Chapter II: Health and Welfare Concerns and Emissions Benefits

This chapter describes the public health and welfare concerns associated with the pollutants emitted by heavy-duty vehicles, and the emission reductions that are expected to occur as a result of today's action. Specifically, we present information on the ambient air pollution situation that is likely to exist without this rule between 2007 and 2030 for ambient pollutants of concern (e.g., ozone, particulate matter). In addition, this chapter presents information on the expected emission reductions based on our projected national heavy-duty vehicle emissions with and without the new standards for nitrogen oxides (NOx), non-methane hydrocarbons (NMHC), particulate matter (PM), sulfur dioxide (SOx), carbon monoxide (CO), and air toxics.

A. Health and Welfare Concerns

When revising emissions standards for heavy-duty vehicles, the Agency considers the effects of air pollutants emitted from heavy-duty vehicles on public health and welfare.[1] As discussed in more detail below, the outdoor, or ambient, air quality in many areas of the country is expected to violate federal health-based ambient air quality standards for ground level ozone and particulate matter during the time when this rule will take effect. In addition, some studies have found public health and welfare effects from ozone and PM at concentrations that do not constitute a violation of their respective NAAQS. Other studies have associated diesel exhaust with cancer and noncancer health effects. Of particular concern is human epidemiological evidence linking diesel exhaust to an increased risk of lung cancer. Emissions from heavy-duty vehicles also contribute to a variety of environmental and public welfare effects such as impairment of visibility/ regional haze, acid deposition, eutrophication/ nitrification, and POM deposition. As described in more detail throughout this chapter, the standards finalized in this rule will result in a significant improvement in ambient air quality and public health and welfare.

1. Health and Welfare Concerns Raised During Public Hearings

Throughout the five public hearings held around the country on the heavy-duty engine and diesel fuel rule, the Agency received strong public support at each venue for increasing the stringency of heavy-duty truck and bus emission standards, and for further controls on sulfur in diesel fuel, in order to enable the necessary exhaust emission control. Public officials and representatives of environmental, public health, or community-based organizations testified regularly about the link between public health ailments, such as asthma and lung cancer, and air pollution caused by diesel exhaust and particulate matter. A common theme revolved around the

notion that since asthma is an incurable disease, it was of utmost importance to help reduce the severity and frequency of attacks by reducing environmental triggers such as ozone, particulate matter and diesel exhaust. Many testifiers expressed a strong sentiment that the public and the auto industry have done their fair share to clean up cars and keep them clean through regular inspections and maintenance, and it was time for the diesel truck industry to do the same.

In different ways, many noted that the impact of diesel soot is compounded by the fact that it is discharged at street level where people live and breathe. A common complaint was the close proximity of bus depots, transfer terminals, and heavily-trafficked roadways to homes and apartment buildings, and in particular, to hospitals, playgrounds, and schools. Cyclists described the stinging eyes and choking caused by breathing fumes from buses and trucks along city streets, especially when trucks accelerated after stopping at an intersection. Two testifiers cited to health studies that they said reported an association between those living in homes located near heavily-trafficked streets and increased incidences of childhood asthma and leukemia.

By far the most poignant testimony was about how air pollution has impaired the health and well-being of children. As our recent reviews of the NAAQS have documented, we heard concerns expressed by citizens that childhood asthma accounts for 10.1 million missed school days in the United States each year, and that asthma is the leading cause of hospitalizations in New York City for children aged 0-14. At times, parents and their children testified together. Kyle Damitz, accompanied by his mother, entered the following testimony:

> I have come here today to tell you how our bad air affects kids like me with asthma. . . . During these ozone days, I will almost always have an asthma attack if I go outside. On good days, I take two pills in the morning and three pills at bedtime. I do an IV treatment every two weeks. On a bad asthma day, I take four pills in the morning, more at lunch, and again, more at bedtime.. . . I came here today to . . . ask you to help make breathing for kids with asthma easier. By making the air cleaner, you are giving asthmatics a chance to breathe easier. If our air was cleaner, I would be able to take less medicine, be able to play outside more. If you make our air cleaner, I will be able to live longer.

Many testifiers took the Agency to task for not acting sooner on heavy-duty vehicles. Reacting to industry testimony requesting additional time to comply with the standards, testifiers representing their constituents, their communities, environmental organizations, or themselves, expressed the simple desire to be healthy as soon as possible. Some compared the annual human cost of air pollution – quantified by thousands of hospitalizations, emergency room and doctors visits, asthma attacks -- with per vehicle cost of $1,600, and stated their belief that the

regulations are cost effective. Others suggested that several billion dollars spent on improving the environmental performance of trucks and buses was reasonable in light of the petroleum industry's multi-billion dollar profits in the first quarter of 2000.

Major industries represented during these public hearings were the heavy-duty vehicle engine manufacturers, the oil industry, and the commercial truckers. While each had a different perspective, most supported the underlying intent of this rule to improve public health and welfare, and some also supported the specific requirements as proposed. For those who objected to the proposal, the main thrust of their concerns related to the stringency and public health necessity of the new standards and the diesel fuel sulfur requirement. Largely in their written comments, these industries raised questions about the need for additional reductions in order to meet existing ozone and PM national ambient air quality standards and took exception with the Agency's characterization of diesel exhaust as a human carcinogen at environmental levels of exposure. Some industry commenters also challenged the Agency's reliance on public welfare and environmental effects such as visibility impairment and eutrophication of water bodies because the Agency had insufficiently quantified the benefits that would result from new standards on heavy-duty vehicles and diesel fuel.

The following subsections present the available information on the air pollution situation that is likely to exist without this rule for each ambient pollutant. We also present information on the improvement that is expected to result from this rule. The Agency received a significant number of comments on this section during the public hearings and in written comments from interested parties. Where appropriate, comments are addressed in this section, but the majority are addressed only in the Response to Comment document that accompanies this preamble. Interested parties should refer to the Response to Comment document for the Agency's response to their specific comments.

2. Ozone

This section reviews health and welfare effects of ozone and describes the air quality information that forms the basis of our belief that ozone concentrations in many areas across the country face a significant risk of exceeding the ozone standard between 2007 and 2030. Information on air quality was gathered from a variety of sources, including monitored ozone concentrations from 1997-1999, air quality modeling forecasts conducted for this rulemaking, ozone modeling and information from states that have recently submitted attainment demonstrations, and other state and local air quality information. Studies have found that ozone concentrations at levels that do not exceed the 1-hour ozone standard are associated with impacts on public health and welfare, and this section also summarizes those health effects and provides some information about the potential for ozone at these moderate levels to exist during the time period when this rule will take effect.

a. Health and Welfare Effects of Ozone and its Precursors

Ground-level ozone, the main ingredient in smog, is formed by complex chemical reactions of VOC and NOx in the presence of heat and sunlight. Ozone forms readily in the lower atmosphere, usually during hot summer weather. Volatile Organic Compounds are emitted from a variety of sources, including motor vehicles, chemical plants, refineries, factories, consumer and commercial products, and other industrial sources. Volatile organic compounds also are emitted by natural sources such as vegetation. Oxides of Nitrogen are emitted largely from motor vehicles, off-highway equipment, power plants, and other sources of combustion.

The science of ozone formation, transport, and accumulation is complex. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions involving NOx, VOC, heat, and sunlight.[a] As a result, differences in NOx and VOC emissions and weather patterns contribute to daily, seasonal, and yearly differences in ozone concentrations and differences from city to city. Many of the chemical reactions that are part of the ozone-forming cycle 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 produce more ozone than typically would occur on a single high temperature day. Further complicating matters, ozone also can be transported into an area from pollution sources found hundreds of miles upwind, resulting in elevated ozone levels even in areas with low VOC or NOx emissions.

Emissions of NOx and VOC are precursors to the formation of ozone in the lower atmosphere. For example, relatively small amounts of NOx enable ozone to form rapidly when VOC levels are relatively high, but ozone production is quickly limited by removal of the NOx. Under these conditions, NOx reductions are highly effective in reducing ozone while VOC reductions have little effect. Such conditions are called “NOx 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 NOx limited.

When NOx levels are relatively high and VOC levels relatively low, NOx forms inorganic nitrates but relatively little ozone. Such conditions are called “VOC limited.” Under these conditions, VOC reductions are effective in reducing ozone, but NOx reductions can actually increase local ozone under certain circumstances. Even in VOC limited urban areas, NOx reductions are not expected to increase ozone levels if the NOx reductions are sufficiently large. The highest levels of ozone are produced when both VOC and NOx emissions are present in significant quantities on clear summer days.

[a] Carbon monoxide also participates in the production of ozone, albeit at a much slower rate than most VOC and NOx compounds.

Rural areas are almost always NOx limited, due to the relatively large amounts of biogenic VOC emissions in such areas. Urban areas can be either VOC or NOx limited, or a mixture of both, in which ozone levels exhibit moderate sensitivity to changes in either pollutant.

Ozone concentrations in an area also can be lowered by the reaction of nitric oxide 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 NOx, VOC, and ozone, all of which change with time and location.

Based on a large number of recent studies, EPA has identified several key health effects caused when people are exposed to levels of ozone found today in many areas of the country.[2, 3] Short-term exposures (1-3 hours) to high ambient ozone concentrations have been linked to increased hospital admissions and emergency room visits for respiratory problems. For example, studies conducted in the northeastern U.S. and Canada show that ozone air pollution is associated with 10-20 percent of all of the summertime respiratory-related hospital admissions. Repeated exposure to ozone can make people more susceptible to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma. Prolonged (6 to 8 hours), repeated exposure to ozone can cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure, which over time could lead to premature aging of the lungs and/or chronic respiratory illnesses such as emphysema and chronic bronchitis.

Children and outdoor workers are most at risk from ozone exposure because they typically are active outside, playing and exercising, during the summer when ozone levels are highest. For example, summer camp studies in the eastern U.S. and southeastern Canada have reported significant reductions in lung function in children who are active outdoors. Further, children are more at risk than adults from ozone exposure because their respiratory systems are still developing. Adults who are outdoors and moderately active during the summer months, such as construction workers and other outdoor workers, also are among those most at risk. 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.

Evidence also exists of a possible relationship between daily increases in ozone levels and increases in daily mortality levels. While the magnitude of this relationship is still too uncertain to allow for direct quantification, the full body of evidence indicates the possibility of a positive relationship between ozone exposure and premature mortality.

In addition to human health effects, ozone adversely affects crop yield, vegetation and forest growth, and the durability of materials. Because ground-level ozone interferes with the

ability of a plant to produce and store food, plants become more susceptible to disease, insect attack, harsh weather and other environmental stresses. Ozone causes noticeable foliage damage in many crops, trees, and ornamental plants (i.e., grass, flowers, shrubs, and trees) and causes reduced growth in plants. Studies indicate that current ambient levels of ozone are responsible for damage to forests and ecosystems (including habitat for native animal species). Ozone chemically attacks elastomers (natural rubber and certain synthetic polymers), textile fibers and dyes, and, to a lesser extent, paints. For example, elastomers become brittle and crack, and dyes fade after exposure to ozone.

Volatile organic compound emissions are detrimental not only for their role in forming ozone, but also for their role as air toxics. Some VOCs emitted from motor vehicles are toxic compounds. At elevated concentrations and exposures, human health effects from air toxics can range from respiratory effects to cancer. Other health impacts include neurological, developmental and reproductive effects. The toxicologically significant VOCs emitted in substantial quantities from HDVs are discussed in detail in Section II.A.4 below.

Besides their role as an ozone precursor, NOx emissions produce a wide variety of health and welfare effects.[4] [5] These problems are caused in part by emissions of nitrogen oxides from motor vehicles. Nitrogen dioxide can irritate the lungs and lower resistance to respiratory infection (such as influenza). NOx emissions are an important precursor to acid rain and may affect both terrestrial and aquatic ecosystems. Atmospheric deposition of nitrogen leads to excess nutrient enrichment problems ("eutrophication") in the Chesapeake Bay and several nationally important estuaries along the East and Gulf Coasts. Eutrophication can produce multiple adverse effects on water quality and the aquatic environment, including increased algal blooms, excessive phytoplankton growth, and low or no dissolved oxygen in bottom waters. Eutrophication also reduces sunlight, causing losses in submerged aquatic vegetation critical for healthy estuarine ecosystems. Deposition of nitrogen-containing compounds also affects terrestrial ecosystems. Nitrogen fertilization can alter growth patterns and change the balance of species in an ecosystem. In extreme cases, this process can result in nitrogen saturation when additions of nitrogen to soil over time exceed the capacity of plants and microorganisms to utilize and retain the nitrogen. These environmental impacts are discussed further in Sections II.A.6 and II.A.7.

Elevated levels of nitrates in drinking water pose significant health risks, especially to infants. Studies have shown that a substantial rise in nitrogen levels in surface waters are highly correlated with human-generated inputs of nitrogen in those watersheds.[6] These nitrogen inputs are dominated by fertilizers and atmospheric deposition. Nitrogen dioxide and airborne nitrate also contribute to pollutant haze, which impairs visibility and can reduce residential property values and the value placed on scenic views.

b. Photochemical Ozone Modeling

In conjunction with this rulemaking, the Agency performed a series of ozone air quality modeling simulations for nearly the entire Eastern U.S covering metropolitan areas from Texas to the Northeast.[b] The model simulations were performed for five emissions scenarios: a 2007 baseline projection, a 2020 baseline projection and a 2020 projection with heavy-duty vehicle controls, a 2030 baseline projection, and a 2030 projection with heavy-duty vehicle controls.

The model outputs from the 2007, 2020 and 2030 baselines, combined with current air quality data, were used to identify areas expected to exceed the ozone NAAQS in 2007, 2020 and 2030. These areas became candidates for being determined to be residual exceedance areas which will require additional emission reductions to attain and maintain the ozone NAAQS. The impacts of the heavy-duty vehicle controls were determined by comparing the model results in the future year control runs against the baseline simulations of the same year. This modeling supports the conclusion that there is a broad set of areas with predicted ozone concentrations at or above 0.125 ppm between 2007 and 2030 in the baseline scenarios without additional emission reductions.

The air quality modeling performed for this rule was based upon the same modeling system as was used in the Tier 2 air quality analysis, with the addition of updated inventory estimates for 2007, 2020 and 2030. Consistent with a commitment expressed in the rule proposal, the Agency released the emissions inventory inputs for, and a description of ozone modeling, into the public record (docket number A-99-06), and also onto a website developed expressly for this purpose, on a continuous basis as they were developed. Further discussion of this modeling, including evaluations of model performance relative to predicted future air quality, is provided in the air quality modeling technical support document (TSD).

i. Modeling Methodology, Domains, and Episodes

A variable-grid version of the Urban Airshed Model (UAM-V) was utilized to estimate base and future-year ozone concentrations over the eastern U.S. for the various emissions scenarios. UAM-V simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone. This model is commonly used for purposes of determining attainment/non-attainment as well as estimating the ozone reductions expected to

[b] EPA also performed ozone air quality modeling for the western United States but, as described further in the air quality technical support document, model predictions were well below corresponding ambient concentrations. Given that model performance was degraded to the extent that the directional response of the model to controls may be questionable, and considering the performance relative to that for the East and what is typically expected out of such regulation modeling applications, it was determined that the results of western ozone modeling were not relied on for this rule.

occur from a reduction in emitted pollutants. The following sections provide an overview of the ozone modeling completed as part of this rulemaking. More detailed information is included in the air quality modeling TSD, which is located in the docket for this rule.

The eastern modeling domain covered that portion of the U.S. east of west longitude 99 degrees. The model resolution was 36 km over the outer portions of the domain and 12 km in the inner portion of the grids. A modeling study considered the sensitivity of regional modeling strategies to grid resolution (LADCO, 1999). This study showed that the spatial pattern and magnitude of the ozone changes at 4 km in response to emissions reductions were slightly more pronounced, but generally similar to the modeled changes at 12 km in the Lake Michigan area. The Ozone Transport Assessment Group (OTAG)[c] modeling application also investigated the effects of grid resolution on national/regional control strategies. The OTAG Final Report concluded that: a) peak simulated ozone is generally higher with more highly resolved grids, b) spatial concentration patterns are comparable between the fine and the coarse grid, and c) NOx reductions produce widespread ozone decreases and occasional limited ozone increases with either the fine or the coarse grid (although the increases tend to be larger in magnitude when finer-scale grids are used). More detail on the effect of grid size upon model results is provided in the response to comments and the TSD for this final rule.

Three multi-day meteorological scenarios during the summer of 1995 were used in the model simulations over the eastern U.S.: 12-24 June, 5-15 July, and 7-21 August. These periods featured ozone exceedances at various times over many areas of the eastern U.S.[d] In general, these episodes do not represent extreme ozone events but, instead, are generally representative of ozone levels near local design values. Each of the six emissions scenarios (1996 base year, 2007 baseline, 2020 base, 2020 control, 2030 baseline, 2030 control) were simulated for the three episodes.

ii. Non-emissions Modeling Inputs

The meteorological data required for input into UAM-V (wind, temperature, vertical mixing, etc.) were developed by a separate meteorological model, the Regional Atmospheric Modeling System (RAMS) for the eastern U.S. 1995 episodes. This model provided needed data at every grid cell on an hourly basis. These meteorological modeling results were evaluated

[c] The OTAG modeling project is used as a benchmark for this heavy-duty vehicle and low sulfur diesel fuel modeling because it is the most extensive regional ozone modeling application completed to date in terms of days modeled, areas covered, and efforts of the air pollution modeling community to obtain sound model performance.

[d] Each modeling episode contains three days for which the modeling results are not considered. These days are simulated to minimize the dependence of the modeling results on uncertain initial conditions.

against observed weather conditions before being input into UAM-V and it was concluded that the model fields were adequate representations of the historical meteorology.

The modeling assumed background pollutant levels at the top and along the periphery of the domain. Additionally, initial conditions were assumed to be relatively clean as well. Given the ramp-up days and the expansive domains, it is expected that these assumptions will not affect the modeling results, except in areas near the boundary (e.g., Dallas-Fort Worth TX). The other non-emission UAM-V inputs (land use, photolysis rates, etc.) were developed using procedures employed in the OTAG regional modeling. The development of model inputs is discussed in greater detail in the Air Quality Technical Support Document, which is available in the docket to this final action on heavy-duty vehicles.

iii. Model Performance Evaluation

The purpose of the Heavy Duty Engine base year modeling was to reproduce the atmospheric processes resulting in the observed ozone concentrations over these domains and episodes. One of the fundamental assumptions in air quality modeling is that a model which adequately replicates observed pollutant concentrations in the base year can be used to support future-year policymaking (i.e., assessing the effects of altering the original emissions state).

As with previous regional photochemical modeling studies, the accuracy of the Heavy Duty Engine base year simulations of historical ozone patterns varies by day and by location over this large modeling domain. From a qualitative standpoint, there appears to be considerable similarity on most days between the observed and simulated ozone patterns. Additionally, where possible to discern, the model appears to follow the day-to-day variations in synoptic-scale ozone fairly closely.

The values of the two primary measures of model performance, mean normalized bias and mean normalized gross error, indicate that the Heavy Duty Engine modeling over the eastern U.S. is generally as good as the grid modeling done to support the Tier 2/Sulfur rulemaking, as shown in Table II.A-1. In turn, the performance of the Tier 2/Sulfur modeling was determined to be as good or better than the detailed OTAG regional modeling, which has served as a relative benchmark for acceptable performance from a regional photochemical grid model.

Table II.A-1. Comparison of eastern U.S. regional model performance statistics between the Tier 2/Sulfur modeling and the Heavy Duty Engine modeling. The units are percentages.

Mean Normalized Bias	*Tier 2 June 95*	*Tier 2 July 95*	*Tier 2 August 95*	*HDE June 95*	*HDE July 95*	*HDE August 95*
Domain	-10	-6	+2	-13	-11	+5
Midwest	-11	-13	+7	-15	-16	+10
Northeast	-17	-9	-9	-20	-11	-15
Southeast	-4	+4	+7	-7	-3	+12
Southwest	+2	+8	+6	+1	+3	+11

Mean Normalized Gross Error	*Tier 2 June 95*	*Tier 2 July 95*	*Tier 2 August 95*	*HDE June 95*	*HDE July 95*	*HDE August 95*
Domain	24	24	23	22	23	24
Midwest	24	26	22	22	24	22
Northeast	27	22	24	27	23	24
Southeast	20	24	22	18	21	25
Southwest	24	27	24	22	24	27

Mean normalized bias is defined as the average difference between model predictions and observations (paired in space and time) normalized by the magnitude of the observations. Mean normalized gross error is defined as the average absolute difference between model predictions and observations, paired in space and time, normalized by the magnitude of the observations. EPA guidance on local ozone attainment demonstration modeling (not the purpose of the Heavy Duty Engine modeling) suggests biases be no greater than 15 percent and errors be no greater than 35 percent.

Model performance statistics for the Heavy Duty Engine base case simulations were calculated for the entire grid and numerous smaller sub-grids. The model performance evaluation consisted solely of comparisons against ambient surface ozone data. There was insufficient data available in terms of ozone precursors or ozone aloft to allow for a more complete assessment of model performance. From a regional perspective, the model generally

underestimated observed ozone values (greater than 60 ppb) for the June and July episodes, but predicted higher than observed amounts for the August episode. Errors average about 22-24 percent. The general tendency of the model, as discussed above, is to underestimate observed ozone concentrations. This tendency should lead to a conservative estimate of future-year air quality need.

c. Results of Photochemical Ozone Modeling

The determination that an area is at risk of exceeding the ozone standard in the future was made for all areas with current design values greater than or equal to 0.125 ppm (or within a 10 percent margin) and with modeling evidence that exceedances will persist into the future. The following sections provide background on methods for analysis of attainment and maintenance. Those interested in greater detail should review the Air Quality Modeling Technical Support Document, which is available in the docket to this rule.

i. Air Quality Design Values

An ozone design value is the concentration that determines whether a monitoring site meets the NAAQS for ozone. Because of the way they are defined, design values are determined based on three consecutive-year monitoring periods. A 1-hour design value is the fourth highest daily maximum 1-hour average ozone concentration measured over a three-year period at a given monitor. The full details of these determinations (including accounting for missing values and other complexities) are given in Appendices H and I of 40 CFR Part 50. As discussed in these appendices, design values are truncated to whole part per billion (ppb). Due to the precision with which the standards are expressed (0.12 parts per million (ppm) for the 1-hour), a violation of the 1-hour standard is defined as a design value greater than or equal to 0.125 ppm.

For a county, the design value is the highest design value from among all the monitors with valid design values within that county. If a county does not contain an ozone monitor, it does not have a design value. For most of our analyses, county design values are consolidated where possible into design values for consolidated metropolitan statistical areas (CMSA) or metropolitan statistical areas (MSA). The design value for a metropolitan area is the highest design value among the included counties. Counties that are not in metropolitan areas are treated separately and are not considered in this analysis. For the purposes of defining the current design value of a given area, the 1997-1999 design values were chosen to provide the most recent set of air quality data for identifying areas likely to have an ozone problem in the future. The1997-1999 design values are listed in the Air Quality Modeling Technical Support Document, which is available in the docket to this rule.

ii. Method for Projecting Future Exceedances

The exceedance method was used for interpreting the future-year modeling results to determine where there is an appreciable risk of future nonattainment in the 2007, 2020 and 2030 Base and Control Cases. As part of this method, the modeling grid cells are first assigned to individual areas. The daily maximum 1-hour ozone values predicted in grid cells assigned to an area are then checked to identify whether there are any predictions greater than or equal to 0.125 ppm. Areas with current measured violations of the one-hour ozone standard (or within a 10 percent margin), and one or more model-predicted exceedances, are projected to have the potential for a nonattainment problem in the future.

iii. Areas at Risk of Future Exceedances Based on Ozone Predictive Modeling

The Agency conducted ozone modeling based on inventories developed with and without reductions from this rulemaking for three future years: 2007, 2020, and 2030. The year 2007 was chosen because it is the first year of implementation for the new standards adopted in today's action. It is also the year that ten major urban areas with a history of persistent and elevated ozone concentrations must demonstration attainment. The year 2020 was chosen because of its relevance to the ability of many areas to maintain the ozone standard. The year 2030 was chosen to provide the reader with a full sense of the reductions in ambient ozone concentrations likely to be achieved once the existing fleet of heavy-duty vehicles is replaced with vehicles meeting the standards finalized today.

The predictive ozone modeling is based on emissions inventories which have been updated and improved subsequent to the Agency's recent rulemaking on light duty vehicles and gasoline sulfur, also known as Tier 2. Areas presented in Table II.A-2 have 1997-1999 air quality data indicating violations of the 1-hour ozone NAAQS, or are within 10 percent of the standard, and are predicted to have exceedances in 2007, 2020 or 2030 without the reductions from this rule. Table II.A-2 lists those metropolitan areas with predicted exceedances of the 1-hour ozone standard in 2007, 2020, or 2030 without emission reductions from this rule (i.e., base cases). These areas are listed in columns with a "b" after the year (e.g., 2020b). Table II.A-2 also lists those metropolitan areas with predicted exceedances of the 1-hour ozone standard in 2020 and 2030, with emission reductions from this rule (i.e., control case). These areas are listed in columns with a "c" after the year (e.g., 2020c). An area was considered likely to have future exceedances if exceedances were predicted by the model, and the area is currently violating the 1-hour ozone standard, or is within ten percent of violating the 1-hour ozone standard.

Photochemical ozone modeling conducted for this rulemaking was based in part on updated national emissions inventories for all sources. National emission trends for NOx predict a significant decline from 1996 to 2007, a leveling off of the downward trend between 2007 to 2020, and an increase in NOx inventories from 2020 to 2030. By 2030, national NOx levels are

estimated to reach levels that are within ten percent of 2007 levels. Predictions of national VOC emissions indicate a reduction from 1996 to 2007, followed by an increase between 2007 and 2030 resulting in 2030 levels that are estimated to be 10 percent greater than VOC emissions levels in 2007. In metropolitan ozone nonattainment areas, such as Charleston, Chicago and Houston, NOx or VOC emissions in 2030 are predicted to reach or exceed 2007 levels. These estimated national and metropolitan area emissions inventories of ozone precursors are consistent with the conclusions reached by analysis of ozone modeling conducted for this rule that additional reductions are needed in order to enable areas to reach and maintain attainment of the ozone standard between 2007 and 2030.

In addition, the substantial reductions from today's rule will greatly lower ozone concentrations which will help federal and State efforts to bring about attainment with the current 1-hour ozone standard. As described in the Air Quality Modeling Technical Support Document for this rule, EPA performed regional scale ozone modeling for the Eastern U.S. to assess the impacts of the controls in this rule on predicted 1-hour ozone exceedances. The results of this modeling were examined for those 37 areas in the East for which EPA's modeling predicted exceedances in 2007, 2020 and/or 2030 and current 1-hour design values are above the standard or within 10 percent of the standard. The results for these areas combined indicate that there will be substantial reductions in the number of exceedances and the magnitude of high ozone concentrations in both 2020 and 2030 due to this rule. The modeling also indicates that without the rule exceedances would otherwise increase by 37 percent between 2020 and 2030 as growth in emissions offsets the reductions from Tier 2 and other current control programs.

For all areas combined, the rule is forecast to provide a 33 percent reduction in exceedances in 2020 and a 38 percent reduction in 2030. The total amount of ozone above the standard is expected to decline by nearly 37 percent in 2020 and 44 percent in 2030. Also, daily maximum ozone exceedances are lowered by 5 ppb on average in 2020 and nearly 7 ppb in 2030. The modeling forecasts an overall net reduction of 39 percent in exceedances from 2007, which is close to the start of this program, to 2030 when controls fully in place. In addition, the results for each individual area indicates that all areas are expected to have less exceedances in 2030 with the HDV controls than without this rule.

Table II.A-2. Eastern Metropolitan Areas with Modeled Exceedances of the 1-Hour Ozone Standard in 2007, 2020, or 2030 With and Without Emission Reductions from this Rule

MSA or CMSA / State	2007b	2020b	2020c	2030b	2030c	pop (1999)
Atlanta, GA MSA	x	x	x	x	x	3.9
Barnstable-Yarmouth, MA MSA *	x			x		0.2
Baton Rouge, LA MSA	x	x	x	x	x	0.6
Beaumont-Port Arthur, TX MSA	x	x	x	x	x	0.4
Benton Harbor, MI MSA *	x	x	x	x	x	0.2
Biloxi-Gulfport-Pascagoula, MS MSA *	x	x	x	x	x	0.3
Birmingham, AL MSA	x	x	x	x	x	0.9
Boston-Worcester-Lawrence, MA CMSA	x	x	x	x	x	5.7
Charleston, WV MSA *	x	x	x	x		0.3
Charlotte-Gastonia-Rock Hill, NC MSA	x	x	x	x	x	1.4
Chicago-Gary-Kenosha, IL CMSA	x	x	x	x	x	8.9
Cincinnati-Hamilton, OH-KY-IN CMSA *	x	x	x	x	x	1.9
Cleveland-Akron, OH CMSA *	x	x	x	x	x	2.9
Detroit-Ann Arbor-Flint, MI CMSA	x	x	x	x	x	5.4
Grand Rapids-Muskegon-Holland, MI MSA*	x	x	x	x	x	1.1
Hartford, CT MSA	x	x	x	x	x	1.1
Houma, LA MSA *	x	x	x	x	x	0.2
Houston-Galveston-Brazoria, TX CMSA	x	x	x	x	x	4.5
Huntington-Ashland, WV-KY-OH MSA	x	x	x	x	x	0.3
Lake Charles, LA MSA *	x	x		x	x	0.2
Louisville, KY-IN MSA	x	x	x	x	x	1
Macon, GA MSA	x	x		x		0.3
Memphis, TN-AR-MS MSA	x	x	x	x	x	1.1
Milwaukee-Racine, WI CMSA	x	x	x	x	x	1.7
Nashville, TN MSA	x	x	x	x	x	1.2
New London-Norwich, CT-RI MSA	x	x	x	x	x	0.3
New Orleans, LA MSA *	x	x	x	x	x	1.3
New York-Northern NJ-Long Island, NY-NJ-CT-PA CMSA	x	x	x	x	x	20.2
Norfolk-Virginia Beach-Newport News, VA-NC MSA *	x	x		x	x	1.6
Orlando, FL MSA *	x	x	x	x	x	1.5
Pensacola, FL MSA	x					0.4
Philadelphia-Wilmington-Atlantic City, PA-NJ-DE-MD CMSA	x	x	x	x	x	6
Providence-Fall River-Warwick,RI-MAMSA*	x	x	x	x	x	1.1
Richmond-Petersburg, VA MSA	x	x	x	x	x	1
St. Louis, MO-IL MSA	x	x	x	x	x	2.6
Tampa-St. Petersburg, FL MSA *	x	x	x	x		2.3
Washington-Baltimore	x	x	x	x	x	7.4
Total number of areas	37	35	32	36	32	
Population	91.2	90.6	88.5	90.8	87.8	91.4

* These areas have registered 1997-1999 ozone concentrations within 10 percent of standard.

The inventories that underlie the photochemical ozone modeling conducted for this rulemaking included reductions from all current or committed federal, State and local controls and, for the control case, the Heavy-Duty Vehicle and Diesel Fuel Sulfur Program itself. It did not did not attempt to examine the prospects of areas attaining or maintaining the ozone standard with possible future controls (i.e., controls beyond current or committed federal, State and local controls). Therefore, Table II.A-2 should be interpreted as indicating what areas are at risk of ozone violations in 2007, 2020 or 2030 without federal or State measures that may be adopted and implemented after this rulemaking is finalized. We expect many of the areas listed in Table II.A-2 to adopt additional emission reduction programs, but the Agency is unable to quantify or rely upon future reductions from additional State programs since they have not yet been adopted.

The Agency recently redesignated Cincinnati-Hamilton, OH-KY-IN to attainment on June 19, 2000. This determination is based on four years of clean air quality monitoring data from 1996 to 1999 (1999 data was not considered in the Tier 2 air quality analysis or the proposal for this rulemaking), and a downward emissions trend. In today's action, Cincinnati-Hamilton is considered to have some risk of registering exceedances of the 1-hour ozone standard during the time period when the HD vehicle standards would take effect. This determination is based on air quality monitoring analysis and 1999 data with concentrations within 10 percent of the standard. Given these circumstances, the risk of future exceedances occurring in the Cincinnati-Hamilton area is most prevalent in the time period beyond the end date of Cincinnati's proposed 10-year maintenance plan (i.e., after 2010). As discussed in more detail in the relevant portions of the response to comment document for the Cincinnati-Hamilton attainment determination, any emissions and ozone modeling system used to predict future ozone involves approximations and uncertainties, and are best treated as indicators of risk rather than absolute forecasts. Thus a determination made in this rule that there is some risk of future exceedances during the relevant time period is not inconsistent with EPA approval of Cincinnati's redesignation to attainment, and its approval of Cincinnati's 10-year maintenance plan.[7]

d. Ozone Modeling and Analysis in 1-Hour State Implementation Plan Submittals and Other Local Ozone Modeling

i. Overview

We have compared and supplemented our own ozone modeling with other modeling studies, submitted to us as state implementation plan (SIP) revisions, or brought to our attention through our consultations with states on SIP revisions that are in development. The ozone modeling in the SIP revisions has the advantage of using emission inventories that are more specific to the area being modeled, and of using meteorological conditions selected specifically for each area. Also, the SIP revisions included other evidence and analysis, such as analysis of air quality and emissions trends, observation based models that make use of data on concentrations of ozone precursors, alternative rollback analyses, and information on the

responsiveness of the air quality model. For some areas, we decided that the predictions of future ozone concentrations from our modeling were less reliable than conclusions that could be drawn from this additional evidence and analysis. For example, in some areas our episodes did not capture the meteorological conditions that have caused high ozone, while local modeling did so. Thus, these local analyses are considered to be more extensive than our own modeling for estimating whether there would be NAAQS nonattainment without further emission reductions, when interpreted by a weight of evidence method which meets our guidance for such modeling.

We have reviewed and recently proposed action on SIP submissions from 13 states and the District of Columbia covering 10 serious and severe 1-hour ozone nonattainment areas. We received these submissions as part of the three-phase SIP process described by EPA guidance memos or as part of a request for an attainment date extension. These submissions also provided ozone modeling results for two attainment areas in a downwind state. These submissions contain local ozone modeling which we considered along with the results of the EPA ozone modeling conducted for this rule. We have also considered ozone modeling submitted as part of an attainment date extension requests for Beaumont-Port Arthur, TX, and Dallas/Fort Worth, TX. Finally, we have considered information in the most recent SIP submittal from California for the South Coast Air Basin. Table II.A-3 lists the areas involved, whether the modeling indicates attainment without further reductions and the Federal Register citation for our proposed action if applicable. This section discusses the background for the submissions and our findings base on them.

It is important to note that the information contained in this section on current and future ozone nonattainment is current as of December 1, and there may have been recent developments in some areas that are not incorporated here.

The local modeling analyses generally cover a modeling domain encompassing one or a few closely spaced nonattainment areas and a limited upwind area. Because of this limited domain, states have been able to use grid cells of 4 or 5 kilometers on a side, in keeping with EPA guidance for such modeling. The future attainment date examined differs from State to State depending on its current (or proposed extended) attainment deadline. In the State modeling, ozone episode days were selected by the respective states based on days with high ozone in the local domain being modeled. In all cases, we are proposing to find that the selection of episode days met our guidance. The local modeling also may make use of location-specific emission data and control programs than is practicable to include in regional-scale modeling by EPA as described above.

The SIP submissions for the 13 states and the District of Columbia covering 10 nonattainment areas contain many legally required elements in addition to the attainment demonstrations. After considering the attainment demonstrations and these other elements, we have proposed appropriate action on each of these submissions. In many cases, we have

proposed alternative actions on our part, based on whether the state submits additional SIP elements which we have described as necessary. We also explained what each state must provide us in order to allow us to take final approval or conditional approval action.

More specific descriptions of the ozone modeling contained in the SIPs, for areas where we have recently proposed action on a submittal, and more explanation of our evaluation of it can be obtained in the individual Federal Register notices and in the technical support document prepared for each action.

Table II.A-3. Nonattainment Areas For Which EPA Has Proposed Action On SIP Submissions Containing 1-hour Ozone Attainment Demonstrations or Otherwise Has Considered Results of Local Ozone Modeling

Nonattainment Area (Major Metro Area)	Affected States	Attainment Date	Indicates Attainment Without "Further Reductions"
Western Massachusetts* (Springfield)	MA	2003 (Requested Extension)	Yes
Greater Connecticut (Hartford and other MSAs)*	CT	2007 (Requested Extension)	Yes, but CT's extension request is based on Greater CT's inabiity to attain because it is affected by transport from the NYC Metropolitan Area.
New York City*	NY, CT, NJ	2007	No
Philadelphia*	PA, NJ, DE, MD	2005	No
Baltimore*	MD	2005	No
Washington, D.C.*	MD, VA, D.C.	2005 (Requested Extension)	Yes
Atlanta*	GA	2003 (Requested Extension)	No
Houston*	TX	2007	No
Chicago*	IL, IN	2007	Revised SIP attainment modeling accounts for reductions from this rule.
Milwaukee*	WI	2007	Revised SIP attainment modeling accounts for reductions from this rule.
Benton Harbor	MI	N/A	Revised SIP attainment modeling accounts for reductions from this rule.
Grand Rapids	MI	N/A	Revised SIP attainment modeling accounts for reductions from this rule.
Dallas	TX	2007 (Requested Extension)	No
Beaumont-Port Arthur	TX	2007 (Requested Extension)	EPA weight of evidence proposed approval based in part of reductions from this rule.
South Coast Air Basin	CA	2010	No

* Proposed for Action in December 16, 1999 Federal Register (64 FR 70318).

ii. Local Ozone Modeling in SIP Submissions

The EPA provides that states may rely on a modeled attainment demonstration supplemented with additional evidence to demonstrate attainment. In order to have a complete modeling demonstration submission, states have submitted the required modeling analysis and identified any additional evidence that EPA should consider in evaluating whether the area will attain the standard.

For purposes of demonstrating attainment, the CAA requires serious and severe areas to use photochemical grid modeling or an analytical method EPA determines to be as effective. The EPA has issued guidance on the air quality modeling that is used to demonstrate attainment with the 1-hour ozone NAAQS.[8] The photochemical grid model is set up using meteorological conditions conducive to the formation of ozone. Emissions for a base year are used to evaluate the model's ability to reproduce actual monitored air quality values and to predict air quality changes in the attainment year due to the emission changes which include growth up to and controls implemented by the attainment year. A modeling domain is chosen that encompasses the nonattainment area. Attainment is demonstrated when all predicted concentrations inside the modeling domain are at or below the NAAQS or at an acceptable upper limit above the NAAQS permitted under certain conditions by EPA's guidance. When the predicted concentrations are above the NAAQS, an optional weight of evidence determination, which incorporates but is not limited to other analyses such as air quality and emissions trends, may be used to address uncertainty inherent in the application of photochemical grid models.

The EPA guidance identifies the features of a modeling analysis that are essential to obtain credible results. First, the State must develop and implement a modeling protocol. The modeling protocol describes the methods and procedures to be used in conducting the modeling analyses and provides for policy oversight and technical review by individuals responsible for developing or assessing the attainment demonstration (State and local agencies, EPA Regional offices, the regulated community, and public interest groups). Second, for purposes of developing the information to put into the model, the State must select air pollution days, i.e., days in the past with bad air quality, that are representative of the ozone pollution problem for the nonattainment area. Third, the State needs to identify the appropriate dimensions of the area to be modeled, i.e., the domain size. The domain should be larger than the designated nonattainment area to reduce uncertainty in the boundary conditions and should include large upwind sources just outside the nonattainment area. In general, the domain is considered the local area where control measures are most beneficial to bring the area into attainment. Fourth, the State needs to determine the grid resolution. The horizontal and vertical resolutions in the model affect the dispersion and transport of emission plumes. Artificially large grid cells (too few vertical layers and horizontal grids) may dilute concentrations and may not properly consider impacts of complex terrain, complex meteorology, and land/water interfaces. Fifth, the State needs to generate meteorological conditions that describe atmospheric conditions and emissions

inputs. Finally, the State needs to verify the model is properly simulating the chemistry and atmospheric conditions through diagnostic analyses and model performance tests. Once these steps are satisfactorily completed, the model is ready to be used to generate air quality estimates to support an attainment demonstration.

The modeled attainment test compares model predicted 1-hour daily maximum concentrations in all grid cells for the attainment year to the level of the NAAQS. A predicted concentration above 0.124 ppm ozone indicates that the area is expected to exceed the standard in the attainment year and a prediction at or below 0.124 ppm indicates that the area is expected to attain the standard. This type of test is often referred to as an exceedance test. The EPA's guidance recommends that states use either of two modeled attainment or exceedance tests for the 1-hour ozone NAAQS: a deterministic test or a statistical test.

The deterministic test requires the State to compare predicted 1-hour daily maximum ozone concentrations for each modeled day[e] to the attainment level of 0.124 ppm. If none of the predictions exceed 0.124 ppm, the test is passed.

