Of these three studies, we prefer using the Pope et al. study as the basis for developing the primary PM mortality estimates in this analysis. Pope et al. studied the largest cohort, had the broadest geographic scope, and effectively controlled for potentially significant sources of confounding.

Pope et al. examined a much larger population (over 295,000) and many more locations (50 metropolitan areas) than either the Dockery study or the Abbey study. The Dockery study covered a cohort of over 8,000 individuals in six U.S. cities, and the Abbey study covered a cohort of 6,000 people in California. In particular, the cohort in the Abbey study was considered substantially too small and too young to enable the detection of small increases in mortality risk. The study was therefore omitted from consideration in this analysis. Even though Pope et al. (1995) reports a smaller premature mortality response to elevated PM than Dockery et al. (1993), the results of the Pope study are nevertheless consistent with those of the Dockery study.

Pope et al., (1995) is unique in that it followed a largely white and middle class population. The use of this study population reduces the potential for confounding because it decreases the likelihood that differences in premature mortality across locations were attributable to differences in socioeconomic status or related factors rather than PM. However, the demographics of the study population may also produce a downward bias in the PM mortality coefficient, because short-term studies indicate that the effects of PM tend to be significantly greater among groups of lower socioeconomic status.

Although it is the strongest of the PM cohort studies, Pope et al. does have some limitations. For example, Pope et al. did not consider the migration of cohort members across study cities, which would cause exposures to be more similar across individuals than those indicated by assigning city-specific annual average pollution levels to each member of the cohort. As intercity migration increases among cohort members, the exposure experienced by migrating individuals will tend toward an intercity mean. If this migration is significant and is ignored, approximating true differences in exposure levels by differences in city-specific annual average PM levels will exaggerate changes in exposure, resulting in a downward bias of the PM coefficient. This occurs because a given difference in mortality rates is being associated with a larger difference in PM levels than that actually experienced by individuals in the study cohort. When the relationship between elevated PM exposure and premature mortality derived from the

Pope et al. study is applied in the present analysis, the effect of the potential mis-specification of exposure due to migration in the underlying study is to underestimate PM-related mortality reduction benefits attributable to the CAAA.

Also, Pope et al. only included PM when estimating a C-R function. Because PM concentrations are correlated with the concentrations of other criteria air pollutants (e.g., ozone), and because these other pollutants may be correlated with premature mortality (see Appendix D), the PM risk estimate may be overestimated because it includes the mortality impacts of these confounders. However, in an effort to avoid overstating benefits, and because the evidence associating mortality with PM exposure is stronger than for other pollutants, the 812 Prospective analysis uses PM as a surrogate for PM and related criteria pollutants.

Although we use the Pope study exclusively to derive our primary estimates of avoided mortality, the C-R function based on Dockery et al. (1993) may provide a reasonable alternative estimate. While the Dockery et al. study used a smaller sample of individuals from fewer cities than the study by Pope et al., 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. We present an alternative estimate of the premature adult mortality associated with long-term PM exposure based on Dockery et al. (1993) in Chapter 8 and in Appendix D. We emphasize, however, that the estimate based on Pope et al. (1995) is our primary estimate of the effect of the 1990 Amendments on this important health effect.

Health Effects Modeling Results

This section presents a summary of the differences in health effects resulting from improvements in air quality between the Pre-CAAA and Post-CAAA scenarios. Table 5-3 summarizes the CAAA-related avoided health effects in 2010 for each study included in the analysis. The mean estimate is presented as the Primary Central estimate, the 5th percentile observation from the statistical uncertainty modeling is presented as the Primary Low estimate, and the 95th percentile observation is presented as the Primary High estimate of the number of avoided cases of each endpoint.[11] To provide context for these results, Table 5-3 also expresses the mean reduction in incidence for each adverse health effect as a percentage of the baseline incidence of that effect (extrapolated to the appropriate future year) for the population considered (e.g., adults over 30 years of age). In general, because the differences in air quality between the Pre- and Post-CAAA scenarios are expected to increase from 1990 to 2010 and because population is also expected to increase during that time, the health benefits attributable to the CAAA are expected to increase consistently from 1990 to 2010. More detailed results are presented in Appendix D.

Avoided Premature Mortality Estimates

Table 5-3 summarizes the avoided mortality due to reductions in PM exposure in 2010 between the Pre- and Post-CAAA scenarios. As this table shows, our Primary Central estimate implies that PM reductions due to the CAAA in 2010 will result in 23,000 avoided deaths, with a Primary Low and Primary High bound on this estimate of 14,000 and 32,000 avoided deaths, respectively. The Primary Central estimate of 23,000 avoided deaths represents roughly one percent of the projected annual non-accidental mortality of adults aged 30 and older in the year 2010. Additionally, Table 5-4 summarizes the distribution of avoided mortality for 2010 by age cohort, along with the expected remaining lifespan (i.e., the life years lost) for the average person in each age cohort. The majority of the estimated deaths occur in people over the age of 65 (due to their higher baseline mortality rates), and this group has a shorter life expectancy relative to other age groups. The life years lost estimates might be higher if data were available for PM-related mortality in the under 30 age group.

[11] The Primary Low, Primary Central and Primary High health benefit estimates represent points on a distribution of estimated incidence changes for each health effect. This distribution reflects the uncertainty associated with the coefficient of the C-R function for each health endpoint. More information about C-R function uncertainty and the uncertainty modeling that generates the results distributions is presented in Appendix D.

Table 5-3
Change in Incidence of Adverse Health Effects Associated with Criteria Pollutants in 2010 (Pre-CAAA minus Post-CAAA) – 48 State U.S. Population (avoided cases per year)

		2010			% of Baseline Incidences for the mean estimates [a]
Endpoint	**Pollutant**	**5th %**	**mean**	**95th %**	**2010**
Mortality					
ages 30 and older	PM	14,000	23,000	32,000	1.00%
Chronic Illness					
chronic bronchitis	PM	5,000	20,000	34,000	3.14%
chronic asthma	O_3	1,800	7,200	12,000	3.83%
Hospitalization					
respiratory admissions	PM, CO, NO_2, SO_2, O_3	13,000	22,000	34,000	0.62%
cardiovascular admissions	PM, CO, NO_2, SO_2, O_3	10,000	42,000	100,000	0.86%
emergency room visits for asthma	PM, O_3	430	4,800	14,000	0.55%
Minor Illness					
acute bronchitis	PM	0	47,000	94,000	5.06%
upper respiratory symptoms	PM	280,000	950,000	1,600,000	0.86%
lower respiratory symptoms	PM	240,000	520,000	770,000	3.57%
respiratory illness	NO_2	76,000	330,000	550,000	10.44%
moderate or worse asthma[c]	PM	80,000	400,000	720,000	0.24%
asthma attacks[c]	O_3, PM	920,000	1,700,000	2,500,000	1.04%
chest tightness, shortness of breath, or wheeze	SO_2	290	110,000	520,000	0.003%
shortness of breath	PM	26,000	91,000	150,000	1.69%
work loss days	PM	3,600,000	4,100,000	4,600,000	0.94%
minor restricted activity days / any of 19 respiratory symptoms[d]	O_3, PM	25,000,000	31,000,000	37,000,000	2.15%
restricted activity days[c]	PM	10,000,000	12,000,000	13,000,000	1.00%

[a] The baseline incidence generally is the same as that used in the C-R function for a particular health effect. However, there are a few exceptions. To calculate the baseline incidence rate for respiratory-related hospital admissions, we used admissions for persons of all ages for International Classification of Disease (ICD) codes 460-519; for cardiovascular admissions, we used admissions for persons of all ages for ICD codes 390-429; for emergency room visits for asthma, we used the estimated ER visit rate for persons of all ages; for chronic bronchitis we used the incidence rate for individuals 27 and older; for the pooled estimate of minor restricted activity days and any-of-19 respiratory symptoms, we used the incidence rate for minor restricted activity days.

[b] Percentage is calculated as the ratio of avoided mortality to the projected baseline annual non-accidental mortality for adults aged 30 and over. Non-accidental mortality was approximately 95% of total mortality for this subpopulation in 2010.

[c] These health endpoints overlap with the "any-of-19 respiratory symptoms" category. As a result, although we present estimates for each endpoint individually, these results are not aggregated into the total benefits estimates.

[d] Minor restricted activity days and any-of-19 respiratory symptoms have overlapping definitions and are pooled.

Non-Fatal Health Impacts

We report non-fatal health effects estimates in a similar manner to estimates of premature mortalities: as a range of estimates for each quantified health endpoint, with the range dependent on the quantified uncertainties in the underlying concentration-response functions. The range of results for 2010 only is characterized in Table 5-3 with 5th percentile, mean, and 95th percentile estimates which correspond to the Primary Low, Primary Central, and Primary High estimates, respectively. All estimates are expressed as new cases avoided in 2010, with the following exceptions. Hospital admissions reflect admissions for a range of respiratory and cardiovascular diseases, and these results, along with emergency room visits for asthma, do not necessarily represent the avoidance of new cases of disease (i.e., air pollution may simply exacerbate an existing condition, resulting in an emergency room visit or hospital admission). Further, each admission is only counted once, regardless of the length of stay in the hospital. "Shortness of breath" is expressed in terms of symptom days: that is, one "case" represents one child experiencing shortness of breath for one day. Likewise, "Restricted Activity Days" and "Work Loss Days" are expressed in person-days.

Avoided Health Effects of Other Pollutants

This section discusses the health effects associated with non-criteria air pollutants regulated by the Clean Air Act Amendments of 1990. It first discusses the effects of pollutants known as "air toxics", and then summarizes the effects associated with stratospheric ozone depleting substances.

Avoided Effects of Air Toxics

In addition to addressing the control of criteria pollutants, the Clean Air Act Amendments revamped regulations for air toxics — defined as non-criteria pollutants which can cause adverse effects to human health and to ecological resources — under section 112 of the Act. Among other changes, the 1990 Amendments establish a list of air toxics to be regulated, require EPA to establish air toxic emissions standards based on maximum achievable control technology (MACT standards), and include a provision that requires EPA to establish more stringent air toxic standards if MACT controls do not sufficiently protect the public health against residual risks. Control of air toxics is expected to result both from these changes and from incidental control due to changes in criteria pollutant programs.

Table 5-4
Mortality Distribution by Age in Primary Analysis (2010 only), Based on Pope et al. (1995)[a]

Age Group	Proportion of Premature Mortality by Age [b]	Life Expectancy (years)
Infants	not estimated	--
1-29	not estimated	--
30-34	1%	48
35-44	4%	38
45-54	6%	29
55-64	12%	21
65-74	24%	14
75-84	30%	9
85+	24%	6

[a] Results based on PM-related mortality incidence estimates for the 48 state U.S. population.

[b] Percentages may not sum to 100 percent due to rounding.

For several decades, the primary focus of risk assessments and control programs designed to reduce air toxics has been cancer. According to present EPA criteria, over 100 air toxics are known or suspected carcinogens. EPA's 1990 Cancer Risk study indicated that as many as 1,000 to 3,000 cancers annually may be attributable to the air toxics for which assessments were available (virtually all of this estimate came from assessments of about a dozen well-studied pollutants).[12] We note, however, that the results of this analysis are based, in part, on conservative, upper-bound estimates of chemical specific risk factors.

In addition to cancer, inhalation of air toxics compounds can cause a wide variety of health effects, including neurotoxicity, respiratory problems, and adverse reproductive and developmental effects. However, there has been considerably less work done to assess the magnitude of non-cancer effects from air toxics.

Air toxics can also cause adverse health effects via non-inhalation exposure routes. Persistent bioaccumulating pollutants, such as mercury and dioxins, can be deposited into water or soil and subsequently taken up by living organisms. The pollutants can biomagnify through the food chain and exist in high concentrations when consumed by humans in foods such as fish or beef. The resulting exposures can cause adverse effects in humans.

Finally, there are a host of other potential ecological and welfare effects associated with air toxics, for which very little exists in the way of quantitative analysis. Toxic effects of these pollutants have the potential to disrupt both terrestrial and aquatic ecosystems and contribute to adverse welfare effects such as fish consumption advisories in the Great Lakes.[13]

Unfortunately, the effects of air toxics emissions reductions could not be quantified for the present study. Unlike criteria pollutants, monitoring data for air toxics are relatively scarce, and the data that do exist cover only a handful of pollutants. Emissions inventories are very limited and inconsistent, and air quality modeling has only been performed for a few source categories. In addition, the scientific literature on the effects of air toxics is generally much weaker than that available for criteria pollutants. Appendix I presents a list of research needs identified by the Project Team which, if met, would enable at least a partial assessment of air toxics benefits in future section 812 prospective studies.

Avoided Health Effects for Provisions to Protect Stratospheric Ozone

We estimate benefits of stratospheric ozone protection programs by relying on analyses conducted to support a series of regulatory support documents for these provisions. The series of basic steps to arrive at physical effects estimates — from emissions estimation, atmospheric modeling, exposure assessment, and dose-response characterization — is similar to that used to estimate effects of criteria pollutants, but the details of each modeling step are vastly different. The emissions and atmospheric modeling yields estimates of changes in ultraviolet-b (UV-b) radiation, and the exposure and dose-response analyses then yield estimates of the effects of changes in UV-b radiation, including human health, welfare, and ecological effects. Appendix G provides a detailed description of the methodology and sources used to generate these estimates. Several of the benefits can be identified but cannot yet be reliably quantified, and so are described qualitatively.

The quantified physical effects estimates of sections 604 and 606 of Title VI, the provisions that provide the primary controls on production and release of CFCs and HCFCs generate about 98 percent of the monetized quantified benefits estimate. The quantified health benefits include the following: reduced incidences of mortality and morbidity associated with skin cancer (melanoma and nonmelanoma); and reduced incidences of cataracts

[12] These pollutants included PIC (products of incomplete combustion), 1,3-butadiene, hexavalent chromium, benzene, formaldehyde, chloroform, asbestos, arsenic, ethylene dibromide, dioxin, gasoline vapors, and ethylene dichloride. See U.S. EPA, Cancer Risk from Outdoor Exposure to Air Toxics. EPA-450/1-90-004f. Prepared by EPA/OAR/OAQPS.

[13] U.S. EPA, Office of Air Quality Planning and Standards. "Deposition of Air Pollutants to the Great Waters, First Report to Congress," May 1994. EPA-453/R-93-055.

and their associated pain and suffering.[14] Using the change in UV radiation dose, we estimate the number of additional cases of skin cancer (melanoma and nonmelanoma) and cataracts. Because the baseline levels of all of these UV-related health effects tend to be higher for older people and for those with lighter skins, EPA's method for projecting future incremental skin cancers and cataracts incorporates these factors in its benefits estimates.[15] We present a brief summary of these benefits in Table 5-5, and the analysis is described in detail in Appendix G.

To calculate the number of deaths from melanoma, the model uses a dose response function similar to the C-R functions for criteria pollutants. For nonmelanoma, the model estimates the number of deaths by assuming that a fixed percentage of the total nonmelanoma cases will result in death.[16] We estimate that from 1990 to 2165 sections 604 and 606 will result in 6.3 million avoided deaths from skin cancer, 27.5 million avoided cataract cases, and 299.0 million cases of non-fatal skin cancers (melanoma and nonmelanoma). The unquantified effects of sections 604 and 606 include avoided pain and suffering from skin cancer and human health and environmental benefits outside the United States.

Table 5-5
Major Health Benefits of Provisions to Protect Stratospheric Ozone
(CAAA Sections 604, 606, And 609)

Health Effects- Quantified	Estimate	Basis for Estimate
Melanoma and nonmelanoma skin cancer (fatal)	6.3 million lives saved from skin cancer in the U.S. between 1990 and 2165	Dose-response function based on UV exposure and demographics of exposed populations.[1]
Melanoma and nonmelanoma skin cancer (non-fatal)	299 million avoided cases of non-fatal skin cancers in the U.S. between 1990 and 2165	Dose-response function based on UV exposure and demographics of exposed populations.[1]
Cataracts	27.5 million avoided cases in the U.S. between 1990 and 2165	Dose-response function uses a multivariate logistic risk function based on demographic characteristics and medical history. [1]
Health Effects- Unquantified		
Skin cancer: reduced pain and suffering		
Reduced morbidity effects of increased UV. For example, • reduced actinic keratosis (pre-cancerous lesions resulting from excessive sun exposure) • reduced immune system suppression.		

Notes:
[1] For more detail see EPA's *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988).
[2] Note that the ecological effects, unlike the health effects, do not reflect the accelerated reduction and phaseout schedule of section 606.
[3] Benefits due to the section 606 methyl bromide phaseout are not included in the benefits total because annual incidence estimates are not currently available.

[14] Quantitative estimates presented in Appendix G also include reduced crop damage associated with UV-b radiation and tropospheric ozone; reduced damage to fish harvests associated with UV-b radiation; and reduced polymer degradation from UV-b radiation. The derivation of these effects is described in more detail in Chapter 7.

[15] The dose-response equation is (fractional change in incidence) = (fractional change in UV-b dose + 1)^b -1, where b (the biological amplification factor) equals the percent change in incidence associated with a one percent change in dose. More information about the origins of the models can be found in Appendix G.

[16] Scotto, Fears, and Fraumeni, U.S. Department of Health and Human Services, NIH, "Incidence of Nonmelanoma Skin Cancer in the United States," 1981, pages 2, 7, and 13.

Uncertainty in the Health Effects Analysis

As discussed above, and in greater detail in Appendix D, a number of important assumptions and uncertainties in the physical effects analysis may influence the estimate of monetary benefits presented in this study. Several of these key uncertainties, their potential directional bias (i.e., overestimation or underestimation), and the potential significance of each of these uncertainties for the overall net benefit results of the analysis are summarized in Table 5-6. As shown in this table, the decisions made to overcome the problems of limited data, the inadequacy of the currently available scientific literature, and other constraints do not clearly bias the overall results of this analysis in one particular direction.

Table 5-6
Key Uncertainties Associated with Human Health Effects Modeling (continued)

Potential Source of Error	Direction of Potential Bias for Net Benefits Estimate	Likely Significance Relative to Key Uncertainties in Net Benefit Estimate*
Application of C-R relationships only to those subpopulations matching the original study population.	Underestimate.	Potentially major. The C-R functions for several health endpoints (including PM-related premature mortality) were applied only to subgroups of the U.S. population (e.g., adults over 30) and thus may underestimate the whole population benefits of reductions in pollutant exposures. In addition, the demographics of the study population in the Pope et al. study (largely white and middle class) may result in an underestimate of PM-related mortality, because the effects of PM tend to be significantly greater among groups of lower socioeconomic status.
No quantification of health effects associated with exposure to air toxics.	Underestimate	Potentially major. According to EPA criteria, over 100 air toxics are known or suspected carcinogens, and many air toxics are also associated with adverse health effects such as neurotoxicity, reproductive toxicity, and developmental toxicity. Unfortunately, current data and methods are insufficient to develop (and value) quantitative estimates of the health effects of these pollutants.
Use of long-term global warming estimates in Title VI analysis that show more severe warming than is now generally anticipated.	Overestimate (for Title VI estimate only)	Potentially major. Global warming can accelerate the pace of stratospheric ozone recovery; if warming is less severe than anticipated at the time the Title VI analyses were conducted, the modeled pace of ozone recovery may be overestimated, suggesting benefits of the program could be delayed, perhaps by many years. The magnitude of estimated Title VI benefits suggests that the impact of delaying benefits could be major.
The quantitative analysis of Title VI (see next section) does not account for potential increases in averting behavior (i.e., people's efforts to protect themselves from UV-b radiation).	Unable to determine based on current information.	Potentially major. Murdoch and Thayer (1990) estimate that the cost-of-illness estimates for nonmelanoma skin cancer cases between 2000 and 2050 may be almost twice the estimated cost of averting behavior (application of sunscreen). Our Title VI analysis relies on epidemiological studies, which incorporate averting behavior as currently practiced. Omission of future increases in averting behavior, however, may overstate the benefits of reduced emissions of ozone-depleting chemicals. Benefits could be understated if individuals alter their behaviors in ways that could increase exposure or risk (e.g., sunbathing more frequently). A recent European study by Autier et al. (1999) found that the use of high sun protection factor (SPF) sun screen is associated with increased frequency and duration of sun exposure.

