Chapter II: Health and Welfare Concerns and Emissions Benefits

This chapter describes the public health and welfare concerns associated with the pollutants impacted by this rulemaking, and the emission reductions that are expected to occur as a result of the proposed new standards for heavy-duty vehicles. In addition, the results of our analysis of heavy-duty vehicle inventory levels with and without the proposed new standards are presented and discussed 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 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 would take effect. In addition, some studies have found public health and welfare effects from ozone and fine PM at concentrations that do not constitute a violation of their respective NAAQS. Other studies have associated diesel exhaust with a variety of 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. The standards proposed in this proposal would result in a significant improvement in ambient air quality and public health and welfare.

1. 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 in 2007 or later. Information on air quality was gathered from a variety of sources, including monitored ozone concentrations from 1995 to 1998, air quality modeling forecasts conducted for the recently-promulgated Tier 2 rule, 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 proposal may take effect.

a. Health and Welfare Effects of Ozone

Ground-level ozone, the main ingredient in smog, is formed by complex chemical reactions of volatile organic compounds (VOC) and nitrogen oxides (NOx) in the presence of heat and sunlight. Ozone forms readily in the lower atmosphere, usually during hot summer weather. VOCs are emitted from a variety of sources, including motor vehicles, chemical plants, refineries, factories, consumer and commercial products, and other industrial sources. VOCs also are emitted by natural sources such as vegetation. NOx is 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, small amounts of NOx enable ozone to form rapidly when VOC levels are 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 low can be NOx limited.

When NOx levels are high and VOC levels relatively low, NOx forms inorganic nitrates but 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. The highest levels of ozone are produced when both VOC and NOx emissions are present in significant quantities.

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.

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

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, 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, chronic bronchitis and chronic asthma.

Children 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.

VOC 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. (See Section II.A-5).

b. General Description of the Tier 2 Ozone Modeling

The Agency believes that there is a significant risk that an appreciable number of areas will violate the 1-hour ozone NAAQS during the time when these proposed standards would apply to HD vehicles. This is based, in part, on the air quality modeling performed during the Tier 2 rulemaking, and assumes reductions from Tier 2 and other controls currently in place. This subsection describes the methods used in the Tier 2 air quality modeling analysis.

In its Tier 2/Sulfur rulemaking efforts, the Agency performed ozone air quality modeling for nearly the entire Eastern U.S. covering metropolitan areas from Texas to the Northeast, and for a western U.S. modeling domain. In addition, the Agency reviewed ozone attainment modeling and other evidence covering 15 of these areas (see Table II.A-2) from State Implementation Plan (SIP) submittals or from modeling underway to support SIP revisions. The local modeling only addressed

the current and requested attainment date in each area. Based on the air quality modeling and local information compiled and reviewed during the Tier 2 rulemaking process, the Agency made attainment and nonattainment predictions.

In the Tier 2 needs assessment, a series of air quality modeling simulations were completed to support, among other things, a determination of the need for additional emissions reductions in order to meet the 1-hour ozone NAAQS and to assess the impact of the Tier 2/Sulfur rule on future ozone levels. The model simulations were performed for five emissions scenarios: a 1996 base year, a 2007 baseline projection, a 2007 projection with Tier 2/Sulfur controls, a 2030 baseline projection, and a 2030 projection with Tier 2/Sulfur controls.

In conjunction with current air quality data, the model output from the 2007 and 2030 baselines was used to identify areas expected to exceed the ozone NAAQS in 2007 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 Tier 2/Sulfur controls were determined by comparing the model results in the future year control runs against the National Low Emission Vehicle Program/high sulfur baseline simulations of the same year.

Modeling Methodology

A variable-grid version of the Urban Airshed Model (UAM-V) was utilized to estimate base and future-year ozone concentrations over the continental 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 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 Tier 2 Air Quality Modeling Technical Support Document (TSD), which is located in the docket for this proposal.

Modeling domains

Two separate modeling domains were utilized in the Tier 2/Sulfur analyses. The first covered that portion of the U.S. east of west longitude 99 degrees. The second covered the remainder of the U.S. west of west longitude 99 degrees. The model resolution was 36 km over the outer portions of each domain and 12 km in the inner portion of the grids. A recent modeling study (LADCO, 1999) considered the sensitivity of regional modeling strategies to grid resolution. 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)[b] modeling application also investigated the effects of grid resolution on national/regional control strategies (e.g., Tier 2/Sulfur). 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 proposal.

Modeling episodes

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.[c]. In general, these episodes do not represent extreme ozone events but, instead, are generally representative of ozone levels near local design values. Five simulations were completed for the June and July episodes (1996 base, 2007 baseline, 2007 control, 2030 baseline, 2030 control). Three simulations were completed for the August episode (1996 base, 2007 baseline, 2007 control).

Two episodes were modeled for the western U.S. domain: 5-15 July 1996 and 18-31 July 1996. Again, these 19 days contained design value level ozone exceedances over most of the western U.S. allowing for an assessment of emission controls in polluted, but not infrequent, conditions. The primary purpose of simulating the western episodes was to provide data for the benefits/cost analysis for 2030. Thus, no 2007 simulations were made for the West.

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, and the Fifth-Generation National Center for Atmospheric Research (NCAR) / Penn State University (PSU) Mesoscale Model (MM5) for the western U.S. 1996 episodes. These models provided needed data at every grid cell on an hourly basis. These meteorological modeling results were evaluated 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.

[b] The OTAG modeling project is used as a benchmark for the Tier 2/Sulfur 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.

[c] 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.

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 Tier 2 Air Quality Technical Support Document, which is available in the docket to this proposal on heavy-duty vehicles.

Model performance evaluation

The purpose of the Tier 2/Sulfur 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 ozone modeling is that a model which closely replicates observed ozone in the base year can be used to support future-year policymaking.

As with previous regional photochemical modeling studies, the accuracy of the Tier 2/Sulfur model 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 regional-scale ozone trends fairly closely.

The values of two primary measures of model performance, mean normalized bias and mean normalized gross error, indicate that the Tier 2/Sulfur modeling over the eastern U.S. is generally as good or better than the grid modeling done for OTAG[d], as shown in Table II.A-1. As OTAG did not perform any modeling for the West, no comparison back to OTAG is possible for the Tier 2 western U.S. model performance. Mean normalized bias is defined as the average difference between model predictions and observations (paired in space and time) normalized by the observations. Mean gross error is defined as the average absolute difference between model predictions and observations, paired in space and time, normalized by the observations. EPA guidance on local ozone attainment demonstration modeling (not the purpose of the Tier 2 modeling) suggests biases be less than 5-15 percent and error be less than 30-35 percent.

[d] Again, the OTAG application is used as a relative benchmark for model performance because it is the most detailed modeling to date over this region.

Table II.A-1 Comparison of eastern U.S. regional model performance statistics between the Ozone Transport Assessment Group (OTAG) modeling used to support the NO_X SIP call and the Tier 2/Sulfur modeling. The units are percentages.

Mean Normalized Bias	*OTAG 1988 Episode*	*OTAG 1991 Episode*	*OTAG 1993 Episode*	*OTAG 1995 Episode*	*Tier 2 June 95 Episode*	*Tier 2 July 95 Episode*	*Tier 2 August 95 Episode*
Domain	-8	-4	+1	+4	-10	-6 (-4)[e]	+2
Midwest	-15	-8	-8	-5	-11	-13 (-8)	+7
Northeast	-3	-6	-8	+8	-17	-9 (-9)	-9
Southeast	+2	+15	+21	+9	-4	+4 (+5)	+7
Southwest	-6	+6	+2	+12	+2	+8 (+8)	+6

Mean Normalized Gross Error	*OTAG 1988 Episode*	*OTAG 1991 Episode*	*OTAG 1993 Episode*	*OTAG 1995 Episode*	*Tier 2 June 95 Episode*	*Tier 2 July 95 Episode*	*Tier 2 August 95 Episode*
Domain	28	25	27	25	24	24 (24)	23
Midwest	27	26	25	24	24	26 (25)	22
Northeast	29	23	23	26	27	22 (21)	24
Southeast	28	25	32	27	20	24 (24)	22
Southwest	22	24	23	29	24	27 (26)	24

In general, the model underestimates ozone for the June and July eastern episodes in 1995 and, especially, both western episodes in 1996. The under prediction bias in the western U.S. modeling averages about 40 percent. The model is slightly biased toward overestimation in the August 1995 eastern episode. Although the overall tendency is to underestimate the observed ozone, there are several instances in which overestimations occurred. The net effect is expected to be an underestimate of the total extent of future-year exceedances, although some individual areas may be overstated.

c. Factors in Attainment/Nonattainment and Maintenance Discussion

Tables II.A-3 and A-4 list those metropolitan areas that EPA believes may need additional emission reductions in order to reduce the risk of failing to attain or maintain the 1-hour ozone NAAQS. This belief is based on an analysis performed for the Tier 2 rulemaking. The Tier 2 determination 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

[e] Values in parentheses are for the 10-15th only. These dates correspond with OTAG episode days.

maintenance. Those interested in greater detail should review the Tier 2 Air Quality Modeling Technical Support Document, which is available in the docket to this proposal on heavy-duty vehicles.

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. For the purposes of defining the current design value of a given area, the higher of the 1995-1997 and 1996-1998 design values were chosen to provide greater confidence in identifying areas likely to have an ozone problem in the future. The 1995-1997 and 1996-1998 design values are listed in the Tier 2 Air Quality Modeling Technical Support Document, which is available in the docket to this proposal on heavy-duty vehicles.

Method for projecting future exceedances

The exceedance method was used for interpreting the future-year modeling results to determine where nonattainment is expected to occur in the 2007 and 2030 Base Cases[f]. 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.

[f] 2030 is the relevant baseline scenario for the western U.S. domain

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

Overview

We have reviewed and recently proposed action on SIP submissions from 14 States covering 10 serious and severe 1-hour ozone nonattainment areas. We received these submissions as part of the three-phase SIP process allowed 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 described above. We have also considered ozone modeling submitted as part of an attainment date extension request for Beaumont-Port Arthur, TX, but have not yet taken action on that request. We have also reviewed a status report on the results of modeling being conducted in anticipation of submittal to EPA as part of an extension request for Dallas, TX. Finally, we have considered information in the most recent SIP submittal from California for the South Coast Air Basin. Table II.A-2 lists the areas involved, our overall conclusion as to whether the modeling demonstrates attainment without further reductions in addition to those obtained under the Tier 2 program, the Federal Register citation for our proposed action if applicable. This section discusses the background for the submissions and our conclusions from them.

It is important to note that the information contained in this section on current and future ozone nonattainment is current as of March 15, and there may have been recent developments in some areas that are not incorporated here. We will update this information for the final rulemaking document.

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. This fine grid size is an important factor in how much weight we have given to this set of evidence. 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, the selection of episode days met our guidance. The local modeling also may make use of more information on the local emission inventory and control program than is impracticable to include in broad scale modeling by EPA as described above.

The SIP submissions for these 14 States 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-2
Nonattainment Areas For Which EPA Has Recently 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*	*Demonstrates Attainment Without "Further Reductions"*	*Proposed for Action in December 16, 1999 Federal Register (64 FR 70318)*
Western Massachusetts (Springfield)	MA	2003 (Requested Extension)	Yes	Yes
Greater Connecticut (Hartford and other MSAs)	CT	2007 (Requested Extension)	Yes	Yes
New York City	NY, CT, NJ	2007	No	Yes
Philadelphia	PA, NJ, DE, MD	2005	No	Yes
Baltimore	MD	2005	No	Yes
Washington, D.C.	MD, VA, D.C.	2005 (Requested Extension)	Yes (with Tier 2)	Yes
Atlanta	GA	2003 (Requested Extension)	No	Yes
Houston	TX	2007	No	Yes
Chicago*	IL, IN	2007	Yes	Yes
Milwaukee*	WI	2007	Yes	Yes
Benton Harbor*	MI	N/A	Yes	No
Grand Rapids*	MI	Not Applicable	Yes	No
Dallas	TX	2007 (Requested	No	No

Nonattainment Area (Major Metro Area)	*Affected States*	*Attainment Date*	*Demonstrates Attainment Without "Further Reductions"*	*Proposed for Action in December 16, 1999 Federal Register (64 FR 70318)*
		Extension)		
Beaumont-Port Arthur	TX	2007 (Requested Extension)	No	Yes
South Coast Air Basin	CA	2010	No	No

*Revised modeling in progress.

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.[7] 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[g] 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

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

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.

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.[h] For the purpose of this proposal, 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.

Conclusions from the Local Modeling in SIP Submittals

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 proposal 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 beyond those predicted from the Tier 2 program.

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); and Milwaukee, WI areas demonstrated attainment through the control measures contained in the submitted attainment strategy. [i] We expect that Illinois, Wisconsin, and Indiana will submit further SIP revisions for Chicago and Milwaukee prior to our taking final action on our recent proposals regarding the submissions they made earlier. However, these new revisions will be based on a new round of modeling conducted by the Lake Michigan Air Directors Consortium (LADCO) on behalf of the States. While we have not received this modeling, we have received a progress report on it.[8] As described in greater detail in the following section, the Agency expects to rely in part on the reductions from the proposed new standards for heavy-duty vehicles 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. The reliance on these new controls will also impact our judgement about the future attainment prospects of Benton Harbor and Grand Rapids-Muskegon areas.

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[h] 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.

[i] Revised local modeling may affect this situation (see section e for details).

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, our weight of evidence analysis indicates that the Tier 2/Sulfur program is likely to provide all of the additional emission reductions 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.

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. However, many of the measures in that strategy are not yet adopted or fully committed. 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 were considered. The modeling for Atlanta assumed implementation of the NOx SIP Call outside the local modeling domain, but lesser NOx reductions within the domain. The difference in NOx reduction within the modeling domain is small, and it is apparent that even if the full NOx reductions from the SIP call had been assumed attainment would still not be demonstrated without reductions from measures which are additional reductions.

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

Other Local Ozone Modeling

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). The modeling analysis indicates nonattainment in 2007 under an emissions scenario that includes additional reductions.

We have also become aware of recent modeling by the State of Texas for the Dallas-Fort Worth metropolitan area.[10] Dallas continues to have concentrations above the 1-hour ozone standard after its 1999 attainment date, and Texas has made known its intent to seek an attainment date extension for this area. We have recently indicated to Texas that we will propose to approve its request for an attainment date extension to 2007, provided that the State can meet several necessary

conditions one of which is to demonstrate attainment by that date. The State is conducting modeling analyses to identify its options for reaching attainment in Dallas by 2007. This modeling has been made public. The modeling results to date indicate that even with the emission reductions expected from the Tier 2/Sulfur program, Dallas will be in nonattainment in 2007.

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. However, the air quality situation and a recent SIP revision for one area in California support the conclusion that there is an overall need for further reductions in order to attain and maintain.

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 30 million have 1996-1998 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, represents only a small fraction of the emission reductions needed in the South Coast to attain the NAAQS.

We expect that California will be submitting one or more revisions since it appears that some serious classification nonattainment areas in California with an attainment deadline of 1999 have not met that date. These areas are San Diego and the San Joaquin Valley. San Joaquin has had too many exceedances to be eligible for an extension and EPA has informally indicated its intent to bump-up the area to severe classification. San Diego might be eligible for a 1-year attainment date extension under the provisions of CAA section 181(a)(5). We have not yet received an indication of California's intention in this regard, or any modeling which assesses whether these areas can attain before 2004 relying only on baseline measures.

Attainment of the 1-hour standard in the South Coast Air Basin, Southeast Desert, Sacramento, and Ventura nonattainment areas by their future attainment dates (2010 for the South Coast, 2007 for Southeast Desert, and 2005 for Sacramento and Ventura) remains the goal of California and EPA, but will be a challenging task. The difficulty of the task is reflected in recent litigation and settlement negotiations over both the design and the implementation of the attainment plans in the South Coast, for example. The most recent State SIP update for the South Coast indicates that it still needs additional measures to reduce emissions of NOx and VOCs to attain the 1-hour standard in 2010.

e. Current and Future Exceedances of the 1-Hour Ozone Standard

EPA proposes to find that there is a significant risk to public health and welfare from elevated levels of ground-level ozone above the 1-hour NAAQS during the time period when this proposal would take effect, and that the reductions in oxides of nitrogen (NOx) and volatile organic compounds (VOCs) projected from the proposed new standards would benefit public health and

welfare by reducing such levels. This belief is based upon the Agency's recent and extensive ozone air quality modeling and analysis performed for the Tier 2 rulemaking, as well as a review of local ozone modeling and other local factors. Because ozone concentrations causing violations of the 1-hour ozone standard are well established to endanger public health and welfare, this information supports the proposed new standards for heavy-duty vehicles. In addition, there is a large body of scientific literature indicating health effects related to ozone exposures that do not constitute 1-hour violations. In the absence of this rule, we believe that prolonged exposure to moderate ozone levels can reasonably be anticipated to occur in the future.

i. Current and Future Nonattainment Status With the 1-Hour Ozone NAAQS

Exposure to levels of ozone that are not in compliance with the 1-hour ozone NAAQS are a serious public health and welfare concern. The following sections discuss the risk that areas of the country will exhibit ozone levels that fail to comply with this NAAQS.

Over the last decade, emissions have declined and national air quality has improved and for all six criteria pollutants, including ozone.[11] Some of the greatest emissions reductions have taken place in densely-populated urban areas, where emissions are heavily influenced by mobile sources such as cars and trucks. For example, VOC and NOx emissions in several urban areas in the Northeast declined by 15 percent and 14 percent from 1990 to 1996.[12] When ozone trends are normalized for annual weather variations between 1989 and 1998, they reveal a downward trend in the early 1990's followed by a leveling off, or an upturn in ozone levels, over the past several years in many urban areas.[13]

Despite impressive improvements in air quality over the last decade, present concentrations of ground-level ozone continue to endanger public health and welfare in many areas. As of December, 1999, 92 million people (1990 census) lived in 32 metropolitan areas designated nonattainment under the 1-hour ozone NAAQS.[14] In addition, there are 14 areas with a 1996 population of 17 million people not currently listed as non-attainment areas because the 1-hour ozone standard was revoked for these areas (we have proposed to re-instate the standard).[15] These 14 areas are relevant to this proposal because ozone concentrations that violate the health-based ozone standard endanger public health and welfare independent of the applicability of the 1-hour standard or an area's official attainment or nonattainment status. Ozone also has negative environmental impacts. For example, exposure of vegetation to ozone can inhibit photosynthesis, and alter carbohydrate allocation, which in turn can suppress the growth of crops, trees, shrubs and other plants.

While there are still many areas where recent ozone levels have caused violations of the 1-hour ozone NAAQS, federal and State programs and policies continue to move the nation toward cleaner air. In all cases where violations of the 1-hour NAAQS have recently been observed, there are statutory and regulatory obligations that apply and processes in operation – or that will be in operation again assuming the 1-hour NAAQS is restored – to move areas with violations of the 1-hour ozone standard into attainment. Later portions of this section review more specifically what

obligations and processes apply to which areas. Many States are required to get EPA approval for SIPs which will show that their nonattainment areas will achieve the 1-hour NAAQS for ozone, either by the deadlines established in the Clean Air Act or by an extended deadline.[j] Some areas will need to submit attainment demonstrations if the ozone standard is reinstated. For other areas, there is no specific attainment date and associated requirement for an attainment demonstration, but a general requirement to provide for attainment and maintenance does apply and EPA may take action to require a State to amend its SIP or implement it fully. In addition, there are the 14 previously cited areas with recent violations for which the standard does not currently apply. Most of these areas have maintenance plans, and assuming the standard is restored in these areas those with recent violations of the 1-hour ozone NAAQS standard will need to implement any contingency measures in their maintenance plans that are triggered by such violations.

EPA believes that there is a significant risk that despite efforts by EPA and States to reach attainment through SIPs and to continue to attain through implementation of maintenance plans, some areas will experience violations of the 1-hour NAAQS during the time period when this proposal would achieve its emission reductions. Our belief regarding the risk of future violations of the 1-hour NAAQS is based upon our consideration of predictive ozone air quality modeling and analysis we performed for U.S. metropolitan areas for the recent Tier 2 rulemaking as well as the predictive ozone modeling and other information that has come to us through the SIP process and other local air quality modeling for certain areas. We have assessed this information in light of our understanding of the factors that influence ozone concentrations, the challenges and uncertainties in ozone air quality planning and implementation, and the uncertainties inherent in all predictive ozone modeling.

The next four sections present lists of areas for which the Agency has reason to believe may experience violations of the ozone standard in the future. The first section presents information in two tables. The first table lists 33 areas that were predicted by Tier 2 modeling to have exceedances in either 2007 or 2030, and the second table lists nine areas for which we have other evidence of a risk of future exceedances. The second through fourth sections discuss the air quality prospects for these 42 areas, which are divided into three groups based on the similarity of their situations. The second section examines the 10 areas that have statutorily-defined attainment dates of 2007 or 2010, and their need for additional reductions in order to attain, and then maintain, the ozone standard. Some of these areas have requested attainment date extensions to 2007 (including two requests on which we have not yet proposed any action). The third section examines the air quality prospects of a list of 26 areas that have in common that they have recently exceeded the standard, and have attainment deadlines prior to 2007 (or no specific attainment deadline). The fourth section examines the prospects of six areas with predicted exceedances in 2007 or 2030, and recent (1995-1998) ozone design values within 10% of the ozone standard.