The statistical test takes into account the fact that the form of the 1-hour ozone standard allows exceedances. If, over a three-year period, the area has an average of one or fewer exceedances per year, the area is not violating the standard. Thus, if the State models a very extreme day, the statistical test provides that a prediction above 0.124 ppm up to a certain upper limit may be consistent with attainment of the standard. (The form of the 1-hour standard allows for up to three readings above the standard over a three-year period before an area is considered to be in violation.)

The acceptable upper limit above 0.124 ppm is determined by examining the size of exceedances at monitoring sites which *meet or attain* the 1-hour NAAQS. For example, a monitoring site for which the four highest 1-hour average concentrations over a three-year period are 0.136 ppm, 0.130 ppm, 0.128 ppm and 0.122 ppm is attaining the standard. To identify an acceptable upper limit, the statistical likelihood of observing ozone air quality exceedances of the standard of various concentrations is equated to severity of the modeled day. The upper limit generally represents the maximum ozone concentration level observed at a location on a single day and it would be the only level above the standard that would be expected to occur no more than an average of once a year over a three-year period. Therefore, if the maximum ozone concentration predicted by the model is below the acceptable upper limit, in this case 0.136 ppm, then EPA might conclude that the modeled attainment test is passed. Generally, exceedances well above 0.124 ppm are very unusual at monitoring sites meeting the NAAQS. Thus, these upper limits are rarely significantly higher than the attainment level of 0.124 ppm.

[e] The initial, "ramp-up" days for each episode are excluded from this determination.

When the modeling does not conclusively demonstrate that the area will attain, additional analyses may be presented to help determine whether the area will attain the standard. As with other predictive tools, there are inherent uncertainties associated with modeling and its results. For example, there are uncertainties in some of the modeling inputs, such as the meteorological and emissions data bases for individual days and in the methodology used to assess the severity of an exceedance at individual sites. The EPA's guidance recognizes these limitations, and provides a means for considering other evidence to help assess whether attainment of the NAAQS is likely. The process by which this is done is called a weight of evidence (WOE) determination.

Under a WOE determination, the State can rely on and EPA will consider factors such as other modeled attainment tests, e.g., a rollback analysis; other modeled outputs, e.g., changes in the predicted frequency and pervasiveness of exceedances and predicted changes in the design value; actual observed air quality trends; estimated emissions trends; analyses of air quality monitored data; the responsiveness of the model predictions to further controls; and, whether there are additional control measures that are or will be approved into the SIP but were not included in the modeling analysis. This list is not an exclusive list of factors that may be considered and these factors could vary from case to case. The EPA's guidance contains no limit on how close a modeled attainment test must be to passing to conclude that other evidence besides an attainment test is sufficiently compelling to suggest attainment. However, the further a modeled attainment test is from being passed, the more compelling the WOE needs to be.

Special explanation is necessary on the issue of how the NOx SIP Call/Regional Ozone Transport Rule has been handled by states in their local ozone modeling. In most of the local ozone modeling in these SIP revisions, upwind NOx reductions have been assumed to occur through implementation of the NOx SIP Call/Regional Ozone Transport Rule in some or all of the states subject to that rule, even though all states' rules to implement those reductions have not yet been adopted. Where upwind and local implementation of the NOx SIP Call is assumed, our conclusion that the modeling shows that an area cannot attain the NAAQS means that it cannot attain even with the prior implementation of the NOx SIP Call.[f] For the purpose of this rule, EPA has incorporated the emission reductions from the NOx SIP Call into its evaluation of whether further reductions are needed. Absent such reductions, the need for additional reductions is even greater.

[f] Our recent proposals on the SIPs explain how we propose to approach the approval of 1-hour attainment SIPs themselves with respect to the NOx SIP Call. To summarize, we have proposed to approve a SIP which assumes implementation of the NOx SIP Call provided that the State is committed to implementing the NOx reductions within the in-State portion of the modeling domain of the subject nonattainment area. Reductions outside the domain and in other states may be assumed even if a commitment is currently lacking for those areas.

iii. Conclusions from the Local Modeling in SIP Submittals

As discussed previously, we have recently been able to review ozone modeling and other evidence on the likelihood of attainment for ten major metropolitan nonattainment areas. The local modeling only addresses the current and requested attainment date in each area. For the areas involved, these dates fall between 2003 and 2007. The State and local ozone modeling therefore does not address attainment prospects beyond 2007. In December, 1999, the Agency proposed to approve attainment demonstrations for these 10 areas, in some cases with, and in others without, a requirement that states adopt additional measures. More recently, we proposed to approve an attainment demonstration for St. Louis.

All of the states have made use of the weight of evidence concept in their attainment demonstrations. EPA has proposed to find that some of the demonstrations are adequate, while for others additional reductions are needed to attain. We are in some cases proposing to approve demonstrations that depend on emission reductions from measures that the State has not yet adopted and has not yet made a legally enforceable commitment to adopt and implement. Before we take final and unconditional action on an attainment demonstration in such a case, the state will have to adopt all the necessary rules or make enforceable commitments to adopt them.

These State-specific conclusions are not final and we are not making them final via this rule on heavy-duty vehicles. In our final actions on these SIP revisions, we may deviate from our proposal for one or more areas, based on the full record of the rulemaking for each, including any comments received after today. However, we have used the ozone attainment assessments as described below in analyzing the need for additional emission reductions in these areas.

For the New York Metro area, Philadelphia, Baltimore, and Houston nonattainment areas, the EPA has proposed to determine that additional emission reductions beyond those provided by the SIP submission are necessary for attainment. A portion of that reduction will be achieved by federal actions, such as the Tier 2/Sulfur program. In the case of Washington DC, the Tier 2/Sulfur program will provide additional emission reductions needed to keep local emissions in 2005 at or below the levels needed to attain. However, as discussed subsequently, there is still a risk of future nonattainment in the Washington, DC area in 2007 and later due to inherent uncertainties in air quality forecasting and future exceedances predicted by Tier 2 air quality modeling.

As a result of EPA's review of the states' SIP submittals, EPA believes that the ozone modeling submitted by the applicable states for the Chicago, IL, Greater CT (Hartford and New London metropolitan areas), Southeast Desert, and Milwaukee, WI areas demonstrated attainment through the control measures contained in the submitted attainment strategy. Illinois, Indiana, and Wisconsin must submit further SIP revisions, including updated modeling for the Chicago and Milwaukee nonattainment areas by December 2000. For these areas, the updated

regional ozone modeling conducted by the Lake Michigan Air Directors Consortium on behalf of the states relies in part on reductions from this rulemaking.[g] Thus, the 2007 attainment demonstrations for these areas will be based in part on reductions from this rule.

Greater Connecticut and the Southeast Desert are subject to transport from upwind areas that need additional reductions in order to reach attainment in 2007 (New York City), or 2010 (South Coast Air Basin).[h] If attainment is not achieved by New York and South Coast, it is unlikely that Greater Connecticut and the Southeast Desert will achieve attainment. Since New York and the South Coast need further reductions that this rule will in part satisfy, reductions from this rule will also assist downwind nonattainment areas such as Greater Connecticut and the Southeast Desert to reach attainment in 2007 and maintain the ozone NAAQS in future years.

Atlanta's statutory attainment date as a serious 1-hour ozone nonattainment area was November 1999, which it has not met. Georgia has requested an attainment date extension for Atlanta to November 15, 2003 and has proposed an emission control program to achieve attainment by that date. The EPA has proposed to assign Atlanta an attainment date of November 2003 based on a successful demonstration by the State that the control strategy described in the SIP will achieve attainment by this date. All of the measures in that strategy – as well as the measures identified as "additional measures" that were not modeled but needed for attainment in the weight of evidence analyses – have been adopted. It is clear from the amount of emission reductions from these measures that the nonattainment status of Atlanta would extend into the 2004 and later period if only "previous" emission reductions (i.e., reductions in the modeled strategy) were considered. The modeling for Atlanta assumed implementation of the NOx SIP Call outside the local modeling domain. The NOx reductions relied on in the Atlanta SIP local modeling domain are slightly greater than the NOx reductions that are expected to be achieved under the NOx SIP call.

The specific reasons for reaching these conclusions are explained in the individual Federal Register notices.

[g] Lake Michigan Air Directors Consortium. Midwest Subregional Modeling: 1-Hour Attainment Demonstration - Tier II/Low Sulfur Controls. November 8, 1999.

[h] EPA approved the South Coast's "additional measures" relying on new technologies under Clean Air Act section 182(e) in 1995. 60 FR 43379 (August 21, 1995). Emissions reduction shortfall was quantified at 60 tons per day (tpd) for NOx, and 79 tps of VOC. These measures are discussed in the 1994 California SIP (Volume I, p. I33, I-35 and Volume II, p. I-29, I-31). In addition, EPA found shortfalls remaining in the mobile source emissions reductions needed for attainmetn of the 1-hour ozone standard in the South Coast (64 FR 39923-27, July 23, 1999).

iv. Other Local Ozone Modeling and Ozone Nonattainment Prospects

The photochemical ozone modeling conducted for this rule did not predict exceedances for a number of areas for which other available information, such as local ozone modeling, inventory and air quality trends, demonstrates a risk of future exceedances between 2007 and 2030. Table II.A-4 lists these eight areas. These eight areas will be discussed in subsequent sections along with the 37 areas with predicted ozone exceedances in 2007, 2020, or 2030 (Table II.A-2).

We have received ozone modeling for the Beaumont-Port Arthur nonattainment area.[9] Beaumont-Port Arthur is a moderate ozone nonattainment area which continues to have concentrations above levels of the 1-hour ozone NAAQS. Presently, the State of Texas is seeking our approval for a demonstration that Beaumont-Port Arthur is impacted by ozone transport from the Houston area, in order to support a request that we extend its attainment deadline to 2007 which would be the same as the deadline for Houston. We proposed action on this request on April 16, 1999 (64 FR 18864) and extended the comment period on June 3, 1999 (64 FR 29822). Our Proposed Action indicated that we would approve the attainment date extension request if Texas met several necessary conditions, one of which was submission of an approvable demonstration of attainment showing attainment by that date. Texas submitted revisions to the Beaumont/Port Arthur SIP on November 15, 1999 and April 28, 2000. The modeling analysis in these SIP revisions indicates that after implementation of the State's adopted control strategy nonattainment continues. The State supplied additional evidence to indicate that the area is likely to attain the Standard based on EPA's weight of evidence guidance. We have considered the additional reductions from this rule as part of our proposed action on the State's attainment demonstration.

Texas also submitted a modeling analysis and attainment date extension request for the Dallas-Fort Worth metropolitan area on April 28, 2000.[10] The State has requested to extend the attainment date to 2007. The SIP revision includes the State's adopted control strategy and a modeling analysis and weight of evidence demonstration. Our preliminary finding is that the combined modeling and weight of evidence analysis has little if any margin of safety for demonstrating attainment. The Agency's expects that any future proposed determination of the SIP attainment demonstration submitted by Dallas, TX, using meteorology conditions and other inputs selected to be locally applicable, would rely in part on reductions from this rule.

Table II.A-4. Areas With Some Risk of Ozone Violations between 2007 and 2030 Based on Information Other Than Predictive Ozone Modeling

Metropolitan Areas	Basis for Need of Additional Reductions	1999 Population (in millions)
Dallas, TX	Agency expects to rely on reductions from this rule in its proposed weight of evidence determination for the Dallas SIP.	4.9
South Coast Air Basin, CA (Los Angeles-Riverside-San Bernardino)	Emission reduction shortfalls (NOx and VOC) identified in current SIP.	16.0
San Diego, CA	Transport from South Coast Air Basin.	2.8
Southeast Desert, CA	Transport from South Coast Air Basin, significant ozone levels and number of exceedance days (1997-1999).	0.5
Sacramento, CA	Significant ozone levels and number of exceedance days (1997-1999).	1.7
Ventura County, CA	Transport from South Coast Air Basin	0.7
San Joaquin Valley, CA	Area needs to revise SIP for bump-up to severe (2005); significant ozone levels and number of exceedance days (1997-1999)	3.2
San Francisco, CA	Area needs to revise its SIP, significant ozone levels and number of exceedance days (1997-1999).	6.9
8 areas		**36.8**

We have not received any recent ozone modeling from California, because California submitted and we approved the SIPs for nonattainment areas in California some time ago. It is appropriate for us to consider the need for further emission reductions in order for areas in California to attain and maintain. California contains many of the most ozone-impacted areas in the nation. Nine areas in California currently designated as nonattainment (and two counties currently designated as being in attainment) with a population of approximately 32 million have

1997-1999 design values above the 1-hour ozone NAAQS. Seven of the nonattainment areas have approved SIPs, including demonstrations of attainment for their required date. Emissions reductions expected from federal programs, such as the Tier 2/Sulfur rule, represent only a small fraction of the emission reductions needed in the South Coast to attain the NAAQS.

Ozone levels in the South Coast Air Basin have declined over the last two decades, but this area continues to register some of the highest ozone concentrations and the greatest number of exceedance days in the nation. In the three year period from 1997 to 1999, the South Coast recorded a peak ozone level of 0.211 ppm and averaged 39 days above the 1-hour ozone standard. The South Coast has an approved SIP, but it contains shortfalls that must be filled if the area is to reach attainment in 2010. The South Coast generates ozone and ozone precursors that affect the air quality and attainment prospects of downwind areas such as Ventura County,[i] San Diego, and the Southeast Desert.

The transport of ozone and its precursors from the South Coast to downwind areas such as the San Diego, Ventura County and the Southeast Desert has been established by the California Air Resources Board.[j] In addition to receiving transport from the South Coast, the Southeast Desert registered a significant peak ozone concentrations of 0.170 ppm and exceeded the 1-hour ozone standard 24 days on average between 1997-1999. While these areas may have earlier attainment dates, their ability to attain the ozone NAAQS depends in part on attainment by the South Coast. Reductions from this action will provide NOx and VOC reductions needed to help fill shortfalls identified in the South Coast's approved SIP. By extension, since attainment in the South Coast would assist efforts to reach attainment in Ventura and the Southeast Desert by their respective deadlines (2005 and 2007), these two areas, along with San Diego, are also dependent on South Coast reductions associated with today's action to maintain attainment in the future.

We expect that California will be submitting one or more revisions since it appears that one unclassified nonattainment area with a 2000 attainment deadline and one serious classification nonattainment area in California with an attainment deadline of 1999 have not met that date. These areas are San Francisco and the San Joaquin Valley. San Francisco had a violation in its attainment year (2000), which may require the area to submit a revised attainment

[i] *Assessment and Mitigation of the Impacts of Transported Pollutants on Ozone Concentrations within California*, California Air Resources Board, June, 1990. This photochemical grid model analysis found that on some days emissions from the South Coast Air Basin contribute in a significant way to ozone concentrations in Ventura County.

[j] Regulations on "Rulemaking on the Assessment of the Impacts of Transported Pollutants on Ozone Concentrations in California" were approved by the California Office of Administrative Law on August 27, 1997. Note that for purposes of the CARB's transport assessment, the Southeast Desert is divided into two air basins: the Mojave Desert Air Basin and the Salton Sea Air Basin (Title 17, CA Code of Regulations, 60104, 60109, 60114).

plan. From 1997 to 1999, the area registered a peak ozone value of 0.139 ppm and averaged about 3 exceedance days per year. San Francisco does not currently have an attainment classification.

San Joaquin has had too many exceedances to be eligible for an extension and EPA has proposed to bump-up the area to severe classification with a 2005 attainment date. From 1997 to 1999, the area recorded a peak ozone level of 0.161 ppm and exceeded the ozone standard 14 days on average. In fact, since 1991, the area has consistently registered peak ozone levels of around 0.160 ppm. The magnitude and persistence of peak ozone levels in San Joaquin Valley is an important factor to consider when attempting to assess future attainment prospects.

Sacramento has an approved SIP. However, between 1997-1999, the area registered a peak ozone concentration of 0.148 ppm and five exceedances days per year on average. These ozone levels and number of exceedance days suggest that this area has some risk of not attaining the standard by its attainment date.

San Diego is subject to transport from the South Coast Air Basin, and has registered significant ozone levels from 1997-1999. This area was granted a 1-year attainment date extension under the provisions of CAA section 181(a)(5), and appears to be eligible for a second 1-year attainment date extension based on clean air in 2000.

v. Areas At Risk of Exceeding the 1-Hour Ozone Standard in the Future

This section collects the information previously presented on the attainment prospects of areas across the nation based on both photochemical ozone modeling and other local factors such as magnitude and persistence of ozone exceedances, emissions inventory trends, local modeling, SIP status, and transport from areas with later attainment dates.[k] The Agency's conclusions about the risk of future nonattainment is provided in Table II.A-6, which is separated into two broad groups: (1) those areas with attainment dates in 2007 or 2010 that will benefit from reductions from this rule to attain and maintain the standard; and (2) those areas with attainment dates prior to 2007 that will benefit from reductions from this rule to maintain the standard after their attainment dates. Because ozone concentrations causing violations of the 1-hour ozone standard are well established to endanger public health and welfare, this indicates that it is appropriate for the Agency to set new standards for heavy-duty vehicles.

[k] In the proposal, we relied on photochemical ozone modeling performed for recently promulgated standards on light duty vehicles, or Tier 2. The results presented in this final rulemaking for heavy-duty vehicles and diesel fuel are largely consistent with the findings presented in the proposal, with small differences due to updated emissions inventories. As stated in the proposal, the ozone modeling methodologies used in the proposal and presented here in the final rule are identical.

vi. Areas with 2007 or 2010 Attainment Deadlines

The Clean Air Act requires states to submit a SIP to provide for attainment of the 1-hour ozone standard which includes a demonstration of attainment (including air quality modeling) for their nonattainment areas, as well as emission control measures needed to attain by the attainment date. Once the attainment date arrives, areas that have not attained the standard based on monitoring data are subject to applicable provisions of the Clean Air Act, including the possibility of being required to adopt additional emission control measures. Areas that have attained the standard have the option of applying for redesignation to attainment status, which can permit adjustments in the emission control program.

Table II.A-5 identifies ten ozone nonattainment areas with attainment dates of 2007 or 2010. These ten areas are also listed on the top section of Table II.A-6, which is located at the end of this subsection. Each of these areas will need additional reductions to attain the ozone standard, and will also be able to rely on additional reductions from today's action in order to maintain the standard. There are specific emission reduction shortfalls in attainment SIPs submitted for New York, Houston and the South Coast Air Basin based on the local ozone modeling and other evidence. The Agency has not identified a shortfall in the attainment demonstrations submitted by Greater Connecticut (Hartford and New London, CT), but we have proposed to approve an extension date to 2007 based on Greater Connecticut being unable to attain because it is affected by transport from the New York metropolitan area. Transport of ozone and its precursors from the South Coast to the Southeast Desert, San Diego, and Ventura County hinders the ability of these areas to attain the standard. There is some risk that New York will fail to attain the standard by 2007, and thus a transferred risk that Connecticut will also fail. A similar situation exists in Southern California, where attainment of the South Coast is a precondition of the ability of three downwind areas -- Southeast Desert, San Joaquin Valley, and Ventura County to reach attainment by their respective attainment dates. Additional reductions from this rule will assist New York and Greater Connecticut, and the South Coast and its downwind nonattainment areas, in reaching the standard by each areas' respective attainment date, and maintaining the standard from attainment to 2030.

Chicago and Milwaukee originally submitted modeling which did not indicate a need for additional local reductions. However, required, updated modeling for these two areas relies in part on reductions from this rule.[1] Moreover, the ozone modeling for this rulemaking predicted exceedances in Chicago and Milwaukee in 2007, 2020 and 2030.

[1] *Technical Support Document, Midwest Subregional Modeling: 1-Hour Attainment Demonstration for Lake Michigan Area* and *Emissions Inventory*, Illinois Environmental Protection Agency, Indiana Department of Environmental Management, Michigan Department of Environmental Quality, Wisconsin Department of Natural Resources, September 27, 2000, at 14 and at 8.

Dallas and Beaumont Port-Arthur, TX have requested attainment date extensions to 2007 on the grounds that 2007 is the attainment date for Houston and that local air quality is affected by transport from Houston. We have proposed to grant an extension to Beaumont-Port Arthur. We have not yet proposed any action on Dallas. The State of Texas has developed an attainment plan for Dallas, which is a precondition for granting extensions based on transport. In a recently proposed action on attainment SIP submitted for Beaumont-Port Arthur, we have proposed approval based in part on the Agency's weight of evidence determination that included in its consideration expected reductions from today's action. We expect that we will also consider reductions from today's action in our action on the Dallas/Fort Worth plan. Furthermore, EPA's ozone modeling indicated exceedances in Beaumont Port-Arthur in 2007. Although there were no exceedances predicted in the future-year scenarios for Dallas in the modeling by EPA, the episodes used by the state in their local modeling did predict future-year exceedances. We do currently believe these two areas are likely to violate the NAAQS between 2007 and 2030, without more emission reductions in the local areas, and/or from the upwind Houston area, and/or from today's action.

The Los Angeles (South Coast Air Basin) ozone attainment demonstration is fully approved, but it is based in part on reductions from new technology measures that have yet to be identified. The 2007 attainment demonstration for the Southeast Desert area is also approved. However, a transport situation exists between the Southeast Desert areas and the South Coast Air Basin, such that attainment in the Southeast Desert depends on progress in reducing ozone levels in the South Coast Air Basin.

Table II.A-5. Metropolitan Areas With Established or Requested 2007 or 2010 Attainment Deadlines

Metropolitan Area	*Attainment Dates*	*Future Attainment Prospects*	*Metropolitan Area 1999 Population (in millions)*
New York City, NY-NJ-CT	2007	VOC and NOx Shortfall	20.0
Houston, TX	2007	NOx Shortfall	4.5
Hartford, CT	2007 (requested extension)	Contingent on New York Attainment	1.1
New London, CT	2007 (requested extension)	Contingent on New York Attainment	0.3
Chicago, IL-IN	2007	Updated modeling relies in part on reductions from this rule.	8.9
Milwaukee, WI	2007	Updated modeling relies in part on reductions from this rule.	1.6
Dallas, TX	2007 (requested extension)	Local modeling shows nonattainment in 2007	4.9
Beaumont-Port Arthur, TX	2007 (requested extension)	Local modeling shows nonattainment in 2007	0.4
Los Angeles, CA	2010	Approved attainment demonstration, but needs additional reductions to attain	16
Southeast Desert, CA	2007	Approved attainment demonstration, but contingent on South Coast Attainment	0.5
10 Metropolitan Areas		**Total Population (in millions)**	**58.4**

Therefore, these 10 nonattainment areas with about 58 million people will need to rely in part on the reductions from today's action to attain the 1-hour ozone standard by 2007 or 2010, and maintain the standard from 2007/2010 and 2030. We expect to rely in part on these reductions in reaching our final conclusion as to whether each area for which we have reviewed an attainment demonstration is more likely than not to attain on its respective date, whether or not the State formally relies on these reductions as part of its strategy to fill the identified shortfall in its attainment demonstration, if any. This is especially true for those areas that have shortfalls in their attainment demonstrations, or that have air quality modeling that suggests additional reductions are needed. The NOx and VOC reductions in the early years of this program may prove to be a critical part of a range of actions necessary for these areas to overcome their shortfalls and reach attainment.

The emission reductions from this rule will also help these areas reach attainment at lower overall cost, with less impact on small businesses. Following implementation of new controls for regional NOx reductions, states will have already adopted emission reduction requirements for most large sources of VOC and NOx for which cost-effective control technologies are known and for which they have authority to control. Those that must adopt measures to complete their attainment demonstrations therefore will have to consider their remaining alternatives. Many of the alternatives that states may consider could be very costly, and the emissions impact from each additional emissions source subjected to new emissions controls could be considerably smaller than the emissions impact of the standards being proposed today. Therefore, the emission reductions from the standards we are finalizing today will ease the need for states to find first-time reductions from the mostly smaller sources that have not yet been controlled, including area sources that are closely connected with individual and small business activities. The emission reductions from the standards being finalized today will also reduce the need for states to seek even deeper reductions from large and small sources already subject to emission controls.

The Southeast Desert has an approved attainment demonstration, and we have proposed to approve attainment demonstrations in some of the other nine areas without additional emission reductions from local measures. Even if all shortfalls were filled for each area, this would not mean that there is no danger that ozone levels in these areas will exceed the NAAQS, in the absence of today's action Agency approval of an attainment demonstration generally indicates our belief that a nonattainment area is reasonably likely to attain by the applicable attainment date with the emission controls in the SIP. However, such approval does not indicate that attainment is certain. Moreover, no ozone forecasting is 100 percent certain, so attainment by these deadlines is not certain, even though we believe it is more likely than not. There are significant uncertainties inherent in predicting future air quality, such as unexpected economic growth, unexpected VMT growth, the year-to-year variability of meteorological conditions conducive to ozone formation, and modeling approximations. Ozone formation is highly dependent on local weather conditions. In fact, the variability in observed ozone due to

meteorology can be larger than the ozone reductions yielded from a significant emission reduction.[m] There is at least some risk in each of these ten areas that even assuming all shortfalls are filled, attainment will not be reached by the applicable dates without further emission reductions. The Agency's mid-course review in the SIP process -- as well as the Clean Air Act's provisions for contingency measures -- is part of our strategy for dealing with some of these uncertainties, but does not ensure successful attainment.

Where we have proposed a specific amount of additional reductions needed for attainment, there is a risk that violations would occur in 2007 even if the additional measures for this amount of reduction are adopted. In addition to all of the factors mentioned above in connection with the Southeast Desert and the areas for which we did not identify a shortfall, there is uncertainty in the conclusion about the existence and size of the shortfall. The shortfalls were identified through consideration of a variety of evidence, without actual ozone modeling on the effect of the additional emissions reductions.

Given the political, human, and economic factors involved, until the affected states actually submit their emission control measures to make up the shortfalls, there is some risk that the eight areas presently without approved attainment demonstrations will not adopt fully approvable SIPs. In addition, some of these SIPs assume reductions in NOx emissions in upwind areas in other states. Until those controls are adopted and implemented, those reductions are somewhat uncertain. Also, success in implementing all the in-state measures in the SIPs once they are developed and approved is somewhat uncertain, and this contributes to the risk that 2007 attainment will not happen. This possibility contributes to the risk that each of these areas will have violations in 2007 despite all efforts to reach attainment.

If an area with a 2007 attainment date does fail to demonstrate actual attainment of the 1-hour ozone NAAQS based on 2005-2007 ozone data, the Clean Air Act allows EPA to grant it up to two one-year extensions, provided there has not been more than one exceedance of the standard in the year prior to the attainment year. The emission reductions from the rule in 2008 and 2009 will be even larger than the reductions in 2007, and can play an important role in allowing an area that needs these extensions to attain in 2008 or 2009.

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[m] An analysis of ambient 1-hour design values for three, 3-year time periods between 1994 and 1998 for monitoring sites in the East indicates a 10 percent swing in the 90 percent percentile design values. Thus, if an area just attains in 2007, there is a risk that it could fall back into nonattainment in subsequent time periods due to year-to-year variations in meteorology, assuming emissions do not change or change very little. The net NOx emissions reductions due to Tier 2 in 2007 is 4 percent considering all Eastern States collectively. The Tier 2 modeling indicates that this level of NOx reductions results in ozone reduction on the order of generally 1-3 ppb ozone. The 1-3 ppb reductions associated with the 4 percent Tier 2 NOx reductions are small compared to the effects of variations in ozone due to meteorology. It is important to note that the episodes modeled by the Agency, though not "worst case," may be somewhat more severe for most areas than meteorological conditions associated with recent design values. Thus, modeling with these episodes that indicates attainment for an area is likely to be conservative.

The Agency regards the continuing reductions from the 2007 heavy-duty rule as part of the federal/State effort not only to reach attainment in the 2007 to 2009 time frame, but to ensure that attainment is maintained in the future. The ability of states to maintain the ozone NAAQS once attainment is reached has proved challenging, and the recent recurrence of violations of the NAAQS in some other areas increases the Agency's concern about continuing maintenance of the standard in these ten areas (and other areas discussed later) once attainment is achieved. Agency uncertainty about the prospects of continued maintenance of the standard is also due, in part, to the fact that State attainment demonstrations generally do not model beyond their particular attainment date, and EPA does not insist that states prepare maintenance plans prior to their request for redesignation to attainment after they have attained. Local modeling and our review of the SIPs did not address whether additional reductions from fleet turnover would offset factors that might cause violations after their attainment dates.

Recurrent nonattainment is especially problematic for areas where high population growth rates lead to significant annual increases in vehicle trips and vehicle miles traveled. Another factor that plays a role in long-term maintenance is meteorology. Our guidance to states on ozone modeling for attainment demonstrations is to select high ozone days that are representative of their current ozone design values. Analysis of these conditions are then used to predict future ozone and in evaluating control strategies. When assessing the risk of air pollution that would endanger public health and welfare during the period when the heavy-duty rule will reduce emissions, we think it is appropriate to consider the possibility that year-to-year variability of meteorological conditions conducive to ozone formation may be worse than this sometime in the future. In considering the period for many years beyond 2007, it is possible that some years will have meteorological conditions conducive to ozone formation substantially worse than assumed in the ozone modeling in the attainment demonstration. Moreover, ozone modeling conducted for this rule predicted exceedances in 2020 and 2030, which adds to the Agency's uncertainty about the prospect of continued attainment for these areas.

To conclude, a total of ten metropolitan areas need additional measures to meet the shortfalls in the applicable attainment demonstrations, or are subject to ozone transport from an upwind area that has an identified shortfall. EPA finds that the states responsible may need, among other reductions, the level of reductions provided by this rule in order to fill the shortfalls. We expect to rely in part on these reductions in reaching our final conclusion as to whether each of the eight areas for which we have recently reviewed an attainment demonstration is more likely than not to attain on its respective date, whether or not the State formally relies on these reductions as part of its strategy to fill the identified shortfall in its attainment demonstration. As to all ten areas, even if all shortfalls were filled by the states, there is some risk that at least some of the areas will not attain the standards by their attainment dates of 2007, or 2010 for Los Angeles. In that event, the reductions associated with this program, which increase substantially after 2007, would help assure that any residual failures to attain are remedied. Finally, there is also some risk that the areas will be unable to maintain attainment after 2007. Considered

collectively, there is a significant risk that some areas would not be in attainment throughout the period when the rule will reduce heavy-duty vehicle emissions.

vii. Areas with Pre-2007 Attainment Dates or No Attainment Date

The next group of 20 areas have required attainment dates prior to 2007, or have no attainment date but are subject to a general obligation to have a SIP that provides for attainment and maintenance. These 20 areas are found in the middle of Table II.A-6, which compiles information about the 45 areas of concern. Table II.A-6 is located at the end of this subsection. EPA and the states are pursuing the established statutory processes for attaining, and maintaining the ozone standard, where it presently applies, and EPA has re-instated the ozone standard to the remaining areas. The Agency's finding that there is a significant risk that future air quality would exceed the ozone standard at some time in the 2007 and later period is based on three factors: (1) recent exceedances in 1997- 1999, (2) predicted exceedances in 2007 or 2030 after accounting for reductions from Tier 2 and other federal, local, state or regional controls currently in place or required, and (3) our assessment of the magnitude of recent violations, the year-to-year variability of meteorological conditions conducive to ozone formation, transport from areas with later attainment dates, and uncertainty inherent in SIP attainment planning.

In addition, only a subset have yet adopted specific control measures that have allowed the Agency to approve an attainment plan, and until the SIPs are actually submitted, reviewed and approved, there is some risk that these areas will not adopt fully approvable SIPs. Furthermore, some of these areas are not under a current requirement to obtain EPA approval for an attainment plan. The mechanisms to get to attainment in areas without a requirement to submit an attainment demonstration are less automatic, and more uncertain. Even with suitable plans, implementation success is uncertain, and therefore there is some risk that 2007 attainment, or maintenance thereafter, would not happen. Maintenance plans are not required to contain enforceable measures beyond those in the conforming SIP, and all current maintenance plans will expire prior to the time when the bulk of reductions from this rule will be achieved.

Seven metropolitan areas listed in Table II.A-6 contain a 1-hour ozone nonattainment area, or areas, for which we have approved, or proposed to approve, an attainment demonstration for an attainment date of 2003 or 2005 (including granted or requested extensions). These areas include Atlanta, Philadelphia, Washington DC, Baltimore, Sacramento, Ventura County, and the San Joaquin Valley. For Atlanta, Baltimore, and Philadelphia, we have proposed that specific further emission reductions are needed in order to attain by the applicable attainment date. We have proposed to approve Washington, D.C.'s attainment demonstration without requiring additional local emission reductions beyond what the State is required to implement or has already said it will implement. However, air quality modeling conducted for this rule predicted

exceedances for Washington DC.[n] Baltimore has predicted exceedances under our ozone modeling and has a recognized emissions shortfalls in its attainment demonstration. We have given final approval to the attainment demonstrations for the listed areas in California. Ventura County's air quality (like that of Southeast Desert and San Diego) is greatly affected by transport from the South Coast Air Basin, and has a significant risk of registering ozone exceedances until the South Coast achieves attainment in 2010 or thereafter. Sacramento has a shortfall identified in its approved SIP. The San Joaquin Valley has an approved SIP, but has recently registered some of the highest ozone levels in the nation.

Subject to consideration of comments on our proposed approvals or other new information, we consider it more likely than not that these seven areas with proposed or final attainment demonstrations will attain by their deadlines, provided the identified additional reductions are achieved. However, as noted above for the areas with 2007 or 2010 attainment dates, there are inherent uncertainties in ozone modeling, attainment planning, and control plan implementation. All of the uncertainties and risk factors discussed above in connection with the 2007 and 2010 areas also apply to these areas. As with most of the 2007 and 2010 areas, ozone modeling predicted ozone exceedances in 2007 for many of these areas. There is some risk in each of these areas that attainment will not be reached by its deadline. Furthermore, nonattainment might persist beyond the deadline into the period when additional reductions from the this rule can assist with attainment. Recurrent nonattainment is especially problematic for areas like Atlanta, GA and Sacramento, CA, where high population growth rates lead to significant annual increases in vehicle trips and vehicle miles traveled.

There are eight metropolitan areas still subject to the 1-hour ozone NAAQS which have attainment dates of 1999 or earlier, but have experienced concentrations above the level of the 1-hour ozone NAAQS. These are Baton Rouge, Birmingham, Cincinnati, Louisville, San Diego, San Francisco (moderate, but with a 2000 attainment date), and St. Louis.[o] Ozone modeling

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[n] It is important to note that modeling conducted for this rulemaking shows that areas are at risk of exceeding the ozone standard in 2007, 2020 and 2030, and that this modeling is not related to the modeling analysis performed for the Washington, D.C. nonattainment area, which demonstrates attainment by 2007 when combined with weight of evidence arguments.

[o] Ozone monitoring data showing 1997-1999 violations in Baton Rouge, Phoenix, San Diego, Sacramento, San Francisco, Southeast Desert, Ventura County and the San Joaquin Valley may in some cases still be in need of final confirmation. San Diego had a 1999 attainment date, which it did not meet. However, it experienced only one exceedance in 1999 and so is eligible for an extension to 2000, and then to 2001 if there is only one exceedance in 2000. The occurrence of only a single exceedance in 1999 arguably was attributable to unusual meteorology, and there is a good risk that attainment will not be reached even by 2001. San Francisco was originally classified as a moderate area with a 1996 attainment deadline. In 1995, the area was redesignated to attainment, but subsequently violated the NAAQS. The area was again designated nonattainment and given a 2000 attainment deadline. Data from 1998 make it clear that this area will not attain based on 1998-2000 monitoring data. Based on air quality

predicted 2007, 2020 and/or 2030 exceedances for all of the areas outside of California. The California areas have recent exceedances. San Diego is impacted by South Coast's air quality and recent violations prevent San Francisco from attaining in 2000. In addition, San Francisco is without an approved attainment plan. For some of these areas, we have not yet received, or have not proposed approval of, a SIP revision with a plan to correct the recent violations. Many of these areas may require an attainment date extension while retaining their current classification, or reclassification to a higher classification with a later attainment date. The present absence of an attainment plan increases the risk that nonattainment will persist into the 2007 and later period.

There are another eight areas of concern because of recent concentrations above the level of the 1-hour ozone NAAQS and modeled predictions of 2007 nonattainment, for which the 1-hour ozone NAAQS was recently reapplied, and are re-classified as attainment and have maintenance plans spanning 10 year periods ending between 2005 and 2008. These 8 areas are Charlotte, Grand Rapids, Huntington, Indianapolis, Memphis, Nashville, Houma, and Richmond. Houma (LaFourche Parish), LA does not have a specific attainment date.

EPA has recently reinstated the 1-hour ozone standard. There were seven areas designated attainment with maintenance plans that had violations since revocation between 1996-1998. Four of these areas -- Charlotte-Gastonia, NC, Huntington-Ashland, WV-KY, Nashville, TN, Richmond, VA – also have predicted exceedances. Recent exceedances in these four areas will likely trigger any contingency measures in the maintenance plans that are tied to new ozone violations. However, contingencies tied to air quality were not always a required element or enforced while the standard was revoked in these maintenance plans, and the SIPs may not yet contain adequate provisions to bring these areas into consistent attainment. Our ozone modeling predicted that, even with federal and regional controls in place at the time, these areas are likely to exceed the standard in 2007, 2020 and 2030. EPA will monitor the situation in these areas, and has options for working with the affected states towards further emission reductions if needed. At this time, the Agency has not identified the specific next steps that states might appropriately take to address this situation.

A group of four areas have had the ozone standard revoked, are without maintenance plans, have experienced recent exceedances, and are predicted by ozone modeling to be nonattainment in 2007 if more emission reductions are not implemented. The ozone standard was reinstated for two of these areas -- Boston and Providence. Benton Harbor was officially an unclassifiable/ attainment area prior to the revocation of the 1-hour standard. Massachusetts and Rhode Island have been required to develop and submit new attainment demonstrations for their areas. For all the reasons discussed above in connection with other areas facing the need to

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monitoring data not considered in the Tier 2 analysis and on 10 year emissions projections, the Agency has proposed to redesignate Cincinnati into attainment.

develop and implement an attainment plan, we find that there is some risk that these areas will not consistently attain the standard in 2007 and beyond without additional controls such as those proposed in this rulemaking. For Benton Harbor, there is no automatic requirement for preparation of a new attainment demonstration, adding to the uncertainty about 2007 attainment. There is some risk that these four areas will not attain the standard by 2007 or thereafter without additional control from today's action.

As with other areas discussed above, the absence of enforceable local controls that are demonstrated to be adequate to restore attainment in these areas on a long term basis supports the Agency's finding that there is some risk in these areas that air quality may violate the ozone standard in the 2007 and later period. There will remain risks even if a new plan is developed, adopted, and implemented. All maintenance plans must be revisited eight years after redesignation, and extended another 10 years. When these areas do face the task of planning for maintenance in the period beyond their current maintenance plan, the emission reductions from this rule will help them in reducing the risk of violations in that period.

For all of these 20 areas, EPA and the states are pursuing the established statutory processes for attaining and maintaining the ozone standard. However, only a subset have yet adopted specific control measures that has allowed or, we expect, will allow the Agency to approve an attainment plan. Despite the presence of statutory and regulatory requirements for those six areas, there is thus some uncertainty in whether states will adopt and implement measures to provide the additional reductions needed to attain by 2007. Given the political, human, and economic factors involved, until the SIPs are actually submitted there is some risk that the areas presently without approved attainment demonstrations will not adopt fully approvable SIPs. In addition, some of these SIPs assume reductions in NOx emissions in upwind areas in other states, under the Regional Ozone Transport Rule. Until those controls are adopted and implemented, those reductions are uncertain. Also, success in implementing all the in-state measures in the SIPs once they are developed and approved is uncertain, and this contributes to the risk that 2007 attainment will not happen. This possibility contributes to the risk that each of these areas will have violations in 2007 or thereafter despite all efforts to achieve attainment.

viii. Areas within 10 percent of Violating the Ozone Standard

There are 15 additional metropolitan areas for which the available ozone modeling and other evidence is less clear regarding the need for additional reductions. Our own ozone modeling predicted these 15 areas to need further reductions to avoid exceedances in 2007, 2020 and/ or 2030. The recent air quality monitoring data for these areas shows ozone levels with less than a 10 percent margin below the NAAQS. We believe there is still a risk of that future ozone levels will be above the NAAQS because of the year-to-year variability of meteorological conditions conducive to ozone formation.

ix. Conclusion

In sum, without these reductions, there is a significant risk that an appreciable number of the 45 areas, with a population of 128 million people in 1999, will violate the 1-hour ozone standard during the time period when these standards will apply to heavy-duty vehicles. The evidence summarized in this section, and presented in more detail in the air quality modeling TSD, supports the Agency's finding that emissions of NOx and VOC from heavy-duty vehicles between 2007 and 2030 will contribute to a national ozone air pollution problem that warrants regulatory action under section 202(a)(3) of the Act.

