Chapter 8:   Health and Environmental Impacts

8.1 Health and Environmental Effects of Non-GHG Pollutants

8.1.1 Health Effects Associated with Exposure to Non-GHG Pollutants

In this section we will discuss the health effects associated with non-GHG pollutants, specifically: particulate matter, ozone, nitrogen oxides (NO_X), sulfur oxides (SO_X), carbon monoxide and air toxics. These pollutants would not be directly regulated by the standards, but the standards would affect emissions of these pollutants and precursors. Reductions in these pollutants would be co-benefits of the final rule (that is, benefits in addition to the benefits of reduced GHGs).

8.1.1.1 Background on Particulate Matter

Particulate matter (PM) is a generic term for a broad class of chemically and physically diverse substances. It can be principally characterized as discrete particles that exist in the condensed (liquid or solid) phase spanning several orders of magnitude in size. Since 1987, EPA has delineated that subset of inhalable particles small enough to penetrate to the thoracic region (including the tracheobronchial and alveolar regions) of the respiratory tract (referred to as thoracic particles). Current National Ambient Air Quality Standards (NAAQS) use $PM_{2.5}$ as the indicator for fine particles (with $PM_{2.5}$ referring to particles with a nominal mean aerodynamic diameter less than or equal to 2.5 µm), and use PM_{10} as the indicator for purposes of regulating the coarse fraction of PM_{10} (referred to as thoracic coarse particles or coarse-fraction particles; generally including particles with a nominal mean aerodynamic diameter greater than 2.5 µm and less than or equal to 10 µm, or $PM_{10-2.5}$). Ultrafine particles (UFPs) are a subset of fine particles, generally less than 100 nanometers (0.1 µm) in aerodynamic diameter.

Particles span many sizes and shapes and consist of numerous different chemicals. Particles originate from sources and are also formed through atmospheric chemical reactions; the former are often referred to as "primary" particles, and the latter as "secondary" particles. In addition, there are also physical, non-chemical reaction mechanisms that contribute to secondary particles. Particle pollution also varies by time of year and location and is affected by several weather-related factors, such as temperature, clouds, humidity, and wind. A further layer of complexity comes from a particle's ability to shift between solid/liquid and gaseous phases, which is influenced by concentration, meteorology, and temperature.

Fine particles are produced primarily by combustion processes and by transformations of gaseous emissions (e.g., SO_X, NO_X and VOCs) in the atmosphere. The chemical and physical properties of $PM_{2.5}$ may vary greatly with time, region, meteorology and source category. Thus, $PM_{2.5}$ may include a complex mixture of different chemicals including sulfates, nitrates, organic compounds, elemental carbon and metal compounds. These particles can remain in the atmosphere for days to weeks and travel through the atmosphere hundreds to thousands of kilometers.[1]

8.1.1.2 Particulate Matter Health Effects

This section provides a summary of the health effects associated with exposure to ambient concentrations of PM.[A] The information in this section is based on the information and conclusions in the Integrated Science Assessment (ISA) for Particulate Matter (December 2009) prepared by EPA's Office of Research and Development (ORD).[B]

The ISA concludes that ambient concentrations of PM are associated with a number of adverse health effects.[C] The ISA characterizes the weight of evidence for different health effects associated with three PM size ranges: $PM_{2.5}$, $PM_{10-2.5}$, and UFPs. The discussion below highlights the ISA's conclusions pertaining to these three size fractions of PM, considering variations in both short-term and long-term exposure periods.

8.1.1.2.1   Effects Associated with Short-term Exposure to $PM_{2.5}$

The ISA concludes that cardiovascular effects and all-cause cardiovascular- and respiratory-related mortality are causally associated with short-term exposure to $PM_{2.5}$.[2] It also concludes that respiratory effects are likely to be causally associated with short-term exposure to $PM_{2.5}$, including respiratory emergency department (ED) visits and hospital admissions for chronic obstructive pulmonary disease (COPD), respiratory infections, and asthma; and exacerbation of respiratory symptoms in asthmatic children.

8.1.1.2.2   Effects Associated with Long-term Exposure to $PM_{2.5}$

The ISA concludes that there are causal associations between long-term exposure to $PM_{2.5}$ and cardiovascular effects, such as the development/progression of cardiovascular disease (CVD), and premature mortality, particularly from cardiopulmonary causes.[3] It also concludes that long-term exposure to $PM_{2.5}$ is likely to be causally associated with respiratory effects, such as reduced lung function growth, increased respiratory symptoms, and asthma development. The ISA characterizes the evidence as suggestive of a causal relationship for associations between long-term $PM_{2.5}$ exposure and reproductive and developmental outcomes, such as low birth weight and infant mortality. It also characterizes the evidence as suggestive of a causal relationship between $PM_{2.5}$ and cancer incidence, mutagenicity, and genotoxicity.

[A] Personal exposure includes contributions from many different types of particles, from many sources, and in many different environments. Total personal exposure to PM includes both ambient and nonambient components; and both components may contribute to adverse health effects.
[B] The ISA is available at http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546
[C] The ISA evaluates the health evidence associated with different health effects, assigning one of five "weight of evidence" determinations: causal relationship, likely to be a causal relationship, suggestive of a causal relationship, inadequate to infer a causal relationship, and not likely to be a causal relationship. For definitions of these levels of evidence, please refer to Section 1.5 of the ISA.

8.1.1.2.3 Effects Associated with $PM_{10-2.5}$

The ISA summarizes evidence related to short-term exposure to $PM_{10-2.5}$. $PM_{10-2.5}$ is the fraction of PM_{10} particles that is larger than $PM_{2.5}$.[4] The ISA concludes that available evidence is suggestive of a causal relationship between short-term exposures to $PM_{10-2.5}$ and cardiovascular effects, such as hospitalizations for ischemic heart disease. It also concludes that the available evidence is suggestive of a causal relationship between short-term exposures to $PM_{10-2.5}$ and respiratory effects, including respiratory-related ED visits and hospitalizations and pulmonary inflammation. The ISA also concludes that the available literature suggests a causal relationship between short-term exposures to $PM_{10-2.5}$ and mortality. Data are inadequate to draw conclusions regarding health effects associated with long-term exposure to $PM_{10-2.5}$.[5]

8.1.1.2.4 Effects Associated with Ultrafine Particles

The ISA concludes that the evidence is suggestive of a causal relationship between short-term exposures to UFPs and cardiovascular effects, including changes in heart rhythm and vasomotor function (the ability of blood vessels to expand and contract).[6]

The ISA also concludes that there is suggestive evidence of a causal relationship between short-term UFP exposure and respiratory effects. The types of respiratory effects examined in epidemiologic studies include respiratory symptoms and asthma hospital admissions, the results of which are not entirely consistent. There is evidence from toxicological and controlled human exposure studies that exposure to UFPs may increase lung inflammation and produce small asymptomatic changes in lung function. Data are inadequate to draw conclusions regarding health effects associated with long-term exposure to UFPs.[7]

8.1.1.3 Background on Ozone

Ground-level ozone pollution is typically formed by the reaction of VOCs and NO_X in the lower atmosphere in the presence of sunlight. These pollutants, often referred to as ozone precursors, are emitted by many types of pollution sources such as highway and nonroad motor vehicles and engines, power plants, chemical plants, refineries, makers of consumer and commercial products, industrial facilities, and smaller area sources.[D]

The science of ozone formation, transport, and accumulation is complex. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions, many of which are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up and result in more ozone than typically occurs on a single high-temperature day. Ozone can be transported hundreds of miles downwind of precursor emissions, resulting in elevated ozone levels even in areas with low VOC or NO_X emissions.

[D] Area sources are defined as sources that emit less than 10 tons per year of a criteria or hazardous air pollutant or less than 25 tons per year of a combination of pollutants.

The highest levels of ozone are produced when both VOC and NO_X emissions are present in significant quantities on clear summer days. Relatively small amounts of NO_X enable ozone to form rapidly when VOC levels are relatively high, but ozone production is quickly limited by removal of the NO_X. Under these conditions NO_X reductions are highly effective in reducing ozone while VOC reductions have little effect. Such conditions are called "NO_X-limited." Because the contribution of VOC emissions from biogenic (natural) sources to local ambient ozone concentrations can be significant, even some areas where man-made VOC emissions are relatively low can be NO_X-limited.

Ozone concentrations in an area also can be lowered by the reaction of nitric oxide (NO) with ozone, forming nitrogen dioxide (NO_2); as the air moves downwind and the cycle continues, the NO_2 forms additional ozone. The importance of this reaction depends, in part, on the relative concentrations of NO_X, VOC, and ozone, all of which change with time and location. When NO_X levels are relatively high and VOC levels relatively low, NO_X forms inorganic nitrates (i.e., particles) but relatively little ozone. Such conditions are called "VOC-limited." Under these conditions, VOC reductions are effective in reducing ozone, but NO_X reductions can actually increase local ozone under certain circumstances. Even in VOC-limited urban areas, NO_X reductions are not expected to increase ozone levels if the NO_X reductions are sufficiently large. Rural areas are usually NO_X-limited, due to the relatively large amounts of biogenic VOC emissions in such areas. Urban areas can be either VOC- or NO_X-limited, or a mixture of both, in which ozone levels exhibit moderate sensitivity to changes in either pollutant.

8.1.1.4 Ozone Health Effects

Exposure to ambient ozone contributes to a wide range of adverse health effects.[E] These health effects are well documented and are critically assessed in the EPA ozone air quality criteria document (ozone AQCD) and EPA staff paper.[8,9] We are relying on the data and conclusions in the ozone AQCD and staff paper, regarding the health effects associated with ozone exposure.

Ozone-related health effects include lung function decrements, respiratory symptoms, aggravation of asthma, increased hospital and emergency room visits, increased asthma medication usage, and a variety of other respiratory effects. Cellular-level effects, such as inflammation of lungs, have been documented as well. In addition, there is suggestive evidence of a contribution of ozone to cardiovascular-related morbidity and highly suggestive evidence that short-term ozone exposure directly or indirectly contributes to non-accidental and cardiopulmonary-related mortality, but additional research is needed to clarify the underlying mechanisms causing these effects. In a recent report on the estimation of ozone-related premature mortality published by the National Research Council (NRC), a panel of experts and

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[E] Human exposure to ozone varies over time due to changes in ambient ozone concentration and because people move between locations which have notable different ozone concentrations. Also, the amount of ozone delivered to the lung is not only influenced by the ambient concentrations but also by the individuals breathing route and rate.

reviewers concluded that short-term exposure to ambient ozone is likely to contribute to premature deaths and that ozone-related mortality should be included in estimates of the health benefits of reducing ozone exposure.[10] People who appear to be more susceptible to effects associated with exposure to ozone include children, asthmatics and the elderly. Those with greater exposures to ozone, for instance due to time spent outdoors (e.g., children and outdoor workers), are also of concern.

Based on a large number of scientific studies, EPA has identified several key health effects associated with exposure to levels of ozone found today in many areas of the country. Short-term (1 to 3 hours) and prolonged exposures (6 to 8 hours) to ambient ozone concentrations have been linked to lung function decrements, respiratory symptoms, increased hospital admissions and emergency room visits for respiratory problems.[11, 12, 13, 14, 15, 16] Repeated exposure to ozone can increase susceptibility to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.[17, 18, 19, 20, 21] Repeated exposure to sufficient concentrations of ozone can also cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure, which over time could affect premature aging of the lungs and/or the development of chronic respiratory illnesses, such as emphysema and chronic bronchitis.[22, 23, 24, 25]

Children and adults who are outdoors and active during the summer months, such as construction workers, are among those most at risk of elevated ozone exposures.[26] Children and outdoor workers tend to have higher ozone exposure because they typically are active outside, working, playing and exercising, during times of day and seasons (e.g., the summer) when ozone levels are highest.[27] For example, summer camp studies in the Eastern United States and Southeastern Canada have reported statistically significant reductions in lung function in children who are active outdoors.[28, 29, 30, 31, 32, 33, 34, 35] Further, children are more at risk of experiencing health effects from ozone exposure than adults because their respiratory systems are still developing. These individuals (as well as people with respiratory illnesses, such as asthma, especially asthmatic children) can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during prolonged periods of moderate exertion.[36, 37, 38, 39]

8.1.1.5 Background on Nitrogen Oxides and Sulfur Oxides

Sulfur dioxide (SO_2), a member of the sulfur oxide (SO_X) family of gases, is formed from burning fuels containing sulfur (e.g., coal or oil), extracting gasoline from oil, or extracting metals from ore. Nitrogen dioxide (NO_2) is a member of the nitrogen oxide (NO_X) family of gases. Most NO_2 is formed in the air through the oxidation of nitric oxide (NO) emitted when fuel is burned at a high temperature. SO_2 and NO_2 can dissolve in water droplets and further oxidize to form sulfuric and nitric acid which react with ammonia to form sulfates and nitrates, both of which are important components of ambient PM. The health effects of ambient PM are discussed in Section 8.1.1.2. NO_X along with non-methane hydrocarbons (NMHC) are the two major precursors of ozone. The health effects of ozone are covered in Section 8.1.1.4.

8.1.1.6 Health Effects of SO_2

This section provides an overview of the health effects associated with SO_2. Additional information on the health effects of SO_2 can be found in the EPA Integrated Science Assessment for Sulfur Oxides.[40] Following an extensive evaluation of health evidence from epidemiologic and laboratory studies, the U.S. EPA has concluded that there is a causal relationship between respiratory health effects and short-term exposure to SO_2. The immediate effect of SO_2 on the respiratory system in humans is bronchoconstriction. Asthmatics are more sensitive to the effects of SO_2 likely resulting from preexisting inflammation associated with this disease. In laboratory studies involving controlled human exposures to SO_2, respiratory effects have consistently been observed following 5-10 min exposures at SO_2 concentrations ≥ 0.4 ppm in asthmatics engaged in moderate to heavy levels of exercise, with more limited evidence of respiratory effects among exercising asthmatics exposed to concentrations as low as 0.2-0.3 ppm. A clear concentration-response relationship has been demonstrated in these studies following exposures to SO_2 at concentrations between 0.2 and 1.0 ppm, both in terms of increasing severity of respiratory symptoms and decrements in lung function, as well as the percentage of asthmatics adversely affected.

In epidemiologic studies, respiratory effects have been observed in areas where the mean 24-hour SO_2 levels range from 1 to 30 ppb, with maximum 1 to 24-hour average SO_2 values ranging from 12 to 75 ppb. Important new multicity studies and several other studies have found an association between 24-hour average ambient SO_2 concentrations and respiratory symptoms in children, particularly those with asthma. Generally consistent associations also have been observed between ambient SO_2 concentrations and emergency department visits and hospitalizations for all respiratory causes, particularly among children and older adults (≥ 65 years), and for asthma. A limited subset of epidemiologic studies have examined potential confounding by copollutants using multipollutant regression models. These analyses indicate that although copollutant adjustment has varying degrees of influence on the SO_2 effect estimates, the effect of SO_2 on respiratory health outcomes appears to be generally robust and independent of the effects of gaseous and particulate copollutants, suggesting that the observed effects of SO_2 on respiratory endpoints occur independent of the effects of other ambient air pollutants.

Consistent associations between short-term exposure to SO_2 and mortality have been observed in epidemiologic studies, with larger effect estimates reported for respiratory mortality than for cardiovascular mortality. While this finding is consistent with the demonstrated effects of SO_2 on respiratory morbidity, uncertainty remains with respect to the interpretation of these associations due to potential confounding by various copollutants. The U.S. EPA has therefore concluded that the overall evidence is suggestive of a causal relationship between short-term exposure to SO_2 and mortality. Significant associations between short-term exposure to SO_2 and emergency department visits and hospital admissions for cardiovascular diseases have also been reported. However, these findings have been inconsistent across studies and do not provide adequate evidence to infer a causal relationship between SO_2 exposure and cardiovascular morbidity.

8.1.1.7 Health Effects of NO_2

Information on the health effects of NO_2 can be found in the EPA Integrated Science Assessment (ISA) for Nitrogen Oxides.[41] The EPA has concluded that the findings of epidemiologic, controlled human exposure, and animal toxicological studies provide evidence that is sufficient to infer a likely causal relationship between respiratory effects and short-term NO_2 exposure. The ISA concludes that the strongest evidence for such a relationship comes from epidemiologic studies of respiratory effects including symptoms, emergency department visits, and hospital admissions. The ISA also draws two broad conclusions regarding airway responsiveness following NO_2 exposure. First, the ISA concludes that NO_2 exposure may enhance the sensitivity to allergen-induced decrements in lung function and increase the allergen-induced airway inflammatory response following 30-minute exposures of asthmatics to NO_2 concentrations as low as 0.26 ppm. In addition, small but significant increases in non-specific airway hyperresponsiveness were reported following 1-hour exposures of asthmatics to 0.1 ppm NO_2. Second, exposure to NO_2 has been found to enhance the inherent responsiveness of the airway to subsequent nonspecific challenges in controlled human exposure studies of asthmatic subjects. Enhanced airway responsiveness could have important clinical implications for asthmatics since transient increases in airway responsiveness following NO_2 exposure have the potential to increase symptoms and worsen asthma control. Together, the epidemiologic and experimental data sets form a plausible, consistent, and coherent description of a relationship between NO_2 exposures and an array of adverse health effects that range from the onset of respiratory symptoms to hospital admission.

Although the weight of evidence supporting a causal relationship is somewhat less certain than that associated with respiratory morbidity, NO_2 has also been linked to other health endpoints. These include all-cause (nonaccidental) mortality, hospital admissions or emergency department visits for cardiovascular disease, and decrements in lung function growth associated with chronic exposure.

8.1.1.8 Health Effects of Carbon Monoxide

Information on the health effects of carbon monoxide (CO) can be found in the EPA Integrated Science Assessment (ISA) for Carbon Monoxide.[42] The ISA concludes that ambient concentrations of CO are associated with a number of adverse health effects.[F] This section provides a summary of the health effects associated with exposure to ambient concentrations of CO.[G]

[F] The ISA evaluates the health evidence associated with different health effects, assigning one of five "weight of evidence" determinations: causal relationship, likely to be a causal relationship, suggestive of a causal relationship, inadequate to infer a causal relationship, and not likely to be a causal relationship. For definitions of these levels of evidence, please refer to Section 1.6 of the ISA.

[G] Personal exposure includes contributions from many sources, and in many different environments. Total personal exposure to CO includes both ambient and nonambient components; and both components may contribute to adverse health effects.

Human clinical studies of subjects with coronary artery disease show a decrease in the time to onset of exercise-induced angina (chest pain) and electrocardiogram changes following CO exposure. In addition, epidemiologic studies show associations between short-term CO exposure and cardiovascular morbidity, particularly increased emergency room visits and hospital admissions for coronary heart disease (including ischemic heart disease, myocardial infarction, and angina). Some epidemiologic evidence is also available for increased hospital admissions and emergency room visits for congestive heart failure and cardiovascular disease as a whole. The ISA concludes that a causal relationship is likely to exist between short-term exposures to CO and cardiovascular morbidity. It also concludes that available data are inadequate to conclude that a causal relationship exists between long-term exposures to CO and cardiovascular morbidity.