[j] Some States have petitioned the Agency for additional time to attain the standard, either through a one-year extension, an extension based on overwhelming transport from an upwind area with a later attainment date, or an extension based on the timing of regional reductions in NOx emissions. Some areas face reclassification to a higher ozone classification, with a later attainment deadline.

ii. Ozone predictions made in the Tier 2 rulemaking and other information on ozone attainment prospects

In conjunction with its Tier 2 rulemaking efforts, the Agency performed ozone air quality modeling for nearly the entire Eastern U.S covering metropolitan areas from Texas to the Northeast, and for a western modeling domain. The ozone modeling we did as part of the Tier 2 rulemaking predicted that without further emission reductions, a significant number of areas now experiencing ozone violations across the nation are at risk of failing to meet the 1-hour ozone NAAQS in 2007 and beyond, even with Tier 2 and other controls currently in place.

The general pattern observed from the Tier 2 ozone modeling is a broad reduction between 1996 and 2007 in the geographic extent of ozone concentrations above the 1-hour NAAQS, and in the frequency and severity of exceedances. Despite this improvement from 1996 to 2007, many ozone exceedances were predicted to occur in 2007 and 2030 even with reductions from Tier 2 standards and other controls currently in place, affecting 33 areas across the nation. Assuming no additional emission reductions beyond those that will be achieved by current control programs,[k] a slight decrease below 2007 levels in modeled concentrations and frequencies of exceedances was predicted for 2030 for most areas. Exceedances were still predicted in 2030 in most of the areas where they were predicted in 2007.[l]

Although we did not model ozone concentrations for years between 2007 and 2030, we expect that they would broadly track the national emissions trends. Based on these emission trends alone, national ozone concentrations, on average, would be projected to decline after 2007, but begin to increase around 2015 or 2020 due to economic growth until they reach the 2030 levels just described. However, the change in ozone levels from the expected NOx reduction is relatively small compared to the effects of variations in ozone due to meteorology. Furthermore, in some areas, where growth exceeds national averages, emissions levels would begin increasing sooner and reach higher levels in 2030.

Table II.A-3 lists the 33 areas with predicted 1-hour ozone exceedances in 2007 and/or 2030 based on the Tier 2 modeling, after accounting for the emission reductions from the Tier 2 program and other controls.[m] There are areas that are not included in this table that will be discussed shortly.

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[k] Current control programs assumed for the predictions summarized here included the Tier 2/Gasoline Sulfur program and some specific programs that are legally required but not yet fully adopted, such as the Regional Ozone Transport Rule and not-yet-adopted MACT standards that will affect VOC emissions.

[l] Achieving attainment with the ozone standard is only one measure of air quality improvement. EPA found that the Tier 2 program significantly lowers the model-predicted number of exceedances of the ozone standard by one tenth in 2007, and by almost one-third in 2030 (Tier 2 RIA).

[m] Table II.A-3 excludes areas for which the Tier 2 modeling predicted exceedances in 1996 but for which the actual ozone design values in 1995-1997 and 1996-1998 were both less than 90 percent of the NAAQS. For these areas, we considered the ozone model's predictions of 2007 or 2030 exceedances to be too uncertain to play a

A factor to consider with respect to the ozone predictions in Table II.A-3 is that recent improvements to our estimates of the current and future mobile source NOx inventory have resulted in an increase in our estimate of aggregate NOx emissions from all sources by about eight percent since the air quality modeling performed for the Tier 2 rule. The adjusted NOx inventory level in 2015 is greater than the NOx inventory used in the Tier 2 air quality analysis for 2030. If we were to repeat the ozone modeling now for the 2015 time frame, using the new emissions estimates, it is would likely predict exceedances in 2015 for all the areas that had 2030 exceedances predicted in the modeling done for the Tier 2 rulemaking. As summarized in Table II.A-3, the Tier 2 modeling predicted that there will be 33 areas in 2007 or 2030 with about 89 million people predicted to exceed the 1-hour ozone standard, even after Tier 2 and other controls currently in place.

supportive role in our rulemaking determinations. Also, 2007 ozone was not modeled for western areas. For 2030, all areas were modeled for fewer episode days which, along with a general model under-prediction bias, may result in an underestimation of 2030 exceedances. Without these factors, there could have been more western areas listed in Table II.A-3, and more areas with predicted exceedances in 2030.

Table II.A-3:
Metropolitan Areas with Predicted Exceedances in 2007 or 2030 from Tier 2 Air Quality Modeling With Emission Reductions from Tier 2 and Other Current/Committed Controls

CMSA/MSAs	*2007 Control Case*	*2030 Control Case*	*1996 Population*
Boston, MA CMSA	X	X	5.6
Chicago, IL CMSA	X	X	8.6
Cincinnati, OH	X		1.9
Cleveland, OH CMSA*	X	X	2.9
Detroit, MI CMSA*	X	X	5.3
Houston, TX CMSA	X	X	4.3
Milwaukee, WI CMSA	X	X	1.6
New York City, NY CMSA	X	X	19.9
Philadelphia, PA CMSA	X	X	6.0
Washington,-Baltimore, DC-VA-WV-MD CMSA	X	X	7.2
Atlanta, GA MSA	X	X	3.5
Barnstable, MA MSA	X	X	0.2
Baton Rouge, LA MSA	X	X	0.6
Benton Harbor, MI MSA	X	X	0.2
Biloxi, MS MSA*	X	X	0.3
Birmingham, AL MSA	X	X	0.9
Charlotte, NC MSA	X	X	1.3
Grand Rapids, MI MSA	X	X	1.0
Hartford, CT MSA	X	X	1.1
Houma, LA MSA	X	X	0.2
Huntington, WV MSA	X		0.3
Indianapolis, IN MSA	X		1.5
Louisville, KY MSA	X	X	1.0

Memphis, TN MSA	X	X	1.1
Nashville, TN MSA	X	X	1.1
New London, CT MSA	X	X	1.3
New Orleans, LA MSA*	X	X	0.3
Pensacola, FL MSA*	X		0.4
Pittsburgh, PA MSA		X	2.4
Providence, RI MSA	X	X	1.1
Richmond, VA MSA	X		0.9
St. Louis, MO MSA	X	X	2.5
Tampa, FL MSA*	X	X	2.2
33 areas / 88.7 million people	**32 areas/ 86.3 million people**	**28 areas/ 83.7 million people**	

*These areas have registered recent (1995-1998) ozone levels within 10% of the 1-hour ozone standard.
**Based on more recent air quality monitoring data not considered in the Tier 2 analysis, and on 10-year emissions projections, we expect to redesignate Cincinnati-Hamilton to attainment soon.

It should also be noted that the ozone modeling for the Tier 2 rulemaking did not look at the effect on ozone attainment and maintenance beyond current/committed controls and the Tier 2/Gasoline Sulfur Program itself. Therefore, Table II.A-3 should be interpreted as indicating what areas are at risk of ozone violations in 2007 or 2030 without federal or State measures that may be adopted and implemented after this rulemaking is proposed. We expect many of these areas to adopt additional emission reduction programs, but the Agency is unable to quantify the future reductions from additional State programs since they have not yet been adopted.

Table II.A-3 reflects only the ozone predictions made in the modeling for the Tier 2 rulemaking. The Tier 2 modeling did not predict 2007 or 2030 exceedances for a number of areas for which other available ozone modeling has shown 2007 violations, or for which the history and current degree of nonattainment indicates some risk of ozone violations in 2007 or beyond. Table II.A-4 lists these nine additional areas. Local ozone modeling for Dallas and Beaumont-Port Arthur, TX, using meteorology conditions and other inputs selected to be locally applicable, has shown exceedances in 2007. We attribute the absences of such predicted exceedances in the Tier 2 modeling to the use of episodes that did not represent the meteorological conditions that are most conducive to ozone in these areas. The Tier 2 modeling also did not predict exceedances in any nonattainment areas of California, which we attribute to an obvious and substantial tendency to underpredict ozone across the large-grid western modeling domain. These nine areas will be discussed in subsequent sections along with the 33 areas shown in Table II.A-3.

Table II.A-4:
Additional Areas With Some Risk of Ozone Violations in 2007 or Beyond

Metropolitan Areas	*1996 Population (in millions)*
South Coast Air Basin, CA (Los Angeles-Riverside-San Bernardino)	15.5
San Diego, CA	2.8
Southeast Desert, CA	0.4
Sacramento, CA	1.5
Ventura County, CA	0.7
San Joaquin Valley, CA	2.7
San Francisco, CA	6.2
Dallas, TX	4.6
Beaumont-Port Arthur, TX	0.4
9 areas	**34.8**

As described 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.

The Agency has recently proposed to redesignate Cincinnati-Hamilton, OH-KY-IN in attainment, and to approve its 10-year maintenance plan. This determination is based on four years of clean air quality monitoring data from 1996 to 1999 (1999 data was not considered in Tier 2 air quality analysis or this rulemaking), and a downward emissions trend. In Today's proposal, 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 proposed determination is based on Tier 2 air quality monitoring analysis and a violation in the 1995 to 1997 time period. With the 1999 data, the same method used now would place Cincinnati in the list of areas with recent concentrations within 10% 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 (ie, after 2010). As discussed in more detail later in this section, and also 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 proposal 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.[16]

iii. 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.

Ten ozone nonattainment areas with attainment dates of 2007 will be able to take advantage of the expected reductions from the proposed rule in their attainment demonstrations. Los Angeles, with its approved attainment demonstration for an attainment date of 2010, will also benefit. These 10 areas are listed in Table II.A-5

The clearest evidence of the need for more reductions is for New York and Houston for which we have proposed that specific additional reductions are needed for attainment based on the local ozone modeling and other evidence. The Agency has not identified a shortfall in the attainment demonstration submitted by Greater Connecticut (Hartford and New London, CT), but we have proposed to approve an extension date to 2007 due to overwhelming transport from the New York metropolitan area. There is some risk that New York will fail to attainment the standard by 2007, and thus a transferred risk that Connecticut will also fail. Additional reductions from this proposal will assist New York and Greater Connecticut in reaching the standard in 2007, and maintaining the standard thereafter. The ozone modeling for the Tier 2 rulemaking predicted exceedances in New York, Houston, and Greater Connecticut in both 2007 and 2030, in the absence of emission reductions beyond the Tier 2 program.

Chicago and Milwaukee submitted modeling which did not indicate a need for additional reductions, but they may join this group based on the revised modeling now underway. The ozone modeling for the Tier 2 rulemaking predicted exceedances in Chicago and Milwaukee in both 2007 and 2030, in the absence of emission reductions beyond the Tier 2 program.

Local modeling for Dallas and Beaumont Port-Arthur, TX shows violations in 2007, but we have not yet proposed action on attainment date extensions or attainment demonstrations for these areas. The Tier 2 ozone modeling did not indicate exceedances in Dallas and Beaumont Port-Arthur, TX, because the episodes used did not represent the meteorological conditions that are associated with higher ozone levels in these two areas. Both areas have requested an attainment date extension 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 do currently believe these two areas are likely to violate the NAAQS in 2007 and beyond, without more emission reductions in the local areas and/or from the upwind Houston area. We have proposed to grant an extension to Beaumont-Port Arthur, or in the alternative to reclassify it the next higher classification with a new attainment date well before 2007. We have not yet proposed any action on Dallas. The State of Texas is developing attainment plans for both areas, which are a precondition for granting extensions based on transport.

A national program to reduce VOC and NOx from heavy-duty vehicles is essential to achieving reductions in California nonattainment areas, due to interstate travel by these vehicles. 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 (as well as with Ventura County and San Diego), such that attainment in the Southeast Desert may depend 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 1996 Population (in millions)*
New York City, NY-NJ-CT	2007	VOC and NOx Shortfall	19.9
Houston, TX	2007	NOx Shortfall	4.3
Hartford, CT	2007 (requested extension)	Contingent on New York Attainment	1.1
New London, CT	2007 (requested extension)	Contingent on New York Attainment	1.3
Chicago, IL-IN	2007	Revised modeling in progress	8.6
Milwaukee, WI	2007	Revised modeling in progress	1.6
Dallas, TX	2007 (requested extension)	Local modeling shows nonattainment in 2007	4.6
Beaumont-Port Arthur, TX	2007 (requested extension)	Local modeling shows nonattainment in 2007	0.4
Los Angeles, CA	2010	Approved attainment demonstration, but needs significant additional reductions to attain	15.5
Southeast Desert, CA	2007	Approved attainment demonstration, but contingent on South Coast Attainment	0.4
10 Metropolitan Areas	**Total Population (in millions)**		**57.7**

Therefore, these 10 nonattainment areas with about 58 million people may rely in part on the reductions from this proposal to attain the 1-hour ozone standard. 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. While the NOx and VOC reductions are relatively small in the early years of this program, they may nevertheless prove to be a critical part of a range of actions necessary for these areas to overcome their shortfalls. We will start to work with these areas so that they can rely on the HD reductions once the rule is promulgated.

The HD reductions would 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 proposing today would 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 proposed today would 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 and without having accounted for the reductions from this proposed heavy-duty vehicle rule. This does not mean that there is no danger that ozone levels in these areas will exceed the NAAQS, in the absence of the proposed rule. 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, weather variations from year to year, 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.[n] There is at least some risk in each of these ten areas that

[n] 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% swing in the 90% 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

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, under the Regional Ozone Transport Rule. 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 violation in the year prior to the attainment year. The emission reductions from the proposed rule in 2008 and 2009 will be even larger than the reductions in 2007, and can play an important role is allowing an area that needs these extensions to attain in 2008 or 2009.

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

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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.

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 could reduce emissions, we think it is appropriate to consider the possibility that meteorological conditions 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 meteorology substantially worse than assumed in the ozone modeling in the attainment demonstration. Moreover, Tier 2 modeling predicted exceedances after 2007, which adds to the Agency's uncertainty about the prospect of continued attainment for these areas.

To conclude, a total of eight 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 believes 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 proposed rule would reduce heavy-duty vehicle emissions.

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

The next group of 26 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 26 areas are found in the middle of Table II.A-6, which compiles information about the 42 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 proposed to re-apply the ozone standard to the remaining areas. The Agency's belief 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 1995-1997 or 1996-1998, (2) predicted exceedances in 2007 or 2030 after accounting for reductions from Tier 2 and other local or regional controls currently in place or required, and (3) our assessment of the magnitude of recent violations, the variability of meteorological conditions, 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.

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, EPA Tier 2 air quality modeling predicted exceedances for Washington DC. Baltimore has predicted exceedances under Tier 2 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 and San Joaquin Valley have approved SIPs, and are not subject to transport by an area with a later attainment date.

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, Tier 2 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 proposed heavy-duty vehicle 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, Pittsburgh, San Diego, San Francisco (moderate, but with a 2000 attainment date), and St. Louis.[o] Tier 2 modeling predicted 2007 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 Tier 2 predictions of 2007 nonattainment, for which the 1-hour ozone NAAQS does not presently apply, but 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.[p] Houma (LaFourche Parish), LA does not have a specific attainment date. If and when the ozone standard is re-instated, the recent exceedances will likely trigger any contingency measures in their maintenance plans that are tied to new ozone violations. However, contingencies tied to air quality were not a required element in these maintenance plans, and the SIPs may not yet contain adequate provisions

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

[p] Preliminary data from the 1997 to 1999 monitoring period indicates that one additional major metropolitan area may be added to this group.

to bring these areas into consistent attainment. Our Tier 2 modeling predicted that, even with Tier 2 and other controls in place at the time, these areas are likely to exceed the standard in 2007 and/or 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 4 areas has had the ozone standard revoked, are without maintenance plans, have experienced recent exceedances, and are predicted by Tier 2 modeling to be nonattainment in 2007 if more emission reductions are not implemented. These areas include Barnstable, Boston, Benton Harbor, and Providence. (Benton Harbor was officially an unclassifiable/ attainment area prior to the revocation of the 1-hour standard.) We have proposed that these areas return to their prior nonattainment classification if the standard is reinstated, in which case Massachusetts and Rhode Island may be 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 develop and implement an attainment plan, we believe 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 2007or thereafter without additional control such as those proposed in this rulemaking.

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 belief 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 the proposed rule would be of help to them in reducing the risk of violations in that period.

For all of these 26 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.

v. Areas within 10 percent of Violating the Ozone Standard

There are six additional metropolitan areas, with another 11 million people in 1996, for which the available ozone modeling and other evidence is less clear regarding the need for additional reductions. These areas include Biloxi-Gulfport-Pascagoula, MS, Cleveland-Akron, OH, Detroit-Ann Arbor-Flint, MI, New Orleans, LA, Pensacola, FL, and Tampa, FL. Our own ozone modeling predicted these six areas to need further reductions to avoid exceedances in 2007 or 2030. The recent air quality monitoring data for these six areas shows ozone levels with less than a 10 percent margin below the NAAQS. This suggests that ozone concentrations in these areas may remain below the NAAQS for some time, but we believe there is still a risk of that future ozone levels will be above the NAAQS because meteorological conditions may be more severe in the future.

vi. Conclusion

We have reviewed the air quality situation of three broad groups of areas: those areas with recent violations of the ozone standard and attainment dates in 2007 or 2010, (2) those areas with recent violations and attainment dates (if any) prior to 2007, and (3) those areas with recent ozone concentrations within 10% of a violation of the 1-hour ozone standard, predicted exceedances, and without proposed or approved SIP attainment demonstrations. In general, the evidence summarized in this section supports a conclusion that emissions of NOx and VOC from heavy-duty vehicles in 2007 and later will contribute to a national ozone air pollution problem that warrants regulatory attention under section 202(a)(3) of the Act.

In sum, without these reductions, there is a significant risk that an appreciable number of the 42 areas, with a population of 123 million people in 1996, would violate the 1-hour ozone standard during the time period when these proposed standards would apply to heavy-duty vehicles. The 42 areas consist of the 27 areas with predicted exceedances in 2007 or 2030 under Tier 2 air quality modeling and recent violations of the 1-hour ozone standard, plus seven California areas (South Coast Air Basin, San Diego, Ventura County, Southeast Desert; San Francisco, San Joaquin Valley, Sacramento), two Texas areas (Dallas and Beaumont-Port Arthur), and six areas that have recent ozone concentrations within 10% of exceeding the standard and predicted exceedances. Under the mandates and authorities in the Clean Air Act, federal, State, and local governments are working to bring ozone levels into compliance with the 1-hour NAAQS through SIP attainment and maintenance plans, and ensure that future air quality continues to achieve this health-based standard. The new standards in this proposal are an integral part of these important efforts.

Table II.A-6

Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
Areas with 2007/ 2010 Attainment Dates					
New York City, NY-NJ-CT		19.9	Severe 17	2007	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70364, 70348, 70380)
Houston, TX		4.3	Severe 17	2007	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70548)
Hartford, CT		1.1	Serious	requested 2007	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70332)
New London, CT		1.3	Serious	requested 2007	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70332)
Chicago, IL-IN		8.6	Severe 17	2007	Proposed approval/disapproval

Table II.A-6

Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
Milwaukee, WI		1.6	Severe 17	2007	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70531)
Dallas, TX		4.6	Serious	requested 2007	
Beaumont-Port Arthur, TX		0.4`	Moderate	requested 2007	
Los Angeles, CA		15.5	Extreme	2010	
Southeast Desert, CA		0.4	Severe 17	2007	
	Subtotal of 10 areas	***57.7***			
Areas with Pre-2007 Attainment Dates**					
Atlanta, GA		3.5	Serious	requested 2003	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70478)

Table II.A-6

Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
Philadelphia-Wilmington-Atlantic City, PA-NJ-DE-MD		6.0	Severe-15	2005	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70380, 70444, 70412, 70428)
Sacramento, CA		1.5	Severe-15	2005	
San Joaquin Valley, CA		2.7	Serious	1999	possible future reclassification and change of attainment date to 2005
Ventura County, CA		0.7	Severe-15	2005	
Washington-Baltimore, DC-MD-VA-WV		7.2	Baltimore--Severe-15 Metro Washington--Serious	Baltimore--2005; Metro Washington--requested 2005	Proposed approval/disapproval of the attainment demonstration in the alternative (64 FR 70460, 70397)

Table II.A-6

Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
Charlotte-Gastonia, NC	x	1.3	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Grand Rapids, MI	x	1.0	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Huntington-Ashland, WV-KY	x	0.3	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Indianapolis, IN	x	1.5	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Memphis, TN	x	1.1	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Nashville, TN	x	1.1	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Barnstable-Yarmouth, MA	x	0.2	Serious (Part of former Boston NA area)		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Boston-Worcester-Lawrence, MA	x	5.6	Serious		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)

Table II.A-6

Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
Houma, LA		0.2	Nonattainment--incomplete data area	incomplete data area	
Providence-Fall River-Warwick, RI-MA	x	1.1	Serious		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Richmond-Petersburg, VA	x	1.0	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Benton Harbor, MI	x	0.2	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Baton Rouge, LA		0.6	Serious	1999	
Birmingham, AL		0.9	Marginal	1993	Proposed call for SIP revision (64 FR 70205)
Cincinnati-Hamilton, OH-KY-IN*		1.9	Moderate	Extended to 1998	Proposed redesignation to attainment (65 FR 3630)

Table II.A-6

Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
Louisville, KY-IN		0.3	Moderate	Requested 2003	
Pittsburgh, PA MSA		2.4	Moderate	1997	Received attainment date extension from 1996
San Diego, CA		2.8	Serious	1999	
San Francisco Bay Area, CA		6.2	Moderate	2000	Area was originally nonattainment, then redesignated to attainment, then redesignated back to nonattainment
St. Louis, MO-IL		2.5	Moderate	Requested 2003	
	Subtotal of 26 areas	***53.8***			
Areas with Pre-2007 Attainment Dates and Recent Concentrations within 10% of an Exceedance					

Table II.A-6

Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
Biloxi-Gulfport-Pascagoula, MS MSA	x	0.3	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Cleveland-Akron, OH CMSA	x	2.9	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Detroit-Ann Arbor-Flint, MI CMSA	x	5.3	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
New Orleans, LA MSA	x	0.3	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Pensacola, FL MSA	x	0.4	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
Tampa, FL MSA	x	2.2	[formerly attainment]		Proposal to reinstate the 1-hour NAAQS (64 FR 57424)
	Subtotal of 6 areas	***11.4***			
Total 1996 Population of All Metropolitan Areas at Risk of Exceeding the Ozone Standard in 2007 or Thereafter					

Table II.A-6					
Metropolitan Area / State	*NAAQS Revoked & Proposed for Reinstatement of Standard (x= yes)*	*1996 Population (in millions)*	*Classification (previous classification for revoked areas)*	*Attainment Date*	*Recent Development, Action, or Proposed Action*
42 Areas Total Population		**122.9 million**			

* Based on more recent air quality monitoring data not considered in the Tier 2 analysis, and on 10 year emissions projections, we expect to redesignate Cincinnati-Hamilton to attainment soon.