Table II.A-6. Areas and 1999 Populations at Risk of Exceeding the Ozone Standard between 2007 and 2030

MSA/ CMSA / State	1999 Population (in millions)
Areas with 2007/ 2010 Attainment Dates (Established or Requested)	
Beaumont-Port Arthur, TX	0.4
Chicago-Gary-Kenosha, IL-IN-WI	8.9
Dallas-Fort Worth, TX	4.9
Hartford, CT	1.1
Houston-Galveston-Brazoria, TX	4.5
Los Angeles-Riverside-Orange County, CA	16.0
Milwaukee-Racine, WI	1.6
New London-Norwich, CT-RI	0.3
New York-Northern New Jersey-Long Island, NY-NJ-CT-PA	20.2
Southeast Desert, CA	0.5
10 areas	***58.4***
Areas with Pre-2007 Attainment Dates or No Specific Attainment Date, with a Recent History of Nonattainment.	
Atlanta, GA	3.9
Baton Rouge, LA	0.6
Birmingham, AL	0.9
Boston-Worcester-Lawrence, MA-HN-ME-CT	5.7
Charlotte-Gastonia-Rock Hill, NC-SC	1.4
Detroit-Ann Arbor-Fling, MI MSA	5.5
Huntington-Ashland, WV-KY-OH	0.3
Louisville, KY-IN	1.0
Macon, GA MSA	0.3
Memphis, TN-AR-MS	1.1
Nashville, TN	1.2
Philadelphia-Wilmington- Atlantic City, PA-NJ-DE-MD	6
Richmond-Petersburg, VA	1
Sacramento-Yolo, CA	1.7
San Diego, CA	2.8
San Francisco-Oakland-San Jose, CA	6.9
San Joaquin Valley, CA	3.2
St. Louis, MO-IL	2.6
Ventura County, CA	0.7
Washington, DC-Baltimore, DC, MD, VA MSA	7.4
20 Areas	***54.2***

Table II.A-6. Areas and 1999 Populations at Risk of Exceeding the Ozone Standard between 2007 and 2030

Areas with Pre-2007 Attainment Dates and Recent Concentrations within 10 Percent of an Exceedance, But With No Recent History of Nonattainment.	
Barnstable-Yarmouth, MA	0.2
Benton Harbor, MI	0.2
Biloxi-Gulfport-Pascagoula, MS MSA	0.4
Charleston, WV MSA	0.3
Cincinnati-Hamilton, OH-KY-IN	2.0
Cleveland-Akron, OH CMSA	2.9
Grand Rapids-Muskegon-Holland, MI MSA	1.1
Houma, LA	0.2
Lake Charles, LA	0.2
New Orleans, LA MSA	1.3
Norfolk-Virginia Beach-Newport News, VA-NC MSA	1.6
Orlando, FL MSA	1.5
Pensacola, FL MSA	0.4
Providence-Fall River-Warwick, RI-MA	1.1
Tampa-St. Petersburg-Clearwater, FL MSA	2.3
15 areas	***15.7***
Total Areas: 45	**Population: 128**

e. Public Health and Welfare Concerns from Prolonged and Repeated Exposures to Ozone

There exists a large body of scientific literature regarding health and welfare effects of ozone. Initially, research indicates that there were harmful effects resulting from peak ozone levels (e.g., one-hour concentrations above 0.125 ppm). However, in recent years, research has shown that harmful effects can occur from much lower, sustained levels of exposure. Studies of prolonged exposures, those lasting about 7 hours, showed health effects from exposures to ozone concentrations as low as 0.08 ppm. Prolonged and repeated exposures to ozone at these levels are common in areas that do not attain the 1-hour NAAQS, and also occur in areas where ambient concentrations of ozone are in compliance with the 1-hour NAAQS. Thus, adverse health effects from this type of ozone exposure can reasonably be anticipated to occur in the future in the absence of this rule. Adverse welfare effects can also be anticipated, primarily from damage to vegetation at ozone levels below peak levels.

i. Health and Welfare Effects

Studies of acute health effects from ozone have reported ozone exposure to cause or be statistically associated with transient pulmonary function responses, transient respiratory symptoms, effects on exercise performance, increased airway responsiveness, increased susceptibility to respiratory infection, increased hospital and emergency room visits, and transient pulmonary respiratory inflamation. Such acute health effects have been observed following prolonged exposures at moderate levels of exertion at concentrations of ozone as low as 0.08 ppm, the lowest concentration tested. The effects are more pronounced as concentrations increase, affecting more subjects or having a greater effect on a given subject in terms of functional changes or symptoms. A detailed summary and discussion of the large body of ozone health effects research may be found in Chapters 6 through 9 (Volume 3) of the 1996 Criteria Document for ozone.[11]

The following is a brief summary focusing on studies on the effects of exposures to concentrations of ozone just at and below peak ozone concentrations. Tables II.A-7 through II.A-11 of this section are excerpted from the 1996 Criteria Document, with only studies that used peak ozone concentrations or below retained.

It has long been established by exposure chamber studies that single, short-term (1 to 3 hour) exposures to ozone concentrations at or above peak levels produce a variety of respiratory function effects in exposed subjects. Tables II.A-7 and II.A-8 summarize these studies, for healthy and diseased subjects, and also indicate that equally short-term exposures to concentrations below peak levels have not shown these effects. More recent studies have sought to investigate whether similar effects occur following longer exposures to lower levels of ozone. These studies are summarized here in Tables II.A-9 and II.A-10. Exposures of 6.6 hours to ozone concentrations of 0.08 , 0.10, and 0.12 ppm were used in these chamber exposures studies, and are reported to cause decrements in lung function (reduced ability to take a deep breath), increased respiratory symptoms (cough, shortness of breath, pain upon deep inspiration), increased airway responsiveness (an indication that airways are predisposed to broncho-constriction, which is characteristic of asthma), and increased airway inflammation in adults. The effects are more pronounced as concentrations increase, affecting more subjects or having a greater effect on a given subject in terms of functional changes or symptoms. Earlier studies found these effects in heavily exercising adults exposed to ozone on a short-term basis, but the level of exertion involved was high enough to be unusual among people conducting their normal activities. The more recent studies with 6.6 hour exposures at 0.08 and 0.10 ppm observed these functional changes and symptoms when subjects were exerting themselves at only moderate levels. This means that much of the population could experience these effects from ambient concentrations while conducting their normal activities at moderate exertion levels.

With regard to chronic health effects, the collective data from these chamber studies have many ambiguities, but provide suggestive evidence of chronic effects in humans. Table II.A-11 summarizes studies associating a single prolonged exposure to ozone at 0.08 and 0.10 ppm with lung inflammation. There is a biologically plausible basis for considering the possibility that repeated inflammation associated with exposure to ozone over a lifetime, as can occur with exposure to 8-hour ozone levels as low as 0.08 ppm, may result in sufficient damage to respiratory tissue such that individuals later in life may experience a reduced quality of life, although such relationships remain highly uncertain.

A number of "summer camp" studies of children and adolescents, and other types of epidemiological studies involving exposure to ambient concentrations of ozone, confirm that ozone concentrations are correlated with lung function changes, as indicated by the chamber studies. The studies are not summarized in table form here. Changes reported at low ozone concentrations in these studies are comparable to those observed in the chamber studies, although comparisons are difficult because of differences in experimental design and analytical approach. Studies published since 1986 have supported a direct association between ambient ozone/oxidant concentrations and acute respiratory morbidity in asthmatics, although it is difficult to clearly differentiate the independent effects of ozone from those of copollutants. Conclusions from the field studies on asthmatics are based on observations over a range of ozone exposures extending below the 0.12 ppm level of the 1-hour NAAQS.

Over 20 epidemiology studies of aggregate populations have investigated the relationship between ozone concentrations and hospital admissions/ hospital visits. The studies are not summarized in table form here. Significant associations are seen between ozone and hospital admissions/visits at exposures below 0.12 ppm 1-hour daily maximum ozone.

Ozone also has many welfare effects, with damage to plants being of most concern. Plant damage affects crop yields, forestry production, and ornamentals. The adverse effect of ozone on forests and other natural vegetation can in turn cause damage to associated ecosystems, with additional resulting economic losses. Ozone concentrations of 0.10 ppm can be phytotoxic to a large number of plant species, and can produce acute injury and reduced crop yield and biomass production. Ozone concentrations within the range of 0.05 to 0.10 ppm have the potential over a longer duration of creating chronic stress on vegetation that can result in reduced plant growth and yield, shifts in competitive advantages in mixed populations, decreased vigor, and injury. Ozone effects on vegetation are presented in more detail in Chapter 5, Volume II of the 1996 Criteria Document.

Table II.A-7. Controlled Exposure of Healthy Human Subjects to Ozone *

Ozone Concentration		Exposure Duration and Activity	Exposure Conditions	Number and Gender of Subjects	Subject Character-istics	Observed Effect(s)	Reference
ppm	*$\mu g/m^3$*						
Healthy Exercising Adult Subjects							
0.08 0.10 0.12 0.14 0.16	157 196 235 274 314	2 h IE (4 × 15 min at $\dot{V}_E$ = 68 L/min)	Tdb = 32 °C RH = 38%	24 M	Young, healthy adults, 18 to 33 years old	No significant changes in pulmonary function measurements.	Linn et al. (1986)
0.12 0.18 0.24	235 353 470	1 h competitive simulation exposures at mean $\dot{V}_E$ = 87 L/min	Tdb = 23 to 26 °C RH = 45 to 60%	10 M	10 highly trained competitive cyclists, 19 to 29 years old	Decrease in FVC and FEV_1 for 0.18- and 0.24-ppm O_3 exposure compared with FA exposure; decrease in exercise time for subjects unable to complete the competitive simulation at 0.18 and 0.24 ppm O_3, respectively.	Schelegle and Adams (1986)
0.12 0.18 0.24 0.30 0.40	235 353 470 588 784	2.5 h IE (4 × 15 min treadmill exercise [$\dot{V}_E$ = 65 L/min])	Tdb = 22 °C RH = 40%	20 M 22 M 20 M 21 M 20 M 29 M	Young, healthy adults, 18 to 30 years old	Significant decrease in FVC, FEV_1, and $FEF_{25-75\%}$ at 0.12 ppm O_3; decrease in V_T and increase in f and SR_{aw} at 0.24 ppm O_3.	McDonnell et al. (1983)

Table II.A-7. Controlled Exposure of Healthy Human Subjects to Ozone *

0.12 0.18 0.24 0.30 0.40	235 353 470 588 784	2 × 2.5 h IE (4 × 15 min treadmill exercise [$\dot{V}_E$ = 35 L/min/m^2 BSA]). Exposure separated by 48 ± 30 days and 301 ± 77 days	Tdb = 22 °C RH = 40%	8 M 8 M 5 M 5 M 6 M	Young, healthy adults, 18 to 30 years old	Pulmonary function variables SR_{aw} and $\dot{V}_E$ were not significantly different in repeat exposures, indicating that the response to 0.18 ppm O_3 or higher is reproducible.	McDonnell et al. (1985b)
0.12 0.18 0.24 0.30 0.40	235 353 470 588 784	2 × 2.5 h IE (4 × 15 min treadmill exercise [$\dot{V}_E$ = 35 L/min/m^2 BSA])	Tdb = 22 °C RH = 40%	290 M	Young, healthy adults, 18 to 32 years old	O_3 concentration and age predicted FEV_1 decrements; it was concluded that age is a significant predictor of response (older subjects being less responsive to O_3).	McDonnell et al. (1993)
0.12 0.18 0.24 0.30 0.40	235 353 470 588 784	2.5 h IE (4 × 15 min treadmill exercise [$\dot{V}_E$ = 25 L/min/m^2 BSA])	Tdb = 22 °C RH = 40%	17 WM/15 BM/15 WF/ 15BF 15 WM/15 BM/15 WF/ 16BF 15 WM/17 BM/17 WF/ 15BF 16 WM/15 BM/17 WF/ 16BF 15 WM/15 BM/15 WF/ 15BF 15 WM/15 BM/15 WF/ 15BF	Young, healthy whites and blacks, 18 to 35 years old	Decreases in FEV_1 for all levels of O_3 as compared with FA; increase in SR_{aw} with 0.18 ppm O_3 and greater compared with FA; black men and women had larger FEV_1 decrements than white men, and black men had larger FEV_1 decrements than white women.	Seal et al. (1993)

Table II.A-7. Controlled Exposure of Healthy Human Subjects to Ozone *

0.12 0.20	235 392	1 h CE (mean $\dot{V}_E$ = 89 L/min)	Tdb = 31 °C	15 M 2 F	Highly trained competitive cyclists, 19 to 30 years old	Decrease in $\dot{V}_{Emax}$, $\dot{V}O_{2max}$, V_{Tmax}, work load, ride time, FVC, and FEV_1 with 0.20 ppm O_3 exposure during maximal exercise conditions, but not significant with 0.12 ppm O_3 exposure, as compared to FA exposure.	Gong et al. (1986)
0.10 0.15 0.20 0.25	196 294 392 490	2 h IE (4 × 14 min treadmill at mean $\dot{V}_E$ = 70.2 L/min)	Tdb = 22 °C RH = 50%	20 M	Young, healthy NS, 25.3 ± 4.1 (SD) years old	FVC, FEV_1, $FEF_{25-75\%}$, SG_{aw}, IC, and TLC all decreased with (1) increasing O_3 concentration, and (2) increasing time of exposure; threshold for response was above 0.10 ppm but below 0.15 ppm O_3.	Kulle et al. (1985)

* See Appendix A of the 1996 Ozone Criteria Document for abbreviations and acronyms.

Table II.A-8. Ozone Exposure in Subjects with Preexisting Disease

Ozone Concentration ppm	*Ozone Concentration* $\mu g/m^3$	*Exposure Duration and Activity*	*Exposure Condition*	*Number and Gender of Subjects*	*Subject Character-istics*	*Observed Effect(s)*	*Reference*
Subjects with Chronic Obstructive Pulmonary Disease							
0.12	236	1 h IE (2 × 15 min light bicycle ergometry)	Tdb = 25 °C RH = 50%	18 M, 7 F	8 smokers, 14 ex-smokers, 3 nonsmokers; FEV_1/FVC = 32 to 66%	No significant changes in pulmonary function measurements; small significant decrease in arterial O_2 saturation.	Linn et al. (1982a)
Adult Subjects with Asthma							
0.10 0.25 0.40	196 490 784	1 h light IE (2 × 15 min on treadmill, $\dot{V}_E$ = 27 L/min)	Tdb = 21 ° C RH = 40%	12 M, 9 F, 19 to 40 years old	Stable mild asthmatics with FEV_1 > 70% and methacholine responsiveness	No significant differences in FEV_1 or FVC were observed for 0.10 and 0.25 ppm O_3-FA exposures or postexposure exercise challenge; 12 subjects exposed to 0.40 ppm O_3 showed significant reduction in FEV_1.	Weymer et al. (1994)
0.12	236	1 h rest	NA	7 M, 8 F	Never smoked, mild stable asthmatics with exercise-induced asthma	Exposure to 0.12 ppm O_3 did not affect pulmonary function. Preexposure to 0.12 ppm O_3 at rest did not affect the magnitude or time course of exercise-induced bronchoconstriction.	Fernandes et al. (1994)
0.12	236	0.75 h IE $\dot{V}_E$ = 30 L/min (15 min rest, 15 min exercise, 15 min rest) followed by 15 min exercise inhaling 0.10 ppm SO_2	Tdb = 22 ° C RH = 75%	8 M, 5 F, 12 to 18 years old	Asthmatics classified on basis of positive clinical history and methacholine challenge. Asymptomatic at time of study.	Filtered air followed by SO_2 and O_3 alone did not cause significant changes in pulmonary function. Ozone followed by SO_2 resulted in significant decrease in FEV_1 (8%) and $\dot{V}_{max50\%}$ (15%) and a significant increase in R_T (19%).	Koenig et al. (1990)

Table II.A-8. Ozone Exposure in Subjects with Preexisting Disease

0.12 0.24	236 472	1.5 h IE, $\dot{V}_E$ = 25 L/min	Tdb = 22 ° C RH = 65%	4 M, 4 F (nonasthmatics); 18 to 35 years old; 5 M, 5 F (asthmatics); 18 to 41 years old	Physician-diagnosed asthma confirmed with methacholine challenge test. All nonsmokers and asymptomatic at time of study. Nine were atopic.	No significant changes in pulmonary and nasal function measurements in either asthmatics or nonasthmatics. Significant increase in nasal lavage white cell count and epithelial cell following O_3 exposure in asthmatics only.	McBride et al. (1994)
0.12	236	6.5 h/day IE (6 × 50 min) (2 days of exposure), $\dot{V}_E$ = 28 L/min (asthmatic), $\dot{V}_E$ = 31 L/min (healthy)	NA	8 M, 7 F (nonasthmatics); 22 to 41 years old; 13 M, 17 F (asthmatics); 18 to 50 years old	Asthmatics classified on basis of positive clinical history, previous physician diagnosis, and low PD_{20}. Mild to severe asthmatics.	Significant increase in bronchial reactivity to methacholine in both asthmatics and nonasthmatics. FEV_1 decreased 8.6% in asthmatics and 1.7% in nonasthmatics, with difference not being significant.	Linn et al. (1994)
0.12	236	1 h rest	NA	4 M, 3 F, 21 to 64 years old	Mild, stable asthma	Increase in bronchial responsiveness to allergen; no change in baseline airway function.	Molfino et al. (1991)
Adolescent Subjects with Asthma							
0.12	235	1 h rest	Tdb = 22 °C RH ≥ 75%	4 M, 6 F (normals), 13 to 18 years old; 4 M, 6 F (asthmatics), 11 to 18 years old	Asthmatics had a history of atopic extrinsic asthma and exercise-induced bronchospasm	Decrease in FRC with O_3 exposure in asthmatics; no consistent significant changes in pulmonary functional parameters in either group or between groups.	Koenig et al. (1985)

Table II.A-8. Ozone Exposure in Subjects with Preexisting Disease

0.12	235	1 h IE (2 × 15 min treadmill walking at mean $\dot{V}_E$ = 32.5 L/min)	Tdb = 22 °C RH ≥ 75%	5 M, 8 F (normals), 12 to 17 years old; 9 M, 3 F (asthmatics), 12 to 17 years old	Asthmatics selected from a clinical practice and had exercise-induced bronchospasm	Decrease in maximal flow at 50% of FVC in asthmatics with O_3 exposure compared to FA; no significant changes with combined O_3-NO_2 exposure.	Koenig et al. (1988)
0.12 0.18	235 353	40 min IE (1 × 10 min treadmill walking at mean $\dot{V}_E$ = 32.5 L/min)	NA	4 M, 9 F (normals), 14 to 19 years old; 8 M, 8 F (asthmatics), 12 to 19 years old	Asthmatics had allergic asthma, positive responses to methacholine, and exercise-induced bronchospasm	Decrease in FEV_1 and increase in R_T in normals and asthmatics with 0.12 and 0.18 ppm O_3 exposure compared to FA; no consistent differences between normals and asthmatics.	Koenig et al. (1987)

Table II.A-9. Pulmonary Function Effects After Prolonged Exposures to Ozone

Ozone Concentration *ppm*	*$\mu g/m^3$*	*Exposure Duration and Activity*	*Exposure Conditions*	*Number and Gender of Subjects*	*Subject Character-istics*	*Observed Effect(s)*	*Reference*
0.08 0.10 0.12	157 196 235	6.6 h IE (6 × 50 min) $\dot{V}_E \approx 39$ L/min	18 °C 40% RH	22 M	Healthy NS, 18 to 33 years old	FVC and FEV_1 decreased throughout the exposure; FEV_1 decrease at end exposure was 7.0, 7.0, and 12.3%, respectively. FEV_1 change >15% occurred in 3, 5, and 9 subjects at 0.08, 0.10, and 0.12 ppm, respectively. Methacholine responsiveness increased by 56, 89, and 121%, respectively.	Horstman et al. (1990)
		See Horstman et al. (1990) and Folinsbee et al. (1988)				A lognormal model was fitted to FEV_1 data. Model parameters indicate O_3 concentration had greater effect than $\dot{V}_E$ or duration (estimated exponent for $[O_3] \approx 4/3$).	Larsen et al. (1991)
0.08 0.10	157 196	6.6 h IE (6 × 50 min) $\dot{V}_E = 40$ L/min	18 °C 40% RH	38 M	Healthy NS, mean age 25 years old	FEV_1, decreased 8.4% at 0.08 ppm and 11.4% at 0.10 ppm. Symptoms of cough, PDI, and SB increased with O_3 exposure.	McDonnell et al. (1991)
0.08	157	6.6 h IE (6 × 50 min) $\dot{V}_E = 35$ to 38 L/min (1 day of air, 2 days of O_3)	25 °C 48% RH	5 F, 6 M	Healthy NS, 30 to 45 years old	FVC decreased 2.1%, FEV_1 decreased 2.2% on first day of O_3 exposure; no change on second O_3 day.	Horvath et al. (1991)

Table II.A-9. Pulmonary Function Effects After Prolonged Exposures to Ozone

0.12	235	6.6 h IE (6 × 50 min) $\dot{V}_E$ = 42.6 L/min	18 °C 40% RH (1 exposure to clean air; 1 exposure to O_3)	10 M	Healthy NS, 18 to 33 years old	FEV_1 decreased by 13% after 6.6 h. FVC dropped 8.3%. Cough and PDI increased with O_3 exposure. Airway responsiveness to methacholine doubled after O_3 exposure.	Folinsbee et al. (1988)
0.12	235	6.5 h/day IE (6 × 50 min) (2 days of exposure) $\dot{V}_E$ = 28 L/min (asthmatic) $\dot{V}_E$ = 31 L/min (healthy)	21 °C 50% RH	15 (8 M, 7 F) 30 (13 M, 17 F)	Healthy NS, 22 to 41 years old Asthmatic NS, 18 to 50 years old	Bronchial reactivity to methacholine increased with O_3 exposure in healthy subjects. FEV_1 decreased 2% (pre- to postexposure) in healthy subjects and 7.8% in asthmatics. Responses were generally less on the second day. Two healthy subjects and four asthmatics had FEV_1 decreases >10%.	Linn et al. (1994)
0.12	235	6.6 h IE (6 × 50 min) $\dot{V}_E$ = 38.8 L/min	18 °C 40% RH (5 consecutive days of exposure to O_3, 1 day exposure to CA)	17 M	Healthy NS, mean age 25 ± 4 years old	FEV_1 decreased by 12.8, 8.7, 2.5, and 0.6 and increased by 0.2 on Days 1 to 5 of O_3 exposure, respectively. Methacholine airway responsiveness increased by >100% on all exposure days. Symptoms increased on the first O_3 day, but were absent on the last 3 exposure days.	Folinsbee et al. (1994)

Table II.A-9. Pulmonary Function Effects After Prolonged Exposures to Ozone

(a) 0.12 (b) Varied from 0.0 to 0.24 (increased by 0.06 ppm/h then decreased by 0.06 ppm/h)	235	8 h IE (8 × 30 min) $\dot{V}_E$ = 40 L/min	22 °C 40% RH <3 μg/m^3 TSP	23 M	Healthy NS, 20 to 35 years old	(a) FEV_1 decreased 5% by 6 h and remained at this level through 8 h. (b) FEV_1 change mirrored O_3 concentration change with a lag time of ≈ 2 h. Max decrease of 10.2% after 6 h. FEV_1 change was reduced in last 2 h of exposure.	Hazucha et al. (1992)

* See Appendix A of the 1996 Ozone Criteria Document for abbreviations and acronyms.

Table II.A-10. Increased Airway Responsiveness Following Ozone Exposures *

*Ozone Concentration ***		*Exposure Duration and Activity*	*Exposure Conditions*	*Number and Gender of Subjects*	*Subject Character-istics*	*Observed Effect(s)*	*Reference*
ppm	*$\mu g/m^3$*						
0.08 0.10 0.12	157 196 235	6.6 h IE at ≈39 L/min	18 °C 40% RH	22 M	Healthy NS, 18 to 32 years old	33, 47, and 55% decreases in cumulative dose of methacholine required to produce a 100% increase in SR_{aw} after exposure to O_3 at 0.08, 0.10, and 0.12 ppm, respectively.	Horstman et al. (1990)
0.10 0.32 1.00	196 627 1,960	2 h	NA	14	Health NS, 24 ± 2 years old	Increased airway responsiveness to methacholine immediately after exposure at the two highest concentrations of O_3.	König et al. (1980)
0.12 0.20	235 392	1 h at $\dot{V}_E$ = 89 L/min followed by 3 to 4 min at ≈150 L/min	31 °C 35% RH	15 M, 2 F	Elite cyclists, 19 to 30 years old	Greater than 20% increase in histamine responsiveness in one subject at 0.12 ppm O_3 and in nine subjects at 0.20 ppm O_3.	Gong et al. (1986)
0.12	235	6.6 h with IE at ≈25 L/min/m^2 BSA	NA	10 M	Healthy NS, 18 to 33 years old	Approximate doubling of mean methacholine responsiveness after exposure. On an individual basis, no relationship between O_3-induced changes in airway responsiveness and FEV_1 or FVC.	Folinsbee et al. (1988)
0.12 ppm O_3-100 ppb SO_2 0.12 ppm O_3-0.12 ppm O_3 Air-100 ppb SO_2		45 min in first atmosphere and 15 min in second IE	75% RH 22 °C	8 M, 5 F	Asthmatic, 12 to 18 years old	Greater declines in FEV_1 and $\dot{V}_{max50\%}$ and greater increase in respiratory resistance after O_3-SO_2 than after O_3-O_3 or air-SO_2.	Koenig et al. (1990)
Air-antigen 0.12 ppm O_3-antigen		1 h at rest	NA	4 M, 3 F	Asthmatic, 21 to 64 years old	Increased bronchoconstrictor response to inhaled ragweed or grass after O_3 exposure compared to air.	Molfino et al. (1991)

* See Appendix A of the 1996 Ozone Criteria Document for abbreviations and acronyms.

Table II.A-11. Bronchoalveolar Lavage Studies of Inflammatory Effects from Controlled Human Exposure to Ozone

Ozone Concentration[b] *ppm*	*$\mu g/m^3$*	*Exposure Duration*	*Activity Level ($\dot{V}_E$)*	*Number and Gender of Subjects*	*Observed Effect(s)*	*Reference*
0.08 0.10	157 196	6.6 h	IE (40 L/min) six 50-min exercise periods + 10 min rest; 35 min lunch	18 M, 18 to 35 years old	BAL fluid 18 h after exposure to 0.1 ppm O_3 had significant increases in PMNs, protein, PGE_2, fibronectin, IL-6, lactate dehydrogenase, and α-1 antitrypsin compared with the same subjects exposed to FA. Similar but smaller increases in all mediators after exposure to 0.08 ppm O_3 except for protein and fibronectin. Decreased phagocytosis of yeast by alveolar macrophages was noted at both concentrations.	Devlin et al. (1990, 1991) Koren et al. (1991)

ii. Ozone Concentrations

This section summarizes the results of analyses of model-adjusted ozone air quality concentrations and the anticipated air quality impact of reductions in emissions expected to result from implementation of the heavy duty engine and vehicle standards and highway diesel fuel sulfur control requirements. Specifically, it provides information on the number of people estimated to live in metropolitan counties in which ozone monitors are predicted to repeatedly experience certain levels of ozone of potential concern over prolonged periods, i.e., 8-hours.

Heavy-duty vehicles contribute a substantial fraction of ozone precursors in any metropolitan area. Available health studies (summarized above) have indicated health effects (e.g., lung function decrements, respiratory symptoms, and pulmonary inflammation) at ozone concentrations between 0.08 ppm and 0.12 ppm over prolonged exposures (6.6 hours in most chamber studies). An 8-hour averaging period was chosen as a convenient and appropriate metric for describing current and future ozone patterns relevant to this concentration range. Another important metric is the number of days with ozone levels between 0.08 and 0.12 ppm because repeated exposure to ozone in this concentration range may be associated with long term health effects related to pulmonary inflammation.

To provide a quantitative estimate of the number of people anticipated to reside in areas in which ozone concentrations are predicted to experience multiple days with 8-hour ozone in the range of 0.08 to 0.12 ppm and higher, we performed regional modeling for 6 different scenarios (1996 base, 2007 base, 2020 base and control, 2030 base and control) for the eastern United States. This modeling is further described in section A.2.6 “Photochemical Ozone Modeling.” Our analysis relies on projected county-level population from the U.S. Department of Census for the period representing each year analyzed.

For each of the counties analyzed, we determined the number of days for periods on which the highest model-adjusted 8-hour concentration at any monitor in the county was predicted, for example, to be between 0.08 and 0.12 ppm (after rounding from 3 decimal places). We then grouped the counties which had days with ozone in this range according to the number of days this was predicted to happen, and summed their projected populations. We repeated this for ozone ranges of 0.09 to 0.12 ppm, 0.10 to 0.12 ppm, 0.11 to 0.12 ppm and greater than or equal to 0.12 ppm.

In the 2007 base case (i.e., before the application of emission reductions resulting from this rule), we estimated that 116 million, or 93 percent of the total population considered in this analysis, are predicted to live in areas with at least 2 days with model-adjusted 8-hour average concentrations of 0.08 ppm or higher. The number of people involved is predicted to diminish as the lower end of the concentration range increases or as the number of days predicted to experience such peak 8-hour average concentrations increases. The number of people predicted

to live in areas with at least 2 days with model-adjusted 8-hour average concentrations of 0.08 ppm or higher is estimated to increase in the 2020 base case to 122 million people, although this is estimated to represent a smaller percentage (87 percent) of the total projected population considered in the analysis. However, both the number of people (139 million) and the relative percentage (91 percent) of the total population considered in the analysis is projected to grow in the 2030 base case.

3. Particulate Matter

a. Health and Welfare Effects of Ambient Particulate Matter

Particulate matter (PM) represents a broad class of chemically and physically diverse substances that exist as discrete particles (liquid droplets or solids) over a wide range of sizes. Coarse PM are those particles which have a diameter in the range of 2.5 to 10 microns, and fine particles are those particles which have a diameter less than 2.5 microns. Typically, PM is also classified as PM_{10} (all particles less than 10 microns) or $PM_{2.5}$ (all particles less than 2.5 microns). Human-generated sources of particles include a variety of stationary sources (including power generating plants, industrial operations, manufacturing plants, waste disposal) and mobile sources (light- and heavy-duty on-road vehicles, and off-highway vehicles such as construction, farming, industrial, locomotives, marine vessels and other sources). Particles may be emitted directly to the atmosphere (primary particles) or may be formed by transformations of gaseous emissions of sulfur dioxide, nitrogen oxides or volatile organic compounds (secondary particles). Secondary PM is dominated by sulfate in the eastern U.S. and nitrate in the western U.S.[12] Essentially all (>90 percent) of the direct mobile source PM emissions and their secondary formation products are in the fine PM size range. Mobile sources can reasonably be estimated to contribute to ambient secondary nitrate and sulfate PM in proportion to their contribution to total NOx and SOx emissions.

The chemical and physical properties of PM vary greatly with time, region, meteorology, and source category, thus complicating the assessment of health and welfare effects. At elevated concentrations, particulate matter can adversely affect human health, visibility, and materials. Components of particulate matter (e.g., sulfuric or nitric acid) also contribute to acid deposition, nitrification of surface soils and water and eutrophication of surface water as will be discussed below.

Key EPA findings regarding the health risks posed by ambient particulate matter can be found in the Air Quality Criteria for Particulate Matter and are summarized as follows:

a. Health risks posed by inhaled particles are affected both by the penetration and deposition of particles in the various regions of the respiratory tract, and by the biological responses to these deposited materials.

b. The risks of adverse effects associated with deposition of ambient particles in the thorax (tracheobronchial and alveolar regions of the respiratory tract) are markedly greater than for deposition in the extrathoracic (head) region. Maximum particle penetration to the thoracic regions occurs during oronasal or mouth breathing.

c. The key health effects categories associated with PM include premature death; aggravation of respiratory and cardiovascular disease, as indicated by increased hospital admissions and emergency room visits, school absences, work loss days, and restricted activity days; changes in lung function and increased respiratory symptoms; changes to lung tissues and structure; and altered respiratory defense mechanisms. Most of these effects have been consistently associated with ambient PM concentrations, which have been used as a measure of population exposure, in a large number of community epidemiological studies. Additional information and insights on these effects are provided by studies of animal toxicology and controlled human exposures to various constituents of PM conducted at higher than ambient concentrations. Although mechanisms by which particles cause effects are not well known, there is general agreement that the cardio-respiratory system is the major target of PM effects.

d. Based on a qualitative assessment of the epidemiological evidence of effects associated with PM for populations that appear to be at greatest risk with respect to particular health endpoints, we have concluded the following with respect to sensitive populations:

1. Individuals with respiratory disease (e.g., chronic obstructive pulmonary disease, acute bronchitis) and cardiovascular disease (e.g., ischemic heart disease) are at greater risk of premature mortality and hospitalization due to exposure to ambient PM.

2. Individuals with infectious respiratory disease (e.g., pneumonia) are at greater risk of premature mortality and morbidity (e.g., hospitalization, aggravation of respiratory symptoms) due to exposure to ambient PM. Also, exposure to PM may increase individuals' susceptibility to respiratory infections.

3. Elderly individuals are also at greater risk of premature mortality and hospitalization for cardiopulmonary problems due to exposure to ambient PM.

4. Children are at greater risk of increased respiratory symptoms and decreased lung function due to exposure to ambient PM.

5. Asthmatic individuals are at risk of exacerbation of symptoms associated with asthma, and increased need for medical attention, due to exposure to PM.

e. There are fundamental physical and chemical differences between fine and coarse fraction particles. The fine fraction contains acid aerosols, sulfates, nitrates, transition metals, diesel exhaust particles, and ultra fine particles and the coarse fraction typically contains high mineral concentrations, silica and resuspended dust. It is reasonable to expect that differences may exist in both the nature of potential effects elicited by coarse and fine PM and the relative concentrations required to produce such effects. Both fine and coarse particles can accumulate in the respiratory system. Exposure to coarse fraction particles is primarily associated with the aggravation of respiratory conditions such as asthma. Fine particles are most closely associated with health effects such as premature death or hospital admissions, and for cardiopulmonary diseases.

With respect to welfare or secondary effects, fine particles have been clearly associated with the impairment of visibility over urban areas and large multi-State regions. Fine particles, or major constituents thereof, also are implicated in materials damage, soiling and acid deposition. Coarse fraction particles contribute to soiling and materials damage.

Particulate pollution is a problem affecting urban and non-urban localities in all regions of the United States. Manmade emissions that contribute to airborne particulate matter (listed above) result principally from combustion sources (stationary and mobile sources) and fugitive emissions from industrial process and non-industrial processes (such as roadway dust from paved and unpaved roads, wind erosion from cropland, construction, etc.). Natural sources also contribute to particulate matter in the atmosphere and include sources such as wind erosion of geological material, sea spray, volcanic emissions, biogenic emanation (e.g., pollen from plants, fungal spores), and wild fires. Emission inventories for the relative contribution of diesel PM to total ambient PM will be discussed below.

Secondary diesel PM includes particles containing sulfuric acid, nitric acid and organic compounds of diesel exhaust origin. Sulfur dioxide (SO_2) and nitrogen oxides (primarily nitric oxide, or NO), are emitted from diesel engines. Sulfur dioxide is converted to sulfuric acid in the presence of oxidizing reactants and water vapor to form (H_2SO_4) droplets which are less than 1 µm in diameter. Because SO_2 is soluble in water, it is scavenged by fog, cloud water, and raindrops. Sulfur emitted from diesel engines is predominantly (~98 percent) in the form of SO_2, a portion of which will form sulfate aerosols by the reaction described above. Off-road equipment, typically use fuel containing 3300 ppm sulfur, and therefore emit more SO_2 than on-road diesel engines which use fuels currently containing an average of 340 ppm sulfur. We estimate that mobile sources are responsible for about seven percent of nationwide SO_2 emissions with diesel engines contributing 80 percent of the mobile source total (the majority of the diesel SO_2 emissions originate from off-highway engines).[13] The portion of this SO_2 which is subsequently converted to sulfuric acid will vary regionally and, especially in the eastern U.S., the contribution of diesel emissions will be minimal.

Nitric oxide (NO) is also oxidized in the atmosphere to form NO_2 and particulate nitrate (nitric acid and ammonium nitrate primarily). Organic aerosols are also formed from atmospheric transformation of hydrocarbons emitted in the gaseous phase from diesel engines. Little research has been conducted to characterize the contribution of diesel exhaust to secondary organic particulates in the ambient air. Some studies suggest that up to 38 percent of the organic aerosol in an urban environment can be secondary in origin, a portion of which would come from diesel exhaust.[14] In a recent modeling study by Kleeman and Cass, 8.96 $\mu g/m^3$ $PM_{2.5}$ (67 percent of the diesel PM2.5 mass) at Riverside, CA was attributed to secondary formation from direct diesel emissions.[15] A portion of the secondary $PM_{2.5}$ was attributed to primary emissions of hydrocarbons (1 percent). The majority (70 percent) of the secondary diesel $PM_{2.5}$ at Riverside was attributed to nitrate formation.

The sources, ambient concentration, and chemical and physical properties of PM_{10} vary greatly with time, region, meteorology, and source category. A first step in developing a plan to attain the PM_{10} NAAQS is to disaggregate ambient PM_{10} into the basic categories of sulfate, nitrate, carbonaceous, and crustal, and then determine the major contributors to each category based on knowledge of local and upwind emission sources. Following this approach, SIP strategies to reduce ambient PM concentrations have generally focused on controlling fugitive dust from natural soil and soil disturbed by human activity, paving dirt roads and controlling of soil on paved roads, reducing emissions from residential wood combustion, and controlling major stationary sources of PM_{10} where applicable. The control programs to reduce stationary, area, and mobile source SO_2, NOx, and VOCs to achieve attainment with the sulfur dioxide and ozone NAAQS also have contributed to reductions in the fine fraction of PM_{10} concentrations. In addition, the EPA standards for PM emissions from highway and off-highway engines are contributing to reducing PM_{10} concentrations. As result of all these efforts, in the last ten years, there has been a downward trend in PM_{10} concentrations, with a leveling off in the later years.[16]

Heavy-duty vehicles contribute to fine particle formation through a number of pollutants. The chemical composition of PM fine varies by region of the country (see Table II.A-12). Sulfate plays a major role in the composition of fine particulate across the country, but typically makes up over half the fine particles found in the Eastern United States. Organic carbon accounts for a large portion of fine particle mass, with a slightly higher fraction in the west. Diesel engines are the principle source of elemental carbon, which makes up about 5-6 percent of particle mass.

Table II.A-12. Percent Contribution to $PM_{2.5}$ by Component, 1998 [p]

	East	West
Sulfate	56	33
Elemental Carbon	5	6
Organic Carbon	27	36
Nitrate	5	8
Crustal Material	7	17

Nationally, nitrate plays a relatively small roles in the make up of fine particles, but it plays a far larger role in southern California. Ammonium nitrate – formed secondarily from NOx and ammonia emissions -- is one of the most significant components of particulate matter pollution in California. During some of the worst episodes of elevated particle levels in the South Coast, ammonium nitrate can account about 65-75 percent of the PM2.5 mass.[q] Reducing ammonium nitrate through controls on NOx sources is a critical part of California's particulate matter strategy. Nationally, the standards finalized in this rule will significantly reduce HDV emissions of SOx, NO_X, VOCs and elemental carbon, and thus contribute to reductions in ambient concentrations of PM_{10} and PM2.5.

i. Current PM_{10} *Nonattainment*

The most recent PM_{10} monitoring data indicates that 14 designated PM_{10} nonattainment areas with a projected population of 23 million violated the PM_{10} NAAQS in the period 1997-1999. Table II.A-13 lists the 14 areas, and also indicates the PM_{10} nonattainment classification, and 1999 projected population for each PM_{10} nonattainment area. The projected population in

[p] National Air Quality and Emissions Trends Report, 1998, EPA 454/R-00-003, March, 2000.

[q] Southern California 1997 PM_{10} Air Quality Management Plan.

1999 was based on 1990 population figures which were then increased by the amount of population growth in the county from 1990 to 1999.

Table II.A-13. PM_{10} Nonattainment Areas Violating the PM_{10} NAAQS in 1997- 1999

Nonattainment Area or County	1999 Population (projected, in millions)
Anthony, NM (Moderate)[B]	0.003
Clark Co [Las Vegas], NV (Serious)	1.200
Coachella Valley, CA (Serious)	0.239
El Paso Co, TX (Moderate) [A]	0.611
Hayden/Miami, AZ (Moderate)	0.004
Imperial Valley, CA (Moderate)	0.122
Los Angeles South Coast Air Basin, CA (Serious)	14.352
Nogales, AZ (Moderate)	0.025
Owens Valley, CA (Serious)	0.018
Phoenix, AZ (Serious)	2.977
San Joaquin Valley, CA (Serious)	3.214
Searles Valley, CA (Moderate)	0.029
Wallula, WA (Moderate)[B]	0.052
Washoe Co [Reno], NV (Moderate)	0.320
Total Areas: 14	23.167

[A] EPA has determined that continuing PM_{10} nonattainment in El Paso, TX is attributable to such transport under section 179(B).

[B] The violation in this area has been determined to be attributable to natural events under section 188(f) of the Act.

