CHAPTER 2: Air Quality, Health, and Welfare Effects

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With this rulemaking, we are acting to extend highway types of emission controls to another major source of diesel engine emissions: nonroad land-based diesel engines. This final rule sets out emission standards for nonroad land-based diesel engines - engines used mainly in construction, agricultural, industrial and mining operations - that will achieve reductions in particulate matter (PM) and NOx standards in excess of 95 percent and 90 percent, respectively. This action also regulates nonroad diesel fuel for the first time by reducing sulfur levels in this fuel more than 99 percent to 15 part per million (ppm). The diesel fuel sulfur requirements will decrease PM and sulfur dioxide (SO_2) emissions for land-based diesel engines, as well as for three other nonroad source categories: commercial marine diesel vessels, locomotives, and recreational marine diesel engines.

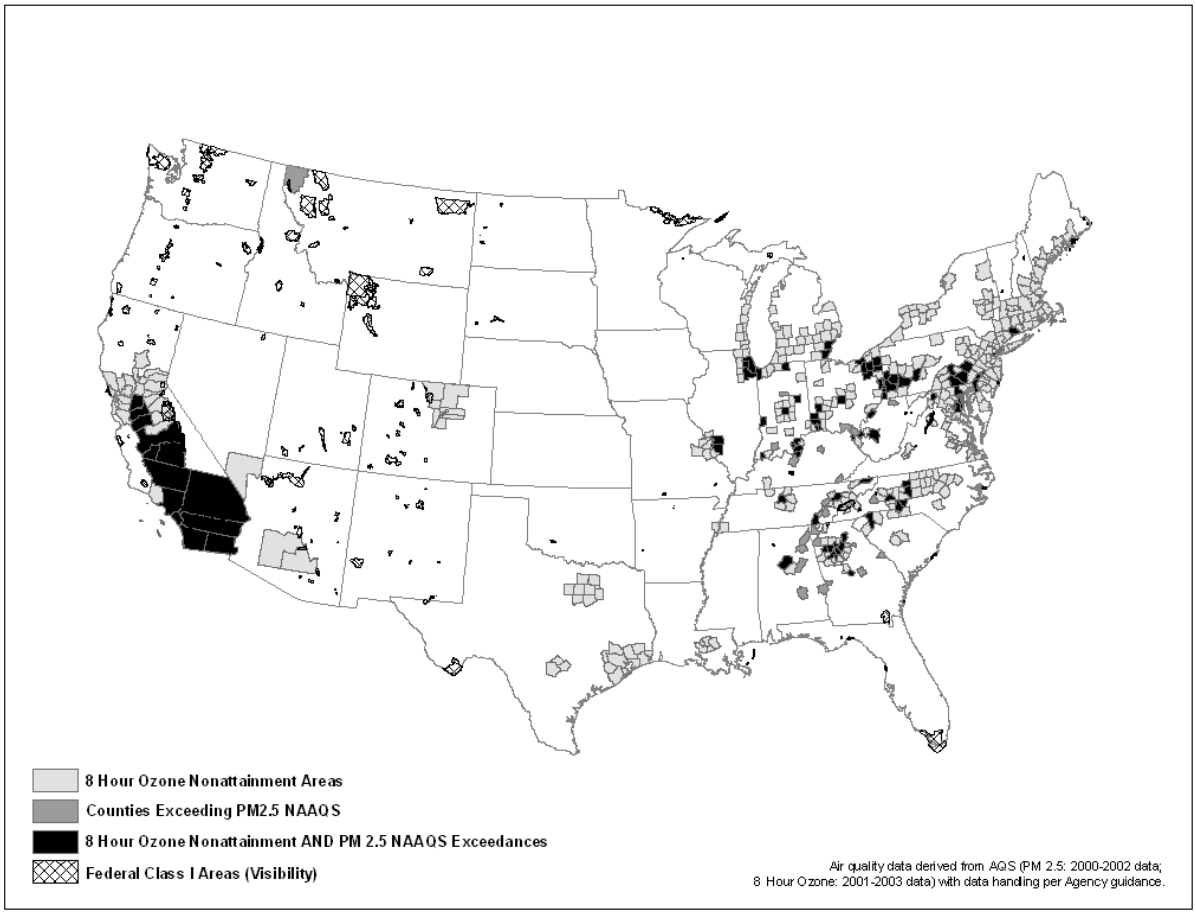
These sources are significant contributors to atmospheric pollution of (among other pollutants) PM, ozone and a variety of toxic air pollutants. In 1996, emissions from these four source categories were estimated to be 40 percent of the mobile source inventory for $PM_{2.5}$ and 25 percent for NOx. Without further control beyond those we have already adopted, by the year 2030, these sources will emit 44 percent of $PM_{2.5}$ from mobile sources, and 47 percent of NOx emissions from mobile sources. Thus, reducing emissions from nonroad sources is critically important to achieving the nation's air quality goals.