Table 5-6
Key Uncertainties Associated with Human Health Effects Modeling (continued)

Potential Source of Error	Direction of Potential Bias for Net Benefits Estimate	Likely Significance Relative to Key Uncertainties in Net Benefit Estimate*
Analysis assumes a causal relationship between PM exposure and premature mortality based on strong epidemiological evidence of a PM/mortality association. However, epidemiological evidence alone cannot establish this causal link.	Unable to determine based on current information.	Potentially major. A basic underpinning of this analysis, this assumption is critical to the estimation of health benefits. However, the assumption of causality is suggested by the epidemiologic evidence and is consistent with current practice in the development of a best estimate of air pollution-related health benefits. At this time, we can identify no basis to support a conclusion that such an assumption results in a known or suspected overestimation bias.
Across-study variance / application of regionally derived C-R estimates to entire U.S.	Unable to determine based on current information.	Potentially major. The differences in the expected changes in health effects calculated using different underlying studies can be large. If differences reflect real regional variation in the PM/mortality relationship, applying individual C-R functions throughout the U.S. could result in considerable uncertainty in health effect estimates.
Estimate of non-melanoma skin cancer mortality resulting from reductions in stratospheric ozone is calculated indirectly, by assuming the mortality rate is a fixed percentage of non-melanoma incidence.	Unable to determine based on current information.	Potentially major. New data on the death rate for non-melanoma skin cancer may significantly influence the Title VI mortality estimate. Some preliminary estimates suggest that this estimate may need to be adjusted downward.
The baseline incidence estimate of chronic bronchitis based on Abbey et al. (1995) excluded 47 percent of the cases reported in that study because those reported "cases" experienced a reversal of symptoms during the study period. These "reversals" may constitute acute bronchitis cases that are not included in the acute bronchitis analysis (based on Dockery et al., 1996).	Underestimate.	Probably minor. The relative contribution of acute bronchitis cases to the overall benefits estimate is small compared to other health benefits such as avoided mortality and avoided chronic bronchitis.
CAAA fugitive dust controls implemented in PM non-attainment areas would reduce lead exposures by reducing the re-entrainment of lead particles emitted prior to 1990. This analysis does not estimate these benefits.	Underestimate	Probably minor. While the health and economic benefits of reducing lead exposure can be substantial (e.g., see section 812 Retrospective Study Report to Congress), most additional fugitive dust controls implemented under the Post-CAAA scenario (e.g., unpaved road dust suppression, agricultural tilling controls, etc) tend to be applied in relatively low population areas.
Exclusion of C-R functions from short-term exposure studies in PM mortality calculations.	Underestimate	Probably minor. Long-term PM exposure studies may be able to capture some of the impact of short-term peak exposure on mortality; however the extent of overlap between the two study types is unclear.

Table 5-6
Key Uncertainties Associated with Human Health Effects Modeling (continued)

Potential Source of Error	Direction of Potential Bias for Net Benefits Estimate	Likely Significance Relative to Key Uncertainties in Net Benefit Estimate*
Age-specific C-R functions for PM related premature mortality not reported by Pope et al. (1995). Estimation of the degree of life-shortening associated with PM-related mortality used a single C-R function for all applicable age groups.	Unable to determine based on current information.	Unknown, possibly major when using a value of life years approach. Varying the estimate of degree of prematurity has no effect on the aggregate benefit estimate when a value of statistical life approach is used, since all incidences of premature mortality are valued equally. Under the alternative approach based on valuing individual life-years, the influence of alternative values for numbers of average life-years lost may be significant.
Assumption that PM-related mortality occurs over a period of five-years following the critical PM exposure. Analysis assumes that 25 percent of deaths occur in year one, 25 percent in year two, and 16.7 percent in each of the remaining three years.	Unable to determine based on current information.	Probably minor. If the analysis underestimates the lag period, benefits will be overestimated, and vice-versa. However, available epidemiological studies do not provide evidence of the existence or potential magnitude of a lag between exposure and incidence. Thus, an underestimate of the lag seems unlikely. If the assumed lag structure is an overestimate, even if benefits are fully discounted from the future year of death, application of reasonable discount rates over this period would not significantly alter the monetized benefit estimate.
Extrapolation of criteria pollutant concentrations to populations distant from monitors.	Unable to determine based on current information.	Probably minor. Extrapolation method is most accurate in areas where monitor density is high. Monitor density tends to be highest in areas with high criteria pollutant exposures; thus most of this uncertainty affects low exposure areas where benefits are likely to be low. In addition, an enhanced extrapolation method incorporating modeling results is used for areas far (> 50 km) from a monitor.
Exposure analysis in areas beyond 50 km is based on a new technique that relies on the direct use of air quality modeling results in combination with adjusted monitor data.	Unable to determine based on current information.	Probably minor. The new technique is used for less than 10 percent of the country for PM exposure, and less than 15 percent for ozone. The approach we use should be more accurate than the alternative approach of linear interpolation over long distances. The new method nonetheless requires further testing against monitor data to access its accuracy.
Pope et al. (1995) study did not include pollutants other than PM.	Unable to determine based on current information.	Probably minor. If ozone and other criteria pollutants correlated with PM contribute to mortality, that effect may be captured in the PM estimate. Thus, PM is essentially used as a surrogate for a mix of pollutants. This uncertainty does make it difficult to disaggregate avoided mortality benefits by pollutant, however other studies (besides Pope) suggest that PM is the dominant factor in premature mortality.

* The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

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Economic Valuation of Human Health Effects

Chapter 6

The reduced incidence of physical effects is a valuable measure of health benefits for individual endpoints; however, to compare or aggregate benefits across endpoints, the benefits must be monetized. Assigning a dollar value to avoided incidences of each effect permits us to sum monetized benefits realized as a result of the CAAA, and compare them with the associated costs.

In the 812 prospective analysis, we have two broad categories of benefits, health and welfare benefits. Human health effects include mortality and morbidity endpoints, which are presented in this chapter. Welfare effects include agricultural and ecological benefits, visibility, and worker productivity, which are covered in the following chapter. We obtain valuation estimates from the economic literature, and report them in "dollars per case reduced for health effects" and "dollars per unit of avoided damage for welfare effects".[1] Similar to estimates of physical effects provided by health studies, we report each of the monetary values of benefits applied in this analysis in terms of a central estimate and a probability distribution around that value. The statistical form of the probability distribution varies by endpoint. For example, we use a Weibull distribution to describe the estimated dollar value of an avoided premature mortality, while we assume the estimate for the value of a reduced case of acute bronchitis is uniformly distributed between a minimum and maximum value.

Although human health benefits of the 1990 Amendments are attributed to reduced emissions of criteria pollutants (Titles I through V) and reduced emission of ozone depleting substances (Title VI), this chapter focuses only on the valuation of human health effects attributed to the reduction of criteria pollutants. The chapter begins with an brief review of the economic concepts behind valuing human health effects in a cost-benefit context and a summary of the unit values applied to health endpoints. We follow with a discussion of how we derive valuation estimates for specific health effects. We then present the results of this analysis. We conclude the chapter with a review of the uncertainties associated with benefits valuation.

Our analysis indicates that the benefit of avoided premature mortality risk reduction dominates the overall net benefit estimate. This is, in part, due to the high monetary value assigned to the avoidance of premature mortality relative to the unit value of other health endpoints. Because of the critical importance of this endpoint in the study's results, this chapter pays particular attention to the major challenges to valuing mortality risk reductions and the limitations of the estimate we apply in this analysis. There are also significant reductions in short term and chronic health effects and a substantial number of health (and welfare) benefits that we could not quantify or monetize.

Valuation of Benefit Estimates

In an environmental benefit-cost analysis, the dollar value of an environmental benefit (e.g., a health-related improvement due to environmental quality) enjoyed by an individual is the dollar amount such that the person would be indifferent between experiencing the benefit and possessing the money. In general, the dollar amount required to compensate a person for exposure to an adverse effect is roughly the same as the dollar amount a person is willing to pay to avoid the effect. Thus, economists speak of "willingness-to-pay" (WTP) as the appropriate measure of the value of avoiding an adverse

[1] The literature reviews and process for developing valuation estimates are described in detail in Appendix I and in referenced supporting reports.

effect. For example, the value of an avoided respiratory symptom would be a person's WTP to avoid that symptom.

For most 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 that are not exchanged in the market, such as most environmental "goods," valuation is not so straightforward. Nevertheless, a value may be inferred from observed behavior, such as through estimation of the WTP for mortality risk reductions based on observed 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.

Wherever possible in this analysis, we use estimates of mean WTP. In cases where WTP estimates are not available, we use the cost of treating or mitigating the effect as an alternative 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 avoiding a health effect. They tend to reflect the direct expenditures related to treatment and not the utility an individual derives from improved health status or avoided health effect. As noted above, we use a range of values for most environmental effects, to support the primary central estimate of net benefits. Table 6-1 summaries the mean unit value estimates that we use in this analysis. We present the full range of values in Appendix H, including those used to derive the primary low and primary high estimates, as well as values used to generate an alternative value for avoiding premature mortality.

Valuation of Premature Mortality

Some forms of air pollution increase the probability that individuals will die prematurely. We use concentration-response functions for mortality that express the increase in mortality risk as cases of "ex-

Table 6-1
Health Effects Unit Valuation (1990 dollars)

Endpoint	Pollutant	Valuation (mean est.)	
Mortality	PM_{10}	$4,800,000	per case
Chronic Bronchitis	PM_{10}	$260,000	per case
Chronic Asthma	O_3	$25,000	per case
Hospital Admissions			
All Respiratory	SO_2, NO_2, PM_{10} & O_3	$6,900	per case
All Cardiovasular	SO_2, NO_2, & CO PM_{10} & O_3	$9,500	per case
Emergency Room Visits for Asthma	PM_{10} & O_3	$194	per case
Respiratory Illness and Symptoms			
Acute Bronchitis	PM_{10}	$45	per case
Asthma Attack or Moderate or Worse Asthma Day	PM_{10} & O_3	$32	per case
Acute Respiratory Symptoms	SO_2, NO_2, PM_1, & O_3	$18	per case
Upper Respiratory Symptoms	PM_1	$19	per case
Lower Respiratory Symptoms	PM_{10}	$12	per case
Shortness of Breath, Chest Tightness, or Wheeze	PM_{10} & SO_2	$5.30	per day
Work Loss Days	PM_{10}	$83	per day
Mild Restricted Activity Days	PM_{10} & O_3	$38	per day

cess premature mortality" per time period (e.g., per year). The benefit, however, is the avoidance of small increases in the risk of mortality. By summing individuals' WTP to avoid small increases in risk over enough individuals, we can infer the value of a statistical premature death avoided.[2] For expository purposes, we express this valuation as "dollars per mortality avoided," or "value of a statistical life" (VSL), even though the actual valuation is of small changes in mortality risk experience by a large number of people. The economic benefits associated with avoiding premature mortality were the largest category of monetized benefits in the section 812 CAA retrospective analysis (U.S. EPA 1997) and continue to be the largest source of monetized benefits for this prospective analysis. Mortality benefits, however, are also the largest contributor to the range of uncertainty in monetized benefits. For a more detailed discussion of the factors affecting the valuation of premature mortality see Appendix H.

The health science literature on air pollution indicates that several human characteristics affect the degree to which mortality risk affects an individual. For example, some age groups appear to be more susceptible to air pollution than others (e.g., the elderly and children). Health status prior to exposure also affects susceptibility. At risk individuals include those who have suffered strokes or are suffering from cardiovascular disease and angina (Rowlatt, et al. 1998). An ideal economic benefits estimate of mortality risk reduction would reflect these human characteristics, in addition to an individual's willingness to pay (WTP) to improve one's own chances of survival plus WTP to improve other individuals' survival rates.[3] The ideal measure would also take into account the specific nature of the risk reduction commodity that is provided to individuals, as well as the context in which risk is reduced. To measure this value, it is important to assess how reductions in air pollution reduce the risk of dying from the time that reductions take effect onward, and how individuals value these changes. Each individual's survival curve, or the probability of surviving beyond a given age, should shift as a result of an environmental quality improvement. For example, changing the current probability of survival for an individual also shifts future probabilities of that individual's survival. This probability shift will differ across individuals because survival curves are dependent on such characteristics as age, health state, and the current age to which the individual is likely to survive

Although a survival curve approach provides a theoretically preferred method for valuing the economic benefits of reduced risk of premature mortality associated with reducing air pollution, the approach requires a great deal of data to implement. The economic valuation literature does not yet include good estimates of the value of this risk reduction commodity. As a result, in this study we value avoided premature mortality risk using the value of statistical life approach, supplemented by an alternative valuation based on a value of statistical life years lost approach. We provide a review of the relevant literature and a more detailed discussion of our selected approach in Appendix H.

As in the retrospective, we use a mortality risk valuation estimate which is based on an analysis of 26 policy-relevant value-of-life studies (see Table 6-2). 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. We used the best estimate from each of the 26 studies to construct a distribution of mortality risk valuation estimates for the section 812 study. A Weibull distribution, with a mean of $4.8 million and standard deviation of $3.24 million, provided the best fit to the 26 estimates. There is considerable uncertainty associated with this approach. We discuss this issue in detail later in this chapter and in Appendix H.

In addition, we developed alternative calculations based on a life-years lost approach. To employ the value of statistical life-year (VSLY) approach, we first estimated the age distribution of those lives projected to be saved by reducing air pollution. Based on life expectancy tables, we calculate the life-years saved

[2] Because people are valuing small decreases in the risk of premature mortality, it is expected deaths that are inferred. For example, suppose that a given reduction in pollution confers on each exposed individual a decrease in mortal risk of 1/100,000. Then among 100,000 such individuals, one fewer individual can be expected to die prematurely . If each individual's WTP for that risk reduction is $50, then the implied value of a statistical premature death avoided is $50 x 100,000 = $5 million.

[3] For a more detailed discussion of altruistic values related to the value of life, see Jones-Lee (1992).

Table 6-2
Summary of Mortality Valuation Estimates (millions of $1990)

Study	Type of Estimate	Valuation (millions 1990$)
Kneisner and Leeth (1991) (US)	Labor Market	0.6
Smith and Gilbert (1984)	Labor Market	0.7
Dillingham (1985)	Labor Market	0.9
Butler (1983)	Labor Market	1.1
Miller and Guria (1991)	Cont. Value	1.2
Moore and Viscusi (1988a)	Labor Market	2.5
Viscusi, Magat, and Huber (1991b)	Cont. Value	2.7
Gegax et al. (1985)	Cont. Value	3.3
Marin and Psacharopoulos (1982)	Labor Market	2.8
Kneisner and Leeth (1991) (Australia)	Labor Market	3.3
Gerking, de Haan, and Schulze (1988)	Cont. Value	3.4
Cousineau, Lacroix, and Girard (1988)	Labor Market	3.6
Jones-Lee (1989)	Cont. Value	3.8
Dillingham (1985)	Labor Market	3.9
Viscusi (1978, 1979)	Labor Market	4.1
R.S. Smith (1976)	Labor Market	4.6
V.K. Smith (1976)	Labor Market	4.7
Olson (1981)	Labor Market	5.2
Viscusi (1981)	Labor Market	6.5
R.S. Smith (1974)	Labor Market	7.2
Moore and Viscusi (1988a)	Labor Market	7.3
Kneisner and Leeth (1991) (Japan)	Labor Market	7.6
Herzog and Schlottman (1987)	Labor Market	9.1
Leigh and Folson (1984)	Labor Market	9.7
Leigh (1987)	Labor Market	10.4
Garen (1988)	Labor Market	13.5

Source: Viscusi, 1992 and EPA analysis.

from each statistical life saved within each age and gender cohort. To value these statistical life-years, we hypothesized a conceptual model which depicted the relationship between the value of life and the value of life-years. As noted in Chapter 5, the average number of life-years saved across all age groups for which data were available is 14 for PM-related mortality. The average for PM, in particular, differs from the 35-year expected remaining lifespan derived from existing wage-risk studies.[4] Using the same distribution of value of life estimates used above (i.e. the Weibull distribution with a mean estimate of $4.8 million), we estimated a distribution for the value of a life-year and combined it with the total number of estimated life-years lost. The details of these calculations are presented in Appendix H.

Valuation of Specific Health Effects

Chronic Bronchitis

The best available estimate of WTP to avoid a case of chronic bronchitis (CB) comes from Viscusi et al. (1991). The Viscusi study, however, describes to the respondents a severe case of CB. We employ an estimate of WTP to avoid a pollution-related case of CB that is based on adjusting the WTP to avoid a severe case, as estimated by Viscusi et al. (1991), to account for the likelihood that an average case of pollution-related CB is not as severe.

We use the mean of a distribution of WTP estimates as the central tendency estimate of WTP to avoid a pollution-related case of chronic bronchitis 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., 1991; (2) the severity level of an average pollution-related case of CB (relative to that of the case described by Viscusi et al., 1991); 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.[5] The expected value of this distribution,

[4] See, for example, Moore and Viscusi (1988) or Viscusi (1992).

[5] The statistical uncertainty analysis technique we used, which is also known as simulation modeling, is a probabilistic approach to characterizing the uncertainty or the distribution of potential values around a central estimate.

which is about $260,000, is taken as the central tendency estimate of WTP to avoid a pollution-related case of CB. We describe the three underlying distributions, and the generation of the resulting distribution of WTP, in Appendix H.

Chronic Asthma

The valuation of this health endpoint requires an estimate which reflects an individual's desire to avoid the effects of chronic asthma throughout his or her lifetime. We derive this valuation estimate from two studies that solicit values from individuals diagnosed as asthmatics. Blumenschein and Johannesson (1998) generate an estimate of monthly WTP, while O'Conor and Blomquist (1997) generate an annual WTP estimate. In order to extend monthly and annual WTP estimates over an individual's lifetime, we adjusted the reported estimates to reflect the average life-years remaining and age distribution of the adult U.S. population, given that chronic asthma is not expected to affect the average life expectancy. The mean estimate of WTP to avoid a case of chronic asthma resulting from this method is approximately $25,000.

Respiratory-Related Ailments

In general, the values we assign to the respiratory-related ailments in Table 6-1 are a combination of WTP estimates for individual symptoms comprising each ailment. For example, a willingness to pay estimate to avoid the combination of specific upper respiratory symptoms defined in the concentration-response relationship measured by Pope et al. (1991) is not available. While that study defines upper respiratory symptoms as one suite of ailments (runny or stuffy nose; wet cough; and burning, aching, or red eyes), the valuation literature reports individual WTP estimates for three closely matching symptoms (head/sinus congestion, cough, and eye irritation). We therefore use these available WTP estimates and a benefits transfer procedure to estimate the value of avoiding those symptoms defined in the concentration-response study.

To capture the uncertainty associated with the valuation of respiratory-related ailments, we incorporated a range of values reflecting the fact that an ailment, as defined in the concentration-response relationship, could be comprised of just one symptom or several. At the high end of the range, the valuation represents an aggregate of WTP estimates for several individual symptoms. The low end represents the value of avoiding a single mild symptom.

Minor Restricted Activity Days

An individual suffering from a single severe pollution-related symptom or combination of symptoms may experience a Minor Restricted Activity Day (MRAD). Krupnick and Kopp (1988) argue that mild symptoms will not be sufficient to result in a MRAD, so that WTP to avoid a MRAD should exceed WTP to avoid any single mild symptom. On the other hand, WTP to avoid a MRAD should not exceed the WTP to avoid a work loss day (which results when the individual experiences more severe symptoms). No studies report an estimate for WTP to avoid a day of minor restricted activity. Therefore, we derive for this analysis a value from WTP estimates for avoiding combinations of symptoms which may result in a day of minor restricted activity ($38 per day). The uncertainty range associated with this value extends from the highest value for a single symptom to the value for a work loss day. Furthermore, a distributional form is used which reflects our expectations that the actual value is likely to be closer to the central estimate than either extreme.

Hospital Admissions, Cardiovascular and Respiratory

The valuation of this benefits category reflects the value of reduced incidences of hospital admissions due to respiratory or cardiovascular conditions. We use avoided hospital admissions as a measure as opposed to the number of avoided cases of respiratory or cardiovascular conditions, because of the availability of C-R relationships for the hospital admissions endpoint. Hospital admissions reflect a class of health effects linked to air pollution which are acute in nature but more severe than the symptom-day measures discussed above.

As described in Chapter 5, our approach to estimating the number of incidences for this category involves reliance on several concentration-response (C-R) functions. Each concentration response func-

tion provides an alternative definition of either respiratory effects or cardiovascular effects, and may be based on different pollutants. For valuation of the incidences, 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, developed by Elixhauser (1993), we construct cost of illness (COI) estimates that are specific to the suite of health effects defined by each C-R function. For example, we use twelve distinct C-R functions to quantify the expected change in respiratory admissions.[6] Consequently in this analysis, we develop twelve separate COI estimates, each reflecting the unique composition of health effects considered in the individual studies.