In addition to the 14 PM_{10} nonattainment areas that are currently violating the PM_{10} NAAQS listed in Table II.A-13, there are 25 unclassifiable areas that have recently recorded ambient concentrations of PM_{10} above the PM_{10} NAAQS. EPA adopted a policy in 1996 that allows areas with PM_{10} exceedances that are attributable to natural events to retain their designation as unclassifiable if the State is taking all reasonable measures to safeguard public health regardless of the sources of PM_{10} emissions. Areas that remain unclassifiable areas are not required under the Clean Air Act to submit attainment plans, but we work with each of these areas to understand the nature of the PM_{10} problem and to determine what best can be done to reduce it. With respect to the monitored violations reported in 1997-99 in the 25 areas designated as unclassifiable, we have not yet excluded the possibility that factors such as a one-time monitoring upset or natural events, which ordinarily would not result in an area being designated as nonattainment for PM_{10}, may be responsible for the problem. Emission reductions

from today's action will assist these currently unclassifiable areas to achieve ambient PM_{10} concentrations below the current PM_{10} NAAQS.

ii. Risk of Future Exceedances of the PM_{10} Standard

The new standards for heavy-duty vehicles will benefit public health and welfare through reductions in direct diesel particles and NOx, VOCs, and SOx which contribute to secondary formation of particulate matter. Because ambient particle concentrations causing violations of the PM_{10} standard are well established to endanger public health and welfare, this information supports the new standards for heavy-duty vehicles. The reductions from today's rule will assist states as they work with the Agency through implementation of local controls including development and adoption of additional controls as needed to move their areas into attainment by the applicable deadline, and maintain the standards thereafter.

The Agency's PM inventory analysis performed for this rulemaking predicts that without additional reductions 10 areas face a significant risk of failing to meet or to maintain the PM_{10} NAAQS even with federal, State and local controls currently in place. EPA has evaluated projected emissions for this analysis rather than future air quality because REMSAD, the model EPA has used for analyses related to this rule, was designed principally to estimate long-term average concentrations of fine particulate matter and its ability to predict short-term PM_{10} concentrations has not been satisfactorily demonstrated. In contrast with ozone, which is the product of complex photochemical reactions and therefore difficult to directly relate to precursor emissions, ambient PM_{10} concentrations are more heavily influenced by direct emissions of particulate matter and can therefore be correlated more meaningfully with emissions inventories. In the west, where most of the PM_{10} nonattainment areas are located, coarse PM is comprised of 70 percent particles composed of minerals, with only small fractions attributable to gaseous pollutants such as SOx, NOx and ammonia.[r]

Table II.A-14 presents information about these ten areas and subdivides them into two groups. The first group of six areas are designated PM_{10} nonattainment areas which had recent monitored violations of the PM_{10} NAAQS in 1997-1999 and increasing inventories of PM_{10} from 1996 to 2030. These areas have a population of 19 million. Included in the group are the nonattainment areas that are part of the Los Angeles, Phoenix and Las Vegas (Clark County) metropolitan areas, where traffic from heavy-duty vehicles is substantial. These six areas will benefit from the reductions in emissions that will occur from the new standards for heavy-duty vehicles, as will other areas impacted by heavy-duty vehicle emissions.

[r] *Air Quality Criteria for Particulate Matter*, External Review Draft, EPA 600/P-99/002a, Volume 1, October 1999, at 4.43.

The second group of four counties listed in Table II.A-14 with a total of nine million people in 1999 also had predicted exceedances of the PM_{10} standard. These four areas registered, in either 1997 or 1998, single-year annual average monitored PM_{10} levels of at least 90 percent of the PM_{10} NAAQS, these areas did not exceed the formal definition of the PM_{10} NAAQS over the three-year period ending in 1999. For each of these four areas (ie., Cuyahoga, Harris, New York, and San Diego), inventories of total PM_{10} are predicted to increase between 1996, when these areas recorded values within 10 percent of the PM_{10} standard, and 2030 when this rule will take full effect. For some of these areas, total PM_{10} inventories are predicted to decline or stay relatively constant from 1996 to 2007, and then increase after 2007. Based on these inventory projections, the small margin of attainment which the four areas currently enjoy will likely erode between 1996 and 2030, and for some areas before 2007, if additional actions to reduce the growth of future emissions are not taken. We therefore consider these four areas to each individually have a significant risk of exceeding the PM_{10} standard between 2007 and 2030 without further emission reductions. The emission reductions from the new standards for heavy-duty vehicles will help these areas attain and maintain the PM_{10} NAAQS in conjunction with other processes that are currently moving these areas towards attainment.

Table II.A-14. Areas with Significant Risk of Exceeding the PM_{10} NAAQS without Further Emission Reductions between 2007 and 2030

Area	Percent Increases in PM_{10} Emissions (1996-2030)	1999 Population (projected) (millions)
Areas Currently Exceeding the PM_{10} standard		
Clark Co., NV (Las Vegas)	41%	1.217
El Paso, TX *	14%	0.611
Hayden/Miami, AZ	4%	0.004
Los Angeles South Coast Air Basin, CA	14%	14.352
Nogales, AZ	3%	0.025
Phoenix, AZ	24%	3.012
Subtotal for 6 Areas		*19.22*
Areas within 10% of Exceeding the PM_{10} Standard		
Cuyahoga Co., OH (Cleveland)	28%	1.37
Harris, Co., TX (Houston)	37%	3.26
New York Co., NY	14%	1.55
San Diego Co., CA	13%	2.83
Subtotal for 4 Areas		*9.01*
10 Areas		**28.23 million**

* EPA has determined that PM_{10} nonattainment in this area is attributable to international transport. While reductions in heavy-duty vehicle emissions cannot be expected to result in attainment, they will help reduce the degree of PM_{10} nonattainment.

EPA recognizes that the SIP process is ongoing and that many of the 14 current nonattainment areas in Table II.A-13 are in the process of, or will be adopting and implementing additional control measures to achieve the PM_{10} NAAQS in accordance with their attainment dates under the Clean Air Act. EPA believes, however, that as in the case of ozone, there are uncertainties inherent in any demonstration of attainment that is premised on forecasts of emission levels in future years. Even if these areas adopt and submit SIPs that EPA is able to approve as demonstrating attainment of the PM_{10} standard, and attain the standard by the appropriate attainment dates, the inventory analysis conducted for this rule and the history of PM_{10} levels in these areas indicates that there is still a significant risk that these areas will need the reductions from the heavy-duty vehicle standards adopted today to maintain the PM_{10} standards in the long term (ie, between 2007 and 2030). In addition, this list does not fully consider the possibility that there are other areas which are now meeting the PM_{10} NAAQS that have at least a significant probability of requiring further reductions to continue to maintain it.

iii. Conclusion

In sum, the Agency believes that ten areas listed in Table II.A-14 have a significant risk of experiencing particulate matter levels that violate the PM_{10} standard from 2007 to 2030. In addition, this list does not fully consider the possibility that there are other areas which are now meeting the PM_{10} NAAQS that have at least a significant probability of requiring further reductions to continue to maintain it.

b. Public Health and Welfare Concerns from Exposure to Fine PM

i. Health Effects Studies

There are many studies supporting the Agency's belief that ambient PM causes health and welfare effects even in areas where PM_{10} concentrations are below the level of PM_{10} NAAQS. This science points to fine PM in particular as being more strongly associated with serious health effects, such as premature mortality, than coarse fraction PM. The health and welfare studies support a conclusion that fine PM patterns, that can reasonably be anticipated to occur in the future, are a serious public health and welfare concern warranting a requirement to reduce emissions from heavy-duty vehicles, even where they may not constitute violation of the PM_{10} NAAQS.

The strongest evidence for ambient PM exposure health risks is derived from epidemiologic studies. The following brief summary focuses on studies completed in the last 10 years on the health and welfare effects of PM. A detailed summary and discussion of the large body of PM health effects research may be found in Chapters 10 to 13 of the 1996 Air Quality Criteria for Particulate Matter (known as the Criteria Document or CD).

Many epidemiologic studies have shown statistically significant associations of ambient PM levels with a variety of human health endpoints in sensitive populations, including mortality, hospital admissions and emergency room visits, respiratory illness and symptoms, and physiologic changes in mechanical pulmonary function. The epidemiologic science points to fine PM as being more strongly associated with some health effects, such as premature mortality, than coarse fraction PM, which is associated with other health effects.

Associations of both short-term and long-term PM exposure with most of these endpoints have been consistently observed. Peer-reviewed studies in a variety of locations implicate PM exposure in increased mortality at levels well below the current 24-hour PM_{10} NAAQS of 150 $\mu g/m^3$ and annual PM_{10} NAAQS of 50 $\mu g/m^3$. This section will briefly highlight the short-term exposure studies first and then some of the longer-term exposure studies.

The general internal consistency of the epidemiologic data base and available findings have led to increasing public health concern, due to the severity of several studied endpoints and the frequent demonstration of associations of health and physiologic effects with ambient PM levels at or below the current PM_{10} NAAQS. Time-series analyses strongly suggest a positive effect on daily mortality across the entire range of ambient PM levels. Relative risk (RR) estimates for daily mortality in relation to daily ambient PM concentration are consistently positive, and statistically significant (at $P \leq 0.05$), across a variety of statistical modeling approaches and methods of adjustment for effects of relevant covariates such as season, weather, and co-pollutants. Questions remain about the influence of other factors and other issues, and are described in detail in the Criteria Document. However, even considering the uncertainties, the Agency believes that the weight of epidemiologic evidence suggests that ambient PM exposure has affected the public health of U.S. populations.

Within the body of evidence, there is considerable agreement among different studies that the elderly are particularly susceptible to effects from both short-term and long-term exposures to PM, especially if they have underlying respiratory or cardiac disease. These effects include increases in mortality and increases in hospital admissions. Children, especially those with respiratory diseases, may also be susceptible to pulmonary function decrements associated with exposure to PM or acid aerosols. Respiratory symptoms and reduced activity days have also been associated with PM exposures in children.

Numerous time-series analyses published in the late 1980s and early 1990s demonstrate significant positive associations between daily mortality or morbidity and 24-hour concentrations of ambient particles indexed by various measures (black smoke, TSP, PM_{10}, $PM_{2.5}$, etc.) in numerous U.S. metropolitan areas and in other countries (e.g., Athens, São Paulo, Santiago).[s] These studies collectively suggest that PM alone or in combination with other commonly occurring air pollutants (e.g., SO_2) is associated with daily mortality and morbidity, the effect of PM appearing to be most consistent. In both the historic and recent studies, the association of PM exposure with mortality has been strongest in the elderly and for respiratory and cardiovascular causes of death.

Table II.A-15 summarizes effect estimates (relative risk information) derived from epidemiologic studies demonstrating health effects associations with ambient 24-hour PM_{10} concentrations in U.S. and Canadian cities. The evidence summarized in Table II.A-17 leaves little doubt that PM concentrations typical of contemporary U.S. urban air sheds are correlated with detectable increases in risk of human mortality and morbidity. Evidence from studies that looked at PM indicators other than PM_{10}, summarized in Table II.A-16, also suggests that fine

[s] In the tables summarizing the studies, relative risks with lower confidence intervals greater than 1.0 are statistically significant at the 95 percent confidence level. In Table II A-17, for example, the first entry showing Portage, WI, with a confidence interval of 0.98 - 1.09 is not statistically significant.

particles may be important contributors to the observed PM-health effects associations given the increased risks (of mortality, hospitalization, respiratory symptoms, etc.) associated with several different fine particle indicators (e.g., $PM_{2.5}$, $SO_4^=$, H^+). In particular, more recent reanalyses of the Harvard Six-City Study by Schwartz et al. (1996a) examined the effects on daily mortality of 24-hour concentrations of fine particles ($PM_{2.5}$), inhalable particles ($PM_{15/10}$), or coarse fraction particles ($PM_{15/10}$ minus $PM_{2.5}$) as exposure indices. Overall, these analyses suggest that, in general, the association between excess mortality and thoracic particles appears to be stronger for the fine than the coarse fraction.

In addition to short-term exposure effects, mortality and morbidity effects associated with long-term exposure to PM air pollution have been assessed in cross-sectional studies and more recently, in prospective cohort studies. A number of older cross-sectional studies provided indications of increased mortality associated with chronic exposures to ambient PM (indexed mainly by TSP or sulfate measurements); however, unresolved questions regarding adequacy of statistical adjustments for other potentially important covariates tended to limit the degree of confidence that could be placed on such studies.

Table II.A-17 summarizes some more recent studies using improved methods to examine relationships between chronic PM exposures indexed by different particle size indicators (PM_{15}, $PM_{2.5}$, PM_{15} to $PM_{2.5}$). These studies observed associations between increased risk of mortality/morbidity and chronic (annual average) exposures to PM_{10} or fine particle indicators in contemporary North American urban air sheds.

Since the completion of the 1996 PM Criteria Document (CD), many new epidemiological studies have been published. The PM Criteria Document for the current PM NAAQS review is now being prepared, and in the CD these many new studies will be reviewed, summarized, and integrated with what was learned in previous reviews. EPA will await the completion of the current PM CD before drawing conclusions regarding the findings of this new body of literature regarding the PM NAAQS.

Separate from the NAAQS review, however, new peer-reviewed studies may be considered for use in Regulatory Impact Analyses or other such analyses. EPA believes it appropriate to use the more recent scientific findings for these purposes, especially where the new information adds value to the analyses. Some of these new studies are described below, and the findings of these studies will be incorporated in the larger review of the literature contained in the next PM CD.

Two new Health Effects Institute (HEI) funded studies have received substantial attention from both scientists and the public: a multi-city analysis of mortality and morbidity associations with PM_{10} and other air pollutants (Samet et al., 2000) and the reanalysis of two previous studies of mortality associations with long-term exposure to PM (Krewski et al., 2000).

The multi-city study, National Morbidity, Mortality and Air Pollution Study (NMMAPS), evaluated associations between air pollutants and mortality in 90 U.S. cities, and also evaluated associations between air pollutants and hospital admissions among the elderly in 14 U.S. cities. The authors report: "Together, the 2 sets of analyses - that of mortality in 90 cities and hospitalization in 14 cities - provide new and strong evidence linking particulate air pollution to adverse health effects." (Samet et al., 2000, p. 42)

In the Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality, data were obtained from the original investigators for two previous studies (Dockery et al., 1993; Pope et al., 1995). The extensive analyses included replication and validation of the previous findings, as well as sensitivity analyses using alternative analytic techniques, including different methods of covariate adjustment, exposure characterization, and exposure-response modeling. The authors concluded: "The risk estimates reported by the Original Investigators were remarkably robust to alternative specifications of the underlying risk models, thereby strengthening confidence in the original findings." (Krewski et al., 2000, p. 234)

Some of these new epidemiology studies have presented interesting new findings related to mobile source emissions. For example, Laden et al. (2000) used factor analysis with indicators of particulate matter from several sources, and reported that among these sources, particulate matter from mobile sources had the largest association with mortality in six U.S. cities. Mar et al. (2000) conducted a similar analysis using data from Phoenix, Arizona, and report that mortality from cardiovascular diseases was associated with motor vehicle exhaust-related pollutants. An additional new analysis uses the results of a number of new epidemiological studies to assess the public health impact of outdoor and traffic-related pollution for three European countries. The authors report findings of "considerable" public health impacts for both mortality and morbidity (e.g., bronchitis, exacerbation of existing asthma) effects (Kunzli et al., 2000). These new studies suggest that particles from mobile source emissions play a role in ambient PM-related health effects.

In conclusion, the weight of epidemiologic evidence suggests that PM exposures are correlated with a variety of serious health effects at levels well below the current 24-hour PM_{10} NAAQS of 150 $\mu g/m^3$ and annual PM_{10} NAAQS of 50 $\mu g/m^3$. Similarly, although relatively few cohort studies of long-term PM exposure and mortality are available, they are consistent in direction and magnitude of excess risk with a larger body of cross-sectional annual mortality studies, and most show positive associations of PM exposure with mortality.

Table II.A-15. Effect Estimates Per 50 $\mu g/m^3$ Increase in 24-hour PM_{10} Concentrations From U.S. And Canadian Studies

Study Location	*RR (± CI*) Only PM in Model*	*Reported PM_{10} Levels Mean (Min/Max)*[†]
Increased Total Short-term Exposure Mortality		
Six Cities[A]		
Portage, WI	1.04 (0.98, 1.09)	18 (±11.7)
Boston, MA	1.06 (1.04, 1.09)	24 (±12.8)
Topeka, KS	0.98 (0.90, 1.05)	27 (±16.1)
St. Louis, MO	1.03 (1.00, 1.05)	31 (±16.2)
Kingston/Knoxville, TN	1.05 (1.00, 1.09)	32 (±14.5)
Steubenville, OH	1.05 (1.00, 1.08)	46 (±32.3)
St. Louis, MO[C]	1.08 (1.01, 1.12)	28 (1/97)
Kingston, TN[C]	1.09 (0.94, 1.25)	30 (4/67)
Chicago, IL[H]	1.04 (1.00, 1.08)	37 (4/365)
Chicago, IL[G]	1.03 (1.02, 1.04)	38 (NR/128)
Utah Valley, UT[B]	1.08 (1.05, 1.11)	47 (11/297)
Birmingham, AL[D]	1.05 (1.01, 1.10)	48 (21, 80)
Los Angeles, CA[F]	1.03 (1.00, 1.055)	58(15/177)
Increased Hospital Admissions (for Elderly > 65 yrs.)		
Respiratory Disease		
Toronto, CAN[I]	1.23 (1.02, 1.43) [‡]	30-39 [§]
Tacoma, WA[J]	1.10 (1.03, 1.17)	37 (14, 67)
New Haven, CT[J]	1.06 (1.00, 1.13)	41 (19, 67)
Cleveland, OH[K]	1.06 (1.00, 1.11)	43 (19, 72)
Spokane, WA[L]	1.08 (1.04, 1.14)	46 (16, 83)
Chronic Obstructive Pulmonary Disease		
Minneapolis, MN[N]	1.25 (1.10, 1.44)	36 (18, 58)
Birmingham, AL[M]	1.13 (1.04, 1.22)	45 (19, 77)
Spokane, WA[L]	1.17 (1.08, 1.27)	46 (16, 83)
Detroit, MI[O]	1.10 (1.02, 1.17)	48 (22, 82)

Table II.A-15 continues on next page.

Table II.A-15 (cont'd). Effect Estimates per 50 $\mu g/m^3$ Increase in 24-hour PM_{10} Concentrations from U.S. and Canadian Studies

Study Location	*RR (±CI*) Only PM in Model*	*RR (±CI*) Other Pollutants in Model*	*Reported PM_{10} Levels Mean (Min/Max)†*
Pneumonia			
Minneapolis, MN[N]	1.08 (1.01, 1.15)	—	36 (18,58)
Birmingham, AL[M]	1.09 (1.03, 1.15)	—	45 (19, 77)
Spokane, WA[L]	1.06 (0.98, 1.13)	—	46 (16, 83)
Detroit, MI[O]	—	1.06 (1.02, 1.10)	48 (22, 82)
Ischemic HD			
Detroit, MI[P]	1.02 (1.01, 1.03)	1.02 (1.00, 1.03)	48 (22, 82)
Increased Respiratory Symptoms			
Lower Respiratory			
Six Cities[Q]	2.03 (1.36, 3.04)	Similar RR	30 (13,53)
Utah Valley, UT[R]	1.28 (1.06, 1.56)[τ]	—	46 (11/195)
	1.01 (0.81, 1.27)[π]		
Utah Valley, UT[S]	1.27 (1.08, 1.49)	—	76 (7/251)
Cough			
Denver, CO[X]	1.09 (0.57, 2.10)	—	22 (0.5/73)
Six Cities[Q]	1.51 (1.12, 2.05)	Similar RR	30 (13, 53)
Utah Valley, UT[S]	1.29 (1.12, 1.48)	—	76 (7/251)
Decrease in Lung Function			
Utah Valley, UT[R]	55 (24, 86)[**]	—	46 (11/195)
Utah Valley, UT[S]	30 (10, 50)[**]	—	76 (7/251)
Utah Valley, UT[W]	29 (7,51)[***]	—	55 (1,181)

* CI = Confidence Interval.
† Min/Max 24-h PM_{10} in parentheses unless noted otherwise as standard deviation (± S.D), 10 and 90 percentile (10, 90). NR = not reported.
τ Children.
π Asthmatic children and adults.
§ Means of several cities.
‡ RR refers to total population, not just>65 years.
** PEFR decrease in ml/sec.
*** FEV_1 decrease.

Table II.A-15 References

A Schwartz et al. (1996a).
B Pope et al. (1992, 1994)/O_3.
C Dockery et al. (1992)/O_3.
D Schwartz (1993).
F Kinney et al. (1995)/O_3, CO.
G Ito and Thurston (1996)/O_3.
H Styer et al. (1995).
I Thurston et al. (1994)/O_3.
J Schwartz (1995)/SO_2.
K Schwartz et al. (1996b).
L Schwartz (1996).
M Schwartz (1994e).
N Schwartz (1994f).
O Schwartz (1994d).
P Schwartz and Morris (1995)/O_3, CO, SO_2.
Q Schwartz et al. (1994).
R Pope et al. (1991).
S Pope and Dockery (1992).
T Schwartz (1994g).
W Pope and Kanner (1993).
X Ostro et al. (1991).

Table II.A-16. Effect Estimates per Variable Increments in 24-hour Concentrations of Fine Particle Indicators ($PM_{2.5}$, $SO_4^=$, H^+) From U.S. and Canadian Studies

Short-term Exposure Mortality	*Indicator*	*RR (± CI*) per 25 $\mu g/m^3$ PM Increase*	*Reported PM Levels Mean (Min/Max)†*
Six City[A]			
Portage, WI	$PM_{2.5}$	1.030 (0.993, 1.071)	11.2 (±7.8)
Topeka, KS	$PM_{2.5}$	1.020 (0.951, 1.092)	12.2 (±7.4)
Boston, MA	$PM_{2.5}$	1.056 (1.038, 1.0711)	15.7 (±9.2)
St. Louis, MO	$PM_{2.5}$	1.028 (1.010, 1.043)	18.7 (±10.5)
Kingston/Knoxville, TN	$PM_{2.5}$	1.035 (1.005, 1.066)	20.8 (±9.6)
Steubenville, OH	$PM_{2.5}$	1.025 (0.998, 1.053)	29.6 (±21.9)
Increased Hospitalization			
Ontario, CAN[B]	$SO_4^=$	1.03 (1.02, 1.04)	R = 3.1-8.2
Ontario, CAN[C]	$SO_4^=$	1.03 (1.02, 1.04)	R = 2.0-7.7
	O_3	1.03 (1.02, 1.05)	
NYC/Buffalo, NY[D]	$SO_4^=$	1.05 (1.01, 1.10)	NR
Toronto[D]	H^+ (Nmol/m^3)	1.16 (1.03, 1.30)‡	28.8 (NR/391)
	$SO_4^=$	1.12 (1.00, 1.24)	7.6 (NR, 48.7)
	$PM_{2.5}$	1.15 (1.02, 1.78)	18.6 (NR, 66.0)
Increased Respiratory Symptoms			
Southern California[F]	$SO_4^=$	1.48 (1.14, 1.91)	R = 2-37
Six Cities[G]	$PM_{2.5}$	1.19 (1.01, 1.42)**	18.0 (7.2, 37)***
(Cough)	$PM_{2.5}$ Sulfur	1.23 (0.95, 1.59)**	2.5 (3.1, 61)***
	H^+	1.06 (0.87, 1.29)**	18.1 (0.8, 5.9)***
Six Cities[G]	$PM_{2.5}$	1.44 (1.15-1.82)**	18.0 (7.2, 37)***
(Lower Resp. Symp.)	$PM_{2.5}$ Sulfur	1.82 (1.28-2.59)**	2.5 (0.8, 5.9)***
	H^+	1.05 (0.25-1.30)**	18.1 (3.1, 61)***
Decreased Lung Function			
Uniontown, PA[E]	$PM_{2.5}$	PEFR 23.1 (-0.3, 36.9) (per 25 $\mu g/m^3$)	25/88 (NR/88)

* CI = Confidence Interval.

† Min/Max 24-h PM indicator level shown in parentheses unless otherwise noted as (± S.D.), 10 and 90 percentile (10,90) or R = range of values from min-max, no mean value reported. NR = not reported.

‡ Change per 100 nmoles/m^3.

** Change per 20 $\mu g/m^3$ for $PM_{2.5}$; per 5 $\mu g/m^3$ for $PM_{2.5}$ sulfur; per 25 nmoles/m^3 for H^+.

*** 50th percentile value (10,90 percentile).

Table II.A-16 References

[A] Schwartz et al. (1996a).

[B] Burnett et al. (1994).

[C] Burnett et al. (1995) O_3.

[D] Thurston et al. (1992, 1994)

[E] Neas et al. (1995).

[F] Ostro et al. (1993).

[G] Schwartz et al. (1994).

Table II.A-17. Effect Estimates per Increments* in Annual Mean Levels of Fine Particle Indicators from U.S. and Canadian Studies

Type of Health Effect & Location	*Indicator*	*Change in Health Indicator per Increment in PM**	*Range of City PM Levels Means ($\mu g/m^3$)*
Increased total chronic mortality in adults		Relative Risk (95% CI)	
Six City[B]	$PM_{15/10}$	1.42 (1.16-2.01)	18-47
	$PM_{2.5}$	1.31 (1.11-1.68)	11-30
	$SO_4^=$	1.46 (1.16-2.16)	5-13
ACS Study[C] (151 U.S. SMSA)	$PM_{2.5}$	1.17 (1.09-1.26)	9-34
	$SO_4^=$	1.10 (1.06-1.16)	4-24
Increased bronchitis in children		Odds Ratio (95% CI)	
Six City[D]	$PM_{15/10}$	3.26 (1.13, 10.28)	20-59
Six City[E]	TSP	2.80 (1.17, 7.03)	39-114
24 City[F]	H^+	2.65 (1.22, 5.74)	6.2-41.0
24 City[F]	$SO_4^=$	3.02 (1.28, 7.03)	18.1-67.3
24 City[F]	$PM_{2.1}$	1.97 (0.85, 4.51)	9.1-17.3
24 City[F]	PM_{10}	3.29 (0.81, 13.62)	22.0-28.6
Southern California[G]	$SO_4^=$	1.39 (0.99, 1.92)	—
Decreased lung function in children			
Six City[D]	$PM_{15/10}$	No significant changes	20-59
Six City[E]	TSP	No significant changes	39-114
24 City[I,J]	H^+ (52 nmoles/m^3)	-3.45% (-4.87, -2.01) FVC	—
24 City[I]	$PM_{2.1}$ (15 $\mu g/m^3$)	-3.21% (-4.98, -1.41) FVC	—
24 City[I]	$SO_4^=$ (7 $\mu g/m^3$)	-3.06% (-4.50, -1.60) FVC	—
24 City[I]	PM_{10} (17 $\mu g/m^3$)	-2.42% (-4.30, -.0.51) FVC	—

* Estimates calculated annual-average PM increments assume: a 100 $\mu g/m^3$ increase for TSP; a 50 $\mu g/m^3$ increase for PM_{10} and PM_{15}; a 25 $\mu g/m^3$ increase for $PM_{2.5}$; and a 15 $\mu g/m^3$ increase for $SO_4^=$, except where noted otherwise; a 100 nmole/m^3 increase for H^+.

Table II.A-17 References

[B] Dockery et al. (1993)
[C] Pope et al. (1995)
[D] Dockery et al. (1989)
[E] Ware et al. (1986)
[F] Dockery et al. (1996)
[G] Abbey et al. (1995a,b,c)
[I] Raizenne et al. (1996)
[J] Pollutant data same as for Dockery et al. (1996)

Statistically significant increased mortality from daily exposures to fine PM was observed in cities with longer-term average fine PM concentrations in the range of 16 to 21 ug/m3. It is reasonable to anticipate that populations exposed to similar or higher levels, now and in the 2007 and later time frame, will also experience cases of premature mortality attributable to short term exposures to fine PM. In addition to mortality, statistically significant relationships between daily fine PM levels (or close indicators of fine PM) and increased respiratory symptoms, decreased lung functions, and increased hospitalizations, have also been observed in U.S. cities.

ii. Current and Future Exposures

At the beginning of 1999, State environmental agencies began operating a broad network of monitoring stations for the measurement of fine particulate matter (measured as particulate matter having an aerometric diameter less than or equal to 2.5 micrometers, or PM2.5), using the Federal Reference Method for PM2.5 mass established when the PM2.5 national ambient air quality standard was promulgated (62 FR 38763, July 18, 1997). The data that have been submitted to EPA from this network are available in summary form via the internet on EPA's website (http://www.epa.gov/aqspubl1/annual_summary.html). Copies of raw data may be obtained by contacting the Information Management Group, Information Transfer and Program Integration Division within the Office of Air Quality Planning and Standards. Monitors are generally located within metropolitan statistical areas, although some monitors intended to measure upwind PM2.5 concentrations are located outside of metropolitan areas. Monitors in this network report a 24-hour average PM2.5 concentration for each day of successful monitoring.

At present, virtually all States have completed the quality assurance review and certification process. Data which have been certified as valid are considered to be reliable, although for the purposes of characterizing air quality in areas to which people may be exposed, there must also be a sufficient number of valid samples during the period in question. For the purposes of this analysis, we have only included data certified by the States as valid, and have included only data from sites recording eleven or more valid samples in each calendar quarter. These data are not sufficient for determining whether given areas should be designated under the Clean Air Act as attainment or nonattainment with the PM2.5 NAAQS. Under EPA regulations, this would require consideration of 3 years of valid data. However, these data provide a sufficient basis to estimate the number of people who lived in monitored counties in 1999 in which annual average concentrations of PM2.5 equaled or exceeded certain specified values.

In this analysis, we focus on the long-term average concentrations of $PM_{2.5}$. Accordingly, we analyze the 1999 $PM_{2.5}$ monitoring data, as available, quality assured, and certified by the states, to estimate the long-term average concentration at each monitor for the final rule. These data will not be sufficient for predicting attainment or nonattainment with the $PM_{2.5}$ NAAQS, which requires three years of data. However, for the purpose of this analysis, the currently available monitor data will suffice.

Current 1999 PM2.5 monitored values, which cover about a third of the nation's counties, indicate that at least 40 million people live in areas where long term ambient fine particulate matter levels are at or above 16 µg/m3 (37 percent of the population in the areas with monitors), which is the low end of the range of long term average PM2.5 concentrations in cities where statistically significant associations were found with serious health effects, including premature mortality (EPA, 1996).[t]

Our REMSAD modeled predictions allow us to also estimate the affected population for the counties which do not currently have PM2.5 monitors. According to our national modeled predictions, there were a total of 76 million people (1996 populations) living in areas with modeled annual average PM2.5 concentrations at or above 16 ug/m3 (29 percent of the population).[u]

The REMSAD model also allows us to estimate future PM2.5 levels. However, the most appropriate method of making these projections relies on the model to predict changes between current and future states. Thus, we have estimated future conditions only for the areas with current PM2.5 monitored data (which, as just noted, covers about a third of the nation's counties). For these counties, REMSAD predicts the current level of 37 percent of the population living in areas where fine PM levels are at or above 16 $\mu g/m^3$ to increase to 59 percent in 2030.

It is reasonable to anticipate that sensitive populations exposed to similar or higher levels, now and in the 2007 and later time frame, will also be at increased risk of premature mortality associated with exposures to fine PM. In addition, statistically significant relationships have also been observed in U.S. cities between PM levels and increased respiratory symptoms and decreased lung functions in children.

Since EPA's examination in the mid-1990s of the epidemiological and toxicological evidence of the health effects of PM, many new studies have been published that reevaluate or extend the initial research. The Agency is currently reviewing these new studies to stay abreast of the literature and adjust as necessary its assessment of PM's health effects. It is worth noting that within this new body of scientific literature, there are two new studies funded by the Health Effects Institute, a EPA-industry jointly funded group, that have generally confirmed the mid-1990s findings of the Agency about the association of fine particles and premature mortality and various other respiratory and cardiovascular effects. HEI's *National Morbidity, Mortality and Air Pollution Study* (NMMAPS), evaluated associations between air pollutants and mortality in 90 U.S. cities, and also evaluated associations between air pollutants and hospital admissions

[t] EPA (1996) Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information OAQPS Staff Paper. EPA-452\R-96-013.

[u] REMSAD modeling for PM2.5 annual average concentrations. Total 1996 population in all REMSAD grid cells is 263 million

among the elderly in 14 U.S. cities.[v] In HEI's *Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality*, data were obtained from the original investigators for two previous studies.[w] [x] The extensive analyses included replication and validation of the previous findings, as well as sensitivity analyses using alternative analytic techniques, including different methods of covariate adjustment, exposure characterization, and exposure-response modeling.[y]

In conclusion, we believe that in the period 2007 to 2030, when the standards adopted in today's action will help reduce ambient PM2.5 concentrations, a significant portion of the US population may be exposed to ambient PM2.5 concentrations that studies have found may cause adverse health effects.

4. Diesel Exhaust

The following section presents information about the health hazard and potential risk to public health and welfare posed by exposure to diesel exhaust. The finding of a health hazard addresses the question of whether exposure to an agent is likely to cause an adverse human effect, whereas a discussion of risk is an attempt to provide information on the possible exposure-related impact of the hazard for an exposed population. In this section, we describe in some detail the cancer, chronic noncancer, and acute health effects associated with exposure to diesel exhaust and provide the Agency's current position on the potential for environmental concern. Ambient concentrations and exposure to diesel particulate matter are also described to put the hazard conclusions in perspective.

a. Cancer and Noncancer Effects of Diesel Exhaust

The EPA has concluded that diesel exhaust is likely to be carcinogenic to humans by inhalation at occupational and environmental levels of exposure.[17] Available evidence shows that exposure to diesel exhaust may also cause adverse noncancer health effects with episodic,

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[v] Samet JM, Zeger SL, Dominici F, Curriero F, Coursac I, Dockery DW, Schwartz J, Zanobetti A. 2000. The National Morbidity, Mortality and Air Pollution Study: Part II: Morbidity, Mortality and Air Pollution in the United States. Research Report No. 94, Part II. Health Effects Institute, Cambridge MA, June 2000.

[w] Dockery, D.W., Pope, C.A., III, Xu, X., Spengler, J.D., Ware, J.H., Fay, M.E., Ferris, B.G., Speizer, F.E. (1993) An association between air pollution and mortality in six U.S. cities. N. Engl. J. Med. 329:1753-1759.

[x] Pope, C. A., III, Thun, M. J., Namboodiri, M. M., Dockery, D. W., Evans, J. S., Speizer, F. E., Heath, C. W., Jr. (1995) Particulate air pollution as a predictor of mortality in a prospective study of U.S. adults. Am. J. Respir. Crit. Care Med. 151: 669-674.

[y] Krewski D, Burnett RT, Goldbert MS, Hoover K, Siemiatycki J, Jerrett M, Abrahamowicz M, White WH.(2000) Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge MA, July 2000

acute exposures, as well as noncancer and cancer effects to the respiratory system at longer term, chronic exposures. The draft *Health Assessment Document for Diesel Exhaust* (draft Assessment), was reviewed in public session by the Clean Air Scientific Advisory Committee (CASAC) on October 12-13, 2000.[18] CASAC found that the Agency's conclusion that diesel exhaust is likely to be carcinogenic to humans is scientifically sound. CASAC concurred with the draft Assessment's findings with the proviso that EPA provide modifications and clarifications on certain topics. The Agency expects to produce the finalized Assessment in early 2001. Information presented here is consistent with that to be provided in the final Assessment.

In the draft Assessment, the Agency presents evidence to support its determination that exposure to diesel exhaust is likely to pose a carcinogenic hazard to humans. The most compelling information to suggest a carcinogenic hazard is the consistent association that has been observed between increased lung cancer and diesel exhaust exposure in certain occupationally exposed workers working in the presence of diesel engines. In its review of the published literature, EPA found that about 30 individual epidemiologic studies show increased lung cancer risk associated with diesel emissions. In the draft Assessment EPA evaluated 22 studies that were most relevant for risk assessment, 16 of which reported significant increased lung cancer risks, ranging from 20 to 167 percent, associated with diesel exhaust exposure. These studies are of varying quality in terms of design and controlling for factors that might confound a lung cancer response.

Published analytical results of pooling the positive study results show that on average the lung cancer risks were increased by 33 to 47 percent within a range of 20-89 percent across the studies. Individual epidemiological studies numbering about 30 show increased lung cancer risks of 20 to 89 percent within the study populations depending on the study. The magnitude of the pooled risk increases is not precise owing to uncertainties in the individual studies, the most important of which is a continuing concern about whether smoking effects have been accounted for adequately and in some cases whether other PM exposures were also present. While not all studies have demonstrated an increased risk (six of 34 epidemiological studies summarized by the Health Effects Institute[19] reported relative risks less than 1.0), the fact that an increased risk has been consistently noted in the majority of epidemiological studies strongly supports the determination that exposure to diesel exhaust is likely to pose a carcinogenic hazard to humans.

Additional evidence supporting the identification of a cancer hazard for diesel exhaust includes the observation tumors in animals following applications of various fractions of the diesel exhaust mixture to skin, and implantation of diesel particles in respiratory tissue. Recognizing that diesel exhaust is a complex mixture of carbon particles and associated organics and other inorganics, it is unclear what fraction or combination of fractions is responsible for the carcinogenicity and other respiratory effects. It has been shown, however, that the carbon particles as well as the organics have the potential to be active toxicological agents, either because of the potential to be irritants which cause inflamation, or because of a capacity to produce mutagenic and/or carcinogenic activity. In the case of the organics (which exist both in particle and gaseous states in diesel exhaust) some have potent mutagenic and carcinogenic

properties. In addition, some evidence for the bioavailability of these particle adsorbed compounds has been demonstrated which supports a hypothesis that the adsorbed organics are bioavailable to the lung as well as being transported to sites distant from the lung.

While much of the available evidence for a cancer hazard in humans comes from occupational exposures which generally have higher exposures than in the ambient environment, there is a basis to infer that the lung cancer hazard extends to ambient environmental exposures. The basis for the ambient environmental cancer hazard recommendation is due, in part, to the observation that some ambient environmental concentrations and thus exposures are close to or overlap low-end occupational exposure estimates as discussed below. This potential overlap in exposures suggests that little extrapolation is necessary or, conversely, that there is no margin or only a small margin of safety for some in the general population when compared to occupational exposures where increased cancer risk is observed. Key to the extrapolation is the assumption that across any population showing a risk, that risk would be proportional to total lifetime exposure. The proportional assumption is always made by EPA unless there is evidence to the contrary, and in the case of diesel exhaust, the extrapolation of occupational risk to environmental exposure levels is more confidently judged to be appropriate due to the potential for small exposure differences.

Additional evidence for treating diesel exhaust as a carcinogen at ambient levels of exposure is provided by the observation of the presence of small quantities of many mutagenic and some carcinogenic compounds in the diesel exhaust. A carcinogenic response believed to be caused by such agents is assumed not to have a threshold unless there is direct evidence to the contrary. This is an EPA risk assessment policy choice in the absence of clear contrary evidence. In addition, there is evidence that at least some of the organic compounds associated with diesel particulate matter are extracted by lung fluids (i.e., are bioavailable) and, therefore, are available in some quantity to the lungs as well as entering the bloodstream and being transported to other sites in the body.

In the late 1980s, the International Agency for Research on Cancer (IARC) determined that diesel exhaust is "probably carcinogenic to humans" and the National Institute for Occupational Safety and Health classified diesel exhaust a "potential occupational carcinogen."[20] [21] Based on IARC findings, the State of California identified diesel exhaust in 1990 as a chemical known to the State to cause cancer. In 1996, the International Programme on Chemical Safety of the World Health Organization listed diesel exhaust as a "probable" human carcinogen.[22] In 1998, the California Office of Environmental Health Hazard Assessment (OEHHA, California EPA) identified diesel PM as a toxic air contaminant due to the noncancer and cancer hazard and because of the potential magnitude of the cancer risk.[23] Most recently, the U.S. Department of Health and Human Services National Toxicology Program designated diesel exhaust particles as "reasonably anticipated to be a human carcinogen" in its Ninth Report on Carcinogens.[24] The concern for a carcinogenicity hazard resulting from diesel exhaust exposures is longstanding and widespread.

The adverse noncancer effects of diesel exhaust are also of concern to the Agency. Acute (usually episodic, short duration, high concentration) exposures to diesel exhaust have been associated with a variety of inflammation-related symptoms such as headache, eye discomfort, asthma-like reactions, nausea and exacerbation or initiation of allergenic hypersensitivity. No specific recommendations are made by EPA at this juncture about safe or unsafe exposures to protect from acute effects, since the onset of acute effects is so variable in the population and the available acute health effects data lacks adequate detail regarding dose-response relationships. The potential allergenic effects area of diesel exhaust are of growing interest in the health research community and as additional information emerges, additional review may be warranted.

Chronic (frequent or continuous, long duration, lower concentrations) diesel exhaust exposure, at sufficient inhalation levels, is judged to constitute a chronic noncancer respiratory hazard for humans. For chronic diesel exhaust exposure, EPA is completing the development of an inhalation reference concentration (RfC) for diesel exhaust exposure. The RfC is an estimate of the continuous human inhalation exposure (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious noncancer effects during a lifetime. While the limited amount of human data are suggestive of respiratory distress, animal test data are quite definitive in providing a basis to anticipate a hazard to the human lung based on the irritant and inflammatory reactions in the lung of test animals. Thus, EPA believes that chronic diesel exhaust exposure, at sufficient exposure levels, increases the hazard and risk of an adverse health effect. Based on CASAC advice regarding the use of the animal data to derive the RfC, the Agency will provide an RfC based on diesel exhaust effects in test animals of approximately 5 $\mu g/m^3$.