In 2030, we estimate that this program will reduce over 129,000 tons $PM_{2.5}$ and 738,000 tons of NOx. It will also virtually eliminate nonroad diesel SO_2 emissions, which amounted to approximately 236,000 tons in 1996, and would otherwise grow to approximately 379,000 tons by 2030.

These dramatic reductions in nonroad emissions are a critical part of the effort by Federal, State, local and Tribal governments to reduce the health related impacts of air pollution and to reach attainment of the National Ambient Air Quality Standard (NAAQS) for PM and ozone, as well as to improve environmental effects such as visibility. These emission reductions will be directly helpful to the 474 partial and full counties nationwide that have been recently designated as nonattainment areas for the 8-hour ozone standard and the $PM_{2.5}$ areas that will be designated later this year. Based on the most recent monitoring data available for this rule, such problems are widespread in the United States. There are almost 65 million people living in 120 counties with $PM_{2.5}$ levels exceeding the $PM_{2.5}$ NAAQS (based on 2000-2002), and about 159 million people living in 474 partial and full counties that are in nonattainment for either failing to meet the 8-hour ozone NAAQS or for contributing to poor air quality in a nearby area. Figure 2.-1 illustrates the widespread nature of these problems. Shown in this figure are counties exceeding either or both of the $PM_{2.5}$ NAAQS or designated 8-hour ozone nonattainment areas plus mandatory Federal Class I areas, which have particular needs for reductions in haze.

As described in Chapter 9, the air quality improvements expected from this rulemaking will produce major benefits to human health and welfare, with a combined value in excess of three quarters of a trillion dollars between 2007 and 2036. By the year 2030, we expect that this rule will annually prevent approximately 12,000 premature deaths and 15,000 nonfatal heart attacks. By 2030, it will also prevent 13,000 annual acute bronchitis attacks in children, 280,000 upper and lower respiratory symptoms in children, nearly 1 million lost work days among adults because of their own symptoms, and 5.9 million days where adults have to restrict their activities due to symptoms in 2030.

Figure I-1. Air Quality Problems are Widespread.



In this chapter and chapter 3, we describe in more detail the air pollution problems associated with emissions from nonroad diesel engines and air quality information that we are relying upon in this rulemaking. To meet these emission standards, engine manufacturers directly control emissions of NOx, PM, non-methane hydrocarbons (NMHC), and to a lesser extent, carbon monoxide (CO). Gaseous air toxics from nonroad diesel engines will also decrease as a consequence of the new emission standards. In addition, there will be a substantial reduction in

SO_2 emissions resulting from the decreasing sulfur level in diesel fuel. SO_2 is transformed in the atmosphere to form PM (sulfate) and can also pose a public health hazard in the gas phase.

From a public health perspective, we are primarily concerned with nonroad engine contributions to atmospheric levels of particulate matter in general (diesel PM in particular), various gaseous air toxics emitted by diesel engines, and ozone.[A] We will first review important public health effects caused by these pollutants, briefly describing the human health effects, and we will then review the current and expected future ambient levels of directly or indirectly caused pollution. Our presentation will show that substantial further reductions of these pollutants, and the underlying emissions from nonroad diesel engines, will be needed to protect public health.