Animal studies show various neurological effects with in-utero CO exposure. Controlled human exposure studies report inconsistent neural and behavioral effects following low-level CO exposures. The ISA concludes the evidence is suggestive of a causal relationship with both short- and long-term exposure to CO and central nervous system effects.

A number of epidemiologic and animal toxicological studies cited in the ISA have evaluated associations between CO exposure and birth outcomes such as preterm birth or cardiac birth defects. The epidemiologic studies provide limited evidence of a CO-induced effect on preterm births and birth defects, with weak evidence for a decrease in birth weight. Animal toxicological studies have found associations between perinatal CO exposure and decrements in birth weight, as well as other developmental outcomes. The ISA concludes these studies are suggestive of a causal relationship between long-term exposures to CO and developmental effects and birth outcomes.

Epidemiologic studies provide evidence of effects on respiratory morbidity such as changes in pulmonary function, respiratory symptoms, and hospital admissions associated with ambient CO concentrations. A limited number of epidemiologic studies considered copollutants such as ozone, SO_2, and PM in two-pollutant models and found that CO risk estimates were generally robust, although this limited evidence makes it difficult to disentangle effects attributed to CO itself from those of the larger complex air pollution mixture. Controlled human exposure studies have not extensively evaluated the effect of CO on respiratory morbidity. Animal studies at levels of 50-100 ppm CO show preliminary evidence of altered pulmonary vascular remodeling and oxidative injury. The ISA concludes that the evidence is suggestive of a causal relationship between short-term CO exposure and respiratory morbidity, and inadequate to conclude that a causal relationship exists between long-term exposure and respiratory morbidity.

Finally, the ISA concludes that the epidemiologic evidence is suggestive of a causal relationship between short-term exposures to CO and mortality. Epidemiologic studies provide evidence of an association between short-term exposure to CO and mortality, but limited evidence is available to evaluate cause-specific mortality outcomes associated with CO exposure. In addition, the attenuation of CO risk estimates which was often observed in copollutant models contributes to the uncertainty as to whether CO is acting alone or as an indicator for other combustion-related pollutants. The ISA also concludes that there is not likely to be a causal relationship between relevant long-term exposures to CO and mortality.

8.1.1.9 Health Effects of Air Toxics

Motor vehicle emissions contribute to ambient levels of air toxics known or suspected as human or animal carcinogens, or that have noncancer health effects. The population experiences an elevated risk of cancer and other noncancer health effects from exposure to air toxics.[43] These compounds include, but are not limited to, benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, diesel particulate matter and exhaust organic gases, polycyclic organic matter (POM), and naphthalene. These compounds were identified as national or regional risk drivers in past National-scale Air Toxics Assessments (NATA) and have significant inventory contributions from mobile sources. Although the 2002 NATA did not quantify cancer risks associated with exposure to diesel exhaust, EPA has concluded that diesel exhaust ranks with the other emissions that the 2002 NATA suggests pose the greatest relative risk. According to NATA for 2002, mobile sources were responsible for 47 percent of outdoor toxic emissions, over 50 percent of the cancer risk, and over 80 percent of the noncancer hazard. Data from the 2002 National Emissions Inventory (NEI), which is the basis for NATA, show that thirty percent of national diesel PM emissions are attributable to heavy-duty vehicles.[44]

Noncancer health effects can result from chronic,[H] subchronic,[I] or acute[J] inhalation exposures to air toxics, and include neurological, cardiovascular, liver, kidney, and respiratory effects as well as effects on the immune and reproductive systems. According to the 2002 NATA, nearly the entire U.S. population was exposed to an average concentration of air toxics that has the potential for adverse noncancer respiratory health effects. This will continue to be the case in 2030, even though toxics concentrations will be lower.[45]

The NATA modeling framework has a number of limitations which prevent its use as the sole basis for setting regulatory standards. These limitations and uncertainties are discussed on the 2002 NATA website.[46] Even so, this modeling framework is very useful in identifying air toxic pollutants and sources of greatest concern, setting regulatory priorities, and informing the decision making process.

8.1.1.9.1   Diesel Exhaust PM

Heavy-duty diesel engines emit diesel exhaust (DE), a complex mixture comprised of carbon dioxide, oxygen, nitrogen, water vapor, carbon monoxide, nitrogen compounds, sulfur compounds and numerous low-molecular-weight hydrocarbons. A number of these gaseous hydrocarbon components are individually known to be toxic including aldehydes, benzene and 1,3-butadiene. The diesel particulate matter (DPM) present in diesel exhaust consists of fine particles (< 2.5μm), including a subgroup with a large number of ultrafine particles (< 0.1 μm).

[H] Chronic exposure is defined in the glossary of the Integrated Risk Information (IRIS) database (http://www.epa.gov/iris) as repeated exposure by the oral, dermal, or inhalation route for more than approximately 10% of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).
[I] Defined in the IRIS database as exposure to a substance spanning approximately 10% of the lifetime of an organism.
[J] Defined in the IRIS database as exposure by the oral, dermal, or inhalation route for 24 hours or less.

These particles have large surface areas which make them an excellent medium for adsorbing organics, and their small size makes them highly respirable and able to deposit deep in the lung. Diesel PM contains small quantities of numerous mutagenic and carcinogenic compounds associated with the particles (and also organic gases). In addition, while toxic trace metals emitted by heavy-duty diesel engines represent a very small portion of the national emissions of metals (less than one percent) and are a small portion of diesel PM (generally much less than one percent of diesel PM), we note that several trace metals of potential toxicological significance and persistence in the environment are emitted by diesel engines. These trace metals include chromium, manganese, mercury and nickel. In addition, small amounts of dioxins have been measured in highway engine diesel exhaust, some of which may partition into the particulate phase. Dioxins are a major health concern but diesel engines are a minor contributor to overall dioxin emissions.

Diesel exhaust varies significantly in chemical composition and particle sizes between different engine types (heavy-duty, light-duty), engine operating conditions (idle, accelerate, decelerate), and fuel formulations (high/low sulfur fuel).[47] Also, there are emission differences between on-road and nonroad engines because the nonroad engines are generally of older technology. After being emitted, diesel exhaust undergoes dilution as well as chemical and physical changes in the atmosphere. The lifetime for some of the compounds present in diesel exhaust ranges from hours to days.

A number of health studies have been conducted regarding diesel exhaust. These include epidemiologic studies of lung cancer in groups of workers and animal studies focusing on non-cancer effects specific to diesel exhaust exposure. Diesel exhaust PM (including the associated organic compounds which are generally high molecular weight hydrocarbon types but not the more volatile gaseous hydrocarbon compounds) is generally used as a surrogate measure for diesel exhaust.

8.1.1.9.1.1 *Potential Cancer Effects of Exposure to Diesel Exhaust*

Exposure to diesel exhaust is of specific concern because it has been judged by EPA to pose a lung cancer hazard for humans at environmental levels of exposure.

EPA's 2002 final "Health Assessment Document for Diesel Engine Exhaust" (the EPA Diesel HAD) classified exposure to diesel exhaust as likely to be carcinogenic to humans by inhalation at environmental exposures, in accordance with the revised draft 1996/1999 EPA cancer guidelines.[48,49] In accordance with earlier EPA guidelines, exposure to diesel exhaust would similarly be classified as probably carcinogenic to humans (Group B1).[50,51] A number of other agencies (National Institute for Occupational Safety and Health, the International Agency for Research on Cancer, the World Health Organization, California EPA, and the U.S. Department of Health and Human Services) have made similar classifications.[52, 53,54,55,56] The Health Effects Institute has prepared numerous studies and reports on the potential carcinogenicity of exposure to diesel exhaust.[57,58,59]

More specifically, the EPA Diesel HAD states that the conclusions of the document apply to diesel exhaust in use today including both on-road and nonroad engines. The EPA Diesel HAD acknowledges that the studies were done on engines with generally older technologies and

that "there have been changes in the physical and chemical composition of some DE [diesel exhaust] emissions (onroad vehicle emissions) over time, though there is no definitive information to show that the emission changes portend significant toxicological changes."

For the Diesel HAD, EPA reviewed 22 epidemiologic studies on the subject of the carcinogenicity of exposure to diesel exhaust in various occupations, finding increased lung cancer risk, although not always statistically significant, in 8 out of 10 cohort studies and 10 out of 12 case-control studies which covered several industries. Relative risk for lung cancer, associated with exposure, ranged from 1.2 to 1.5, although a few studies show relative risks as high as 2.6. Additionally, the Diesel HAD also relied on two independent meta-analyses, which examined 23 and 30 occupational studies respectively, and found statistically significant increases of 1.33 to 1.47 in smoking-adjusted relative lung cancer risk associated with diesel exhaust. These meta-analyses demonstrate the effect of pooling many studies and in this case show the positive relationship between diesel exhaust exposure and lung cancer across a variety of diesel exhaust-exposed occupations.[60,61,62]

EPA generally derives cancer unit risk estimates to calculate population risk more precisely from exposure to carcinogens. In the simplest terms, the cancer unit risk is the increased risk associated with average lifetime exposure of 1 $\mu g/m^3$. EPA concluded in the Diesel HAD that it is not currently possible to calculate a cancer unit risk for diesel exhaust due to a variety of factors that limit the current studies, such as a lack of standard exposure metric for diesel exhaust and the absence of quantitative exposure characterization in retrospective studies.

In the absence of a cancer unit risk, the Diesel HAD sought to provide additional insight into the significance of the diesel exhaust-cancer hazard by estimating possible ranges of risk that might be present in the population. An exploratory analysis was used to characterize a possible risk range by comparing a typical environmental exposure level for highway diesel sources to a selected range of occupational exposure levels. The occupationally observed risks were then proportionally scaled according to the exposure ratios to obtain an estimate of the possible environmental risk. If the occupational and environmental exposures are similar, the environmental risk would approach the risk seen in the occupational studies whereas a much higher occupational exposure indicates that the environmental risk is lower than the occupational risk. A comparison of environmental and occupational exposures showed that for certain occupations the exposures are similar to environmental exposures while, for others, they differ by a factor of about 200 or more.

A number of calculations are involved in the exploratory analysis of a possible risk range, and these can be seen in the EPA Diesel HAD. The outcome was that environmental risks from diesel exhaust exposure could range from a low of 10^{-4} to 10^{-5} to as high as 10^{-3}, reflecting the range of occupational exposures that could be associated with the relative and absolute risk levels observed in the occupational studies. Because of uncertainties, the analysis acknowledged that the risks could be lower than 10^{-4} or 10^{-5}, and a zero risk from diesel exhaust exposure was not ruled out.

As mentioned in Section 8.1.1.9, EPA recently assessed air toxic emissions and their associated risk (the National-Scale Air Toxics Assessment or NATA for 2002), and we concluded that diesel exhaust ranks with other emissions that the national-scale assessment

suggests pose the greatest relative risk.[63] This national assessment estimates average population inhalation exposures to DPM for nonroad as well as on-highway sources. These are the sum of ambient levels in various locations weighted by the amount of time people spend in each of the locations.

In summary, even though EPA does not have a specific carcinogenic potency with which to accurately estimate the carcinogenic impact of exposure to diesel exhaust, the likely hazard to humans together with the potential for significant environmental risks leads us to conclude that diesel exhaust emissions from heavy-duty diesel engines present public health issues of concern to this rule.

8.1.1.9.1.2   *Other Health Effects of Exposure to Diesel Exhaust*

Noncancer health effects of acute and chronic exposure to diesel exhaust emissions are also of concern to the EPA. The Diesel HAD established an inhalation Reference Concentration (RfC) specifically based on animal studies of diesel exhaust exposure. An RfC is defined by EPA as "an estimate of a continuous inhalation exposure to the human population, including sensitive subgroups, with uncertainty spanning perhaps an order of magnitude, which is likely to be without appreciable risks of deleterious noncancer effects during a lifetime." EPA derived the RfC from consideration of four well-conducted chronic rat inhalation studies showing adverse pulmonary effects.[64,65,66,67] The diesel RfC is based on a "no observable adverse effect" level of 144 $\mu g/m^3$ that is further reduced by applying uncertainty factors of 3 for interspecies extrapolation and 10 for human variations in sensitivity. The resulting RfC derived in the Diesel HAD is 5 $\mu g/m^3$ for diesel exhaust as measured by DPM. This RfC does not consider allergenic effects such as those associated with asthma or immunologic effects. There is growing evidence that exposure to diesel exhaust can exacerbate these effects, but the exposure-response data is presently lacking to derive an RfC. The EPA Diesel HAD states, "With DPM [diesel particulate matter] being a ubiquitous component of ambient PM, there is an uncertainty about the adequacy of the existing DE [diesel exhaust] noncancer database to identify all of the pertinent DE-caused noncancer health hazards."

While there have been relatively few human studies associated specifically with the noncancer impact of exposure to DPM alone, DPM is a component of the ambient particles studied in numerous epidemiologic studies. The conclusion that health effects associated with ambient PM in general are relevant to DPM is supported by studies that specifically associate observable human noncancer health effects with exposure to DPM. As described in the Diesel HAD, these studies identified some of the same health effects reported for ambient PM, such as respiratory symptoms (cough, labored breathing, chest tightness, wheezing), and chronic respiratory disease (cough, phlegm, chronic bronchitis and suggestive evidence for decreases in pulmonary function). Symptoms of immunological effects such as wheezing and increased allergenicity are also seen. Studies in rodents, especially rats, show the potential for human inflammatory effects in the lung and consequential lung tissue damage from chronic diesel exhaust inhalation exposure. The Diesel HAD concludes "that acute exposure to DE [diesel exhaust] has been associated with irritation of the eye, nose, and throat, respiratory symptoms (cough and phlegm), and neurophysiological symptoms such as headache, lightheadedness, nausea, vomiting, and numbness or tingling of the extremities."[68] There is also evidence for an

immunologic effect such as the exacerbation of allergenic responses to known allergens and asthma-like symptoms.[69,70,71]

The Diesel HAD briefly summarizes health effects associated with ambient PM and discusses the $PM_{2.5}$ NAAQS. There is a much more extensive body of human data, which is also mentioned earlier in the health effects discussion for $PM_{2.5}$ (Section 8.1.1.2 of this RIA), showing a wide spectrum of adverse health effects associated with exposure to ambient PM, of which diesel exhaust is an important component. The $PM_{2.5}$ NAAQS is designed to provide protection from the non-cancer and premature mortality effects of $PM_{2.5}$ as a whole.

8.1.1.9.1.3   Ambient Levels of Diesel Exhaust PM

Because DPM is part of overall ambient PM and cannot be easily distinguished from overall PM, we do not have direct measurements of DPM in the ambient air. DPM concentrations are estimated using ambient air quality modeling based on DPM emission inventories. DPM concentrations were recently estimated as part of the 2002 NATA.[72] Ambient impacts of mobile source emissions were predicted using the Assessment System for Population Exposure Nationwide (ASPEN) dispersion model.

Concentrations of DPM were calculated at the census tract level in the 2002 NATA. Table 8-1 below summarizes the distribution of ambient DPM concentrations at the national scale. The median DPM concentration calculated nationwide is 0.89 μg/m^3. Over 30% of the DPM and diesel exhaust organic gases can be attributed to onroad diesels. A map of ambient diesel PM concentrations is provided in Figure 8-1. Areas with high median concentrations are clustered in the Northeast, Great Lake States, California, and the Gulf Coast States, and are also distributed throughout the rest of the U.S.

2002 NATA
Diesel PM Concentrations (ug/m3)
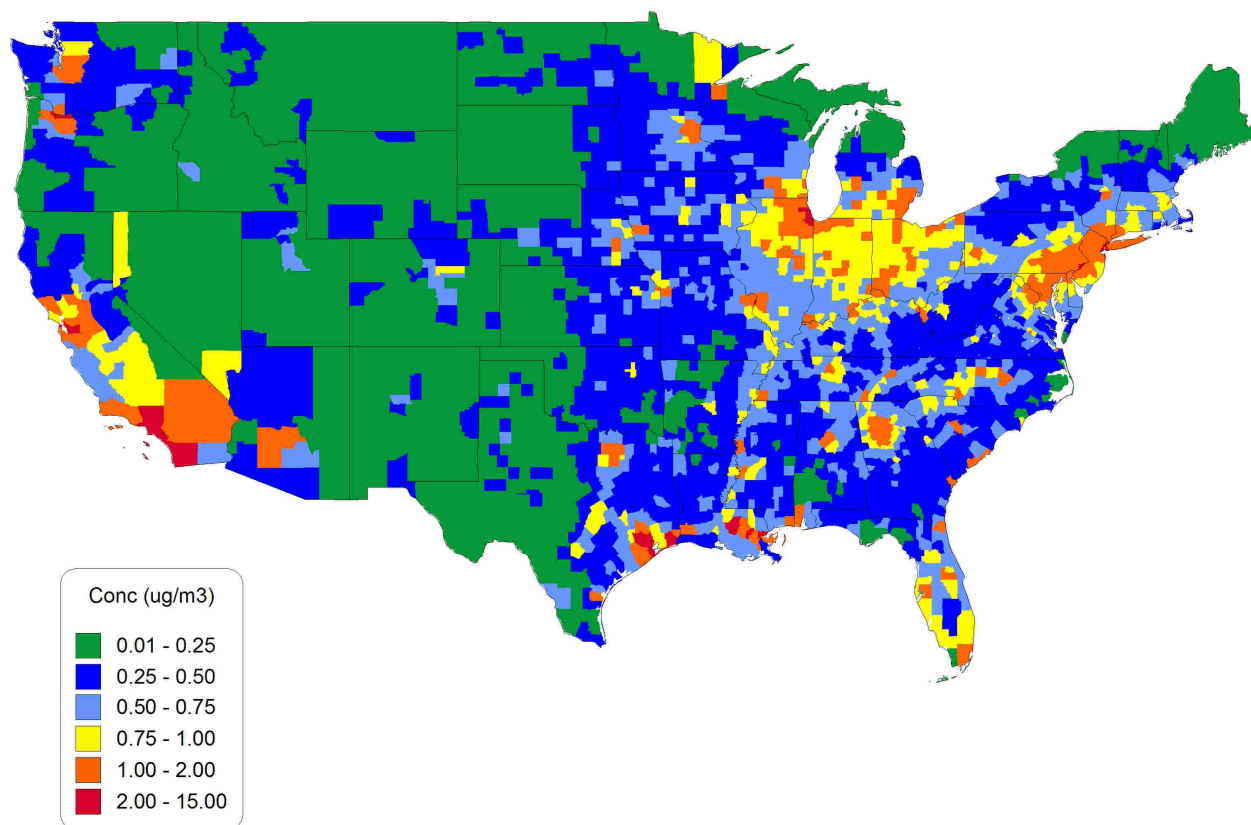
Figure 8-1 Estimated County Ambient Concentration of Diesel Particulate Matter

Table 8-1 Distribution of Census Tract Ambient Concentrations of DPM at the National Scale in 2002 NATA[a]

	NATIONWIDE (MG/M^3)
5th Percentile	0.21
25th Percentile	0.54
Median	0.89
75th Percentile	1.34
95th Percentile	2.63
Onroad Contribution to Mean	31%

Note:
[a] This table is generated from data contained in the diesel particulate matter Microsoft Access database file found in the Tract-Level Ambient Concentration Summaries section of the 2002 NATA webpage (http://www.epa.gov/ttn/atw/nata2002/tables.html).