In addition, it is also instructive to recognize that diesel exhaust particulate matter is part of ambient fine PM. A qualitative comparison of adverse effects of exposure to ambient fine PM and diesel exhaust particulate matter shows that the respiratory system is adversely affected in both cases, though a wider spectrum of adverse effects has been identified for ambient fine PM. Relative to the diesel PM database, there is a wealth of human data for fine PM noncancer effects. Since diesel exhaust PM is a component of ambient fine PM, the fine PM health effects data base can be informative. The final Assessment will discuss the fine PM health effects data and its relation to evaluating health effects associated with diesel exhaust.

b. The Link Between Diesel Exhaust and Diesel Particulate Matter

Diesel exhaust includes components in the gas and particle phases. Gaseous components of diesel exhaust include nitrogen compounds, sulfur compounds, organic compounds, carbon monoxide, carbon dioxide, water vapor, and excess air (nitrogen and oxygen). Among these gas-phase constituents, at least one of the organic compounds is a known human carcinogen (e.g., benzene) while possible or probable human carcinogens are present (e.g., formaldehyde, acetaldehyde, 1,3-butadiene), along with compounds for which the Agency has set inhalation reference concentrations as a guidance to protect the public from noncancer health effects (e.g., acetaldehyde, acrolein, naphthalene).

Diesel particulate matter is either directly emitted from diesel-powered engines (primary particulate matter) or is formed from the gaseous compounds emitted by a diesel engine (secondary particulate matter). After emission from the tail-pipe, diesel exhaust undergoes dilution, reaction and transport in the atmosphere. The primary emission is considered 'fresh', while 'aged' diesel exhaust is considered to have undergone chemical and physical transformation. In an urban or industrial environment, or downwind of an area with large emission sources, diesel exhaust may enter an atmosphere with high concentrations of compounds capable of transforming some diesel particulate matter organic constituents into compounds which exhibit greater toxicity than the primary emitted particle. The formation of nitroarenes is one example of atmospheric transformation of a diesel exhaust organic compound to a more toxicologically significant compound.[25] Some assessments report up to 16 organic compounds in primary and secondary diesel exhaust with known or suspected carcinogenic activity or other toxicologically significant effects.[26]

Primary diesel particles mainly consist of carbonaceous material, with a small contribution from sulfuric acid and ash (trace metals). Many of these particles exist in the atmosphere as a carbon core with a coating of organic carbon compounds, or as sulfuric acid and ash, sulfuric acid aerosols, or sulfate particles associated with organic carbon.[27] While representing a very small portion (less than one percent) of the national emissions of metals, and representing a small portion of diesel particulate matter (one to five percent), we note that several trace metals that may have general toxicological significance depending on the specific species are also emitted by diesel engines in small amounts including chromium, manganese, mercury and nickel. In addition, small amounts of dioxins have been measured in diesel exhaust, some of which may partition into the particle phase.

Approximately 80-95 percent of diesel particle mass is in the size range from 0.05-1.0 micrometers with a mean particle diameter of about 0.2 micrometers. These fine particles have a very large surface area per gram of mass, which make them excellent carriers for adsorbed inorganic and organic compounds that can effectively reach the lowest airways of the lung. Approximately 50-90 percent of the number of particles in diesel exhaust are in the ultrafine size range from 0.005-0.05 micrometers, averaging about 0.02 micrometers. While accounting for the majority of the number of particles, ultrafine diesel particulate matter accounts for 1-20 percent of the mass of diesel particulate matter.

Diesel particulate matter is mainly attributable to the incomplete combustion of fuel hydrocarbons as well as engine oil and other fuel components such as sulfur. Diesel exhaust particles are part of ambient $PM_{2.5}$, since diesel engines are used to power numerous types of equipment in many places. Some geographic areas may have higher diesel particulate loading because of the number of engines that exhaust into the ambient air. While diesel particulate matter contributes to ambient levels of $PM_{2.5}$, the high content of elemental carbon with the adsorbed organic compounds and the high number of ultrafine particles (organic carbon and sulfate) in diesel exhaust distinguish it from other noncombustion sources of $PM_{2.5}$. In addition, diesel particulate matter from mobile source diesel engines is emitted into the breathing zone of

humans and thus has a greater potential for human exposure (per kg of emissions) compared to other combustion particles emitted out of stacks.

While some of the cancer risk may be associated with exposure to the gaseous components of diesel exhaust, studies suggest that the particulate component plays a substantial role in carcinogenicity and noncancer effects. Investigations show that diesel particles (the elemental carbon core plus the adsorbed organics) induce lung cancer at high doses, and that the particles, independent of the gaseous compounds, elicit an animal lung cancer response. The presence of non-diesel elemental carbon particles, as well as the organic-laden diesel particles, correlate with an adverse inflammatory effect in the respiratory system of animals. Additional evidence suggesting the importance of the role of particulate matter in diesel exhaust includes the observation that the extractible particle organics collectively produce cancer and mutagenic toxicity in experimental test systems. Many of the individual organic compounds are mutagenic or carcinogenic in their own right. EPA believes that exposure to whole diesel exhaust is best described, as many researchers have done over the years, by diesel exhaust concentrations expressed in units of mass concentration, i.e., micrograms/m^3. This dosimeter does not directly quantify the gaseous component of diesel exhaust exposure.

Overall, information suggests that the diesel particle may be playing a key role(s) in contributing to the chronic noncancer and carcinogenicity hazards associated with exposure to diesel exhaust: both as a mechanism of delivery for many of the organics and trace metals into the respiratory system, and as a physical irritant in and of itself. Given the available information, it is a reasonable and prudent step to protect public health by proposing regulations on diesel exhaust. Today's action will reduce exposure to both the particulate phase and the gaseous component of diesel exhaust as a result of the particulate matter and NMHC standards adopted. The emission standards and fuel sulfur limit would not directly limit emissions of trace metals, but may indirectly do so by encouraging engine designs with better control of engine oil consumption.[z]

c. Ambient Concentrations and Exposure to Diesel Exhaust

As stated previously, the current Agency position is that diesel exhaust is likely to be carcinogenic to humans and that this cancer hazard exists for occupational as well as ambient levels of exposure. To provide a context in which to assess the potential hazard from ambient levels of diesel exhaust, EPA uses the mass concentration of diesel particulate matter (as do many researchers) as the exposure metric for whole diesel exhaust. A summary of diesel particulate matter concentrations is found in Table II.A-21 and levels of ambient exposure and occupational exposure for some job categories are presented in Table II.A-22.

[z] We are also proposing in today's action to prohibit the introduction of used motor oil into the fuel delivery system which would reduce the trace metal content of the fuel (See Section VIII).

i. Ambient Concentrations

Information about ambient concentrations of diesel particulate matter and the relative contribution of diesel engines to ambient particulate matter levels is available from source-receptor models, dispersion models, and elemental carbon measurements. The most commonly used receptor model for quantifying concentrations of diesel particulate matter at a receptor site is the chemical mass balance model (CMB). Input to the CMB model includes particulate matter measurements made at the receptor site as well as measurements made of each of the source types suspected to impact the site. Because of problems involving the elemental similarity between diesel and gasoline emission profiles and their co-emission in time and space, it is useful to carefully quantify chemical molecular species that provide markers for separation of these sources. Recent advances in chemical analytical techniques have facilitated the development of sophisticated molecular source profiles, including detailed speciation of organic compounds which allow the apportionment of particulate matter to gasoline and diesel sources with increased certainty. Older studies that made use of only elemental source profiles have been published and are summarized here, but are subject to more uncertainty. It should be noted that since receptor modeling is based on the application of source profiles to ambient measurements, the CMB estimates of diesel particulate matter concentrations do not distinguish between on-road and off-highway sources. In addition, this model accounts for primary emissions of diesel particulate matter only; the contribution of secondary aerosols is not included.

Dispersion models estimate ambient levels of particulate matter at a receptor site on the basis of emission factors for the relevant sources and the investigator's ability to model the advection, mixing, deposition, and chemical transformation of compounds from the source to the receptor site. Dispersion models can provide the ability to distinguish on-road from off-highway diesel sources and can be used to estimate the concentrations of secondary aerosols from diesel exhaust. Dispersion modeling is being conducted by EPA to estimate concentrations of, and exposures to several toxic species, including diesel particulate matter.

Elemental carbon (EC) is a major component of diesel particulate matter, contributing approximately 60 to 80 percent of diesel particulate mass, depending on engine technology, fuel type, duty cycle, lube oil consumption, and state of engine maintenance.[28] [29] [30] [31] In most ambient environments, diesel particulate matter is one of the major contributors to EC, with other potential sources including gasoline exhaust; combustion of coal, oil, or wood; charbroiling; cigarette smoke; and road dust. Because of the large portion of EC in diesel particulate matter, and the fact that diesel exhaust is one of the major contributors to EC in most ambient environments, diesel particulate matter concentrations can be bounded using EC measurements. One approach for calculating diesel particulate matter concentrations from EC measurements is presented in the draft *Health Assessment for Diesel Exhaust*.[32] The surrogate diesel particulate matter calculation is a useful approach for estimating diesel particulate matter in the absence of a more sophisticated modeling analysis for locations where EC concentrations are available.

Annual average diesel particulate matter concentrations measured during or after 1988 in urban areas are generally greater than 0.6 micrograms/m^3 and range up to 3.6 micrograms/m^3 in the South Coast Air Basin and 2.4 micrograms/m^3 in Phoenix, AZ (Table II.A-21). Diesel particulate matter concentrations measured on individual days in urban areas are as high as 46.7 micrograms/m^3 in Manhattan, NY, 22 micrograms/m^3 in Phoenix, AZ and 13.3 micrograms/m^3 in Riverside, CA, the latter of which includes both primary and secondary diesel particulate matter. In two dispersion model studies in Southern California, secondary formation of diesel particulate matter accounted for 27 to 67 percent of the total diesel particulate matter concentrations on individual days of 2.6 micrograms/m^3 and 13.3 micrograms/m^3, respectively.[33] [34] Off-highway diesel engines also operate in urban areas, and may have contributed to the ambient diesel particulate matter concentrations reported for CMB studies, depending on the sampling location. Dispersion modeling conducted in Southern California reported that the on-road contribution to the reported diesel particulate matter levels ranged from 63-89 percent of the total diesel particulate matter.[35]

Table II.A-21. Ambient Diesel Particulate Matter Concentrations from Receptor Modeling, Dispersion Modeling and Elemental Carbon Measurements

Location	*Year of Sampling*	*Diesel PM_{10} & $PM_{2.5}$ $\mu g/m^3$ (mean)*	*Diesel PM % of Total PM*	*Type of Data*
West LA, CA	1982, annual	4.4	18	Source-Receptor Model: Based on ambient measurements at receptor sites.
Pasadena, CA	1982, annual	5.3	19	
Rubidoux, CA	1982, annual	5.4	13	
Downtown LA, CA[36]	1982, annual	11.6	36	
Phoenix area, AZ[37]	1989-90, Winter	4-22[A]	9-20	
Phoenix, AZ[38]	1994-95, Nov-	0-5.3 (2.4)	0-27	
California, 15 Air Basins[39]	1988-92, annual	0.2-3.6[A]	[B]	
Manhattan, NY[40]	1993, Spring 3	13.2-46.7[A]	31-68	
Welby, CO	1996-97, Winter 60 days	0-7.3 (1.7)	0-26	
Brighton, CO[41]	1996-97, Winter 60 days	0-3.4 (1.2)	0-38	
Azusa, CA	1982, annual	1.4[D]	5	Dispersion Model: Based on emission rates from the majority of PM sources contributing to the area studied.
Pasadena, CA	1982, annual	2.0[D]	7	
Anaheim, CA	1982, annual	2.7[D]	12	
Long Beach, CA	1982, annual	3.5[D]	13	
Downtown LA, CA	1982, annual	3.5[D]	11	
Lennox, CA	1982, annual	3.8[D]	13	
West LA, CA[42]	1982, annual	3.8[D]	16	
Claremont, CA[43]	18-19 Aug 1987	2.4 (4.0)[C D]	8	
Long Beach, CA	24 Sept 1996	1.9(2.6)[C]	8	
Fullerton, CA	24 Sept 1996	2.4(3.9)[C]	9	
Riverside, CA[44]	25 Sept 1996	4.4(13.3)[C]	12	
Boston, MA	1995, annual	0.8-1.7 (1.1)	6-12	Diesel PM based on EC measurements.
Rochester, NY[45]	1995, annual	0.4-0.8 (0.5)	3-6	
Washington, DC[46]	1992-1995,	1.0-2.2 (1.5)	5-12	
South Coast Air Basin[47]	1995-1996,	2.4-4.5[E]	[B]	

[A] PM_{10}. The reader should note that 80-95 percent of diesel PM is $PM_{2.5}$.

[B] Not Available.

[C] Value in parenthesis includes secondary diesel PM (nitrate, ammonium, sulfate and hydrocarbons) due to atmospheric reactions of primary diesel emissions of NOx, SO_2 and hydrocarbons.

[D] On-road diesel vehicles only; All other values are for on-road plus off-highway diesel emissions.

[E] The Multiple Air Toxics Exposure Study in the South Coast Air Basin reported average annual values for 8 sites in the South Coast Basin.

In addition to these studies, investigations of the concentrations of diesel particulate matter in some microenvironments and "hotspot" areas have been conducted. One such study in Manhattan, NY collected ambient particulate matter near a bus stop on Madison Avenue during a three day period in 1993.[48] Source apportionment applied to these samples indicated that diesel particulate matter concentrations ranged from 13.2 to 46.7 micrograms/m^3 and this study attributed, on average, 53 percent of the total PM_{10} to diesel exhaust. Interpretation of the results of this study require some caution due to the methods used to apportion sources. Concentrations of diesel particulate matter in the vicinity of bus stops may be indicative of concentrations also experienced by urban dwellers who live and/or work in the vicinity of large on-road diesel emission sources and these concentrations may contribute significantly to exposures among some urban dwellers. Kinney et al. (2000) reported elemental carbon concentrations from personal monitors worn by study participants who were located on sidewalks at four intersections in Harlem, NY. The elemental carbon concentrations ranged from 1.5 micrograms/m^3 to 6 micrograms/m^3 and were reported to be associated with diesel bus and truck counts.

In an additional study to assess diesel particulate matter concentrations near heavily traveled roadways, the California Air Resources Board (ARB) collected data on ambient elemental carbon concentrations near the Long Beach Freeway for 3 days in December 1993.[49] Using emission estimates from their mobile source emissions model, and elemental/organic carbon composition profiles for diesel and gasoline exhaust, tire wear, and road dust, ARB estimated that the contribution of freeway diesel traffic resulted in diesel particulate matter concentrations ranging from 0.7 micrograms/m^3 to 4.0 micrograms/m^3 above background concentrations.

A study designed to investigate relationships between diesel exhaust exposure and respiratory health of children in the Netherlands found that schools within 400 meters of a freeway had average elemental carbon concentrations of 3.4 micrograms/m^3, while schools more than 400 meters from freeways had average elemental carbon concentrations of 1.4 micrograms/m^3.[50]

Recently the South Coast Air Quality Management District completed their Multiple Air Toxics Exposure Study in the South Coast Air Basin (MATES-II) to investigate spatial differences in risk from air toxics exposures in the Basin.[51] For this study, elemental carbon concentrations were measured as a surrogate for diesel particulate matter every sixth day for a one year period from April 1998 through March 1999 at eight locations throughout the South Coast Basin. Annual average elemental carbon concentrations ranged from 2.4 micrograms/m^3 to 4.5 micrograms/m^3 across the eight-site network. Monthly mean elemental carbon values peaked during winter months with maximum monthly elemental carbon reaching 13.4 micrograms/m^3.

In a separate study, the California ARB measured elemental carbon concentrations in vehicles on Los Angeles roadways as a surrogate for diesel particulate matter. In-vehicle concentrations of diesel particulate matter are an important microenvironmental exposure for

many people.[52] Diesel particulate matter concentrations in the vehicle were estimated to range from approximately 2.8 micrograms/m^3 to 36.6 micrograms/m^3 with the higher concentrations measured when the vehicle followed a HDDV.

ii. Occupational and Population Exposures

A distinction must be made between ambient concentrations and the concentration of diesel particulate matter to which people are exposed. Ambient concentrations reflect outdoor levels of diesel particulate while exposure depends on both the concentrations of diesel particulate matter and the time spent in various microenvironments where people are exposed. Since people typically spend a large portion of their day indoors and indoor diesel particulate matter concentrations are lower than outdoor concentrations (in the absence of an indoor diesel PM source), then the concentrations to which most people are exposed are expected to be lower than ambient diesel particulate matter concentrations. Exposure to diesel exhaust is most commonly measured in terms of diesel particulate matter and is reported as such in the following section. This information is summarized in the draft *Health Assessment for Diesel Exhaust* and briefly summarized here.

Exposure to diesel exhaust has been measured for several occupationally exposed groups including miners, railroad workers, diesel forklift operators, firefighters, truck drivers, dockworkers and mechanics. Diesel exhaust occupational exposures (typically measured as respirable dust) reported for workers in non-coal mines using diesel-powered shuttle cars range from approximately 38 to 1,280 micrograms/m^3.[53] [54] Diesel exhaust exposures measured among railroad workers (as smoking-adjusted respirable particulate) ranged from 39 micrograms/m^3 for engineers/firers, to 134 micrograms/m^3 for locomotive shop workers and 191 micrograms/m^3 for hostlers.[55] Diesel exhaust exposure among firefighters operating diesel engine vehicles ranges from 4-748 micrograms/m^3 which also encompasses the range of diesel exhaust exposures reported for diesel forklift dockworkers (18.6-64.7 micrograms/m^3).[56] [57] [58] [59] Diesel exhaust exposures measured for truck drivers, mechanics and dockworkers using elemental carbon as a surrogate for diesel particulate matter ranged from 2.0-7.0 micrograms/m^3 for road and local truckers and from 4.8 to 28.0 micrograms/m^3 for dockworkers and mechanics.[60]

For several occupational categories, the occupational exposure and/or environmental equivalent of the occupational exposure overlap with some current ambient concentrations and also overlap with exposure estimates provided by the Hazardous Air Pollutant Exposure Model described below (Table II.A-22). The relevance of the comparison between estimated occupational exposures and ambient exposures to diesel exhaust is discussed in section d. Potential for Cancer Risk, below.

Table II.A-22. Occupational and Population Exposure to Diesel Exhaust

Year of Sampling	*Locations*	***Diesel PM, ug/m³***
Occupational Exposure for a Minimum 8-Hour Workday		
1980's	Non-coal Miners [A]	38 - 1,280
1980's	Railroad Workers [B]	39 - 191
1980's	Diesel Forklift Dockworkers [C]	9 - 61
1980 and 1990's	Firefighters/Fire Station Employees [D]	4-748
1990's	Public Transit Workers, Airport Ground Crew [E]	7-98
1990	Long- and Short-Haul Truckers, Dockworkers, Mechanics [F]	2 - 28
Ambient Exposure Estimates (On-Road) [G]		
1990	National Annual Average	0.84
1990	Urban Annual Average	0.92
1990	Urban Annual Average Outdoor Workers	1.1
1990	Range of Annual Average for Most Highly Exposed by City	0.83 - 4.0
California Exposure Estimates (On-Road & Nonroad) [H]		
1990	California Annual Average	2.1
1995	Projected California Annual Average	1.5
2000	Projected California Annual Average	1.3
2010	Projected California Annual Average	1.2

[A] Watts (1995) and Säverin et al., (1999)
[B] Woskie et al. (1988)
[C] NIOSH (1990); Zaebst et al. (1991)
[D] Friones et al. (1991); NIOSH (1992); Birch and Carey (1996)
[E] Birch and Carey (1996)
[F] Zaebst et al. (1991)
[G] HAPEM-MS3 exposure results for 1990 for on-road sources only. Methodology is described below. These estimates are for the average population and the uncertainty associated with them is large. In particular, in areas where diesel vehicles comprise a higher-than-average portion of the vehicle fleet, exposures will be substantially higher than predicted average exposure estimates.
[H] California EPA (1998).

To estimate population exposures to diesel particulate matter the EPA currently uses the Hazardous Air Pollutant Exposure Model - Mobile Source 3 (HAPEM-MS3).[61] This model provides national and urban-area specific exposures to diesel particulate matter from on-road sources only. Results for 1990 are presented in Table II.A-22. Modeled atmospheric concentrations and exposure estimates of diesel PM from on-highway and nonroad sources have

recently been developed as part of the National Air Toxics Assessment (NATA) National-Scale Analysis. Results from the National-Scale Analysis are currently in draft form and are undergoing technical review by States and EPA's Scientific Advisory Board after which time the data may change. Information on the National-Scale Analysis can be found on the Agency's Urban Air Toxics Website.[62] Table II.A-22 also includes exposure estimates for on-road and nonroad sources modeled by the California EPA's California Population Indoor Exposure Model (CPIEM). Results from this model are presented below and described in more detail in California ARB's "Proposed Identification of Diesel Exhaust as a Toxic Air Contaminant Appendix III Part A: Exposure Assessment".[63]

The HAPEM-MS3 model estimates personal exposures to diesel particulate matter using a ratio to ambient CO measurements. Since most ambient CO comes from motor vehicles, we believe CO exposure is a reasonable surrogate for exposure to other motor vehicle emissions, including emissions of toxic compounds. The HAPEM-MS3 model is based on the carbon monoxide (CO) probabilistic NAAQS exposure model (pNEM/CO), which is used to estimate the frequency distribution of population exposures to CO and the resulting carboxyhemaglobin levels. The pNEM/CO model has undergone evaluation and the results of this evaluation are considered applicable to HAPEM-MS3.[64] The HAPEM-MS3 model simulates the movement of individuals between home and work and through 37 microenvironments. CO concentrations are based on ambient measurements made in 1990 and are related to exposures of individuals in a ten km radius around the sampling site.

Exposure modeling was conducted for 1990. CO concentration data from ten urban areas were used to model 1990 exposures. These areas were Atlanta, GA, Chicago, IL, Denver, CO, Houston, TX, Minneapolis, MN, New York, NY, Philadelphia, PA, Phoenix, AZ, Spokane, WA, and St. Louis, MO. These areas were selected because a large percentage of the population lived within reasonable proximity to CO monitors, and also to represent good geographic coverage of the U.S. The HAPEM model links human activity patterns with ambient CO concentration to arrive at average exposure estimates for 22 different demographic groups (e.g., outdoor workers, children 0 to 17, working men 18 to 44, women 65+) and for the total population. The model simulates the movement of individuals between home and work and through a number of different microenvironments. The CO concentration in each microenvironment is determined by multiplying ambient concentration by a microenvironmental factor derived from regression analysis of ambient and personal monitor data. Each microenvironmental factor has a multiplicative term, which represents ambient exposure, and an additive term, which represents exposure to emissions originating within microenvironments. These factors were derived by IT Corporation using paired ambient and personal exposure monitor measurements from CO studies in Denver and Washington.[65] [66] In our modeling, we set the additive term to zero, to eliminate non-ambient sources of CO, such as gas stoves. The multiplicative term has a component that represents penetration from the ambient air into the microenvironment, and a factor that represents the proximity of the microenvironment to monitors. Thus, even though a compound may have a penetration of close to one, the microenvironmental factor could be significantly less

than one if the microenvironment is typically found a significant distance from where CO monitors are located.

With the 1990 CO exposure estimates generated by the HAPEM-MS3 model for each urban area, EPA determined the fraction of exposure that was a result of on-road motor vehicle emissions. This calculation was accomplished by scaling the exposure estimates (which reflect exposure to total ambient CO) by the fraction of the 1990 CO emissions inventory from on-road motor vehicles, determined from the EPA Emission Trends database.[67] [68] Nationwide urban CO exposure from on-road motor vehicles was estimated by first calculating a population-weighted average CO exposure for the ten modeled areas. This number was adjusted by applying a ratio of population-weighted annual average CO for urban areas in the entire country versus average ambient CO concentration for the modeled areas. To estimate rural exposure, the urban estimate was scaled downward using estimates of urban versus rural exposure from the 1993 *Motor Vehicle-Related Air Toxics Study*.[69]

Motor vehicle diesel particulate matter and CO emission rates reported by EPA[70] are used to calculate mobile source diesel particulate matter exposures. Methods for the development of particulate matter emissions used to calculate population exposures can be found in "Analysis of the Impacts of Control Programs on Motor Vehicle Toxic Emissions and Exposure in Urban Areas and Nationwide: Volumes I and II".[71] Diesel particulate matter (DPM) exposures are calculated as in Equation 1, using a ratiometric approach to CO.

$$DPM_{ug/m^3} = (CO_{ug/m^3} / CO_{g/mi}) \times DPM_{g/mi}$$

Equation 1. Ratiometric Calculation of Diesel Particulate Matter Based on CO Exposures.

To estimate diesel particulate matter emissions, we used EPA's PART5 model. PART5 is similar in structure and function to the MOBILE series of models and calculates exhaust and non-exhaust (e.g., road dust) particulate emissions for each vehicle class included in the MOBILE models. PART5 is currently being modified to account for deterioration, in-use emissions, poor maintenance and tampering effects, all of which would increase emission factors. As a result, we believe that HAPEM-MS3 exposure estimates, based on PART5 emission factors, may underestimate true exposures. A comparison of PART5 HDDV emission factors with a comprehensive review of HDDV emission factors reported from in-use chassis dynamometer testing[72] and modeling performed by CARB suggests that PART5 may underestimate HDDV emissions by up to 50 percent. Diesel PM exposures reported here were adjusted to account for new data demonstrating higher HDDV VMT compared with the HDDV VMT presented in the "Analysis of the Impacts of Control Programs on Motor Vehicle Toxic Emissions and Exposure in Urban Areas and Nationwide: Volumes I and II". A complete description of the HAPEM-MS3 model can be found in "Final Technical Report on the Analysis of Carbon Monoxide Exposure for Fourteen Cities Using HAPEM-MS3".[73]

Our methodology for modeling exposure to diesel particulate matter using HAPEM-MS3 has certain limitations and uncertainties. Our use of HAPEM-MS3 to estimate population exposures to air toxics was peer reviewed for the 1993 Motor Vehicle Related Air Toxics Study[74] and more recently for the EPA (1999) report summarized here.[75] [76] [77] Important aspects of our modeling approach are addressed in these comments and are summarized briefly here.

A validation study conducted for the pNEM/CO model on which HAPEM-MS3 is based, indicates that CO exposures for the population in the 5th percentile were overestimated by approximately 33 percent, while those with exposures in the 98th percentile were underestimated by about 30 percent. Based on this finding, we expect that HAPEM-MS3 also underestimates exposures in the highly exposed populations. To assess exposures for highly exposed populations, we have used 1990 CO concentrations relevant to the most highly exposed populations to estimate 1990 diesel particulate matter exposures for different demographic groups in this population.[78]

Two aspects of the HAPEM-MS3 model which result in some uncertainty in diesel particulate matter exposure estimates are: 1) HAPEM-MS3 assumes that the highway fleet (gasoline plus diesel) emissions ratio of CO to diesel particulate matter can be used as an adjustment factor to convert estimated CO personal exposure to diesel particulate matter exposure estimates; and 2) the model does not account for physical and chemical differences between diesel particulate matter and CO. Even though gasoline vehicles emit the large majority of CO, gasoline and diesel highway vehicles travel on the same roadways and we are making the assumption that diesel vehicles will comprise a constant fraction of on-road traffic. Diesel particulate matter and CO are both relatively long-lived atmospheric species (1-3 days) except under certain conditions such as precipitation which will more readily remove particulate matter. Our exposure modeling assumes that for the average person in a modeled air district, CO and diesel particulate matter are well mixed. We are not attempting to assess exposure in microscale environments in which these assumptions may not be valid. While our assumptions have inherent uncertainties, we find that exposure estimates provided by the HAPEM-MS3 model are lower than the majority of ambient diesel particulate matter concentrations. This comparison provides some indication that HAPEM-MS3 exposure estimates are in the range of reasonable exposure estimates for the average population. It is noteworthy that these exposure estimates underestimate exposures for the more highly exposed populations in part due to the underestimate of CO exposures in the 98th percentile (discussed above), underestimates of emission factors by PART5, and the inability to assess small spatial and temporal scale environments.

While EPA continues efforts toward improving exposure estimates, the results of current HAPEM-MS3 exposure modeling are used here to compare exposure ranges to ambient concentration data for the purposes of characterizing potential environmental risk.

Diesel particulate matter exposure was assessed by on-road vehicle class and found to be due almost entirely to emissions from HDDVs. Nationally in 1996, 99 percent of diesel

particulate matter exposure from on-road vehicles is attributable to HDDVs and the rest is generated mainly by LDDTs. We estimate that in 1990, exposure to diesel particulate matter ranged from 0.84 micrograms/m^3 for the general population to 1.1 micrograms/m^3 for outdoor workers (Table II.A-22). Since HDDV traffic, and therefore exposure to diesel particulate matter, varies for different urban areas, we used HAPEM-MS3 to estimate annual average population exposures for ten urban areas.[79] Modeled 1990 diesel particulate matter exposures in Minneapolis, MN (1.0 micrograms/m^3), New York, NY (1.6 micrograms/m^3), Phoenix, AZ (1.3 micrograms/m^3), and Spokane, WA (1.2 micrograms/m^3) were all higher than the 1990 urban exposure average of 0.92 micrograms/m^3 for 1990.[aa]

Since HAPEM-MS3 is suspected to underestimate exposures in the highly exposed populations, we have used 1990 CO concentrations relevant to the most highly exposed populations to estimate 1990 diesel particulate matter exposures for different demographic groups in this population.[80] The highest estimated diesel particulate matter exposures ranged up to 4.0 micrograms/m^3 for outdoor children in New York. The highest exposed demographic groups were those who spend a large portion of their time outdoors. It is important to note that these exposure estimates are lower than the total exposure to diesel particulate matter since they reflect only diesel particulate matter from on-road sources.

Annual average exposure to on-road HDDV particulate matter was modeled for 1990 and 1996. We expect annual average nationwide exposures to change proportionally with the change in the PM emissions inventory. These estimates are for the average population and the uncertainty associated with them is significant. In particular, in areas where diesel vehicles comprise a higher-than-average portion of the vehicle fleet, exposures may be substantially higher than predicted average exposure estimates.

The exposure estimates using HAPEM-MS3 are substantially lower than those reported by California EPA which range from 1.5 micrograms/m^3 in 1995, to 1.3 micrograms/m^3 in 2000.[81] One significant reason for the difference is that the California estimate is for diesel PM_{10} from all sources, including off-highway, while HAPEM estimates exposures for highway vehicles only. Other reasons may be differences in estimates of emission rates, exposure patterns, the concentration of diesel vehicle traffic, or the spatial distribution of diesel engine emissions.

HAPEM-MS3 exposure estimates for the general population are also lower than annual average diesel particulate matter concentrations reported from most receptor and dispersion models. We have modeled exposure for two urban areas for which there is an estimate of ambient diesel particulate concentrations (Phoenix, AZ and Denver, CO). In these locations, the annual average exposure estimates are up to a factor of two lower than ambient concentrations.

[aa] Memorandum to air docket, May 1, 2000, Determination of demographic groups with the highest annual averaged modeled diesel PM exposure. Pamela Brodowicz, Office of Transportation and Air Quality.

For example, the modeled annual average exposure for the general population in Phoenix in 1996 is 1.3 $\mu g/m^3$ and recent sampling conducted in 1994-1995 in Phoenix indicates that concentrations of diesel particulate matter are 2.4 micrograms/m^3. In Denver, CO the 1996 exposure estimate for the general population is 0.8 micrograms/m^3 and the winter sampling conducted during the Northern Front Range Air Quality Study indicates that in Welby and Brighton, CO, average ambient concentrations of diesel particulate matter are 1.7 micrograms/m^3 and 1.2 micrograms/m^3, respectively. This difference in exposure estimates and ambient concentrations is expected since a large portion of time is spent indoors by most people (where diesel PM concentrations are lower than outdoors) and the HAPEM-MS3 exposure estimates do not include the influence of off-highway sources of diesel particulate matter. Our emissions inventory suggests that mobile sources account for approximately 98 percent of all diesel particulate matter emissions and that on-road HDDVs emit approximately one-third of the diesel particulate matter with the rest attributable to off-highway equipment.[82] Reductions in on-road diesel particulate matter emissions resulting from today's action will have a substantial impact on population exposure to diesel particulate matter.

The discrepancy between exposure and ambient concentrations is small for those who spend a large portion of their day out-of-doors or for those whose microenvironmental exposures permit greater intrusion of outdoor air (such as those whose occupations require that they spend substantial time in motor vehicles). For these more highly exposed demographic groups HAPEM-MS3 still underestimates exposure. Given the ambient concentration data available from some hotspot studies, exposure to diesel particulate matter for the highly exposed subset could be quite large and is likely to overlap some occupational exposures to a large degree.

d. Potential for Cancer Risk

The EPA has concluded that diesel exhaust is likely to be carcinogenic to humans by inhalation at occupational and ambient levels of exposure. While the available evidence leads to EPA's conclusion that diesel exhaust is a likely human lung carcinogen, the evidence is insufficient to develop a confident estimate of cancer unit risk. The absence of quantitative estimates of the lung cancer unit risk for diesel exhaust limits our ability to characterize the precise magnitude of the cancer impact. Given the absence of a unit risk estimate, we provide a perspective on the possible risks to gain a better understanding of the potential significance of the cancer hazard for the general population.

With respect to the estimation of a unit risk for diesel exhaust, risk assessments using epidemiological studies in the peer-reviewed literature which have attempted to assess the lifetime risk of lung cancer in workers occupationally exposed to diesel exhaust suggest that lung cancer risk may range from 10^{-4} to 10^{-2}. [83] [84] [85] The Agency recognizes the significant uncertainties in these studies, and has not used these estimates to assess the possible cancer unit risk associated with ambient exposure to diesel exhaust.

In the draft Assessment, EPA acknowledged the limitations in confidently characterizing a unit risk and provided a discussion of the possible cancer risk consistent with occupational epidemiological findings of increased risk and relative exposure ranges in the occupational and environmental settings. Such an approach does not produce estimates of cancer unit risk. Rather, this approach provides a perspective on the possible magnitude of environmental cancer risk and thus insight about the possible significance of the hazard. We describe here two approaches to gauge the magnitude of potential cancer risk from ambient exposure to diesel exhaust. A more complete description of the approaches and the methods used can be found in the draft Assessment.

One approach to provide a perspective on the possible magnitude of the environmental cancer risk involves examining the differences between the levels of occupational and ambient exposures, and assuming that cancer risk posed by exposure to diesel exhaust is linearly proportional with cumulative lifetime exposure. Risks to the general public are considered to be of concern if the differences between occupational and ambient exposure are small (i.e., within one to two orders of magnitude), as they would approach workers' risk as observed in epidemiologic studies of past occupational exposures.

To compare differences between occupational and ambient exposures, it is necessary to convert occupational exposure estimates to continuous exposure (e.g., an environmental equivalent exposure). The relationship between occupational exposure and environmental equivalent exposure is calculated based on a typical set of assumptions to account for the difference between the amount of air breathed by a worker during their working lifetime compared to an individual in the general population during their 70-year lifetime (environmental equivalent exposure = 0.21 × occupational exposure).[bb] The environmental equivalent exposures for the occupational exposures presented in Table II.A-23 range from 0.4 to 269 micrograms/m^3.

The environmental equivalent exposure is then compared to ambient diesel exhaust exposure by calculating an exposure margin (EM) which is the ratio of the environmental equivalent exposure to ambient exposure. Table II.A-23 presents the ratios of environmental equivalent exposure to ambient exposures. An EM of one or less indicates that ambient exposure is comparable to occupational exposure (expressed as the environmental equivalent exposure). An EM greater than one means that the occupational exposure is greater than the ambient exposure. Table II.A-23 shows that the EMs based on the average nationwide ambient exposure (0.84 µg/m^3) may be less than one for low-end occupational exposure and start to approach three orders of magnitude for high end occupational exposure. The EMs based on a high-end ambient exposure (i.e., 4.0 µg/m^3) range from less than one to less than two orders of magnitude. This exposure analysis only addresses on-road sources for DE exposure. With additional diesel

[bb] The fraction of a worker exposure relevant to a 70-year lifetime exposure is typically calculated by multiplying the fraction of air inhaled during a typical work shift by the fraction of a week, year and life during which a worker is exposed: (10m^3/shift / 20m^3/day) * (5 days / 7days) * (48 weeks / 52 weeks) * (45 years / 70 years) = 0.21.

exhaust exposures from nonroad sources, there is a potential small margin of exposure and hence a greater concern for diesel exhaust-induced cancer risk.

Table II.A-23. Occupational and Population Exposure to Diesel Exhaust, Environmental Equivalent Exposures and Exposure Margins

Occupational Group	Estimated Occupational Exposure, $\mu g/m^3$	Environmental Equivalent Exposure, $\mu g/m^3$	Exposure margin ratio using 0.84 $\mu g/m^3$ ambient exposure	Exposure margin ratio using 4.0 $\mu g/m^3$ ambient exposure
Non-coal Miners[A]	38-1,280	8-269	10-320	2-67
U.S. Railroad Workers[B]	39-191	8-40	10-48	2-10
Firefighters[C]	4-748	0.8-157	1-187	0.2-39
Public Transit Workers, Dockworkers[D]	2-98	0.4-21	0.5-25	0.1-5

[A] Watts (1995) and Säverin et al., (1999).
[B] Woskie et al. (1988).
[C] Friones et al. (1991); NIOSH (1992); Birch and Carey (1996).
[D] Birch and Carey (1996); Zaebst et al. (1991); NIOSH (1990).

The potential overlap and small margins between occupational and ambient diesel exhaust exposures demonstrated in this analysis, is a significant public health concern for an environmental pollutant that is viewed as a likely human carcinogen. Several factors including the carcinogenicity of diesel, differences in human susceptibility, and our current lack of information regarding exposure to diesel exhaust from non-road sources all affirm the Agency's concern regarding the small difference between ambient concentrations and exposures and occupational exposure levels where the presence of diesel exhaust correlates with an increased risk of lung cancer.

To further characterize the significance of the potential environmental cancer hazard, the Agency is using a three step process based on general epidemiological principles to evaluate the available information. First, the risk of excess lung cancer attributed to occupational exposure to diesel exhaust is estimated. Second, the exposure margin between occupational and ambient exposures is considered. Finally, a perspective on the diesel exhaust hazard significance is derived by proportioning the excess risk from step one by the diesel exhaust exposure margins provided from step two. This approach is expanded upon below and is explained in more detail in the draft Assessment.[86]

In its review of the published literature, EPA found that about 30 individual epidemiological studies show increased lung cancer risk associated with diesel emissions. In the draft Assessment EPA evaluated 22 studies that were the most relevant for risk assessment, 16 of which reported significant increased lung cancer risks, ranging from 20 to 167 percent, associated with diesel exhaust exposure. Questions remain about the influence of other factors (e.g., effect of smoking, other particulate sources), the quality of the individual epidemiologic studies, exposure levels, and consequently the precise magnitude of the increased risk of lung cancer. Two published analytic studies pooled many of the 30 individual epidemiological studies and after adjusting for smoking reported a relative risk increase of 1.35 and 1.47. [87] [88] For the purpose of this analysis, we have used these pooled studies to select a relative risk of 1.4 as a reasonable estimate of the increased lung cancer attributed to exposure to diesel exhaust in occupational settings.

The relative risk of 1.4 means that the occupationally exposed workers experienced an extra risk that is 40 percent higher than the 5 percent background lifetime lung cancer risk in the U.S. population.[cc] Thus, using the relationship *[excess risk = (relative risk-1) × background risk]*, the diesel exhaust-exposed workers would have an excess risk of developing lung cancer of 2 percent (10^{-2}) due to occupational exposure to diesel exhaust [(1.4 -1) × 0.05)= 0.02]. In this analysis, we refer to this value as the occupational population risk.[dd] This is not a unit risk value.

Since the risk is assumed to be proportional to cumulative lifetime exposure, lower exposures among the general population compared to the occupational population, decrease the occupational population risk proportionally. As discussed above, occupational and ambient exposure estimates indicate that the exposure margins (i.e., the EM ratio) between occupational and ambient exposures may range from 0.5-320 when comparing occupational environmental equivalent exposure to the nationwide average ambient exposure of 0.84 $\mu g/m^3$. If lifetime risks decrease proportionately with reduced exposure, and if one assumes that past occupational exposures were at the high end, then the risk from average ambient exposure could be between 10^{-5} and 10^{-4} ($0.02 \div 320 = 6 \times 10^{-5}$). If occupational exposures were closer to 50 $\mu g/m^3$, a value

[cc] The background rate of 0.05 is an approximated lifetime risk calculated by the method of lifetable analysis using age-specific lung cancer mortality data and probability of death in the age group taken from the National Health Statistics (HRS) monographs of Vital Statistics of the U.S. (Vol. 2, Part A, 1992). Similar values based on two rather crude approaches can also be obtained: (1) $59.8 \times 10^{-5} / 8.8 \times 10^{-3} = 6.8 \times 10^{-2}$ where 59.8×10^{-5} and 8.8×10^{-3} are respectively the crude estimates of lung cancer deaths (including intrathoracic organs, estimated to be less than 105 of the total cases) and total deaths for 1996 reported in Statistical Abstract of the U.S. (Bureau of the Census, 1998, 118th Edition), and (2) 156,900/270,000,000 × 76 = 0.045, where 156,900 is the projected lung cancer deaths for the year 2000 as reported in Cancer Statistics 9J of American Cancer Society, Jan/Feb 2000), 270,000,000 is the current U.S. population, and 76 is the expected lifespan.