Following discussion of health effects, we will discuss a number of welfare effects associated with emissions from diesel engines. These effects include atmospheric visibility impairment, ecological and property damage caused by acid deposition, eutrophication and nitrification of surface waters, environmental threats posed by polycyclic organic matter (POM) deposition, and plant and crop damage from ozone. Once again, the information available to us indicates a continuing need for further nonroad emission reductions to bring about improvements in air quality.

2.1 Particulate Matter

Particulate matter (PM) represents a broad class of chemically and physically diverse substances. It can be principally characterized as discrete particles that exist in the condensed (liquid or solid) phase spanning several orders of magnitude in size. PM_{10} refers to particles with an aerodynamic diameter less than or equal to a nominal 10 micrometers. Fine particles refer to those particles with an aerodynamic diameter less than or equal to a nominal 2.5 micrometers (also known as $PM_{2.5}$), and coarse fraction particles are those particles with an aerodynamic diameter greater than 2.5 microns, but less than or equal to a nominal 10 micrometers. Ultrafine PM refers to particles with diameters of less than 100 nanometers (0.1 micrometers). The health and environmental effects of PM are in some cases related to the size of the particles. Specifically, larger particles (greater than 10 micrometers) tend to be deposited nasally and in the larger conducting airways, and they are removed by the respiratory clearance mechanisms whereas smaller particles (PM_{10}) are deposited deeper in the lungs. Also, fine particles scatter light obstructing visibility.

In addition to directly emitted particles, nonroad diesel engines currently emit high levels of NOx, which reacts in the atmosphere to form secondary $PM_{2.5}$ (namely ammonium nitrate).

[A]Ambient PM from nonroad diesel engine is associated with the direct emission of diesel PM and sulfate PM, and with PM formed indirectly in the atmosphere by NOx and SO_2 emissions (and to a lesser extent NMHC emissions). Both NOx and NMHC can participate in the atmospheric chemical reactions that produce ozone.

Nonroad diesel engines also emit SO_2 and HC, which react in the atmosphere to form secondary $PM_{2.5}$ (namely sulfates and organic carbonaceous $PM_{2.5}$). Both types of directly and indirectly formed particles from nonroad engines are found principally in the fine fraction. Thus, this discussion will focus on fine particles ($PM_{2.5}$). Ambient fine particles are a complex mixture generally composed of sulfate, nitrate, chloride, ammonium compounds, organic carbon, elemental carbon, and metals. Fine particles can remain in the atmosphere for days to weeks and travel through the atmosphere hundreds to thousands of kilometers, while coarse particles generally tend to deposit to the earth within minutes to hours and within tens of kilometers from the emission source.

2.1.1 Health Effects of Particulate Matter

Scientific studies show ambient PM concentrations (which are attributable to a number of sources including diesel) contribute to a series of adverse health effects. These health effects are discussed in detail in the EPA Air Quality Criteria Document for PM (PM Criteria Document) as well as the draft updates of this document released in the past year.[1] EPA's Health Assessment Document for Diesel Engine Exhaust (Diesel HAD) also reviewed health effects information related to diesel exhaust as a whole including diesel PM, which is one component of ambient PM.[2] We are relying on the data and conclusions in these documents regarding the effects of particulate matter. We also present additional recent studies. Taken together this information supports the conclusion that PM-related emissions from nonroad diesel engines have been associated with adverse health effects.

We received a number of public comments on specific health studies, and we are relying on the discussions and conclusions presented in the PM Criteria Document and Diesel HAD in which EPA prepared detailed evaluations of the body of scientific information and subjected those evaluations to extensive public and expert peer review. Additional information is available in the Summary and Analysis of Public Comments that accompanies this final rule.