8.1.1.9.1.4   Exposure to Diesel Exhaust PM

Exposure of people to diesel exhaust depends on their various activities, the time spent in those activities, the locations where these activities occur, and the levels of diesel exhaust pollutants in those locations. The major difference between ambient levels of diesel particulate and exposure levels for diesel particulate is that exposure levels account for a person moving from location to location, the proximity to the emission source, and whether the exposure occurs in an enclosed environment.

8.1.1.9.1.4.1   Occupational Exposures

Occupational exposures to diesel exhaust from mobile sources can be several orders of magnitude greater than typical exposures in the non-occupationally exposed population.

Over the years, diesel particulate exposures have been measured for a number of occupational groups resulting in a wide range of exposures from 2 to 1280 μg/m^3 for a variety of occupations. As discussed in the Diesel HAD, the National Institute of Occupational Safety and Health (NIOSH) has estimated a total of 1,400,000 workers are occupationally exposed to diesel exhaust from on-road and nonroad vehicles.

8.1.1.9.1.4.2   Elevated Concentrations and Ambient Exposures in Mobile Source Impacted Areas

Regions immediately downwind of highways or truck stops may experience elevated ambient concentrations of directly-emitted $PM_{2.5}$ from diesel engines. Due to the unique nature of highways and truck stops, emissions from a large number of diesel engines are concentrated in a small area. Studies near roadways with high truck traffic indicate higher concentrations of components of diesel PM than other locations.[73,74,75] High ambient particle concentrations have also been reported near trucking terminals, truck stops, and bus garages.[76,77,78] Additional discussion of exposure and health effects associated with traffic is included below in Section 8.1.1.10.

8.1.1.9.2   Benzene

The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia) by all routes of exposure, and concludes that exposure is associated with additional health effects, including genetic changes in both humans and animals and increased proliferation of bone marrow cells in mice.[79,80,81] EPA states in its IRIS database that data indicate a causal relationship between benzene exposure and acute lymphocytic leukemia and suggest a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. The International Agency for Research on Carcinogens (IARC) has determined that benzene is a human carcinogen and the U.S. Department of Health and Human Services (DHHS) has characterized benzene as a known human carcinogen.[82,83]

A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with long-term exposure to benzene.[84,85] The most sensitive noncancer effect observed in humans, based on current data, is the depression of the absolute lymphocyte count in blood.[86,87] In addition, recent work,

including studies sponsored by the Health Effects Institute (HEI), provides evidence that biochemical responses are occurring at lower levels of benzene exposure than previously known.[88,89,90,91] EPA's IRIS program has not yet evaluated these new data.

8.1.1.9.3   1,3-Butadiene

EPA has characterized 1,3-butadiene as carcinogenic to humans by inhalation.[92,93] The IARC has determined that 1,3-butadiene is a human carcinogen and the U.S. DHHS has characterized 1,3-butadiene as a known human carcinogen.[94,9596] There are numerous studies consistently demonstrating that 1,3-butadiene is metabolized into genotoxic metabolites by experimental animals and humans. The specific mechanisms of 1,3-butadiene-induced carcinogenesis are unknown; however, the scientific evidence strongly suggests that the carcinogenic effects are mediated by genotoxic metabolites. Animal data suggest that females may be more sensitive than males for cancer effects associated with 1,3-butadiene exposure; there are insufficient data in humans from which to draw conclusions about sensitive subpopulations. 1,3-butadiene also causes a variety of reproductive and developmental effects in mice; no human data on these effects are available. The most sensitive effect was ovarian atrophy observed in a lifetime bioassay of female mice.[97]

8.1.1.9.4   Formaldehyde

Since 1987, EPA has classified formaldehyde as a probable human carcinogen based on evidence in humans and in rats, mice, hamsters, and monkeys.[98] EPA is currently reviewing recently published epidemiological data. For instance, research conducted by the National Cancer Institute (NCI) found an increased risk of nasopharyngeal cancer and lymphohematopoietic malignancies such as leukemia among workers exposed to formaldehyde.[99,100] In an analysis of the lymphohematopoietic cancer mortality from an extended follow-up of these workers, NCI confirmed an association between lymphohematopoietic cancer risk and peak exposures.[101] A recent National Institute of Occupational Safety and Health (NIOSH) study of garment workers also found increased risk of death due to leukemia among workers exposed to formaldehyde.[102] Extended follow-up of a cohort of British chemical workers did not find evidence of an increase in nasopharyngeal or lymphohematopoietic cancers, but a continuing statistically significant excess in lung cancers was reported.[103]

In the past 15 years there has been substantial research on the inhalation dosimetry for formaldehyde in rodents and primates by the CIIT Centers for Health Research (formerly the Chemical Industry Institute of Toxicology), with a focus on use of rodent data for refinement of the quantitative cancer dose-response assessment.[104,105,106] CIIT's risk assessment of formaldehyde incorporated mechanistic and dosimetric information on formaldehyde. However, it should be noted that recent research published by EPA indicates that when two-stage modeling assumptions are varied, resulting dose-response estimates can vary by several orders of magnitude.[107,108,109,110] These findings are not supportive of interpreting the CIIT model results as providing a conservative (health protective) estimate of human risk.[111] EPA research also examined the contribution of the two-stage modeling for formaldehyde towards characterizing the relative weights of key events in the mode-of-action of a carcinogen. For example, the model-based inference in the published CIIT study that formaldehyde's direct mutagenic action

is not relevant to the compound's tumorigenicity was found not to hold under variations of modeling assumptions.[112]

Based on the developments of the last decade, in 2004, the working group of the IARC concluded that formaldehyde is carcinogenic to humans (Group 1), on the basis of sufficient evidence in humans and sufficient evidence in experimental animals - a higher classification than previous IARC evaluations. After reviewing the currently available epidemiological evidence, the IARC (2006) characterized the human evidence for formaldehyde carcinogenicity as "sufficient," based upon the data on nasopharyngeal cancers; the epidemiologic evidence on leukemia was characterized as "strong."[113] EPA is reviewing the recent work cited above from the NCI and NIOSH, as well as the analysis by the CIIT Centers for Health Research and other studies, as part of a reassessment of the human hazard and dose-response associated with formaldehyde.

Formaldehyde exposure also causes a range of noncancer health effects, including irritation of the eyes (burning and watering of the eyes), nose and throat. Effects from repeated exposure in humans include respiratory tract irritation, chronic bronchitis and nasal epithelial lesions such as metaplasia and loss of cilia. Animal studies suggest that formaldehyde may also cause airway inflammation – including eosinophil infiltration into the airways. There are several studies that suggest that formaldehyde may increase the risk of asthma – particularly in the young.[114,115]

8.1.1.9.5 Acetaldehyde

Acetaldehyde is classified in EPA's IRIS database as a probable human carcinogen, based on nasal tumors in rats, and is considered toxic by the inhalation, oral, and intravenous routes.[116] Acetaldehyde is reasonably anticipated to be a human carcinogen by the U.S. DHHS in the 11th Report on Carcinogens and is classified as possibly carcinogenic to humans (Group 2B) by the IARC.[117,118] EPA is currently conducting a reassessment of cancer risk from inhalation exposure to acetaldehyde.

The primary noncancer effects of exposure to acetaldehyde vapors include irritation of the eyes, skin, and respiratory tract.[119] In short-term (4 week) rat studies, degeneration of olfactory epithelium was observed at various concentration levels of acetaldehyde exposure.[120,121] Data from these studies were used by EPA to develop an inhalation reference concentration. Some asthmatics have been shown to be a sensitive subpopulation to decrements in functional expiratory volume (FEV1 test) and bronchoconstriction upon acetaldehyde inhalation.[122] The agency is currently conducting a reassessment of the health hazards from inhalation exposure to acetaldehyde.

8.1.1.9.6 Acrolein

Acrolein is extremely acrid and irritating to humans when inhaled, with acute exposure resulting in upper respiratory tract irritation, mucus hypersecretion and congestion. The intense irritancy of this carbonyl has been demonstrated during controlled tests in human subjects, who suffer intolerable eye and nasal mucosal sensory reactions within minutes of exposure.[123] These data and additional studies regarding acute effects of human exposure to acrolein are

summarized in EPA's 2003 IRIS Human Health Assessment for acrolein.[124] Evidence available from studies in humans indicate that levels as low as 0.09 ppm (0.21 mg/m^3) for five minutes may elicit subjective complaints of eye irritation with increasing concentrations leading to more extensive eye, nose and respiratory symptoms.[125] Lesions to the lungs and upper respiratory tract of rats, rabbits, and hamsters have been observed after subchronic exposure to acrolein.[126] Acute exposure effects in animal studies report bronchial hyper-responsiveness.[127] In a recent study, the acute respiratory irritant effects of exposure to 1.1 ppm acrolein were more pronounced in mice with allergic airway disease by comparison to non-diseased mice which also showed decreases in respiratory rate.[128] Based on these animal data and demonstration of similar effects in humans (e.g., reduction in respiratory rate), individuals with compromised respiratory function (e.g., emphysema, asthma) are expected to be at increased risk of developing adverse responses to strong respiratory irritants such as acrolein.

EPA determined in 2003 that the human carcinogenic potential of acrolein could not be determined because the available data were inadequate. No information was available on the carcinogenic effects of acrolein in humans and the animal data provided inadequate evidence of carcinogenicity.[129] The IARC determined in 1995 that acrolein was not classifiable as to its carcinogenicity in humans.[130]

8.1.1.9.7 *Polycyclic Organic Matter (POM)*

POM is generally defined as a large class of organic compounds which have multiple benzene rings and a boiling point greater than 100 degrees Celsius. Many of the compounds included in the class of compounds known as POM are classified by EPA as probable human carcinogens based on animal data. One of these compounds, naphthalene, is discussed separately below. Polycyclic aromatic hydrocarbons (PAHs) are a subset of POM that contain only hydrogen and carbon atoms. A number of PAHs are known or suspected carcinogens. Recent studies have found that maternal exposures to PAHs (a subclass of POM) in a population of pregnant women were associated with several adverse birth outcomes, including low birth weight and reduced length at birth, as well as impaired cognitive development at age three.[131,132] EPA has not yet evaluated these recent studies.

8.1.1.9.8 *Naphthalene*

Naphthalene is found in small quantities in gasoline and diesel fuels. Naphthalene emissions have been measured in larger quantities in both gasoline and diesel exhaust compared with evaporative emissions from mobile sources, indicating it is primarily a product of combustion. EPA released an external review draft of a reassessment of the inhalation carcinogenicity of naphthalene based on a number of recent animal carcinogenicity studies.[133] The draft reassessment completed external peer review.[134] Based on external peer review comments received, additional analyses are being undertaken. This external review draft does not represent official agency opinion and was released solely for the purposes of external peer review and public comment. The National Toxicology Program listed naphthalene as "reasonably anticipated to be a human carcinogen" in 2004 on the basis of bioassays reporting clear evidence of carcinogenicity in rats and some evidence of carcinogenicity in mice.[135] California EPA has released a new risk assessment for naphthalene, and the IARC has reevaluated naphthalene and re-classified it as Group 2B: possibly carcinogenic to humans.[136]

Naphthalene also causes a number of chronic non-cancer effects in animals, including abnormal cell changes and growth in respiratory and nasal tissues.[137]

8.1.1.9.9   Other Air Toxics

In addition to the compounds described above, other compounds in gaseous hydrocarbon and PM emissions from vehicles would be affected by today's proposed action. Mobile source air toxic compounds that would potentially be impacted include ethylbenzene, propionaldehyde, toluene, and xylene. Information regarding the health effects of these compounds can be found in EPA's IRIS database.[K]

8.1.1.10 Exposure and Health Effects Associated with Traffic

Populations who live, work, or attend school near major roads experience elevated exposure concentrations to a wide range of air pollutants, as well as higher risks for a number of adverse health effects. While the previous sections of this RIA have focused on the health effects associated with individual criteria pollutants or air toxics, this section discusses the mixture of different exposures near major roadways, rather than the effects of any single pollutant. As such, this section emphasizes traffic-related air pollution, in general, as the relevant indicator of exposure rather than any particular pollutant.

Concentrations of many traffic-generated air pollutants are elevated for up to 300-500 meters downwind of roads with high traffic volumes.[138] Numerous sources on roads contribute to elevated roadside concentrations, including exhaust and evaporative emissions, and resuspension of road dust and tire and brake wear. Concentrations of several criteria and hazardous air pollutants are elevated near major roads. Furthermore, different semi-volatile organic compounds and chemical components of particulate matter, including elemental carbon, organic material, and trace metals, have been reported at higher concentrations near major roads.

Populations near major roads experience greater risk of certain adverse health effects. The Health Effects Institute published a report on the health effects of traffic-related air pollution.[139] It concluded that evidence is "sufficient to infer the presence of a causal association" between traffic exposure and exacerbation of childhood asthma symptoms. The HEI report also concludes that the evidence is either "sufficient" or "suggestive but not sufficient" for a causal association between traffic exposure and new childhood asthma cases. A review of asthma studies by Salam et al. (2008) reaches similar conclusions.[140] The HEI report also concludes that there is "suggestive" evidence for pulmonary function deficits associated with traffic exposure, but concluded that there is "inadequate and insufficient" evidence for causal associations with respiratory health care utilization, adult-onset asthma, COPD symptoms, and allergy. A review by Holguin (2008) notes that the effects of traffic on asthma may be modified by nutrition status, medication use, and genetic factors.[141]

[K] U.S. EPA Integrated Risk Information System (IRIS) database is available at: www.epa.gov/iris

The HEI report also concludes that evidence is "suggestive" of a causal association between traffic exposure and all-cause and cardiovascular mortality. There is also evidence of an association between traffic-related air pollutants and cardiovascular effects such as changes in heart rhythm, heart attack, and cardiovascular disease. The HEI report characterizes this evidence as "suggestive" of a causal association, and an independent epidemiological literature review by Adar and Kaufman (2007) concludes that there is "consistent evidence" linking traffic-related pollution and adverse cardiovascular health outcomes.[142]

Some studies have reported associations between traffic exposure and other health effects, such as birth outcomes (e.g., low birth weight) and childhood cancer. The HEI report concludes that there is currently "inadequate and insufficient" evidence for a causal association between these effects and traffic exposure. A review by Raaschou-Nielsen and Reynolds (2006) concluded that evidence of an association between childhood cancer and traffic-related air pollutants is weak, but noted the inability to draw firm conclusions based on limited evidence.[143]

There is a large population in the U.S. living in close proximity of major roads. According to the Census Bureau's American Housing Survey for 2007, approximately 20 million residences in the U.S., 15.6% of all homes, are located within 300 feet (91 m) of a highway with 4+ lanes, a railroad, or an airport.[144] Therefore, at current population of approximately 309 million, assuming that population and housing are similarly distributed, there are over 48 million people in the U.S. living near such sources. The HEI report also notes that in two North American cities, Los Angeles and Toronto, over 40% of each city's population live within 500 meters of a highway or 100 meters of a major road. It also notes that about 33% of each city's population resides within 50 meters of major roads. Together, the evidence suggests that a large U.S. population lives in areas with elevated traffic-related air pollution.

People living near roads are often socioeconomically disadvantaged. According to the 2007 American Housing Survey, a renter-occupied property is over twice as likely as an owner-occupied property to be located near a highway with 4+ lanes, railroad or airport. In the same survey, the median household income of rental housing occupants was less than half that of owner-occupants ($28,921/$59,886). Numerous studies in individual urban areas report higher levels of traffic-related air pollutants in areas with high minority or poor populations.[145,146,147]

Students may also be exposed in situations where schools are located near major roads. In a study of nine metropolitan areas across the U.S., Appatova et al. (2008) found that on average greater than 33% of schools were located within 400 m of an Interstate, US, or state highway, while 12% were located within 100 m.[148] The study also found that among the metropolitan areas studied, schools in the Eastern U.S. were more often sited near major roadways than schools in the Western U.S.

Demographic studies of students in schools near major roadways suggest that this population is more likely than the general student population to be of non-white race or Hispanic ethnicity, and more often live in low socioeconomic status locations.[149,150,151] There is some inconsistency in the evidence, which may be due to different local development patterns and measures of traffic and geographic scale used in the studies.[148]

8.1.2   Environmental Effects Associated with Exposure to Non-GHG Pollutants

In this section we will discuss the environmental effects associated with non-GHG pollutants, specifically: particulate matter, ozone, NO_X, SO_X and air toxics.

8.1.2.1 Visibility Degradation

Emissions from heavy-duty vehicles contribute to poor visibility in the U.S. through their emissions of primary $PM_{2.5}$ and secondary $PM_{2.5}$ precursors such as NO_X. Airborne particles degrade visibility by scattering and absorbing light. Good visibility increases the quality of life where individuals live and work, and where they engage in recreational activities.

EPA is pursuing a two-part strategy to address visibility. First, EPA has concluded that $PM_{2.5}$ causes adverse effects on visibility in various locations, depending on PM concentrations and factors such as chemical composition and average relative humidity, and has set secondary $PM_{2.5}$ standards.[L] The secondary $PM_{2.5}$ standards act in conjunction with the regional haze program. EPA's regional haze rule (64 FR 35714) was put in place in July 1999 to protect the visibility in Mandatory Class I Dederal areas. There are 156 national parks, forests and wilderness areas categorized as Mandatory Class I Federal areas (62 FR 38680-81, July 18, 1997).[M] Visibility can be said to be impaired in both $PM_{2.5}$ nonattainment areas and mandatory class I federal areas. Figure 8-2 shows the location of the 156 Mandatory Class I Federal areas.

[L] The existing annual primary and secondary PM2.5 standards have been remanded and are being addressed in the currently ongoing PM NAAQS review.

[M] These areas are defined in CAA section 162 as those national parks exceeding 6,000 acres, wilderness areas and memorial parks exceeding 5,000 acres, and all international parks which were in existence on August 7, 1977.

Figure 8-2 <u>Mandatory Class I Federal Areas in the U.S.</u>

8.1.2.1.1   Visibility Monitoring

In conjunction with the U.S. National Park Service, the U.S. Forest Service, other Federal land managers, and State organizations in the U.S., the U.S. EPA has supported visibility monitoring in national parks and wilderness areas since 1988. The monitoring network was originally established at 20 sites, but it has now been expanded to 110 sites that represent all but one of the 156 Mandatory Federal Class I areas across the country (see Figure 8-2). This long-term visibility monitoring network is known as IMPROVE (Interagency Monitoring of Protected Visual Environments).