The use of COI estimates suggests we likely understand the WTP to avoid these effects. The valuation of any given health effect, such as hospitalization, should reflect the value of avoiding associated pain and suffering and lost leisure time, in addition to medical costs and lost work time. While the probability distributions in this analysis characterize a range of potential costs associated with hospitalization, they do not account for the omission of factors from the COI estimates such as pain and suffering. Consequently, the valuations for these endpoints most likely understate the true social values for avoiding hospital admissions due to respiratory or cardiovascular conditions.

Stratospheric Ozone Provisions

We develop monetary estimates of the health benefits due to stratospheric ozone provisions based on estimated incidences presented in a series of existing regulatory support analyses. To ensure consistency with the valuation strategy of this analysis, however, we adjust certain parameters used in the existing regulatory analyses of Title VI provisions. Specifically, we re-evaluate the physical effects change projected in the RIAs using the discount rate and the value of statistical life adopted throughout the rest of our present study. The net effect of these changes is to reduce the estimates of benefits from those found in the regulatory source support documents. The most important change is the discount rate. Because the benefits of stratospheric ozone protection accrue over several hundred years, the discount rate chosen can have an especially large influence on the benefits estimate. The central estimate employed in this analysis is five percent; the rate used in the source documents is two percent.

The value of statistical life (VSL) estimate is also an important factor in the calculations, because the vast majority of benefits of stratospheric ozone protection result from avoided fatal skin cancer cases. To reflect the uncertainty of the VSL estimates, we employ the same statistical uncertainty aggregation approach used in the criteria pollutant analysis, using a Weibull distribution of VSL estimates as an input. Appendix G describes the details of these and other changes made to ensure consistency between our stratospheric ozone provision benefits analysis and our criteria pollutant analyses.

Results of Benefits Valuation

We combine the number of reduced incidences of our health endpoints with our estimated values of avoiding the health effect to generate total annual monetized human health benefits in 2000 and 2010. We attribute to Titles I through V of the CAAA total annual human health benefits of $68 billion in 2000 and $110 billion in 2010. We summarize the Post-CAAA 2010 monetized benefit in Table 6-3. The table provides our central estimate, in addition to the 5th and 95th percentile estimates for each benefit category.

There are two aspects of our results that warrant discussion. The first is the valuation of premature mortality due to PM exposure. The second is our strategy to avoid double-counting when aggregating health benefits. As discussed in Chapter 5, premature mortality is attributed to PM exposure and our primary estimate reflects a lag between PM exposure and premature mortality. While this lag does not alter the number of estimated incidences, it does alter the monetization of benefits. Because we value the "event" rather than the present risk, in this analysis we assume that the value of avoided future premature mortality should be discounted. Therefore, the type of lag structure employed plays a direct role in the valuation of this endpoint.

[6] For more detailed discussion of the various health effects considered by each C-R function and methodology for estimating the number of avoided hospital admissions, see Appendix D.

Table 6-3
Results of Human Health Benefits Valuation, 2010

	Monetary Benefits (in millions 1990$)		
	5th %ile	Mean	95th %ile
Mortality			
Ages 30+	$ 14,000	$ 100,000	$ 250,000
Chronic Illness			
Chronic Bronchitis	$ 360	$ 5,600	$ 18,000
Chronic Asthma	40	180	300
Hospitalization			
All Respiratory	$ 75	$ 130	$ 200
Total Cardiovascular	93	390	960
Asthma-Related ER Visits	0.1	1	3
Minor Illness			
Acute Bronchitis	$ 0	$ 2	$ 5
URS	4	19	39
LRS	2	6	12
Respiratory Illness	1	6	15
Mod/Worse Asthma[1]	2	13	29
Asthma Attacks[1]	20	55	100
Chest tightness, Shortness of Breath, or Wheeze	0	0.6	3
Shortness of Breath	0	0.5	1.2
Work Loss Days	300	340	380
MRAD/Any-of-19	680	1,200	1,800
Total Benefits in 2010[2]	-	$ 110,000	-

Note:

[1] Moderate to worse asthma and asthma attacks are endpoints included in the definition of MRAD/Any-of-19 respiratory effects. Although valuation estimates are presented for these categories, the values are not included in total benefits to avoid the potential for double-counting.

[2] Summing 5th and 95th percentile values would yield a misleading estimate of the 5th and 95th percentile estimate of total health benefits. For example, the likelihood that the 5th percentile estimates for each endpoint would simultaneously be drawn during the statistical uncertainty analysis is much less than 5 percent. As a result, we present only the total mean.

The primary analysis reflects a five-year lag structure. Under this scenario, 50 percent of the estimated cases of avoided mortality occur within the first two years. The remaining 50 percent are then distributed across the next three years. Our valuation of avoided premature mortality applies a five percent discount rate to the lagged estimates over the periods 2000 to 2005 and 2010 to 2015. We discount over the period between the initial PM exposure change (either 2000 or 2010) and timing of the incidence.

Many of the monetized health benefit categories include overlapping health endpoints, creating the potential for double-counting. In an effort to avoid overstating the benefits, we do not aggregate all of the quantified health effects. For example, asthma attacks and moderate to worse asthma are considered components of the endpoint, "Any of 19 Respiratory Symptoms". Consequently, we present the results but do not include them in our reported total benefits figures. In other cases, there are endpoints included in our aggregation of benefit that appear to have overlapping health effects. For those benefit categories that describe similar health effects, it is important to keep in mind that estimated incidences are based on unique portions of the population.

Valuation Uncertainties

We addressed many valuation uncertainties explicitly and quantitatively by expressing values as distributions (see Appendix H for a complete description of distributions employed), using a computerized statistical technique to apply the valuations to physical effects (see Chapters 5 and 8) with the mean of each valuation distribution providing the foundation for the primary central estimate of total net benefits. This approach does not, of course, guarantee that all uncertainties have been adequately characterized, nor that the valuation estimates are unbiased. It is possible that the actual WTP to avoid an air pollution-related impact is outside of the range of estimates used in this analysis. Nevertheless, we assume that the distributions employed are reasonable approximations of the ranges of uncertainty, and that there is no compelling reason to believe that the mean values employed are systematically biased (except for the cost of illness values, which probably underestimate WTP). There are, however, a limited number of health endpoints

for which a different valuation approach may yield results significantly different from out primary central benefit estimate. For example, using a value of statistical life year approach in lieu of the value of statistical life method for valuing avoided premature mortality yields a mean estimate for this benefit which is approximately 45 percent lower than our primary central estimate. For those few endpoints where reasonable alternative valuation paradigms yield significantly different results from our preferred approach, see our discussion in Chapter 8.

The potential for biases as introduced by benefits transfer methodology is applicable to all benefits categories and, as noted in Table 6-4, the direction of its bias is unknown. Because changes in mortality risk are the single most important component of aggregate benefits, mortality risk valuation is also the dominant component of the quantified uncertainty. This category accounts for over 90 percent of total annual estimates under the Post-CAAA scenario. The second largest benefits category, reduced risk of chronic bronchitis, valued at approximately $5.6 billion per year in 2010, accounts for roughly five percent of the total estimated benefits. Consequently, any uncertainty concerning mortality risk valuation beyond that addressed by the quantitative uncertainty assessment (i.e., that related to the Weibull distribution with a mean value of $4.8 million) deserves note.

Mortality Risk Benefits Transfer

One issue that merits special attention is the uncertainties and possible biases related to the "benefits transfer" from the 26 valuation source studies to valuation of reductions in PM-related mortality rates. Given the limitations of the current literature, we address this source of uncertainty qualitatively in this section. Although each of the mortality risk valuation source studies (see Table 6-2) estimate the average WTP for a given reduction in mortality risk, the degree of reduction in risk being valued varies across studies and is not necessarily the same as the degree of mortality risk reduction estimated in this analysis. The transferability of estimates of the value of a statistical life from the 26 studies to the section 812 benefit analysis rests on the assumption that, within a reasonable range, WTP for reductions in mortality risk is linear in risk reduction. For example, suppose a study estimates that the average WTP for a reduction in mortality risk of 1/100,000 is $50, but that the actual mortality risk reduction resulting from a given pollutant reduction is 1/10,000. If WTP for reductions in mortality risk is linear in risk reduction, then a WTP of $50 for a reduction of 1/100,000 implies a WTP of $500 for a risk reduction of 1/10,000 (which is ten times the risk reduction valued in the study). Under the assumption of linearity, the estimate of the value of a statistical life does not depend on the particular amount of risk reduction being valued. This assumption has been shown to be reasonable provided the change in the risk being valued is within the range of risks evaluated in the underlying studies (Rowlatt et al. 1998).

Although the particular amount of mortality risk reduction being valued in a study may not affect the transferability of the WTP estimate from the study

Table 6-4
Valuation of CAAA Benefits: Potential Sources and Likely Direction of Bias

Benefits Category	Factor	Likely Direction of Bias in WTP Estimates Used in this Study
Premature Mortality	Age	Uncertain, perhaps overestimate
	Degree of Risk Aversion	Underestimate
	Income	Uncertain
	Voluntary vs. Involuntary	Underestimate
	Catastrophic vs. Protracted Death	Uncertain, perhaps underestimate
	Discounting over a latency period	Uncertain, perhaps underestimate
Chronic Bronchitis	Severity-level	Uncertain
	Elasticity of WTP with respect to severity	Uncertain
All other benefit endpoints	Benefits Transfer	Uncertain

to the benefit analysis, the characteristics of the study subjects and the nature of the mortality risk being valued in the study could be important. Certain characteristics of both the population affected and the mortality risk facing that population are believed to affect the average WTP to reduce risk. The appropriateness of the mean of the WTP estimates from the 26 studies for valuing the mortality-related benefits of reductions in pollutant concentrations therefore depends not only on the quality of the studies (i.e., how well they measure what they are trying to measure), but also on (1) the extent to which the subjects in the studies are similar to the population affected by changes in air pollution and (2) the extent to which the risks being valued are similar.

The substantial majority of the 26 studies relied upon are wage-risk (or labor market) studies. Compared with the subjects in these wage-risk studies, the population most affected by air pollution-related mortality risk changes is likely to be, on average, older and probably more risk averse. Some evidence suggests that approximately 85 percent of those identified in short-term ("episodic") studies who die prematurely from PM-related causes are over 65.[7] The average age of subjects in wage-risk studies, in contrast, would be well under 65, and probably closer to 40 years of age.

The direction of bias resulting from the age difference is unclear. We could argue that, because an older person has fewer expected years left to lose, his or her WTP to reduce mortality risk would be less than that of a younger person. This hypothesis is supported by one empirical study, Jones-Lee et al. (1985), which found WTP to avoid mortality risk at age 65 to be about 90 percent of what it is at age 40. On the other hand, there is reason to believe that those over 65 are, in general, more risk averse than the general population. This would imply that older populations are likely to select occupations that are relatively less risky than workers represented in wage-risk studies or the general population. Although the list of 26 studies used here excludes studies that consider only much-higher-than-average occupational risks, there is nevertheless likely to be some selection bias in the remaining studies, because these studies are likely to be based on samples of workers who are, on average, more risk-loving than the general population. In contrast, older people as a group exhibit more risk-averse behavior.

There is substantial evidence that the income elasticity of WTP for health risk reductions is positive (although there is uncertainty about the exact value of this elasticity). This implies that individuals with higher incomes and/or greater wealth should be willing to pay more to reduce risk, all else equal, than individuals with lower incomes or wealth. The comparison between the income, both actual and potential, or wealth of the workers in the wage-risk studies versus that of the population of individuals most likely to be affected by changes in pollution concentrations, however, is unclear. One could argue that because the elderly are relatively wealthy, the affected population is also wealthier, on average, than are the wage-risk study subjects, who tend to be middle-aged (on average) blue-collar workers. On the other hand, the workers in the wage-risk studies will have potentially more years remaining in which to acquire streams of income from future earnings. On net, the potential income comparison is unclear.

Although there may be several ways in which job-related mortality risks differ from air pollution-related mortality risks, the most important difference may be that job-related risks are incurred voluntarily, or generally assumed to be, whereas air pollution-related risks are incurred involuntarily. There is some evidence[8] that people will pay more to reduce involuntarily incurred risks than risks incurred voluntarily. If this is the case, WTP estimates based on wage-risk studies may understate WTP to reduce involuntarily incurred air pollution-related mortality risks.

Another important difference related to the nature of the risk may be that some workplace mortality risks tend to involve sudden, catastrophic events, whereas air pollution-related risks tend to involve longer periods of disease and suffering prior to death. Some evidence suggests that WTP to avoid a risk of a protracted death involving prolonged suffering and loss of dignity and personal control is greater than the WTP to avoid a risk (of identical magnitude) of sudden death. To the extent that the mortality risks

[7] See Schwartz and Dockery (1992), Ostro et al. (1995), and Chestnut (1995).

[8] See, for example, Violette and Chestnut, 1983.

addressed in this assessment are associated with longer periods of illness or greater pain and suffering than are the risks addressed in the valuation literature, the WTP measurements employed in the present analysis would reflect a downward bias.

Economic assessment of WTP for lagged mortality effects also introduces uncertainty. For lack of a more refined technique, our analysis relies on the simplifying assumption that lagged mortality risks can be valued at the time of the occurrence of death, rather than at the time of exposure. In subsequent development of the annual and present value estimates, we therefore discount the dollar benefits estimate as if the full benefit accrues only in the year of death. There are several reasons to believe that this approach underestimates willingness to pay. Most importantly, while death may occur after a lag period, morbidity effects may appear at any time prior to death, including immediately upon exposure. It is not clear that other dose-response assessments capture the full range of morbidity effects, direct and indirect, that might be associated with a latent fatal exposure. Other potentially important factors include the use of a financial discount rate, which may or may not accurately represent the rate at which individuals might discount delayed health benefits and the effect of knowledge of a fatal exposure on valuation of a delayed effect, in other words whether the valuation is affected by a prior diagnosis of a fatal condition.

We summarize the potential sources of bias introduced by relying on wage-risk studies to derive an estimate of the WTP to reduce air pollution-related mortality risk in Table 6-4; the overall effect of these multiple biases is addressed in Table 6-5. Among these potential biases, it is disparities in age and income between the subjects of the wage-risk studies and those affected by air pollution which have thus far motivated specific suggestions for quantitative adjustment;[9] however, the appropriateness and the proper magnitude of such potential adjustments remain unclear given presently available information. These uncertainties are particularly acute given the possibility that age and income biases might offset each other in the case of pollution-related mortality risk aversion. Furthermore, the other potential biases discussed above, and summarized in Table 6-4, add additional uncertainty regarding the transferability of WTP estimates from wage-risk studies to environmental policy and program assessments.

[9] Chestnut, 1995; IEc, 1992.

Table 6-5
Key Uncertainties Associated with Valuation of Health Benefits

Potential Source of Error	Direction of Potential Bias for Net Benefits	Likely Significance Relative to Key Uncertainties on Net Benefits Estimate[1]
Benefits transfer for mortality risk valuation, including differences in age, income, degree of risk aversion, the nature of the risk, and treatment of latency between mortality risks presented by PM and the risks evaluated in the available economic studies.	Unable to determine based on currently available information	Potentially major. The mortality valuation step is clearly a critical element in the net benefits estimate, so any uncertainties can have a large effect. As discussed in the text, however, information on the combined effect of these known biases is relatively sparse, and it is therefore difficult to assess the overall effect of multiple biases that work in opposite directions.
Benefits transfer for chronic bronchitis, including adjustments made to better match the severity of the risks modeled in the available economic studies.	Unable to determine based on currently available information	Probably minor. Benefits of avoided chronic bronchitis account for about five percent of total benefits, limiting the effect on net benefits to a maximum of about seven percent. Steps taken in the study to adjust for severity using the best available empirical information likely limit the effect to much less than this maximum value.
Inability to value some quantifiable morbidity endpoints, such as impaired lung function.	Underestimate	Probably minor. Reductions in lung function are a well-established effect, based on clinical evaluations of the impact of air pollutants on human health, and the effect would be pervasive, affecting virtually every exposed individual. There is therefore a potential for a major impact on benefits estimates. The lack of a clear symptomatic presentation of the effect, however, could limit individual WTP to avoid lung function decrements.

Note: [1] The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

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Ecological and Other Welfare Effects

Chapter 7

EPA's traditional focus in environmental benefits assessment has been on quantifying beneficial impacts of environmental regulation on human health. As we have learned more about the effects of anthropogenic stressors on ecological systems, however, pursuit of environmental programs targeted on reductions of damage to the environment have become more common. The CAAA Title IV provisions, collectively referred to as the Acid Rain Program, are a good example. These provisions are in place largely as the result of a major research effort to better understand and quantify the effects of sulfur and nitrogen oxides on natural systems susceptible to acid rain. Although the benefits of this program include improvements in human health, the initial impetus was protection of ecological resources.

We have designed this first section 812 prospective analysis to be responsive to the increased focus on the importance of ecological resources by devoting a great deal of effort to characterizing and, where possible, quantifying and monetizing the impacts of air pollutants on natural systems. This increased focus is also partly a result of the outcome of EPA's retrospective analysis, in which we identified an increased understanding of and focus on ecological effects as one of the important research directions for the first prospective and subsequent analyses. This chapter presents the results of these efforts.

This chapter consists of four sections. First, we provide an overview of our approach to estimating the effects of air pollution on ecological systems. Second, we provide a characterization of these effects in qualitative terms. The second section concludes with a summary of the process for selecting specific impacts which can be quantified and monetized using currently available methods. Third, we present the results of our quantitative and economic analyses. Finally, we discuss major uncertainties of the ecological and other welfare effects analyses.

Overview of Approach

Our analysis of ecological effects involves three major steps:

- First, we identify and characterize ecological effects from air pollution.
- Second, we develop and implement selection criteria for more in-depth assessment of ecological impacts.
- Third, we perform quantitative and qualitative analyses to characterize a portion of the benefits of the 1990 CAAA provisions.

The first step involves taking a broad view of pollutants controlled under the CAAA and their documented effects on ecological systems, both as individual pollutants and, to the extent possible, as one component in multiple-stressor effects on ecosystems and their components. We organize our analysis in terms of major pollutant classes and by the level of biological organization at which impacts are measured (e.g., regional ecosystem, local ecosystem, community, population, individual, etc.).

After completing the first step on a broad level, the second step involves narrowing the scope of subsequent analyses. While it is desirable to focus effort on those impacts that are of greatest importance, in practice the state of the science in ecological assessment largely dictates the subsequent focus of the analysis. There exist only a handful of comprehensive ecological assessments from which to draw conclusions about those effects that are most important either ecologically or in economic terms, and those studies are potentially controversial in their methods and conclusions, in part because of the incomplete understanding of many of these effects. As a result, the categories of effects ultimately chosen for assessment here are necessarily limited by available

methods and data. As scientific understanding and impact assessment methods grow more comprehensive, however, we expect that the focus of subsequent analyses will be on those effects whose avoidance would have the greatest potential ecological and/or economic value.

The third step involves implementing a wide range of analyses to more exhaustively characterize specific effects of air pollution on ecological systems. We provide quantitative estimates of the benefits of the 1990 CAAA for the following effects:

- eutrophication of estuaries associated with airborne nitrogen deposition;
- acidification of freshwater bodies associated with airborne nitrogen and sulfur deposition; and
- reduced forest growth associated with ozone exposure.

In addition, in this chapter we present the methods and results for quantitative analysis of other welfare effects, including reduced agricultural yields associated with ozone exposure, the impact of ambient particulate matter on visibility, the effects of ozone on farm worker productivity, and the effects of stratospheric ozone on crop and fisheries yields. These effects have been identified as important categories of benefits in many previous analyses, including the section 812 retrospective analysis. As a result, these effects were not considered in the same three step process used for other service flows.

We attempted to conduct quantitative analyses of two other benefits categories: the accumulation of toxics in freshwater fisheries associated with airborne toxics deposition; and aesthetic degradation of forests associated with ozone and airborne toxics exposure. However, we found that, while some quantitative methods exist to evaluate these benefits, key links are missing in the analytic process. This in turn prevents development of defensible benefits estimates which can be reasonably associated with the air quality and air pollutant deposition patterns developed from our Post-CAAA and Pre-CAAA scenarios. See Appendix E for more detailed discussion of these service flows. In addition, in assessing nitrogen deposition impacts to estuarine systems, we relied on a displaced cost approach with results that we chose to omit from the primary benefits estimate because of uncertainties in the methodology. These results are nonetheless reported in this chapter, but are used for the purposes of sensitivity testing only.

Because the breadth and complexity of air pollutant-ecosystem interactions do not allow for comprehensive quantitative analysis of all the ecological benefits of the CAAA, we stress the importance of continued consideration of those impacts not valued in this report in policy decision-making and in further technical research. Judging from the geographic breadth and magnitude of the relatively modest subset of impacts that we find sufficiently well-understood to quantify and monetize, it is apparent that the economic benefits of the CAAA's reduction of air pollution impacts on ecosystems are substantial.