2.1.1.1 Short-Term Exposure-Mortality and Morbidity Studies

As detailed in the PM Criteria Document, health effects associated with short-term variation in ambient PM have been indicated by numerous epidemiologic studies showing associations between exposure and increased hospital admissions for ischemic heart disease,[3] heart failure,[4] respiratory disease,[5, 6, 7, 8] including chronic obstructive pulmonary disease (COPD) and pneumonia.[9, 10, 11] Short-term elevations in ambient PM have also been associated with increased cough, lower respiratory symptoms, and decrements in lung function.[12, 13, 14] Short-term variations in ambient PM have also been associated with increases in total and cardiorespiratory daily mortality in individual cities[15, 16, 17, 18] and in multi-city studies.[19, 20, 21]

Several studies specifically address the contribution of PM from mobile sources in these time-series studies. Analyses incorporating source apportionment by factor analysis with daily time-series studies of daily death also established a specific influence of mobile source-related $PM_{2.5}$ on daily mortality[22] and a concentration-response function for mobile source-associated $PM_{2.5}$ and daily mortality.[23] Another recent study in 14 U.S. cities examined the effect of PM_{10}

exposures on daily hospital admissions for cardiovascular disease (CVD). They found that the effect of PM_{10} was significantly greater in areas with a larger proportion of PM_{10} coming from motor vehicles, indicating that PM_{10} from these sources may have a greater effect on the toxicity of ambient PM_{10} when compared with other sources.[24]

In 2002, questions were raised about the default convergence criteria and standard error calculations made using generalized additive models (GAM), which has been the statistical model of choice in many of the time-series epidemiologic studies. A number of time-series studies were reanalyzed using alternative methods, typically GAM with more stringent convergence criteria and an alternative model such as generalized linear models (GLM) with natural smoothing splines. Since then, the Health Effects Institute convened an expert panel to review the results of and the results of the reanalyses have been compiled and reviewed in a recent HEI publication.[25] In most, but not all, of the reanalyzed studies, it was found that risk estimates were reduced and confidence intervals increased with the use of GAM with more stringent convergence criteria or GLM analyses; however, the reanalyses generally did not substantially change the findings of the original studies, and the changes in risk estimates with alternative analysis methods were much smaller than the variation in effects across studies. The HEI review committee concluded the following:

a. While the number of studies showing an association of PM with mortality was slightly smaller, the PM association persisted in the majority of studies.
b. In some of the large number of studies in which the PM association persisted, the estimates of PM effect were substantially smaller.
c. In the few studies in which investigators performed further sensitivity analyses, some showed marked sensitivity of the PM effect estimate to the degree of smoothing and/or the specification of weather.

As discussed in Chapter 9, examination of the original studies used in our economic benefits analysis found that the health endpoints that are potentially affected by the GAM issues include: reduced hospital admissions, reduced lower respiratory symptoms, and reduced premature mortality due to short-term PM exposures. It is important to note that the benefits estimates derived from the long-term exposure studies, which account for a major share of the economic benefits described in Chapter 9, are not affected. Similarly, the time-series studies and case-crossover studies employing generalized linear models or other parametric methods are not affected.

2.1.1.2 Long-Term Exposure Mortality and Morbidity Studies

Short-term studies provide one way of examining the effect of short-term variations in air quality on morbidity and mortality. However, they do not allow for an evaluation of the effect of long-term exposure to air pollution on human mortality and morbidty.[26] Longitudinal cohort studies allow for analysis of such effects.

As discussed in the PM Criteria Document, the newer morbidity studies that combine the features of cross-sectional and cohort studies provide the best evidence for chronic exposure

effects. The Gauderman *et al.* studies both found significant decreases in lung function growth among southern California school children to be related to $PM_{2.5}$ and/or PM_{10} levels.[27],[28] However, Peters *et al.* reported no relationship between respiratory symptoms and annual average PM_{10} levels in 12 southern California communities.[29] Long-term (months to years) exposure to PM was linked with decreased lung function and increased incidence of respiratory disease such as bronchitis (PM Criteria Document 1996, p. V-26, Abbey et al. 1995). The results of studies using long-term and short-term PM exposure data were reported to be consistent with one another. In addition, toxicology studies using surrogate particles or PM components, generally at high concentrations, and autopsy studies of humans and animals reported evidence of pulmonary effects, including morphological damage (e.g., changes in cellular structure of the airways) and changes in resistance to infection.