IMPROVE provides direct measurement of fine particles that contribute to visibility impairment. The IMPROVE network employs aerosol measurements at all sites, and optical and scene measurements at some of the sites. Aerosol measurements are taken for PM_{10} and $PM_{2.5}$ mass, and for key constituents of $PM_{2.5}$, such as sulfate, nitrate, organic and elemental carbon, soil dust, and several other elements. Measurements for specific aerosol constituents are used to calculate "reconstructed" aerosol light extinction by multiplying the mass for each constituent by its empirically-derived scattering and/or absorption efficiency, with adjustment for the relative humidity. Knowledge of the main constituents of a site's light extinction "budget" is critical for source apportionment and control strategy development. In addition to this indirect method of assessing light extinction, there are optical measurements which directly measure light extinction or its components. Such measurements are taken principally with either a transmissometer,

which measures total light extinction, or by combining the PM light scattering measured by integrating nephelometers with the PM light absorption measured by an aethalometer. Scene characteristics are typically recorded three times daily with 35 millimeter photography and are used to determine the quality of visibility conditions (such as effects on color and contrast) associated with specific levels of light extinction as measured under both direct and aerosol-related methods. Directly measured light extinction is used under the IMPROVE protocol to cross check that the aerosol-derived light extinction levels are reasonable in establishing current visibility conditions. Aerosol-derived light extinction is used to document spatial and temporal trends and to determine how proposed changes in atmospheric constituents would affect future visibility conditions.

Annual average visibility conditions (reflecting light extinction due to both anthropogenic and non-anthropogenic sources) vary regionally across the U.S. Visibility is typically worse in the summer months and the rural East generally has higher levels of impairment than remote sites in the West. Figures 9-9 through 9-11 in the PM ISA detail the percent contributions to particulate light extinction for ammonium nitrate and sulfate, EC and OC, and coarse mass and fine soil, by season.[152]

8.1.2.2 Plant and Ecosystem Effects of Ozone

There are a number of environmental or public welfare effects associated with the presence of ozone in the ambient air.[153] In this section we discuss the impact of ozone on plants, including trees, agronomic crops and urban ornamentals.

The Air Quality Criteria Document for Ozone and related Photochemical Oxidants notes that, "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant."[154] Like carbon dioxide (CO_2) and other gaseous substances, ozone enters plant tissues primarily through apertures (stomata) in leaves in a process called "uptake."[155] Once sufficient levels of ozone (a highly reactive substance), or its reaction products, reaches the interior of plant cells, it can inhibit or damage essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns.[156,157] If enough tissue becomes damaged from these effects, a plant's capacity to fix carbon to form carbohydrates, which are the primary form of energy used by plants is reduced,[158] while plant respiration increases. With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance, leading to reduced growth and/or reproduction. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, more susceptible to disease, insect attack, harsh weather (e.g., drought, frost) and other environmental stresses. Furthermore, there is evidence that ozone can interfere with the formation of mycorrhiza, essential symbiotic fungi associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont.[159,160]

This ozone damage may or may not be accompanied by visible injury on leaves, and likewise, visible foliar injury may or may not be a symptom of the other types of plant damage

described above. When visible injury is present, it is commonly manifested as chlorotic or necrotic spots, and/or increased leaf senescence (accelerated leaf aging). Because ozone damage can consist of visible injury to leaves, it can also reduce the aesthetic value of ornamental vegetation and trees in urban landscapes, and negatively affects scenic vistas in protected natural areas.

Ozone can produce both acute and chronic injury in sensitive species depending on the concentration level and the duration of the exposure. Ozone effects also tend to accumulate over the growing season of the plant, so that even lower concentrations experienced for a longer duration have the potential to create chronic stress on sensitive vegetation. Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of ozone uptake through closure of stomata)[161,162,163] Other resistance mechanisms may involve the intercellular production of detoxifying substances. Several biochemical substances capable of detoxifying ozone have been reported to occur in plants, including the antioxidants ascorbate and glutathione. After injuries have occurred, plants may be capable of repairing the damage to a limited extent.[164]

Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and response to ozone, it is not possible to identify threshold values above which ozone is consistently toxic for all plants. The next few paragraphs present additional information on ozone damage to trees, ecosystems, agronomic crops and urban ornamentals.

Ozone also has been conclusively shown to cause discernible injury to forest trees.[165,166] In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts. Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function.[167,168]

Because plants are at the base of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone impacts at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors.[169] In most instances, responses to chronic or recurrent exposure in forested ecosystems are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems.[170,171,172] It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States.

Laboratory and field experiments have also shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN

results show that "several economically important crop species are sensitive to ozone levels typical of those found in the United States."[173] In addition, economic studies have shown reduced economic benefits as a result of predicted reductions in crop yields associated with observed ozone levels.[174,175,176]

Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels. It is estimated that more than $20 billion (1990 dollars) are spent annually on landscaping using ornamentals, both by private property owners/tenants and by governmental units responsible for public areas.[177] This is therefore a potentially costly environmental effect. However, in the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative analysis has been conducted.

Air pollution can have noteworthy cumulative impacts on forested ecosystems by affecting regeneration, productivity, and species composition.[178] In the U.S., ozone in the lower atmosphere is one of the pollutants of primary concern. Ozone injury to forest plants can be diagnosed by examination of plant leaves. Foliar injury is usually the first visible sign of injury to plants from ozone exposure and indicates impaired physiological processes in the leaves.[179]

In the U.S. this indicator is based on data from the U.S. Department of Agriculture (USDA) Forest Service Forest Inventory and Analysis (FIA) program. As part of its Phase 3 program, formerly known as Forest Health Monitoring, FIA examines ozone injury to ozone-sensitive plant species at ground monitoring sites in forest land across the country. For this indicator, forest land does not include woodlots and urban trees. Sites are selected using a systematic sampling grid, based on a global sampling design.[180,181] At each site that has at least 30 individual plants of at least three ozone-sensitive species and enough open space to ensure that sensitive plants are not protected from ozone exposure by the forest canopy, FIA looks for damage on the foliage of ozone-sensitive forest plant species. Monitoring of ozone injury to plants by the USDA Forest Service has expanded over the last 10 years from monitoring sites in 10 states in 1994 to nearly 1,000 monitoring sites in 41 states in 2002.

8.1.2.2.1 Recent Ozone Data for the U.S.

There is considerable regional variation in ozone-related visible foliar injury to sensitive plants in the U.S. The U.S. EPA has developed an environmental indicator based on data from the U.S. Department of Agriculture (USDA) Forest Service Forest Inventory and Analysis (FIA) program which examines ozone injury to ozone-sensitive plant species at ground monitoring sites in forest land across the country (This indicator does not include woodlots and urban trees). Sites are selected using a systematic sampling grid, based on a global sampling design.[182, 183] Because ozone injury is cumulative over the course of the growing season, examinations are conducted in July and August, when ozone injury is typically highest. The data underlying the indicator in

Figure 8-3 are based on averages of all observations collected in 2002, the latest year for which data are publicly available at the time the study was conducted, and are broken down by U.S. EPA Region. Ozone damage to forest plants is classified using a subjective five-category biosite index based on expert opinion, but designed to be equivalent from site to site. Ranges of

biosite values translate to no injury, low or moderate foliar injury (visible foliar injury to highly sensitive or moderately sensitive plants, respectively), and high or severe foliar injury, which would be expected to result in tree-level or ecosystem-level responses, respectively.[184, 185]

The highest percentages of observed high and severe foliar injury, those which are most likely to be associated with tree or ecosystem-level responses, are primarily found in the Mid-Atlantic and Southeast regions. In EPA Region 3 (which comprises the States of Pennsylvania, West Virginia, Virginia, Delaware, Maryland and Washington D.C.), 12% of ozone-sensitive plants showed signs of high or severe foliar damage, and in Regions 2 (States of New York, New Jersey), and 4 (States of North Carolina, South Carolina, Kentucky, Tennessee, Georgia, Florida, Alabama, and Mississippi) the values were 10% and 7%, respectively. The sum of high and severe ozone injury ranged from 2% to 4% in EPA Region 1 (the six New England States), Region 7 (States of Missouri, Iowa, Nebraska and Kansas), and Region 9 (States of California, Nevada, Hawaii and Arizona). The percentage of sites showing some ozone damage was about 45% in each of these EPA Regions.
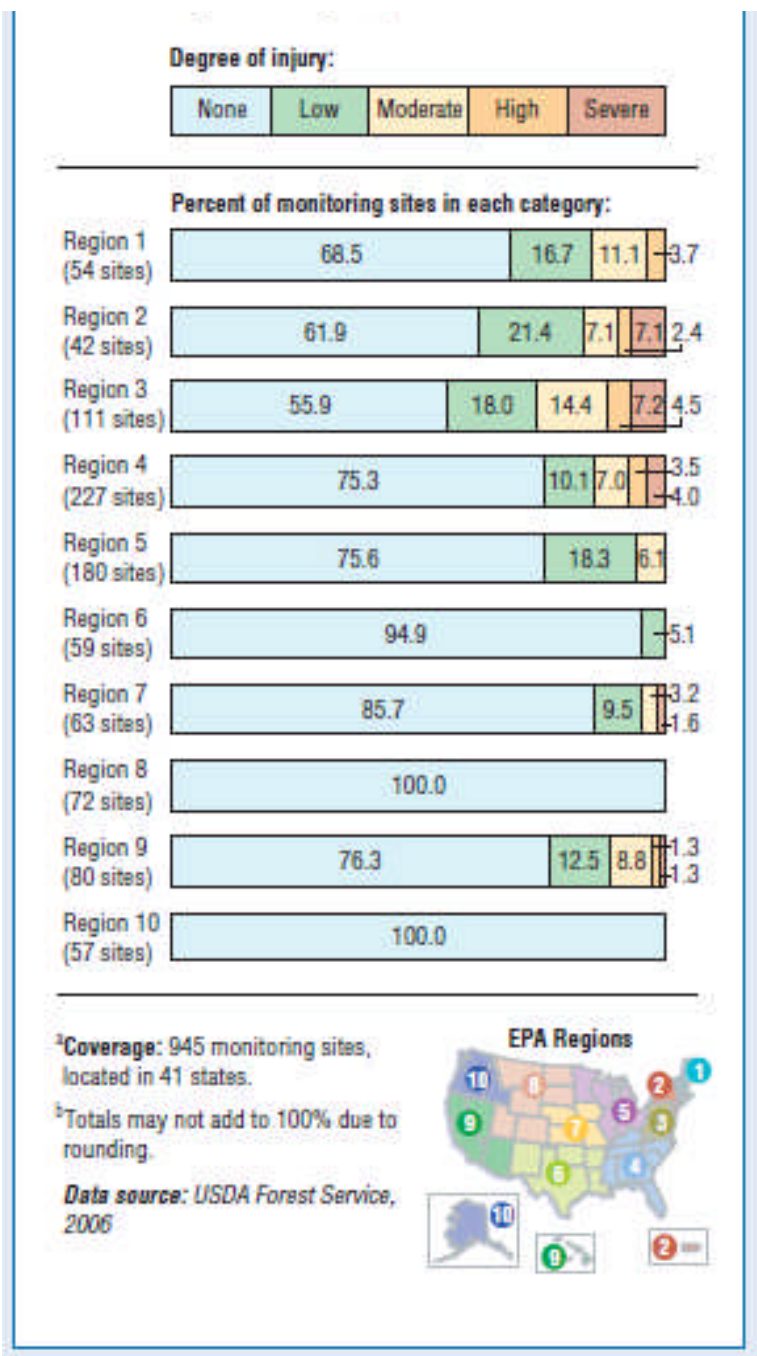
Figure 8-3 Ozone Injury to Forest Plants in U.S. by EPA Regions, 2002[ab]

8.1.2.2.1.1 Indicator Limitations

Field and laboratory studies were reviewed to identify the forest plant species in each region that are highly sensitive to ozone air pollution. Other forest plant species, or even genetic

variants of the same species, may not be harmed at ozone levels that cause effects on the selected ozone-sensitive species.

Because species distributions vary regionally, different ozone-sensitive plant species were examined in different parts of the country. These target species could vary with respect to ozone sensitivity, which might account for some of the apparent differences in ozone injury among regions of the U.S.

Ozone damage to foliage is considerably reduced under conditions of low soil moisture, but most of the variability in the index (70%) was explained by ozone concentration.[186] Ozone may have other adverse impacts on plants (e.g., reduced productivity) that do not show signs of visible foliar injury.[187]

Though FIA has extensive spatial coverage based on a robust sample design, not all forested areas in the U.S. are monitored for ozone injury. Even though the biosite data have been collected over multiple years, most biosites were not monitored over the entire period, so these data cannot provide more than a baseline for future trends.

8.1.2.3 Ozone Impacts on Forest Health

Air pollution can impact the environment and affect ecological systems, leading to changes in the biological community (both in the diversity of species and the health and vigor of individual species). As an example, many studies have shown that ground-level ozone reduces the health of plants including many commercial and ecologically important forest tree species throughout the United States.[188]

When ozone is present in the air, it can enter the leaves of plants, where it can cause significant cellular damage. Since photosynthesis occurs in cells within leaves, the ability of the plant to produce energy by photosynthesis can be compromised if enough damage occurs to these cells. If enough tissue becomes damaged it can reduce carbon fixation and increase plant respiration, leading to reduced growth and/or reproduction in young and mature trees. Ozone stress also increases the susceptibility of plants to disease, insects, fungus, and other environmental stressors (e.g., harsh weather). Because ozone damage can consist of visible injury to leaves, it also reduces the aesthetic value of ornamental vegetation and trees in urban landscapes, and negatively affects scenic vistas in protected natural areas.

Assessing the impact of ground-level ozone on forests in the eastern United States involves understanding the risks to sensitive tree species from ambient ozone concentrations and accounting for the prevalence of those species within the forest. As a way to quantify the risks to particular plants from ground-level ozone, scientists have developed ozone-exposure/tree-response functions by exposing tree seedlings to different ozone levels and measuring reductions in growth as "biomass loss." Typically, seedlings are used because they are easy to manipulate and measure their growth loss from ozone pollution. The mechanisms of susceptibility to ozone within the leaves of seedlings and mature trees are identical, though the magnitude of the effect may be higher or lower depending on the tree species. [189]

Some of the common tree species in the United States that are sensitive to ozone are black cherry (*Prunus serotina*), tulip-poplar (*Liriodendron tulipifera)*, eastern white pine (*Pinus strobus)*. Ozone-exposure/tree-response functions have been developed for each of these tree species, as well as for aspen (*Populus tremuliodes*), and ponderosa pine (*Pinus ponderosa*). Other common tree species, such as oak (*Quercus* spp.*)* and hickory (*Carya* spp.), are not nearly as sensitive to ozone. Consequently, with knowledge of the distribution of sensitive species and the level of ozone at particular locations, it is possible to estimate a "biomass loss" for each species across their range.

8.1.2.4 Particulate Matter Deposition

Particulate matter contributes to adverse effects on vegetation and ecosystems, and to soiling and materials damage. These welfare effects result predominately from exposure to excess amounts of specific chemical species, regardless of their source or predominant form (particle, gas or liquid). The following characterizations of the nature of these environmental effects are based on information contained in the 2009 PM ISA and the 2005 PM Staff Paper as well as the Integrated Science Assessment for Oxides of Nitrogen and Sulfur- Ecological Criteria.[190,191,192]

8.1.2.4.1   Deposition of Nitrogen and Sulfur

Nitrogen and sulfur interactions in the environment are highly complex. Both are essential, and sometimes limiting, nutrients needed for growth and productivity. Excesses of nitrogen or sulfur can lead to acidification, nutrient enrichment, and eutrophication of aquatic ecosystems.[193]

The process of acidification affects both freshwater aquatic and terrestrial ecosystems. Acid deposition causes acidification of sensitive surface waters. The effects of acid deposition on aquatic systems depend largely upon the ability of the ecosystem to neutralize the additional acid. As acidity increases, aluminum leached from soils and sediments, flows into lakes and streams and can be toxic to both terrestrial and aquatic biota. The lower pH concentrations and higher aluminum levels resulting from acidification make it difficult for some fish and other aquatic organisms to survive, grow, and reproduce. Research on effects of acid deposition on forest ecosystems has come to focus increasingly on the biogeochemical processes that affect uptake, retention, and cycling of nutrients within these ecosystems. Decreases in available base cations from soils are at least partly attributable to acid deposition. Base cation depletion is a cause for concern because of the role these ions play in acid neutralization, and because calcium, magnesium and potassium are essential nutrients for plant growth and physiology. Changes in the relative proportions of these nutrients, especially in comparison with aluminum concentrations, have been associated with declining forest health.

At current ambient levels, risks to vegetation from short-term exposures to dry deposited particulate nitrate or sulfate are low. However, when found in acid or acidifying deposition, such particles do have the potential to cause direct leaf injury. Specifically, the responses of forest trees to acid precipitation (rain, snow) include accelerated weathering of leaf cuticular surfaces, increased permeability of leaf surfaces to toxic materials, water, and disease agents; increased leaching of nutrients from foliage; and altered reproductive processes—all which serve to

weaken trees so that they are more susceptible to other stresses (e.g., extreme weather, pests, pathogens). Acid deposition with levels of acidity associated with the leaf effects described above are currently found in some locations in the eastern U.S.[194] Even higher concentrations of acidity can be present in occult depositions (e.g., fog, mist or clouds) which more frequently impacts higher elevations. Thus, the risk of leaf injury occurring from acid deposition in some areas of the eastern U.S. is high. Nitrogen deposition has also been shown to impact ecosystems in the western U.S. A study conducted in the Columbia River Gorge National Scenic Area (CRGNSA), located along a portion of the Oregon/Washington border, indicates that lichen communities in the CRGNSA have shifted to a higher proportion of nitrophilous species and the nitrogen content of lichen tissue is elevated.[195] Lichens are sensitive indicators of nitrogen deposition effects to terrestrial ecosystems and the lichen studies in the Columbia River Gorge clearly show that ecological effects from air pollution are occurring.

Some of the most significant detrimental effects associated with excess nitrogen deposition are those associated with a condition known as nitrogen saturation. Nitrogen saturation is the condition in which nitrogen inputs from atmospheric deposition and other sources exceed the biological requirements of the ecosystem. The effects associated with nitrogen saturation include: (1) decreased productivity, increased mortality, and/or shifts in plant community composition, often leading to decreased biodiversity in many natural habitats wherever atmospheric reactive nitrogen deposition increases significantly above background and critical thresholds are exceeded; (2) leaching of excess nitrate and associated base cations from soils into streams, lakes, and rivers, and mobilization of soil aluminum; and (3) fluctuation of ecosystem processes such as nutrient and energy cycles through changes in the functioning and species composition of beneficial soil organisms.[196]

In the U.S. numerous forests now show severe symptoms of nitrogen saturation. These forests include: the northern hardwoods and mixed conifer forests in the Adirondack and Catskill Mountains of New York; the red spruce forests at Whitetop Mountain, Virginia, and Great Smoky Mountains National Park, North Carolina; mixed hardwood watersheds at Fernow Experimental Forest in West Virginia; American beech forests in Great Smoky Mountains National Park, Tennessee; mixed conifer forests and chaparral watersheds in southern California and the southwestern Sierra Nevada in Central California; the alpine tundra/subalpine conifer forests of the Colorado Front Range; and red alder forests in the Cascade Mountains in Washington.

Excess nutrient inputs into aquatic ecosystems (i.e. streams, rivers, lakes, estuaries or oceans) either from direct atmospheric deposition, surface runoff, or leaching from nitrogen saturated soils into ground or surface waters can contribute to conditions of severe water oxygen depletion; eutrophication and algae blooms; altered fish distributions, catches, and physiological states; loss of biodiversity; habitat degradation; and increases in the incidence of disease.