[dd] As used in this document, population risk is defined as the risk (i.e. a mathematical probability) that lung cancer might be observed in the population after a lifetime exposure to diesel exhaust. Exposure levels may be occupational lifetime or environmental lifetime exposures. A population risk in the magnitude of 10^{-2} translates as the risk of lung cancer being evidenced in one person in one hundred over a lifetime exposure.

that is represented in several data sets shown in Table II.A-23 (with an equivalent environmental exposure of 11 $\mu g/m^3$ and a corresponding EM of 13), then risks from ambient exposure would approach 10^{-3} ($0.02 \div 13 = 2 \times 10^{-3}$).

This analysis establishes a reasonable basis for concern that the general population faces possible lifetime environmental cancer risk ranging from 10^{-5} to 10^{-3}. Adding to this concern is recognition that segments of the population may be additionally exposed to nonroad sources of diesel exhaust which would increase the risk.

The environmental risk estimates included in the Agency's risk perspective are meant only to gauge the possible magnitude of risk to provide a means to understand the potential significance of the lung cancer hazard. The conversion of relative risk to population risk is not specific to the diesel exhaust data as it would apply to any pollutant exposure for which cancer risk increases are observed and there is a known background rate for the cancer in question. The environmental risk estimates are not to be construed as cancer unit risk estimates and are not suitable for use in analyses which would estimate possible lung cancer cases in exposed populations.

EPA recognizes that, as in all such risk assessments, there are uncertainties in the assessment of an environmental risk range. For diesel exhaust, these uncertainties include limitations in exposure data, uncertainty with respect to the most accurate characterization of the risk increases observed in the occupational epidemiological studies, chemical changes in diesel exhaust over time, and extrapolation of the risk from occupational to ambient exposures. As with any such risk assessment for a carcinogen, despite EPA's thorough examination of the available epidemiologic evidence and exposure information, at this time EPA can not rule out the possibility that the lower end of the risk range includes zero.[ee] However, it is the Agency's best scientific judgement that the assumptions and other elements of this analysis are reasonable and appropriate for identifying the risk potential based on the scientific information currently available.

The Agency believes that the risk estimation techniques that were used in the draft Assessment to gauge the potential for and possible magnitude of risk are reasonable and the CASAC panel has concurred with the Assessment's discussion of the possible environmental

[ee] EPA's scientific judgment (which CASAC has supported) is that diesel exhaust is likely to be carcinogenic to humans. Notably, similar scientific judgements about the carcinogenicity of diesel exhaust have been recently made by the National Toxicology Program of the Department of Health and Human Services, NIOSH, WHO, and OEHA of the State of California. In the risk perspective discussed above, EPA recognizes the possibility that the lower end of the environmental risk range includes zero. The risks could be zero because (1) some individuals within the population may have a high tolerance level to exposure from diesel exhaust and therefore are not susceptible to the cancer risks from environmental exposure and (2) although EPA has not seen evidence of this, there could be a threshold of exposure below which there is no cancer risk.

risk range with an understanding that some clarifications and caveats would be added to the final version of the Assessment.

In the absence of having a unit cancer risk to assess environmental risk, EPA has considered the relevant epidemiological studies and principles for their assessment, the risk from occupational exposure as assessed by others, and relative exposure differences between occupational and ambient levels of diesel exhaust exposure.

While uncertainty exists in estimating the possible magnitude of the environmental risk range, the likely hazard to humans together with the potential for significant environmental risks leads the Agency to believe that diesel exhaust emissions should be reduced in order to protect the public's health. We believe that this is a prudent measure in light of:

- the designation that diesel exhaust is likely to be carcinogenic to humans,
- the exposure of the entire population to various levels of diesel exhaust,
- the consistent observation of significantly increased lung cancer risk in workers exposed to diesel exhaust, and
- the potential overlap and/or relatively small difference between some occupational settings where increased lung cancer risk is reported and ambient exposures.

Today's action will reduce exposure to the toxic gaseous component of diesel exhaust as a result of the NMHC standard and we expect that the particulate matter standard in today's action will result in the implementation of particulate matter control technology (catalyzed particulate traps) that will significantly reduce particulate matter and additionally remove gaseous hydrocarbons.

5. Gaseous Air Toxics

This section summarizes our analysis of the impact of the proposed HDV standards on exposure to gaseous air toxics. Heavy-duty vehicle emissions contain several substances that are known, likely, or possible human or animal carcinogens, or that have serious noncancer health effects. These substances include, but are not limited to, benzene, formaldehyde, acetaldehyde, 1,3-butadiene, acrolein, and dioxin. For the purposes of the exposure estimates presented in this section, we have chosen to focus on those compounds in heavy duty vehicle exhaust that are known, likely, or possible carcinogens and that have significant emissions from heavy-duty vehicles.

a. Health Effects

i. Benzene

Highway mobile sources account for 48 percent of nationwide emissions of benzene and HDVs account for 7 percent of all highway vehicle benzene emissions.[89] Benzene is an aromatic hydrocarbon which is present as a gas in both exhaust and evaporative emissions from motor vehicles. Benzene in the exhaust, expressed as a percentage of total organic gases (TOG), varies depending on control technology (e.g., type of catalyst) and the levels of benzene and other aromatics in the fuel, but is generally about three to five percent. The benzene fraction of evaporative emissions depends on control technology and fuel composition and characteristics (e.g., benzene level and the evaporation rate) and is generally about one percent.[90]

The EPA has recently reconfirmed that benzene is a known human carcinogen by all routes of exposure.[91] Respiration is the major source of human exposure. Long-term respiratory exposure to high levels of ambient benzene concentrations has been shown to cause cancer of the tissues that form white blood cells. Among these are acute nonlymphocytic leukemia,[ff] chronic lymphocytic leukemia and possibly multiple myeloma (primary malignant tumors in the bone marrow), although the evidence for the latter has decreased with more recent studies.[92,93] Leukemias, lymphomas, and other tumor types have been observed in experimental animals exposed to benzene by inhalation or oral administration. Exposure to benzene and/or its metabolites has also been linked with genetic changes in humans and animals[94] and increased proliferation of mouse bone marrow cells.[95] The occurrence of certain chromosomal changes in individuals with known exposure to benzene may serve as a marker for those at risk for contracting leukemia.[96]

The latest assessment by EPA places the excess risk of developing acute nonlymphocytic leukemia at 2.2×10^{-6} to $7.7 \times 10^{-6}/\mu g/m^3$. There is a risk of about two to eight excess acute nonlymphocytic leukemia cases in one million people exposed to $1 \mu g/m^3$ over a lifetime (70 years).[97] This range of unit risk represents the maximum likelihood (MLE) estimate of risk, not an upper confidence limit (UCL).

[ff] Leukemia is a blood disease in which the white blood cells are abnormal in type or number. Leukemia may be divided into nonlymphocytic (granulocytic) leukemias and lymphocytic leukemias. Nonlymphocytic leukemia generally involves the types of white blood cells (leukocytes) that are involved in engulfing, killing, and digesting bacteria and other parasites (phagocytosis) as well as releasing chemicals involved in allergic and immune responses. This type of leukemia may also involve erythroblastic cell types (immature red blood cells). Lymphocytic leukemia involves the lymphocyte type of white bloods cell that are responsible for the immune responses. Both nonlymphocytic and lymphocytic leukemia may, in turn, be separated into acute (rapid and fatal) and chronic (lingering, lasting) forms. For example; in acute myeloid leukemia (AML) there is diminished production of normal red blood cells (erythrocytes), granulocytes, and platelets (control clotting) which leads to death by anemia, infection, or hemorrhage. These events can be rapid. In chronic myeloid leukemia (CML) the leukemic cells retain the ability to differentiate (i.e., be responsive to stimulatory factors) and perform function; later there is a loss of the ability to respond.

A number of adverse noncancer health effects, blood disorders such as preleukemia and aplastic anemia, have also been associated with low-dose, long-term exposure to benzene.[98] People with long-term exposure to benzene may experience harmful effects on the blood-forming tissues, especially the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components, such as red blood cells and blood platelets, leading to anemia (a reduction in the number of red blood cells), leukopenia (a reduction in the number of white blood cells), or thrombocytopenia (a reduction in the number of blood platelets, thus reducing the ability for blood to clot). Chronic inhalation exposure to benzene in humans and animals results in pancytopenia,[gg] a condition characterized by decreased numbers of circulating erythrocytes (red blood cells), leukocytes (white blood cells), and thrombocytes (blood platelets).[99,100] Individuals that develop pancytopenia and have continued exposure to benzene may develop aplastic anemia,[hh] whereas others exhibit both pancytopenia and bone marrow hyperplasia (excessive cell formation), a condition that may indicate a preleukemic state.[101 102] The most sensitive noncancer effect observed in humans is the depression of absolute lymphocyte counts in the circulating blood.[103]

ii. 1,3-Butadiene

Highway mobile sources account for approximately 42 percent of the annual emissions of 1,3-butadiene and HDVs account for approximately 15 percent of the highway vehicle portion.[104] 1,3-Butadiene is formed in vehicle exhaust by the incomplete combustion of fuel. It is not present in vehicle evaporative emissions, because it is not present in any appreciable amount in fuel. 1,3-Butadiene accounts for 0.4 to 1.0 percent of total organic gas exhaust, depending on control technology and fuel composition.[105]

1,3-Butadiene was classified by EPA as a Group B2 (probable human) carcinogen in 1985.[106] This classification was based on evidence from two species of rodents and epidemiologic data. In the EPA1998 draft Health Risk Assessment of 1,3-Butadiene, that was reviewed by the Science Advisory Board (SAB), the EPA proposed that 1,3-butadiene is a known human carcinogen based on human epidemiologic, laboratory animal data, and supporting data

[gg] Pancytopenia is the reduction in the number of all three major types of blood cells (erythrocytes, or red blood cells, thrombocytes, or platelets, and leukocytes, or white blood cells). In adults, all three major types of blood cells are produced in the bone marrow of the vertebra, sternum, ribs, and pelvis. The bone marrow contains immature cells, known as multipotent myeloid stem cells, that later differentiate into the various mature blood cells. Pancytopenia results from a reduction in the ability of the red bone marrow to produce adequate numbers of these mature blood cells.

[hh] Aplastic anemia is a more severe blood disease and occurs when the bone marrow ceases to function, i.e.,these stem cells never reach maturity. The depression in bone marrow function occurs in two stages - hyperplasia, or increased synthesis of blood cell elements, followed by hypoplasia, or decreased synthesis. As the disease progresses, the bone marrow decreases functioning. This myeloplastic dysplasia (formation of abnormal tissue) without acute leukemiais known as preleukemia. The aplastic anemia can progress to AML (acute mylogenous leukemia).

such as the genotoxicity of 1,3-butadiene metabolites. [107] The Environmental Health Committee of EPA's Scientific Advisory Board (SAB), reviewed the draft document in August 1998 and recommended that 1,3-butadiene be classified as a probable human carcinogen, stating that designation of 1,3-butadiene as a known human carcinogen should be based on observational studies in humans, without regard to mechanistic or other information.[108] In applying the 1996 proposed Guidelines for Carcinogen Risk Assessment, the Agency relies on both observational studies in humans as well as experimental evidence demonstrating causality and therefore the designation of 1,3-butadiene as a known human carcinogen remains applicable.[109] The Agency has revised the draft Health Risk Assessment of 1,3-Butadiene based on the SAB and public comments. The draft Health Risk Assessment of 1,3-Butadiene will undergo the Agency consensus review, during which time additional changes may be made prior to its public release and placement on the Integrated Risk Information System (IRIS).

The SAB panel recommended that EPA calculate the lifetime cancer risk estimates based on the human data from Delzell et al. 1995[110] and account for the highest exposure of "360 ppm-year" for 70 years. Based on this calculation[111] the maximum likelihood estimate of lifetime cancer risk from continuous 1,3-butadiene exposure is 2.21×10^{-6}/microgram/m^3. This estimate implies that approximately 2 people in one million exposed to 1 microgram/m^3 1,3-butadiene continuously for their lifetime (70 years) would develop cancer as a result of their exposure.

An adjustment factor of 3 can be applied to this potency estimate to reflect evidence from rodent studies suggesting that extrapolating the excess risk of leukemia in a male-only occupational cohort may underestimate the total cancer risk from 1,3-butadiene exposure in the general population.[112] First, studies in both rats and mice indicate that 1,3-butadiene is a multi-site carcinogen. It is possible that humans exposed to 1,3-butadiene may also be at risk of cancers other than leukemia and that the epidemiologic study had insufficient power to detect excess cancer risks for other tissues or sites in the body. Second, both the rat and mouse studies suggest that females are more sensitive to 1,3-butadiene-induced carcinogenicity than males, and the female mammary gland was the only 1,3-butadiene-related tumor site common to both species. Use of a 3-fold adjustment to the potency estimate of 2.21×10^{-6}/microgram/m^3 derived from the occupational epidemiologic study yields a upper bound cancer potency estimate of 1.4×10^{-5}/microgram/m^3, which roughly corresponds to a combination of the human leukemia and mouse mammary gland tumor risk estimates, at least partially addressing the concerns that the leukemia risk estimated from the occupational data may underestimate total cancer risk to the general population, in particular females.

1,3-Butadiene also causes a variety of noncancer reproductive and developmental effects in mice and rats (no human data) when exposed to long-term, low doses of butadiene.[113] The most sensitive effect was reduced litter size at birth and at weaning. These effects were observed in studies in which male mice exposed to 1,3-butadiene were mated with unexposed females. In humans, such an effect might manifest itself as an increased risk of spontaneous abortions, miscarriages, still births, or very early deaths. Long-term exposures to 1,3-butadiene should be kept below its reference concentration of 4.0 microgram/m^3 to avoid appreciable risks of these

reproductive and developmental effects.[114] EPA has developed a draft chronic, subchronic, and acute RfC values for 1,3-butadiene exposure as part of the draft risk characterization mentioned above. The RfC values will be reported on IRIS.

iii. Formaldehyde

Highway mobile sources contribute approximately 24 percent of the national emissions of formaldehyde, and HDVs account for approximately 36 percent of the highway portion.[115] Formaldehyde is the most prevalent aldehyde in vehicle exhaust. It is formed from incomplete combustion of both gasoline and diesel fuel and accounts for one to four percent of total organic gaseous emissions, depending on control technology and fuel composition. It is not found in evaporative emissions.

Formaldehyde exhibits extremely complex atmospheric behavior.[116] It is formed by the atmospheric oxidation of virtually all organic species, including biogenic (produced by a living organism) hydrocarbons. Mobile sources contribute both primary formaldehyde (emitted directly from motor vehicles) and secondary formaldehyde (formed from photooxidation of other VOCs emitted from vehicles).

EPA has classified formaldehyde as a probable human carcinogen based on limited evidence for carcinogenicity in humans and sufficient evidence of carcinogenicity in animal studies, rats, mice, hamsters, and monkeys.[117] Epidemiological studies in occupationally exposed workers suggest that long-term inhalation of formaldehyde may be associated with tumors of the nasopharyngeal cavity (generally the area at the back of the mouth near the nose), nasal cavity, and sinus. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to formaldehyde causes an increase in the incidence of squamous (epithelial) cell carcinomas (tumors) of the nasal cavity. The distribution of nasal tumors in rats suggests that not only regional exposure but also local tissue susceptibility may be important for the distribution of formaldehyde-induced tumors.[118] Research has demonstrated that formaldehyde produces mutagenic activity in cell cultures.[119]

The MLE estimate of a lifetime extra cancer risk from continuous formaldehyde exposure is about $1.3 \times 10^{-6}/\mu g/m^3$. In other words, it is estimated that approximately 1 person in one million exposed to 1 $\mu g/m^3$ formaldehyde continuously for their lifetime (70 years) would develop cancer as a result of this exposure. The agency is currently conducting a reassessment of risk from inhalation exposure to formaldehyde.

Formaldehyde exposure also causes a range of noncancer health effects. At low concentrations (0.05-2.0 ppm), irritation of the eyes (tearing of the eyes and increased blinking) and mucous membranes is the principal effect observed in humans. At exposure to 1-11 ppm, other human upper respiratory effects associated with acute formaldehyde exposure include a dry or sore throat, and a tingling sensation of the nose. Sensitive individuals may experience these effects at lower concentrations. Forty percent of formaldehyde-producing factory workers

reported nasal symptoms such as rhinitis (inflammation of the nasal membrane), nasal obstruction, and nasal discharge following chronic exposure.[120] In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 5 ppm.[121] Formaldehyde exposure may also cause bronchial asthma-like symptoms in non-asthmatics.[122] [123]

Immune stimulation may occur following formaldehyde exposure, although conclusive evidence is not available. Also, little is known about formaldehyde's effect on the central nervous system. Several animal inhalation studies have been conducted to assess the developmental toxicity of formaldehyde: The only exposure-related effect noted in these studies was decreased maternal body weight gain at the high-exposure level. No adverse effects on reproductive outcome of the fetuses that could be attributed to treatment were noted. An inhalation reference concentration (RfC), below which long-term exposures would not pose appreciable noncancer health risks, is not available for formaldehyde at this time.

iv. Acetaldehyde

Highway mobile sources contribute 29 percent of the national acetaldehyde emissions and HDVs are responsible for approximately 33 percent of the highway emissions.[124] Acetaldehyde is a saturated aldehyde that is found in vehicle exhaust and is formed as a result of incomplete combustion of both gasoline and diesel fuel. It is not a component of evaporative emissions. Acetaldehyde comprises 0.4 to 1.0 percent of total organic gas exhaust, depending on control technology and fuel composition.[125]

The atmospheric chemistry of acetaldehyde is similar in many respects to that of formaldehyde.[126] Like formaldehyde, it is produced and destroyed by atmospheric chemical transformation. Mobile sources contribute to ambient acetaldehyde levels both by their primary emissions and by secondary formation resulting from their VOC emissions. Acetaldehyde emissions are classified as a probable human carcinogen. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to acetaldehyde causes an increase in the incidence of nasal squamous cell carcinomas (epithelial tissue) and adenocarcinomas (glandular tissue).[ii] [jj] The MLE estimate of a lifetime extra cancer risk from continuous acetaldehyde exposure is about $0.78 \times 10^{-6} / \mu g/m^3$. In other words, it is estimated that less than 1 person in one million exposed to 1 $\mu g/m^3$ acetaldehyde continuously for their lifetime (70 years) would develop cancer as a result of their exposure. The agency is currently conducting a reassessment of risk from inhalation exposure to acetaldehyde.

[ii] Environmental Protection Agency, Health assessment document for acetaldehyde, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC, EPA-600/8-86/015A (External Review Draft), 1987.

[jj] Environmental Protection Agency, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, 1992.

Noncancer effects in studies with rats and mice showed acetaldehyde to be moderately toxic by the inhalation, oral, and intravenous routes.[127] [128] [129] The primary acute effect of exposure to acetaldehyde vapors is irritation of the eyes, skin, and respiratory tract. At high concentrations, irritation and pulmonary effects can occur, which could facilitate the uptake of other contaminants. Little research exists that addresses the effects of inhalation of acetaldehyde on reproductive and developmental effects. The *in vitro* and *in vivo* studies provide evidence to suggest that acetaldehyde may be the causative factor in birth defects observed in fetal alcohol syndrome, though evidence is very limited linking these effects to inhalation exposure. Long-term exposures should be kept below the reference concentration of 9 $\mu g/m^3$ to avoid appreciable risk of these noncancer health effects.[130]

v. Acrolein

Highway mobile sources contribute 16 percent of the national acrolein emissions and HDVs are responsible for approximately 39 percent of these highway mobile source emissions. Acrolein is extremely toxic to humans from the inhalation route of exposure, with acute exposure resulting in upper respiratory tract irritation and congestion. The Agency developed a reference concentration for inhalation (RfC) of acrolein of 0.02 micrograms/m^3 1993. Although no information is available on its carcinogenic effects in humans, based on laboratory animal data, EPA considers acrolein a possible human carcinogen.[131]

vi. Dioxins

Recent studies have confirmed that dioxins are formed by and emitted from heavy-duty diesel trucks and are estimated to account for 1.2 percent of total dioxin emissions in 1995. In the environment, the pathway of immediate concern is the food pathway (e.g., human ingestion of certain foods, e.g. meat and dairy products contaminated by dioxin) which may be affected by deposition of dioxin from the atmosphere. EPA classified dioxins as probable human carcinogens in 1985. Recently EPA has proposed, and the Scientific Advisory Board has concurred, to classify one dioxin compound, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin as a human carcinogen and the complex mixtures of dioxin-like compounds as likely to be carcinogenic to humans using the draft 1996 carcinogen risk assessment guidelines.[132] Using the 1986 cancer risk assessment guidelines, the hazard characterization for 2,3,7,8-tetrachlorodibenzo-*p*-dioxin is 'known' human carcinogen and the hazard characterization for complex mixtures of dioxin-like compounds is 'probable' human carcinogens. Acute and chronic noncancer effects have also been reported for dioxin.

b. Assessment of Exposure

This subsection describes the analysis conducted by the Agency to evaluate the impact of HDV standards on exposure to gaseous toxics present in significant quantities in heavy duty vehicle exhaust: benzene, formaldehyde, acetaldehyde, and 1,3-butadiene. The information in

this section is based on the 1999 'Analysis of the Impacts of Control Programs on Motor Vehicle Toxics Emissions and Exposure in Urban Areas and Nationwide' ('1999 Study').[133]

In these analyses, emissions of benzene, formaldehyde, acetaldehyde, and 1,3-butadiene were estimated using a toxic emission factor model, MOBTOX5b. This model is based on a modified version of MOBILE5b, which estimates emissions of regulated pollutants, and applies toxic fractions to total organic gas (TOG) estimates. The TOG basic emission rates used in this modeling incorporated the available elements for MOBILE6 used to develop the VOC inventory for this rule. The model accounted for differences in toxic fractions between technology groups, driving cycles, and normal versus high emitters. Impacts of fuel formulations were also addressed in the modeling.

We modeled toxic emissions for 10 urban areas and 16 geographic regions selected to encompass a broad range of I/M programs, fuel parameters, and temperature regimes. These urban areas and geographic regions are listed in Table II.A-24. The intent of the selection was to best characterize the different combinations of I/M programs, fuel parameters, and temperature regimes needed to perform accurate nationwide toxic emissions estimates. Every U.S. county in the country was then "mapped" to one of these modeled areas or regions (i.e., the emission factor for the modeled area was also used for the area "mapped" to it). Mapping was done based on a combination of geographic proximity, I/M program, and fuel control programs. Details of this process are provided in the 1999 Study. We then multiplied the resulting county level emission factors by county-level VMT estimates from EPA's Emission Trends Database and summed the results across all counties to come up with nationwide emissions in tons.

Table II.A-24. Metropolitan Areas and Regions Included in Toxic Emissions Modeling

Chicago, IL	Atlanta, GA	Florida
Denver, CO	Western WA/ OR	Northeast States – non-I/M and non-RFG
Houston, TX	Northern CA	Northeast States - I/M and non-RFG
Minneapolis, MN	Southern CA	Northeast States - non-I/M and RFG
New York, NY	ID/ MT/ WY	Ohio Valley – non-I/M and non-RFG
Philadelphia, PA	UT/ NM/NV	Ohio Valley – I/M and non-RFG
Phoenix, AZ	West TX	Ohio Valley – I/M and RFG
Spokane, WA	ND/ SD/ NB/ IA/ KS/ Western MO	Northern MI/ WI
St. Louis, MO	AR/ MS/ AL/ SC/ Northern LA	

Modeling for these areas was accomplished on a seasonal basis. Information on fuel properties for 1990 and 1996 was obtained from surveys conducted by the National Institute for Petroleum and Energy Research (NIPER) and the American Automobile Manufacturers Association (AAMA). Fuel parameters for 2007 and 2020 were projected from 1996 baseline values using information from a February 26, 1999 report from Mathpro to the American Petroleum Institute.[134] Data from the EPA Emission Trends Database and other agency sources were used to develop appropriate local modeling parameters for I/M programs, Stage II refueling controls, fuel RVP, average ambient temperature, and other inputs.

These emissions data were used as input to the HAPEM-MS3 exposure model to assess ambient exposures to the four gaseous toxics discussed in this section. With the 1990 CO exposure estimates generated by the HAPEM-MS3 model for each urban area, EPA determined the fraction of exposure that was a result of on-road motor vehicle emissions. This calculation was accomplished by scaling the exposure estimates (which reflect exposure to total ambient CO) by the fraction of the 1990 CO emissions inventory from on-road motor vehicles, determined from the EPA Emission Trends database.[135] [136] Nationwide urban CO exposure from on-road motor vehicles was estimated by first calculating a population-weighted average CO exposure for the ten modeled areas. This number was adjusted by applying a ratio of population-weighted annual average CO for urban areas in the entire country versus average ambient CO concentration for the modeled areas. To estimate rural exposure, the urban estimate was scaled downward using estimates of urban versus rural exposure from the 1993 *Motor Vehicle-Related Air Toxics Study*.[137]

Modeled on-road CO exposure for 1990 was divided by 1990 CO grams per mile emission estimates to create a conversion factor. The conversion factor was applied to modeled toxic emission estimates (in grams per mile terms) to determine exposure to on-road toxic emissions, as shown in Equation 2:

$$TOX_{Exposure(\mu g/m3)} = [CO_{Exposure(\mu g/m3)}/CO_{EF(g/mi)}]_{1990} \times TOX_{EF(g/mi)} \qquad (2)$$

where TOX reflects one of the four toxic pollutants considered in this study.

The ambient exposure estimates for calendar years 1996, 2007, and 2020 were adjusted for VMT growth relative to 1990. Exposure estimates were adjusted to account for the VOC emissions modeling conducted for this rulemaking.

To account for atmospheric loss of 1,3-butadiene that varies seasonally[kk], exposure estimates were adjusted using the following multiplicative factors: 0.44 for summer, 0.70 for spring and fall, and 0.96 for winter.[138] These factors account for the difference in reactivity

[kk] Seasons were defined as Spring (March, April, May); Summer (June, July, August); Fall (September, October, November); Winter (December, January, February).

between relatively inert CO, which is being used as the tracer for toxics exposure, and 1,3-butadiene. In contrast, estimated exposure to formaldehyde and acetaldehyde was based on direct emissions. For these pollutants, removal of direct emissions in the afternoon was assumed to be offset by secondary formation. We evaluated the validity of this assumption by comparing our results to draft average ambient concentration estimates from the 1996 National Air Toxics Assessment (NATA). The NATA for 1996 used the same inventory applied to the analysis presented here for motor vehicle toxics. The Assessment System for Population Exposure Nationwide (ASPEN) dispersion model was used in the NATA to estimate ambient concentrations of several mobile source toxics, including aldehydes. Assumptions applied in the ASPEN model include an estimate that 68 percent of formaldehyde is primary emissions (i.e. direct emission as opposed to secondary formation in the atmosphere), while only about 20 percent of acetaldehyde is assumed to be primary emissions. The comparison between ASPEN concentrations and HAPEM-MS3 exposures indicated fairly good agreement for formaldehyde, but suggested the HAPEM-MS3 exposure estimates for acetaldehyde may be low by a factor of three. Thus, our acetaldehyde exposure estimates were adjusted upward by a factor of three to match draft ambient concentration estimates from the National Air Toxics Assessment.

HAPEM-MS3 does not account for exposures originating within microenvironments. For instance, the model would not account for exposure to evaporative benzene emissions indoors from vehicles parked in attached garages, or to vehicles during refueling.

Table II.A-25 presents annual average nationwide exposure estimates from all highway motor vehicles for benzene, acetaldehyde, formaldehyde and 1,3-butadiene. The projected contribution of HDVs to the highway motor vehicle exposures estimates in 2007 is 13 percent for benzene, 51 percent for acetaldehyde, 59 percent for formaldehyde, and 9 percent for 1,3-butadiene. With today's standards in place, exposure to toxics from all HDVs in 2020 would be reduced by 7 percent for benzene, 20 percent for acetaldehyde, 23 percent for formaldehyde, and 7 percent for 1,3-butadiene. And exposure to toxics from all highway sources in 2020 (Table II.A-25) would be reduced by 2 percent for benzene, 15 percent for acetaldehyde, 18 percent for formaldehyde, and 5 percent for 1,3-butadiene.

Table II.A-25. Modeled Average 50-State Ambient Exposure to Gaseous Toxics from All Highway Motor Vehicles (µg/m^3) in 1990, 1996, 2007, and 2020 without 2007 HDV Standards and for 2020 with 2007 HDV Standards

Toxic	*1990*	*1996*	*2007*	*2020*	*2020*[A]	*Percent Reduction in 2020 with 2007 HDV Standards*[B]
Benzene	1.07	0.71	0.38	0.28	0.28	2%
Acetaldehyde	0.51	0.38	0.21	0.22	0.18	15%
Formaldehyde	0.57	0.37	0.18	0.17	0.14	18%
1,3-Butadiene	0.11	0.08	0.03	0.03	0.03	5%

[A] Exposure estimates with the 2007 Heavy-Duty Vehicle Standards.
[B] Percent reductions use exposures calculated to four decimal places.

Separately, exposure estimates were also generated for the 10 urban areas listed in Table II.A-24. In Denver, CO, Minneapolis, MN, Spokane, WA, Atlanta, GA and Phoenix, AZ, exposure to these four gaseous toxic compounds resulting from HDV emissions is projected to be higher than the national average in 2007. Of the cities modeled, Denver, and Phoenix are projected to have two-fold higher exposure estimates for acetaldehyde, formaldehyde and 1,3-butadiene from HDVs compared with the national average in 2007.

6. Visibility/Regional Haze

Visibility impairment is the haze that obscures what we see, and is caused by the presence of tiny particles in the air. These particles cause light to be scattered or absorbed, thereby reducing visibility. Visibility impairment, also called regional haze, is a complex problem that relates to natural conditions and also several pollutants. Visibility in our national parks and monuments, and many urban areas of the country, continues to be obscured by regional and local haze.

The principle cause of visibility impairment is fine particles, primarily sulfates, but also nitrates, organics, and elemental carbon and crustal matter. Particles between 0.1 and one micrometers in size are most effective at scattering light, in addition to being of greatest concern for human health. Of the pollutant gases, only NO_2 absorbs significant amounts of light; it is partly responsible for the brownish cast of polluted skies. However, it is responsible for less than ten percent of visibility reduction.

In the eastern U.S., reduced visibility is mainly attributable to secondary particles, particularly those less than a few micrometers in diameter. Based on data collected by the Interagency Monitoring of Protected Visual Environments (IMPROVE) network for visibility monitoring, sulfate particles account for about 50-70 percent of annual average light extinction in eastern locations. Sulfate plays a particularly significant role in the humid summer months, most notably in the Appalachian, northeast, and mid-south regions. Nitrates, organic carbon, and elemental carbon each account for between 10–15 percent of total light extinction in most eastern locations. Rural areas in the eastern U.S. generally have higher levels of impairment than most remote sites in the western U.S., generally due to the eastern U.S.'s higher levels of man-made pollution, higher estimated background levels of fine particles, and higher average relative humidity levels.

The relative contribution of individual pollutants to visibility impairment vary geographically. While secondary particles still dominate in the West, direct particulate emissions from sources such as woodsmoke contribute a larger percentage of the total particulate load than in the East. In the rural western U.S., sulfates also play a significant role, accounting for about 25–40 percent of estimated total light extinction in most regions. In some areas, such as the Cascades region of Oregon, sulfates are estimated to account for over 50 percent of annual average light extinction. Organic carbon typically is estimated to be responsible for 15–35 percent of total light extinction in the rural western U.S. and elemental carbon (absorption) accounts for about 15–25 percent, so the total carbonaceous contribution is between 30 and 60 percent. Soil dust (coarse PM) accounts for about 10–20 percent. Nitrates typically account for less than 10 percent of visibility impairment.[139]

The CAA requires EPA to address visibility impairment, or visual air quality, through a number of programs. These programs include the national visibility program under sections 169a and 169b of the Act, the Prevention of Significant Deterioration program for the review of potential impacts from new and modified sources, and the secondary NAAQS for PM_{10} and $PM_{2.5}$. The national visibility program established in 1980 requires the protection of visibility in 156 mandatory Federal Class I areas across the country (primarily national parks and wilderness areas). The CAA established as a national visibility goal, "the prevention of any future, and the remedying of any existing, impairment of visibility in mandatory Federal class I areas in which impairment results from manmade air pollution." The Act also calls for State programs to make "reasonable progress" toward the national goal. In July 1999, EPA promulgated a program to address regional haze in the nation's national parks and wilderness areas (see 64 FR 35714, July 1, 1999).

Since mobile sources contribute to visibility-reducing PM, control programs that reduce the mobile source emissions of direct and indirect PM would have the effect of improving visibility. Western Governors, in commenting on the Regional Haze Rule and on protecting the 16 Class I areas on the Colorado Plateau, stated that, "...the federal government must do its part in regulating emissions from mobile sources that contribute to regional haze in these areas..." and called on EPA to make a "binding commitment to fully consider the Commission's

recommendations related to the ... federal national mobile source emissions control strategies", including Tier 2 vehicle emissions standards.[140] The Grand Canyon Visibility Transport Commission's report found that reducing total mobile source emissions is an essential part of any program to protect visibility in the Western U.S.[141] The Commission identifies mobile source pollutants of concern as VOC, NO_X, and elemental and organic carbon.

Visibility is greatly affected by ambient $PM_{2.5}$ concentration, with $PM_{2.5}$ concentrations below the NAAQS being sufficient to impair visibility. Black elemental carbon particles are a dominant light adsorbing species in the atmosphere [142], and a major component of diesel exhaust. The reductions in ambient $PM_{2.5}$ from the standards in this rulemaking are expected to contribute to visibility improvements across the U.S. The geographical pattern of the improvement mirrors that of the $PM_{2.5}$ reductions. Visibility improvements have value to Americans in both recreational areas traditionally known for scenic vistas, and in the urban areas where people spend most of their time.

7. Acid Deposition

Acid deposition, or acid rain as it is commonly known, occurs when SO_2 and NOx react in the atmosphere with water, oxygen, and oxidants to form various acidic compounds that later fall to earth in the form of precipitation or dry deposition of acidic particles.[ll] It contributes to damage of trees at high elevations and in extreme cases may cause lakes and streams to become so acidic that they cannot support aquatic life. In addition, acid deposition accelerates the decay of building materials and paints, including irreplaceable buildings, statues, and sculptures that are part of our nation's cultural heritage. To reduce damage to automotive paint caused by acid rain and acidic dry deposition, some manufacturers use acid-resistant paints, at an average cost of $5 per vehicle--a total of $61 million per year if applied to all new cars and trucks sold in the U.S.

Acid deposition primarily affects bodies of water that rest atop soil with a limited ability to neutralize acidic compounds. The National Surface Water Survey (NSWS) investigated the effects of acidic deposition in over 1,000 lakes larger than 10 acres and in thousands of miles of streams. It found that acid deposition was the primary cause of acidity in 75 percent of the acidic lakes and about 50 percent of the acidic streams, and that the areas most sensitive to acid rain were the Adirondacks, the mid-Appalachian highlands, the upper Midwest and the high elevation West. The NSWS found that approximately 580 streams in the Mid-Atlantic Coastal Plain are acidic primarily due to acidic deposition. Hundreds of the lakes in the Adirondacks surveyed in the NSWS have acidity levels incompatible with the survival of sensitive fish species. Many of the over 1,350 acidic streams in the Mid-Atlantic Highlands (mid-Appalachia) region have already experienced trout losses due to increased stream acidity. Emissions from U.S. sources

[ll] Much of the information in this subsection was excerpted from the EPA document, *Human Health Benefits from Sulfate Reduction*, written under Title IV of the 1990 Clean Air Act Amendments, U.S. EPA, Office of Air and Radiation, Acid Rain Division, Washington, DC 20460, November 1995.

contribute to acidic deposition in eastern Canada, where the Canadian government has estimated that 14,000 lakes are acidic. Acid deposition also has been implicated in contributing to degradation of high-elevation spruce forests that populate the ridges of the Appalachian Mountains from Maine to Georgia. This area includes national parks such as the Shenandoah and Great Smoky Mountain National Parks.

The SOx and NOx reductions from today's rule would help reduce acid rain and acid deposition, thereby helping to reduce acidity levels in lakes and streams throughout the country and help accelerate the recovery of acidified lakes and streams and the revival of ecosystems adversely affected by acid deposition. Reduced acid deposition levels would also help reduce stress on forests, thereby accelerating reforestation efforts and improving timber production. Further deterioration of our historic buildings and monuments, and of buildings, vehicles, and other structures exposed to acid rain and dry acid deposition also would be slowed, and the costs borne to prevent acid-related damage may also decline. While the reduction in sulfur and nitrogen acid deposition would be roughly proportional to the reduction in SOx and NOx emissions, respectively, the precise impact of today's rule would differ across different areas.

8. Eutrophication and Nitrification

Nitrogen deposition into bodies of water can cause problems beyond those associated with acid rain. The Ecological Society of America has included discussion of the contribution of air emissions to increasing nitrogen levels in surface waters in a recent major review of causes and consequences of human alteration of the global nitrogen cycle in its *Issues in Ecology* series.[mm] Long-term monitoring in the United States, Europe, and other developed regions of the world shows a substantial rise of nitrogen levels in surface waters, which are highly correlated with human-generated inputs of nitrogen to their watersheds. These nitrogen inputs are dominated by fertilizers and atmospheric deposition.

Human activity can increase the flow of nutrients into those waters and result in excess algae and plant growth. This increased growth can cause numerous adverse ecological effects and economic impacts, including nuisance algal blooms, dieback of underwater plants due to reduced light penetration, and toxic plankton blooms. Algal and plankton blooms can also reduce the level of dissolved oxygen, which can also adversely affect fish and shellfish populations. This problem is of particular concern in coastal areas with poor or stratified circulation patterns, such as the Chesapeake Bay, Long Island Sound, or the Gulf of Mexico. In such areas, the "overproduced" algae tends to sink to the bottom and decay, using all or most of the available oxygen and thereby reducing or eliminating populations of bottom-feeder fish and

[mm] Vitousek, Peter M., John Aber, Robert W. Howarth, Gene E. Likens, et al. 1997. Human Alteration of the Global Nitrogen Cycle: Causes and Consequences. *Issues in Ecology*. Published by Ecological Society of America, Number 1, Spring 1997.

shellfish, distorting the normal population balance between different aquatic organisms, and in extreme cases causing dramatic fish kills.

Collectively, these effects are referred to as eutrophication, which the National Research Council recently identified as the most serious pollution problem facing the estuarine waters of the United States (NRC, 1993). Nitrogen is the primary cause of eutrophication in most coastal waters and estuaries.[nn] On the New England coast, for example, the number of red and browntides and shellfish problems from nuisance and toxic plankton blooms have increased over the past two decades, a development thought to be linked to increased nitrogen loadings in coastal waters. We believe that airborne NOx contributes from 12 to 44 percent of the total nitrogen loadings to United States coastal water bodies. For example, some estimates assert that approximately one-quarter of the nitrogen in the Chesapeake Bay comes from atmospheric deposition.

Excessive fertilization with nitrogen-containing compounds can also affect terrestrial ecosystems.[oo] Research suggests that nitrogen fertilization can alter growth patterns and change the balance of species in an ecosystem, providing beneficial nutrients to plant growth in areas that do not suffer from nitrogen over-saturation. In extreme cases, this process can result in nitrogen saturation when additions of nitrogen to soil over time exceed the capacity of the plants and microorganisms to utilize and retain the nitrogen. This phenomenon has already occurred in some areas of the U.S.

Deposition of nitrogen from heavy-duty vehicles contributes to these effects. In the Chesapeake Bay region, modeling shows that mobile source deposition occurs in relatively close proximity to highways, such as the 1-95 corridor which covers part of the Bay surface. The NOx reductions from the standards for heavy-duty vehicles should reduce the eutrophication problems associated with atmospheric deposition of nitrogen into watersheds and onto bodies of water, particularly in aquatic systems where atmospheric deposition of nitrogen represents a significant portion of total nitrogen loadings.

[nn] Much of this information was taken from the following EPA document: *Deposition of Air Pollutants to the Great Waters-Second Report to Congress*, Office of Air Quality Planning and Standards, June 1997, EPA-453/R-97-011.

[oo] Terrestrial nitrogen deposition can act as a fertilizer. In some agricultural areas, this effect can be beneficial.