** The list includes certain areas that are currently not violating the 1-hour ozone standard.

f. 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.[17]

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 (Studies with Exposures of 0.12 ppm and Below Only)[a]

Ozone Concentration		Exposure Duration and Activity	Exposure Conditions	Number and Gender of Subjects	Subject Characteristics	Observed Effect(s)	Reference
ppm	*g/m³*						
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)

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)

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)

[a]See Appendix A of the 1996 Ozone Criteria Document for abbreviations and acronyms.

Table II.A-8. Ozone Exposure in Subjects with Preexisting Disease (Studies with Exposures of 0.12 ppm and Below Only) [a]

Ozone Concentration *ppm*	*g/m³*	*Exposure Duration and Activity*	*Exposure Condition*	*Number and Gender of Subjects*	*Subject Characteristics*	*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)
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)
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 (Studies with Exposures at 0.12 ppm and Below Only)

Ozone Concentration *ppm*	*g/m³*	*Exposure Duration and Activity*	*Exposure Conditions*	*Number and Gender of Subjects*	*Subject Characteristics*	*Observed Effect(s)*	*Reference*
0.08 0.10 0.12	157 196 235	6.6 h IE (6 × 50 min) $\dot{V}_E$ 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 $[O_3]$ 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)

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)

(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 g/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)

[a]See Appendix A of the 1996 Ozone Criteria Document for abbreviations and acronyms.

Table II.A-10. Increased Airway Responsiveness Following Ozone Exposures (Studies with Exposures at 0.12 ppm and Below Only [a]

Ozone Concentration[b]		*Exposure Duration and Activity*	*Exposure Conditions*	*Number and Gender of Subjects*	*Subject Characteristics*	*Observed Effect(s)*	*Reference*
ppm	*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)

[a]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 (Studies with Exposures at 0.12 ppm and Below Only [a]

Ozone Concentration[b] *ppm*	*g/m³*	*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 presents information on the number of people that live in metropolitan areas where ozone monitors have repeatedly recorded moderate levels of ozone over a prolonged period. The focus is on metropolitan areas other than those addressed in the above discussion of current and prospective attainment of the 1-hour ozone NAAQS. 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 illustration of the number of people residing in 1-hour ozone attainment areas where ozone monitoring data shows patterns involving multiple days with 8-hour ozone in the range of 0.08 to 0.12 ppm, we have analyzed the ozone monitoring data from one recent year, 1998. We considered each ozone-monitored county that is part of a metropolitan statistical area with all counties meeting the 1-hour ozone NAAQS based on 1996-1998 data, which together have a 1990 population of 76 million. For each county, we determined the number of days in 1998 on which the highest recorded 8-hour concentration of any monitor in the county was, for example, 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 happened, and summed their 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 0.12 ppm only. The full set of results are presented in a technical memorandum to the docket. Almost 71 million people, or 93 percent of all the population considered in the analysis, live in areas with at least 2 days with concentrations of 0.08 ppm or higher, the most stringent pattern considered. Moreover, we estimate that 62 million people lived in areas with 2 or more days with concentrations of 0.09 ppm or higher, excluding areas currently violating the 1-hour NAAQS. Fewer people are involved if only higher concentrations are considered, or if only areas with more than 2 days are considered.

The patterns of ozone that may exist in the 2007 and 2030 are also of interest. This analysis predicts that without additional emission reductions beyond adopted/committed controls, in addition to the population that resides in areas where there is a risk of violations of the 1-hour ozone NAAQS, there will be a large population residing in Eastern U.S. metropolitan areas with repeated, prolonged concentrations of ozone in the range of 0.08 ppm to 0.12 ppm. In 2007, our analysis predicts that about 33 million people (excluding those counties at risk of violating the 1-hour ozone standard) will live in Eastern metropolitan counties with at least two days of monitored 8-hour ozone concentrations at 0.08 to 0.12 ppm. This same analysis finds that 20 million people would be exposed in 2007 at 8-hour ozone concentrations between 0.09 and 0.12 ppm for two days, and 3.1 million people would be exposed at 8-hour concentrations between 0.10 and 0.12. In 2030, our

analysis predicts that 30 million people will be exposed to at least 2 days of ozone concentrations within the range of 0.08 to 0.12 ppm; 16 million to ozone levels between 0.09 and 0.12; and 2 million exposed to ozone levels between 0.10 and 0.12 ppm. The bulk of the emission reductions from this proposal would take effect between 2007 and 2030. The relatively small difference in the number of people with predicted exposures in 2007 and 2030 serves to illustrate the importance of further reductions in ozone precursors.

Another important metric is the number of days that are predicted to fall within the range of ozone levels that studies have shown may cause adverse health effects. As previously stated, 33 million people (excluding those counties at risk of violating the 1-hour ozone standard) are predicted to live in Eastern metropolitan counties in 2007 with at least two days of monitored 8-hour ozone concentrations at 0.08 to 0.12 ppm. Our analysis found that the 30 million people willbe exposed in 2007 to at least 3 days of monitored 8-hour ozone concentrations at 0.08 to 0.12 ppm; 28 million people willbe exposed to at least 4 days of monitored 8-hour ozone concentrations at 0.08 to 0.12 ppm; and 26 million people willbe exposed to at least 5 days of monitored 8-hour ozone concentrations at 0.08 to 0.12 ppm. By 2030, the analysis predicts 30 million people woule be exposed for at least two days of monitored 8-hour ozone concentrations at 0.08 to 0.12 ppm, and 28 million people will be exposed to at least 3 days of 8-hour ozone concentrations between 0.08 and 0.12 ppm; and 26 million people will be exposed to at least 4 days of 8-hour ozone concentrations between 0.08 and 0.12 ppm.[q] Again, the relatively small decline in the populations exposed repeatedly to ozone concentrations between 0.08 and 0.12 in 2007 and 2030 strongly suggests that current controls programs are not sufficient to address these impacts, and that additional reductions are needed to adequately protect public health and welfare.

2. 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.[18] Essentially all (>90

[q] Technical Memorandum to the Docket, May 8, 2000.

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).[19] 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.[20] 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.[21] 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 emissions of sulfur dioxide, oxides of nitrogen, and volatile organic compounds in order 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.[22]

i. Current PM_{10} Nonattainment

The most recent PM_{10} monitoring data indicates that 12 designated PM_{10} nonattainment areas, with a population of 19 million in 1990, violated the PM_{10} NAAQS in the period 1996-1998. Table II.A-15 lists the 12 areas. The table also indicates the classification and 1990 population for each area.

Table II.A-15
PM_{10} Nonattainment Areas Violating the PM_{10} NAAQS in 1996-1998 [a]

Area	*Classification*	*1990 Population (millions)*
Clark Co., NV	Serious	0.741
El Paso, TX[b]	Moderate	0.515
Hayden/Miami, AZ	Moderate	0.003
Imperial Valley, CA[b]	Moderate	0.092
Owens Valley, CA	Serious	0.018
San Joaquin Valley, CA	Serious	2.564
Mono Basin, CA	Moderate	0.000
Phoenix, AZ	Serious	2.238
Fort Hall Reservation, ID	Moderate	0.001
Los Angeles South Coast Air Basin, CA	Serious	13.00
Nogales, AZ	Moderate	0.019
Wallula, WA[c]	Moderate	0.048
TOTAL POPULATION		19.24

[a] In addition to these designated nonattainment areas, there are 15 unclassified counties, with a 1996 population of 4.2 million, for which the State has reported PM_{10} monitoring data for this period indicating a PM_{10} NAAQS violation. Although we do not believe that we are limited to considering only designated nonattainment areas as part of this rulemaking, we have focused on the designated areas in the case of PM_{10}. An official designation of PM_{10} nonattainment indicates the existence of a confirmed PM_{10} problem that is more than a result of a one-time monitoring upset or a result of PM_{10} exceedances attributable to natural events. We have not yet excluded the possibility that one or the other of these is responsible for the monitored violations in 1996-1998 in the 15 unclassified areas. We adopted a policy in 1996 that allows areas whose PM_{10} exceedances are attributable to natural events to remain unclassified if the State is taking all reasonable measures to safeguard public health regardless of the source of PM_{10} emissions. Areas that remain unclassified are not required 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.

[b] EPA has determined that PM10 nonattainment in these areas is attributable to international transport. While reductions in heavy-duty vehicle emissions cannot be expected to result in attainment, they would reduce the degree of PM10 nonattainment to some degree.

[c] The violation in this area has been determined to be attributable to natural events.

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

The proposed new standards for heavy-duty vehicles would 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 proposed new standards for heavy-duty vehicles. The Agency's recent PM modeling analysis performed for the Tier 2 rulemaking predicts that a significant number of areas across the nation are at risk of failing to meet the PM_{10} NAAQS even with Tier 2 and other controls currently in place. These reductions would assist States as they work with the Agency through SIP development and implementation of local controls to move their areas into attainment by the applicable deadline, and maintain the standards thereafter.

The Agency believes that the PM_{10} concentrations in 10 areas shown in Table II.A-16 have a significant risk of exceeding the PM_{10} standard without further emission reductions during the time period when this proposalwould take effect. This belief is based on the PM_{10} modeling conducted for the Tier 2 rulemaking. Table II.A-16 presents information about these 10 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 1996-1998 and were predicted to be in nonattainment in 2030 in our PM_{10} air quality modeling. These areas have a population of over 19 million. Included in the group are the nonattainment areas that are part of the Los Angeles, Phoenix, and Las Vegas metropolitan areas, where traffic from heavy-duty vehicles is substantial. These six areas would clearly benefits from the reductions in emissions that would occur from the proposed new standards for heavy-duty vehicles.

The second group of four counties listed in Table II.A-16 with a total of 8 million people in 1996 also had predicted exceedances of the PM10 standard. However, while 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 1998.[r] Unlike the situation for ozone, for which precursor emissions are generally declining over the next 10 years or so before beginning to increase, we estimate that emissions of PM_{10} will rise steadily unless new controls are implemented. The small margin of attainment which the four areas currently enjoy will likely erode; the PM air quality modeling suggests that it will be reversed. We therefore consider these four areas to each individually have a significant risk of exceeding the PM_{10} standard without further emission reductions. The emission reductions from the proposed new standards for heavy-duty vehicles would help these areas with attainment and maintain in conjunction with other processes that are

[r] In fact, in two of these areas, New York Co., NY and Harris Co., TX, the average PM_{10} level in 1998 was above the 50 micrograms per cubic meter value of the NAAQS. These two areas are not characterized in Table II.B-4 as areas with a high risk of failing to attain and maintain because lower PM_{10} levels in 1996 and 1997 caused their three-year average PM_{10} level to be lower than the NAAQS. Official nonattainment determinations for the annual PM_{10} NAAQS are made based on the average of 12 quarterly PM_{10} averages.

currently moving these areas towards attainment.

Table II.A-16
Areas With Significant Risk of Exceeding the PM_{10} NAAQS Without Further Emission Reductions

Area	*1990 Population (millions)*
Areas Currently Exceeding the PM_{10} standard	
Clark Co., NV	0.741
El Paso, TX[a]	0.515
Imperial Valley, CA[a]	0.092
San Joaquin Valley, CA	2.564
Phoenix, AZ	2.238
Los Angeles South Coast Air Basin, CA	13.00
Subtotal for 6 Areas	*19.15*
Areas within 10% of Exceeding the PM_{10} Standard	
New York Co., NY	1.49
Cuyahoga Co., OH	1.41
Harris, Co., TX	2.83
San Diego Co., CA	2.51
Subtotal for 4 Areas	*8.24*
Total 1996 Population of All 10 Areas at Risk of Exceeding the PM_{10} Standard	
10 Areas Total 1990 Population	**27.39**

[a] EPA has determined that PM10 nonattainment in these areas is attributable to international transport. While reductions in heavy-duty vehicle emissions cannot be expected to result in attainment, they would reduce the degree of PM10 nonattainment to some degree.

Future concentrations of ambient particulate matter may be influenced by the potentially significant influx of diesel-powered cars and light trucks into the light duty vehicle fleet. At the present time, virtually all cars and light trucks being sold are gasoline fueled. However, the possibility exists that diesels will become more prevalent in the car and light-duty truck fleet, since automotive companies have announced their desire to increase their sales of diesel cars and light trucks. For the Tier 2 rulemaking, the Agency performed a sensitivity analysis using A.D.Little's "most likely" increased growth scenario of diesel penetration into the light duty vehicle fleet which culminated in a 9 percent and 24 percent penetration of diesel vehicles in the LDV and LDT markets, respectively, in 2015 (see Tier 2 RIA, Table III.A.-13). This scenario is relevant for the purpose of this rulemaking because, according to the analysis performed in Tier 2, an increased number of diesel-powered light duty vehicles will increase LDV PM emissions by about 13 percent in 2010 rising to 19 percent in 2030, even with the stringent new PM standards established under

the Tier 2 rule. If manufacturers elect to certify a portion of their diesel-powered LDVs to the least-stringent PM standard available under the Tier 2 bin structure, the increase in LDV PM emissions could be even greater, thus potentially exacerbating PM_{10} nonattainment problems.

Because the types and sources of PM_{10} are complex and vary from area to area, the best projections of future PM_{10} concentrations are the local emission inventory and air quality modeling analyses that States have developed or are still in the process of developing for their PM_{10} attainment plans. In its Tier 2 analysis, EPA employed a much simpler modeling approach, known as the source-receptor matrix approach, which relates emission reductions to PM_{10} reductions on a national scale. This approach is presently our established air quality model for purposes of quantifying the health and welfare related economic benefits of PM reductions from major regulatory actions. One application of this modeling approach was for the Regulatory Impact Analysis for the establishment of the new PM NAAQS.[23] This model is also used to determine PM_{10} (and $PM_{2.5}$) concentration reductions to estimate economic benefits of proposed motor vehicle programs. In both applications, we model a base case emissions scenario corresponding to controls currently in place or committed to by States. As such, this scenario is an appropriate baseline for determining if further reductions in emissions are needed in order to attain and maintain the PM_{10} NAAQS.

The source-receptor matrix approach is appropriate for determining that a current nonattainment area has a high risk of remaining in PM_{10} nonattainment at a future date. Therefore, in its Tier 2 analysis, the Agency cross-matched the predictions of annual average PM_{10} nonattainment for 2030 from our final economic benefits analysis for Tier 2 and the list of current PM_{10} nonattainment areas. The Agency used the more recent modeling for 2030 rather than the earlier modeling for 2010, because the former incorporated the more recent estimates of emissions inventories that were also used for the ozone modeling.

EPA recognizes that the SIP process is ongoing and that several of the six current nonattainment areas in Table II.A-16 are in the process of, or will be adopting 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 promised on forecasts of emission levels and meteorology in future years. Therefore, even if these areas adopt and submit SIPs that EPA is able to approve as demonstrating attainment of the PM_{10} standard, the modeling conducted for Tier 2 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 proposed heavy-duty vehicle standards to maintain the PM_{10} standards in the long term. The other four areas in Table II.A-16 also have a significant risk of experiencing violations of the PM_{10} standard.

iii. Conclusion

In sum, the Agency believes that all 10 areas have a significant risk of experiencing particulate matter levels that violate the PM_{10} standard during the time period when this rule would take effect. These 10 areas have a combined population of 27 million, and are located throughout the nation. 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 at PM concentrations 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 g/m^3 and annual PM_{10} NAAQS of 50 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 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-17 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-18, also suggests that fine 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

[s] In the tables summarizing the studies, relative risks with lower confidence intervals greater than 1.0 are statistically significant at the 95% 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.

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-19 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.

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 g/m^3 and annual PM_{10} NAAQS of 50 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-17 Effect Estimates Per 50 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)

Study Location	*RR (± CI*) Only PM in Model*	*Reported PM_{10} Levels Mean (Min/Max)†*
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)

*CI: Confidence Interval

Table II.A-17 (cont'd). Effect Estimates per 50 µg/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)[ττ]		
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

References:

[a]Schwartz et al. (1996a). [L]Schwartz (1996). [X]Ostro et al. (1991)

[b]Pope et al. (1992, 1994)/O_3.
[c]Dockery et al. (1992)/O_3.
[d]Schwartz (1993).
[g]Ito and Thurston (1996)/O_3.
[f]Kinney et al. (1995)/O_3, CO.
[h]Styer et al. (1995).
[*]Means of several cities.
[I]Thurston et al. (1994)/O_3.
[**]PEFR decrease in ml/sec.
[J]Schwartz (1995)/SO_2.
[***]FEV_1 decrease.
[K]Schwartz et al. (1996b).
[‡]RR refers to total population, not just>65 years.
[M]Schwartz (1994e).
[N]Schwartz (1994f).
[O]Schwartz (1994d).
[Q]Schwartz et al. (1994).
[P]Schwartz and Morris (1995)/O_3, CO, SO_2.
[R]Pope et al. (1991).
[S]Pope and Dockery (1992).
[T]Schwartz (1994g)
[W]Pope and Kanner (1993).
[†]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.
[ι]Children.
[ɪɪ]Asthmatic children and adults.

Table II.A-18 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 g/m³ 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/m³)	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			

Short-term Exposure Mortality	*Indicator*	*RR (± CI*) per 25 g/m³ PM Increase*	*Reported PM Levels Mean (Min/Max)†*
Uniontown, PA[E]	$PM_{2.5}$	PEFR 23.1 (-0.3, 36.9) (per 25 g/m³)	25/88 (NR/88)

*CI: Confidence Interval

References:

[A]Schwartz et al. (1996a) unless [B]Burnett et al. (1994)
[C]Burnett et al. (1995) O_3 reported.
[D]Thurston et al. (1992, 1994)
[E]Neas et al. (1995)
[F]Ostro et al. (1993)
[G]Schwartz et al. (1994)

[†]Min/Max 24-h PM indicator level shown in parentheses otherwise noted as (± S.D.), 10 and 90 percentile (10,90) or R = range of values from min-max, no mean value reported.

[*]Change per 100 nmoles/m³
[**]Change per 20 g/m³ for $PM_{2.5}$; per 5 g/m³ for $PM_{2.5}$ sulfur; per 25 nmoles/m³ for H^+.
[***]50th percentile value (10,90 percentile)

Table II.A-19 Effect Estimates per Increments[a] 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[a]*	*Range of City PM Levels Means (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,h]	$PM_{15/10}$	NS Changes	20-59
Six City[e]	TSP	NS 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 g/m^3)	3.21% (-4.98, -1.41) FVC	—
24 City[i]	$SO_4^=$ (7 g/m^3)	3.06% (-4.50, -1.60) FVC	—
24 City[i]	PM_{10} (17 g/m^3)	2.42% (-4.30, -.0.51) FVC	—

[a]Estimates calculated annual-average PM increments assume: a 100 g/m^3 increase for TSP; a 50 g/m^3 increase for PM_{10} and PM_{15}; a 25 g/m^3 increase for $PM_{2.5}$; and a 15 g/m^3 increase for $SO_4^=$, except where noted otherwise; a 100 $nmole/m^3$ increase for H^+.
[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)
[h]NS Changes = No significant changes.
[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

State environmental agencies began operation of the first part of a broad network of $PM_{2.5}$ monitoring stations at the beginning of 1999, using the Federal Reference Method for $PM_{2.5}$ mass that we established as part of the setting of the $PM_{2.5}$ NAAQS.[24] The data that has been submitted to EPA from this network is accessible via the internet on an EPA website.[25] While more than 680 monitoring locations have reported $PM_{2.5}$ data from 1999 as of the date of this draft RIA, relatively few have reported sufficient data to determine an annual average concentration, and the established quality assurance process for the submitted data has not been completed. Monitors are generally located within metropolitan statistical areas, although some monitors intended to measure upwind $PM_{2.5}$ concentrations are located outside of metropolitan areas.