Characterization of Impacts of Air Pollution on Ecological Systems

The purpose of this section is to provide an overview of potential interactions between air pollutants and the natural environment. We identify major single pollutant-environment interactions, as well as the synergistic impacts of ecosystem exposure to multiple air pollutants. Although a wide variety of complex air pollution-environment interactions are described or hypothesized in the literature, for the purposes of this analysis we focus on major aspects of ecosystem-pollutant interactions. We do this by limiting our review according to the following criteria:

- Pollutants regulated by the CAAA.
- Known interactions between pollutants and natural systems as documented in peer-reviewed literature.
- Pollutants present in the atmosphere in sufficient amounts after 1990 to cause significant damages to natural systems.

Our understanding of air pollution effects on ecosystems has progressed considerably during recent decades. Previously, air pollution was regarded primarily as a local phenomenon and concern was associated with the vicinity of industrial facilities,

power plants or urban areas. The pollutants of concern were gaseous (e.g., sulfur dioxide and ozone) or heavy metals (e.g., lead) and the observed effects were visible stress- specific symptoms of injury (e.g., foliar chlorosis). The most typical approach to documenting the effects of specific pollutants was a dose-response experiment, where the objective was to develop a regression equation describing the relationship between exposure and some easily measured effect (e.g., growth, yield or mortality). As analytic methods improved and ecology progressed, a broader range of effects of air pollutants was identified and understanding of the mechanisms of effect improved. Observations made on various temporal scales (e.g., long-term studies) and spatial scales (e.g., watershed studies) led to the recognition that air pollution can affect all organizational levels of biological systems.

Our current understanding of ecosystem impacts can be organized by the pollutants of concern and by the level of biological organization at which impacts are directly measured. We attempt to address both dimensions of categorization in this overview. In Table 7-1 we summarize the major pollutants of concern, and the documented acute and long-term ecological impacts associated with them.

The summary in Table 7-1 is a highly condensed version of the results of our characterization of ecological impacts. In addition to the pollutant-specific effects outlined in the table, it is important to identify the level of biological organization and types of ecosystems that are susceptible to these types of effects. Tables 7-2 through 7-4 provide more detail on pollutant-specific impacts at a range of levels of biological organization. It is important to note that the interactions listed are intended to illustrate the range of possible adverse effects. For a more complete review of air-pollutant-induced effects on ecosystems, see Appendix E.

Effects of Mercury and Ozone

Table 7-2 summarizes the effects of mercury and ozone on ecological systems. To illustrate the nature of our review of effects, consider the second row in Table 7-2. This row summarizes the effects of the air pollutants mercury and ozone at the "individual" level of biological organization. As indicated in the table, in a general sense air pollutants can induce a direct physiological response in individuals (analogous to that experienced by humans exposed to pollutants), or an indirect effect either through impacts on the individual's surroundings or by weakening the individual and making it more susceptible to other stressors. Mercury has several direct effects to fauna, including effects to the central nervous system and the liver, while the documented direct effects of ozone tend to be to a variety of plant functions. Indirect effects of mercury are not well understood, but the indirect effects of ozone may serve to compound the direct effects to plants by also making the plants more susceptible to drought or heat stress, for example. This type of cataloging of

Table 7-1
Classes of Pollutants and Ecological Effects

Pollutant Class	Major Pollutants and Precursor Emissions	Acute Effects	Long-term Effects
Acidic Deposition	Sulfuric acid, nitric acid Precursor emissions: Sulfur dioxide, nitrogen oxides	Direct toxic effects to plant leaves and aquatic organisms.	Progressive deterioration of soil quality. Chronic acidification of surface waters.
Nitrogen Deposition	Nitrogen compounds (e.g., nitrogen oxides)		Saturation of terrestrial ecosystems with nitrogen. Progressive nitrogen enrichment of coastal estuaries.
Hazardous Air Pollutants (HAPs)	Mercury, dioxins	Direct toxic effects to animals.	Conservation of mercury and dioxins in biogeochemical cycles and accumulation in the food chain.
Ozone	Tropospheric ozone Precursor emissions: Nitrogen Oxides and Volatile Organic Compounds (VOCs)	Direct toxic effects to plant leaves.	Alterations of ecosystem wide patterns of energy flow and nutrient cycling.

Table 7-2
Interactions of Mercury and Ozone with Natural Systems At Various Levels of Organization

		Examples of Interactions	
Spatial Scale	**Type of Interaction**	**Mercury in streams and lakes**	**Ozone**
Molecular and cellular	Chemical and biochemical processes	Mercury enters the body of vertebrates and binds to sulfhydril groups (i.e. proteins).	Oxidation of enzymes of plants. Disruption of the membrane potential.
Individual	Direct physiological response.	Neurological effects in vertebrates. Behavioral abnormalities. Damages to the liver.	Direct injuries include visible foliar damage, premature needle senescence, reduced photosynthesis, altered carbon allocation, and reduction of growth rates and reproductive success.
	Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress.	Few interactions known. Damages through increased sensitivity to other environmental stress factors could occur, for example, through impairment of immune response.	Increased sensitivity to biotic and abiotic stress factors like pathogens and frost. Disruption of plant-symbiont relationship (mychorrhiza), and symbionts.
Population	Change of population characteristics like productivity or mortality rates.	Reduced reproductive success of fish and bird species. Increased mortality rates, especially in earlier life stages.	Reduced biological productivity. Selection for less sensitive individuals. Possibly microevolution for ozone resistance.
Community	Changes of community structure and competitive patterns	Loss of species diversity of benthic invertebrates.	Alteration of competitive patterns. Selective advantage for ozone-resistant species. Loss of ozone sensitive species and individuals. Reduction in productivity.
Local Ecosystem (e.g.,landscape element)	Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc.	Not well understood.	Alterations of ecosystem-wide patterns of energy flow and nutrient cycling.
Regional Ecosystem (e.g., watershed)	Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity.	Not well understood.	Region-wide loss of sensitive species.

effects, while limited in its direct usefulness in a cost-benefit framework, nonetheless does convey the wide range of documented effects of air pollutants on ecological resources. These tables and the accompanying text, found in Appendix E, also provide a framework for determining the extent to which important factors may not be well characterized by quantitative analysis, setting the stage for prioritization of research needs.

Effects of Nitrogen Deposition

Table 7-3 provides a summary of the effects of nitrogen deposition on natural systems. These impacts are manifest in both terrestial and coastal estuarine systems. In both types of systems, nitrogen can be a growth-enhancing nutrient. As shown in the rows characterizing individual and population level impacts, the effects on many varieties of plants are beneficial. This growth can have other harmful effects, however. For example, excessive growth of

Table 7-3
Interactions Between Nitrogen Deposition and Natural Systems At Various Levels of Organization

		Examples of Interactions	
Spatial Scale	**Type of Interaction**	**Eutrophication and Nitrogen Saturation of Terrestrial Landscapes**	**Eutrophication of Coastal Estuaries**
Molecular and cellular	Chemical and biochemical processes	Assimilation of nitrogen by plants and microorganisms	Assimilation of nitrogen by plants and microorganisms.
Individual	Direct physiological response.	Increases in leaf- size of terrestrial plants.	Increase in growth of marine plants.
	Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress.	Decreased resistance to biotic and abiotic stress factors like pathogens and frost. Disruption of plant-symbiont relationships with mycorrhiza fungi.	Injuries to marine fauna through oxygen depletion of the environment. Loss of physical habitat due to loss of sea-grass beds. Injury through increased shading. Toxic blooms of plankton.
Population	Change of population characteristics like productivity or mortality rates.	Increase in biological productivity and growth rates of some species.	Increase in biological productivity. Increase of growth rates (esp. of algae and marine plants).
Community	Changes of community structure and competitive patterns	Alteration of competitive patterns. Selective advantage for fast growing species and individuals that efficiently use additional nitrogen. Loss of species adapted to nitrogen-poor environments.	Excessive algal growth. Changes in species composition. Decrease in sea-grass beds.
Local Ecosystem (e.g., landscape element)	Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc.	Magnification of the biogeochemical nitrogen cycle. Progressive saturation of microorganisms, soils, and plants with nitrogen.	Magnification of the nitrogen cycle. Depletion of oxygen, increased shading through algal growth.
Regional Ecosystem (e.g., watershed)	Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity.	Leaching of nitrogen from terrestrial sites to streams and lakes. Acidification of aquatic bodies. Eutrophication of estuaries.	Additional input of nitrogen from nitrogen-saturated terrestrial sites within the watershed.

marine organisms can lead to eutrophy, a state where the enhanced surface growth of plants shields bottom growing plants from sunlight, causing them to die and, in extreme cases, lead to low dissolved oxygen, or anoxic, conditions that impair a wide range of species and ecological functions. These effects are described in the table in the rows characterizing effects at the community and ecosystem levels. For this reason, isolated analysis of the effects of nitrogen on individuals or populations may provide misleading results; by the same token, analyses which ignore the beneficial effects of nitrogen in certain types of systems may lead to similarly misleading results. These complex linkages across biological levels of organization suggest that, when feasible, a systems level approach to ecological assessments is preferable to isolated analyses of effects at lower orders of organization.

Effects of Acid Deposition

Table 7-4 provides a summary of the effects of acid deposition on forest and freshwater systems. The direct effects of acid deposition in lakes and streams include effects on fish species, as charaterized in the row describing individual-level effects. These

Table 7-4
Interactions Between Acid Deposition and Natural Systems At Various Levels of Organization

		Examples of Interactions	
Spatial Scale	Type of Interaction	Acidification of Forests	Acidification of Streams and Lakes
Molecular and cellular	Chemical and biochemical processes	Damages to epidermal layers and cells of plants through deposition of acids.	Impairment of ion interactions of fish at the cellular level.
Individual	Direct physiological response	Increased loss of nutrients via foliar leaching.	Decreases in pH and increase in aluminum ions causes pathological changes in gill structure of fish.
	Indirect effects: Response to altered environmental factors or alterations of the individual's ability to cope with other kinds of stress.	Cation depletion in the soil causes nutrient deficiencies in plants. Concentrations of aluminum ions in soils can reach phytotoxic levels. Increased sensitivity to other stress factors like pathogens and frost.	Aluminum ions in the water column can be toxic to many aquatic organisms through impairment of gill regulation. Acidification can indirectly affect submerged plant species, because it reduces the availability of dissolved carbon dioxide (CO_2).
Population	Change of population characteristics like productivity or mortality rates.	Decrease of biological productivity of sensitive organisms. Selection for less sensitive individuals. Microevolution of resistance.	Decrease of biological productivity of sensitive organisms. Selection for less sensitive individuals. Microevolution of resistance.
Community	Changes of community structure and competitive patterns	Alteration of competitive patterns. Selective advantage for acid-resistant species. Loss of acid sensitive species and individuals. Decrease in productivity. Decrease of species richness and diversity.	Alteration of competitive patterns. Selective advantage for acid-resistant species. Loss of acid sensitive species and individuals. Decrease in productivity. Decrease of species richness and diversity.
Local Ecosystem (e.g., landscape element)	Changes in nutrient cycle, hydrological cycle, and energy flow of lakes, wetlands, forests, grasslands, etc.	Progressive depletion of nutrient cations in the soil. Increase in the concentration of mobile aluminum ions in the soil.	Measurable declines of decomposition of some forms of organic matter, potentially resulting in decreased rates of nutrient cycling.
Regional Ecosystem (e.g., watershed)	Biogeochemical cycles within a watershed. Region-wide alterations of biodiversity.	Leaching of sulfate, nitrate, aluminum, and calcium to streams and lakes. Acidification of aquatic bodies.	Additional acidification of aquatic systems through processes in terrestrial sites within the watershed.

effects are not as straightforward as they might appear, however, because it is not only the acidity (pH) of the water itself that causes the effect but the increased leaching of metals, particularly aluminum, which takes place in acidic (low pH) environments that contributes substantially to the effects on fish. These effects will vary widely from place to place according to the mineral content of the soil near the lake and the lakebed sediment, as well as the natural resistance of the lake in absorbing acid deposition (i.e., its buffering capacity). Other important effects characterized in the table include the ability of acid deposition to deplete cation concentrations in terrestrial ecosystems; increase the concentration of aluminum in soils; and leach nutrients, sulfates, and metals to surrounding streams and lakes. Effects of note at the individual level include foliar damage to trees.

A few general points emerge from our review of ecological effects:

- Air pollutants have indirect effects that are at least as important as direct toxic effects on living organisms. Indirect effects include those in which the pollutant alters the physical or chemical environment (e.g., soil properties), the plant's ability to compete for limited resources (e.g., water, light), or the plant's ability to withstand pests or pathogens. Examples are excessive availability of nitrogen, depletion of nutrient cations in the soil by acid deposition, mobilization of toxic elements such as aluminum, and changes in winter hardiness. As is true for other complex interactions, indirect effects are more difficult to observe than direct toxic relationships between air pollutants and biota, and there may be a variety of interactions that have not yet been detected.

- There is a group of pollutants that tend to be conserved in the landscape after they have been deposited to ecosystems. These conserved pollutants are transformed through biotic and abiotic processes within ecosystems, and accumulate in biogeochemical cycles. These pollutants include, but are not limited to, hydrogen ions (H+), sulfur (S) and nitrogen (N) containing substances, and mercury (Hg). Chronic deposition of these pollutants can result in progressive increases in concentrations and cause injuries due to cumulative effects. Indirect, cumulative damages caused by chronic exposure (i.e., long-term, moderate concentrations) to these pollutants may increase in magnitude over time frames of decades or centuries with very subtle annual increments of change. Examples are N-saturation of terrestrial ecosystems, cation depletion of terrestrial ecosystems, acidification of streams and lakes, and accumulation of mercury in aquatic food webs.

- Damages to ecosystems are most likely caused by a combination of environmental stress factors. These include anthropogenic factors such as air pollution and other environmental stress factors such as low temperature, excess or limited water, and limited availability of nutrients. The specific combinations of factors differ among regions and ecosystems where declines have been observed. Accurately predicting the impacts of multiple stress factors is an extremely difficult task, but this is an area of very active research among ecologists.

- Pollutant-environment interactions are complicated by the fact that biotic and abiotic factors in ecosystems change dramatically over time. Besides oscillations on a daily basis, and changes in a seasonal rhythm, there are long-range successional developments over time periods of years, decades, or even centuries. These temporal variations occur in polluted and pristine ecosystems, and no single point in time or space can be defined as representative of the entire system.

Selection of Service Flows Potentially Amenable to Economic Analysis

Based on this broad overview of effects, we identify a set of pollutant-environment interactions which are amenable to more detailed quantification and monetization. We evaluate the long list of effects and seek categories where a defensible link exists between changes in air pollution emissions and the quality or quantity of the ecological service flow, and where economic models are available to monetize these changes. The use of these criteria greatly constrains the range of impacts that can be treated quantitatively. While the previous section identifies many pollutant-ecosystem interactions, only a handful are understood and have been modeled to an extent sufficient to reliably quantify their impact.

The theoretical basis of economic benefits assessment is that ecosystems provide services to mankind, and that those services have economic value. The application of this theory requires the isolation of service flows that have market values or are otherwise amenable to available methods for determining value in the absence of formal markets. Available methods do not exist to comprehensively value all service flows for any particular ecosystem or aggregation of ecosystems. Generally, we are limited

to those service flows that are either sources of material inputs or associated with natural amenities that involve active recreation. Impacts to these service flows that can be valued tend to manifest themselves immediately and can be readily measured and assessed in terms of the established cause and effect relationships.

Based on the constraints of economic valuation methods and data, we select from the host of ecosystem impacts identified in the previous section a set of service flows as candidate endpoints for analysis. The list of service flows establishes the potential scope of economic analysis for ecological effects feasible in the context of the present study. Table 7-5 presents the service flow impacts that we quantitatively estimate in this analysis plus those effects that currently cannot be quantified for each of the four ecological pollutant categories discussed in Table 7-1.

From the list of effects in Table 7-5, we further limited the quantitative and qualitative analyses conducted to reflect the available model coverage. The results are summarized in Table 7-6. The relatively short list of effects in Tables 7-5 and 7-6 demonstrates that, of the great number of known impacts of air pollution, only a subset can be assessed quantitatively. Note that for one category of effects, nitrogen deposition impacts to estuarine systems, we relied on a displaced cost approach (described below)

Table 7-5
Ecological Effects of Air Pollutants

Pollutant	Quantified Effects	Unquantified Effects
Acidic Deposition	Impacts to recreational freshwater fishing	Impacts to commercial forests (e.g., timber, non-timber forest products)
		Impacts to commercial freshwater fishing
		Watershed damages (water filtration flood control)
		Impacts to recreation in terrestrial ecosystems (e.g. forest aesthetics, nature study)
		Reduced existence value and option values for nonacidified ecosystems (e.g. biodiversity values)
Nitrogen Deposition	Additional costs of alternative or displaced nitrogen input controls for eastern estuaries	Impacts to commercial fishing, agriculture, and forests
		Watershed damages (water filteration, flood control)
		Impacts to recreation in estuarine ecosystems (e.g. Recreational fishing, aesthetics, nature study)
		Reduced existence value and option values for non-eutrophied ecosystems (e.g. biodiversity values)
Tropospheric Ozone Exposure	Reduced commercial timber yields and reduced tons of carbon sequestered	Impacts to recreation in terrestrial ecosystems (e.g. forest aesthetics, nature study)
		Reduced existence value and option values for ozone-impacted ecosystems
Hazardous Air Pollutant (HAPS) Deposition	No service flows quantified	Impacts to commercial and recreational fishing from toxification of fisheries
		Reduced existence value and option values for non-toxified ecosystems (e.g. biodiversity values)

Table 7-6
Summary of Endpoints Selected for Quantitative Analysis

Endpoint	Analysis	Geographic Scope
Lake acidification impacts on recreational fishing	Quantification of improved fishing with monetization of recreational value	Case study of New York State
Estuarine eutrophication impacts on recreational and commercial fishing	Quantification of improved fishing with monetization of displaced costs of alternative eutrophication control methods	Case studies of Chesapeake Bay, Long Island Sound, and Tampa Bay (with illustrative extensions to East Coast estuaries)
Ozone impacts on commercial timber sales	Quantification of improved timber growth with monetization of commercial timber revenues	National assessment
Ozone impacts on carbon sequestration in commercial timber	Quantification of improved carbon sequestration	National assessment

that we chose to omit from the primary benefits estimate because of uncertainties in the methodology. These results are nonetheless reported in this chapter, but are used for the purposes of sensitivity testing only. In the next section we discuss the methods, results, and caveats of the analyses of these selected endpoints.

Results

In this section we summarize the methods used for, and results obtained from, our quantitative and economic analyses of selected service flows. We first review the methods for each analysis, and then present a summary of key quantitative results. For a more detailed description of methods and results, see Appendix E.

Estuarine Eutrophication Associated with Airborne Nitrogen Deposition

Atmospherically derived nitrogen makes up a sizable fraction of total nitrogen inputs in estuaries in the eastern United States. Airborne nitrogen deposition accounts for a significant fraction of the total nitrogen loads to coastal estuaries, particularly on the East and Gulf coasts. For example, the most recent estimates for the Chesapeake Bay indicate airborne deposition accounts for over 40 percent of the total nitrogen load to the estuary; in Galveston Bay, the share is almost 50 percent. When nitrogen enters estuaries it can cause eutrophication, or an increased nutrient load that, in excess, changes the ecosystem's structure and function and affects ecological service flows. Many state governments and multi-state regional authorities have expressed increasing concern about the control of airborne nitrogen deposition as an important source of nitrogen loading.

Our analysis of the effects of nitrogen deposition followed two tracks. We first attempted to quantify the service flows affected by and the damages associated with eutrophication, and derive dose-response relationships and valuation strategies for each of the key service flow categories (for example, recreational fishing). The derivation of dose-response relationships between atmospheric nitrogen loading and ecological effects, however, is complicated by the dynamic nature of ecological systems. In addition to being characterized by non-linear, "threshold" type responses, estuarine ecosystems are simultaneously influenced by a variety of stressors (both anthropogenic and natural). This makes it difficult to quantify the nature and magnitude of ecological changes expected to result from a change in a single stressor such as nutrient loading. Further, if the state of the ecosystem has changed (as from oligotrophic[1] to eutrophic) the removal of the initial stressor does not necessarily mean a rapid return to the prior state. This complicates the quantitative benefits assessment of controlling nitrogen deposition through the CAAA.