Atmospheric deposition of nitrogen is a significant source of total nitrogen to many estuaries in the United States. The amount of nitrogen entering estuaries that is ultimately attributable to atmospheric deposition is not well-defined. On an annual basis, atmospheric nitrogen deposition may contribute significantly to the total nitrogen load, depending on the size and location of the watershed. In addition, episodic nitrogen inputs, which may be ecologically important, may play a more important role than indicated by the annual average concentrations.

Estuaries in the U.S. that suffer from nitrogen enrichment often experience a condition known as eutrophication. Symptoms of eutrophication include changes in the dominant species of phytoplankton, low levels of oxygen in the water column, fish and shellfish kills, outbreaks of toxic alga, and other population changes which can cascade throughout the food web. In addition, increased phytoplankton growth in the water column and on surfaces can attenuate light causing declines in submerged aquatic vegetation, which serves as an important habitat for many estuarine fish and shellfish species.

Severe and persistent eutrophication often directly impacts human activities. For example, losses in the nation's fishery resources may be directly caused by fish kills associated with low dissolved oxygen and toxic blooms. Declines in tourism occur when low dissolved oxygen causes noxious smells and floating mats of algal blooms create unfavorable aesthetic conditions. Risks to human health increase when the toxins from algal blooms accumulate in edible fish and shellfish, and when toxins become airborne, causing respiratory problems due to inhalation. According to a NOAA report, more than half of the nation's estuaries have moderate to high expressions of at least one of these symptoms – an indication that eutrophication is well developed in more than half of U.S. estuaries.[197]

8.1.2.4.2 *Deposition of Heavy Metals*

Heavy metals, including cadmium, copper, lead, chromium, mercury, nickel and zinc, have the greatest potential for impacting forest growth.[198] Investigation of trace metals near roadways and industrial facilities indicate that a substantial load of heavy metals can accumulate on vegetative surfaces. Copper, zinc, and nickel have been documented to cause direct toxicity to vegetation under field conditions. Little research has been conducted on the effects associated with mixtures of contaminants found in ambient PM. While metals typically exhibit low solubility, limiting their bioavailability and direct toxicity, chemical transformations of metal compounds occur in the environment, particularly in the presence of acidic or other oxidizing species. These chemical changes influence the mobility and toxicity of metals in the environment. Once taken up into plant tissue, a metal compound can undergo chemical changes, exert toxic effects on the plant itself, accumulate and be passed along to herbivores or can re-enter the soil and further cycle in the environment. Although there has been no direct evidence of a physiological association between tree injury and heavy metal exposures, heavy metals have been implicated because of similarities between metal deposition patterns and forest decline. This hypothesized relationship/correlation was further explored in high elevation forests in the northeastern U.S. These studies measured levels of a group of intracellular compounds found in plants that bind with metals and are produced by plants as a response to sublethal concentrations of heavy metals. These studies indicated a systematic and significant increase in concentrations of these compounds associated with the extent of tree injury. These data strongly imply that metal stress causes tree injury and contributes to forest decline in the northeastern United States.[199] Contamination of plant leaves by heavy metals can lead to elevated soil levels. Trace metals absorbed into the plant frequently bind to the leaf tissue, and then are lost when the leaf drops. As the fallen leaves decompose, the heavy metals are transferred into the soil.[200,201] Upon entering the soil environment, PM pollutants can alter ecological processes of energy flow and nutrient cycling, inhibit nutrient uptake, change ecosystem structure, and affect ecosystem biodiversity. Many of the most important effects occur in the soil. The soil environment is one of the most dynamic sites of biological interaction in nature. It is inhabited by microbial

communities of bacteria, fungi, and actinomycetes. These organisms are essential participants in the nutrient cycles that make elements available for plant uptake. Changes in the soil environment that influence the role of the bacteria and fungi in nutrient cycling determine plant and ultimately ecosystem response.[202]

The environmental sources and cycling of mercury are currently of particular concern due to the bioaccumulation and biomagnification of this metal in aquatic ecosystems and the potent toxic nature of mercury in the forms in which is it ingested by people and other animals. Mercury is unusual compared with other metals in that it largely partitions into the gas phase (in elemental form), and therefore has a longer residence time in the atmosphere than a metal found predominantly in the particle phase. This property enables mercury to travel far from the primary source before being deposited and accumulating in the aquatic ecosystem. The major source of mercury in the Great Lakes is from atmospheric deposition, accounting for approximately eighty percent of the mercury in Lake Michigan.[203,204] Over fifty percent of the mercury in the Chesapeake Bay has been attributed to atmospheric deposition.[205] Overall, the National Science and Technology Council identifies atmospheric deposition as the primary source of mercury to aquatic systems.[206] Forty-four states have issued health advisories for the consumption of fish contaminated by mercury; however, most of these advisories are issued in areas without a mercury point source.

Elevated levels of zinc and lead have been identified in streambed sediments, and these elevated levels have been correlated with population density and motor vehicle use.[207,208] Zinc and nickel have also been identified in urban water and soils. In addition, platinum, palladium, and rhodium, metals found in the catalysts of modern motor vehicles, have been measured at elevated levels along roadsides.[209] Plant uptake of platinum has been observed at these locations.

8.1.2.4.3   Deposition of Polycyclic Organic Matter

Polycyclic organic matter (POM) is a byproduct of incomplete combustion and consists of organic compounds with more than one benzene ring and a boiling point greater than or equal to 100 degrees centigrade.[210] Polycyclic aromatic hydrocarbons (PAHs) are a class of POM that contains compounds which are known or suspected carcinogens.

Major sources of PAHs include mobile sources. PAHs in the environment may be present as a gas or adsorbed onto airborne particulate matter. Since the majority of PAHs are adsorbed onto particles less than 1.0 μm in diameter, long range transport is possible. However, studies have shown that PAH compounds adsorbed onto diesel exhaust particulate and exposed to ozone have half lives of 0.5 to 1.0 hours.[211]

Since PAHs are insoluble, the compounds generally are particle reactive and accumulate in sediments. Atmospheric deposition of particles is believed to be the major source of PAHs to the sediments of Lake Michigan.[212,213] Analyses of PAH deposition in Chesapeake and Galveston Bay indicate that dry deposition and gas exchange from the atmosphere to the surface water predominate.[214,215] Sediment concentrations of PAHs are high enough in some segments of Tampa Bay to pose an environmental health threat. EPA funded a study to better characterize the sources and loading rates for PAHs into Tampa Bay.[216] PAHs that enter a water body through gas exchange likely partition into organic rich particles and can be biologically recycled,

while dry deposition of aerosols containing PAHs tend to be more resistant to biological recycling.[217] Thus, dry deposition is likely the main pathway for PAH concentrations in sediments while gas/water exchange at the surface may lead to PAH distribution into the food web, leading to increased health risk concerns.

Trends in PAH deposition levels are difficult to discern because of highly variable ambient air concentrations, lack of consistency in monitoring methods, and the significant influence of local sources on deposition levels.[218] Van Metre et al. noted PAH concentrations in urban reservoir sediments have increased by 200-300% over the last forty years and correlate with increases in automobile use.[219]

Cousins et al. estimate that more than ninety percent of semi-volatile organic compound (SVOC) emissions in the United Kingdom deposit on soil.[220] An analysis of PAH concentrations near a Czechoslovakian roadway indicated that concentrations were thirty times greater than background.[221]

8.1.2.4.4   *Materials Damage and Soiling*

The effects of the deposition of atmospheric pollution, including ambient PM, on materials are related to both physical damage and impaired aesthetic qualities. The deposition of PM (especially sulfates and nitrates) can physically affect materials, adding to the effects of natural weathering processes, by potentially promoting or accelerating the corrosion of metals, by degrading paints, and by deteriorating building materials such as concrete and limestone. Only chemically active fine particles or hygroscopic coarse particles contribute to these physical effects. In addition, the deposition of ambient PM can reduce the aesthetic appeal of buildings and culturally important articles through soiling. Particles consisting primarily of carbonaceous compounds cause soiling of commonly used building materials and culturally important items such as statues and works of art.

8.1.2.5 Environmental Effects of Air Toxics

Emissions from producing, transporting and combusting fuel contribute to ambient levels of pollutants that contribute to adverse effects on vegetation. Volatile organic compounds (VOCs), some of which are considered air toxics, have long been suspected to play a role in vegetation damage.[222] In laboratory experiments, a wide range of tolerance to VOCs has been observed.[223] Decreases in harvested seed pod weight have been reported for the more sensitive plants, and some studies have reported effects on seed germination, flowering and fruit ripening. Effects of individual VOCs or their role in conjunction with other stressors (e.g., acidification, drought, temperature extremes) have not been well studied. In a recent study of a mixture of VOCs including ethanol and toluene on herbaceous plants, significant effects on seed production, leaf water content and photosynthetic efficiency were reported for some plant species.[224]

Research suggests an adverse impact of vehicle exhaust on plants, which has in some cases been attributed to aromatic compounds and in other cases to nitrogen oxides.[225,226,227] The impacts of VOCs on plant reproduction may have long-term implications for biodiversity and survival of native species near major roadways. Most of the studies of the impacts of VOCs on vegetation have focused on short-term exposure and few studies have focused on long-term

effects of VOCs on vegetation and the potential for metabolites of these compounds to affect herbivores or insects.

8.2 Air Quality Impacts of Non-GHG Pollutants

8.2.1 Introduction

Chapter 6 of this DRIA presents the projected emissions changes due to the proposed rule. Once the emissions changes are projected the next step is to look at how the ambient air quality would be impacted by those emissions changes. Although the purpose of this proposal is to address greenhouse gas emissions, this proposed rule would also impact emissions of criteria and hazardous air pollutants. Section 8.2.2 describes current ambient levels of PM, ozone, and some air toxics without the standards being proposed in this rule. No air quality modeling was done for this DRIA to project the impacts of the proposed rule. Air quality modeling will be done for the final rule, however, and those plans are discussed in Section 8.2.3.

8.2.2 Current Levels of Pollutants

8.2.2.1 Particulate Matter

As described in Section 8.1.1.1, PM causes adverse health effects, and the EPA has set national standards to provide requisite protection against those health effects. There are two National Ambient Air Quality Standards (NAAQS) for $PM_{2.5}$: an annual standard (15 $\mu g/m^3$) and a 24-hour standard (35 $\mu g/m^3$). The most recent revisions to these standards were in 1997 and 2006. In 2005 the U.S. EPA designated nonattainment areas for the 1997 $PM_{2.5}$ NAAQS (70 FR 19844, April 14, 2005).[N] As of January 6, 2010, approximately 88 million people live in the 39 areas that are designated as nonattainment for the 1997 $PM_{2.5}$ NAAQS. These $PM_{2.5}$ nonattainment areas are comprised of 208 full or partial counties. On October 8, 2009, the EPA issued final nonattainment area designations for the 2006 24-hour $PM_{2.5}$ NAAQS (74 FR 58688, November 13, 2009). These designations include 31 areas composed of 120 full or partial counties with a population of over 70 million. In total, there are 54 $PM_{2.5}$ nonattainment areas composed of 245 counties with a population of 101 million people.

States with $PM_{2.5}$ nonattainment areas will be required to take action to bring those areas into compliance in the future. Most 1997 $PM_{2.5}$ nonattainment areas are required to attain the 1997 $PM_{2.5}$ NAAQS in the 2010 to 2015 time frame and then be required to maintain the 1997 $PM_{2.5}$ NAAQS thereafter.[228] The 2006 24-hour $PM_{2.5}$ nonattainment areas will be required to attain the 2006 24-hour $PM_{2.5}$ NAAQS in the 2014 to 2019 time frame and then be required to maintain the 2006 24-hour $PM_{2.5}$ NAAQS thereafter.[229] The heavy-duty vehicle standards proposed here first apply to model year 2014 vehicles.

[N] A nonattainment area is defined in the Clean Air Act (CAA) as an area that is violating an ambient standard or is contributing to a nearby area that is violating the standard.

8.2.2.2 Ozone

As described in Section 8.1.1.3, ozone causes adverse health effects, and the EPA has set national standards to protect against those health effects. The primary and secondary NAAQS for ozone are 8-hour standards set at 0.075 ppm. The most recent revision to the ozone standards was in 2008; the previous 8-hour ozone standards, set in 1997, had been set at 0.08 ppm. In 2004, the U.S. EPA designated nonattainment areas for the 1997 8-hour ozone NAAQS (69 FR 23858, April 30, 2004). As of January 6, 2010, there are 51 8-hour ozone nonattainment areas for the 1997 ozone NAAQS composed of 266 full or partial counties with a total population of over 122 million. On January 6, 2010, EPA proposed to reconsider the 2008 ozone NAAQS to ensure that they are requisite to protect public health with an ample margin of safety, and requisite to protect public welfare. EPA intends to complete the reconsideration by August 31, 2010. If, as a result of the reconsideration, EPA promulgates different ozone standards, the new 2010 ozone standards would replace the 2008 ozone standards and the requirement to designate areas for the replaced 2008 standards would no longer apply. Because of the significant uncertainty the reconsideration proposal creates regarding the continued applicability of the 2008 ozone NAAQS, EPA has extended the deadline for designating areas for the 2008 NAAQS by one year. This will allow EPA to complete its reconsideration of the 2008 ozone NAAQS before determining whether designations for those standards are necessary.

If EPA promulgates new ozone standards in 2010, EPA intends to accelerate the designations process for the primary standard so that the designations would be effective in August 2011. EPA is considering two alternative schedules for designating areas for a new seasonal secondary standard, an accelerated schedule or a 2-year schedule.

States with ozone nonattainment areas are required to take action to bring those areas into compliance in the future. The attainment date assigned to an ozone nonattainment area is based on the area's classification. Most ozone nonattainment areas are required to attain the 1997 8-hour ozone NAAQS in the 2007 to 2013 time frame and then be required to maintain it thereafter.[O] In addition, there will be attainment dates associated with the designation of nonattainment areas as a result of the reconsideration of the 2008 ozone NAAQS. If the ozone NAAQS reconsideration action is completed on the proposed schedule, the primary NAAQS attainment dates would be in the 2014-2031 time frame. The heavy-duty vehicle standards proposed here first apply to model year 2014 vehicles.

[O] The Los Angeles South Coast Air Basin 8-hour ozone nonattainment area is designated as severe and will have to attain before June 15, 2021. The South Coast Air Basin has requested to be reclassified as an extreme nonattainment area which will make their attainment date June 15, 2024. The San Joaquin Valley Air Basin 8-hour ozone nonattainment area is designated as serious and will have to attain before June 15, 2013. The San Joaquin Valley Air Basin has requested to be reclassified as an extreme nonattainment area which will make their attainment date June 15, 2024.

8.2.2.3 Air Toxics

The majority of Americans continue to be exposed to ambient concentrations of air toxics at levels which have the potential to cause adverse health effects.[230] The levels of air toxics to which people are exposed vary depending on where people live and work and the kinds of activities in which they engage, as discussed in detail in U.S. EPA's most recent Mobile Source Air Toxics (MSAT) Rule.[231] According to the National Air Toxics Assessment (NATA) for 2002, mobile sources were responsible for 47 percent of outdoor toxic emissions and over 50 percent of the cancer risk.[232] Nearly the entire U.S. population was exposed to an average concentration of air toxics that has the potential for adverse noncancer respiratory health effects. EPA recently finalized vehicle and fuel controls to reduce mobile source air toxics.[233] In addition, over the years, EPA has implemented a number of mobile source and fuel controls resulting in VOC reductions, which also reduce air toxic emissions. Modeling from the recent MSAT rule suggests that the mobile source contribution to ambient benzene concentrations is projected to decrease over 40% by 2015, with a decrease in ambient benzene concentration from all sources of about 25%. Although benzene is used as an example, the downward trend is projected for other air toxics as well. See the RIA for the final MSAT rule for more information on ambient air toxics projections.[234]

8.2.3   Impacts of Future Air Quality

Air quality models use mathematical and numerical techniques to simulate the physical and chemical processes that affect air pollutants as they disperse and react in the atmosphere. Based on inputs of meteorological data and source information, these models are designed to characterize primary pollutants that are emitted directly into the atmosphere and secondary pollutants that are formed as a result of complex chemical reactions within the atmosphere. Photochemical air quality models have become widely recognized and routinely utilized tools for regulatory analysis by assessing the effectiveness of control strategies. These models are applied at multiple spatial scales from local, regional, national, and global.

Full-scale photochemical air quality modeling is necessary to accurately project levels of criteria and air toxic pollutants. For the final rule, a national-scale air quality modeling analysis will be performed to analyze the impacts of the standards on $PM_{2.5}$, ozone, and selected air toxics (i.e., benzene, formaldehyde, acetaldehyde, acrolein and 1,3-butadiene). The length of time needed to prepare the necessary emissions inventories, in addition to the processing time associated with the modeling itself, has precluded us from performing air quality modeling for this proposal.

Section VII of the preamble presents projections of the changes in criteria pollutant and air toxics emissions due to the proposed standards; the basis for those estimates is set out in Chapter 6 of the DRIA. The atmospheric chemistry related to ambient concentrations of $PM_{2.5}$, ozone and air toxics is very complex, and making predictions based solely on emissions changes is extremely difficult. However, based on the magnitude of the emissions changes predicted to result from the proposed standards, we expect that there will be a relatively small change in ambient air quality, pending a more comprehensive analysis for the final rule.

For the final rule, EPA intends to use a 2005-based Community Multi-scale Air Quality (CMAQ) modeling platform as the tool for the air quality modeling. The CMAQ modeling system is a comprehensive three-dimensional grid-based Eulerian air quality model designed to estimate the formation and fate of oxidant precursors, primary and secondary PM concentrations and deposition, and air toxics, over regional and urban spatial scales (e.g., over the contiguous U.S.).[235,236,237] The CMAQ model is a well-known and well-established tool and is commonly used by EPA for regulatory analyses, for instance the recent ozone NAAQS proposal, and by States in developing attainment demonstrations for their State Implementation Plans.[238] The CMAQ model version 4.7 was most recently peer-reviewed in February of 2009 for the U.S. EPA.[239]

CMAQ includes many science modules that simulate the emission, production, decay, deposition and transport of organic and inorganic gas-phase and particle-phase pollutants in the atmosphere. EPA intends to use the most recent version of CMAQ which reflects updates to version 4.7 to improve the underlying science. These include aqueous chemistry mass conservation improvements, improved vertical convective mixing and lowered CB05 mechanism unit yields for acrolein from 1,3-butadiene tracer reactions which were updated to be consistent with laboratory measurements.

The CMAQ modeling domain will encompass all of the lower 48 States and portions of Canada and Mexico. The modeling domain will include a large continental U.S. 36 km grid and two 12 km grids (an Eastern U.S. and a Western U.S. domain), as shown in Figure 8-4. The modeling domain will contain 14 vertical layers with the top of the modeling domain at about 16,200 meters, or 100 millibars (mb).