Additional data are available regarding long-term PM exposures and mortality. To date, four major cohorts in the U.S. have examined mortality and long-term exposure to $PM_{2.5}$. These studies are described in detail in the PM Criteria Document and we are relying on the analyses and conclusions in that document for these studies. Many of the issues raised in public comment are addressed by the Criteria Document (as detailed in the Summary and Analysis of public comments document.) In addition to the U.S. studies, there are additional data from Europe and Canada. A cohort in the Netherlands evaluated exposure to mobile source-related pollutants.[30] Another study examines exposure-mortality relationships with income in southern Ontario, Canada.[31]

Two major U.S. cohort studies, the Harvard Six Cities and the American Cancer Society studies, suggest an association between exposure to ambient $PM_{2.5}$ measured in the city of residence and premature mortality from cardiorespiratory causes.[32, 33] As discussed in the PM Criteria Document, these two prospective cohort studies tracked health outcomes in discrete groups of people over time. Subsequent reanalysis of these studies have confirmed the findings of these articles, and a recent extension of the ACS cohort study found statistically significant increases in lung cancer mortality risk associated with ambient $PM_{2.5}$.[34] This most recent finding is of special interest in this rulemaking, because of the association of diesel exhaust and lung cancer in occupational studies of varying design.

More recently, the Adventist Health Study on Smog (AHSMOG) in California indicated that long-term exposure to PM_{10} resulted in a significant risk of premature mortality in men, although risks were not elevated among women.[35] In another AHSMOG analysis, ambient $PM_{2.5}$ estimates made from visibility data at an airport were used to compare the effects of PM_{10} and $PM_{2.5}$ for the cohort.[36] No statistically significant increase in risk was observed with any component of PM. Among men, the $PM_{2.5}$ coefficient on mortality from all natural causes was consistently larger than the coarse fraction of PM_{10}. Among women, no elevation in mortality risk was found for any PM index.

Another study evaluated in the PM Criteria Document examining long-term exposure to ambient PM and mortality is the Electric Power Research Institute (EPRI)-Washington University mortality study in American Veterans.[37] The Veterans Study was originally designed as a means of assessing the efficacy of anti-hypertensive drugs in reducing morbidity and

mortality in a population with pre-existing high blood pressure (in this case, male veterans) (Lipfert et al., 2000). Unlike previous long-term analyses, this study found some associations between premature mortality and ozone but found inconsistent results for PM indicators. A variety of issues associated with the study design, including sample representativeness and loss to follow up, make this cohort a poor choice for extrapolating to the general public. Furthermore, the selective nature of the population in the veteran's cohort and methodological weaknesses may have resulted in estimates of relative risk that are biased relative to a relative risk for the general population.

The Hoek et al. (2002) study examines a cohort of residents of the Netherlands who were recruited as part of the Netherlands Cohort study on Diet and Cancer (NLCS).[38] Five thousand study participants were selected at random from the larger cohort, which consisted of persons aged 55 to 69 in 1986, with follow up until 1994. In 1986, all participants filled out questionnaires on diet and other risk factors. All participants with full questionnaire data were included in the study. Each participants' home address was mapped by street address. Individual exposures to ambient pollutants were assigned by matching residential address to an exposure metric via geographic information system (GIS). "Black smoke" – widely used in Europe as a surrogate of particulate elemental carbon – and NO_2 had been previously assessed as a function of regional background, urban background, and contribution from local traffic based on proximity to busy roads.[39] Results of the survival analysis indicated that residential black smoke predicted from regional, urban, and intra-urban variation was associated with a relative risk (RR) of cardiopulmonary mortality per 10 ug/m^3 of 1.71 (with a 95 percent confidence interval (CI) of [1.10, 2.67]) and an RR for all-cause mortality of 1.31 [0.95, 1.80]. In a model including background black smoke and proximity to a major roadway, the cardiopulmonary mortality RR associated with living near a busy road was 1.95 [1.09, 3.51]. This study is of particular interest in this rule, because of the strong focus on mobile source pollutants in the exposure assessment portion of the study. This study also highlights the "near-roadway" health concerns, discussed later.