[1] Oligotrophy refers to a state of relatively low nutrient enrichment and low productivity of aquatic ecosystems. In contrast, eutrophy refers to a state of relatively high nutrient loading and higher productivity, sometimes leading to overenrichment and reduction in ecological service flows due to water quality decline.

Our second track relies on a displaced cost approach to benefit estimation. To reduce excess nutrient loads (including nitrogen) to local estuaries, many coastal communities are pursuing a range of abatement options. These options include wastewater and stormwater discharge point source controls as well as urban non-point and agricultural non-point source controls for runoff from the land. If atmospheric nitrogen depostion is reduced, the need for these types of expenditure to control other sources of nitrogen loading is also lessened, and the displaced control expenditures represent a benefit to society.

Displaced or avoided cost approaches are not always justified. In order to establish that the costs would truly be avoided, and to ensure that the avoidance of that cost represents a real benefit to society, we need to show that realistic and enforceable nitrogen reduction goals exist for each evaluated estuary. Without specific targets or reduction goals, it is not possible to suggest that there are specific control expenditures to be displaced. Therefore, we choose case study estuaries that most closely meet this criterion: Chesapeake Bay, Long Island Sound, and Tampa Bay. These areas have established nitrogen reduction programs that rely primarily on reductions of effluent from point sources as well as reductions in non-point source discharges. Information on the reduction goal and potential abatement options for meeting those goals allows us to estimate the portion of the goal that can be met by the CAAA, as well as the associated cost savings.[2]

The benefits valuation derived using the displaced-costs approach should be interpreted cautiously for two reasons. First, it is an estimation of capital costs that serve more purposes than mitigating nitrogen inputs into the estuaries of concern. Water treatment works are intended to provide waste water treatment for a variety of pollutants and may be required even in the absence of deposition of airborne nitrogen. Second, the nitrogen loading targets for the estuaries are not concrete, strictly enforced limits, based on certain knowledge of the capacity of the estuaries to accept nitrogen inputs. Instead, the targets may change over time as knowledge of the effects of nitrogen to these estuaries change. For these reasons, and because of the uncertainty about the ability of local and regional entities to enforce the nitrogen reduction targets, we calculate estimates of displaced costs for these three estuaries but do not include them in the primary benefits estimate for the CAAA.

Our approach involves three basic steps. First, we estimate the total loading of nitrogen to each of the three target estuaries. We use nitrogen deposition estimates from the RADM model, generated for each 80 km x 80 km grid cell in the eastern U.S. We then estimate the ultimate fate of deposited nitrogen through a GIS-based model of nitrogen "pass-through." The pass-through is the share of nitrogen deposited that is ultimately transported to the estuarine waters rather than retained by the land. Pass-through factors vary by land use, from about 20 percent (for forests and wetlands) to 100 percent (for open water). We estimate the nitrogen loading for each scenario, and the within-year, cross-scenario differences are the reduced nitrogen deposition attributed to the CAAA. We present these estimates in the second column of Table 7-7.

Second, we estimate the marginal costs of alternative abatement actions which could be implemented in the three case study estuaries. We develop our displaced-cost estimate by assuming that decision makers will choose to forego the most costly nitrogen abatement projects first. That is, we assume that reduced deposition and the resulting loadings reduction will eliminate the need for additional point or non-point source controls at the high end of the marginal cost curve. We summarize those results in the third and fourth columns of Table 7-7.

Third, we multiply the reduced nitrogen loading attributed to the CAAA by the marginal cost estimate to arrive at a range of estimates of displaced cost, ensuring that the reduction in airborne nitrogen is less than or equal to the potential tonnage reduction achieved by the displaced, high marginal cost abatement strategies. We present our results in the last column of Table 7-7. Our estimates suggest that the displaced cost is substantial for the large Chesapeake Bay and Long Island Sound estuaries, and more modest for Tampa Bay. The Chesapeake

[2] With increasing populations, controls of alternative sources (e.g., automobile and utility emissions) may be needed simply to meet the original target or goal. If the CAA amendments are necessary just to achieve the target reductions, then we are actually measuring alternative costs and not avoided costs.

Table 7-7
Estimated Displaced Costs for Three Estuaries

Estuary	Reduced N Deposition in 2010(millions of pounds)	Low Marginal Cost($/lb/yr.)	High Marginal Cost ($/lb/yr.)	Estimated Annual Displaced Costs in 2010 ($millions)
Long Island Sound	12.8	$2	$8	$26-$100 Central Estimate: $63
Chesapeake Bay	58.1	$6	$22	$350-$1,300 Central Estimate: $820
Tampa Bay	1.8	$6	$38	$11 - $68 Central Estimate: $40

Bay and Long Island Sound watersheds together account for about 40 percent of the total estuarine watershed area on the East (Atlantic) coast that is sensitive to nitrogen deposition, while Tampa Bay accounts for about two percent of the sensitive watershed area for the Gulf coast.

Acidification of Freshwater Fisheries

During the 1970s and 1980s, "acid rain" came to be known to the public as a phenomenon that injures trees, forests, and water bodies throughout Europe and in some areas of the United States and Canada. One of the goals of the CAAA was to address the problem of acidification of terrestrial and aquatic ecosystems caused by acidic deposition. To assess this effect we conducted a quantitative analysis of benefits derived from a reduction in acidification of aquatic bodies as they relate to recreational fishing in the Adirondacks region of New York State.

As discussed earlier in this chapter, acidification of water bodies is a complex process. Airborne acids, in the form of sulfur and nitrogen compounds, are deposited to water bodies and surrounding drainage areas, with the potential to change the pH of the water body. Many water bodies are relatively resistant and can absorb a great deal of deposition before pH changes substantially. This buffering capacity is referred to as acid neutralizing capacity (ANC). Once pH begins to be affected, a series of interactions occur, the most important of which is the leaching of aluminum from sediments and surrounding soil and the suspension of this metal in the water column. While acidic pH presents a direct stress to aquatic organisms, it is the combined effect of pH and aluminum exposure that presents the greatest risk. Lakes in the Adirondacks region of New York State are particularly susceptible to acidification because they have low baseline ANC, relative to water bodies in other areas of the country.

Because of these complex physical and chemical interactions, acidification stress is typically evaluated by application of a model that simulates these processes, and requires data on individual lake chemistry and sediment composition. We relied on the scenario-specific atmospheric deposition data (both sulfur and nitrogen) from the RADM air quality model (see Chapter 4 and Appendix C) as an input to EPA's Model of Acidification of Groundwater in Catchments (MAGIC). MAGIC generates several measures of the impact of sulfur and nitrogen deposition on lake acidity, including ANC and pH.[3] We used the pH outputs to classify lakes where recreational fishing might be impaired, and those estimates were used in an economic model of recreational fishing behavior in New York State to develop economic estimates of the impact of acid rain on recreational fishing resources in that state.

We summarize the results of our analysis of economic benefits of avoided Adirondacks acidification attributable to the CAAA in 2010 in Table 7-8. The range of annual benefits from the CAAA are $12 million to $49 million using the low-end assumptions on the threshold of effect (pH 5.0), and $82 to $88 million for the high-end assumptions on the effects threshold (pH 5.4). Higher pH (or, less acidic) threshold assumptions lead to greater damage estimates, because more lakes cross the less acidic threshold. We calculate our benefits results by comparing

[3] For more information on EPA's MAGIC model see Cosby et al. (1985a), as referenced in Appendix E.

Table 7-8
Annual Economic Impact of Acidification in 2010 (Millions of 1990 Dollars)

		Range of Economic Impact		
Year	**Scenario**	**Low Estimate**	**Central Estimate**	**High Estimate**
1990	Base Year	$61		$320
2010	Post-CAAA	$24 to $61		$261 to $281
	Pre-CAAA	$73		$349 to $363
Range of CAAA Benefits in 2010		**$12 to $49**	**$50**	**$82 to $88**

the suite of Post-CAAA 2010 estimates of total damages to the corresponding suite of estimates using Pre-CAAA deposition. The impact of nitrogen saturation in the surrounding terrestrial environment is reflected in the range of estimates presented in Table 7-8. If surrounding soils are saturated, less deposited nitrogen will remain on the land and more nitrogen will enter the water bodies, increasing the stress on the aquatic ecosystem. This phenomenon is reflected by the higher damage estimates for saturated versus non-saturated scenarios, other factors equal, although our model shows no effect of saturation in the 2010 Pre-CAAA low estimate. The results we present are in line with those generated from previous analyses that find annual benefits to the Adirondacks of halving utility emissions to be approximately in the millions to tens of millions of dollars.[4]

Reduced Timber Growth Associated with Ozone Exposure

The third category of effects we quantify is improved commercial timber growth through the reduction of tropospheric ozone concentrations attributable to the CAAA. There is substantial scientific evidence to suggest that elevated ozone concentrations in the troposphere disrupt ecosystems by damaging and slowing the growth of vegetation. In this analysis, we examine one aspect of these impacts, reduced commercial timber growth. Much of the literature on the effects of ozone on tree growth is based on laboratory exposures of seedlings or leaf-scale experiments in the field. Estimates from those studies have been used in previous analyses, making use of professional judgment as an interpretive tool, but always with strong caveats about the potential applicability of the seedling and leaf-scale results to tree growth and, in particular, the rate of accumulation of wood mass that is important for commercial timber production.[5] In an attempt to overcome these issues, we sought to find a concentration-response relationship that would provide a more defensible and broadly applicable basis for estimating effects on tree growth.

Our analysis makes use of the Net Photosynthesis and Evapo-Transpiration model II (PnET II), a biological model of timber stand productivity to estimate the impacts of ozone on timber yields. The PnET II model was designed to estimate the combined effects of several stressors on the rate of net primary productivity (NPP), a measure of the rate of photosynthesis. NPP in a tree does not necessarily all go towards accumulation of wood mass; some may be allocated to root growth, leaf growth, or other tree functions. The PnET II model provides a means to measure both NPP and wood mass growth, as well as the effect on trees of several stressors combined. One important stressor to acknowledge in an analysis of the effects of ozone on trees is drought stress. Ozone has the effect of reducing water loss in trees by stimulating the closing of stomata through which water is transpired. As a result, in drought stress conditions, ozone can have beneficial effects on tree growth. The PnET II model reflects the impact of this factor in combination with other direct effects of ozone on tree function.

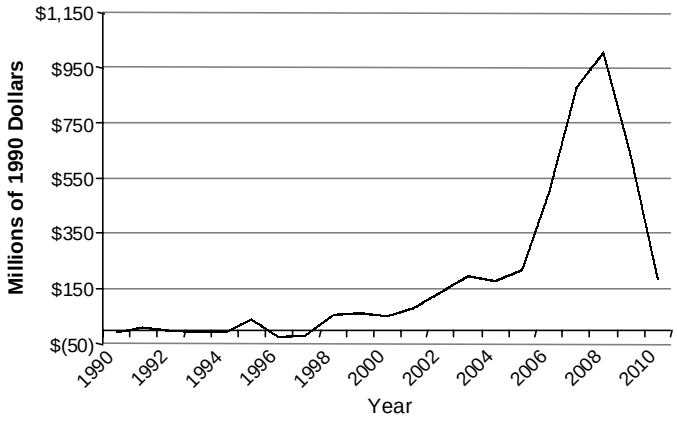
We used the PnET II model to provide estimates of timber stand responses to ozone exposure under each of the scenarios examined in this analysis. We aggregated tree growth results by region, with separate estimates for hardwoods and softwoods, and used them as inputs to the Timber Assessment Market

[4] For alternative estimates see, for example, Englin et al. (1991), Mullen and Menz (1985), and Morey and Shaw (1990), as referenced in Appendix E.

[5] See de Steiger et al. (1990) for an example of the generation of tree growth dose-response estimates based on professional judgement.

Model (TAMM), an economic model of the forest sector maintained by the United States Forest Service. There are three stages to the economic estimation. First, forest growth rate information generated by PnET II is provided to the Aggregate Timber Land Assessment System (ATLAS), the forest inventory tracking component of TAMM. Growth rate information is provided for each of the forest production regions defined by TAMM.[6] Second, ATLAS generates an estimate of forest inventories in each major region, which in turn serves as input to the market component of TAMM. Third, TAMM estimates the future harvests and market responses in each region.

Figure 7-1
Annual Economic Welfare Benefit of Mitigating Ozone Impacts on Commercial Timber: Difference Between the Pre-CAAA and Post-CAAA Scenarios



Our analysis suggests that there is a significant and measurable difference in timber harvests attributable to ozone exposure under the Post-CAAA and Pre-CAAA scenarios. At the outset of our modeling period, the early 1990s, virtually no change is measured in forest harvest volumes. This result occurs because increases in growth rates do not substantively affect timber volume over a short period of time. By the end of our modeling period, nearing 2010, increased growth rates over the previous decade(s) begin to affect overall forest yields of harvestable timber. This is observed in Figure 7-1 as an increasing annual benefit estimate over the modeling period. The shape of the benefits time-series reveals a production spike in the 2007 to 2008 period. This spike is due to a large anticipated harvest of Southeast U.S. timber due to forest maturity during this period. The spike would occur even in the absence of the CAAA, but is elevated by the CAAA due to increased growth rates projected under the Post-CAAA scenario. Although this change is small in percentage terms relative to total economic surplus generated by the timber sector, it contributes to a large portion of the commercial timber benefits estimate over the 1990-2010 period.

We calculate the cumulative value of annual benefits based on the discounted stream of the annual differences in consumer and producer surplus from commercial timber harvests under the Post-CAAA and Pre-CAAA ozone exposure scenarios from 1990 to 2010. Discounting annual benefits to 1990 using a five percent discount rate, the total cumulative benefits estimate is approximately $1.9 billion. These estimates are incorporated into the primary central estimate by developing a range of annual estimates for the year 2000, based on model results for the period 1998 to 2002, and the year 2010, based on model results for the period 2005 to 2010. The averaging of results across several years to generate our target year results avoids the potential problem of a particular year's results (such as for 2010) mischaracterizing the full time series of estimates when we later calculate the net present value of effects.

Reduced Carbon Sequestration Associated with Reduced Timber Growth

Forest ecosystems help mitigate increasing atmospheric concentrations of carbon dioxide by sequestering carbon from the atmosphere. These ecosystems convert atmospheric carbon into biological structures (e.g., wood) or substances needed in the tree's physiological processes. As described above, however, ozone reduces the growth of forests, thereby limiting the amount of carbon that is sequestered. Sequestered carbon can help mitigate global climate change that has been linked to anthropogenic emissions of carbon and other greenhouse gases.

[6] TAMM includes Canadian as well as U.S. timber production regions because of the important influence of Canadian timber supply on the U.S. market. This analysis reflects modeling of Canadian timber regions and their impact on U.S. production, but we did not simulate changes in ozone in Canadian regions.

We used the timber inventory output of the TAMM/ATLAS modeling system (described above), in combination with a forest carbon model (FORCARB), to estimate changes in carbon storage in each of four ecosystem components: trees, forest understory, forest floor, and soil. The estimates from FORCARB, however, do not account for "leakages" of carbon back to the atmosphere as wood or wood products decay and decompose over time. To estimate the amount of carbon that is sequestered over the long-term, we used a second model, HARVCARB, to estimate the life-cycle of harvested forest timber and thereby adjust the forest carbon sequestration estimates of FORCARB.

The results of these calculations yield estimates of long-term increases in carbon storage as a result of the CAAA provisions of 8 million metric tons of carbon per year by the year 2000, and 29 million metric tons of carbon per year by the year 2010. Because of the great uncertainties in assessing the mitigating effect of carbon sequestration on global climate change, and the economic value of avoiding climate change, we do not attempt to monetize this category of benefit.

Other Categories of Ecological Benefits

There were two additional categories of ecological effects for which we considered developing economic estimates; however, we abandoned the exercise when key portions of the analysis proved to be excessively problematic. Aesthetic degradation of forests, the first of these additional categories, was supported by a benefits transfer of contingent valuation studies of individual willingness to pay to avoid foliar damage. This category of effects, however, proved too difficult to link to the specific air quality scenarios we evaluated. In other words, available scientific methods and data on the visual appearance of forest stands and their impact on perceived forest aesthetics make it difficult to precisely describe changes in forest aesthetics. Evaluation of the second additional effect category, toxification of freshwater fisheries, was limited by the lack of toxic deposition and exposure data as well as by the limitations of available economic estimates of the impacts of toxics on recreational and commercial fishery resources. (See Appendix E for a more detailed discussion of these service flows). These and many other ecological benefit categories could not be quantified given current data and methods and are thus not reflected in our overall benefits estimates.

Valuation of Other Effects

Agricultural Benefits

As discussed earlier in this chapter, tropospheric ozone affects the growth of a wide range of plant species, including agricultural crops. Our agricultural benefits analysis relies on crop-yield loss C-R functions derived from the National Crop Loss Assessment Network (NCLAN) research and a national economic model of the agricultural sector (AGSIM). The NCLAN-derived relationships use a sum of hourly ozone concentration at or above 0.06 ppm (SUM06) as a measure of ozone exposure for the May to September ozone season; these exposure estimates are derived from the ozone air quality modeling results discussed in Chapter 4. Where the C-R functions require a longer time period of ozone concentrations, for example, for winter crops or when the growing or harvest season for summer crops extends beyond the end of September, we rely on 1990 monitor data to estimate ozone exposure, conservatively using the same estimates for both Pre-CAAA and Post-CAAA scenarios. The NCLAN functions cover the following crops: corn, cotton, peanuts, sorghum, soybeans, and winter wheat.

The AGSIM agricultural sector model takes the yield loss information, incorporates agricultural price, farm policy, and other data for each year, and then estimates production levels for each crop and the economic benefits to consumers and producers associated with these production levels. The crop coverage in the AGSIM model includes a wider range of crops than the NCLAN data inputs, adding barley, oats, hay, rice, and cottonseed. The broader crop coverage ensures that the model addresses price and production quantity effects on potential substitute crops that might be related to the effects in the six NCLAN crops. We estimate economic effects using a range of C-R outcomes for several crops, to reflect the variation in ozone sensitivity among the various crop cultivars. Our central estimate is the expected value of the range of results that emerge from the economic model.

Our results indicate significant beneficial effects of ozone reductions in the agricultural sector. Our Primary Central estimate of the benefit in 2000 is $450 million; the annual benefit rises to $550 million in 2010. Our estimated uncertainty around the Primary Central estimates, however, is very broad. For example, in 2010, the Primary Low estimate is $7.1 million, and the Primary High is $1,100 million. The uncertainty range reflects variation in the ozone response of crop cultivars and uncertainty about the suitability of alternative crop cultivars for the soil types and climate conditions in various agricultural regions. See Appendix F for more details on the methods and results of the C-R functions and economic modeling for agricultural effects.

Visibility

As outlined in Chapter 4, air pollution impairs visibility in both residential and recreational settings. An individual's willingness to pay to avoid reductions in visibility differs in these two settings. Impairments in residential visibility are experienced throughout an individual's daily life and activities. Visibility in recreational settings, on the other hand, is experienced by visitors to areas with notable vistas. For the purposes of this report, we interpret recreational settings applicable for this category of effects to include National Parks throughout the nation. Other recreational settings may also be applicable, for example National Forests, state parks, or even hiking trails or roadside areas, but a lack of suitable economic valuation literature to identify these other areas, as well as a lack of visitation data in some cases, prevents us from generating estimates for those recreational vista areas.

We derive a residential visibility valuation function from the Chestnut and Dennis (1997) published estimates for the Eastern U.S. These estimates are based on original research conducted by McClelland et al. (1990) in two Eastern cities (Atlanta and Chicago). Because of technical concerns about the study's methodology, however, we calculate a benefits estimate but omit the results from the primary benefits estimates.[7] For recreational visibility, we derive values from the the Chestnut and Rowe (1989) study of WTP for visibility in three park regions in the Western, Southwestern, and Eastern U.S.[8] In both cases, the valuation function takes the following form:

$$HHWTP = B * \ln(VR1/VR2)$$

where:

HHWTP = annual WTP per household for visibility changes
VR1 = the starting annual average visual range
VR2 = the annual average visual range after the change in air quality
B = the estimated visibility coefficient.

The form of this valuation function is designed to reflect the way individuals perceive and express value for changes in visibility. In general terms, expressed WTP for visibility changes varies with the percentage change in visual range, a measure that is closely related to, though not exactly analogous to, the Deciview index used in Chapter 4. We use a central B coefficient for residential visibility of 141, as reported in Chestnut and Dennis (1997). For recreational visibility, the coefficients vary based on the region of study and whether the household is within or outside of the National Park region studied. In-region coefficients are higher than those for out-of-region households. The in-region estimates for California, the Southwest, and Southeast are $105, $137, and $65, respectively; the corresponding out-of-region estimates are $73, $110, and $40, respectively. The derivation and application of these valuation functions are described in more detail in Appendix H. The results of this procedure suggest visibility is an important category of CAAA benefits; the Primary Central estimate for 2010, for example, indicates annual recreational visibility benefits of $2.9 billion.