Figure 8-4 CMAQ 12-km Eastern and Western US Modeling Domains
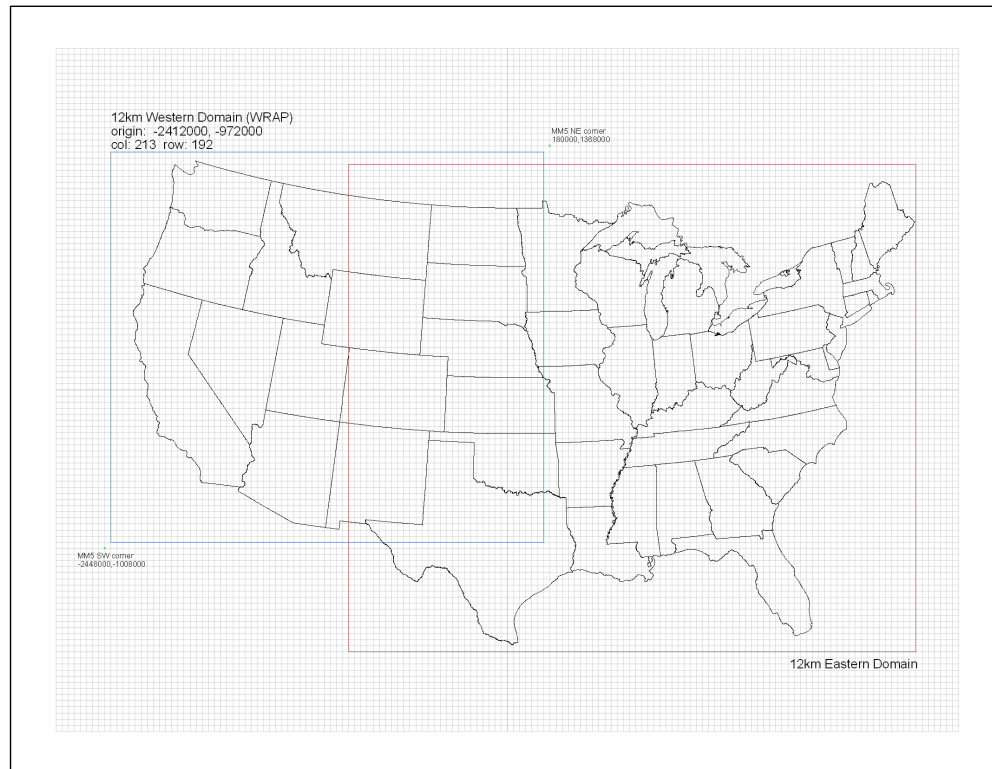
The key inputs to the CMAQ model include emissions from anthropogenic and biogenic sources, meteorological data, and initial and boundary conditions. The CMAQ meteorological input files will be derived from simulations of the Pennsylvania State University / National Center for Atmospheric Research Mesoscale Model[240] for the entire year of 2005. This model, commonly referred to as MM5, is a limited-area, nonhydrostatic, terrain-following system that solves for the full set of physical and thermodynamic equations which govern atmospheric motions.[241] The meteorology for the national 36 km grid and the 12 km Eastern and Western U.S. grids will be developed by EPA and described in more detail within the final RIA and the technical support document for the final rule air quality modeling.

The lateral boundary and initial species concentrations will be provided by a three-dimensional global atmospheric chemistry model, the GEOS-CHEM model.[242] The global GEOS-CHEM model simulates atmospheric chemical and physical processes driven by assimilated meteorological observations from the NASA's Goddard Earth Observing System (GEOS). This model will be run for 2005 with a grid resolution of 2 degree x 2.5 degree (latitude-longitude) and 20 vertical layers. The predictions will be used to provide one-way dynamic boundary conditions at three-hour intervals and an initial concentration field for the 36 km CMAQ simulations. The future base conditions from the 36 km coarse grid modeling will be used as the initial/boundary state for all subsequent 12 km finer grid modeling.

8.3 Quantified and Monetized Non-GHG Health and Environmental Impacts

This section discusses the non-GHG health and environmental impacts that can be expected to occur as a result of the proposed heavy-duty vehicle GHG rule. GHG emissions are predominantly the byproduct of fossil fuel combustion processes that also produce criteria and hazardous air pollutants. The vehicles that are subject to the proposed standards are also significant sources of mobile source air pollution such as direct PM, NO_x, VOCs and air toxics. The proposed standards would affect exhaust emissions of these pollutants from vehicles. They would also affect emissions from upstream sources related to changes in fuel consumption. Changes in ambient ozone, $PM_{2.5}$, and air toxics that would result from the proposed standards are expected to affect human health in the form of premature deaths and other serious human health effects, as well as other important public health and welfare effects.

It is important to quantify the health and environmental impacts associated with the proposed standard because a failure to adequately consider these ancillary co-pollutant impacts could lead to an incorrect assessment of their net costs and benefits. Moreover, co-pollutant impacts tend to accrue in the near term, while any effects from reduced climate change mostly accrue over a time frame of several decades or longer.

EPA typically quantifies and monetizes the health and environmental impacts related to both PM and ozone in its regulatory impact analyses (RIAs), when possible. However, EPA was unable to do so in time for this proposal. EPA attempts to make emissions and air quality modeling decisions early in the analytical process so that we can complete the photochemical air quality modeling and use that data to inform the health and environmental impacts analysis. Resource and time constraints precluded the Agency from completing this work in time for the proposal. Instead, we provide a characterization of the health and environmental impacts that will be quantified and monetized for the final rulemaking.

EPA bases its analyses on peer-reviewed studies of air quality and health and welfare effects and peer-reviewed studies of the monetary values of public health and welfare improvements, and is generally consistent with benefits analyses performed for the analysis of the final Ozone National Ambient Air Quality Standard (NAAQS) and the final PM NAAQS analysis, as well as the proposed Portland Cement National Emissions Standards for Hazardous Air Pollutants (NESHAP) RIA, and final NO_2 NAAQS.[243,244, 245,246]

8.3.1 Human Health and Environmental Impacts

To model the ozone and PM air quality benefits of the final rule, EPA will use the Community Multiscale Air Quality (CMAQ) model (see Section 8.2.3 for a description of the CMAQ model). The modeled ambient air quality data will serve as an input to the Environmental Benefits Mapping and Analysis Program (BenMAP).[247] BenMAP is a computer program developed by EPA that integrates a number of the modeling elements used in previous RIAs (e.g., interpolation functions, population projections, health impact functions, valuation functions, analysis and pooling methods) to translate modeled air concentration estimates into health effects incidence estimates and monetized benefits estimates.

Table 8-1 lists the co-pollutant health effect exposure-response functions we will use to quantify the co-pollutant incidence impacts associated with the final heavy-duty vehicles standard.

(Table 8-1 is on the following page)

Table 8-1: Health Impact Functions Used in BenMAP to Estimate Impacts of PM$_{2.5}$ and Ozone Reductions

ENDPOINT	*POLLUTANT*	*STUDY*	*STUDY POPULATION*
Premature Mortality			
Premature mortality – daily time series	O$_3$	Multi-city Bell et al (2004) (NMMAPS study)[248] – Non-accidental Huang et al (2005)[249] - Cardiopulmonary Schwartz (2005)[250] – Non-accidental Meta-analyses: Bell et al (2005)[251] – All cause Ito et al (2005)[252] – Non-accidental Levy et al (2005)[253] – All cause	All ages
Premature mortality —cohort study, all-cause	PM$_{2.5}$	Pope et al. (2002)[254] Laden et al. (2006)[255]	>29 years >25 years
Premature mortality, total exposures	PM$_{2.5}$	Expert Elicitation (IEc, 2006)[256]	>24 years
Premature mortality — all-cause	PM$_{2.5}$	Woodruff et al. (1997)[257]	Infant (<1 year)
Chronic Illness			
Chronic bronchitis	PM$_{2.5}$	Abbey et al. (1995)[258]	>26 years
Nonfatal heart attacks	PM$_{2.5}$	Peters et al. (2001)[259]	Adults (>18 years)
Hospital Admissions			
Respiratory	O$_3$	Pooled estimate: Schwartz (1995) - ICD 460-519 (all resp)[260] Schwartz (1994a; 1994b) - ICD 480-486 (pneumonia)[261,262] Moolgavkar et al. (1997) - ICD 480-487 (pneumonia)[263] Schwartz (1994b) - ICD 491-492, 494-496 (COPD) Moolgavkar et al. (1997) – ICD 490-496 (COPD)	>64 years
		Burnett et al. (2001)[264]	<2 years
	PM$_{2.5}$	Pooled estimate: Moolgavkar (2003)—ICD 490-496 (COPD)[265] Ito (2003)—ICD 490-496 (COPD)[266]	>64 years
	PM$_{2.5}$	Moolgavkar (2000)—ICD 490-496 (COPD)[267]	20–64 years
	PM$_{2.5}$	Ito (2003)—ICD 480-486 (pneumonia)	>64 years
	PM$_{2.5}$	Sheppard (2003)—ICD 493 (asthma)[268]	<65 years
Cardiovascular	PM$_{2.5}$	Pooled estimate: Moolgavkar (2003)—ICD 390-429 (all cardiovascular) Ito (2003)—ICD 410-414, 427-428 (ischemic heart disease, dysrhythmia, heart failure)	>64 years
	PM$_{2.5}$	Moolgavkar (2000)—ICD 390-429 (all cardiovascular)	20–64 years

Asthma-related ER visits	O_3	Pooled estimate: Peel et al (2005)[269] Wilson et al (2005)[270]	All ages All ages
Asthma-related ER visits (con't)	$PM_{2.5}$	Norris et al. (1999)[271]	0–18 years
Other Health Endpoints			
Acute bronchitis	$PM_{2.5}$	Dockery et al. (1996)[272]	8–12 years
Upper respiratory symptoms	$PM_{2.5}$	Pope et al. (1991)[273]	Asthmatics, 9–11 years
Lower respiratory symptoms	$PM_{2.5}$	Schwartz and Neas (2000)[274]	7–14 years
Asthma exacerbations	$PM_{2.5}$	Pooled estimate: Ostro et al. (2001)[275] (cough, wheeze and shortness of breath) Vedal et al. (1998)[276] (cough)	6–18 years[a]
Work loss days	$PM_{2.5}$	Ostro (1987)[277]	18–65 years
School absence days	O_3	Pooled estimate: Gilliland et al. (2001)[278] Chen et al. (2000)[279]	5–17 years[b]
Minor Restricted Activity Days (MRADs)	O_3	Ostro and Rothschild (1989)[280]	18–65 years
	$PM_{2.5}$	Ostro and Rothschild (1989)	18–65 years

Notes:
[a] The original study populations were 8 to 13 for the Ostro et al. (2001) study and 6 to 13 for the Vedal et al. (1998) study. Based on advice from the Science Advisory Board Health Effects Subcommittee (SAB-HES), we extended the applied population to 6 to 18, reflecting the common biological basis for the effect in children in the broader age group. See: U.S. Science Advisory Board. 2004. Advisory Plans for Health Effects Analysis in the Analytical Plan for EPA's Second Prospective Analysis –Benefits and Costs of the Clean Air Act, 1990—2020. EPA-SAB-COUNCIL-ADV-04-004. See also National Research Council (NRC). 2002. *Estimating the Public Health Benefits of Proposed Air Pollution Regulations*. Washington, DC: The National Academies Press.
[b] Gilliland et al. (2001) studied children aged 9 and 10. Chen et al. (2000) studied children 6 to 11. Based on recent advice from the National Research Council and the EPA SAB-HES, we have calculated reductions in school absences for all school-aged children based on the biological similarity between children aged 5 to 17.

8.3.2 Monetized Impacts

Table 8-2 presents the monetary values we will apply to changes in the incidence of health and welfare effects associated with reductions in non-GHG pollutants that will occur when these GHG control strategies are finalized.

Table 8-2: Valuation Metrics Used in BenMAP to Estimate Monetary Co-Benefits

Endpoint	Valuation Method	Valuation (2000$)
Premature mortality	Assumed Mean VSL	$6,300,000
Chronic Illness		
Chronic Bronchitis	WTP: Average Severity	$340,482
Myocardial Infarctions, Nonfatal	Medical Costs Over 5 Years. Varies by age and discount rate. Russell (1998)[281]	---

	Medical Costs Over 5 Years. Varies by age and discount rate. Wittels (1990)[282]	---
Hospital Admissions		
Respiratory, Age 65+	COI: Medical Costs + Wage Lost	$18,353
Respiratory, Ages 0-2	COI: Medical Costs	$7,741
Chronic Lung Disease (less Asthma)	COI: Medical Costs + Wage Lost	$12,378
Pneumonia	COI: Medical Costs + Wage Lost	$14,693
Asthma	COI: Medical Costs + Wage Lost	$6,634
Cardiovascular	COI: Medical Costs + Wage Lost (20-64)	$22,778
	COI: Medical Costs + Wage Lost (65-99)	$21,191
ER Visits, Asthma	COI: Smith et al. (1997)[283]	$312
	COI: Standford et al. (1999)[284]	$261
Other Health Endpoints		
Acute Bronchitis	WTP: 6 Day Illness, CV Studies	$356
Upper Respiratory Symptoms	WTP: 1 Day, CV Studies	$25
Lower Respiratory Symptoms	WTP: 1 Day, CV Studies	$16
Asthma Exacerbation	WTP: Bad Asthma Day, Rowe and Chestnut (1986) [285]	$43
Work Loss Days	Median Daily Wage, County-Specific	---
Minor Restricted Activity Days	WTP: 1 Day, CV Studies	$51
School Absence Days	Median Daily Wage, Women 25+	$75
Worker Productivity	Median Daily Wage, Outdoor Workers, County-Specific	---
Environmental Endpoints		
Recreational Visibility	WTP: 86 Class I Areas	---

Source: Dollar amounts for each valuation method were extracted from BenMAP version 3.0.

8.3.3 Other Unquantified Health and Environmental Impacts

In addition to the co-pollutant health and environmental impacts we will quantify for the analysis of the heavy-duty vehicle GHG standard, there are a number of other health and human welfare endpoints that we will not be able to quantify because of current limitations in the methods or available data. These impacts are associated with emissions of air toxics (including benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and acrolein), ambient ozone, and ambient PM$_{2.5}$ exposures. For example, we have not quantified a number of known or suspected health effects linked with ozone and PM for which appropriate health impact functions are not available or which do not provide easily interpretable outcomes (e.g., changes in heart rate variability).   In addition, we are currently unable to quantify a number of known welfare effects, including reduced acid and particulate deposition damage to cultural monuments and other materials, and environmental benefits due to reductions of impacts of eutrophication in coastal areas. Table 8-3 lists these unquantified health and environmental impacts.

Table 8-3: Unquantified and Non-Monetized Potential Effects

POLLUTANT/EFFECTS	EFFECTS NOT INCLUDED IN ANALYSIS - CHANGES IN:
Ozone Health[a]	Chronic respiratory damage Premature aging of the lungs Non-asthma respiratory emergency room visits

	Exposure to UVb (+/-)[d]
Ozone Welfare	Yields for -commercial forests -some fruits and vegetables -non-commercial crops Damage to urban ornamental plants Impacts on recreational demand from damaged forest aesthetics Ecosystem functions Exposure to UVb (+/-)
PM Health[b]	Premature mortality - short term exposures[c] Low birth weight Pulmonary function Chronic respiratory diseases other than chronic bronchitis Non-asthma respiratory emergency room visits Exposure to UVb (+/-)
PM Welfare	Residential and recreational visibility in non-Class I areas Soiling and materials damage Damage to ecosystem functions Exposure to UVb (+/-)
Nitrogen and Sulfate Deposition Welfare	Commercial forests due to acidic sulfate and nitrate deposition Commercial freshwater fishing due to acidic deposition Recreation in terrestrial ecosystems due to acidic deposition Existence values for currently healthy ecosystems Commercial fishing, agriculture, and forests due to nitrogen deposition Recreation in estuarine ecosystems due to nitrogen deposition Ecosystem functions Passive fertilization
CO Health	Behavioral effects
Hydrocarbon (HC)/Toxics Health[e]	Cancer (benzene, 1,3-butadiene, formaldehyde, acetaldehyde) Anemia (benzene) Disruption of production of blood components (benzene) Reduction in the number of blood platelets (benzene) Excessive bone marrow formation (benzene) Depression of lymphocyte counts (benzene) Reproductive and developmental effects (1,3-butadiene) Irritation of eyes and mucus membranes (formaldehyde) Respiratory irritation (formaldehyde) Asthma attacks in asthmatics (formaldehyde) Asthma-like symptoms in non-asthmatics (formaldehyde) Irritation of the eyes, skin, and respiratory tract (acetaldehyde) Upper respiratory tract irritation and congestion (acrolein)
HC/Toxics Welfare[f]	Direct toxic effects to animals Bioaccumulation in the food chain Damage to ecosystem function Odor

Notes:

[a] In addition to primary economic endpoints, there are a number of biological responses that have been associated with ozone health effects including increased airway responsiveness to stimuli, inflammation in the lung, acute inflammation and respiratory cell damage, and increased susceptibility to respiratory infection. The public health impact of these biological responses may be partly represented by our quantified endpoints.

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

[c] While some of the effects of short-term exposures are likely to be captured in the estimates, there may be premature mortality due to short-term exposure to PM not captured in the cohort studies used in this analysis. However, the PM mortality results derived from the expert elicitation do take into account premature mortality effects of short term exposures.

[d] May result in benefits or disbenefits.

[e] Many of the key hydrocarbons related to this rule are also hazardous air pollutants listed in the Clean Air Act. Please refer to Chapter 8.1.1 for additional information on the health effects of air toxics.

[f] Please refer to Chapter 8.1.2 for additional information on the welfare effects of air toxics.

While there will be impacts associated with air toxic pollutant emission changes that result from the final standard, we will not attempt to monetize those impacts. This is primarily because currently available tools and methods to assess air toxics risk from mobile sources at the national scale are not adequate for extrapolation to incidence estimations or benefits assessment. The best suite of tools and methods currently available for assessment at the national scale are those used in the National-Scale Air Toxics Assessment (NATA). The EPA Science Advisory Board specifically commented in their review of the 1996 NATA that these tools were not yet ready for use in a national-scale benefits analysis, because they did not consider the full distribution of exposure and risk, or address sub-chronic health effects.[286]   While EPA has since improved the tools, there remain critical limitations for estimating incidence and assessing benefits of reducing mobile source air toxics. EPA continues to work to address these limitations; however, we do not anticipate having methods and tools available for national-scale application in time for the analysis of the final rules.[287]

8.4 Changes in Atmospheric CO_2 Concentrations, Global Mean Temperature, Sea Level Rise, and Ocean pH Associated with the Proposal's GHG Emissions Reductions

8.4.1 Introduction

Based on modeling analysis performed by the EPA, reductions in CO_2 and other GHGs associated with the proposed rule will affect climate change projections. Since GHGs are well-mixed in the atmosphere and have long atmospheric lifetimes, changes in GHG emissions will affect atmospheric concentrations of greenhouse gases and future climate for decades to centuries and even millennia. This section provides estimates of the projected change in atmospheric CO_2 concentrations based on the emission reductions estimated for this proposed rule. In addition, this section analyzes the following climate-related variables: global mean surface temperature, sea level rise, and ocean pH. Provided here are estimates for the response in atmospheric CO_2 concentrations, global mean surface temperature, sea level rise, and ocean pH projections to the estimated net global GHG emissions reductions associated with this proposed rule (see Chapter 5 for the estimated net reductions in global emissions over time by GHG).