The Six Cities, ACS, AHSMOG, Veterans, and NLCS Studies are discussed in detail in the draft PM Criteria Document and revised Chapter 8. We are relying on the evaluations and conclusions presented in those documents. The long-term exposure health effects of PM are summarized in Table 2.1.1-1, which is taken directly from Table 9-11 of the draft Air Quality Criteria Document referenced earlier that was released in 2003. This document is continuing to undergo expert and public review. One study discussed below does not appear in the PM Criteria Document because it was published after the date required for inclusion in the Criteria Document.[40]

Finklestein et al. (2003) examined a cohort of 5,228 residents of the Hamilton-Burnling area of southern Ontario, Canada who had been referred for lung function testing between 1985 and 1999.[41] The study was not a random sample of the population in the Hamilton-Burlington area. Total non-accidental and cardiopulmonary mortalities between 1992 and 1999 were determined based on the Ontario Mortality Registry. The subjects' age, sex, postal code, body mass index, and pulmonary function test results were matched with disease diagnosis via the Ontario Health Insurance Plan. Canada's health insurance system allowed the investigators to determine disease

diagnoses during the follow-up period. Postal codes were used to assign "ecological" variables of census-derived mean household income, 24-hour average total suspended particulate (TSP) measured every 6 days, and SO_2 measured continuously during the mid-1990's Air monitoring data came from 9 TSP and 23 SO_2 monitors, which were subject to spatial interpolation techniques. Postal code-specific pollutant concentrations were assigned using GIS. Analysis of the air quality data indicated that TSP and SO_2 tended to be higher in low-income areas. The study group was divided into higher and lower income and pollution strata, based on the median income, and TSP and SO_2 levels at the postal code level. Compared to the high-income, low-pollution group, all other groups had significantly elevated mortality relative risks with income, and each pollutant (in one-pollutant models) was associated with increased risk. Age appeared as an effect modifier, with attenuated effects at elevated age.

The 1996 PM AQCD indicated that past epidemiologic studies of chronic PM exposures collectively indicate increases in mortality to be associated with long-term exposure to airborne particles of ambient origins. The PM effect size estimates for total mortality from these studies also indicated that a substantial portion of these deaths reflected cumulative PM impacts above and beyond those exerted by acute exposure events.

Several advances have been made in terms of further analyses and/or reanalyses of several studies of long-term PM exposure effects on total, cardiopulmonary, or lung cancer mortality. The Harvard Six Cities analyses (as confirmed by the HEI reanalyses) and the recent extension of the ACS study by Pope et al. (2002) probably provide the most credible and precise estimates of excess mortality risk associated with long-term PM2.5 exposures in the United States.

2.1.1.3 Long-Term Exposures and Physiological Response in Individuals

Several studies examined in the PM Criteria Document have examined the effect of long-term exposure to air pollution on individual physiological and organ structure. These studies provide insight into the biological pathways by which air pollution may act to produce adverse health effects. The studies below provide examples of the types of studies examined in the PM Criteria Document.

Studies in Vancouver, BC, and Mexico City, Mexico, have demonstrated increased retention of $PM_{2.5}$ in the lungs of residents of the more highly polluted Mexico City.[42] More recently, comparisons of non-smoking women in Mexico City and Vancouver have shown that particle retention in the lungs of Mexico City women was associated with small airways remodeling.[43] In another study, dogs autopsied in the Mexico City and other less-polluted areas showed that dogs in more polluted areas showed greater respiratory and cardiac pathology indicative of long-term inflammatory stress.[44,45]

One recent study (not addressed in the PM Criteria Document) was conducted in Leicester, UK studying lung cells (alveolar macrophages (AM)) obtained from children undergoing elective surgery.[46] The cells were examined by electron microscope, and the study reported that in all children, some of the AMs contained particles, ranging from 1 to 16 percent of total AM collected. Of particular note, the authors found that a significantly higher fraction of the AM

collected from children living on main roads contained particles as compared to children living on quiet residential roads, and that these particles were composed of single and chain aggregates of ultrafine carbon particles that appeared to be combustion-related. This study is of particular relevance to this rule, given the evidence that exposure to mobile source PM results in greater concentrations of PM in the lung. Given the elevated exposures to carbonaceous PM in occupations that work with nonroad diesel engines (discussed below), this study provides a link between nonroad PM exposure an potential lung and systemic health effects.