9. POM Deposition

EPA's Great Waters Program has identified 15 pollutants whose deposition to water bodies has contributed to the overall contamination loadings to the these Great Waters.[pp] One of these 15 compounds, a group known as polycyclic organic matter (POM), are compounds that are mainly adhered to the particles emitted by mobile sources and later fall to earth in the form of precipitation or dry deposition of particles. The mobile source contribution of the 7 most toxic POM is at least 62 tons/year[qq] and represents only those POM that are adhered to mobile source particulate emissions. The majority of these emissions are produced by diesel engines.

POM is generally defined as a large class of chemicals consisting of organic compounds having multiple benzene rings and a boiling point greater than 100°C. Polycyclic aromatic hydrocarbons are a chemical class that is a subset of POM. POM are naturally occurring substances that are byproducts of the incomplete combustion of fossil fuels and plant and animal biomass (e.g., forest fires). Also, they occur as byproducts from steel and coke productions and waste incineration.

Evidence for potential human health effects associated with POM comes from studies in animals (fish, amphibians, rats) and in human cells culture assays. Reproductive, developmental, immunological, and endocrine (hormone) effects have been documented in these systems. 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.

The particulate reductions from today's rule would help reduce not only the particulate emissions from highway diesel engines but also the deposition of the POM adhered to the particles, thereby helping to reduce health effects of POM in lakes and streams, accelerate the recovery of affected lakes and streams, and revive the ecosystems adversely affected.

10. Carbon Monoxide

We believe that the aftertreatment technology that would be used to meet the standards for NOx, and diesel particles would result in a per-vehicle reduction in excess of 90 percent in CO from baseline levels. As of December 1999, there were 17 CO nonattainment areas with a population of about 30 million people.[143] An additional 24 areas with a combined population of 22 million are designated as CO maintenance areas. The broad trends indicate that ambient levels of CO are declining. The standards being promulgated today would help reduce levels of carbon monoxide (CO).

[pp] Much of this information was taken from the following EPA document: *Deposition of Air Pollutants to the Great Waters-Second Report to Congress*, Office of Air Quality Planning and Standards, June 1997, EPA-453/R-97-011. You are referred to that document for a more detailed discussion.

[qq] *The 1996 National Toxics Inventory*, Office of Air Quality Planning and Standards, October 1999.

B. Heavy-Duty Diesel Inventory Impacts

This part of the environmental impact chapter presents the emission inventory reductions we anticipate from heavy-duty vehicles as a result of our NMHC, NOx, and PM emission standards and as a result of our fuel sulfur standards. This section provides detail on our emissions inventory calculations and catalogs changes from the NPRM analysis. In addition, this section contains a sensitivity analysis of deterioration, tampering, and malmaintenance on PM emissions.

1. Description of Calculation Method

We calculated our emissions reductions by first determining baseline emissions from HDVs then determining the percent reduction by calendar year. The determination of the baseline and controlled inventories is described below.

For the controlled emission inventory, we actually present two cases. These two control cases are labeled as *Air Quality Analysis Case* and *Updated Control Case.* The *Air Quality Analysis Case* is used in the county-by-county, hour-by-hour air quality analyses associated with this rule. This inventory was developed using the assumptions and proposed standards presented in the NPRM for this rule. Because the detailed air quality analyses take several months to perform, we had to begin as soon as the NPRM was finalized and were not able to incorporate any changes in the final standards.

The *Updated Control Case* incorporates changes in the standards and assumptions from the NPRM to the FRM. Although the differences are fairly small, the *Updated Control Case* more precisely represents the reductions associated with the final standards and is used in our cost-effectiveness analysis. These updates only affect the control inventory and do not affect the baseline inventory.

a. Baseline Emissions Inventory

i. HC, NOx, CO, PM, and SOx

In modeling emissions from heavy-duty diesel engines, our intent is to be consistent with the upcoming MOBILE6 model. MOBILE6 is the upcoming version of the MOBILE model that we historically use to develop calendar year specific emission factors for highway vehicles. This model will be publically available in early 2001. However, the new data used develop this model has been made publicly available for stakeholder review. Therefore, we use new published data that was developed for use in the upcoming MOBILE6.

Emissions inventories from HDVs were calculated at the county level for 1996, 2007, 2020, and 2030. MOBILE5 was used to calculate VOC,[rr] NOx, and CO emissions inventories; PART5 was used to calculate PM and SOx. Adjustment factors were then applied to account for the new data collected as part of the development of the upcoming MOBILE6 emission factor model. This methodology is described and detailed inventories are presented in the docket.[144] [145] The adjustment factors used to incorporate the new data and the development of these adjustment factors are also described in the docket.[146] [147] No adjustments were made to the brake and tire wear calculations.

ii. Fuel Consumption

To determine the impact of the low sulfur diesel fuel requirement on vehicle operation costs and on emissions, we first need to calculate the diesel fuel consumption. We calculated HDDE fuel consumption using Equation 1:

$$Gallons_{CY} = j_{class} \left\{ VMT \times j_{MY/age} \left[FC \times TF_{age} \right] \right\} \quad (1)$$

where:

$Gallons_{CY}$ - fuel consumption in gallons/year
class - LHDDE, MHDDE, HHDDE, and urban bus
VMT- total vehicle miles traveled in a given calendar year by class
MY/age - distribution of vehicles in a calendar year by vehicle age
FC - fuel consumption in gallons per mile
TF_{age} - travel fraction of vehicles from each model year in a given calendar year

VMT projections are described in the same report as the calculations of VOC, NOx, CO, PM, and SOx.[148] The travel fraction is described in the memo which details the adjustment factors.[149]

Historical fuel consumption estimates (1987-1996) come from a report performed to support the upcoming MOBILE6 model.[150] These historical fuel consumption estimates suggest that fuel economy is improving. For future fuel consumption estimates, we extrapolate the historical estimates into the future using a constant, linear improvement in terms of miles per gallon. We use a single, weighted average, growth rate for MHDDEs and HHDDEs. This is because a straight projection of the MHDDE and HHDDE fuel economies would suggest that HHDDEs would have better fuel economy than MHDDEs beginning in 2020. We don't believe this is likely because of the lower weight of MHDDEs. Table II.B-3 presents per-vehicle the HDDE fuel economy estimates for selected years.

[rr] Volatile Organic Compounds–This includes exhaust and evaporative hydrocarbon emissions.

Table II.B-1. HDDE Fuel Economy Estimates by Model Year (miles per gallon)

Model Year	*LHDDE*	*MHDDE*	*HHDDE*	*Urban Bus*
1990	10.7	7.7	5.9	3.6
2000	11.8	8.1	6.6	4.0
2010	12.9	8.7	7.3	4.4
2020	14.0	9.4	7.9	4.8

To fully evaluate the effects of the fuel sulfur level standards, we also need to consider other sources that will likely consume low sulfur fuel produced for HDDEs. These sources include light-duty vehicles, off-highway engines, and stationary sources. We refer to the low sulfur fuel used in sources other than highway engines as spillover.

To include the gallons consumed by light-duty diesel vehicles, we use estimates developed for our Tier 2 final rule[151] and fuel economy estimates of 25 mpg and 16.7 mpg for light-duty diesel vehicles (LDDV) and light-duty diesel trucks (LDDT), respectively.[152] We divided the VMT values within each of these light-duty diesel fuel categories by the corresponding MOBILE6 projected fuel economy estimates to derive the diesel fuel consumption for each category per year.

Highway engines are not the only sources that burn highway diesel fuel. Due to limitations of the fuel production and distribution system, a considerable amount of low sulfur diesel fuel is currently consumed in off-highway and other applications. To estimate the amount of highway diesel fuel consumed by other sources, we used data compiled by the Energy Information Administration (EIA) which showed that combined 1996 production plus importation minus exportation of highway diesel fuel was 32.8 billion gallons.[153] We then subtracted our estimates of HDDE and LDV diesel fuel consumption to determine the spillover to sources other than highway engines.

For future years we estimate that spillover will increase as fuel production increases. We recognize that spillover could decrease in future years if the highway fuel cost were to increase significantly with respect to the off-highway fuel cost and if the fuel were redistributed economically. However, we believe the proportion of spillover is largely driven by the limitations of the fuel distribution system and that it is not likely to change substantially in response to this rule.

iii. Crankcase Emissions

We anticipate some benefits in NMHC, NOx, and PM from the closed crankcase requirements for turbocharged HDDEs. Based on limited engine testing, we estimate that crankcase emissions of NMHC and PM from HDDEs are each about 0.01 g/bhp-hr.[154] NOx data varies, but crankcase NOx emissions may be as high as NMHC and PM. Therefore, we use the same crankcase emission factor of 0.01 g/bhp-hr for each of the three constituents.

iv. Air Toxics

We use baseline gaseous toxic emission estimates for heavy duty gasoline vehicles prepared by Sierra Research. Sierra developed inventory estimates for several gaseous mobile source air toxics (MSAT), including acetaldehyde, benzene, 1-3 butadiene, and formaldehyde.[155] The Sierra study provided estimates of toxic emissions under various control scenarios for several years. These specific MSATs were addressed because detailed information on the emission impacts of emission control technologies, fuel properties, and other parameters were available for these compounds.

The emissions of benzene, formaldehyde, acetaldehyde, and 1,3-butadiene were estimated using a toxic emission factor model, MOBTOX5b. This model is based on a modified version of MOBILE5b, which estimates emissions of regulated pollutants, and essentially applies toxic fractions to total organic gas (TOG) estimates. The TOG basic emission rates used in this modeling incorporated available elements from MOBILE6 used to develop the VOC inventory for the Tier 2 final rule. The model accounted for differences in toxic fractions between technology groups, driving cycles, and normal versus high emitting vehicles and engines ("high emitters"). Impacts of fuel formulations were also addressed in the modeling.

Sierra modeled toxic emissions for 10 urban areas and 16 geographic regions. The areas were selected to encompass a broad range of I/M programs, fuel parameters, and temperature regimes. The intent of the selection process was to best characterize the different combinations needed to perform accurate nationwide toxic emissions estimates. Every U. S. county in the country was then "mapped" to one of these modeled areas or regions (i.e., the emission factor for the modeled area was also used for the area "mapped" to it). Mapping was done based on a combination of geographic proximity, I/M program, and fuel control programs.

Modeling for these areas was done on a seasonal basis. Information on fuel properties for was obtained from surveys conducted by the National Institute for Petroleum and Energy Research (NIPER) and the American Automobile Manufacturers Association (AAMA) and additional information from the American Petroleum Institute. Data from the EPA Emission Trends Database and other agency sources were used to develop appropriate local modeling parameters for I/M programs, Stage II refueling controls, fuel RVP, average ambient temperature, and other inputs.

To estimate the effect of the 2007 and later model year heavy-duty engine standards on toxics inventories, we started with the toxics inventories estimated in the Sierra study assuming

all heavy-duty engine programs up until the 2004 model year standards are in effect. Using these "baseline" inventory estimates for 2007 and 2020 and the nationwide vehicle miles traveled estimates from the same study, we then estimated the "baseline" gram per mile emissions for the five toxics (on a nationwide, average basis) for 1996, 2007 and 2020. The emission factors for other years, were interpolated from these estimates.

Finally, we then multiplied the gram per mile estimates by the nationwide vehicle mile traveled estimates developed for this rule, to obtain the heavy-duty gasoline and diesel vehicle toxic inventories used in this analysis. Because benzene has an exhaust and an evaporative component, we applied the percent reduction based on total (exhaust and evaporative) NMHC benefits. For formaldehyde, acetaldehyde, and 1,3-butadiene, which do not have an evaporative component, we applied the percent reduction based on exhaust NMHC only.

b. Controlled Emissions Inventory (Air Quality Analysis Case)

i. HC, NOx, CO, PM, and SOx

To determine the emissions reductions in NMHC, NOx, CO, and PM we look at the percent emission reductions expected from new engines then calculate percent reductions by calendar year using the travel fractions discussed above. For the *Air Quality Analysis Case* we base the calculations on the proposed HDV standards. This methodology is described and detailed inventories are presented in the docket.[156] [157] We assume that manufacturers will design their engine with a compliance margin below the standards. Based on historical certification data, we use an eight percent compliance margin for HDDEs and a 25 percent compliance margin for HDGVs.

Based on our analysis of the aftertreatment technology described in Chapter III, HDDEs meeting the standards should have very low levels of CO. Although the standards give manufacturers the same phase-in for NMHC as for NOx, we model the NMHC reductions to be fully in place for diesel engines in 2007. We believe the use of aftertreatment for PM control will result in HDDEs meeting the NMHC standards in 2007 and will result in 90 percent reductions in CO levels soon as the PM standard goes into effect in 2007. In the *Air Quality Analysis Case*, we assume that particulate traps will result in a 90 percent reduction in NMHC; however, as discussed later, we changed this assumption in the *Updated Control Case.*

We assume that hot soak, diurnal and resting loss emissions from HDGVs would be reduced proportionally to the reduction in the evaporative emission standard. However, we only apply these reductions to the emissions of HDGVs which pass the EPA pressure and EPA purge functional test procedures. We do not claim any benefits from HDGVs which fail these tests.

The majority of the projected PM reductions from HDDEs are directly a result of the PM standard. However, some PM reductions will come from reducing sulfur in the fuel. Reducing sulfur in the fuel decreases the amount of direct sulfate PM (DSPM) emitted from heavy-duty

diesel engines and other engines using highway fuel. This section describes the calculations exhaust emission PM benefits that are directly the result of the 2007 standards. DSPM benefits from the existing fleet are calculated separately and are discussed later. For SOx reductions, we look at the reduction of sulfur in the fuel and the amount of sulfur in the fuel that can be assumed to be converted to SO_2.

The control emission factors and percent reductions by calendar year are described in more detail in the docket.[158] [159]

ii. Direct Sulfate PM

Once the low sulfur diesel fuel requirements go into effect, pre-2007 model year HDDEs will be using low sulfur fuel, as will engines using new PM control technology. Because these pre-2007 engines will be certified with high sulfur fuel, they will achieve reductions in PM beyond their certification levels.

For engines built prior to 2007 that use low sulfur fuel in 2007 and later, we need to calculate the PM benefit associated with the reduction of direct sulfate PM. Equation 2 shows how we calculate this benefit and express it in terms of an emission factor. We did not consider deterioration for DSPM which is consistent with our analysis of total PM. We must calculate the per-vehicle average g/mi reduction independently for each class and calendar year.

$$DSPM_{TONS} = 10^{-6} \times ppmS \times MWR \times S_{conv} \times FF \times FC \times density/2000 \quad (2)$$

where:

$DSPM_{TONS}$ - direct sulfate PM for a given calendar year [short tons]
ppmS = average fuel sulfur level expressed in parts per million
MWR - molecular weight ratio of DSPM measured on a filter to sulfur in the fuel
= 224/32 (224 is the molecular weight of H_2SO_4 hydrated seven times)
S_{conv} - % of sulfur in fuel converted to direct sulfate PM
FF - fraction of VMT from pre-2007 MY fleet
FC - total consumption of fuel intended for HDDEs in gallons
density - fuel density = 7.1 lbs/gallon

For the reduction in average fuel sulfur level, we use 334 ppm. We base this reduction on an average baseline fuel level of 340 ppm S and an average low sulfur fuel level of 7 ppm S with adjustments for sulfur in the oil. We estimate that oil adds the equivalent of about 1 ppm S to the fuel. In the baseline case most of the crankcase vapor is vented to the atmosphere which minimizes the oil burned in the cylinder. In the control case where there are closed crankcase requirements, we consider the oil recovery system discussed in Chapter III.

We use the fuel consumption estimates described above in Section B.1.a.iv. This fuel consumption includes highway fuel burned in heavy-duty engines, light-duty vehicles, and other sources which use distillate fuel.

For engines not using aftertreatment, we assume that 2 percent of the sulfur in the fuel is converted to direct sulfate PM. This conversion rate is consistent with the PART5 emission model. We model the use of low sulfur fuel to begin in mid-2006.

iii. Crankcase Emissions

By routing crankcase vapors to the exhaust upstream of the aftertreatment systems, HDDE manufacturers should be able to reduce crankcase emissions by about the same percentage as for engine-out exhaust. For this analysis, we recognize that the crankcase emissions will be included in the total exhaust emissions when the engine is designed to the standards. Because exhaust emissions would have to be reduced slightly to offset any crankcase emissions, the crankcase emission control is functionally equivalent to a 100 percent reduction in crankcase emissions.

The engine data we use to determine crankcase emission levels is based on new HDDEs. We do not have data on the effect of in-use deterioration of crankcase emissions. However, we expect that these emissions would increase as the engine wears. Therefore, this analysis may underestimate the benefits that would result from our crankcase emission requirements.

iv. Air Toxics

We use the same methodology to calculate the controlled toxics inventory as the baseline inventory. We lack data on how the toxic fractions of the hydrocarbons may change for engines designed to meet the new standards; therefore, we assume for the sake of analysis that the toxic fractions do not change. In other words, we assume the same percent reductions in air toxics as we calculate for hydrocarbons.

c. Controlled Emissions Inventory (Updated Control Case)

The main purpose of the updated control case is to consider changes between the standards proposed in the NPRM and the standards finalized today. For these calculations, we consider the heavy-duty vehicle standards as presented in Table II.B-1 and the standards phase-in dates presented in Table II.B-2. All HDDEs are engine-certified, however; most heavy-duty gasoline vehicles are chassis-certified. We refer to gasoline engines sold as part of a chassis as "completes" and require these engines to be certified on a chassis-based test provided that the vehicle does not have a gross vehicle weight rating more than 14,000 pounds. Other gasoline engines are tested on an engine dynamometer and we refer to these as "incompletes."

Table II.B-2. Heavy-Duty Vehicle Exhaust Emissions Standards

Class	*Units*	*NMHC*	*NOx*	*PM*
HDDE	g/bhp-hr	0.14	0.20	0.01
HDGV, 2b Completes	g/mile	0.195	0.2	0.02
HDGV, 3 Completes	g/mile	0.230	0.4	0.02
HDGV Incompletes	g/bhp-hr	0.14	0.20	0.01

Table II.B-3. Heavy-Duty Vehicle Standards Phase-In (percent of production)

Model Year	*HDDE (NMHC & NOx)*		*HDDE (PM)*[A]		*HDGV*[B]	
	NPRM	*FRM*	*NPRM*	*FRM*	*NPRM*	*FRM*
2007	25%	50%	100%	100%	100%	0%
2008	50%	50%	100%	100%	100%	50%
2009	75%	50%	100%	100%	100%	100%
2010+	100%	100%	100%	100%	100%	100%

[A] This applies to the closed crankcase requirement as well.
[B] This applies to evaporative emission standards as well.

As shown above, the actual values of the standards have not changed since proposal. However, the implementation dates have changed somewhat. One other change is that we assume that diesel engine manufacturers will design their engines to meet the NMHC with a small compliance margin. In the NPRM, we assumed that particulate traps would result in a 90 percent reduction in NMHC. This is discussed in more detail in Chapter III. Other than for NMHC, the net effect of the changes in the FRM from the NPRM for HDDEs is small. However, the FRM implementation dates essentially delay the HDGV standards by a year and a half. The *Updated Control Case* calculates the reductions from HDVs using the same methodology as the *Air Quality Analysis Case* except that the new HDDE NMHC assumptions and FRM implementation dates are used.

Also, we consider the low sulfur diesel temporary compliance flexibilities and hardship provisions in our calculations. These provisions allow as little as 75 percent of highway diesel fuel sales to be 15 ppm sulfur beginning in 2006; increasing to 100% in 2010. In the NPRM, we

proposed to require that all highway diesel fuel meet the standard in 2006. This delay in production affects SOx and DSPM benefits from HDDEs.

2. HDDE Emission Reductions

a. Anticipated Reductions due to the New HDDE Standards

This section looks at tons/year emission inventories of NOx, PM, and NMHC from HDDEs. These are the emissions that we are directly regulating from HDDEs. We present our projected baseline and controlled emissions inventories in addition to our anticipated benefits. Where there is a difference, we present both the results from the *Air Quality Analysis Case* (AQAC) and the *Updated Control Case* (Updated). In addition, this section presents the total production of highway diesel fuel which will be required to meet the low sulfur standard set today.

i. NOx Reductions

Today's standards should result in about a 90 percent reduction in NOx from new engines. Table II.B-4 presents these projections with the estimated NOx benefits for selected years.

Table II.B-4. Nationwide NOx Emissions from HDDEs (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*		*Reduction*	
		AQAC	*Updated*	*AQAC*	*Updated*
2007	2,650	2,620	2,600	29	57
2010	2,440	2,020	2,040	416	403
2015	2,310	1,080	1,090	1,230	1,220
2020	2,350	582	587	1,770	1,760
2030	2,770	291	292	2,480	2,480

ii. PM Reductions from 2007 Model Year and Later

This section just looks at exhaust emission PM benefits that are directly the result of the 2007 standards. DSPM benefits are presented later. For engines meeting the new standards, we consider low sulfur fuel to be necessary to enable the PM control technology. In other words, we

don't claim additional emissions reductions beyond the standard due to reductions in direct sulfate PM except for the difference between certification and average in-use fuel sulfur levels as discussed above.

The new standards should result in about a 90 percent reduction in exhaust PM from new engines. This translates to a 76 percent reduction in total PM_{10} when brake and tire wear are considered. Table II.B-5 presents these projections with the estimated PM benefits for selected years. This table includes brake and tire wear, but does not include the direct sulfate benefits from the existing fleet. These results do not change between the AQAC and Updated analyses.

Table II.B-5. Nationwide PM_{10} Exhaust and Break/Tire Wear Emissions from HDDEs Without Existing Fleet Reductions (thousand short tons per year)

Calendar Year	*Baseline Exhaust*	*Control Exhaust*	*Reduction*	*Brake/Tire Wear*
2007	96	91	5	13
2010	84	57	27	15
2015	80	28	51	17
2020	86	15	71	19
2030	104	8	96	23

iii. NMHC Reductions

Although the standards give manufacturers the same phase-in for NMHC as for NOx, we model the NMHC reductions to be fully in place for diesel engines in 2007. As discussed earlier, we believe the use of aftertreatment for PM control will cause the NMHC levels to meet the standard as soon as the PM standard goes into effect in 2007, but in the *Updated Control Case*, no longer assume a 90 percent reduction due to the particulate trap. This standard will result in about a 30 percent reduction in NMHC from new engines. Table II.B-6 presents these projections with the estimated NMHC reductions for selected years.

Table II.B-6. Nationwide NMHC Exhaust Emissions from HDDEs (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*		*Reduction*	
		AQAC	*Updated*	*AQAC*	*Updated*
2007	184	175	182	9	2
2010	185	132	172	53	13
2015	191	74	156	117	35
2020	206	40	152	166	54
2030	240	25	167	217	74

iv. Fuel Consumption Estimates

Table II.B-7 presents national fuel consumption estimates for HDDEs. Table II.B-8 presents our estimates of low sulfur fuel consumption. This total consumption includes on-highway fuel used by light duty diesel vehicles and spillover into sources other than on-highway. Our total consumption estimates are similar to EIA's production estimates and our highway fuel consumption estimates are consistent with Federal Highway Association estimates of taxed highway diesel fuel use.[160]

Table II.B-7. HDDE Fuel Consumption Estimates by Calendar Year (billion gallons)

Calendar Year	*LHDDE*	*MHDDE*	*HHDDE*	*Urban Bus*
2007	4.26	5.57	26.4	0.86
2010	4.52	5.94	27.9	0.91
2015	4.93	6.53	30.4	0.99
2020	5.30	7.06	32.6	1.06
2030	5.95	8.02	36.5	1.18

Table II.B-8. Consumption of Highway Diesel Fuel Including Spillover (billion gallons)

Calendar Year	*Light-duty*	*Heavy-duty*	*Spillover*	*Total*
2007	0.37	37.1	4.09	41.5
2010	0.39	39.3	4.25	44.0
2015	0.43	42.8	4.51	47.8
2020	0.46	46.0	4.78	51.2
2030	0.53	51.6	5.30	57.5

v. DSPM Reductions from Existing Fleet

Figure II.B-1 shows our national projections (using the *Updated Control Case*) of direct sulfate PM emissions from the pre-2007 engines using HD highway diesel fuel with and without the low sulfur fuel. The low sulfur fuel should result in about a 95 percent reduction in direct sulfate PM from pre-2007 engines. Table II.B-9 presents the estimated DSPM benefits from HDDEs and other engines using the same fuel for selected years.

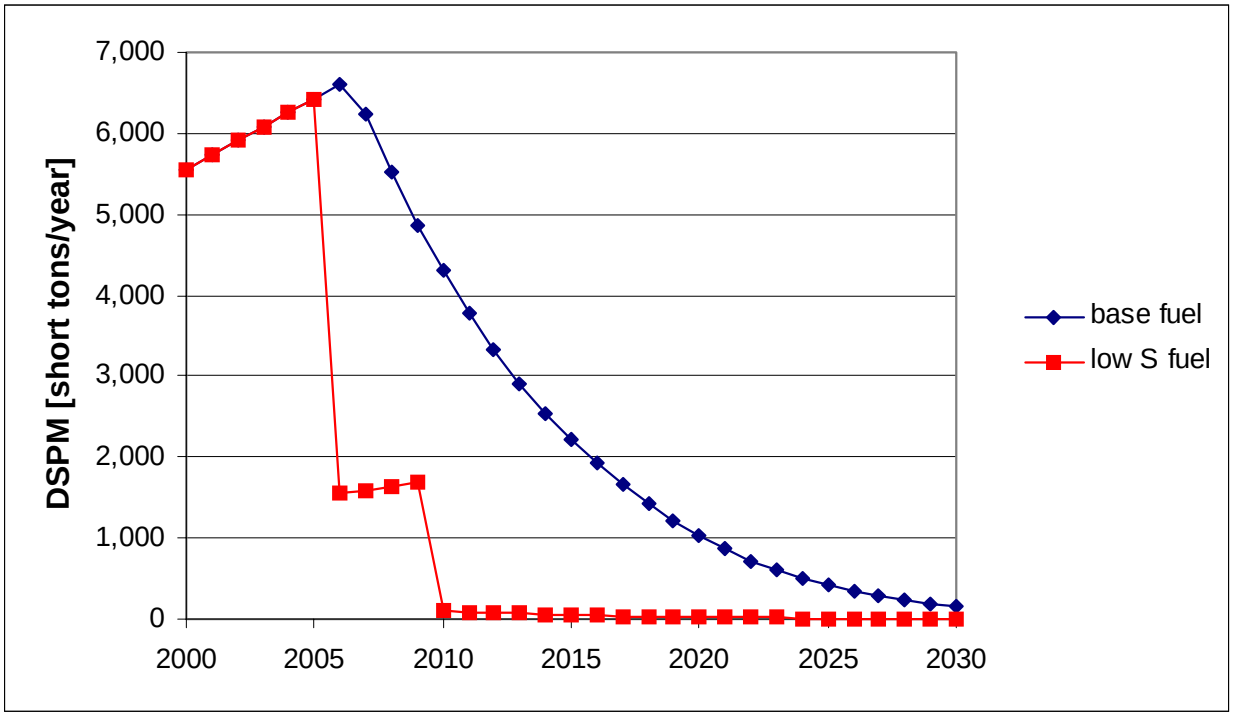

Figure II.B-1. Projected DSPM from Pre-2007 Engines Using Highway Diesel Fuel

Table II.B-9. Existing Fleet PM Reductions From Low Sulfur Fuel (thousand short tons per year)

Calendar Year	*HDDEs*		*Other*		*Total Reductions*	
	AQAC	*Updated*	*AQAC*	*Updated*	*AQAC*	*Updated*
2007	6.07	4.64	0.73	0.56	6.80	5.20
2010	4.19	4.19	0.50	0.50	4.69	4.69
2015	2.16	2.16	0.25	0.25	2.41	2.41
2020	1.00	1.00	0.11	0.11	1.12	1.12
2030	0.15	0.15	0.02	0.02	0.17	0.17

vi. Crankcase Emission Reductions

Table II.B-10 presents our estimates of the baseline crankcase emissions from HDDEs. As described earlier, we assume that the crankcase emissions would be zero for the controlled case. These calculations do not differ between the AQAC and Updated analyses.

Table II.B-10. Crankcase Emissions from Uncontrolled HDDEs (thousand short tons per year)

Calendar Year	*NOx*	*PM*	*NMHC*
2007	0.7	0.7	0.7
2010	3.7	3.7	3.7
2015	7.1	7.1	7.1
2020	9.5	9.5	9.5
2030	12.8	12.8	12.8

vii. Sum of NOx, PM, and NMHC Reductions

As discussed above, we are anticipating large emission reductions in NOx, PM, and NMHC from HDDEs as a result of the new exhaust emission standards. In addition, we are anticipating reductions in PM from the existing fleet due to the low sulfur fuel and reductions from 2007 and later MY engines due to the closed crankcase requirements. Table II.B-11 presents the total projected reductions from HDDEs for this rule for selected years.

Table II.B-11. Total Reductions from HDDEs for this Rule (thousand short tons per year)

Calendar Year	*NOx*		*PM*		*NMHC*	
	AQAC	*Updated*	*AQAC*	*Updated*	*AQAC*	*Updated*
2007	29	58	13	11	10	2
2010	419	406	35	35	57	17
2015	1,240	1,230	61	61	124	43
2020	1,780	1,770	82	82	175	64
2030	2,490	2,490	109	109	229	87

This action is the second of two rules requiring large reductions in NOx emissions from HDDEs. The 2004 standards reduce NOx from 4 g/bhp-hr to about 2.3 g/bhp-hr. The new standards will reduce NOx again by another 2.1 g/bhp-hr in 2007. This is a 95 percent reduction in NOx from new engines. Figure II.B-2 presents (using the *Updated Control Case*) the combined effects of the two standards on national HDDE NOx emissions. This figure also includes crankcase emissions.

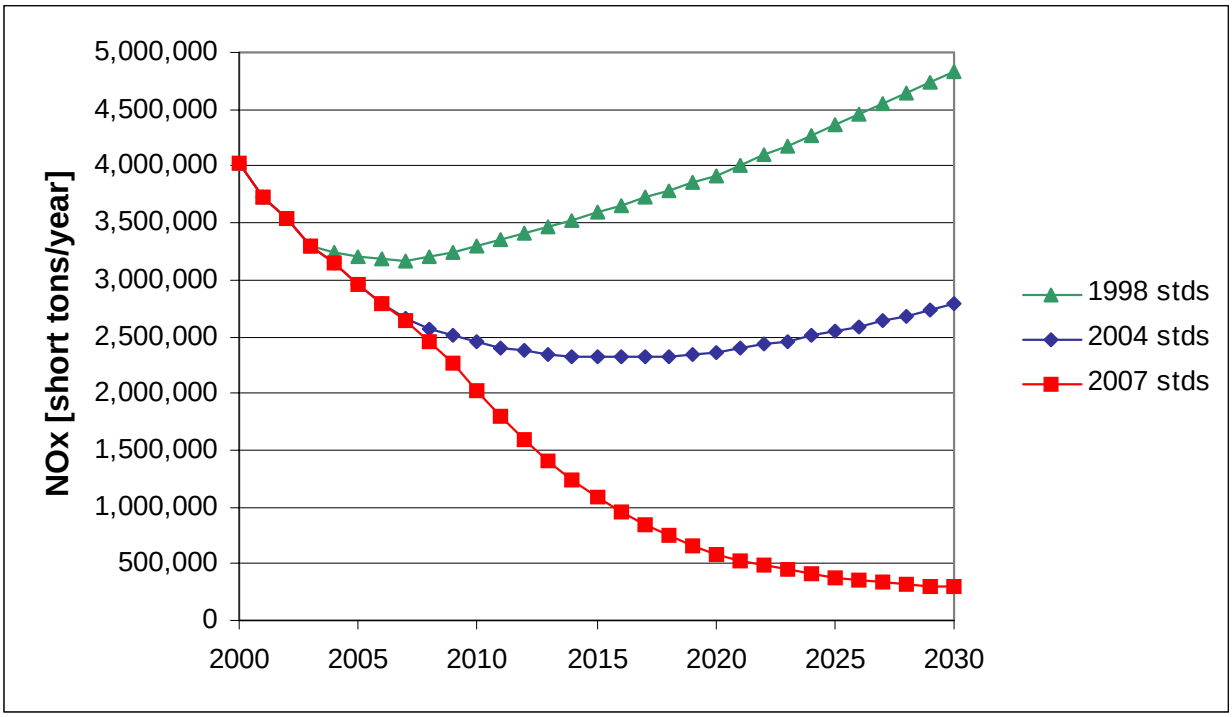

Figure II.B-2. Projected HDDE NOx Emissions Due to 2004 and 2007 Standards

Figure II.B-3 shows (using the *Updated Control Case*) our national projections of total PM emissions with and without the new engine controls. This figure includes brake and tire wear, crankcase emissions, and the direct sulfate PM benefits due to the use of low sulfur fuel by the existing fleet.

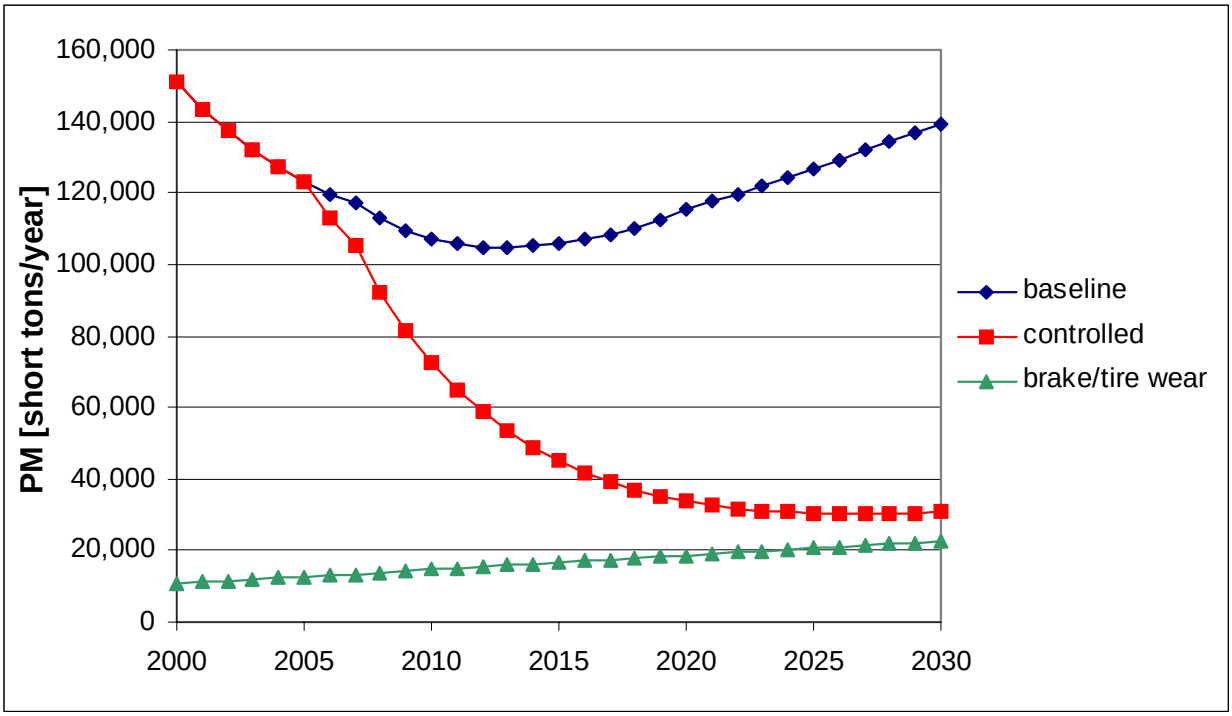

Figure II.B-3. Projected Nationwide PM Emissions from HDDEs

Figure II.B-4 shows (using the *Updated Control Case*) our national projections of total NMHC crankcase and exhaust emissions from HDDEs with and without the new engine controls.

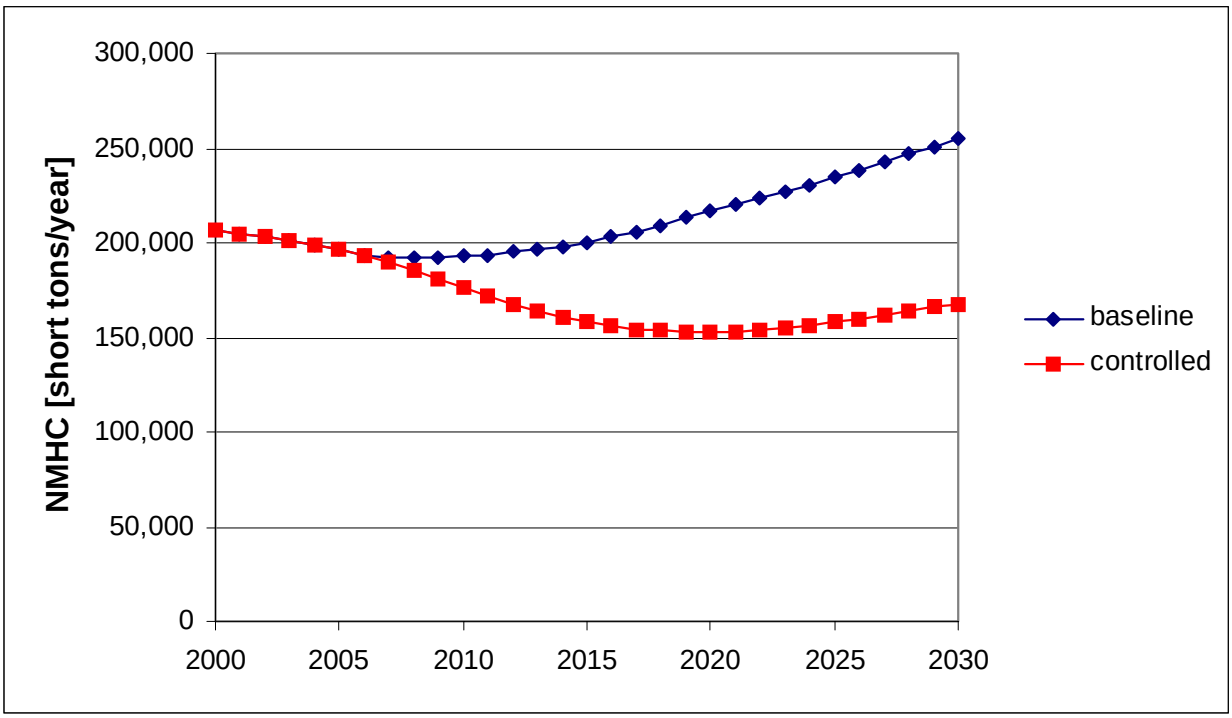

Figure II.B-4. Projected Nationwide NMHC Emissions from HDDEs

b. Additional Reductions due to the New HDDE Standards

This section looks at tons/year emission inventories of carbon monoxide (CO), oxides of sulfur (SOx), and air toxics from HDDEs. Although we are not including explicit new standards for these pollutants in today's action, we believe the new standards will result in reductions in CO, SOx, and air toxics. Here we present our anticipated benefits.

i. CO Reductions

Although the CO standard for HDDEs remains at 37.1 g/bhp-hr, CO emission levels from certified HDDEs are much lower. According to the emission factor report[161] we use for baseline EFs and DFs, baseline emissions for CO range from 1.0 to 1.3 g/bhp-hr for HDDEs. We believe that the exhaust emission control technology that would be used to meet the standards would result in excess of a 90 percent reduction in CO from baseline levels. This is because PM traps have very high oxidation capabilities. We use 90 percent here to be conservative. Using this assumption, Table II.B-12 presents projected reductions in CO from HDDEs. These results do not change between the AQAC and Updated analyses.

Table II.B-12. Reductions in CO from HDDEs (thousand short tons per year)

Calendar Year	*CO Reduction*
2007	56
2010	317
2015	691
2020	982
2030	1,290

ii. SOx Reductions

We assume that all of the sulfur in the fuel not converted to direct sulfate PM is converted to sulfur dioxide. For pre-2007 engines, we assume that 98 percent of the sulfur is converted to SO_2; for 2007 and later engines, we assume that 70 percent of the sulfur is converted to SO_2. Because we are converting from S to SO_2, we use a molecular weight ratio of 64/32. Table II.B-13 presents our estimates of SOx reductions from HDDEs corresponding with the use of low sulfur fuel. Table II.B-13 also presents SOx benefits from other sources using highway diesel fuel as discussed earlier in this chapter.

Table II.B-13. Reductions in SOx from Low Sulfur Fuel (thousand short tons per year)

Calendar Year	*HDDE SOx Reduction*		*Other SOx Reduction*	
	AQAC	*Updated*	*AQAC*	*Updated*
2007	90	70	11	8
2010	96	96	12	11
2015	105	105	13	12
2020	113	113	13	13
2030	127	127	14	14

iii. Air Toxic Reductions

Table II.B-14 shows the estimated air toxics reductions associated with the anticipated reductions in hydrocarbons. The difference between the toxics reductions from the *Air Quality Analysis Case* and the *Updated Control Case* are due to the revised assumption about NMHC reductions due to PM traps.