The 1996 Particulate Matter Criteria Document summarized the then-current state of knowledge of ambient concentrations and exposure to fine PM, based largely on these earlier monitoring programs. Because of the geographic breadth of the federal reference method network, we intend to give it primary consideration in our final assessment of current and likely future population exposures to ambient concentrations of fine PM. As of today, however, not all States have reported data from all $PM_{2.5}$ sites for all of 1999, and we and the States have not reviewed the data for possible errors as much as we intend to do. However, States are required to certify that the 1999 data is complete and accurate by July 2000, at which time we expect the data to be reliable for the purpose of characterizing the size of the population that is presently exposed to $PM_{2.5}$

concentrations similar to those that have been associated with premature mortality and other health effects in epidemiology studies.

For our final analysis of present exposures to fine PM, we plan to focus on the long-term average concentrations of $PM_{2.5}$. Accordingly, we plan to 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 our planned final analysis, the monitor data that may be available as of July 2000 would be sufficient.

Pending the availability of the 1999 $PM_{2.5}$ monitoring data, we have used an air quality model to estimate recent $PM_{2.5}$ concentrations across the U.S. We have conducted preliminary PM air quality modeling for 1996, using the Regional Modeling System for Aerosols and Deposition (REMSAD) air quality model. Using the modeling results from 1996, along with 1990 census data on the population of each modeling grid cell, we have calculated the population residing in grid cells with predicted annual average $PM_{2.5}$ concentrations in various ranges.

Based on our analysis of 1990 populations living in grid cells with modeled 1996 concentrations at various levels, over 113 million people (46 percent of continental US population, 1990) live in areas where long term ambient fine particulate matter levels are at or above 16 $\mu g/m^3$, the long term average $PM_{2.5}$ concentration that prevailed in Boston during the Harvard Six Cities Study (discussed above) which found that acute mortality was statistically significantly associated with daily fine PM concentrations. Most of the grid cells with predicted concentrations of 16 $\mu g/m^3$ are in metropolitan areas that are not experiencing PM_{10} violations. Over 53 million people (21.5 percent of continental US population, 1990) live in areas where 1996 annual average ambient fine particulate matter levels are modeled to be at or above 21 $\mu g/m^3$, the long term average $PM_{2.5}$ concentration that prevailed in Kingston/Knoxville, TN where the same study also found that acute mortality was statistically significantly associated with daily fine PM concentrations. Many of the monitors associated with this concentration range are located in areas that are not violating the PM_{10} NAAQS.

Based on the analysis presented here and described in more detail in a technical memorandum to the docket, we propose to conclude that significant numbers of people presently have exposures to $PM_{2.5}$ at concentrations that have been associated with premature mortality and other adverse effects. As discussed above, we will review this proposed conclusion in light of our planned analysis of the 1999 $PM_{2.5}$ monitoring data.

Future exposures to $PM_{2.5}$ are also relevant in this rulemaking. We plan to use the REMSAD air quality model, and the emission inventory estimates whose current status and planned development are described elsewhere in this draft RIA and in technical documents placed in the docket, to estimate the changes in $PM_{2.5}$ concentrations over the period 1996 to 2030.

With regard to total U.S. emissions, we expect a mixed trend in emissions between 1996 and 2030, given the control programs in place or already required by rule or enforceable commitment. VOC and NOx emissions will decline through about 2015 or 2020, increasing thereafter but not back to the levels of 1999 or even 2007. SO2 emissions will also decline from 1999 levels, with most of the decline happening by 2007. However, in the Tier 2 analysis, emissions of $PM_{2.5}$ were estimated to increase from 1999 levels.[26] This trend will vary from area to area, with areas with higher population and economic growth tending to have less decline in VOC, NOx, and SO2 emissions and a stronger increase in $PM_{2.5}$ emissions. These trends reflect the control programs that are aimed at ozone and PM_{10} attainment, which generally focus on only some of the emissions that contribute to $PM_{2.5}$ concentrations. Based on our current understanding of the emission trends and how they will affect air quality, we do expect that our final estimates of $PM_{2.5}$ concentrations in the 2007 and later period will indicate substantial population exposure to $PM_{2.5}$.

In conclusion, we believe that in the period of 2007 to 2030, when the proposed rule would help to reduce ambient $PM_{2.5}$ concentrations, a significant portion of the US population may be exposed to ambient $PM_{2.5}$ concentrations that studies have found may cause adverse health effects. Before promulgating the final rule, we plan to analyze the 1999 $PM_{2.5}$ monitoring data, as available, quality assured, and certified by the States, and we will make use of the results of additional, updated air quality modeling.

3. Diesel Exhaust

The following section presents information about the health hazard and potential risk posed by exposure to diesel exhaust to public health and welfare. 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 draft Health Assessment Document for Diesel Emissions (i.e. 1999 draft Assessment) is currently being revised based on comments received from the Clean Air Scientific Advisory Committee (CASAC) of EPA's Science Advisory Board.[27] Available evidence shows that exposure to diesel exhaust may cause adverse acute health effects with episodic exposures, as well as chronic noncancer and cancer effects to the respiratory system at longer term exposures. The current EPA position is that diesel exhaust is a likely human carcinogen in the lung and that this

cancer hazard applies to environmental levels of exposure.[t] Diesel exhaust exposure also poses a chronic noncancer hazard for the respiratory system, and can cause various transitory acute effect symptoms from episodic exposures.

In the 1999 draft Assessment, the Agency presented four pieces of 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. Individual epidemiological studies numbering about 30 show increased lung cancer risks of 20 to 89 percent within the study populations depending on the study. These studies are of varying quality in terms of design and controlling for factors that might confound a lung cancer response. Analytical results of pooling the positive study results show that on average the lung cancer risks were increased by 33 to 47 percent. 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. While not all studies have demonstrated an increased risk (six of 34 epidemiological studies summarized by the Health Effects Institute[28] 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.

Additionally, in experimental rat studies, lung cancer has been observed following high exposure to whole diesel exhaust. The rat lung tumor response is considered supportive of a potential for human hazard, though the exposure-response data is not deemed appropriate for estimating risk to humans. Also in separate animal studies, tumors have been observed resulting from 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 is 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 and 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

[t] The EPA designation of diesel exhaust as a likely human carcinogen is subject to further comment by CASAC in 2000. The designation of diesel exhaust as a likely human lung carcinogen under the 1996 Proposed Guidelines for Carcinogen Risk Assessment is very similar to designation of diesel exhaust as a probable B-1 carcinogen under the current 1986 Guidelines for Carcinogen Risk Assessment. The new guidelines, once finalized, will incorporate a narrative approach to assist the risk manager in the interpretation of the agent's mode of action, the weight of evidence, and any risk related exposure-response or protective exposure recommendations.

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 and high exposure animal studies, 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.

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.

The concern for the carcinogenic health hazard resulting from diesel exhaust exposures is widespread and several national and international agencies have designated diesel exhaust or diesel particulate matter as a 'potential' or 'probable' human carcinogen.[29] [30] [31] The International Agency for Research on Cancer (IARC) considers diesel exhaust a 'probable' human carcinogen. Based on IARC findings, the State of California identified diesel exhaust in 1990 as a chemical known to the State to cause cancer and has listed diesel exhaust as a toxic air contaminant.[32] The National Institutes for Occupational Safety and Health has classified diesel exhaust a "potential occupational carcinogen." The World Health Organization recommends that "urgent efforts should be made to reduce [diesel engine] emissions, specifically of particulates, by changing exhaust train techniques, engine design and fuel composition." The Department of Health and Human Services (DHHS) will decide in 2000 whether to list diesel particulate matter in its Report on Carcinogens (ROC) in terms of its lung cancer hazard.

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 at this juncture about safe or unsafe exposures to protect from

acute effects, since most of the effects are temporary and because the onset of acute effects is so variable in the population. 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. The primary evidence for this hazard comes from animal studies where pulmonary inflammation is observed. This response in animals is thought to be predictive of a human hazard. However, humans have not been extensively studied for diesel-specific chronic pulmonary effects. EPA notes that ambient $PM_{2.5}$, of which diesel is a part, has considerable human data regarding noncancer effects. Both $PM_{2.5}$ and diesel PM are believed to cause respiratory effects, though the $PM_{2.5}$ data raises other noncancer health hazard concerns. The draft 1999 Assessment discussed an existing inhalation reference concentration (RfC) for chronic effects that EPA intends to revise in the next draft Assessment in response to CASAC comments. The revised RfC will be reviewed by CASAC at a future meeting. The diesel exhaust RfC is an estimate, with an uncertainty spanning perhaps an order of magnitude, of the continuous human inhalation exposure (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious chronic, exposure-related, noncancer effects during a lifetime.[33]

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 also present (e.g., formaldehyde, acetaldehyde, 1,3-butadiene).

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 and dispersion. 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 an organic compound to a more toxicologically significant diesel exhaust constituent.[34] 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.[35]

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.[36] 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 micron with a mean particle diameter of about 0.2 microns. 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 microns, averaging about 0.02 microns. 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 particulates 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 particulates 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 other 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 adverse 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 does not directly account for the gaseous component of diesel exhaust. Another important aspect is to recognize that diesel exhaust particulate matter is part of ambient $PM_{2.5.}$ A qualitative comparison of adverse effects of exposure to $PM_{2.5}$ and diesel exhaust particulates shows that the respiratory system is adversely affected in both cases, though $PM_{2.5}$ has a wider spectrum of adverse effects for humans. Considerably more $PM_{2.5}$ research also exists than is the case for diesel exhaust. A carcinogenicity hazard for $PM_{2.5}$ has not yet been clearly shown, however.

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 the particulate phase of diesel exhaust. Today's proposal would 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 proposal would result in the implementation of particulate matter control technology (catalyzed particle traps) that would significantly reduce particulate matter and additionally remove gaseous hydrocarbons. The proposed 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.[u]

c. Ambient Concentrations and Exposure to Diesel Exhaust (Diesel Particulate Matter)

As stated previously, the current Agency position under review by CASAC is that diesel exhaust is a likely human carcinogen at 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.

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

[u]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).

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. Results from this model are expected in 2000.

Elemental carbon 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.[37] [38] [39] [40] In most ambient environments, diesel particulate matter is one of the major contributors to elemental carbon, 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 elemental carbon in diesel particulate matter, and the fact that diesel exhaust is one of the major contributors to elemental carbon in most ambient environments, diesel particulate matter concentrations can be bounded using elemental carbon measurements. One approach for calculating diesel particulate matter concentrations from elemental carbon measurements is presented in the draft 1999 Assessment.[41] 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 elemental carbon 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 secondary and primary 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.[42] [43] 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.[44]

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} & $PM_{2.5}$ g/m^3 (mean)*	*Diesel PM % of Total PM*	*Source 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[45]	1982, annual	11.6	36	
Phoenix area, AZ[46]	1989-90, Winter	4-22*	9-20	
Phoenix, AZ[47]	1994-95, Nov-Mar	0-5.3 (2.4)	0-27	
California, 15 Air	1988-92, annual	0.2-3.6*	†	
Manhattan, NY[48]	1993, Spring 3 days	13.2-46.7*	31-68	
Welby, CO	1996-97, Winter 60	0-7.3 (1.7)	0-26	
Brighton, CO[49]	1996-97, Winter 60	0-3.4 (1.2)	0-38	
Azusa, CA	1982, annual	1.4**	5	Dispersion Model: Based on emission rates from the majority of PM sources contributing to the area studied.
Pasadena, CA	1982, annual	2.0**	7	
Anaheim, CA	1982, annual	2.7**	12	
Long Beach, CA	1982, annual	3.5**	13	
Downtown LA, CA	1982, annual	3.5**	11	
Lennox, CA	1982, annual	3.8**	13	
West LA, CA[50]	1982, annual	3.8**	16	
Claremont, CA[51]	18-19 Aug 1987	2.4 (4.0)+**	8 (6)+	
Long Beach, CA	24 Sept 1996	1.9(2.6)+	8 (7)+	
Fullerton, CA	24 Sept 1996	2.4(3.9)+	9 (8)+	
Riverside, CA[52]	25 Sept 1996	4.4(13.3)+	12 (13)+	
Boston, MA	1995, annual	0.7-1.7 (1.1)	3-15	Diesel PM based on elemental carbon measurements.
Rochester, NY	1995, annual	0.4-0.8 (0.5)	2-9	
Quabbin, MA	1995, annual	0.2-0.6 (0.4)	1-6	
Reading, MA	1995, annual	0.4-1.3 (0.6)	2-7	
Brockport, NY[53]	1995, annual	0.2-0.5 (0.3)	1-5	
Washington, DC[54]	1992-1995, annual	1.3-1.8 (1.6)	6-10	
South Coast Air Basin[55]	1995-1996, annual	2.4-4.7‡	†	

*PM10 The reader should note that 80-95% of diesel PM is $PM_{2.5.}$ † Not Available
+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.
**On-road diesel vehicles only; All other values are for on-road plus off-highway diesel emissions
‡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.[56] 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 suggest 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.

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.[57] 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.

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.[58] 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 4.7 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.[59] 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.

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 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. This information is summarized in the draft Assessment and briefly summarized here.

Exposure to diesel particulate matter has been measured for several occupationally exposed groups including miners, railroad workers, diesel forklift operators, firefighters, truck drivers, dockworkers and mechanics. Diesel PM exposures (typically measured as respirable dust) reported for workers in coal mines using diesel-powered shuttle cars range from approximately 100 to 1,000 micrograms/m^3.[60] Diesel PM 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.[61] Diesel PM exposure among firefighters operating diesel engine vehicles ranges from 4-748 micrograms/m^3 which also encompasses the range of diesel PM exposures reported for diesel forklift dockworkers (18.6-64.7 micrograms/m^3).[62] [63] [64] [65] Diesel PM 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 12.1 to 13.8 micrograms/m^3 for dockworkers and mechanics.[66] For several occupational categories, the occupational exposure and/or environmental equivalent exposure overlap (see next paragraph for equivalence conversions) 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).

To understand the relevance of occupational exposure to the general population, an 'environmental exposure equivalent' to an occupational lifetime 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. A rough equivalence of occupational lifetime exposure to environmental lifetime exposure indicates that environmental exposure is 21% of the worker exposure, or a factor of 0.21.[v] Multiplying the estimated worker exposure by 0.21 converts the occupational exposures to 70-year lifetime environmental exposures. These types of conversions are useful for lifetime exposure estimates for the general population, most of which are during the adult years of life. We

[v] 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.

have not considered special exposure considerations for children. The equivalent environmental exposures for the occupational exposures presented in Table II.A-22 range from 0.4 to 210 micrograms/m^3.

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

Year of Sampling	*Locations*	***Diesel PM, ug/m³***
Occupational Exposure for a Minimum 8-Hour Workday and Equivalent Environmental Exposure		
1980's	Miners [a] (*Equivalent Environmental Exposure*)	100 - 1,000 *(21-210)*
1980's	Railroad Workers [b] (*Equivalent Environmental Exposure*)	39 - 191 *(8-40)*
1980's	Diesel Forklift Dockworkers [c] (*Equivalent Environmental Exposure*)	18.6 - 64.7 *(3.9-13.6)*
1980 and 1990's	Firefighters [d] (*Equivalent Environmental Exposure*)	6-748 *(1-157)*
1990	Long- and Short-Haul Truckers, Dockworkers, Mechanics [e] (*Equivalent Environmental Exposure*)	2.0 - 13.8 *(0.4-2.9)*
Ambient Exposure Estimates (On-Road) [f]		
1990	National Annual Average	0.8
1990	Urban Annual Average	0.9
1990	Urban Annual Average Outdoor Workers	1.0
1990	Range of Annual Average for Most Highly Exposed by City	0.8 - 4.0
California Exposure Estimates (On-Road & Off-Road) [h]		
1990	California Annual Average	1.5
2007	Projected California Annual Average	1.3
2020	Projected California Annual Average	1.2

[a] Watts (1995)
[b] Woskie et al. (1988)
[c] NIOSH (1990); Zaebst et al. (1991)
[d] Friones et al. (1991); NIOSH (1992); Birch and Carey (1996)
[e] Zaebst et al. (1991)
[f] HAPEM-MS3 exposure results for 1990 (see below for projections to future years) for on-road sources only. Methodology 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.
[g] California EPA (1998)

To estimate population exposures to diesel particulate matter, and to assess the impact of regulatory options on diesel particulate matter exposures, the EPA currently uses the Hazardous Air Pollutant Exposure Model - Mobile Source 3 (HAPEM-MS3).[67] This model provides national and urban-area specific exposures to diesel particulate matter from on-road sources only. Table II.A-22 also includes exposure estimates for on-road and off-road 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".[68]

The HAPEM-MS3 model estimates personal exposures to diesel particulate matter using a ratio to ambient CO measurements. 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.[69] The HAPEM-MS3 model simulates the movement of individuals between home and work and through 37 microenvironments for 22 different demographic groups. CO concentrations are based on ambient measurements made in 1990 and are related to exposures of individuals in a 10 km radius around the sampling site. 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.

Input to the model includes CO monitoring data for 1990, time-activity data collected in Denver, CO, Washington D.C., and Cincinnati, OH from 1982-1985, microenvironmental data and 1990 census population data. 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] 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%. 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". The HDDV VMT and resulting emissions inventory estimates reflected in the diesel PM exposures presented below are discussed in detail in Section II.B of this draft RIA. 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 in part due to the state of the art in currently available models for assessing population exposures. 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 those in the 98th percentile, 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, albeit with somewhat different spatial and temporal patterns, 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, and to assess the impact of today's proposal on changes in exposures to diesel particulate matter.

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, 97 percent of diesel particulate matter exposure from on-road vehicles is attributable to HDDVs and the rest is generated mainly by LDDTs. If the LDDT market share increases to 4.5 percent of the LDT market share beginning in model year 2004, diesel particulate matter exposures are estimated to increase 9 percent above baseline in 2007 and 12 percent above baseline by 2020. If this increase in the LDDT fleet is observed, 11 percent of the diesel exposure would be attributable to the LDDV fleet. However, regulations promulgated under the Tier 2 rulemaking will limit particulate matter emissions from LDDTs. We project that by 2020, assuming Tier 2 controls, almost all of the diesel particulate matter exposure from on-road sources will be attributable to HDDVs. The values reported here are for diesel particulate matter exposures attributable to on-road HDDVs.

Annual average exposure to on-road HDDV particulate matter was modeled for 1990, 1996, 2007, and 2020 both with and without today's proposed particulate matter standard. We estimate that in 1990, exposure to diesel particulate matter ranged from 0.8 micrograms/m^3 for the general population to 1.0 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 0.9 micrograms/m^3 for 1990.[w]

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 from 0.8 micrograms/m^3 for outdoor workers in St. Louis, to 2.0 micrograms/m^3 for outdoor workers in Spokane, and 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.

To assess the impact of today's proposed 0.01 g/bhp-hr limit on particulate matter emissions from HDDVs, diesel particulate matter exposures were modeled using this standard implemented in

[w]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.

2007 and projected to 2020. Exposures to diesel particulate matter were estimated for 2030 using the relative change in the PM inventory from 2020 to 2030 with and without the 2007 standard. We expect annual average nationwide exposures to change proportionally with the change in the PM emissions inventory. Comparing exposures predicted for 2007, 2020 and 2030 with and without today's proposed controls, we estimate that the proposed particulate matter standard would reduce nationwide annual average diesel particulate matter exposures from on-road motor vehicles five percent in 2007, 85 percent in 2020 compared with uncontrolled exposure levels in 2007 and 92 percent by 2030 compared with 2007 uncontrolled exposure levels (Figure II.A-1)**.**

While exposure for all demographic groups are projected to decrease from 1990 to 2007 (as a result of fleet-turnover and the full implementation of federal regulations that are currently in place), the model indicates that after 2015, diesel particulate matter exposures will begin to increase as controls currently in place (including Tier 2 Light Duty Vehicle standards) are offset by increases in vehicle miles traveled (Figure II.A-1).

The Agency is concerned about the significant negative public health and welfare impacts associated with ambient concentrations of diesel particulate matter, and accompanying exposures. The information presented in Figure II.A-1 represents only those particle emissions from on-road diesel vehicles, which show a downward trend due to federal regulation. There are, however, other significant sources of diesel particulate emissions (i.e., off-highway equipment and diesel generators) that account for a large portion of the diesel PM inventory.