2.1.1.4 Studies of Short-Term Exposures and Physiological Response in Individuals

A number of studies have investigated biological processes and physiological effects that may underlie the epidemiologic findings of earlier studies. This research has found associations between short-term changes in PM exposure with changes in heart beat, force, and rhythm, including reduced heart rate variability (HRV), a measure of the autonomic nervous system's control of heart function.[47, 48, 49, 50, 51, 52] The findings indicate associations between measures of heart function and PM measured over the prior 3 to 24 hours or longer. Decreased HRV has been shown to be associated with coronary heart disease and cardiovascular mortality in both healthy and compromised populations.[53, 54, 55, 56]

Other studies have investigated the association between PM and such systemic factors such as inflammation, blood coagulability and viscosity. It is hypothesized that PM-induced inflammation in the lung may activate a "non-adaptive" response by the immune system, resulting in increased markers of inflammation in the blood and tissues, heightened blood coagulalability, and leukocyte count in the blood. A number of studies have found associations between controlled exposure to either concentrated or ambient PM or diesel exhaust exposure and pulmonary inflammation.[57, 58, 59, 60] A number of studies have also shown evidence of increased blood markers of inflammation, such as C-reactive protein, fibrinogen, and white blood cell count associated with inter-day variability in ambient PM.[61, 62, 63, 64] These blood indices have been associated with coronary heart disease and cardiac events such as heart attack.[65, 66] Studies have also shown that repeated or chronic exposures to urban PM were associated with increased severity of atherosclerosis, microthrombus formation, and other indicators of cardiac risk.[67, 68]

The recent studies examining inflammation, heart rate and rhythm in relation to PM provide some evidence into the mechanisms by which ambient PM may cause injury to the heart. New epidemiologic data have indicated that short-term changes in ambient PM mass is associated with adverse cardiac outcomes like myocardial infarction (MI) or ventricular arrythmia.[69, 70] These studies provide additional evidence that ambient $PM_{2.5}$ can cause both acute and chronic cardiovascular injury, which can result in death or non-fatal effects.

Table 2.1.1-1
Effect Estimates per Increments[a] in Long-term Mean Levels of
Fine and Inhalable Particle Indicators From U.S. and Canadian Studies