Worker Productivity

We base the valuation of worker productivity on a study that measures the decline in worker pro-

[7] The two technical concerns involve the method of adjusting the contingent valuation survey results for non-response, and the failure to include adjustments for the "warm glow" effect, or the tendency of respondents to indicate higher willingness to pay for an environmental good because of a strong desire to improve the environment in general.

[8] The visibility valuation function, and the sources of estimates for the coefficients for the functions, were originally developed as part of the National Acid Precipitation Assessment Program (NAPAP), and were subjected to peer-review as part of that program.

ductivity among outdoor farm workers exposed to ozone (Crocker and Horst, 1981). In our analysis, we estimate the value of reduced productivity at $1 per 10 percent increase in ozone concentration. This estimate reflects valuing reduced productivity in terms of the reduction in percentage of daily income incurred by the average worker engaged in strenuous outdoor labor.

Stratospheric Ozone Provisions

The quantified benefits of stratospheric ozone protection provisions are dominated by the reduced health effects expected from reductions in UV-b radiation; the derivation of health benefits of these provisions is discussed in Chapter 5. We summarize other categories of benefits associated with reduced UV-b radiation in Table 7-9. The quantified benefits include: reduced crop damage; and reduced polymer degradation. To estimate crop damage, we apply the results of existing studies on the relationship between crops and UV-b radiation to the changes in UV-b radiation predicted by the emissions and atmospheric models.[9] The polymer damage function is based on a study by Horst (1986). The estimated total cumulative benefits associated with these ecological and other welfare effects are about 2 percent of the total cumulative benefits of the Title VI provisions.

[9] Sources of dose-response relationship for crops and UV-b: Teramura and Murali (1986) and Rowe and Adams (1987). Source of dose-response relationship for crops and tropospheric ozone: Rowe and Adams (1987).

Table 7-9
Quantified and Unquantified Ecological and Welfare Effects of Title VI Provisions

Ecological Effects- Quantified	Estimate	Basis for Estimate
American crop harvests	Avoided 7.5 percent decrease from UV-b radiation by 2075	Dose-response sources: Teramura and Murali (1986), Rowe and Adams (1987)
American crop harvests	Avoided decrease from tropospheric ozone	Estimate of increase in tropospheric ozone: Whitten and Gery (1986). Dose-response source: Rowe and Adams (1987)
Polymers	Avoided damage to materials from UV-b radiation	Source of UV-b/stabilizer relationship: Horst (1986)
Ecological Effects- Unquantified		
Ecological effects of UV. For example, benefits relating to the following: • recreational fishing • forests • marine ecosystem and fish harvests • avoided sea level rise, including avoided beach erosion, loss of coastal wetlands, salinity of estuaries and aquifers • other crops • other plant species • fish harvests		
Ecological benefits of reduced tropospheric ozone relating to the overall marine ecosystem, forests, man-made materials, crops, other plant species, and fish harvests		
Benefits to people and the environment outside the U.S.		

Notes:
1) For more detail see EPA's *Regulatory Impact Analysis: Protection of Stratospheric Ozone* (1988).
2) Note that the ecological effects, unlike the health effects, do not reflect the accelerated reduction and phaseout schedule of section 606.
3) Benefits due to the section 606 methyl bromide phaseout are not included in the benefits total because the EPA provides neither annual incidence estimates nor a monetary value.

Summary of Quantitative Results

Although the effects of air pollutants on ecological systems are likely to be widespread, many effects may be poorly understood and lack quantitative effects characterization methods and supporting data. In addition, many of our quantitative results reflect an incomplete geographic scope of analysis; for example, we generated monetized acidification results only for the Adirondacks region of New York State. As a result, the quantitative results we generate for the purposes of estimating the benefits of the CAAA reflect only a small portion of the overall impacts of air pollution on ecological systems or ecological service flows.

Despite these limitations, it is important to recognize the magnitude of the monetized ecological benefits that we could estimate and reflect those results in the overall estimates of benefits generated in the larger analysis. Table 7-10 provides a tabular summary of the results documented earlier in this chapter. It is not possible to indicate the degree to which ecological benefits are underestimated, but considering the magnitude of benefits estimated for the select endpoints considered in our analysis, it is reasonable to conclude that a comprehensive benefits assessment would yield substantially greater total benefits estimates.

In Table 7-11 we provide a summary of benefits estimates for other welfare effects, including reduced agricultural yields, impaired visibility, and decreased

Table 7-10
Summary of Evaluated Ecological Benefits (millions 1990$)

Description of Effect	Air Pollutant	Geographic Scale of Economic Estimate	Range of Annual Impact Estimates in 2010	Primary Central Estimate for 2010	Primary Central Cumulative Impact Estimate 1990-2010	Key Limitations
Freshwater acidification	Sulfur and nitrogen oxides	Regional (Adirondacks)	$12 to $88	$50	$260	- Captures only recreational fishing impact - Incomplete geographic coverage leads to underestimate of benefits
Reduced tree growth - Lost commercial timber	Ozone	National	$190 to $1000	$600	$1,900	- Uncertainties in stand-level response to ozone exposure - Uncertainty in future timber markets
	TOTAL MONETIZED ECONOMIC BENEFIT		$200 to $1,100	$650	$2,200	- Partial estimate that omits major unquantifiable benefits categories; see text

Note: Estimates reflect only those benefits categories for which quantitative economic analysis was supported. A comprehensive total economic benefit estimate would likely greatly exceed the estimates in the table. Range of estimates for timber assessment is based on variation in annual point estimates for 2005 through 2010.

Table 7-11
Summary of Other Welfare Benefits (millions 1990$)

Description of Effect	Air Pollutant	Geographic Scale of Economic Estimate	Primary Central Annual Estimate 2000	Primary Central Annual Estimate 2010	Primary Central Cumulative Estimate 1990-2010	Key Limitations
Reduced Agricultural Yields	Ozone	National	$450	$550	$3,900	- Covers only major grain crops - Omits effects on fruits and vegetables
Impaired Recreational Visibility	Particulate Matter	National	$2,000	$2,900	$19,000	- National Parks only - Omits residential visibility benefits
Reduced Worker Productivity	Ozone	National	$460	$710	$4,400	- Reflects effects on workers engaged in strenuous outdoor employment

Note: Estimates reflect only those benefits categories for which quantitative economic analysis was supported.

worker productivity. These estimates add substantially to the total non-health benefits of the CAAA. In particular, our estimates for the annual value of avoiding visibility impairments is $2,900 million by 2010, even through this estimate does not reflect the value of residential visibility improvements.

Uncertainty

Because of the limitations in the available methods and data, the benefits assessment in this report does not represent a comprehensive estimate of the economic benefits of the CAAA. Moreover, the potential magnitude of long-term economic impacts of ecological damages mitigated by the CAAA suggests that great care must be taken to consider those ecosystem impacts that are not quantified here. Significant future analytical work and basic ecological and economic research must be performed to build a sufficient base of knowledge and data to support an adequate assessment of ecological benefits. For the current analysis, this incomplete coverage of effects represents the greatest source of uncertainty in the ecological assessment. This and other key uncertainties are summarized in Table 7-12.

Because the chronic ecological effects of air pollutants may be poorly understood, difficult to observe, or difficult to discern from other influences on dynamic ecosystems, our analysis focuses on acute or readily observable impacts. Disruptions that may seem inconsequential in the short-term, however, can have hidden, long-term effects through a series of interrelationships that can be difficult or impossible to observe, quantify, and model. This factor suggests that many of our qualitative and quantitative results may underestimate the overall, long-term effects of pollutants on ecological systems and resources.

Table 7-12
Key Uncertainties Associated with Ecological Effects Estimation

Potential Source of Error	Direction of Potential Bias for Net Benefits Estimate	Likely Significance Relative to Key Uncertainties in Net Benefit Estimate*
Incomplete coverage of ecological effects identified in existing literature, including the inability to adequately discern the role of air pollution in multiple stressor effects on ecosystems.	Underestimate	Potentially major. The extent of unquantified and unmonetized benefits is largely unknown, but the available evidence suggests the impact of air pollutants on ecological systems may be widespread and significant. At the same time, it is possible that a complete quantification of effects might yield economic valuation results that remain small in comparison to the total magnitude of health benefits.
Omission of the effects of nitrogen deposition as a nutrient with beneficial effects.	Overestimate	Probably minor. Although nitrogen does have beneficial effects as a nutrient in a wide range of ecological systems, nitrogen in excess also has significant and in some cases persistent detrimental effects that are also not adequately reflected in the analysis.
Incomplete assessment of long-term bioaccumulative and persistent effects of air pollutants.	Underestimate	Potentially major. Little is currently known about the longer-term effects associated with the accumulation of toxins in ecosystems, but what is known suggests the potential for major impacts. Future research into the potential for threshold effects is necessary to establish the ultimate significance of this factor.
The PnET II modeling of the effects of ozone on timber yields relies on a simplified mechanism of response (i.e., changes in net primary productivity).	Overestimate	Probably minor. Existing evidence suggests that the growth changes PnET II projects are relatively large, however none of the currently available points of conparison fully address such issues as the impact of stand-level competition, and the net primary productivity results are within the range of results of other studies of environmental and anthropogenic stressors.

*The classification of each potential source of error reflects the best judgement of the section 812 Project Team. The Project Team assigns a classification of "potentially major" if a plausible alternative assumption or approach could influence the overall monetary benefit estimate by approximately five percent or more; if an alternative assumption or approach is likely to change the total benefit estimate by less than five percent, the Project Team assigns a classification of "probably minor."

Comparison of Costs and Benefits

Chapter 8

In this chapter we present our summary of the primary estimates of monetized benefits of the CAAA from 1990 to 2010, compare the benefits estimates with the corresponding costs, and explore some of the major sources of uncertainty in the benefits estimates. We also present the results of our calculations using alternative assumptions for several key input variables.

Monetized Benefits of the CAAA

In this section we provide an overview of the three types of analyses conducted to estimate benefits, present the annual estimates of monetized benefits for the human health, ecological, and welfare analyses, and then present an aggregate measure of benefits from all titles of the CAAA for the full study period.

Overview of Benefits Analyses

Our primary estimates of the monetized economic benefits for the 1990 to 2010 period derive from three distinct analyses: (1) the analysis of changes in human health effects associated with reduced exposures to criteria pollutants and the subsequent valuation of these changes, summarized and described in Chapters 5 and 6; (2) the analysis of monetized ecological and other welfare benefits (e.g., visibility), described in Chapter 7; and (3) the analysis of the benefits of stratospheric ozone protection provisions, summarized briefly in Chapters 5, 6, and 7 and described in detail in Appendix G.

We measure the benefits and present the results from each of these analyses in slightly different ways. For the first two analyses, we generate annual estimates of benefits that result from changes in exposures in two target years of the study, 2000 and 2010. These estimates can be directly compared to the estimates of costs incurred in the target years, because for the most part the annual benefits accrue in the same year as the costs are incurred. There is one exception, however: we model the effect of particulate matter on premature mortality to occur over a period of five years from the time of exposure. In this case, we have accounted for the incidence of premature mortality over the assumed lag period, and discounted the valuation of this effect back to the target year.

The annual estimates provide an indication of the trend in benefits accrued over the 20-year study period. To generate a cumulative measure of benefits over the full 20-year period, we must make an assumption about the level of benefits that would be realized in the years between the target years. We interpolate these values, assuming a linear trend in both costs and benefits over the 1990 to 2000 and 2000 to 2010 periods (assuming benefits and costs in the starting year, 1990, are zero). In one portion of the ecological benefits analysis, acidification, we generate only a single annual estimate for the target year 2010. In that case, we assume a linear trend in annual benefits over the full 20-year study period.

The third analysis, assessing changes in stratospheric ozone and the resulting health effects, is different from the criteria pollutant analyses. The long-term nature of the program, and the significant lag effects associated with the processes of ozone depletion over decades-long time scales, make it difficult to generate a meaningful estimate for any single target year. As a result, we could not generate an annual benefit estimate that could be reliably linked to emissions reductions in a single year and, by extension, compared to the costs incurred to achieve that year's allocation of reductions in stratospheric ozone depleting substances. Instead, we generate an annualized equivalent of the cumulative present value of

benefits and costs of the Title VI program. These annualized equivalents cannot be ascribed to any particular target year.

These fundamental differences in the measurement of benefits affect our presentation of benefits estimates in this chapter. Although we generate and report an annual estimate of costs and benefits of Title VI provisions, we encourage the reader to interpret aggregations of these annual estimates with those from other titles of the CAAA with caution. In particular, we discourage the use of these CAAA Title-specific benefit-cost ratios as the sole, or even primary, basis for comparing the relative economic value of Title VI versus other CAAA titles. The comparative benefit-cost ratios are too sensitive to important, highly uncertain analytical assumptions such as the discount rate.

Summary of Monetized Benefits for Human Health and Welfare Effects

As discussed above, we generate annual estimates for the human health and welfare effects based on exposure analysis conducted for each of the two target years of the analysis, 2000 and 2010. The range of estimates we generate for the monetized benefits of human health effects incorporates both the quantified uncertainty associated with each of the health effect estimates and the quantified uncertainty associated with the corresponding economic valuation strategy. Quantitative estimates of uncertainties in earlier steps of the analysis (i.e., emissions and air quality changes) could not be developed adequately and are therefore not applied in the present study. As a result, the range of estimates for monetized benefits presented in this chapter is more narrow than would be expected with a complete accounting of the uncertainties in all analytical components. The characterization of the uncertainty surrounding economic valuation is discussed in detail in Appendix H. The characterization of the uncertainty surrounding specific health effect estimates is discussed in Appendix D. Below, we discuss the combined effect of these two categories of uncertainty and our techniques for aggregating uncertainty across endpoints and analyses.

We assume that for each endpoint-pollutant combination there are distributions for both the concentration-response function and the valuation coefficients. We combine these distributions by using a computerized, statistical aggregation technique to estimate the mean of the monetized benefit estimate for each endpoint-pollutant combination and to characterize the uncertainty surrounding each estimate.[1]

In the first step of our procedure, we employ statistical analysis to generate mean estimates and quantified uncertainty measures for the C-R function for each endpoint-pollutant combination. For many health and welfare effects, only a single study is available to use as the basis for the C-R function. In this case, the best estimate of the mean of the distribution of C-R coefficients is the reported estimate in the study. The uncertainty surrounding the estimate of the mean C-R coefficient is characterized by the standard error of the reported estimate. This yields a normal distribution, centered at the reported estimate of the mean. If multiple studies are considered for a given C-R function, a normal distribution is derived for each study, centered at the mean estimate reported in the study. On each iteration of the aggregation procedure, a C-R coefficient is selected from an aggregate distribution of C-R estimates for that endpoint. The aggregate distribution of C-R coefficients is determined by a variance-weighted aggregate distribution of values.

In the second step, we estimate incidence for each exposure analysis unit (i.e., 8 km by 8 km cell in a grid pattern) in the 48 contiguous states, and aggregate the results into an estimate of the change in national incidence of the health or welfare effects. Through repeated iterations from the distribution of mean C-R coefficients, we generate a distribution of the estimated change in incidence for each health and welfare effect due to the change in air quality between the Post-CAAA and Pre-CAAA scenarios.

Finally, in the third step we use computerized statistical aggregation methods once again to charac-

[1] The statistical aggregation technique applied is commonly referred to as simulation modeling. The technique involves many re-calculations of results, using different combinations of input parameters each time. For each calculation, values from each input parameter's statistical distribution are selected at random to ensure that the calculation does not always result in extreme values, or rely solely on low end or solely on high end input parameters. The aggregate distribution more accurately reflects a reasonable likelihood of the joint occurrence of multiple input parameters.

terize the overall uncertainty surrounding monetized benefits. For each distinct health and welfare effect, the aggregation procedure selects an estimated incidence change from the distribution of changes for that endpoint, selects a unit value from the corresponding distribution of economic valuation unit values, and multiplies the two to generate a monetized benefit estimate. We then repeat the process many times to generate a distribution of estimated monetized benefits for each endpoint-pollutant combination. Combining the results for the individual endpoints using the aggregation procedure yields a distribution of total estimated monetized benefits for each target year (2000 and 2010).[2] We present the results of this analysis of health effects in Table 6-3 in Chapter 6.

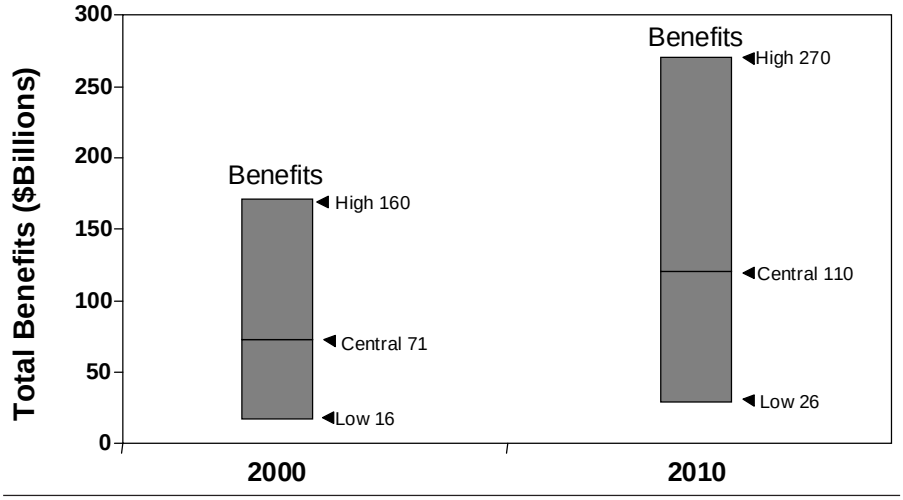
The ecological and welfare results are not currently amenable to the same type of uncertainty analysis. The modeling procedures for estimating the effects of sulfur and nitrogen deposition in acidifying lakes, the effects of ozone in reducing timber and agricultural production, and the effects of particular matter on visibility are all subject to uncertainty and require substantial resources simply to develop single estimates. We describe key uncertainties in Chapter 7 and they are reflected in the ranges of values we present at the end of that chapter. The sources of uncertainty in these estimates, however, cannot as easily be disaggregated among physical effects modeling and valuation components. The endpoints of the ranges we present reflect reasonable alternative choices in key input variables, but the ranges cannot currently be interpreted as points on a statistical distribution of results. For these ecological effects, the central estimate is the midpoint of the ranges of values. We then interpret the endpoints of the range of estimates as the upper and lower bounds of a uniform distribution of values. The uniform distribution is used when we aggregate the ecological and other welfare effects analyses with the analyses of human health.

Annual Benefits Estimates

We present the results of our aggregation of primary annual benefits estimates for Titles I through V in Figure 8-1 below. The figure provides a characterization of both the primary central estimate and the range of values generated by the aggregation procedure described above, for each of the two target years of the analysis (2000 and 2010). The Primary High estimate corresponds to the 95th percentile value from the aggregation, and the Primary Low estimate corresponds to the 5th percentile value. The total benefits estimates are substantial; the Primary Central estimate in 2010 is $110 billion.

Table 8-1 shows the detailed breakdown of benefits estimates for one of the two target years, 2010. As shown in the table, $100 billion of the $110 billion total benefit estimate in 2010, or roughly 90 percent, is attributable to reductions in premature mortality associated with reductions in ambient particulate matter and associated criteria pollutants. The remaining benefits are divided among two broad categories of benefits: avoided morbidity, the largest component of which is avoided chronic bron-

Figure 8-1
Central, Low, and High Primary Benefits Results for Target Years (in billions of 1990 dollars) - Titles I through V



[2] This procedure implicitly assumes independence between the specific aggregation simulation draws from the distribution of health and economic valuation estimates.