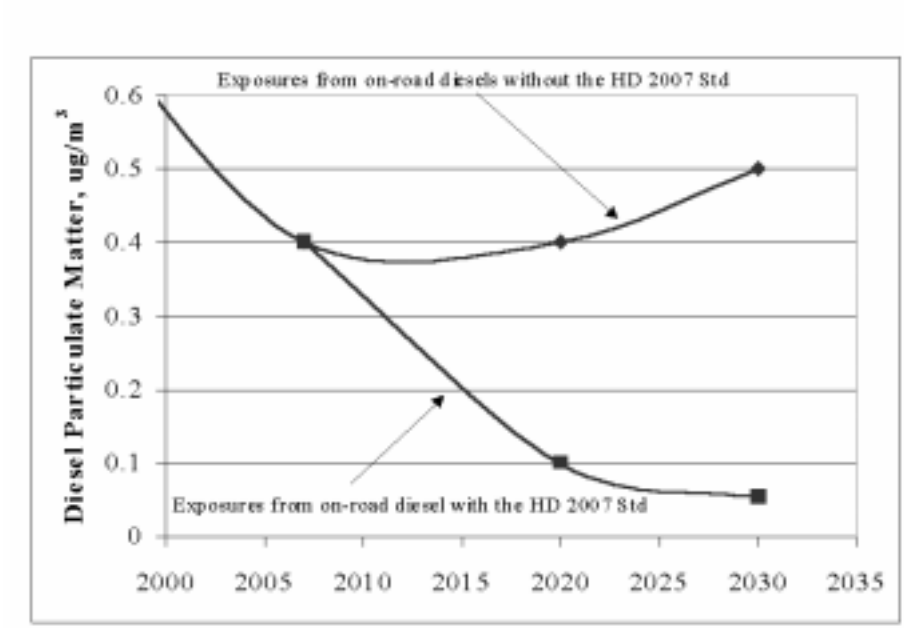


Figure II.A-1.
Nationwide Annual Average Diesel Particulate Matter Preliminary Exposure Estimates ($\mu g/m^3$) from On-road HDDVs in 1990, 1996, 2007, and 2020 With and Without Today's Proposed 2007 HDDV Particulate Matter Standard

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. Estimated exposures in this graph are provided to demonstrate expected trends. Note: heavy-duty vehicles represent an important source of diesel PM, but there are many other sources of diesel PM, mainly off-highway equipment (agricultural, construction, industrial, marine, railroad) that account for a significant portion of the diesel PM inventory.

The exposure estimates reported here 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, and 1.2 micrograms/m^3 in 2010.[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. 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] By proposing standards on particulate matter emissions for HDDVs, reductions in on-road diesel particulate matter emissions proposed in today's rule would 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 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 current Agency position under review by CASAC is that diesel exhaust is a likely human carcinogen and that the hazard observed at occupational exposures is believed to be present at environmental levels of exposure. The extrapolation of the hazard from occupational to environmental levels is the result of considering both the presence of mutagenic and carcinogenic agents in the diesel exhaust as well as noting that exposure differences between environmental levels and low end occupational levels are minimal to modest and thus the uncertainty in extrapolation may be minimal or at least is reduced. For some occupational exposures, the equivalent 70-year lifetime exposures relevant to the general population are overlapping or within a factor of ten of current day ambient concentrations and predicted ambient exposures. For the more highly exposed occupational groups, equivalent 70-year lifetime exposures are at most three orders of magnitude higher than ambient environmental concentrations and predicted related exposures. The extent to which the ambient concentration of diesel PM reflects a person's exposure will vary depending on the amount of time spent outdoors, but for some people (outdoor workers, children who spend a large portion of their day outdoors), ambient concentrations may offer a reasonable estimate of the magnitude of personal exposures. The hazard extrapolation to lower levels of

exposure also requires an assumption that risk is proportional to total lifetime exposure, an assumption nearly always made by EPA unless there is evidence to the contrary.

The potential overlap in occupational and ambient exposures to diesel PM is a significant public health concern for an environmental pollutant which is viewed as a likely human carcinogen. Several factors including the carcinogenicity of diesel, differences in human susceptibility, and the assumption of risk being proportional to exposure 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.

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 suggests 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. While available evidence supports EPA's conclusion that diesel exhaust is a likely human lung carcinogen, 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 an alternative method to gain a better understanding of the potential significance of the cancer hazard for the general population. The Agency agrees with CASAC that a unit risk estimate is not possible at this time.

In the draft 1999 Assessment, EPA acknowledged the limitations in characterizing a unit risk and provided a qualitative discussion of the possible cancer risk that would be consistent with occupational epidemiological findings of increased risk and relative exposure ranges in the occupational and environmental settings. A generic approach was used to qualitatively gauge the potential for cancer risk. It is not intended to be precise, but provides a reasoned scientific judgement of the potential for and possible range of risk in a comparably exposed population under the assumption of risk being proportional to lifetime exposure. Approximate increased risks observed in the diesel occupational studies were used as an example in the generic comparisons that would apply to any population with increased relative risks and known background risk for lung cancer. The following explanation regarding the qualitative range of population risk is discussed in more detail in the draft Assessment that is currently in preparation and is expected to be reviewed by CASAC in 2000.

Multiple diesel exhaust epidemiological studies have demonstrated an increased lung cancer risk of approximately 40 percent over background rates, a rough average from two pooled studies using results from 30 epidemiologic estimates analytically showing that risks ranged from 33 to 47 percent.[86] [87] The results from these pooled studies have some uncertainties, mostly relating to a possible confounding role for smoking in the original studies, although several of the individual studies did control for potential confounding due to smoking.

The consistent finding of increased risk among diesel exhaust exposed workers in the epidemiological studies, combined with our understanding of historic exposures in occupational groups and more recent environmental exposures provides the basis for the approach taken in the draft *Health Assessment Document for Diesel Emissions* to bound the possible magnitude under the assumption that diesel is a human carcinogen. The approach recognizes two conditions: (1) multiple diesel exhaust epidemiological studies show increased lung cancer risks of approximately 40 percent above background lung cancer rates and therefore some level of increased risk has to be present in the occupational populations; and (2) evidence specific to diesel engine emissions supports an often used assumption in public health risk assessment that risk can be viewed as proportional to cumulative lifetime exposure at low as well as higher exposures.

To understand the significance of the potential environmental cancer hazard in the absence of a unit risk estimate which EPA cannot provide at this time, the Agency is using general epidemiological principles to evaluate the available information. First, the risk of excess lung cancer from any cause, where a 40% increase in relative risk has been observed in humans, is estimated. Second, the margin(s) of exposure between the risk in the occupational setting and environmental exposures of interest is considered. Third, a perspective on the diesel exhaust hazard significance is developed by considering the range of potential excess lung cancer risk that can be derived by proportioning the risks from step one by the diesel exhaust exposure margins in step two. This approach is expanded upon below and will be explained in more detail in the updated draft *Health Assessment for Diesel Emissions* which will be publically available in late June 2000.

An approximate lifetime risk of lung cancer for occupationally exposed workers can be estimated using the average increased risk of 40 percent of background cancer levels in occupationally exposed workers. Given an overall background lung cancer risk in the U.S. of approximately five percent, the occupational population risk associated with the 40 percent increase in relative risk is in the magnitude of 10^{-2} (e.g., 40 percent x 5 percent).[x] The Agency believes that this technique used to qualitatively gauge the potential cancer risk is reasonable. The conversion of relative risk to population risk is not specific to the diesel data as it would apply to any population with risk increases and a known background rate for the cancer in question. Since the risk is assumed to be proportional to cumulative lifetime exposure, lower exposures among any population (e.g. the general population) compared to the occupational population, decrease the population risk proportionally. As discussed above, occupational and environmental exposure estimates indicate that exposure differences between occupational environmental exposures range from less than one order of magnitude (10) to three orders of magnitude (1000) (Table II.A-22). The high end of the risk range was derived by considering that there is approximately one order of magnitude difference

[x] This is a population risk, it is not a unit risk. 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.

between the lowest occupational exposures for job categories in which increased relative risk of cancer has been observed (truck workers, some railroad occupations) and environmental exposures. This small difference in exposures suggests that the estimated population risk may be as high as 10^{-3} ($10^{-2}/10$). The low end of the risk range was derived from the observation that the difference between occupational and environmental exposures is roughly three orders of magnitude, suggesting a possible population risk of 10^{-5}.

In the absence of a quantitative 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 margins between occupational and ambient environmental levels of diesel exhaust exposure. Based on this epidemiological and other information, there is the potential that upper bounds on environmental cancer risks from diesel exhaust may exceed 10^{-6} and could be as high as 10^{-3}. While uncertainty exists in estimating risk, 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 this is a prudent measure in light of the designation of diesel exhaust as a likely carcinogen, the exposure of almost the entire population to diesel exhaust, the significant and consistent finding of an increase in lung cancer risk in workers exposed to diesel exhaust, and the potential overlap and/or small difference between some occupational and environmental exposures.

Today's proposal would 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 proposal would result in the implementation of particulate matter control technology (catalyzed particle traps) that would significantly reduce particulate matter and additionally remove gaseous hydrocarbons.

4. 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. We are currently conducting a risk assessment to characterize the risk of cancer in the population that can be attributed to motor vehicle emissions of benzene, 1,3-butadiene, formaldehyde, and acetaldehyde.

a. Health Effects

i. Benzene

Highway mobile sources account for 52 percent of nationwide emissions of benzene and HDVs account for 7 percent of all highway vehicle benzene emissions.[88] 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.[89]

The EPA has recently reconfirmed that benzene is a known human carcinogen by all routes of exposure.[90] 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[y], 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.[91,92] 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[93] and increased proliferation of mouse bone marrow cells.[94] 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.[95]

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).[96] This range of unit risk represents the maximum likelihood (MLE) estimate of risk, not an upper confidence limit (UCL).

[y]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.[97] 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[z], a condition characterized by decreased numbers of circulating erythrocytes (red blood cells), leukocytes (white blood cells), and thrombocytes (blood platelets).[98,99] Individuals that develop pancytopenia and have continued exposure to benzene may develop aplastic anemia,[aa] whereas others exhibit both pancytopenia and bone marrow hyperplasia (excessive cell formation), a condition that may indicate a preleukemic state.[100,101] The most sensitive noncancer effect observed in humans is the depression of absolute lymphocyte counts in the circulating blood.[102]

ii. Formaldehyde

Highway mobile sources contribute approximately 27 percent of the national emissions of formaldehyde, and HDVs account for approximately 35 percent of the highway portion.[103] 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 exhaust TOG emissions, depending on control technology and fuel composition. It is not found in evaporative emissions.

Formaldehyde exhibits extremely complex atmospheric behavior.[104] 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,

[z] 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.

[aa] 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).

mice, hamsters, and monkeys.[105] 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.[106] Research has demonstrated that formaldehyde produces mutagenic activity in cell cultures.[107]

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.

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.[108] In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 5 ppm.[109] Formaldehyde exposure may also cause bronchial asthma-like symptoms in nonasthmatics.[110,111]

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 non-cancer health risks, is not available for formaldehyde at this time.

iii. Acetaldehyde

Highway mobile sources contribute 20 percent of the national acetaldehyde emissions and HDVs are responsible for approximately 33 percent of the highway emissions.[112] 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 exhaust TOG, depending on control technology and fuel composition.[113]

The atmospheric chemistry of acetaldehyde is similar in many respects to that of formaldehyde.[114] 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. The MLE estimate of a lifetime extra cancer risk from continuous acetaldehyde exposure is about 0.78×10^{-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.

Non-cancer effects in studies with rats and mice showed acetaldehyde to be moderately toxic by the inhalation, oral, and intravenous routes.[115, 116 , 117] 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 non-cancer health effects.[118]

iv. 1,3-Butadiene

Highway mobile sources account for approximately 51 percent of the annual emissions of 1,3-butadiene and HDVs account for approximately 15 percent of the highway vehicle portion.[119] 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 exhaust TOG, depending on control technology and fuel composition.[120]

1,3-Butadiene was classified by EPA as a Group B2 (probable human) carcinogen in 1985.[121] This classification was based on evidence from two species of rodents and epidemiologic data. EPA recently prepared a draft assessment to determine if sufficient evidence exists to propose that 1,3-butadiene be classified as a known human carcinogen.[122] However, the Environmental Health Committee of EPA's Scientific Advisory Board (SAB), in reviewing the draft document, issued a majority opinion that 1,3-butadiene should instead be classified as a probable human carcinogen.[123] The SAB panel recommended that EPA calculate the lifetime cancer risk estimates based on the human data from Denzell et al. 1995[124] and account for the highest exposure of "360 ppm-year" for 70 years. Based on this calculation[125] 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.[126] 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.[127] 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.[128]

v. Acrolein

HDVs are responsible for approximately 53 percent of the mobile source highway 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.[129]

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 general, dioxin exposures of concern have primarily been noninhalation exposures associated with human ingestion of certain foods, e.g. beef, vegetables, and dairy products contaminated by dioxin. EPA

has classified dioxin as a probable human carcinogen. Acute and chronic effects have also been reported for dioxin from oral and inhalation routes of exposure.[130]

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 study 'Analysis of the Impacts of Control Programs on Motor Vehicle Toxics Emissions and Exposure in Urban Areas and Nationwide'.[131] A quantitative assessment of the cancer and non-cancer population risks associated with mobile source emissions and exposure to these compounds has not yet been completed.

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 essentially 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.

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.[132, 133] 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 (Table II.A-23). 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*.[134]

Modeled onroad 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 onroad 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. The VMT fractions for light-duty and heavy-duty vehicles used to develop criteria pollutant inventory estimates for this rule and that the Agency plans to use in MOBILE6 are slightly lower for LDVs and higher for HDVs compared to the VMT fractions used to model gaseous toxic emissions and exposure in 1996. New inventories for the gaseous toxics that reflect this shift in VMT would decrease total benzene and 1,3-butadiene emissions for the fleet, since heavy duty diesels emit lower amounts of these compounds per vehicle. Conversely, acetaldehyde and formaldehyde emissions would increase, since heavy duty diesels emit higher amounts of these compounds. It should be noted however, that diesels emit lower amounts of VOC than gasoline engines; thus, emissions of aldehyde precursors would decrease. Toxics emissions and exposure projections are similarly affected by this new information regarding VMT splits.

To account for atmospheric loss of 1,3-Butadiene that varies seasonally[bb], exposure estimates were adjusted using the following multiplicative factors: 0.44 for summer, 0.70 for spring and fall, and 0.96 for winter.[135] These factors account for the difference in reactivity 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. We will be able to make a better comparison when HAPEM4 exposure estimates are available 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.

Ambient toxic exposures were modeled for 10 urban areas and 16 geographic regions (Table II.A-23). These areas were selected to encompass a broad range of I/M programs, fuel parameters,

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

and temperature regimes. The intent of the selection process was to best characterize the different combinations needed to perform accurate nationwide toxic emissions estimates. Each U. S. county was then mapped to a modeled area or region.

Table II.A-23 Areas Included in Toxic Exposure 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	

Table II.A-24 presents annual average nationwide exposure estimates from 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 8 percent for benzene, 40 percent for acetaldehyde, 46 percent for formaldehyde, and 12 percent for 1,3-butadiene.

Separately, exposure estimates were also generated for the 10 urban areas listed in Table II.A-23. 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. With today's proposed standards in place, exposures to the gaseous toxics in 2020 are expected to be reduced by 37 percent for benzene, 74 percent for acetaldehyde, 73 percent for formaldehyde and 70 percent for 1,3-butadiene.

Table II.A-24.
Modeled Average 50-State Ambient Exposure to Highway Motor Vehicle Toxics (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]
Benzene	1.07	0.68	0.35	0.27	0.26
Acetaldehyde	0.51	0.36	0.18	0.18	0.15
Formaldehyde	0.57	0.34	0.15	0.14	0.10
1,3-Butadiene	0.11	0.07	0.03	0.03	0.03

[a] Exposure estimates with the 2007 Heavy-Duty Vehicle Standards

5. 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.[136]

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.[137] 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.[138] 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 [139], and a major component of diesel exhaust. The reductions in ambient $PM_{2.5}$ from the standards proposed 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.

6. 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.[cc] 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 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 proposal 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

[cc] 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.

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 proposal would differ across different areas.

7. 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[dd]. 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 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[ee]. On the New England coast, for example, the number of red and brown tides 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

[dd] 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.

[ee] 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.

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[ff]. 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 proposed 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.

8. 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.[gg] 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[hh] 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

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

[gg] 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.

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

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 proposal 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.

9. Carbon Monoxide

We believe that the aftertreatment technology that would be used to meet the proposed 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.[140] 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).

B. Heavy-Duty Diesel Inventory Impacts

This part of the environmental impact chapter presents the inventory benefits we anticipate from heavy-duty diesel engines as a result of our proposed nonmethane hydrocarbon (NMHC), oxides of nitrogen (NOx), and particulate matter (PM) emission standards for heavy-duty diesel engines. In addition, we describe in detail the method we use to calculate these benefits. For these calculations, we consider the proposed engine-based standards as presented in Table II.B-1.

Table II.B-1: Proposed Engine-Based Emissions Standards and Diesel Fuel Requirements

Model Year	*NMHC [g/bhp-hr]*	*NOx [g/bhp-hr]*	*PM [g/bhp-hr]*	*Fuel Sulfur Limit [ppm]*
2007	0.14 for 25% of production	0.2 for 25% of production	0.01	15
2008	0.14 for 50% of production	0.2 for 50% of production	0.01	15
2009	0.14 for 75% of production	0.2 for 75% of production	0.01	15
2010+	0.14	0.2	0.01	15

In the remainder of this section, we first describe our calculation method. Second, we present reductions in directly regulated emissions of NMHC, NOx, and PM. Third, we discuss other benefits we anticipate in pollutants that we are not proposing new standards for, such as CO, SOx, and air toxics.

1. Description of Calculation Method

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. However, it does not have the flexibility to analyze all of the scenarios needed to support the rulemaking. Consequently, we developed a spreadsheet model which provides consistent results with the MOBILE model, and has the needed flexibility.

a. General Equation

We divide HDDEs into four classes for the purpose of inventory calculations. Table II.B-2 presents these classes which have different characteristics due to the difference in their size and use. Later in this chapter, we discuss some of these differences as they apply to emission modeling. Our standards apply throughout an engine's regulatory useful life. Therefore emissions may be cleaner earlier in an engine's life and dirtier later in its life due to deterioration. We use regulatory useful life in our modeling as the point in the engine's life at which the engine just meets the emissions standards with an assumed compliance margin.

Table II.B-2: HDDE Classes and Regulatory Useful Life

Class	*Description**	*Regulatory Useful Life***
Light HDDE	8,501-19,500 lbs. GVWR	110,000 mi /10 yrs
Medium HDDE	19,501-33,000 lbs. GVWR	185,000 mi /10 yrs
Heavy HDDE	> 33,000 lbs. GVWR	435,000 mi /10 yrs / 22,000 hrs
Urban Bus	characterized by application	435,000 mi /10 yrs / 22,000 hrs

* GVWR refers to gross vehicle weight rating; "urban bus" does not generally include school buses or inter-city buses.

** Whichever occurs first; for the purposes of these calculations, we use 290,000 miles for urban buses because we believe they reach 22,000 hours at approximately 290,000 miles on average.

For our calculations, emissions from HDDEs are primarily a function of per-engine emission factors, in-use deterioration, and vehicle miles traveled. Equation 1 presents the basic calculation we use to determine emissions from HDDEs in short tons per year. Following this section, we supply more detail on the components of this equation.

$$Tons_{CY} = (454 \times 2000)^{-1} \times j_{class} \left\{ VMT \times CF \times j_{MY/age} \left[(ZML_{MY} + DET_{MY/age}) \times TF_{age} \right] \right\} \quad (1)$$

where:

$Tons_{CY}$ - emissions for a given calendar year expressed in short tons
class - LHDDE, MHDDE, HHDDE, and urban bus
VMT- total vehicle miles traveled in a given calendar year by class
CF - conversion factor from g/bhp-hr to g/mi by class
MY/age - distribution of vehicles in a calendar year by vehicle age
ZML_{MY} - zero-mile emission level in g/bhp-hr for a given model year engine
$DET_{MY/age}$ - emissions deterioration as a function of model year and vehicle age
TF_{age} - travel fraction of vehicles from each model year in a given calendar year
$(454 \times 2000)^{-1}$ - conversion from grams to tons

b. Per-Engine EFs and DFs

This section discusses per-engine emission factors (EF)[ii] and deterioration factors (DF) for NMHC, NOx, and PM. For the purposes of this discussion, PM means total PM which includes direct sulfate PM emissions. Both baseline and control factors are discussed here.

[ii] For the purposes of this discussion, EF refers to the deteriorated emission level at the regulatory useful life. ZML refers to the zero mile level which is pre-deterioration.

This proposal includes both engine standards and fuel standards. For the proposed NMHC, NOx and PM standards, we consider the reduced fuel sulfur level to be a factor that enables emission control technology. Therefore, no "additional" benefits are calculated for these constituents solely due to lowering the fuel sulfur limit from 500 to 15 ppm. However, some additional PM benefits do occur for pre-2007 engines that use low sulfur fuel beginning in mid-2006. We discuss emission factors that are driven by the proposed standards here. Later, we discuss direct sulfate PM reductions in the existing fleet (pre-2007 MY) once these engines begin using low sulfur fuel.

For the baseline EFs and DFs presented here, we consider the emission levels which result from the 2004 HDDE emission standards. Although pre-2004 engines exist in the post-2006 fleet, the emission factors for these engines do not affect the projected benefits of the rule because they are a constant in both the baseline and control scenarios. Baseline EFs and DFs from pre-1988 engines are taken from MOBILE5b. Baseline EFs and DFs for 1988 to 2003 model year engines are taken from a report which considered certification data from 1988 to the present.[141] We base these EFs and DFs on our current understanding of what will be used in MOBILE6.