8.4.2 Estimated Projected Reductions in Atmospheric CO_2 Concentrations, Global Mean Surface Temperatures and Sea Level Rise

To assess the impact of the emissions reductions from the proposed rule, EPA estimated changes in projected atmospheric CO_2 concentrations, global mean surface temperature and sea-level rise to 2100 using the GCAM (Global Change Assessment Model, formerly MiniCAM), integrated assessment model[P] coupled with the MAGICC (Model for the Assessment of Greenhouse-gas Induced Climate Change) simple climate model.[Q] GCAM was used to create the globally and temporally consistent set of climate relevant variables required for running MAGICC. MAGICC was then used to estimate the projected change in these variables over time. Given the magnitude of the estimated emissions reductions associated with the rule, a simple climate model such as MAGICC is reasonable for estimating the atmospheric and climate response.

An emissions scenario for the rule was developed by applying the rule's estimated emissions reductions to the GCAM reference (no climate policy or baseline) scenario (used as the basis for the Representative Concentration Pathway RCP4.5[R]). Specifically, the CO_2, N_2O,

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[P] MiniCAM is a long-term, global integrated assessment model of energy, economy, agriculture and land use, that considers the sources of emissions of a suite of greenhouse gases (GHG's), emitted in 14 globally disaggregated regions, the fate of emissions to the atmosphere, and the consequences of changing concentrations of greenhouse related gases for climate change. MiniCAM begins with a representation of demographic and economic developments in each region and combines these with assumptions about technology development to describe an internally consistent representation of energy, agriculture, land-use, and economic developments that in turn shape global emissions.

Brenkert A, S. Smith, S. Kim, and H. Pitcher, 2003: Model Documentation for the MiniCAM. PNNL-14337, Pacific Northwest National Laboratory, Richland, Washington.

[Q] MAGICC consists of a suite of coupled gas-cycle, climate and ice-melt models integrated into a single framework. The framework allows the user to determine changes in greenhouse-gas concentrations, global-mean surface air temperature and sea-level resulting from anthropogenic emissions of carbon dioxide (CO2), methane (CH4), nitrous oxide (N2O), reactive gases (CO, NOx, VOCs), the halocarbons (e.g. HCFCs, HFCs, PFCs) and sulfur dioxide (SO2). MAGICC emulates the global-mean temperature responses of more sophisticated coupled Atmosphere/Ocean General Circulation Models (AOGCMs) with high accuracy.

Wigley, T.M.L. and Raper, S.C.B. 1992. Implications for Climate And Sea-Level of Revised IPCC Emissions Scenarios Nature 357, 293-300. Raper, S.C.B., Wigley T.M.L. and Warrick R.A. 1996. in Sea-Level Rise and Coastal Subsidence: Causes, Consequences and Strategies J.D. Milliman, B.U. Haq, Eds., Kluwer Academic Publishers, Dordrecht, The Netherlands, pp. 11-45.

Wigley, T.M.L. and Raper, S.C.B. 2002. Reasons for larger warming projections in the IPCC Third Assessment Report J. Climate 15, 2945-2952.

[R] This scenario is used because it contains a comprehensive suite of greenhouse and pollutant gas emissions. The four RCP scenarios will be used as common inputs into a variety of Earth System Models for inter-model comparisons leading to the IPCC AR5 (Moss et al. 2008). The MiniCAM RCP4.5 is based on the scenarios presented in Clarke et al. (2007) with non-CO2 and pollutant gas emissions implemented as described in Smith and Wigley (2006). Base-year information has been updated to the latest available data for the RCP process. The final RCP4.5 scenario will be available at the IAMC scenario Web site (www.iiasa.ac.at/web-apps/tnt/RcpDb/).

CH_4, and HFC-134a emissions reductions from Chapter 5 were applied as net reductions to the GCAM global baseline net emissions for each GHG. All emissions reductions were assumed to begin in 2014, with zero emissions change in 2013 and linearly increasing to equal the value supplied (in Chapter 5) for 2018. The emissions reductions past 2050 were scaled with total U.S. road transportation fuel consumption from the GCAM reference scenario. Using MAGICC, the change in atmospheric CO_2 concentrations, global mean temperature, and sea level were projected at five-year time steps to 2100 for both the reference (no climate policy) scenario and the emissions reduction scenario specific to this proposal. To capture some of the uncertainty in the climate system, the changes in projected atmospheric CO_2 concentrations, global mean temperature and sea level were estimated across the most current Intergovernmental Panel on Climate Change (IPCC) range of climate sensitivities, 1.5°C to 6.0°C.[S]

To compute the reductions in atmospheric CO_2 concentration, temperature, and sea level rise specifically attributable to the rule, the output from the proposed rule's emissions scenario was subtracted from the reference (no policy or baseline) emissions case scenario. As a result of the proposal's specified emissions reductions (or preferred alternative), the concentration of atmospheric CO_2 is projected to be reduced by approximately X.X to X.X parts per million (ppm), the global mean temperature is projected to be reduced by approximately 0.00X-0.0XX°C by 2100 and global mean sea level rise is projected to be reduced by approximately 0.0X-0.XX cm by 2100. The reference (no policy) and the specified emission reductions scenarios were subtracted from global emissions for the years 2000-2100. The difference between these two results is the impact of the specified proposed heavy-duty emissions changes on global CO_2 concentrations and other variables.

Figure 8-5 provides the results for the estimated reductions in atmospheric CO_2 concentration associated with the proposed rule. Figure 8-6 provides the estimated reductions in

Clarke, L., J. Edmonds, H. Jacoby, H. Pitcher, J. Reilly, R. Richels, (2007) Scenarios of Greenhouse Gas Emissions and Atmospheric Concentrations. Sub-report 2.1A of Synthesis and Assessment Product 2.1 by the U.S. Climate Change Science Program and the Subcommittee on Global Change Research (Department of Energy, Office of Biological & Environmental Research, Washington, DC., USA, 154 pp.).

Moss, Richard, Mustafa Babiker, Sander Brinkman, Eduardo Calvo, Tim Carter, Jae Edmonds, Ismail Elgizouli, Seita Emori, Lin Erda, Kathy Hibbard, Roger Jones, Mikiko Kainuma, Jessica Kelleher, Jean Francois Lamarque, Martin Manning, Ben Matthews, Jerry Meehl, Leo Meyer, John Mitchell, Nebojsa Nakicenovic, Brian O'Neill, Ramon Pichs, Keywan Riahi, Steven Rose, Paul Runci, Ron Stouffer, Detlef van Vuuren, John Weyant, Tom Wilbanks, Jean Pascal van Ypersele, and Monika Zurek (2008) Towards New Scenarios for Analysis of Emissions, Climate Change, Impacts, and Response Strategies (Intergovernmental Panel on Climate Change, Geneva) 132 pp.

Smith, Steven J. and T.M.L. Wigley (2006) "Multi-Gas Forcing Stabilization with the MiniCAM" Energy Journal (Special Issue #3).

[S] In IPCC reports, equilibrium climate sensitivity refers to the equilibrium change in the annual mean global surface temperature following a doubling of the atmospheric equivalent carbon dioxide concentration. The IPCC states that climate sensitivity is "likely" to be in the range of 2°C to 4.5°C, "very unlikely" to be less than 1.5°C, and "values substantially higher than 4.5°C cannot be excluded." IPCC WGI, 2007, *Climate Change 2007 - The Physical Science Basis,* Contribution of Working Group I to the Fourth Assessment Report of the IPCC, http://www.ipcc.ch/.

projected global mean temperatures associated with the proposed rule. Figure 8-7 provides the estimated reductions in global mean sea level rise associated with the proposed rule.
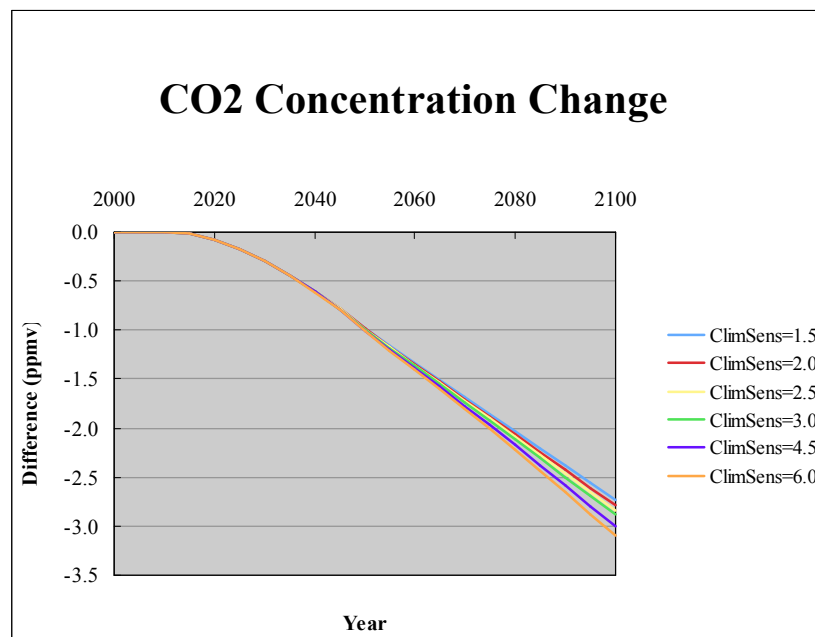

Figure 8-5 Estimated Projected Reductions in Atmospheric CO2 Concentrations (parts per million by volume) from Baseline for the Proposed Heavy-Duty Rule (for climate sensitivities ranging from 1.5-6°C)
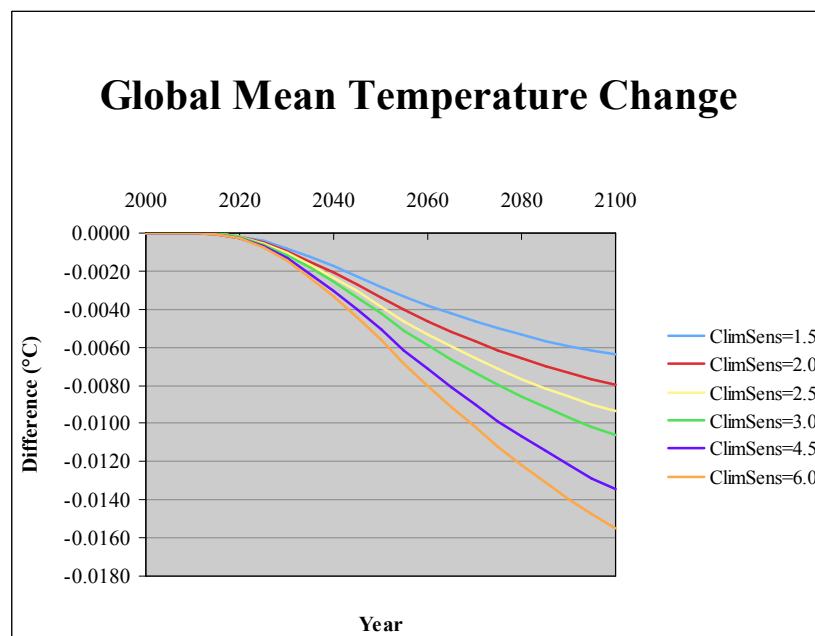

Figure 8-6 Estimated Projected Reductions in Global Mean Surface Temperatures from Baseline for the Proposed Heavy-Duty Rule (for climate sensitivities ranging from 1.5-6°C)
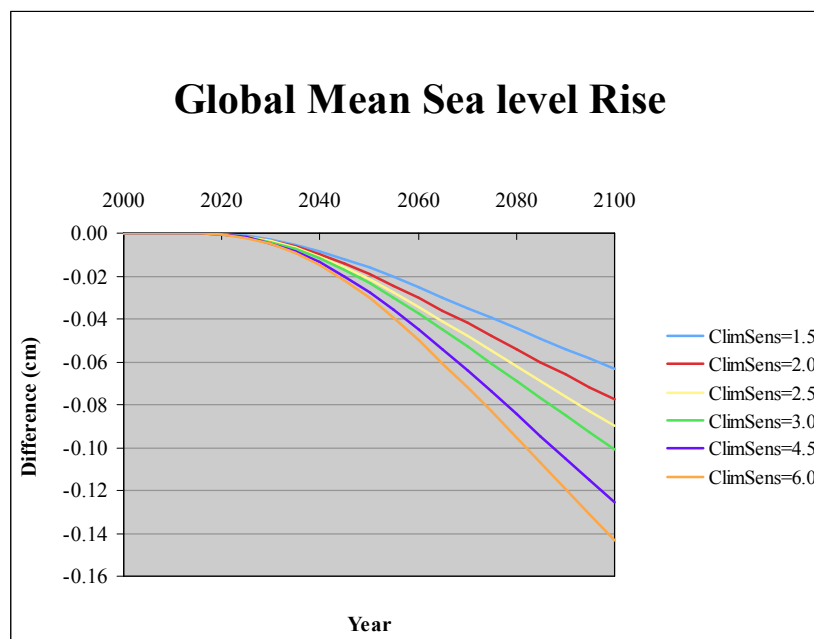
Global Mean Sea level Rise

Figure 8-7   Estimated Projected Reductions in Global Mean Sea Level Rise from Baseline for the Final Proposed Heavy-Duty Rule (for climate sensitivities ranging from 1.5-6°C)

The results in both Figure 8-6 and Figure 8-7 show a relatively small reduction in the projected global mean temperature and sea level respectively, across all climate sensitivities. The projected reductions are small relative to the IPCC's 2100 "best estimates" for global mean temperature increases (1.8 – 4.0ºC) and sea level rise (0.20-0.59m) for all global GHG emissions sources for a range of emissions scenarios.[288]

8.4.3   Estimated Projected Changes in Ocean pH

In today's proposed rule, EPA analyzes another climate-related variable and calculates the projected changes in ocean pH for tropical waters. To calculate the changes in oceanic pH resulting arising from the change in the atmospheric concentration of carbon dioxide (CO2) resulting from this proposal, EPA used the program developed for CO_2 System Calculations (CO2SYS) CO2SYS,[289] version 1.05, a program which performs calculations relating parameters of the carbon dioxide (CO_2) system in seawater..   The program was developed by Ernie Lewis at Brookhaven National Laboratory and Doug Wallace at the Insitut fuer Meereskunde in Germany, supported by the U.S. Department of Energy, Office of Biological and Environmental Research, under Contract No. DE-ACO2-76CH00016.

The program CO2SYS performs calculations relating parameters of the carbon dioxide (CO_2) system in seawater and freshwater. The program uses two of the four measurable parameters of the CO_2 system [total alkalinity (TA), total inorganic CO_2 (TC), pH, and either fugacity (fCO_2) or partial pressure of CO_2 (pCO_2)] to calculate the other two parameters at a set of input conditions (temperature and pressure) and a set of output conditions chosen by the user. EPA ran the DOS version (Lewis and Wallace, 1998)[290] of the program to compute pH under two emissions scenarios as follows:

1) A reference scenario[291] (i.e., business-as-usual, no action) in which atmospheric CO_2 concentrations (also known as partial pressure) in 2100 reached 788.9 parts per million (ppm) [this scenario assumes a mid-range climate sensitivity of 3°C] in 2100.
2) An emissions reduction scenario[292] in which this proposal is implemented, reducing atmospheric CO_2 concentrations by 2.9 ppm to 786 ppm [this scenario assumes a mid-range climate sensitivity of 3°C] in 2100.

In order to determine the change in pH resulting from the emissions reduction, EPA subtracted the reference scenario pH from the emission reduction scenario pH. The emissions reductions result in a 0.00XX increase in pH. The values for pH under the two scenarios varied according to the inputs used. For one set of seawater parameters (described below), the reference scenario (XXX.X ppm) pH was X.XXXX and the emissions reduction scenario (XXX ppm) was X.XXXX.

The CO2SYS program required the input of a number of variables and constants for each scenario (i.e., both the reference case and the proposed rule's emissions reduction). The inputs are described below, with justification for these inputs provided in brackets:

1) Input mode: Single-input [This means that the program calculates pH for one set of input variables at a time, instead of a batch of variables. It does not affect results].
2) Choice of constants: Mehrbach et al. (1973)[293], refit by Dickson and Millero (1987)[294]
3) Choice of fCO_2 or pCO_2: pCO_2 [pCO_2 is the partial pressure of CO2 and can be converted to fugacity (fCO_2) if desired]
4) Choice of KSO4: Dickson (1990)[295] [Lewis and Wallace (1998)[296] recommend using the equation of Dickson (1990) for this dissociation constant. The model also allows the use of the equation of Khoo et al. (1977).[297] Switching this parameter to Khoo et al. (1977) instead of Dickson (1990) had no effect on the difference in pH between the reference and emissions scenario].

Choice of pH scale: Total scale [The model allows pH outputs to be provided on the total scale, the seawater scale, the free scale, and the National Bureau of Standards (NBS) scale. The various pH scales can be interrelated using equations provided by Lewis and Wallace (1998)].

Based on the projected atmospheric CO_2 concentration reductions (average of X.X ppm by 2100) that would result from this proposal, the modeling program calculates an increase in ocean pH of approximately 0.00XX pH units in 2100. Thus, this analysis indicates the projected decrease in atmospheric CO_2 concentrations from today's proposal would result in a increase in ocean pH.

8.4.4 Summary

EPA's analysis of the proposed rule's effect on global climate conditions is intended to quantify these potential reductions using the best available science. While EPA's modeling results of the effect of this proposed rule alone show small differences in climate effects (CO_2 concentration, global mean temperature, sea level rise, and ocean pH), when expressed in terms of global climate endpoints and global GHG emissions, they yield results that are repeatable and

consistent within the modeling frameworks used. A summary of results are provide in Table 8-2, Projected Changes in Climate Variables.

These projected reductions are proportionally representative of changes to U.S. GHG emissions in the transportation sector. While not formally estimated for this proposal, a reduction in projected global mean temperature and sea level rise implies a reduction in the risks associated with climate change. Both figures illustrate that the distribution for projected global mean temperature and sea level rise increases has shifted down. The benefits of GHG emissions reductions can be characterized both qualitatively and quantitatively, some of which can be monetized (see Chapter 8.5). There are substantial uncertainties in modeling the global risks of climate change, which complicates quantification and cost-benefits assessments. Changes in climate variables are a meaningful proxy for changes in the risk of all potential impacts-- including those that can be monetized, and those that have not been monetized but can be quantified in physical terms (e.g., water availability), as well as those that have not yet been quantified or are extremely difficult to quantify (e.g., forest disturbance and catastrophic events such as collapse of large ice sheets and subsequent sea level rise).