Table II.B-14. Reductions in Air Toxics from HDDEs (thousand short tons per year)

Calendar Year	*Benzene*		*Formaldehyde*		*Acetaldehyde*		*1,3-Butadiene*	
	AQAC	*Updated*	*AQAC*	*Updated*	*AQAC*	*Updated*	*AQAC*	*Updated*
2007	0.14	0.02	1.02	0.18	0.38	0.07	0.08	0.01
2010	0.87	0.22	6.47	1.60	2.38	0.59	0.50	0.12
2015	2.01	0.61	15.0	4.52	5.52	1.66	1.17	0.35
2020	2.81	0.92	20.1	6.83	7.71	2.52	1.63	0.53
2030	3.80	1.30	28.3	9.70	10.4	3.57	2.21	0.76

3. HDGV Emission Reductions

This section presents reductions in NOx, exhaust and evaporative NMHC, and air toxics from HDGVs that we anticipate from this rule. Although, medium-duty passenger vehicles (MDPV) are technically part of the HDGV class, they are not included in the standards finalized today. Therefore, emissions from MDPVs are not included in the inventories presented here. MDPVs were recently regulated under the Tier 2 light-duty vehicle rule.

Also, we do not claim benefits for reductions in California for HDGVs due to California's comparably stringent LEV2 standards for these vehicles. However, the charts presented below will include national inventories. In the tables, we will only present emissions reductions that we are claiming for this rule.

The *Air Quality Analysis Case* includes emissions from MDPVs in its baseline, and includes 50-state emissions reductions from the HDGVs regulated under this rule. Therefore, we also present an adjusted AQAC inventory which only includes HDGVs covered by this rule and distinguishes between 49-state and 50-state emission reductions. The *Updated Control Case* not only accounts for the difference between the proposed and final standards, but accounts for MDPVs and California reductions. All of the charts in this section are based on the *Updated*

Control Case; however, the tables present the AQAC (with and without MDPV emissions) and Updated (excludes MDPV emissions) results.

a. NOx Reductions

Figure II.B-5 presents the projected NOx inventory with and without the new standards. We believe the NOx standards will result in more than a 60 percent reduction in NOx from new heavy-duty gasoline vehicles. Tables II.B-15.a and II.B-15.b present these projections with the estimated NOx reductions for selected years for the AQAC and Updated inventories respectively. Table II.B-15a distinguishes between the inventory with and without medium duty passenger vehicles (MDPV). Although these vehicles are classified as HDGVs, they were included in the Tier 2 standards and therefore are not included in today's standards. Table II.B-15b presents the Updated control case which considers a delay in the standards compared to the AQAC inventory. MDPV emissions are excluded from this table.

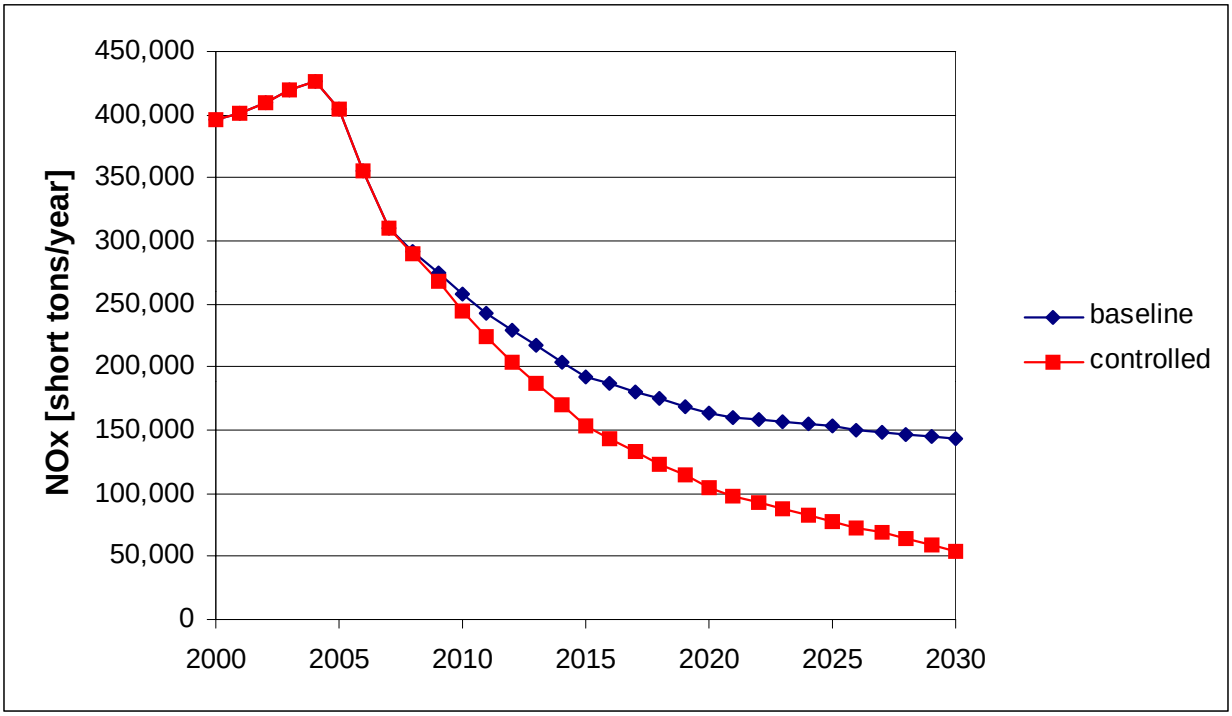

Figure II.B-5. Projected Nationwide Exhaust NOx Emissions from HDGVs

Table II.B-15.a. Estimated Nationwide NOx Emissions from HDGVs Based on the Air Quality Analysis Case (thousand short tons per year)

Calendar Year	*Baseline*		*Controlled*		*Reduction*	
	AQAC	*w/o MDPVs*	*AQAC*	*w/o MDPVs*	*50-state*	*49-state*
2007	381	310	377	306	4	3
2010	316	257	292	233	24	21
2015	236	192	187	144	48	43
2020	200	163	133	96	67	60
2030	175	143	81	49	94	84

Table II.B-15.b. Estimated Nationwide NOx Emissions from HDGVs Based on the Updated Control Case (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Reduction*	
			50-state	*49-state*
2007	310	310	0	0
2010	257	244	13	12
2015	192	154	38	34
2020	163	105	58	52
2030	143	54	88	79

b. Exhaust NMHC Reductions

Figure II.B-6 presents the projected exhaust NMHC inventory with and without the new standards. We believe the NMHC standard will result in about a 30 percent reduction in exhaust NMHC from new heavy-duty gasoline vehicles. Tables II.B-16.a and II.B-16.b present these projections with the estimated exhaust NMHC reductions for selected years for the AQAC and Updated inventories.

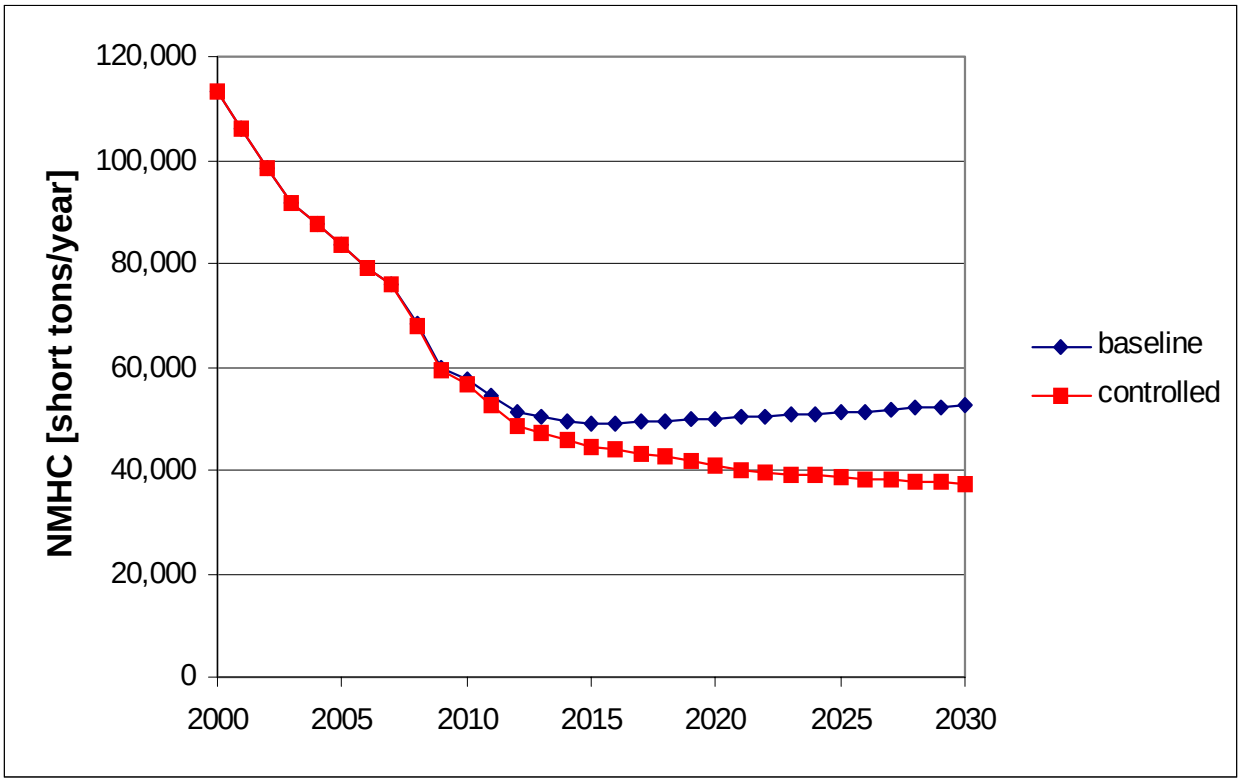

Figure II.B-6. Projected Nationwide Exhaust NMHC Emissions from HDGVs

Table II.B-16.a. Estimated Nationwide Exhaust NMHC Emissions from HDGVs Based on the Air Quality Analysis Case (thousand short tons per year)

Calendar Year	*Baseline*		*Controlled*		*Reduction*	
	AQAC	*w/o MDPVs*	*AQAC*	*w/o MDPVs*	*50-state*	*49-state*
2007	93	76	93	75	0.4	0.4
2010	71	58	68	55	2.4	2.1
2015	60	49	55	44	5.4	4.8
2020	61	50	51	39	10.5	9.4
2030	64	52	49	37	15.6	14.0

Table II.B-16.b. Estimated Nationwide Exhaust NMHC Emissions from HDGVs Based on the Updated Control Case (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Reduction*	
			50-state	*49-state*
2007	76	76	0	0
2010	58	56	1.3	1.1
2015	49	45	4.4	3.9
2020	50	41	9.0	8.1
2030	52	37	15.0	13.5

c. Evaporative Emission Reductions

Evaporative HC emissions include diurnal, resting loss, refueling, and running loss emissions. To estimate evaporative emissions reductions from HDGVs, we used MOBILE5b to calculate percent reductions. We generated average national emission factors giving consideration to northern and southern regions of the country, fuel programs, inspection/maintenance programs, and time of year. This analysis uses the same methodology as was used in the inventory analysis for the Tier 2 light-duty vehicle standards.[162]

Figure II.B-7 presents the projected nonexhaust HC inventory with and without the new standards. We believe the new evaporative emissions standards would result in about a 12 percent reduction in nonexhaust HC from new heavy-duty gasoline vehicles. Tables II.B-17.a and II.B-17.b present these projections with the estimated evaporative emission reductions for selected years for the AQAC and Updated inventories.

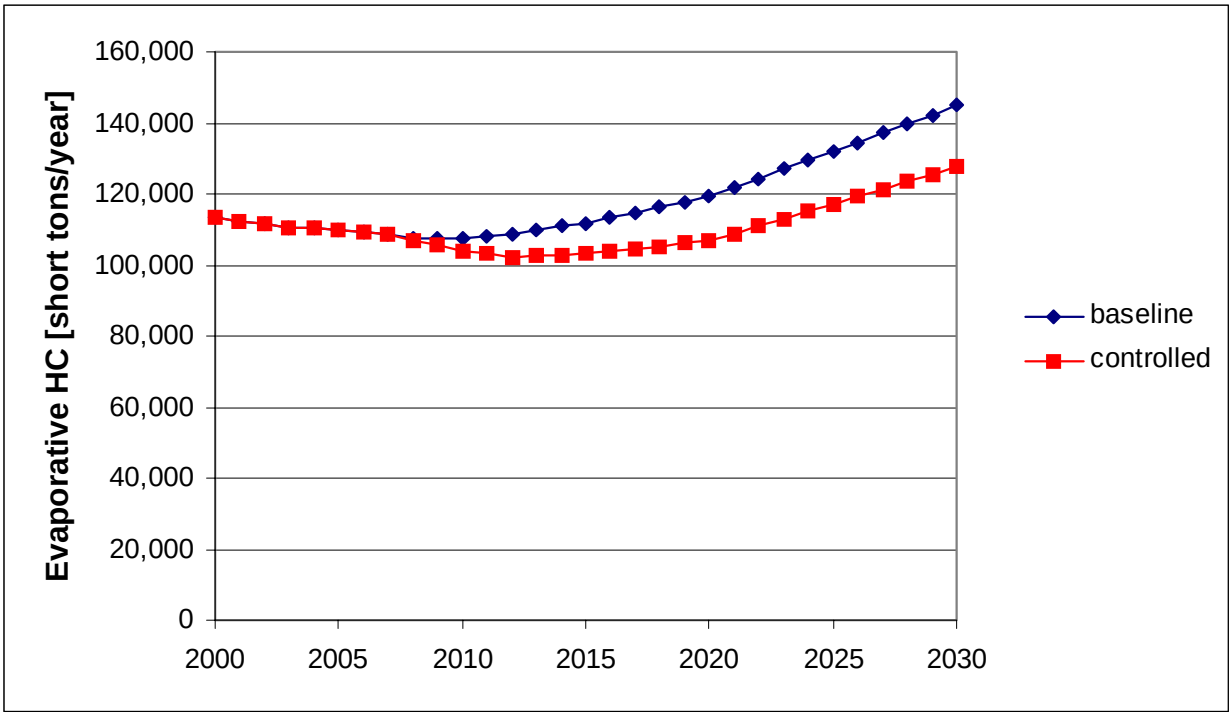

Figure II.B-7. Projected Nationwide Evaporative Emissions from HDGVs

Table II.B-17.a. Estimated Nationwide Evaporative Emissions from HDGVs Based on the Air Quality Analysis Case (thousand short tons per year)

Calendar Year	*Baseline*		*Controlled*		*Reduction*	
	AQAC	*w/o MDPVs*	*AQAC*	*w/o MDPVs*	*50-state*	*49-state*
2007	134	109	133	108	1	1
2010	132	107	126	102	6	5
2015	137	112	127	102	10	9
2020	146	119	133	106	13	12
2030	178	145	160	127	17	16

Table II.B-17.b. Estimated Nationwide Evaporative Emissions from HDGVs Based on the Updated Control Case (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Reduction*	
			50-state	*49-state*
2007	109	109	0.0	0.0
2010	107	104	3.4	3.0
2015	112	103	8.9	8.0
2020	119	107	12.2	10.9
2030	145	128	17.1	15.3

d. Air Toxics Reductions

The air toxics 49-state reductions for HDGVs are presented in Table II.B-18 for the *Air Quality Analysis Case* and the *Updated Control Case.*

Table II.B-18. Estimated 49-State Reductions in Air Toxics from HDGVs (thousand short tons per year)

Calendar Year	*Benzene*		*Formaldehyde*		*Acetaldehyde*		*1,3-Butadiene*	
	AQAC	*Updated*	*AQAC*	*Updated*	*AQAC*	*Updated*	*AQAC*	*Updated*
2007	0.03	0.00	0.02	0.00	0.00	0.00	0.00	0.00
2010	0.24	0.14	0.12	0.06	0.03	0.02	0.02	0.01
2015	0.41	0.36	0.25	0.20	0.07	0.06	0.04	0.03
2020	0.46	0.42	0.29	0.25	0.09	0.08	0.04	0.03
2030	0.68	0.66	0.50	0.48	0.16	0.15	0.07	0.07

4. Total Emission Reductions

Figures II.B-8 through II.B-10 present the total projected emissions of NOx, PM, and NMHC from heavy-duty engines with and without the new exhaust, evaporative, crankcase, and fuel sulfur standards. No reductions are assumed for HDGV PM. Tables II.B-19 through II.B-21

present the total NOx, PM, and NMHC benefits from heavy-duty engines that we anticipate from this rule. Evaporative emission reductions are included in the NMHC benefits. Table II.B-22 presents the total air toxics reductions. All of these projections are based on the *Updated Control Case*. Reductions in California are not included in the tables.

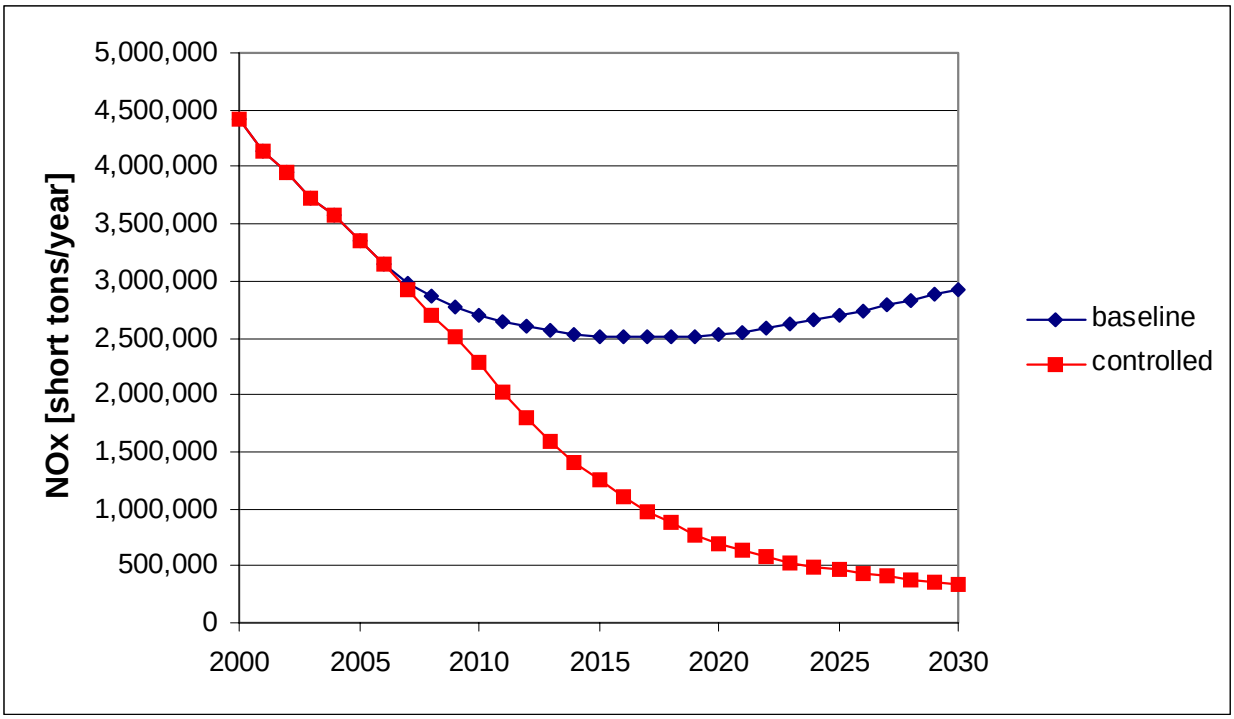

Figure II.B-8. Projected NOx Inventory for Heavy-Duty Highway Vehicles

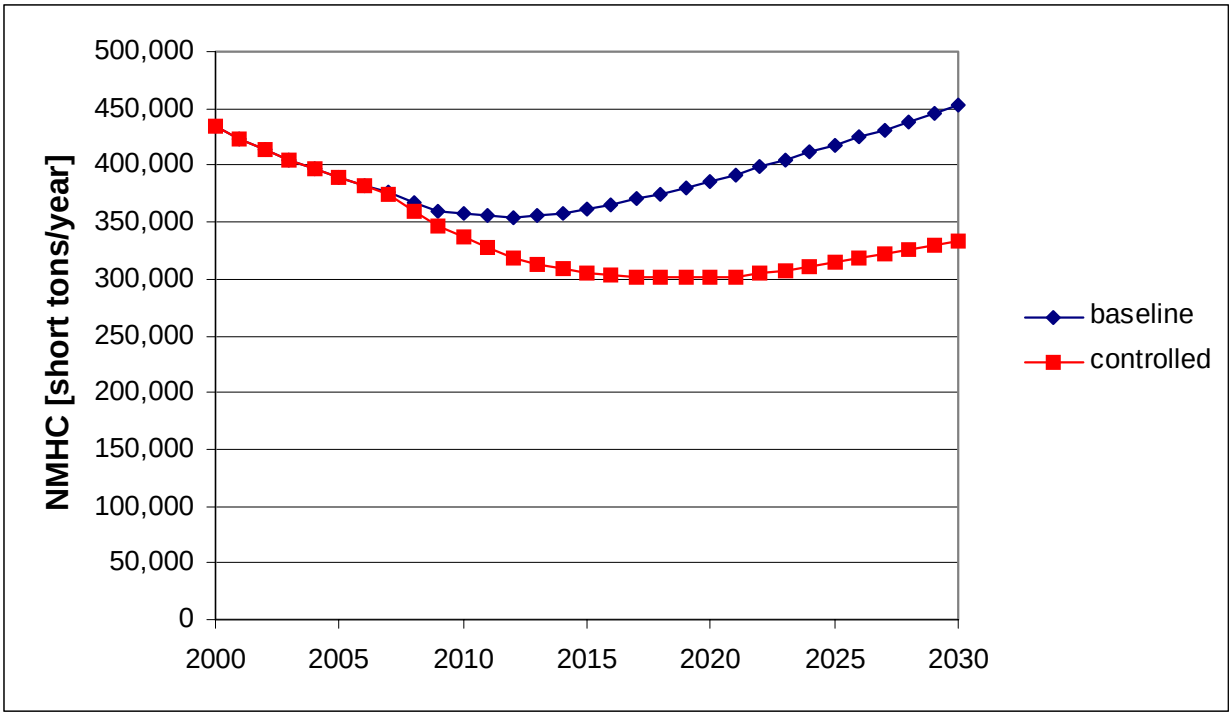

Figure II.B-10. Projected NMHC Inventory for Heavy-Duty Highway Vehicles

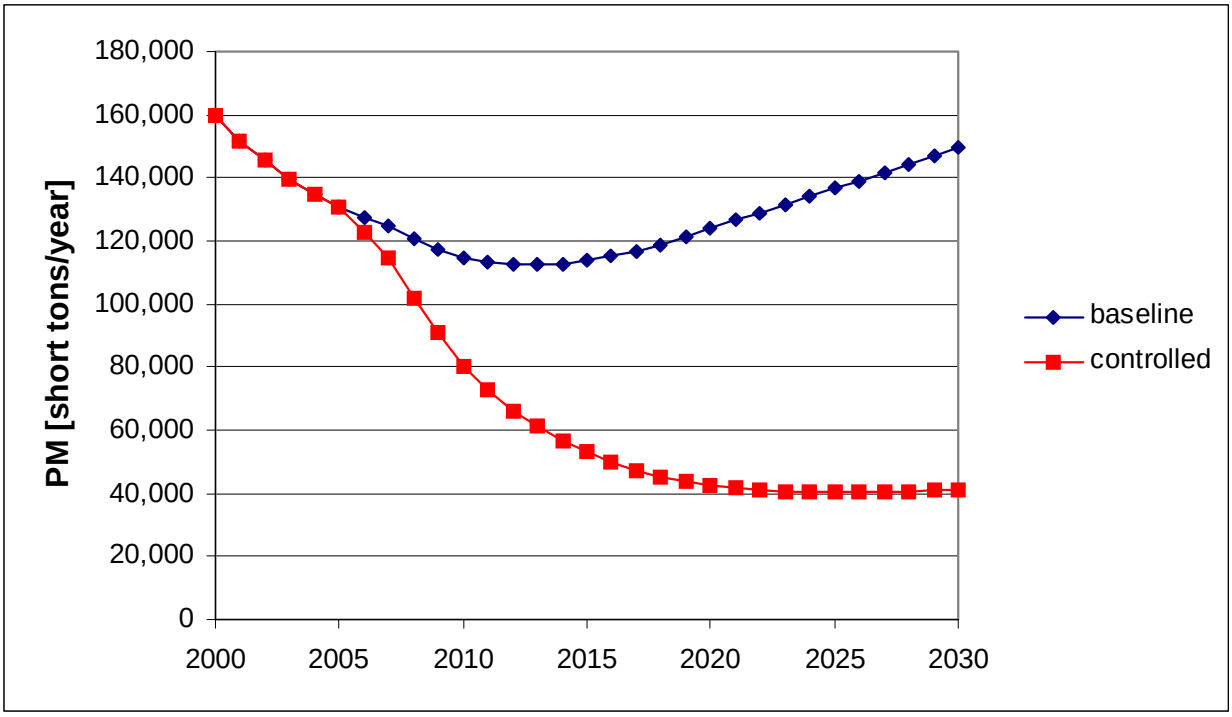

Figure II.B-9. Projected PM Inventory for Heavy-Duty Highway Vehicles

Table II.B-19. Total NOx Emissions and Benefits for This Rule (thousand short tons per year)

Calendar Year	*HDV Baseline*	*HDV Controlled*	*Reduction*
2007	2,970	2,910	58
2010	2,710	2,290	420
2015	2,520	1,250	1,260
2020	2,520	692	1,820
2030	2,930	346	2,570

Table II.B-20. Total PM Emissions and Reductions for This Rule (thousand short tons per year)

Calendar Year	*HDV Baseline*	*HDV Controlled*	*HDV Reduction*	*Other DSPM Reduction**	*Total Reduction*
2007	125	114	11	0.6	12
2010	115	79	35	0.6	36
2015	114	53	61	0.6	61
2020	124	42	82	0.7	82
2030	150	41	109	0.7	109

* From sources other than HDDEs using on-highway low sulfur fuel.

Table II.B-21. Total NMHC Emissions and Reductions for This Rule (thousand short tons per year)

Calendar Year	*HDV Baseline*	*HDV Controlled*	*Reduction*
2007	376	374	2
2010	358	337	21
2015	361	305	54
2020	386	301	83
2030	451	332	115

Table II.B-22. Total Reductions in Air Toxics for This Rule (thousand short tons per year)

Calendar Year	*Benzene*	*Formaldehyde*	*Acetaldehyde*	*1,3-Butadiene*
2007	0.02	0.18	0.07	0.01
2010	0.36	1.67	0.61	0.14
2015	0.96	4.72	1.72	0.38
2020	1.34	7.08	2.60	0.57
2030	1.96	10.2	3.73	0.82

5. Differences from NPRM Inventory

For the NPRM we used a simplified analysis to calculate emissions inventories and reductions from heavy-duty vehicles. For HDDEs we took a top-down approach to modeling emission inventories using a spreadsheet model. For HDGVs we used emission factors generated using a modified MOBILE5 model with the inputs of an average speed, average fuel, and summertime average temperature. Neither of these approaches were sophisticated enough to include county-by-county or hour-by-hour effects on the emission inventories. With that being said, these inventories have proven to be similar to the FRM inventories.

The FRM inventories (as discussed above) are based on complex and time consuming calculations in which emissions were summed in every county in the U.S. on an hourly basis. These inventories were developed for 1996, 2007, 2020, and 2030. Mathematically, this bottom-

up approach gives us more precise results than using national averages. In addition, it allows us to account for more specific effects of ambient conditions, roadway types, fuel parameters, and scrappage rates on HDV emissions.

In this section, we compare the baseline emissions inventories from the NPRM to those presented in the FRM. By looking at baseline inventories we can focus on the calculation methodology separate from the differences caused by the changes in the FRM standards from the proposed standards. The effects of these changes are described in detail earlier in this chapter. We believe that the small changes between the NPRM and FRM inventories reflect better analysis in the FRM inventory.

a. Heavy-Duty Diesel Engines

i. MOBILE Model (NOx, NMHC)

As discussed above, MOBILE5 was used with adjustment factors to calculate HDDE NOx and NMHC in the final rule. The primary difference between the NPRM and FRM analyses is that we only considered operation at a single average speed in the NPRM spreadsheet analysis; therefore, no speed correction was made. However, for the FRM analysis, emissions were calculated for twelve different roadway types. These roadway type distributions differed for each county. Based on the national average distribution of roadway types reported in the FRM analysis, we can roughly calculate the effect of speed on NOx and NMHC inventories. Using the MOBILE speed correction factors, we approximate that the weighted average national speed correction is -5 percent for NOx and -17 percent for NMHC.

Table II.B-23 compares the NPRM and FRM inventories for exhaust NOx and NMHC. We look at exhaust emissions only, because crankcase emissions are still calculated the same way in the FRM analysis as they were in the NPRM analysis. As shown in this table, most of the change from the NPRM to the FRM inventory is due to the application of speed correction factors. We also would expect variation between the two inventories due to the top-down versus bottom-up methodology as discussed earlier.

Table II.B-23. Comparison of NPRM and FRM HDDE Baseline Inventories for NOx and NMHC (thousand short tons per year)

Calendar Year	*Exhaust NOx*			*Exhaust NMHC*		
	NPRM	*FRM*	*% Change*	*NPRM*	*FRM*	*% Change*
2007	2,860	2,650	-7%	218	184	-16%
2020	2,600	2,350	-10%	249	206	-17%
2030	3,000	2,770	-8%	292	240	-18%

ii. PART Model (PM, SOx)

As discussed above, PART5 was used with adjustment factors to calculate HDDE PM and SOx in the final rule. Table II.B-24 shows very good correlation in exhaust PM and SOx between the NPRM and FRM inventories. In this case, the FRM results are less sensitive to roadway distribution because PART does not apply speed correction factors to PM and SOx. We look at exhaust PM because the NPRM inventory did not include brake and tire wear. In addition, we use the same analysis methodology to calculate crankcase emissions in the NPRM and FRM.

Table II.B-24. Comparison of NPRM and FRM HDDE Baseline Inventories for Exhaust PM and SOx (thousand short tons per year)

Calendar Year	*Exhaust PM*			*Exhaust SOx*		
	NPRM	*FRM*	*% Change*	*NPRM*	*FRM*	*% Change*
2007	92	96	4%	91	92	2%
2020	88	86	-2%	112	116	4%
2030	106	104	-2%	126	130	4%

b. Heavy-Duty Gasoline Vehicles

As with the HDDE final analysis, the FRM bases the HDGV NOx and NMHC emissions inventories on the MOBILE5 model with adjustment factors. In this case, the NPRM was also based on MOBILE5 model runs with adjustments to the model year emission factors entered into the model. However, the NPRM analysis was run for a typical summer day and for a single speed of 20 mph. In addition, the NPRM did not consider the effects of inspection/maintenance

or reformulated fuel programs. As a result, we saw similar results as with the Tier 2 Inventory Analysis in which NMHC decreased noticeably with the county-by-county, hour-by-hour analysis compared to the more simple top down analysis.

Table II.B-25 compares the FRM and NPRM baseline inventories for NOx and NMHC. To make a direct comparison, we adjusted the NPRM inventory to be a national inventory rather than just a 49-state inventory. We believe that the differences in the inventories reflect more precise calculations in the county level analysis which results in a better inventory for the FRM.

Table II.B-25. Comparison of NPRM and FRM HDGV Baseline Inventories for NOx and NMHC (thousand short tons per year)

Calendar Year	*Exhaust NOx*			*Exhaust and Evaporative NMHC*		
	NPRM	*FRM*	*% Change*	*NPRM*	*FRM*	*% Change*
2007	307	310	1%	216	185	-15%
2020	159	163	2%	196	169	-14%
2030	138	143	3%	230	197	-14%

6. Sensitivity Analysis for In-Use PM Deterioration

In our analysis of the HDDE emissions inventory, we may underestimate emissions, especially PM, due to engine deterioration in-use. We believe that current modeling represents properly maintained engines, but may not be representative of in-use tampering or malmaintenance. However, data related to this issue is extremely limited and inconclusive and we are in the process of collecting more data on in-use emission deterioration. Once this has been completed we will be better able to decide whether or not we need to update our deterioration rates. If we do update our deterioration rates, we will do so through a similar public process as we are using to create the MOBILE6 model.

Although a substantial amount of work remains before we can update our deterioration factors, we believe it is valuable to get a feel for the potential effects of in-use tampering and malmaintenance on the PM emissions inventory. In this section, we present a sensitivity analysis of these effects.

a. Methodology

Engine, Fuel, and Emissions Engineering, Inc. recently performed a study which suggests that tampering and malmaintenance result in large increases in in-use PM emissions from heavy-

duty diesel engines.[163] The California Air Resources Board (ARB) uses the underlying data in this report in developing its in-use deterioration rates for the EMFAC2000 emission model.[164] The ARB HDDE deterioration rates are presented in Table II.B-26 and are compared to the deterioration factors used in our inventory analysis. No deterioration is assumed for urban buses.

To perform our sensitivity analysis, we use the ARB deterioration rates and the NPRM spreadsheet model to determine the increases in the HDDE exhaust PM inventory due to tampering and malmaintenance. We then applied these increases to the exhaust PM inventory presented above. For 2007 and later model year engines, we assumed that the ratio of the deterioration rate to the emission standard is the same as for 2004 model year engines.

Table II.B-26. Comparison of EMFAC2000 and EPA PM Deterioration Rates for HDDEs (grams per mile per 10,000 miles)

Model Year	*EMFAC2000*			*EPA Analysis*		
	LHDDE	*MHDDE*	*HHDDE*	*LHDDE*	*MHDDE*	*HHDDE*
Pre 1976	0.003	0.016	0.016	0.000	0.000	0.000
1977-79	0.003	0.016	0.017	0.000	0.000	0.000
1980-83	0.004	0.016	0.018	0.000	0.000	0.000
1984-86	0.004	0.021	0.012	0.000	0.000	0.000
1987	0.005	0.017	0.008	0.000	0.000	0.000
1988-99	0.005	0.017	0.008	0.001	0.004	0.005
1990	0.005	0.017	0.008	0.000	0.000	0.000
1991-93	0.002	0.022	0.009	0.000	0.002	0.000
1994-97	0.003	0.018	0.010	0.000	0.002	0.000
1998	0.003	0.012	0.007	0.000	0.000	0.000
1999-02	0.001	0.012	0.003	0.000	0.000	0.000
2003	0.001	0.009	0.003	0.000	0.000	0.000
2004	0.001	0.009	0.003	0.000	0.000	0.000

b. Results

If we consider the EMFAC2000 deterioration rates presented in Table II.B-26, we see an increase of over 50 percent in the HDDE exhaust PM emission inventory compared to the results from the *Updated Control Case*. In 2030, we see an increase in the baseline PM inventory of 48 percent and an increase in the controlled PM inventory of 63 percent. This translates to an exhaust PM reduction of 141,000 tons in 2030 due to the new standards compared to the 96,000 ton PM reduction when tampering and malmaintenance were not considered. Figure II.B-11 presents the exhaust PM inventory with ("High") and without ("FRM") considering tampering and malmaintenance.

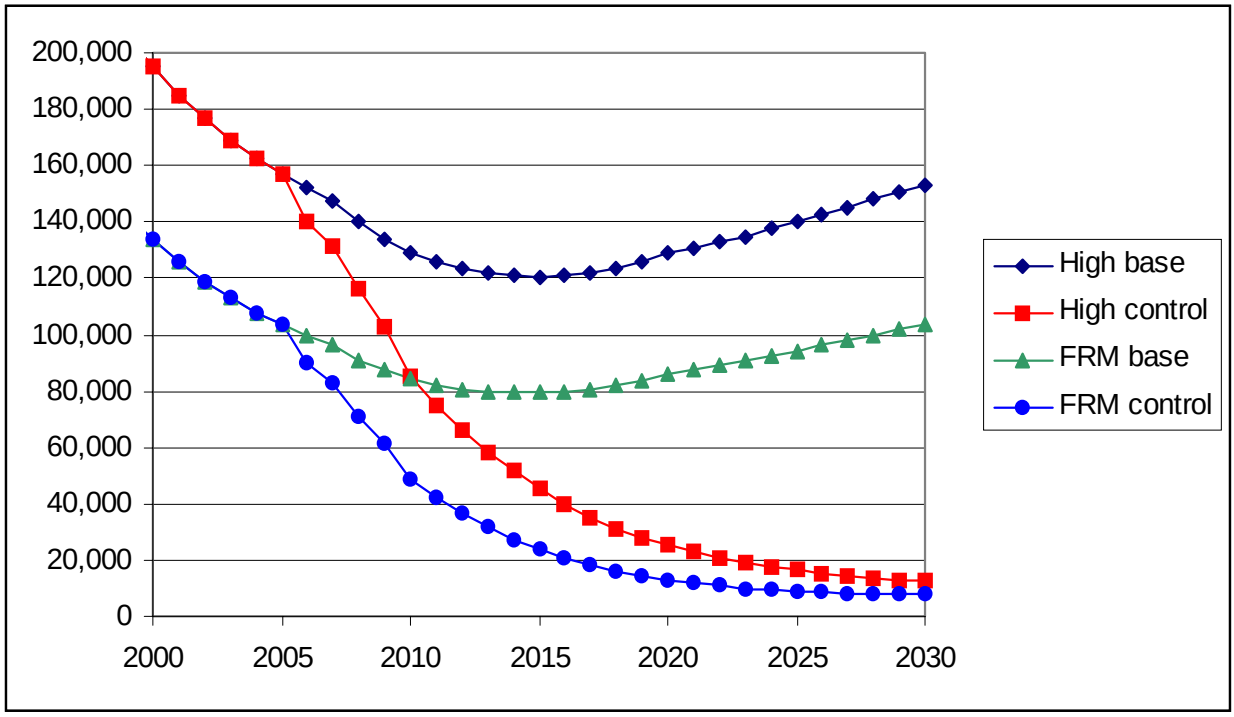

Figure II.B-11. Projected HDDE Exhaust PM Inventory with and without Consideration of Tampering and Malmaintenance

7. Contribution of HDVs to National Inventory

Nationwide, heavy-duty vehicles are projected to contribute about 15 percent of the total NOx inventory, and 28 percent of the mobile source inventory in 2007. Heavy-duty NOx emissions also contribute to fine particulate concentrations in ambient air due to the transformation in the atmosphere to nitrates. The NOx reductions resulting from today's standards will therefore have a considerable impact on the national NOx inventory. All highway vehicles account for 34 percent and heavy-duty highway vehicles account for 20 percent of the mobile source portion of national PM_{10} emissions in 2007. These inventories are based on the analysis performed by Pechan used for the air quality modeling analysis.[165] [166] Because this inventory analysis does not include stationary source emissions from Alaska and Hawaii, Tables II.B-27 through II.B-29 present emissions inventories for the 48 contiguous states.

Table II.B-27. 2007 Baseline Emissions Inventories for 48 Contiguous States (thousand short tons)

Source	*VOC*	*NOx*	*SO_2*	*PM**	*CO*
Heavy-Duty Vehicles	415 (3%)	3,030 (15%)	94 (1%)	126 (4%)	3,850 (4%)
Light-Duty Vehicles	2,600 (18%)	2,950 (14%)	25 (0%)	82 (3%)	39,300 (42%)
Nonroad	2,120 (15%)	4,710 (23%)	1,040 (6%)	408 (14%)	27,200 (29%)
Other	9,140 (64%)	9,890 (48%)	15,900 (93%)	2,210 (78%)	22,600 (24%)
Total	14,300	20,600	17,100	2,830	92,900

* excludes natural and miscellaneous sources

Table II.B-28. 2020 Baseline Emissions Inventories for 48 Contiguous States (thousand short tons)

Source	*VOC*	*NOx*	*SO_2*	*PM**	*CO*
Heavy-Duty Vehicles	420 (3%)	2,550 (14%)	118 (1%)	126 (4%)	4,720 (4%)
Light-Duty Vehicles	1,800 (13%)	1,310 (7%)	31 (0%)	100 (3%)	44,600 (42%)
Nonroad	2,000 (14%)	4,040 (23%)	1,310 (8%)	450 (15%)	33,900 (32%)
Other	10,100 (70%)	9,980 (56%)	14,500 (91%)	2,380 (78%)	23,700 (22%)
Total	14,300	17,900	16,000	3,060	107,000

* excludes natural and miscellaneous sources

Table II.B-29. 2030 Baseline Emissions Inventories for 48 Contiguous States (thousand short tons)

Source	*VOC*	*NOx*	*SO_2*	*PM**	*CO*
Heavy-Duty Vehicles	491 (3%)	2,940 (16%)	133 (1%)	152 (5%)	5,730 (5%)
Light-Duty Vehicles	1,950 (12%)	1,250 (7%)	35 (0%)	114 (3%)	51,200 (42%)
Nonroad	2,230 (14%)	4,320 (23%)	1,490 (9%)	511 (15%)	39,200 (33%)
Other	11,000 (70%)	10,200 (55%)	14,800 (90%)	2,560 (77%)	24,400 (20%)
Total	15,700	18,700	16,400	3,340	120,000

* excludes natural and miscellaneous sources

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