For engines meeting the 2004 NMHC+NOx standard of 2.5 g/bhp-hr, we assume that the mix would be 0.2 g/bhp-hr NMHC and 2.3 g/bhp-hr NOx. We base these emission factors on the judgement that engines not using aftertreatment will be easier to design for low NMHC than for low NOx. This is consistent with statements made in informal discussions with engine manufacturers. We then apply a compliance margin of eight percent to these levels. We base this compliance margin on historical certification data showing past practices. In other words, we assume that the manufacturers will conservatively design their engines to be eight percent below any standards we propose. Therefore, for a NOx standard of 2.3 g/bhp-hr, we use a level of 2.12 g/bhp-hr for the deteriorated emission level at the regulatory useful life of the engine. Table II.B-3 presents baseline EFs and DFs for HDDEs. For the purposes of the HDDE inventory calculations, EF refers to the emission factor at the end of the regulatory useful life.

In cases where the baseline emission factors are below the standard (with compliance margin) we assume that the levels will not increase. For example, baseline NMHC from urban buses is 0.08 g/bhp-hr. We assume that this will not increase to 0.18 g/bhp-hr. Consequently, we assume that urban bus engine manufacturers will use this low NMHC to allow them to design for higher NOx than 2.12 g/bhp-hr under the combined NMHC+NOx standard.

Table II.B-3: Emission Factors and Deterioration Factors for Model Year 2004-2006 Heavy-Duty Diesel Engines

Class	*EF (g/bhp-hr)*			*DF (g/bhp-hr/10^4 mi)*		
	NMHC	*NOx*	*PM*	*NMHC*	*NOx*	*PM*
LHDDE	0.18	2.12	0.08	0.001	0.001	0
MHDDE	0.18	2.12	0.08	0.001	0.001	0
HHDDE	0.18	2.12	0.08	0.001	0.003	0
Urban Bus*	0.08	2.21	0.04	0	0	0

* We assume that urban bus NMHC EFs will not increase in 2004 compared to current certification data.

In this analysis of HDDE emissions, we may underestimate emissions due to engine deterioration in-use. We believe that current modeling only represents properly maintained engines but may not be representative of in-use malmaintenance or tampering. One study[142] shows large deterioration rates for HDDEs. We will consider this study and other information which becomes available in our inventory analysis for the final rule.

For this analysis, we do not include deterioration factors for controlled engines. We understand that this underestimates deterioration and will look more rigorously into this issue prior to the final rule. Also, we use the same compliance margin for controlled engines as for baseline engines.

As discussed in Chapter III, we believe that manufacturers will be using aftertreatment to meet the proposed PM and NOx standards. As a result, we believe that NMHC levels will be well below the standard. Therefore, we anticipate that the aftertreatment described in Chapter III would result in about a 90 percent reduction in NMHC. Manufacturers must design for a fuel sulfur level of 15 ppm at certification; however, we estimate in-use fuel will average 7 ppm S. We therefore assume that PM emissions will be lower in-use due to lower formation of direct sulfate PM. This is discussed in more detail in Chapter III. Table II.B-4 presents the control EFs and DFs.

Table II.B-4: Emission Factors and Deterioration Factors for Heavy-Duty Diesel Engines Meeting the Proposed Standards

Class	*EF (g/bhp-hr)*			*DF (g/bhp-hr/10^4 mi)*		
	NMHC	*NOx*	*PM*	*NMHC*	*NOx*	*PM*
LHDDE	0.02	0.184	0.005	0	0	0
MHDDE	0.02	0.184	0.005	0	0	0
HHDDE	0.02	0.184	0.005	0	0	0
Urban Bus	0.02	0.184	0.005	0	0	0

c. Conversion Factors

Our proposed standards are in terms of grams of pollutant per unit of work performed. We use these units because we believe they best characterize emissions for an engine-based emission standard. However, we use vehicle miles traveled (VMT) to characterize heavy-duty engine operation in our emission inventory calculations. We believe that we can more accurately determine VMT than we can determine the work performed by HDDEs.

To apply VMT to our emissions calculations, we need emission factors in terms of grams per mile. Therefore, in our calculations, we convert the g/bhp-hr figures to g/mi. Because large engines typically perform more work in a mile of travel than small engines, we use separate conversion factors for each class of HDDEs. These numbers are reported in units of bhp-hr/mi and are based on work performed in developing MOBILE6.[143] Table II.B-5 presents the CFs we use for 1996 and later model year engines. For older engines, the CFs do not vary significantly.

Table II.B-5: Conversion Factors for HDDEs (bhp-hr/mi)

LHDDE	*MHDDE*	*HHDDE*	*Urban Bus*
1.23	2.25	2.97	4.68

d. Vehicle Miles Traveled

To determine the tons of emissions in a given calendar year we need to know the total VMT for that calendar year and the travel fraction of each model year of engines. The travel fraction for each model year of engines in a given calendar year is important because engines produced before and after a new standard goes into effect will have different emission levels.

To calculate nationwide emissions from HDDEs, we multiply the total VMT by the emission factors for HDDEs. For this analysis, we base the nationwide annual VMT on Federal Highway Administration estimates of annual VMT by highway category and vehicle type.[144] We then split this VMT by MOBILE class and fuel type. To split the VMT by class and fuel type, we use information on engine registrations by class and per-vehicle operation in miles per year collected for use in MOBILE6.[145] We use the products of the vehicle registrations and per-vehicle operation to determine the VMT fractions. Table II.B-6 presents the resulting breakdown of VMT by class.

Table II.B-6: Total VMT by Class for Heavy Duty Diesel Vehicles [million miles]

calendar year	*light-heavy*	*medium-heavy*	*heavy-heavy*	*urban bus*
2000	41,158	37,013	143,794	2,753
2007	51,362	46,189	179,442	3,435
2010	55,713	50,102	194,643	3,726
2015	63,550	57,149	222,022	4,250
2020	71,386	64,197	249,401	4,774
2030	87,060	78,292	304,160	5,823

These estimates are higher than those used by EPA in its recent proposal for 2004 heavy-duty engine standards due to the use of the new and updated MOBILE6 estimates of the fraction of total VMT that is heavy-duty and the fraction of heavy-duty VMT that is diesel. The predominant changes were to increase VMT estimates of light-heavy duty vehicles and the diesel fraction of heavy-duty vehicles, both of which are consistent with recent trends. The net result is that if the MOBILE values are used to calculated diesel fuel consumption (see section B.2.d), they agree in aggregate very well with Federal Highway Administration estimates. This gives us added confidence that these new estimates are accurate. In addition, the updated VMT fraction results in about a 3 percent reduction in light-duty vehicle VMT. This is reflected in our analysis.

e. VMT by Age

Travel fraction refers to the percentage of total miles driven in a given calendar year coming from each surviving model year of vehicles. In determining the travel fraction of vehicles by age, we considered both the survival rates of HDDEs and the average annual mileage accumulation rates by age. The survival rates give us the distribution of the number of vehicles of each model year in a given calendar year. HDDEs are operated less as they age; therefore, we consider the miles traveled by age when determining our travel fraction. We use the age distributions and VMT by age rates developed for MOBILE6.[146] Table II.B-7 presents survival distribution and mileage accumulation rates by age.

Table II.B-7: Survival Distribution of HDDEs by Age

Vehicle Age	*Survival Distributions*				*Mileage Accumulation Rates*			
	Light	*Medium*	*Heavy*	*Bus*	*Light*	*Medium*	*Heavy*	*Bus*
1*	0.740	0.535	0.535	0.500	28,951	36,493	113,208	45,171
2	1.000	1.000	1.000	1.000	26,479	33,203	102,211	43,731
3	0.932	0.935	0.935	1.000	24,226	30,221	92,288	42,337
4	0.870	0.875	0.875	1.000	22,173	27,519	83,332	40,987
5	0.811	0.818	0.818	1.000	20,301	25,069	75,250	39,681
6	0.756	0.765	0.765	1.000	18,593	22,849	67,954	38,416
7	0.705	0.715	0.715	1.000	17,035	20,836	61,369	37,191
8	0.657	0.669	0.669	1.000	15,613	19,012	55,424	36,005
9	0.613	0.626	0.626	1.000	14,314	17,359	50,059	34,857
10	0.572	0.585	0.585	0.999	13,128	15,861	45,214	33,746
11	0.533	0.547	0.547	0.996	12,043	14,502	40,840	32,670
12	0.497	0.512	0.512	0.989	11,052	13,271	36,892	31,629
13	0.464	0.478	0.478	0.970	10,146	12,155	33,327	30,620
14	0.432	0.447	0.447	0.925	9,317	11,145	30,107	29,644
15	0.403	0.418	0.418	0.832	8,558	10,228	27,200	28,699
16	0.376	0.391	0.391	0.662	7,864	9,397	24,575	27,784
17	0.351	0.366	0.366	0.413	7,227	8,644	22,204	26,898
18	0.327	0.342	0.342	0.197	6,645	7,962	20,063	26,041
19	0.305	0.320	0.320	0.161	6,111	7,342	18,129	25,211
20	0.284	0.299	0.299	0.132	5,622	6,782	16,382	24,407
21	0.265	0.280	0.280	0.108	5,173	6,274	14,804	23,629
22	0.247	0.262	0.262	0.089	4,762	5,814	13,379	22,875
23	0.231	0.245	0.245	0.072	4,384	5,396	12,091	22,146
24	0.215	0.229	0.229	0.059	4,038	5,017	10,928	21,440
25	0.207	0.218	0.218	0.065	3,720	4,674	9,877	20,757
26	0.194	0.204	0.204	0.033	3,427	4,363	8,928	20,095
27	0.177	0.191	0.191	0.033	3,159	4,082	8,069	19,454
28	0.167	0.179	0.179	0.033	2,913	3,826	7,294	18,834
29	0.153	0.165	0.165	0.016	2,686	3,595	6,595	18,234
30	0.119	0.151	0.151	0.016	2,477	3,385	5,962	17,652

* Because a model year of sales is spread over a whole calendar year, we use the convention that the average new vehicle will be on the road less than all of the first calendar year.

To calculate the annual VMT by age for an average HDDV, we multiply the vehicle survival distribution by the vehicle mileage accumulation by age. To get the travel fraction, we divide the

annual VMT by the total average lifetime miles. Table II.B-8 presents the annual VMT by age for an average HDDV and the total average lifetime miles.

Table II.B-8: Average Annual VMT by Age for HDDEs

Vehicle Age	*LHDDE*	*MHDDE*	*HHDDE*	*Urban Bus*
1	21,426	19,511	60,517	22,579
2	26,479	33,203	102,211	43,731
3	22,591	28,263	86,321	42,337
4	19,281	24,067	72,905	40,987
5	16,461	20,503	61,577	39,681
6	14,059	17,476	52,011	38,416
7	12,011	14,904	43,934	37,191
8	10,265	12,718	37,113	35,995
9	8,776	10,859	31,353	34,847
10	7,505	9,279	26,487	33,707
11	6,421	7,934	22,378	32,547
12	5,495	6,790	18,908	31,273
13	4,704	5,816	15,976	29,692
14	4,028	4,987	13,499	27,427
15	3,450	4,280	11,407	23,876
16	2,957	3,678	9,640	18,381
17	2,534	3,164	8,147	11,115
18	2,173	2,725	6,885	5,136
19	1,863	2,350	5,819	4,070
20	1,598	2,030	4,918	3,228
21	1,372	1,756	4,157	2,559
22	1,177	1,522	3,514	2,028
23	1,011	1,321	2,971	1,605
24	868	1,149	2,511	1,275
25	771	1,020	2,177	1,353
26	664	892	1,845	655
27	559	730	1,445	634
28	485	684	1,306	614
29	411	593	1,090	297
30	294	512	903	288
Total*	**201,689**	**244,716**	**713,926**	**567,521**

* This is an average and may be considerably higher for some vehicles.

2. Anticipated Emission Benefits of the Proposed HDDE Standards

This section looks at tons/year emission inventories of NOx, PM, and NMHC from HDDEs. These are the emissions that we propose to directly regulate from HDDEs. We present our projected baseline and controlled emissions inventories in addition to our anticipated benefits.

a. NOx Reductions

We include excess emissions in the NOx projections from some HDDEs in the 1988 through 1998 model years in order to accurately represent the entire HDDE inventory over time. Because HDDEs with excess NOx are no longer produced, the excess NOx emission projections will not affect the benefits of the proposed standards. We use the excess emissions inventory developed by the EPA's Office of Enforcement and Compliance Assurance.[147]

The proposed standards should result in about a 90 percent reduction in NOx from new engines. Table II.B-9 presents these projections with the estimated NOx benefits for selected years.

Table II.B-9: Nationwide NOx Emissions from HDDEs (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	2,860	2,830	32
2010	2,570	2,120	445
2015	2,490	1,130	1,360
2020	2,600	636	1,960
2030	3,000	313	2,690

b. PM Reductions from 2007 and Later MY Engines

The majority of the projected PM reductions from HDDEs are directly a result of the proposed 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 just looks at exhaust emission PM benefits that are directly the result of the proposed 2007 standards. DSPM benefits are discussed later and are presented in Table II.B-14. For engines meeting the proposed standards, we consider low sulfur fuel to be necessary to enable the PM control technology. In other words, we don't claim an additional benefit beyond the proposed standard for reductions in direct sulfate PM

except for the difference between certification and average in-use fuel sulfur levels as discussed above.

The proposed standards should result in about a 90 percent reduction in total PM from new engines. Table II.B-10 presents these projections with the estimated PM benefits for selected years (without the direct sulfate benefits from the existing fleet).

Table II.B-10: Nationwide PM Exhaust Emissions from HDDEs Without Existing Fleet Benefits (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	92	87	5
2010	86	58	28
2015	84	29	54
2020	88	15	75
2030	106	8	98

c. NMHC Reductions

Based on our analysis of the aftertreatment technology described in Chapter III, engines meeting the proposed standards should have very low levels of NMHC. Although the proposed 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 cause the NMHC levels to be well below the proposed standards as soon as the PM standard goes into effect in 2007.

The proposed standards should result in excess of a 90 percent reduction in NMHC from new engines. Table II.B-11 presents these projections with the estimated NMHC benefits for selected years.

Table II.B-11: Nationwide NMHC Exhaust Emissions from HDDEs (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	218	208	11
2010	202	141	60
2015	223	78	146
2020	249	48	202
2030	292	31	261

d. Fuel Consumption Estimates

As described below in Section B.2.e, we need to know the consumption of fuel produced for use in HDDEs to calculate SOx and direct sulfate PM emissions. In addition, we project fuel consumption to calculate fuel costs.

We calculated HDDE fuel consumption using Equation 1 above. However, we substituted fuel consumption estimates in terms gallons of fuel consumed per mile in place of the combined g/bhp-hr emission factor (ZML + DET) and the conversion factor (CF). Historical fuel consumption estimates (1987-1996) come from a report performed to support the upcoming MOBILE6 model.[148] 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. Table II.B-12 presents per-vehicle the HDDE fuel economy estimates for selected years. Table II.B-13 presents national fuel consumption estimates for HDDEs.

Table II.B-12: 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	7.9	6.7	4.0
2010	12.9	8.1	7.5	4.4
2020	14.0	8.3	8.3	4.8

Table II.B-13: HDDE Fuel Consumption Estimates by Calendar Year (billion gallons)

Calendar Year	*LHDDE*	*MHDDE*	*HHDDE*	*Urban Bus*
2007	4.33	5.84	26.5	0.88
2010	4.57	6.28	27.8	0.92
2015	4.99	7.08	30.0	1.00
2020	5.38	7.85	31.9	1.08
2030	6.05	9.34	35.3	1.20

To fully evaluate the effects of the proposed fuel sulfur level standards, we 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[149] and fuel economy estimates of 25 mpg and 16.7 mpg for light-duty diesel vehicles (LDDV) and light-duty diesel trucks (LDDT), respectively.[150] 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.[151] 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. For years beyond 1996, we use the EIA growth rates to project the spillover. Table II.B-14 presents our estimates of low sulfur fuel consumption. 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.[152]

Table II.B-14: Consumption of Highway Diesel Fuel Including Spillover (billion gallons)

Calendar Year	*Light-duty*	*Heavy-duty*	*Spillover*	*Total*
2007	0.37	37.6	4.82	42.8
2010	0.39	39.6	5.01	45.0
2015	0.43	43.0	5.26	48.7
2020	0.46	46.2	5.63	52.3
2030	0.53	51.9	6.26	58.7

e. Direct Sulfate PM Reductions from the Existing Fleet

Once the proposed low sulfur fuel requirements go into effect, pre-2007 model year engines 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.

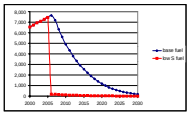
For engines built prior to 2007 that use low sulfur fuel in 2007 and later, we need to calculate the PM benefit associated with the reduction of direct sulfate PM. Equation 2 shows how we calculate this benefit and express it in terms of an emission factor. We did not consider deterioration for DSPM 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.2.d. 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.

Figure II.B-1 shows our national projections of direct sulfate PM emissions from the pre-2007 engines using HD highway diesel fuel with and without the proposed low sulfur fuel. The proposed low sulfur fuel should result in about a 95 percent reduction in direct sulfate PM from pre-2007 engines. Table II.B-15 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-15: Existing Fleet PM Benefits From Low Sulfur Fuel (thousand short tons per year)

Calendar Year	*HDDEs*	*Other*	*Total Benefits*
2007	6.17	0.85	7.02
2010	4.23	0.58	4.81
2015	2.18	0.29	2.48
2020	1.01	0.13	1.15
2030	0.15	0.02	0.17

f. Crankcase Emissions Reductions

We anticipate some benefits in NMHC, NOx, and PM from the proposed 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.[153] 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.

By routing crankcase vapors to the exhaust upstream of the aftertreatment systems, 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 proposed crankcase emission control is functionally equivalent to a 100 percent reduction in crankcase emissions. Table II.B-16 presents our estimates of the baseline crankcase emissions from HDDEs.

The engine data we use to determine crankcase emission levels is based on new engines. 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 proposed crankcase emission requirements.

Table II.B-16: 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	7.2	7.2	7.2
2020	9.7	9.7	9.7
2030	13.0	13.0	13.0

g. Total NOx, PM, and NMHC Benefits

As discussed above, we are anticipating large emission reductions in NOx, PM, and NMHC from HDDEs as a result of the proposed 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 proposed closed crankcase requirements. Table II.B-17 presents the total projected reductions from HDDEs for this proposed rule for selected years.

Table II.B-17: Total Reductions from HDDEs for this Proposed Rule (thousand short tons per year)

Calendar Year	*NOx*	*PM*	*NMHC*
2007	32	13	11
2010	449	36	64
2015	1,370	64	153
2020	1,970	83	211
2030	2,700	111	274

This proposal would be 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. This proposal would 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 the combined effects of the two standards on national HDDE NOx emissions. This figure also includes crankcase emissions.

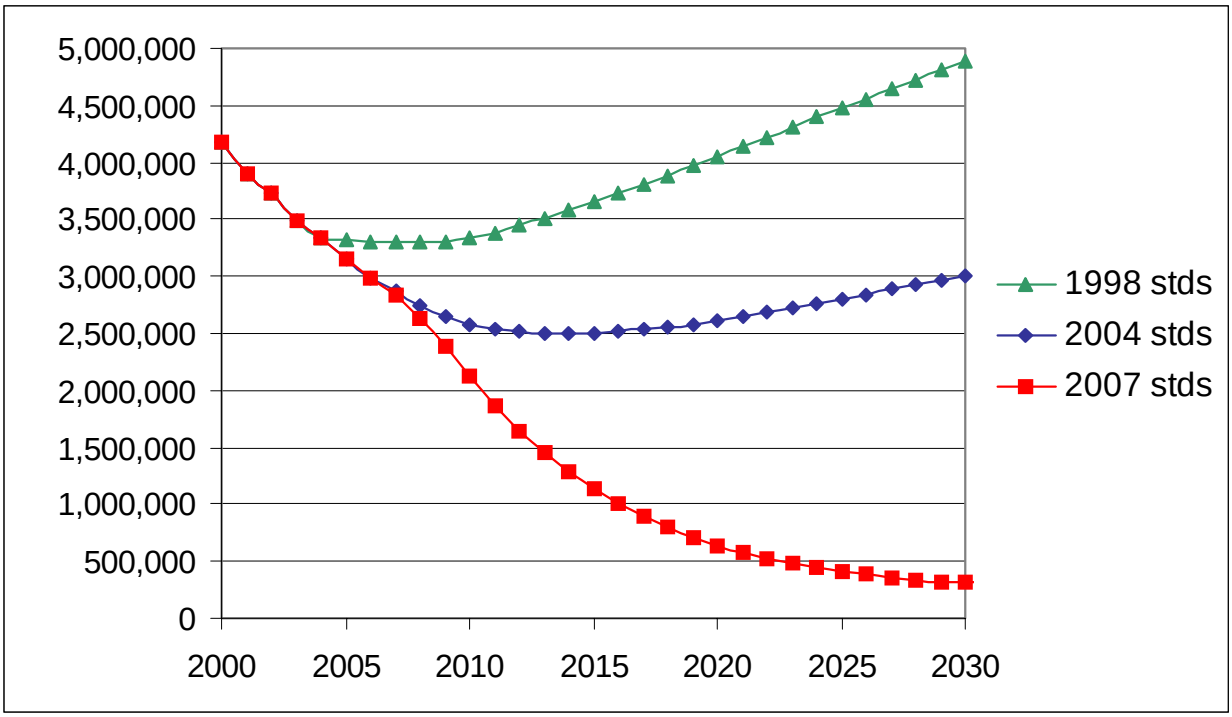


Figure II.B-2: Projected HDDE NOx Emissions Due to 2004 and Proposed 2007 Standards

Figure II.B-3 shows our national projections of total PM emissions with and without the proposed engine controls. This figure includes both crankcase emissions and the direct sulfate PM benefits due to the use of low sulfur fuel by the existing fleet.