8.4.4.1.1 Projected Changes in Climate Variables

Table 8-2 Effect of GHG Emissions Reductions On Projected Changes in Global Climate for the Proposed Heavy-Duty Rule (for climate sensitivities ranging from 1.5-6°C)

VARIABLE	UNITS	YEAR	PROJECTED CHANGE IN YEAR XX	PROJECTED CHANGE IN YEAR YY
Atmospheric CO$_2$ Concentration	ppm	2100	-X.X-X.X	
Global Mean Surface Temperature	° C	2100	-0.00X-0.0XX	
Sea Level Rise	cm	2100	-0.XX-0.XX	
Ocean pH	pH units	2100	0.00XX	

8.5 Monetized CO$_2$ Impacts

We assigned a dollar value to reductions in carbon dioxide (CO$_2$) emissions using recent estimates of the "social cost of carbon" (SCC). The SCC is an estimate of the monetized damages associated with an incremental increase in carbon emissions in a given year. It is intended to include (but is not limited to) changes in net agricultural productivity, human health, property damages from increased flood risk, and the value of ecosystem services due to climate change. The SCC estimates used in this analysis were developed through an interagency process that included EPA, DOT/NHTSA, and other executive branch entities, and concluded in

February 2010. We first used these SCC estimates in the benefits analysis for the final joint EPA/DOT Rulemaking to establish Light-Duty Vehicle Greenhouse Gas Emission Standards and Corporate Average Fuel Economy Standards; see the rule's preamble for discussion about application of the SCC (75 FR 25324; 5/7/10). The SCC Technical Support Document (SCC TSD) provides a complete discussion of the methods used to develop these SCC estimates.[T]

The interagency group selected four SCC values for use in regulatory analyses, which we have applied in this analysis: $5, $22, $36, and $67 per metric ton of CO_2 emissions in 2010, in 2008 dollars.[U,V] The first three values are based on the average SCC from three integrated assessment models, at discount rates of 2.5, 3, and 5 percent, respectively. SCCs at several discount rates are included because the literature shows that the SCC is quite sensitive to assumptions about the discount rate, and because no consensus exists on the appropriate rate to use in an intergenerational context. The fourth value is the 95th percentile of the SCC from all three models at a 3 percent discount rate. It is included to represent higher-than-expected impacts from temperature change further out in the tails of the SCC distribution. Low probability, high impact events are incorporated into all of the SCC values through explicit consideration of their effects in two of the three models as well as the use of a probability density function for equilibrium climate sensitivity. Treating climate sensitivity probabilistically results in more high temperature outcomes, which in turn lead to higher projections of damages.

The SCC increases over time because future emissions are expected to produce larger incremental damages as physical and economic systems become more stressed in response to greater climatic change. Note that the interagency group estimated the growth rate of the SCC directly using the three integrated assessment models rather than assuming a constant annual growth rate. This helps to ensure that the estimates are internally consistent with other modeling assumptions. Table VIII.G.1-1 presents the SCC estimates used in this analysis.

When attempting to assess the incremental economic impacts of carbon dioxide emissions, the analyst faces a number of serious challenges. A recent report from the National

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[T] Docket ID EPA-HQ-OAR-2009-0472-114577, *Technical Support Document: Social Cost of Carbon for Regulatory Impact Analysis Under Executive Order 12866, Interagency Working Group on Social Cost of Carbon*, with participation by Council of Economic Advisers, Council on Environmental Quality, Department of Agriculture, Department of Commerce, Department of Energy, Department of Transportation, Environmental Protection Agency, National Economic Council, Office of Energy and Climate Change, Office of Management and Budget, Office of Science and Technology Policy, and Department of Treasury (February 2010). Also available at http://epa.gov/otaq/climate/regulations.htm

[U] The interagency group decided that these estimates apply only to CO_2 emissions. Given that warming profiles and impacts other than temperature change (e.g. ocean acidification) vary across GHGs, the group concluded "transforming gases into CO_2-equivalents using GWP, and then multiplying the carbon-equivalents by the SCC, would not result in accurate estimates of the social costs of non-CO_2 gases" (SCC TSD, pg 13).

[V] The SCC estimates were converted from 2007 dollars to 2008 dollars using a GDP price deflator (1.021) obtained from the Bureau of Economic Analysis, National Income and Product Accounts Table 1.1.4, *Prices Indexes for Gross Domestic Product*.

Academies of Science (NRC 2009) points out that any assessment will suffer from uncertainty, speculation, and lack of information about (1) future emissions of greenhouse gases, (2) the effects of past and future emissions on the climate system, (3) the impact of changes in climate on the physical and biological environment, and (4) the translation of these environmental impacts into economic damages. As a result, any effort to quantify and monetize the harms associated with climate change will raise serious questions of science, economics, and ethics and should be viewed as provisional.

The interagency group noted a number of limitations to the SCC analysis, including the incomplete way in which the integrated assessment models capture catastrophic and non-catastrophic impacts, their incomplete treatment of adaptation and technological change, uncertainty in the extrapolation of damages to high temperatures, and assumptions regarding risk aversion. The limited amount of research linking climate impacts to economic damages makes the interagency modeling exercise even more difficult. The interagency group hopes that over time researchers and modelers will work to fill these gaps and that the SCC estimates used for regulatory analysis by the Federal government will continue to evolve with improvements in modeling. Additional details on these limitations are discussed in the SCC TSD.

In light of these limitations, the interagency group has committed to updating the current estimates as the science and economic understanding of climate change and its impacts on society improves over time. Specifically, the interagency group has set a preliminary goal of revisiting the SCC values in the next few years or at such time as substantially updated models become available, and to continue to support research in this area.

Applying the global SCC estimates, shown in Table 8-X, to the estimated reductions in domestic CO_2 emissions for the proposed rule, we estimate the dollar value of the climate related benefits for each analysis year. For internal consistency, the annual benefits are discounted back to net present value terms using the same discount rate as each SCC estimate (i.e. 5%, 3%, and 2.5%) rather than 3% and 7%.[W] The SCC estimates are presented in Table 8-X and the associated CO_2 benefit estimates for each calendar year are shown in Table 8-X.

[W] It is possible that other benefits or costs of proposed regulations unrelated to CO2 emissions will be discounted at rates that differ from those used to develop the SCC estimates.

Table 8-3 Social Cost of CO_2, 2010 – 2050[a] (in 2008 dollars)

Year	Discount Rate and Statistic			
	5% Average	3% Average	2.5% Average	3% 95th percentile
2010	$4.80	$21.85	$35.84	$66.26
2015	$5.87	$24.35	$39.21	$74.33
2020	$6.94	$26.85	$42.58	$82.39
2025	$8.45	$30.15	$46.84	$92.25
2030	$9.95	$33.44	$51.10	$102.10
2035	$11.46	$36.73	$55.36	$111.95
2040	$12.97	$40.02	$59.63	$121.81
2045	$14.50	$42.93	$63.00	$130.43
2050	$16.03	$45.84	$66.37	$139.06

[a] The SCC values are dollar-year and emissions-year specific.

Table 8-4 Upstream and Downstream CO_2 Benefits for the Given SCC Value, Calendar Year Analysis[a] (Millions of 2008 dollars)

YEAR	5% (AVERAGE SCC = $5 IN 2010)	3% (AVERAGE SCC = $22 IN 2010)	2.5% (AVERAGE SCC = $36 IN 2010)	3% (95TH PERCENTILE = $66 IN 2010)
2012	$0	$0	$0	$0
2013	$0	$0	$0	$0
2014	$23	$99	$159	$301
2015	$48	$200	$321	$609
2016	$75	$307	$493	$938
2017	$114	$460	$735	$1,406
2018	$157	$622	$993	$1,906
2019	$199	$779	$1,239	$2,389
2020	$241	$933	$1,480	$2,864
2021	$286	$1,086	$1,714	$3,329
2022	$330	$1,233	$1,939	$3,780
2023	$376	$1,381	$2,163	$4,231
2024	$423	$1,530	$2,386	$4,684
2025	$470	$1,678	$2,607	$5,134
2026	$518	$1,823	$2,822	$5,575
2027	$565	$1,964	$3,030	$6,005
2028	$612	$2,101	$3,231	$6,421
2029	$658	$2,235	$3,427	$6,829
2030	$706	$2,370	$3,622	$7,237
2031	$751	$2,498	$3,807	$7,626
2032	$797	$2,624	$3,986	$8,005
2033	$843	$2,751	$4,168	$8,389
2034	$890	$2,878	$4,349	$8,774
2035	$939	$3,008	$4,534	$9,168
2036	$987	$3,139	$4,720	$9,564
2037	$1,037	$3,271	$4,907	$9,964
2038	$1,088	$3,405	$5,095	$10,368
2039	$1,139	$3,540	$5,286	$10,777
2040	$1,191	$3,677	$5,477	$11,189
2041	$1,244	$3,807	$5,653	$11,580

2042	$1,298	$3,938	$5,831	$11,977
2043	$1,354	$4,073	$6,011	$12,382
2044	$1,411	$4,210	$6,196	$12,795
2045	$1,469	$4,349	$6,381	$13,211
2046	$1,528	$4,490	$6,570	$13,637
2047	$1,588	$4,635	$6,763	$14,072
2048	$1,650	$4,782	$6,958	$14,513
2049	$1,713	$4,931	$7,157	$14,963
2050	$1,778	$5,085	$7,361	$15,424
NPV[b]	$8,858	$45,306	$76,810	$138,082

[a] The SCC values are dollar-year and emissions-year specific.
[b] Note that net present value of reduced GHG emissions is calculated differently than other benefits. The same discount rate used to discount the value of damages from future emissions (SCC at 5, 3, 2.5 percent) is used to calculate net present value of SCC for internal consistency. Refer to SCC TSD for more detail.

We also conducted a separate analysis of the CO_2 benefits over the model year lifetimes of the 2014 through 2018 model year vehicles. In contrast to the calendar year analysis, the model year lifetime analysis shows the lifetime impacts of the program on each of these MY fleets over the course of its lifetime. Full details of the inputs to this analysis can be found in RIA chapter 5. The CO_2 benefits of the full life of each of the five model years from 2014 through 2018 are shown in **Error! Reference source not found.**Table 8-X through Table 8-X for each of the four different social cost of carbon values. The CO_2 benefits are shown for each year in the model year life and in net present value. The same discount rate used to discount the value of damages from future emissions (SCC at 5, 3, 2.5 percent) is used to calculate net present value of SCC for internal consistency.

Table 8-5 Upstream and Downstream CO_2 Benefits for the 5% (Average SCC) Value, Model Year Analysis[a]
(Millions of 2008 dollars)

YEAR	MY 2014	MY 2015	MY 2016	MY 2017	MY 2018	SUM
2012	$0	$0	$0	$0	$0	$0
2013	$0	$0	$0	$0	$0	$0
2014	$18	$0	$0	$0	$0	$18
2015	$18	$20	$0	$0	$0	$38
2016	$17	$19	$23	$0	$0	$59
2017	$16	$18	$22	$35	$0	$92
2018	$15	$17	$21	$34	$40	$128
2019	$14	$16	$20	$32	$38	$121
2020	$13	$15	$19	$31	$37	$114
2021	$12	$14	$18	$29	$35	$108
2022	$11	$13	$16	$27	$33	$101
2023	$10	$12	$15	$25	$31	$94
2024	$9	$11	$14	$23	$29	$87
2025	$9	$10	$13	$22	$27	$80
2026	$8	$9	$12	$20	$25	$73
2027	$7	$8	$11	$18	$23	$67
2028	$6	$7	$10	$16	$21	$60
2029	$5	$7	$9	$15	$19	$54
2030	$5	$6	$8	$13	$17	$48

2031	$4	$5	$7	$12	$15	$43
2032	$4	$4	$6	$10	$14	$38
2033	$3	$4	$5	$9	$12	$33
2034	$3	$3	$5	$8	$11	$29
2035	$2	$3	$4	$7	$9	$25
2036	$2	$3	$3	$6	$8	$22
2037	$2	$2	$3	$5	$7	$19
2038	$1	$2	$3	$5	$6	$16
2039	$1	$2	$2	$4	$5	$14
2040	$1	$1	$2	$3	$5	$12
2041	$1	$1	$2	$3	$4	$10
2042	$1	$1	$1	$2	$3	$9
2043	$1	$1	$1	$2	$3	$8
2044	$1	$1	$1	$2	$3	$7
2045	$0	$1	$1	$2	$2	$5
2046	$0	$0	$1	$1	$2	$4
2047	$0	$0	$0	$2	$2	$3
2048	$0	$0	$0	$0	$2	$2
2049	$0	$0	$0	$0	$0	$0
2050	$0	$0	$0	$0	$0	$0
NPV, 5%	$200	$200	$200	$300	$300	$1,100

[a] The SCC values are dollar-year and emissions-year specific.

Table 8-6 Upstream and Downstream CO_2 Benefits for the 3% (Average SCC) SCC Value, Model Year Analysis[a] (Millions of 2008 dollars)

YEAR	MY 2014	MY 2015	MY 2016	MY 2017	MY 2018	SUM
2012	$0	$0	$0	$0	$0	$0
2013	$0	$0	$0	$0	$0	$0
2014	$77	$0	$0	$0	$0	$77
2015	$73	$83	$0	$0	$0	$156
2016	$69	$78	$93	$0	$0	$240
2017	$65	$74	$88	$142	$0	$368
2018	$60	$69	$83	$134	$159	$506
2019	$56	$65	$78	$127	$150	$475
2020	$51	$59	$72	$119	$142	$443
2021	$46	$54	$67	$110	$133	$410
2022	$42	$49	$61	$101	$124	$376
2023	$38	$44	$55	$93	$114	$344
2024	$34	$40	$51	$84	$105	$314
2025	$30	$36	$46	$77	$96	$285
2026	$27	$32	$41	$70	$87	$258
2027	$24	$29	$37	$62	$79	$231
2028	$21	$25	$33	$56	$71	$206
2029	$18	$22	$29	$49	$64	$183
2030	$16	$19	$26	$44	$57	$162
2031	$14	$17	$22	$39	$50	$142
2032	$12	$15	$20	$34	$45	$124
2033	$10	$13	$17	$30	$39	$108

2034	$9	$11	$15	$26	$34	$94
2035	$7	$9	$13	$22	$30	$81
2036	$6	$8	$11	$19	$26	$70
2037	$5	$7	$9	$16	$22	$60
2038	$5	$6	$8	$14	$19	$52
2039	$4	$5	$7	$12	$17	$44
2040	$3	$4	$6	$10	$14	$38
2041	$3	$3	$5	$9	$12	$32
2042	$2	$3	$4	$7	$10	$27
2043	$2	$2	$4	$6	$9	$23
2044	$2	$2	$3	$5	$8	$20
2045	$0	$2	$3	$5	$6	$16
2046	$0	$0	$3	$4	$6	$12
2047	$0	$0	$0	$5	$5	$9
2048	$0	$0	$0	$0	$6	$6
2049	$0	$0	$0	$0	$0	$0
2050	$0	$0	$0	$0	$0	$0
NPV, 3%	$600	$700	$700	$1,100	$1,200	$4,200

[a] The SCC values are dollar-year and emissions-year specific.

Table 8-7 Upstream and Downstream CO_2 Benefits for the from 2.5% (Average SCC) SCC Value, Model Year Analysis[a] (Millions of 2008 dollars)

YEAR	MY 2014	MY 2015	MY 2016	MY 2017	MY 2018	SUM
2012	$0	$0	$0	$0	$0	$0
2013	$0	$0	$0	$0	$0	$0
2014	$125	$0	$0	$0	$0	$125
2015	$118	$133	$0	$0	$0	$251
2016	$110	$125	$149	$0	$0	$385
2017	$104	$118	$140	$228	$0	$589
2018	$96	$111	$132	$214	$254	$807
2019	$88	$103	$124	$201	$239	$756
2020	$81	$94	$115	$188	$225	$702
2021	$73	$86	$105	$174	$210	$647
2022	$66	$77	$96	$159	$194	$592
2023	$59	$70	$87	$145	$179	$539
2024	$53	$63	$79	$131	$164	$490
2025	$47	$56	$71	$119	$149	$443
2026	$42	$50	$64	$108	$135	$399
2027	$37	$44	$57	$96	$123	$357
2028	$32	$39	$50	$86	$110	$317
2029	$28	$34	$44	$76	$98	$280
2030	$24	$30	$39	$67	$87	$247
2031	$21	$26	$34	$59	$77	$217
2032	$18	$22	$30	$51	$68	$189
2033	$15	$19	$26	$45	$59	$164
2034	$13	$16	$22	$39	$52	$142
2035	$11	$14	$19	$34	$45	$123
2036	$10	$12	$16	$29	$39	$105
2037	$8	$10	$14	$25	$33	$90

2038	$7	$9	$12	$21	$29	$77
2039	$6	$7	$10	$18	$25	$66
2040	$5	$6	$9	$15	$21	$56
2041	$4	$5	$7	$13	$18	$48
2042	$3	$4	$6	$11	$15	$40
2043	$3	$4	$5	$9	$13	$34
2044	$3	$3	$4	$8	$11	$30
2045	$0	$4	$4	$7	$10	$23
2046	$0	$0	$4	$6	$8	$18
2047	$0	$0	$0	$7	$7	$14
2048	$0	$0	$0	$0	$8	$8
2049	$0	$0	$0	$0	$0	$0
2050	$0	$0	$0	$0	$0	$0
NPV, 2.5%	$1,000	$1,000	$1,100	$1,700	$1,800	$6,600

[a] The SCC values are dollar-year and emissions-year specific.

Table 8-8 Upstream and Downstream CO_2 Benefits for the 3% (95th Percentile) SCC Value, Model Year Analysis[a] (Millions of 2008 dollars)

YEAR	MY 2014	MY 2015	MY 2016	MY 2017	MY 2018	SUM
2012	$0	$0	$0	$0	$0	$0
2013	$0	$0	$0	$0	$0	$0
2014	$235	$0	$0	$0	$0	$235
2015	$223	$253	$0	$0	$0	$476
2016	$210	$239	$284	$0	$0	$733
2017	$198	$225	$268	$436	$0	$1,127
2018	$185	$212	$253	$411	$488	$1,550
2019	$171	$198	$239	$388	$461	$1,457
2020	$156	$182	$222	$364	$435	$1,359
2021	$142	$166	$204	$338	$408	$1,258
2022	$128	$150	$187	$309	$379	$1,154
2023	$116	$136	$170	$284	$349	$1,055
2024	$104	$123	$155	$258	$322	$962
2025	$93	$110	$141	$235	$294	$873
2026	$82	$99	$126	$213	$268	$788
2027	$73	$88	$113	$191	$243	$707
2028	$64	$77	$100	$171	$218	$630
2029	$56	$68	$89	$151	$195	$559
2030	$49	$59	$78	$134	$173	$493
2031	$42	$52	$68	$118	$154	$434
2032	$36	$45	$60	$103	$136	$380
2033	$31	$39	$52	$90	$119	$331
2034	$27	$33	$45	$78	$104	$287
2035	$23	$28	$38	$68	$91	$248
2036	$19	$24	$33	$58	$79	$214
2037	$16	$21	$28	$50	$68	$183
2038	$14	$17	$24	$43	$58	$157
2039	$12	$15	$21	$37	$50	$134

2040	$10	$12	$18	$31	$43	$114
2041	$8	$10	$15	$27	$37	$97
2042	$7	$9	$13	$23	$32	$83
2043	$6	$7	$11	$19	$27	$70
2044	$7	$6	$9	$16	$23	$61
2045	$0	$7	$8	$14	$20	$49
2046	$0	$0	$9	$12	$17	$38
2047	$0	$0	$0	$14	$14	$29
2048	$0	$0	$0	$0	$17	$17
2049	$0	$0	$0	$0	$0	$0
2050	$0	$0	$0	$0	$0	$0
NPV, 3%	$1,900	$2,000	$2,200	$3,200	$3,600	$13,000

[a] The SCC values are dollar-year and emissions-year specific.

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