Table 8-1
Criteria Pollutant Health and Welfare Benefits in 2010

	Monetary Benefits (in millions 1990$)*		
Benefits Category	**Primary Low**	**Primary Central**	**Primary High**
Mortality			
Ages 30+	14,000	100,000	250,000
Chronic Illness			
Chronic Bronchitis	360	5,600	18,000
Chronic Asthma	40	180	300
Hospitalization			
All Respiratory	76	130	200
Total Cardiovascular	93	390	960
Asthma-Related ER Visits	0.1	1.0	2.8
Minor Illness			
Acute Bronchitis	0.0	2.1	5.2
URS	4.2	19	39
LRS	2.2	6.2	12
Respiratory Illness	0.9	6.3	15
Mod/Worse Asthma[1]	1.9	13	29
Asthma Attacks[1]	20	55	100
Chest Tightness, Shortness of Breath, or Wheeze	0.0	0.6	3.1
Shortness of Breath	0.0	0.5	1.2
Work Loss Days	300	340	380
MRAD/Any-of-19	680	1,200	1,800
Welfare			
Decreased Worker Productivity	710	710	710
Visibility - Recreational	2,500	2,900	3,300
Agriculture (Net Surplus)	7.1	550	1,100
Acidification	12	50	76
Commercial Timber	180	600	1,000
Aggregate Range of Benefits[2]	26,000	110,000	270,000

Note:
* The estimates reflect air quality results for the entire population in the US.
[1] Moderate to worse asthma, asthma attacks, and shortness of breath are endpoints included in the definition of MRAD/Any of 19 respiratory effects. Although valuation estimates are presented for these categories, the values are not included in total benefits to avoid the potential for double-counting.
[2] The Aggregate Range reflects the 5th, mean, and 95th percentile of the estimated credible range of monetary benefits based on quantified uncertainty, as discussed in the text.

Table 8-2
Present Value of Monetized Benefits for 48 State Population

	Present Value (millions 1990$, discounted to 1990 at 5 percent)		
	Primary Low	Primary Central	Primary High
Titles I through V (1990 through 2010)	$160,000	$690,000	$1,600,000
Title VI (1990 through 2165)	$100,000	$530,000	$900,000

chitis, comprises about 60 percent of the non-mortality benefits; and avoided ecological and other welfare effects, the largest component of which is improved recreational visibility, comprises about 40 percent. Note that, because of the aggregation procedure used, and because we round all intermediate results to two significant digits for presentation purposes, the columns of Table 8-1 may not sum to the total estimate presented in the last row.[3]

Aggregate Monetized Benefits

As discussed earlier in this chapter, we linearly interpolate benefit estimates between 1990 and 2000 and between 2000 and 2010 and then aggregate the resulting annual estimates across the entire 1990 to 2010 period of the study to yield a present discounted value of total aggregate benefits for the period. In this section we discuss issues involved in each stage of aggregation, as well as the results of the aggregation.

As noted earlier, air quality modeling was carried out only for the two target years (2000 and 2010). The resulting annual benefit estimates provide a temporal trend of monetized benefits across the period resulting from the annual changes in air quality. They do not, however, characterize the uncertainty associated with the yearly estimates for intervening years. In an attempt to capture uncertainty associated with these estimates, we relied on the ratios of the 5th percentile to the mean and the 95th percentile to the mean in the two target years. In general, these ratios were fairly constant across the target years, for a given endpoint. The ratios were interpolated between the target years, yielding ratios for the intervening years. Multiplying the ratios for each intervening year by the central estimate generated for that year provided estimates of the 5th and 95th percentiles, which we use to characterize uncertainty about the Primary Central estimate.

In Table 8-2 we present the cumulative monetized benefits aggregated from 1990 to 2010. We present the mean estimate from the aggregation procedure, along with the Primary Low (i.e., 5th percentile of the distribution) and Primary High (i.e., 95th percentile of the distribution) estimates, for all provisions of Titles I through V and, then, separately for Title VI. Aggregating the stream of monetized benefits across years involved discounting the stream of monetized benefits estimated for each year to the 1990 present value (using a five percent discount rate).

Aggregate Benefits of Title VI Provisions

As described in summary form in Chapters 5, 6, and 7 and in detail in Appendix G, expected human health benefits from Title VI provisions are substantial. The analysis we conducted is based largely on existing results from EPA Regulatory Impact Analyses for individual rules promulgated under Title VI. To the extent possible, we adjusted existing estimates to reflect both the central estimates and uncertainty characterizations used in the criteria pollutant analysis. We made major adjustments for both the value of statistical life (VSL) and the discount rate. We adjusted the VSL estimate to reflect the Weibull distribution of VSL used in our analysis for other provisions. As discussed in the appendix, the choice of the discount rate for estimated benefits which accrue over decades to century-long time spans presents special problems. Although we argue that a two percent discount rate is more appropriate for such long-term discounting, for consistency in this chapter we present estimates using the five percent discount rate used throughout the rest of this study.

[3] The sum of benefits across endpoints at a given percentile level does not result in the total monetized benefits estimate at the same percentile level in Table 8-1. For example, if the fifth percentile benefits of the endpoints shown in Table 8-1 were added, the resulting total would be substantially less than $30 billion, the fifth percentile value of the distribution of aggregate monetized benefits reported in Table 8-1. This is because the various health and welfare effects are treated as stochastically independent, so that the probability that the aggregate monetized benefit is less than or equal to the sum of the separate five percentile values is substantially less than five percent.

The results of the benefits calculations in Appendix G indicate a cumulative central benefit estimate of $530 billion for Title VI (see Appendix G for details). Using the same aggregation techniques for the valuation analysis described above, but only for the mortality valuation step, we generate a 90 percent confidence interval around this central estimate to derive Primary Low and Primary High estimates of $100 billion to $900 billion, respectively. We present these estimates in Table 8-2 above. The annual human health benefits from Title VI provisions steadily increase until about 2045, then decrease until 2165, the last year in the analysis. About 93 percent of the benefits accrue from 2015 to 2165. These benefit estimates only partially reflect potential averting behaviors, such as remaining indoors or increasing use of sun screens or hats, which may mitigate the effects of the UV-b exposure increases estimated under the Pre-CAAA scenario.

Comparison of Monetized Benefits and Costs

Table 8-3 presents summary quantitative results for the prospective assessment, with costs disaggregated by Title and benefits disaggregated by major category. We present annual, primary estimate results for each of the two target years of the analysis, with all dollar figures expressed as inflation-adjusted 1990 dollars. The final columns provide net present value estimates for costs and benefits from 1990 to 2010 or, in the case of stratospheric ozone protection provisions, 1990 to 2165, discounted to 1990 at five percent. The results indicate that the Primary Central estimate of benefits clearly exceeds the costs of the CAAA, for each of the two target years and for the cumulative estimates of present value over the 1990 to 2010 period.

The estimates in Table 8-3 reflect the difficulty we encountered in reliably disaggregating benefits by CAAA Title or even by pollutant. As the table indicates, a very high percentage of the benefits is attributable to reduced premature mortality associated with reductions in ambient particulate matter and associated criteria pollutants. The CAAA achieves ambient PM reductions through a wide range of provisions controlling emissions of both gaseous precursors of PM that form particles in the atmosphere (sulfur and nitrogen oxides as well as, to a lesser extent, organic constituents) and directly emitted PM (i.e., dust particles). Because the effects of these constituents on ambient PM are nonlinear, and because some precursor pollutants interact with each other in ways which influence the total concentration of particulates in the atmosphere, separating the effects of individual pollutants on the change in ambient PM would require many iterations of our air quality modeling system. These difficulties in separating the effects of individual emissions reductions on the benefits estimates also highlight the need for an integrated air quality modeling system that can more readily analyze multiple scenarios within reasonable time and resource constraints. A tool of this nature could allow us to more reliably and cost-effectively estimate incremental contributions to ambient PM and ozone concentration reductions.

Table 8-3
Summary of Quantified Primary Central Estimate Benefits and Costs (Estimates in million 1990$)

Cost or Benefit Category	Annual Estimates		Present Value
	2000	2010	
Costs:			
Title I	$8,600	$14,500	$85,000
Title II	$7,400	$9,000	$65,000
Title III	$780	$840	$6,600
Title IV	$2,300	$2,000	$18,000
Title V	$300	$300	$2,500
Total Costs, Title I-V	$19,000	$27,000	$180,000
Title VI	$1,400*		$27,000*
Monetized Benefits:			
Avoided Mortality	$63,000	$100,000	$610,000
Avoided Morbidity	$5,100	$7,900	$49,000
Ecological and Welfare Effects	$3,000	$4,800	$29,000
Total Benefits, Title I-V	$71,000	$110,000	$690,000
Stratospheric Ozone	$25,000*		$530,000*

* Annual estimates for Title VI stratospheric ozone protection provisions are annualized equivalents of the net present value of costs over 1990 to 2075 (for costs) or 1990 to 2165 (for benefits). The difference in time scales for costs and benefits reflects the persistence of ozone depleting substances in the atmosphere, the slow processes of ozone formation and depletion, and the accumulation of physical effects in response to elevated UV-b radiation levels.

Table 8-4 provides the results of our comparison of primary benefits estimates to primary cost estimates. In the top half of the table we show both annual and present value estimates for Titles I through V, present value estimates for Title VI, and a total present value for all titles. The "monetized benefits" indicate both the Primary Central estimate (the mean) from our statistical aggregation modeling analysis and the Primary Low and Primary High estimates (5th and 95th percentile values, respectively). In the bottom half of the table we present two alternative methods for comparing benefits to costs. "Net benefits" are the Primary Central estimates of monetized benefits less the Primary Central estimates of costs. The table also notes the benefit/cost ratios implied by the benefit ranges.

The conclusion we draw from Table 8-4 is that, given the particular data, models and assumptions we believe are most appropriate at this time, our analysis indicates that the benefits of the CAAA substantially exceed its costs. Furthermore, the results of the uncertainty analysis imply that it is extremely unlikely that the monetized benefits of the CAAA over the 1990 to 2010 period could be less than its costs. Looking at Titles I through V, the central benefits estimate exceeds costs by a factor of four to one, whether we are looking at annual or present value measures, and the high estimate exceeds costs by more than twice that factor (a ratio of nine or ten to one). Using the Primary Low estimate of benefits, the annual estimates of benefits in 2000 and 2010 are slightly less than the annual costs for that year. The data also suggest that costs for criteria

Table 8-4
Summary Comparison of Benefits and Costs (Estimates in millions 1990$)

	Titles I through V			Title VI	All Titles
	Annual Estimates		Present Value Estimate	Present Value Estimate	
	2000	2010	1990-2010	1990-2165	Total Present Value
Monetized Direct Costs:					
Low [a]			*Not Estimated*		
Central	$19,000	$27,000	$180,000	$27,000	$210,000
High [a]			*Not Estimated*		
Monetized Direct Benefits:					
Low[b]	$16,000	$26,000	$160,000	$100,000	$260,000
Central	$71,000	$110,000	$690,000	$530,000	$1,200,000
High[b]	$160,000	$270,000	$1,600,000	$900,000	$2,500,000
Net Benefits:					
Low	($3,000)	($1,000)	($20,000)	$73,000	$50,000
Central	$52,000	$93,000	$510,000	$500,000	$1,000,000
High	$140,000	$240,000	$1,400,000	$870,000	$2,300,000
Benefit/Cost Ratio:					
Low[c]	less than 1/1	less than 1/1	less than 1/1	less than 4/1	1/1
Central	4/1	4/1	4/1	20/1	6/1
High[c]	more than 8/1	more than 10/1	more than 9/1	more than 33/1	12/1

[a] The cost estimates for this analysis are based on assumptions about future changes in factors such as consumption patterns, input costs, and technological innovation. We recognize that these assumptions introduce significant uncertainty into the cost results; however the degree of uncertainty or bias associated with many of the key factors cannot be reliably quantified. Thus, we are unable to present specific low and high cost estimates.

[b] Low and high benefits estimates are based on primary results and correspond to 5th and 95th percentile results from statistical uncertainty analysis, incorporating uncertainties in physical effects and valuation steps of benefits analysis. Other significant sources of uncertainty not reflected include the value of unquantified or unmonetized benefits that are not captured in the primary estimates and uncertainties in emissions and air quality modeling.

[c] The low benefit/cost ratio reflects the ratio of the low benefits estimate to the central costs estimate, while the high ratio reflects the ratio of the high benefits estimate to the central costs estimate. Because we were unable to reliably quantify the uncertainty in cost estimates, we present the low estimate as "less than X," and the high estimate as "more than Y", where X and Y are the low and high benefit/cost ratios, respectively.

pollutant programs grow somewhat more rapidly than benefits from 1990 to 2000, but that benefits grow more rapidly from 2000 to 2010.

The estimates for Title VI indicate that benefits well exceed costs, even at the low benefits estimate. This conclusion holds despite the relatively high discount rate used for the estimates in Table 8-4 (5 percent) a value that most analysts would consider too high for the long time period over which benefits of this program are discounted (175 years).[4] The total estimates for all titles of the CAAA also indicate benefits in excess of costs for the full range of primary benefits.

Cost-Effectiveness Evaluation

The approach to premature mortality valuation used in our primary estimates is a method that allows us to aggregate the benefits of reducing mortality risks with other monetized benefits of the CAAA. One of the great advantages of the benefit-cost paradigm is that a wide range of quantifiable benefits can be compared to costs to evaluate the economic efficiency of particular actions. Some analysts suggest, however, that presentation of the results of a cost-benefit analysis may mask the key assumptions that are made to quantify all benefits in monetary terms. Another evaluative paradigm, cost-effectiveness analysis, is sometimes suggested as further evidence of whether the benefits of a regulatory program justify its costs. Cost-effectiveness analysis involves estimation of the costs per unit of benefit (e.g., lives saved). This type of analysis is most useful for comparing programs that have similar goals, for example, alternative medical interventions or treatments that can save a life or cure a disease. They are less readily applicable to programs with multiple categories of benefits, such as the CAAA, because the cost-effectiveness calculation is based on quantity of a single benefit category. In other words, we cannot readily convert reductions in new cases of chronic bronchitis, reduced hospital admissions, improvements in visibility, and increased commercial timber and crop yields to a single metric such as "lives saved." For these reasons, we prefer to present our results in terms of monetary benefits.

Despite the risks of oversimplification of benefits, cautiously interpreted cost-effectiveness calculations may provide further evidence of whether the costs incurred to implement the CAAA are a reasonable investment for the nation. The most common cost-effectiveness metric, costs per life saved, can be readily calculated from the information presented in this report. For example, we estimate the total annual direct costs of implementation of Titles I through V in 2010 to be approximately $27 billion. In exchange for this expenditure, in the year 2010 we avoid 23,000 cases of premature mortality and gain estimated non-mortality benefits of about $20 billion. We can generate a net cost per life saved by subtracting from costs the total non-mortality benefits, and then dividing by lives saved. For Titles I through V, we estimate a net cost per life saved of approximately $300,000 ($27 billion minus $20 billion divided by 23,000).[5] Although we are also concerned about many of the uncertain assumptions required to generate cost per life-year saved estimates, we include an estimate for illustrative purposes. For the year 2010, the net cost per life-year saved estimate implied by the primary central case results is $23,000 per life-year ($7 billion divided by 310,000 life-years saved).[6]

Major Sources of Uncertainty

We can obtain additional insights into key assumptions and findings of the present study through further analysis of potentially important variables and inputs. The estimated uncertainty ranges for each endpoint category summarized in Table 8-1 reflect the measured uncertainty associated with two aspects of the analysis: avoided physical effects (both health and welfare benefits) and economic valuation of benefits. In addition, in Chapter 3 we conduct quantitative sensitivity analyses of key components of the direct cost estimates. For many other aspects of our analysis, however, including emissions esti-

[4] The primary central benefit-cost ratio for Title VI using a 3 percent discount rate is 44 to 1, higher than any of those presented in Table 8-4 (see Table 8-6 below). In addition, the ratio using a 2 percent discount rate, the rate used in the underlying RIAs, is 75 to 1. See Appendix G for more detail on the sensitivity of Title VI benefits to the choice of discount rate.

[5] The illustrative calculations presented here do not reflect discounting of the physical incidence of mortality.

[6] Because of Agency concerns regarding discounting of physical effects, the ratio presented here reflects undiscounted life-years saved. If future years were discounted, the implicit cost per life-year saved would be significantly higher.

mates, air quality modeling, and unquantified categories of benefits, we are unable to conduct quantitative analysis of uncertainty. Instead, we have attempted throughout this report to identify and characterize major sources of uncertainty — we present the results of these efforts at the end of Chapters 2 through 7. In this section, we provide a summary evaluation of the relative importance of key sources of uncertainty.

Table 8-5 below provides a summary of both quantified and unquantified sources of uncertainty and our estimates of the impact of these sources of uncertainty on the primary central estimates of benefits and costs. The table covers seven major categories of uncertainties: measurement uncertainties in physical effects and valuation components of the benefits analysis; measurement uncertainties in estimation of direct costs; alternative assumptions for PM-related mortality valuation; alternative assumptions for PM-related mortality risk; unquantified sources of error in emissions and air quality modeling; and omissions of key benefits categories. The table entries cover quantitative analyses of uncertainty, characterization of unquantified uncertainty, and the potential effect of alternative modeling paradigms for costs and benefits. Additional treatment of alternative paradigms is necessary because reasonable people may disagree with our methodological choices regarding these issues, and these choices might be considered to significantly influence the results of the study.

Quantitative Analysis of Physical Effects and Valuation Uncertainties

As discussed previously in this chapter, we have conducted quantitative uncertainty analysis of our benefits estimates to reflect measurement error in two key steps of the analysis: estimation of physical effects and economic valuation. We present the results of our analysis in Figure 8-1 and Table 8-1 above. The procedure used to generate these estimates is well-suited to analysis of uncertainties where the probability of alternative outcomes can be quantitatively characterized in an objective manner. For example, most studies that estimate concentration-response relationships report an estimate of the statistical uncertainty around the central estimate. Because many estimates are available for the value of statistical life, we can use the discrete distribution of the best available estimates as a basis for quantitatively characterizing the probability of alternative values. It is important to recognize, however, that this procedure reflects only a portion of the range of possible sources of uncertainty in our benefits estimates. Other, nonquantified sources of uncertainty must also be factored into conclusions about the ratio of benefits to costs.

As part of our analysis of key contributors to uncertainty in benefits estimates, we also conducted a sensitivity analysis to determine the physical effects estimation and economic valuation variables with the greatest contribution to the quantified measurement uncertainty range. We present the results of this sensitivity analysis in Figure 8-2. In this sen-

Figure 8-2
Analysis of Contribution of Key Parameters to Quantified Uncertainty

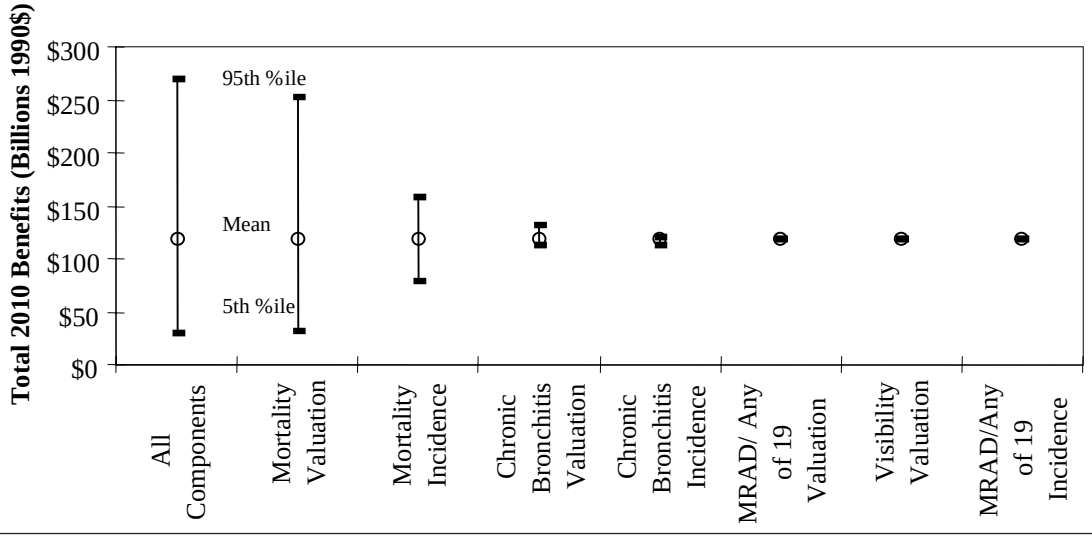


Table 8-5
Summary of Key Sources of Uncertainty and Their Impact on Costs and Benefits

Source of Uncertainty	Description of Alternative Parameter Inputs	Impact on Annual Estimates in 2010	
		Costs	Benefits
Measurement error and uncertainty in the physical effects and economic valuation steps	Use a range of input assumptions to reflect statistical measurement uncertainty in concentration-response functions, modeling of physical effects, and estimation of economic values. Most important input parameters are value of statistical life and estimated relationship between particulate matter and premature mortality (see Chapters 5, 6, and 7).	None	For Titles I through V, effect of the use of alternative input assumptions ranges from a $84 billion decrease (5th percentile) to a $160 billion increase (95th percentile).
Measurement error and uncertainty in direct cost inputs	Use alternative assumptions for key input parameters for six of the highest cost provisions. Conduct sensitivity tests for each provision separately (see Chapter 3, pages 30 to 32). As discussed in Chapter 3 and in this chapter, aggregation of provision-specific results would be inappropriate.	High estimates for some provisions are $1 billion higher than primary estimate. Low estimates are as much as $2 billion below primary estimate	None
Value of statistical life-based estimates do not reflect age at death	Use estimates of the incremental number of life-years lost from exposure to ambient PM and a value of statistical life-year as opposed to measuring number of lives lost and a value of statistical life (see Chapters 5 and 6).	None	Decrease by $47 billion
Basis of estimate of avoided mortality from PM exposure	The Dockery et al. study provides an alternative estimate of the long-term relationship between chronic PM exposure and mortality (see Chapter 5).	None	Increase by $100 to $150 billion
Uncertainties in Title VI health benefits analysis	Major uncertainties include: estimating fatal cancer cases resulting from UV-b exposure; not accounting for future averting behavior; and not accounting for future improvements in the early detection and treatment of melanoma (see Table 5-6).	None	Not quantified, but net effect is probably that benefits estimates are too high.
Uncertainties in emissions and air quality steps	Major uncertainties include: underestimation of $PM_{2.5}$ emissions; omission of changes in primary and organic PM in eastern U.S.; emissions estimation uncertainties in the western U.S.; scarcity of $PM_{2.5}$ monitors; and lack of a fully integrated air quality and emissions modeling system (see Tables 2-5 and 4-7).	Uncertainties in emissions estimates affects some costs, but net effect is minor.	Not quantified, but net effect is probably that benefits estimates are too low.
Omission of potentially important benefits categories from primary estimate	Non-quantified categories of impacts summarized in Chapters 5 and 7. Quantified but omitted categories include household soiling, nitrogen deposition, and residential visibility (see Chapter 7).	None	Increase by at least $8 billion, (does not reflect unquantified categories)

sitivity analysis, we hold constant all inputs to the probabilistic uncertainty analysis except one -- for example, the economic valuation of mortality. We allow that one variable to vary across the estimated range of that variable's uncertainty. The sensitivity analysis isolates the effect of this single source of uncertainty on the total measured uncertainty in estimated aggregate benefits. The first uncertainty bar represents the range associated with the total monetized benefits of the Clean Air Act, based on analysis of quantifiable components of uncertainty, as reported above. This range captures the multiple measurement uncertainties in the quantified benefits estimation. The rest of the uncertainty bars represent the quantified measurement uncertainty ranges generated by single variables. As shown in Figure 8-2, the most important contributors to aggregate quantified measurement uncertainty are mortality valuation and incidence, followed by chronic bronchitis valuation and incidence.