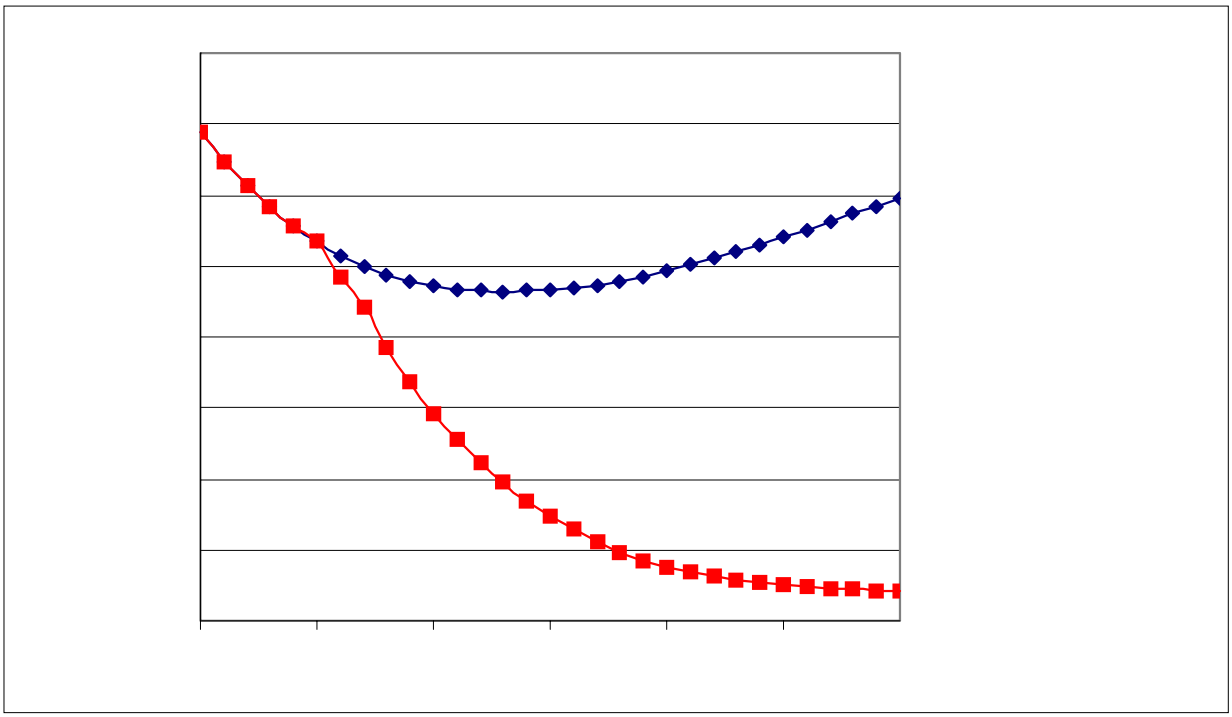


Figure II.B-3: Projected Nationwide PM Emissions from HDDEs

Figure II.B-4 shows our national projections of total NMHC crankcase and exhaust emissions with and without the proposed engine controls.

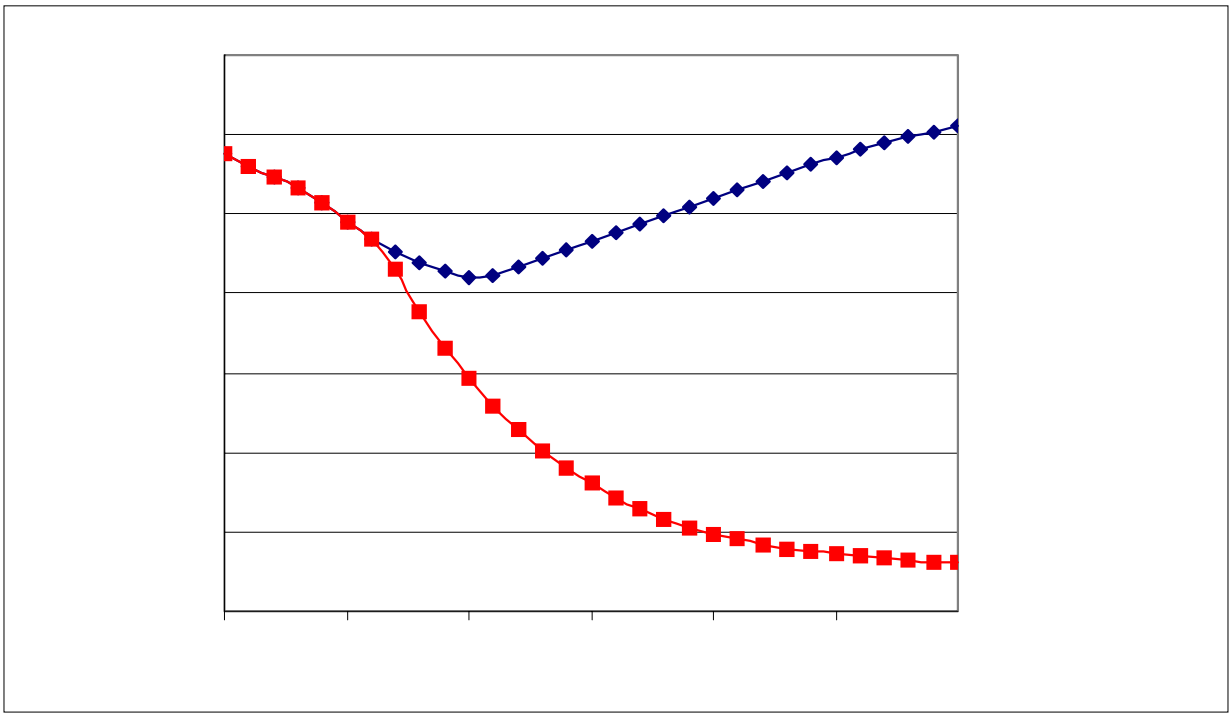


Figure II.B-4: Projected Nationwide NMHC Emissions from HDDEs

3. Additional Benefits of the Proposed 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 our proposed standards, we believe the proposed standards would result in reductions in CO, SOx, and air toxics. Here we present our anticipated benefits.

a. 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 cited above that 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 proposed 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-18 presents projected reductions in CO from HDDEs.

Table II.B-18: Reductions in CO from HDDEs (thousand short tons per year)

Calendar Year	*CO Benefit*
2007	71
2010	405
2015	911
2020	1,250
2030	1,640

b. SOx Reductions

We calculate SOx reductions from HDDEs using the same methodology as for direct sulfate PM (equation 2). However, 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-19 presents our estimates of SOx reductions from HDDEs corresponding with the use of low sulfur fuel. Table II.B-19 also presents SOx benefits from other sources using highway diesel fuel as discussed in Section B.2.d of this chapter.

Table II.B-19: Reductions in SOx from Low Sulfur Fuel (thousand short tons per year)

Calendar Year	*HDDE SOx Benefit*	*Other SOx Benefit*
2007	88	12
2010	93	13
2015	102	14
2020	109	14
2030	123	16

c. Air Toxics Reductions

The term "hydrocarbons" includes many different compounds. Speciation of the hydrocarbons would show that many of the compounds are air toxics such as benzene, formaldehyde, acetaldehyde, and 1,3-butadiene. For this analysis, we estimate air toxics using data collected on heavy-duty diesel engines.[154,155,156,157] According to this data, hydrocarbons from a HDDE include approximately 1.1 percent benzene, 7.8 percent formaldehyde, 2.9 percent acetaldehyde, and 0.6 percent 1,3-butadiene. Table II.B-20 shows the estimated air toxics reductions associated with the anticipated reductions in hydrocarbons. We lack data on how these percentages may change with the use of aftertreatment; we assume for the sake of analysis that they do not change.

Table II.B-20: Reductions in Air Toxics from HDDEs (thousand short tons per year)

Calendar Year	*Benzene*	*Formaldehyde*	*Acetaldehyde*	*1,3-Butadiene*
2007	0.1	0.8	0.3	0.1
2010	0.7	4.7	1.8	0.4
2015	1.6	11.4	4.2	0.9
2020	2.2	15.7	5.9	1.2
2030	2.9	20.4	7.6	1.6

4. Check on HDDE Inventory Calculations

a. Comparison with Tier 2 NPRM

As a check on our analysis, we compared the above inventory projections to HDDE projections that were made in the FRM for the Tier 2 standards for light-duty vehicles.[158] The analysis in the Tier 2 rule is a 47 state inventory built up from analyses that were performed for local air quality modeling. These local calculations were done using MOBILE5b and area specific inputs. We need to adjust the Tier 2 FRM HDDE numbers to be able to directly compare the Tier 2 analysis to our analysis described above. These adjustments are described in a memorandum to the docket[159] and are as follows:

- change from a 47 to a 50 state inventory
- remove tire and brake wear from PM inventory
- update 2004 emission factors
- update conversion factor

- remove speed correction factors for NMHC, NOx, and CO calculations

Another adjustment (not discussed in the memo cited above) that must be made to directly compare the Tier 2 analysis to this NPRM analysis is to update the national HDDE VMT estimates. The Tier 2 analysis used the National Emissions Trends Report[160] projections up to 2010 then extrapolated these projections to 2030 using linear growth. Although we used the same growth as the Tier 2 analysis, we used the updated annual VMT estimates described above. Although both analyses are based on Federal Highway Administration total VMT for motor vehicles, the NPRM analysis uses the updated splits by class and by fuel type cited in Section B.1.d of this chapter. These updated VMT fractions by class and fuel typeresult in a significant increase in our estimates of HDDE VMT.

Table II.B-21: Results of Adjusting the 2030 Tier 2 FRM HDDE Inventory Compared to the Diesel NPRM Exhaust Emission Projections

	NMHC [tons]	*NOx [tons]*	*PM [tons]*	*CO [tons]*
Adjusted Tier 2 FRM	283,000	2,730,000	98,800	1,950,000
Diesel NPRM	292,000	3,000,000	106,000	1,820,000
Difference (%)	3	10	1	-7

The numbers in Table II.B-21 do not include variations between a county-by-county analysis and a national analysis. These variations probably are the reason for the small differences between the adjusted Tier 2 FRM and the Diesel NPRM projections. Also, national average adjustments were applied to the Tier 2 FRM numbers which may also add to the difference. The variations between a county-by-county and a national analysis include:

- registration distribution of vehicles by age
- roadway effects
- VMT distribution of LHDDE, MHDDE, HHDDE, and Urban Bus

In conclusion, we found that the national HDDE inventory analysis for this NPRM is consistent with the Tier 2 analysis built up from local area analyses. However, we needed to adjust the Tier 2 analysis using more recent information in order to make a direct comparison of the two analyses.

b. Comparison with 2004 HDDE NPRM

Our new analysis of the HDDE emissions inventory is built up from the analysis in the draft regulatory impact analysis for the proposed 2004 heavy-duty highway emissions standards. However, we made some improvements to the previous analysis. These improvements include the inclusion of excess NOx emissions from heavy-duty diesel engines, newer annual VMT projections, and our rethinking of how manufacturers will design their engines to meet the combined NMHC and NOx standard. All of these improvements are discussed above in Section B of this chapter. We believe this new analysis more accurately reflects the national HDDE emissions inventory and we intend to use these improvements in the final regulatory impact analysis for the 2004 HDE standards.

C. Heavy-Duty Gasoline Inventory Impacts

In this section, we describe the expected environmental impacts of the proposed exhaust emission standards for heavy-duty gasoline engines and vehicles (HDGV). As we did above for diesel engines, we describe in detail the method we use to calculate these benefits. For these calculations, we consider the chassis-based and engine-based proposed standards as presented in Table II.C-1. We refer to engines sold as part of a chassis as "completes" and require these engines to be certified on a chassis-based test. Other engines are tested on an engine dynamometer and we refer to these as "incompletes."

Table II.C-1: Proposed FTP Chassis-Based Emission Standards

Class	*GVWR*	*NMHC*	*NOx*	*PM*
2b, Completes	8,501-10,000 lbs.	0.195 g/mile	0.2 g/mile	0.02 g/mile
3, Completes	10,001-14,000 lbs.	0.23 g/mile	0.4 g/mile	0.02 g/mile
All, Incompletes	> 8,500 lbs.	0.14 g/bhp-hr	0.20 g/bhp-hr	0.01 g/bhp-hr

In the remainder of this section, we describe our calculation method and we present reductions in emissions of NMHC and NOx. We do not anticipate significant reductions in PM from HDGVs as a result of this proposal.

1. Description of Calculation Method

As with our modeling of emissions from heavy-duty diesel engines, our intent is to be consistent with the upcoming MOBILE6 model. To do this, we run MOBILE5 with new emission rates which consider information that has been developed for MOBILE6.

a. Base Emission Factors (Zero-mile Levels and Deterioration Factors)

To determine the impact of the proposed standards, we first estimate the emission levels of vehicles currently in the fleet. Then we estimate the emission levels of engines that would meet the proposed standards. For the emission factors of existing engines, we use the recently updated zero-mile levels and deterioration factors developed for MOBILE6.[161] For 2004-2006 model year engines we use emission factors included in the draft RIA associated with the recently proposed emission standards for HDGVs.[162] Table II.C-2 presents the zero-mile level and deterioration factor for baseline HDGVs.

Table II.C-2: Zero-Mile Levels and Deterioration Factors for Model Year 2004-2006 Heavy-Duty Gasoline Vehicles

Class [units]	*ZMLs*		*DFs (per 10,000 miles)*	
	NMHC	*NOx*	*NMHC*	*NOx*
2b completes [g/mi]	0.119	0.574	0.008	0.008
3 completes [g/mi]	0.140	0.638	0.009	0.009
incompletes [g/bhp-hr]	0.085	0.510	0.005	0.007

Table II.C-2 reflects a compliance margin of 25 percent. This is consistent with the analysis for the proposed 2004 HDGV standards which based the compliance margin on certification data.

b. Conversion Factors

For engines not sold as part of a complete chassis (incompletes), we express the proposed emission standards for HDGVs in terms of g/bhp-hr. To convert from g/bhp-hr to g/mi, we use conversion factors developed for use in MOBILE6.[163] These conversion factors are a function of fuel density, brake specific fuel consumption and fuel economy. Table II.C-3 presents the conversion factors for HDGVs by class.

Table II.C-3: Conversion Factors for HDGVs (bhp-hr/mi)

Class 2b	*Class 3*	*Class 4*	*Class 5*	*Class 6*	*Class 7*	*Class 8*
1.10	1.15	1.13	1.32	1.31	1.45	1.54

c. Control Emission Factors (Zero-mile Levels and Deterioration Factors)

Our control emission factors are based on a combination of the standards and a compliance margin. For controlled engines, we believe the manufacturers would continue to design their engines with the same compliance margin. For this reason, we use a 25 percent compliance margin for controlled emission factors. Table II.C-4 presents the control emission factors.

Table II.C-4: Zero-Mile Levels and Deterioration Factors for Model Year 2007+ Heavy-Duty Gasoline Vehicles

Class [units]	*ZMLs*		*DFs (per 10,000 miles)*	
	NMHC	*NOx*	*NMHC*	*NOx*
2b completes [g/mi]	0.073	0.110	0.004	0.002
3 completes [g/mi]	0.095	0.233	0.006	0.003
incompletes [g/bhp-hr]	0.061	0.131	0.004	0.002

d. Vehicle Miles Traveled

As for HDDEs, we need to know the total VMT for that calendar year and the travel fraction of each model year of engines. The travel fraction is important because engines produced before and after a new standard goes into effect would have different emission levels.

i. Total Miles

For this analysis, we rely on the annual VMT used in the Tier 2 FRM including every state but California. California is claiming reductions from a LEV2 program. Although our proposed standards are a little more stringent, the LEV2 reductions are roughly similar compared to baseline. Therefore, to simplify our analysis, we do not calculate benefits for California.

We use the same methodology as for HDDEs to determine the annual VMT. We use Federal Highway Administration estimates of total VMT then use information collected for MOBILE6 to split by class and fuel type. We exclude miles traveled by medium-duty passenger vehicles because they are covered by the Tier 2 FRM. Table II.C-5 presents the VMT estimates by class that we use in our analysis.

Table II.C-5: 49 State VMT for Heavy-Duty Gasoline Vehicles [million miles]

Calendar Year	*Class 2B Completes (excluding MDPVs)*	*Class 3 Completes*	*Incompletes*
2000	45,557	1,312	19,920
2007	56,504	1,627	24,706
2010	61,596	1,773	26,933
2015	70,083	2,018	30,644
2020	78,570	2,262	34,355
2030	95,545	2,751	41,777

ii. Travel Fraction

Table II.C-6 presents the HDGV mileage accumulation rates and scrappage rates used in this analysis. The mileage accumulation rates come from recently updated rates for heavy-duty gasoline vehicles developed for the MOBILE6 emissions model.[164] We use scrappage rates taken from a National Highway Traffic Safety Administration (NHTSA) study which are based on light-duty truck (LDT) scrappage rates.[165] The scrappage rate represents the fraction of engines still in the fleet at a given age. The NHTSA study did not include information on HDGVs. We believe the LDT scrappage rates would be similar to those for most HDGVs since three-quarters of all HDGV sales are in the Class 2b truck category, which is the weight category just above the LDT cutoff of Class 2a trucks.

Table II.C-6: Annual Mileage Accumulation, Scrappage, and Composite Mileage Accumulation Rates for Heavy-duty Gasoline Vehicles

Age	*Class 2b/3 Annual Mileage*	*Class 4+ Annual Mileage*	*Scrappage Rate*
1	19,977	21,394	0.998
2	18,779	19,692	0.995
3	17,654	18,125	0.989
4	16,596	16,683	0.980
5	15,601	15,356	0.967
6	14,666	14,134	0.949
7	13,787	13,010	0.924
8	12,961	11,975	0.894
9	12,184	11,022	0.857
10	11,454	10,145	0.816
11	10,768	9,338	0.795
12	10,122	8,595	0.734
13	9,516	7,911	0.669
14	8,946	7,282	0.604
15	8,409	6,703	0.539
16	7,905	6,169	0.476
17	7,432	5,679	0.418
18	6,986	5,227	0.364
19	6,568	4,811	0.315
20	6,174	4,428	0.271
21	5,804	4,076	0.232
22	5,456	3,752	0.198
23	5,129	3,453	0.169
24	4,822	3,178	0.143
25+	4,533	2,926	0.648

Table II.C-7 contains the annual mileage accumulation rates for typical Class 2b/3 vehicles and typical incomplete vehicles factoring the effect of scrappage. For the incomplete vehicles, we sales-weighted the mileage accumulation rates for Class 2b/3 and Class 4+ vehicles in Table II.C-7 based on sales data on incomplete vehicles submitted to us by manufacturers.

Table II.C-7: Annual Mileage Accumulation Rates (Factoring in Scrappage) for Typical Heavy-duty Gasoline Vehicles

Age	*Class 2b/3 Complete Vehicle Annual Mileage*	*Incomplete Vehicle Annual Mileage*
1	19,937	20,524
2	18,685	19,062
3	17,460	17,653
4	16,264	16,299
5	15,086	14,988
6	13,918	13,709
7	12,739	12,441
8	11,587	11,221
9	10,442	10,028
10	9,346	8,903
11	8,561	8,089
12	7,430	6,964
13	6,366	5,921
14	5,403	4,986
15	4,532	4,151
16	3,763	3,420
17	3,107	2,802
18	2,543	2,277
19	2,069	1,839
20	1,673	1,477
21	1,347	1,180
22	1,080	940
23	867	749
24	690	592
25+	2,937	2,505
Lifetime Mileage	197,832	192,722

2. Anticipated Emission Benefits of the Proposed HDGV Standards

To estimate the exhaust NOx and NMHC inventories from heavy-duty gasoline vehicles, we calculate the average emissions of all heavy-duty gasoline vehicles in the fleet for a variety of years. To estimate the fleet average emissions for heavy-duty gasoline vehicles, we ran the MOBILE5b emissions model with the updated information on emission levels, adjustments, and vehicle usage characteristics as described earlier in this chapter. We multiplied these resulting fleet average emission levels by the estimated fleetwide vehicle miles traveled (VMT) for heavy-duty gasoline vehicles for the corresponding year to yield the exhaust emission inventories.

The inventories presented for HDGVs in this section represent nationwide inventories excluding California. For the sake of simplifying the analysis, we are not calculating benefits in California due to their comparably stringent LEV2 standards for these vehicles. A more detailed description of the inventory development has been placed in the docket.[166]

a. NOx Reductions

Figure II.C-1 presents the projected NOx inventory with and without the proposed standards. We believe the proposed NOx standards would result in about a 55 percent reduction in NOx from new heavy-duty gasoline vehicles. Table II.C-8 presents these projections with the estimated NOx benefits for selected years.

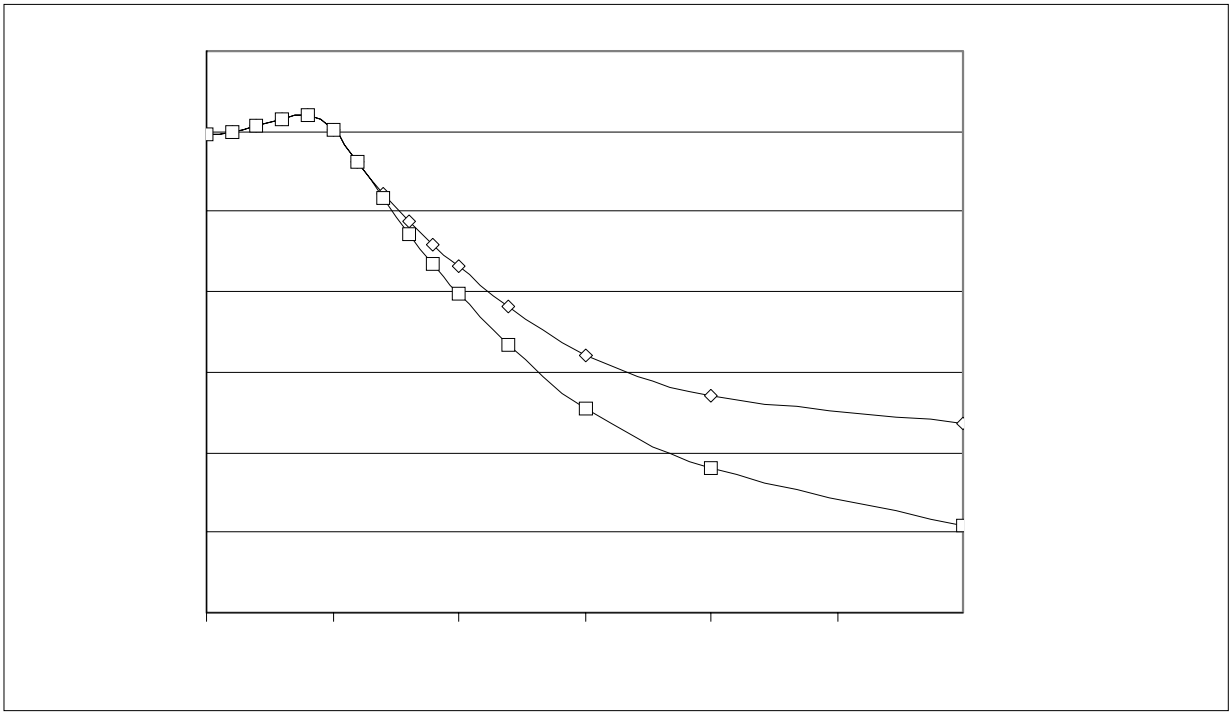


Figure II.C-1: Projected 49-State Exhaust NOx Emissions from HDGVs

Table II.C-8: Estimated 49-State NOx Emissions from HDGVs (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	261	258	3
2010	216	199	16
2015	160	127	33
2020	135	90	45
2030	117	54	63

b. Exhaust NMHC Reductions

Figure II.C-2 presents the projected exhaust NMHC inventory with and without the proposed standards. We believe the proposed NMHC standard would result in about a 28 percent reduction in exhaust NMHC from new heavy-duty gasoline vehicles. Table II.C-9 presents these projections with the estimated NMHC benefits for selected years.

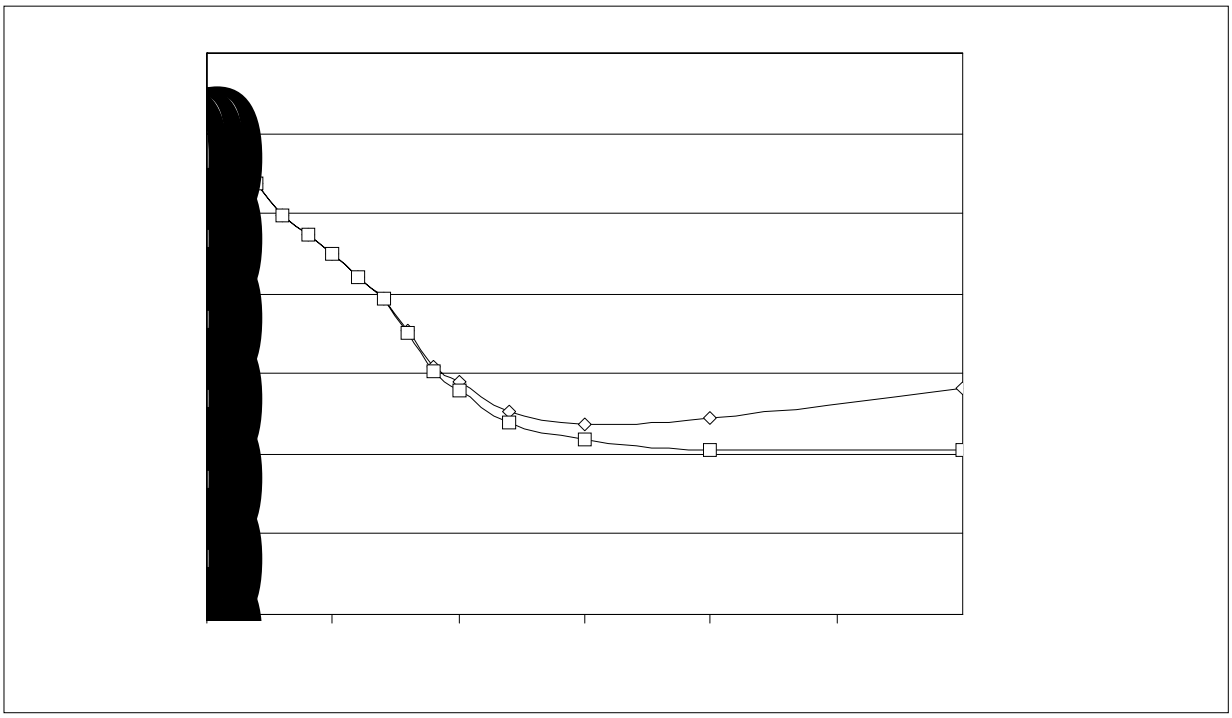


Figure II.C-2: Projected 49-State Exhaust NMHC Emissions from HDGVs

Table II.C-9: Estimated 49-State Exhaust NMHC Emissions from HDGVs (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	80	80	0.2
2010	58	57	1.7
2015	48	44	3.8
2020	49	41	7.6
2030	57	41	15.7

c. Evaporative Emission Reductions

Evaporative HC emissions include diurnal, resting loss, refueling, and running loss emissions. To estimate evaporative emissions from HDGVs, we used MOBILE5b directly, without any further modifications. 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.[167]

We assume that hot soak, diurnal and resting loss emissions 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. Figure II.C-3 presents the projected nonexhaust HC inventory with and without the proposed standards. We believe the proposed evaporative emissions standards would result in about a 10 percent reduction in nonexhaust HC from new heavy-duty gasoline vehicles. Table II.C-10 presents these projections with the estimated HC benefits for selected years.

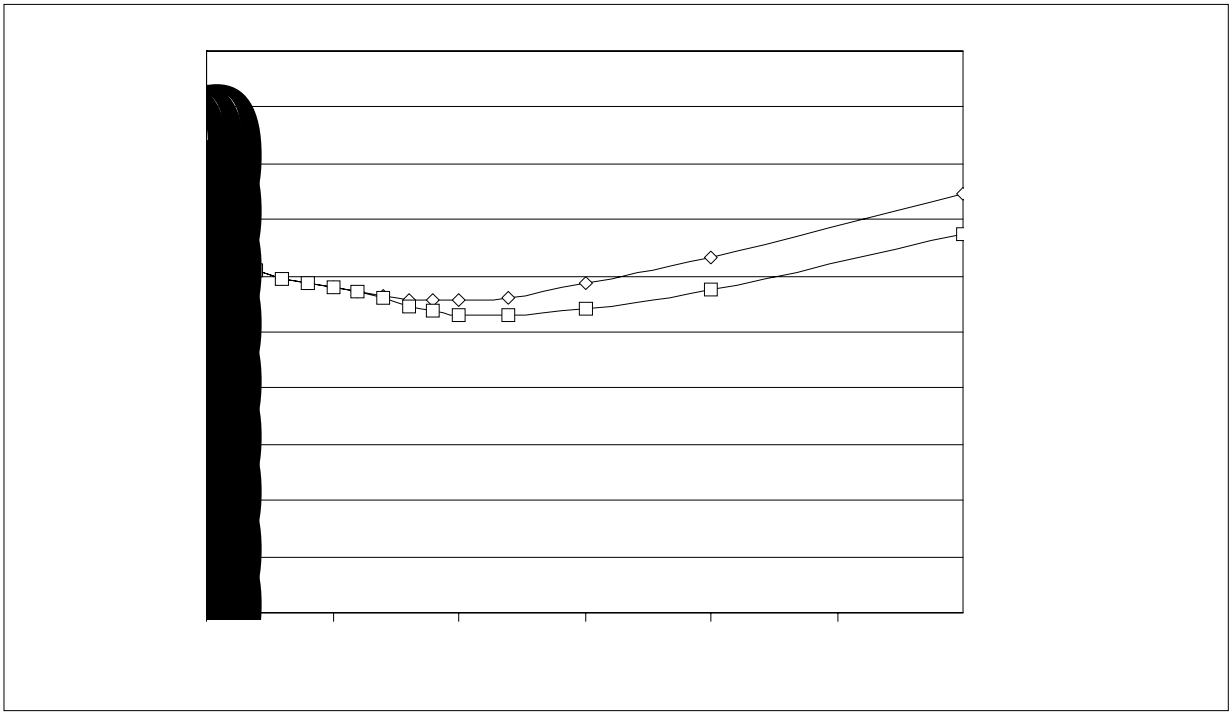


Figure II.C-3: Projected 49-State Evaporative Emissions from HDGVs

Table II.C-10: Estimated 49-State Evaporative Emissions from HDGVs (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	113	112	1
2010	111	106	5
2015	117	109	9
2020	127	115	11
2030	149	135	15

d. Air Toxics Reductions

We use baseline gaseous toxic emission estimates for heavy duty gasoline vehicles prepared by Sierra Research.[168] We developed estimates under controls proposed in this regulation by

applying the ratio of NMHC under new controls and NMHC under baseline conditions to the gaseous toxic emission estimates. Because benzene has an exhaust and an evaporative component, we use the ratio of total NMHC for that toxic; for formaldehyde, acetaldehyde, and 1,3-butadiene, we use the ratio of exhaust NMHC. The air toxics reductions for HDGVs are presented in Table II.C-11.

Table II.C-11: Estimated Reductions in Air Toxics from HDGVs (thousand short tons per year)

Calendar Year	*Benzene*	*Formaldehyde*	*Acetaldehyde*	*1,3-Butadiene*
2007	0.04	0.003	0.01	0.002
2010	0.27	0.03	0.12	0.02
2015	0.48	0.07	0.24	0.04
2020	0.56	0.09	0.27	0.04
2030	0.64	0.10	0.27	0.04

D. Total Emissions Reductions for This Proposal

Figures II.D-1 through II.D-3 present the total projected emissions of NOx, PM, and NMHC from heavy-duty engines with and without the proposed exhaust, evaporative, crankcase, and fuel sulfur standards. Tables II.D-1 through II.D-3 present the total NOx, PM, and NMHC benefits from heavy-duty engines that we anticipate from this proposed rule. Evaporative emission reductions are included in the NMHC benefits. Table II.D-4 presents the total air toxics reductions.

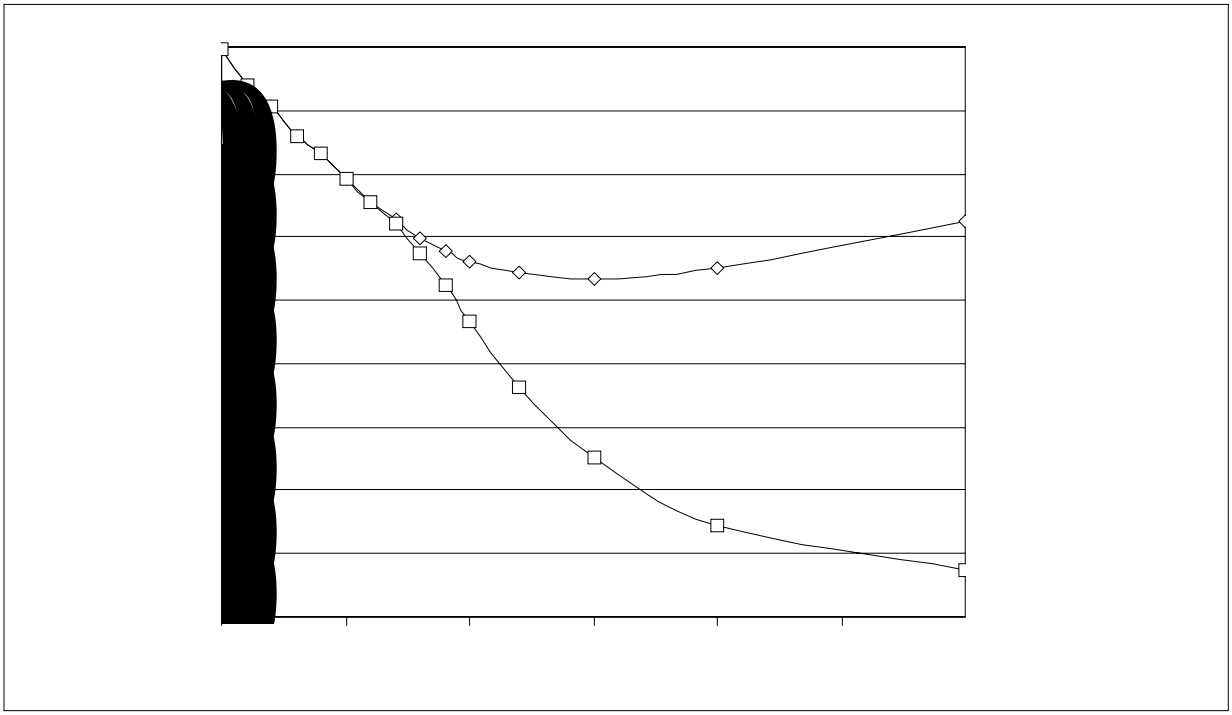


Figure II.D-1: Projected NOx Inventory for Heavy-Duty Highway Vehicles

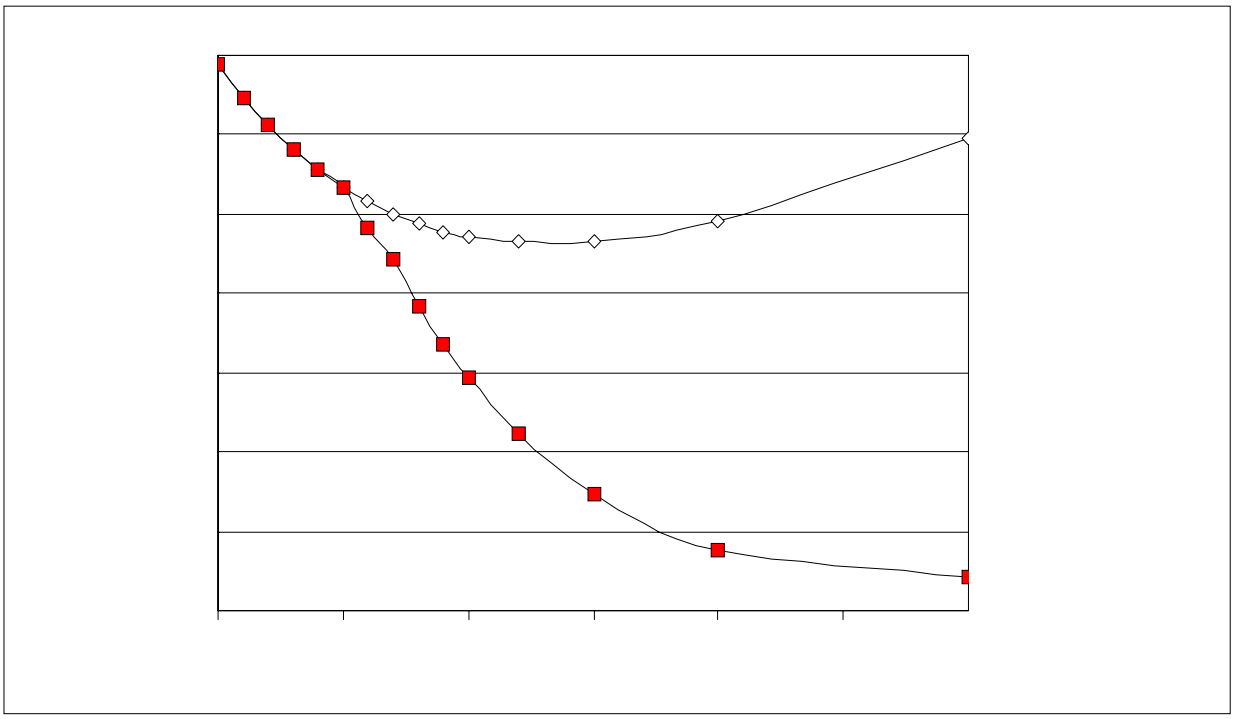


Figure II.D-2: Projected PM Inventory for Heavy-Duty Highway Vehicles

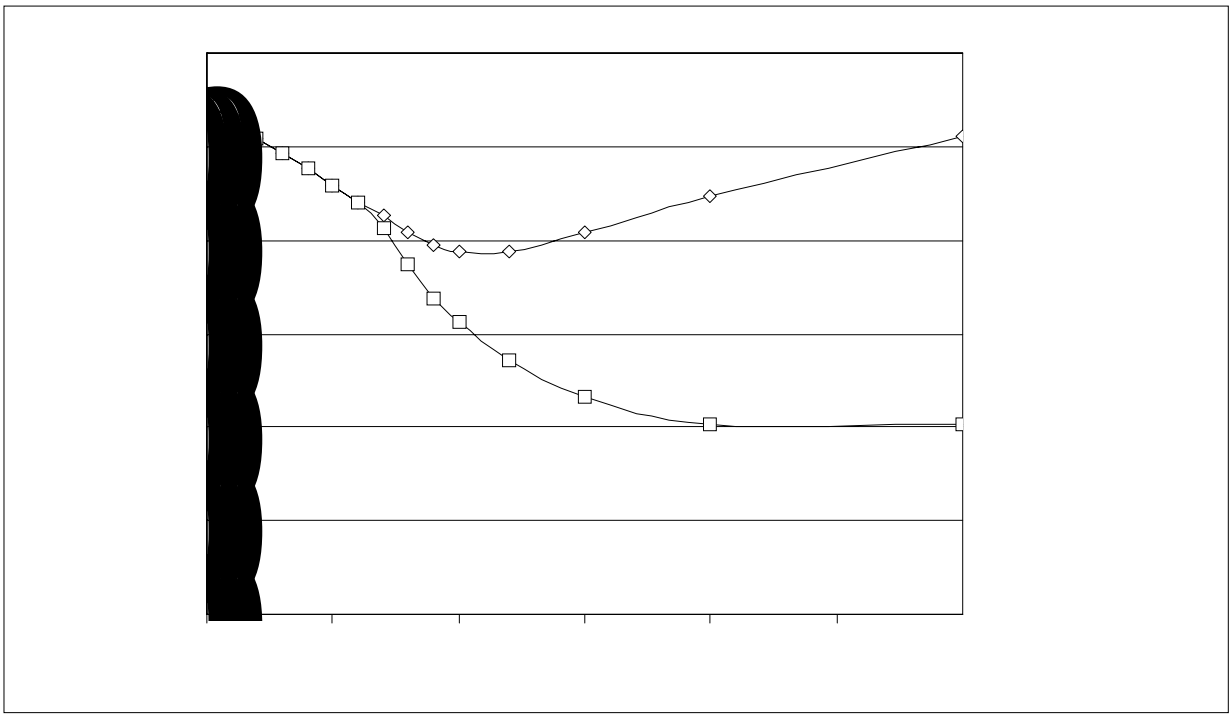


Figure II.D-3: Projected NMHC Inventory for Heavy-Duty Highway Vehicles

Table II.D-1: Total NOx Emissions and Proposed Benefits for this Proposed Rule (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	3,120	3,090	35
2010	2,790	2,320	465
2015	2,660	1,260	1,400
2020	2,740	726	2,020
2030	3,130	367	2,760

Table II.D-2: Total PM Emissions and Proposed Benefits for this Proposed Rule (thousand short tons per year)

Calendar Year	*HDV Baseline*	*HDV Controlled*	*HDV Benefit*	*Other DSPM Benefit*	*Total Benefit*
2007	10	88	12	0.9	13
2010	94	59	36	0.6	36
2015	93	30	64	0.3	64
2020	98	15	83	0.1	83
2030	119	8	111	0.02	111

Table II.D-3: Total NMHC Emissions and Proposed Benefits for this Proposed Rule (thousand short tons per year)

Calendar Year	*Baseline*	*Controlled*	*Benefit*
2007	420	407	12
2010	380	309	71
2015	398	232	165
2020	436	205	230
2030	511	206	305

Table II.D-4: Total Reductions in Air Toxics
(thousand short tons per year)

Calendar Year	*Benzene*	*Formaldehyde*	*Acetaldehyde*	*1,3-Butadiene*
2007	0.2	0.8	0.3	0.1
2010	1.0	4.7	1.9	0.4
2015	2.1	11.4	4.5	0.9
2020	2.8	15.8	6.1	1.2
2030	3.5	20.5	7.9	1.6

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