Measurement Error and Uncertainty in Direct Cost Inputs

As noted in Chapter 3, explicit and implicit assumptions about changes in consumption patterns, input costs, and technological innovation are crucial to estimating the direct compliance costs of the CAAA. For many of the factors contributing to uncertainty, the degree and, in some cases, the direction of the bias are unknown or cannot be determined. Uncertainties and sensitivities can be identified, however, and in many cases the potential measurement errors can be quantitatively characterized. We designed our sensitivity analyses of key input parameters to provide a sense of the relative importance of various input parameters and assumptions necessary to generate estimates of direct costs. The sensitivity tests use ranges of input parameters that include all reasonable alternative estimates that we could identify.

The results indicate that the sensitivity of our primary central cost estimates is not uniform across provisions. Low and high estimates may vary by as much as a factor of two. Unlike our quantitative analysis of benefits, we do not assign probabilities to the likelihood of alternative input parameters. In our judgement, assignment of probabilities to these alternative outcomes would be a largely subjective task; we know of no objective means to develop these probabilities. As a result, it would be inappropriate simply to add up the array of low and the array of high estimates to arrive at an overall range of uncertainty around the central estimates, because it is unlikely that a plausible scenario could be constructed where all the estimates are concurrently either at the high or low end of their individual plausible ranges. A better interpretation of these results is that uncertainty in key input parameters can have a significant effect on the overall uncertainty of our estimates of direct compliance costs and ultimately the net benefits calculation.[7]

PM Mortality Valuation Based on Life-Years Lost

The primary analytical results we present earlier in this chapter assign the same economic value to incidences of premature mortality regardless of the age and health status of those affected. Although this has been the traditional practice for benefit-cost studies conducted within EPA, some argue this may not be the most appropriate method for valuation of premature mortality caused by PM exposure. Some short-term PM exposure studies suggest that a significantly disproportionate share of PM-related premature mortality occurs among persons 65 years of age or older. Combining standard life expectancy tables with the limited available data on age-specific incidence allows rough approximations of the number of life-years lost by those who die prematurely as a result of exposure to PM or, alternatively, the changes in life expectancy of those who are exposed to PM.

The ability to estimate, however roughly, changes in age-specific life expectancy raises the issue of whether available measures of the economic value of mortality risk reduction can, and should, be adapted to measure the value of specific numbers

[7] Although the analysis conducted here is a direct cost analysis, other sources of uncertainty would also need to be considered for a social cost analysis. For example, forecasts of key economic variables (e.g., interest rates), specification of production functions, and the reliability of key supply and demand elasticities are all important factors in social cost modeling that contribute to measurement uncertainty. In addition, most current social cost analyses assume that markets are currently operating under optimally efficient conditions. Emerging literature suggests that a full accounting of the social costs and efficiency impacts of environmental regulations could also include an assessment of the incremental costs that reflect existing market distortions, such as those imposed by the current tax code. Our assessment of uncertainties in direct cost estimates do not reflect these considerations.

of life-years saved.[8] As stated in our retrospective analysis, we have on occasion performed sensitivity calculations that adjust mortality values for those over age 65. Nonetheless, as discussed in Appendix H, the current state of knowledge and available analytical tools do not conclusively support using a life-years lost approach or any other approach which assigns different risk reduction values to people of different ages or circumstances. While we prefer an approach which makes no valuation distinctions based on age or other characteristics of the affected population, we present alternative results based on a VSLY approach below. The method used to develop life years lost estimates is described briefly in Chapter 5 and Appendix D. The method used to develop VSLY estimates is described in Appendix H.

The fourth row of Table 8-5 summarizes the effect of using a VSLY approach on results for 2010. The results indicate that the choice of valuation methodology significantly affects the estimate of the monetized value of reductions in air pollution-related premature mortality. However, the downward adjustment which would result from applying a VSLY approach in lieu of a VSL approach does not change the basic conclusion of this study, since the central estimate of monetized benefits of the CAAA still substantially exceeds the costs of compliance.

We emphasize that the results of the VSLY approach to valuing avoided mortality benefits represent a crude estimate of the value of changes in age-specific life expectancy. These results should be interpreted cautiously, due to the several significant assumptions required to generate a monetized estimate of life years lost from the relative risks reported in the Pope et al., 1995 study and the available economic literature. These assumptions include, but are not limited to: extrapolation of the age distribution of the U.S. population in future years; assumptions about the age-specificity of the relative risk reported by Pope et al., 1995; assumptions about the life expectancy of different age groups, adjustment of the life years lost estimates by an appropriate lag period (if any); assumptions about the age-specificity of the lag period (if any); derivation of VSLY estimates from VSL estimates; assumptions about the variation in VSLY with age; and selection of an appropriate rate at which to discount the lagged estimates of life years lost. Changes in any of these assumptions could significantly affect the VSLY benefit estimate. For example, if we were to assume no lag period for PM-related mortality effects instead of the five-year lag structure described in Chapter 5, VSLY benefit estimates would increase from $53 billion to $61 billion. The specific assumptions we used in generating these results are discussed in Appendix H.

PM Mortality Incidence Using the Dockery Study

As described in Chapter 5, we chose to use the results of the Pope et al. (1995) study to estimate the magnitude of the effect of ambient PM exposure on the incidence of premature mortality. Alternative estimates do exist in the literature, however. Although we chose the Pope study because of its coverage of the largest number of cities and other technical advantages, the Dockery et al. (1993) study provides a credible and reasonable alternative to the Pope study. The Dockery study used a smaller sample of individuals in fewer U.S. cities than the Pope study, but it features improved exposure estimates, a slightly broader study population (including adults aged 25 to 30), and a follow-up period nearly twice as long as that used in the Pope study.

Use of the Dockery study in place of the Pope study would substantially increase the benefits estimate. As shown in the fifth row of Table 8-5, we estimate that using the Dockery study estimates would increase the annual central benefits estimate by $100 to $150 billion, more than doubling the total annual benefits for Titles I through V and, in turn, doubling the estimated benefit-cost ratio.

Uncertainties in Title VI Health Benefits Analysis

As discussed in Chapter 5 and Appendix G, health benefits such as avoided mortality from melanoma and non-melanoma skin cancers constitute the majority of monetized benefits resulting from Title

[8] This issue was extensively discussed during the Science Advisory Board Council review of drafts of the retrospective study. The Council suggested it would be reasonable and appropriate to show PM mortality benefit estimates based on value of statistical life-years (VSLY) saved as well as the value of statistical life (VSL) approach traditionally applied by the Agency to all incidences of premature mortality. Consistent with SAB Council review advice for the present study, we apply the same approach in this analysis.

VI regulations on stratospheric ozone-depleting chemicals. Estimates of avoided mortality from skin cancer due to reduced UV-b exposure between 1990 and 2165 represent over 90 percent of the total health benefits of Title VI. As a result, uncertainties related to avoided mortality estimation under Title VI represent key uncertainties for our overall CAAA benefits estimate. Three main areas of uncertainty are important for our avoided mortality estimates for Title VI: dose-response relationships; predicting averting behavior; and predicting future medical advancements.

Because the literature on the relationship between exposure to ultraviolet rays and melanoma and non-melanoma mortality is not as well developed as that for other health effects, the dose-response functions for both of these endpoints are characterized by significant uncertainty. The association of UV-b exposure with melanoma is controversial, although studies suggest that sunlight exposure is a major environmental risk factor for melanoma. If one assumes that a causal relationship exists between UV-b rays and melanoma, uncertainty still remains about three aspects of the nature of the dose-response relationship. Specifically, the relative contribution of different wavelengths of light to melanoma development, the critical exposure period (e.g., acute, intermittent, or chronic), and the existence (and length) of a latency period between UV exposure and disease are all unclear. The effect of the first two uncertainties on our results cannot be determined from available information. If a significant latency period exists, then the third uncertainty may indicate that our analysis, which does not include a latency period, overestimates avoided melanoma mortality benefits. Because limited data on non-melanoma mortality precluded the development of a dose-response function for this endpoint in the current analysis, our estimate of non-melanoma skin cancer mortality resulting from UV-b exposure is calculated indirectly, by assuming the mortality rate is a fixed percentage of non-melanoma incidence. New data on the death rate for non-melanoma skin cancer may significantly influence this mortality estimate.

Our analysis of avoided mortality also does not incorporate adjustments for future increases in averting behavior (i.e., efforts by individuals to protect themselves from UV-b radiation). Our estimates rely on epidemiological studies that incorporate averting behavior as currently practiced. However, if people would react to increased skin cancer risk in the future by applying sun screen more frequently, spending more time indoors or otherwise reducing their UV-b exposure, then our estimate of avoided mortality would significantly overestimate Title VI benefits. It is not certain, though, that individuals will pursue such behavior, and studies show that those engaging in averting behavior may also alter their behavior in ways that may increase exposure or risk, counteracting the benefits of averting behavior. For example, a recent study of young Europeans by Autier et al. (1999) found that the use of high sun protection factor (SPF) sun screen is associated with increased frequency and duration of sun exposure.

Finally, our analysis does not adjust estimates of future mortality for possible advances in medical technology that could lead to earlier detection and more effective treatment of melanomas. Such advancements could significantly reduce the expected future melanoma mortality, and by not adjusting for such developments, we may be overestimating avoided melanoma mortality. However, future research may also identify additional adverse human health outcomes associated with UV exposure that we have not considered in this analysis, resulting in an underestimate of Title VI benefits.

Uncertainties in Emissions and Air Quality Steps

The emissions estimates presented in this analysis are a critical component of the overall analysis. As the starting point for both costs and benefits, they provide a consistent basis for evaluating the economic efficiency of the CAAA. Characterizing emissions can be very difficult, however, particularly for those source categories where emissions monitoring data are sparse or nonexistent. In general, all our emissions estimates are affected by three major sources of uncertainty: estimation of the base-year inventory, prediction of the growth in pollution-generating activity, and assumptions about future-year controls.

Base-year emissions were estimated using emissions factors that express the relationship between a particular human/industrial activity and the level of

emissions. The accuracy of base-year emissions estimates varies from pollutant to pollutant, depending largely on how directly the selected activity and emissions correlate. We likely estimated 1990 SO_2 emissions with the greatest precision. Sulfur dioxide emissions are generated during combustion of sulfur-containing fuel and are directly related to fuel sulfur content. In addition, we were able to verify these estimates through comparison with Continuous Emission Monitoring (CEM) data. As a result, we were able to accurately estimate SO_2 emissions using emissions factors based on data on fuel usage and fuel sulfur content. Nitrogen oxides are also a product of fuel combustion, allowing us to estimate emissions of this pollutant using the same general technique used to estimate SO_2 emissions. However, the processes involved in the formation of NO_x during combustion are more complicated than those involved in the formation of SO_2; thus, our NO_x emissions estimates are more variable and less certain than SO_2 estimates.

Volatile organic compounds, like SO_2 and NO_x, are products of fuel combustion; however, these compounds are also a product of evaporation. To estimate evaporative emissions of this pollutant we used emissions factors that relate changes in emissions to changes in temperature. Because future meteorological conditions are difficult to predict, the uncertainty associated with forecasting temperature influences the uncertainty in our VOC emissions estimates. The likely significance of this uncertainty, in terms of its impact on the overall monetary benefit present in this analysis, is probably minor.

Of particular importance, however, are uncertainties that affect the estimation of future year emissions of particulate matter and secondarily formed PM precursors. In this analysis we estimated primary $PM_{2.5}$ emissions based on unit emissions that may not accurately reflect the composition and mobility of particles. The ratio of crustal to carbonaceous particulate material, for example, likely is high as a result of overestimation of the fraction of crustal material, primarily composed of fugitive dust, and underestimation of the fraction of carbonaceous material. Because the CAAA have a greater impact on emissions sources that generate carbonaceous particles (mobile sources) than on sources that mainly emit crustal material (area sources), we likely underestimate the impact of the CAAA on reducing $PM_{2.5}$, thereby reducing monetary benefits estimates. The uncertainty associated with estimating the partition of $PM_{2.5}$ emissions components could conceivably have a major impact on the net benefit estimate. Compared to secondary $PM_{2.5}$ precursor emissions, however, changes in primary $PM_{2.5}$ emissions have a relatively small impact on $PM_{2.5}$ related benefits.

Our future-year control assumptions are also a source of uncertainty. Despite our efforts to minimize this uncertainty, whether each of the Post-CAAA controls will be adopted, whether Post-CAAA control programs will be more or less effective than estimated, and whether unanticipated technological shifts will reduce future-year emissions are all unknown. For example, the Post-CAAA scenario includes implementation of a region-wide NO_x control strategy designed to regulate the regional transport of ozone. However, the control program assumed under the Post-CAAA scenario may not reflect the NO_x controls that are actually implemented in a regional ozone transport rule.

In addition to potential inaccuracies in the emissions inventories used as air quality modeling inputs, there are at least three sources of air quality modeling uncertainty that may have a major effect on the precision and accuracy of our projected changes in air quality. First, we estimate changes in PM concentrations in the eastern U.S. based exclusively on changes in the concentrations of sulfate and nitrate particles. By not accounting for changes in organic and primary particulate fractions, we likely underestimate the impact of the CAAA on PM concentrations. Second, by using separate air quality models for individual pollutants and different geographic regions, as opposed to a single integrated model, we were unable to fully capture the interaction among air pollutants or reflect transport of pollutants or precursors across the boundaries of the models covering the western and eastern states. Third, the lack of a well-developed modeling network for $PM_{2.5}$ means we must estimate monitored concentrations of this pollutant based on PM_{10} monitor estimates. The direction and magnitude of bias these limitations impose on net benefits estimate presented in this analysis can not be determined based on current information.

Some model-related uncertainties, however, may be mitigated because this analysis uses the air qual-

ity modeling results in a relative, not absolute, sense. We focus on the change in air quality between the Pre- and Post-CAAA scenarios and not on the ambient concentrations projected by the individual models themselves. Therefore, uncertainties that affect a model's ability to accurately predict the relative change in concentration of a pollutant from one scenario to another are more important in the context of this study than those that affect only the absolute model results. In addition, as summarized in the previous chapters, most of the uncertainties in emissions estimation and air quality modeling contribute to a conservative bias in our benefits results. When faced with alternative approaches to emissions and air quality modeling, we made explicit attempts to choose parameters, assumptions and modeling strategies that would tend to understate benefits.

Omission of Potentially Important Benefits Categories

As described in Chapters 5 through 7 above, and in more detail in Appendix H, the primary estimate reflects application of a strict set of criteria for inclusion of monetized benefits categories. For example, estimates of the value of improved visibility in U.S. residential areas indicate a positive value for this service flow, but the best available residential visibility estimates rely on an unpublished study of values in the eastern U.S. Although our physical effects analysis indicates significant visibility improvements in all regions of the U.S., our application of the results of the economic valuation literature reflect a conservative approach to valuation of improved visibility in the U.S. While we believe our conservative inclusion criteria for the primary benefits reflects the greater uncertainty in measuring some economic values, we also believe that the statutory language of section 812 clearly warns against the practice of assuming a default value of zero for demonstrated categories of benefits. Therefore, the last row of Table 8-5 presents the effect of using a somewhat more inclusive set of criteria for accepting benefits transfer-based economic values. In this alternative case, we included estimates for improved residential visibility, displaced costs from reduced airborne nitrogen loadings to estuaries, and reduced expenditures for household soiling (which are not included in any form in the primary estimate).

In addition to these quantified but omitted categories of benefits, there is a wide range of benefits of the CAAA that we can identify but cannot quantify. We present summaries of unquantified health effects in Chapter 5 (Tables 5-1 and 5-5) and unquantified ecological and welfare effects in Chapter 7 (Tables 7-5 and 7-9). Two of the most important omissions, in our judgement, are the lack of any quantified estimates for the health benefits of air toxics control and the omission of the systemic and long-term ecological effects of mercury and other persistent air pollutants. The importance of these two categories of effects are discussed in Chapters 5 and 7, respectively.

Alternative Discount Rates

In some instances, the choice of discount rate can have an important effect on the results of a benefit-cost analysis; for example, when the distribution of costs and benefits throughout the time period are very different from one another. In this assessment, the discount rate affects annualized costs (i.e., amortized capital expenditures), and the discounting of all costs and benefits to 1990. Table 8-6 summarizes the effect of alternative discount rates on the Primary Central estimate results of this analysis. The estimates we present show that altering the discount rate has only a small effect on annual cost and benefit estimates. In part, this is due to limitations in our ability to conclusively identify costs as annualized capital expenditures or annual operating costs in the underlying estimates. As described in Chapter 3, about $3 billion (or roughly 10 percent) of the 2010 estimate is annualized capital costs. Varying the discount rate, which we also use to represent the cost of capital, affects only this component of costs. The benefits estimates that employ a discount rate include the mortality estimate, where it is used as part of our valuation of the lag effect of PM mortality, and the chronic asthma value, where we use a discount rate to develop a lump-sum value for avoidance of incidence from an annual payment value in the underlying literature.

Not surprisingly, the effect of discount rates on the net present value benefit calculations is greater. Nonetheless, the estimates we present in Table 8-6 show that varying the discount rate assumption also does not change our overall conclusion that the benefits of the CAAA exceed its costs.

Table 8-6

Effect of Alternative Discount Rates on Primary Central Estimates (Estimates in million 1990$)

	Discount Rate Assumption		
	3%	**5%**	**7%**
Annual Costs in 2010:			
Titles I through V	$26,600	$26,800	$26,900
Annual Benefits:			
Titles I through V	$110,000	$110,000	$107,000
Present Value of Costs:			
Titles I through V	$230,000	$180,000	$140,000
Title VI	$43,000	$27,000	$20,000
Present Value of Benefits:			
Titles I through V	$890,000	$690,000	$520,000
Title VI	$1,900,000	$530,000	$240,000
Cumulative Net Benefits:			
Titles I through V	$650,000	$510,000	$380,000
Title VI	$1,860,000	$500,000	$220,000
Benefit/Cost Ratio:			
Titles I through V	4/1	4/1	4/1
Title VI	44/1	20/1	12/1