Type of Health Effect and Location	Indicator	Change in Health Indicator per Increment in PM*	Range of City PM Levels ** Means ($\mu g/m^3$)
Increased Total Mortality in Adults		**Relative Risk (95% CI)**	
Six City[B]	$PM_{15/10}$ (20 $\mu g/m^3$)	1.18 (1.06-1.32)	18-47
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.13 (1.04-1.23)	11-30
	$SO_4^=$ (15 $\mu g/m^3$)	1.46 (1.16-2.16)	5-13
ACS Study[C] (151 U.S. SMSA)	$PM_{2.5}$ (10 $\mu g/m^3$)	1.07 (1.04-1.10)	9-34
	$SO_4^=$ (15 $\mu g/m^3$)	1.10 (1.06-1.16)	4-24
Six City Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$)	1.19 (1.06-1.34)	18.2-46.5
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.13 (1.04-1.23)	11.0-29.6
ACS Study Reanalysis[D]	$PM_{15/10}$ (20 $\mu g/m^3$) (dichot)	1.04 (1.01-1.07)	58.7 (34-101)
	$PM_{2.5}$ (10 $\mu g/m^3$)	1.07 (1.04-1.10)	9.0-33.4
ACS Study Extended Analyses[Q]	$PM_{2.5}$ (10 $\mu g/m^3$)	1.04 (1.01-1.08)	21.1 (SD=4.6)
Southern California[E]	PM_{10} (20 $\mu g/m^3$)	1.091 (0.985-1.212) (males)	51 (±17)
	PM_{10} (cutoff = 30 days/year >100 $\mu g/m^3$)	1.082 (1.008-1.162) (males)	
	PM_{10} (20 $\mu g/m^3$)	0.950 (0.873-1.033) (females)	51 (±17)
	PM_{10} (cutoff = 30 days/year >100 $\mu g/m^3$)	0.958 (0.899-1.021) (females)	
Vetrans Cohort[R]	$PM_{2.5}$ (10 $\mu g/m^3$)	0.90 (0.85, 0.954; males)	5.6-42.3
Increased Bronchitis in Children		**Odds Ratio (95% CI)**	
Six City[F]	$PM_{15/10}$ (50 $\mu g/m^3$)	3.26 (1.13, 10.28)	20-59
Six City[G]	TSP (100 $\mu g/m^3$)	2.80 (1.17, 7.03)	39-114
24 City[H]	H^+ (100 $nmol/m^3$)	2.65 (1.22, 5.74)	6.2-41.0
24 City[H]	$SO_4^=$ (15 $\mu g/m^3$)	3.02 (1.28, 7.03)	18.1-67.3
24 City[H]	$PM_{2.1}$ (25 $\mu g/m^3$)	1.97 (0.85, 4.51)	9.1-17.3
24 City[H]	PM_{10} (50 $\mu g/m^3$)	3.29 (0.81, 13.62)	22.0-28.6
Southern California[I]	$SO_4^=$ (15 $\mu g/m^3$)	1.39 (0.99, 1.92)	—
12 Southern California communities[J] (all children)	PM_{10} (25 $\mu g/m^3$) Acid vapor (1.7 ppb)	0.94 (0.74, 1.19) 1.16 (0.79, 1.68)	28.0-84.9 0.9-3.2 ppb
12 Southern California communities[K] (children with asthma)	PM_{10} (19 $\mu g/m^3$) $PM_{2.5}$ (15 $\mu g/m^3$) Acid vapor (1.8 ppb)	1.4 (1.1, 1.8) 1.4 (0.9, 2.3) 1.1 (0.7, 1.6)	13.0-70.7 6.7-31.5 1.0-5.0 ppb

Type of Health Effect and Location	Indicator	Change in Health Indicator per Increment in PM*	Range of City PM Levels ** Means ($\mu g/m^3$)
Increased Cough in Children		**Odds Ratio (95% CI)**	
12 Southern California communities[J] (all children)	PM_{10} (20 $\mu g/m^3$) Acid vapor (1.7 ppb)	1.05 (0.94, 1.16) 1.13 (0.92, 1.38)	28.0-84.9 0.9-3.2 ppb
12 Southern California communities[K] (children with asthma)	PM_{10} (20 $\mu g/m^3$) $PM_{2.5}$ (10 $\mu g/m^3$) Acid vapor (1.8 ppb)	1.1 (0.7, 1.8) 1.2 (0.8, 1.8) 1.4 (0.9, 2.1)	13.0-70.7 6.7-31.5 1.0-5.0 ppb
10 Canadian Communities[s]	PM_{10} (20 $\mu g/m^3$)	1.19 (1.04,1.35)	13-23
Increased Wheeze in Children			
10 Canadian Communities[s]	PM_{10} (20 $\mu g/m^3$)	1.35 (1.10,1.64)	13-23
Increased Airway Obstruction in Adults			
Southern California[L]	PM_{10} (20$\mu g/m^3$)	1.09 (0.92, 1.30)	NR
Decreased Lung Function in Children			
Six City[F]	$PM_{15/10}$ (50 $\mu g/m^3$)	NS Changes	20-59
Six City[G]	TSP (100 $\mu g/m^3$)	NS Changes	39-114
24 City[M]	H^+ (52 nmoles/m^3)	−3.45% (-4.87, -2.01) FVC	6.2-41.0
24 City[M]	$PM_{2.1}$ (15 $\mu g/m^3$)	−3.21% (-4.98, -1.41) FVC	18.1-67.3
24 City[M]	$SO_4^=$ (7 $\mu g/m^3$)	−3.06% (-4.50, -1.60) FVC	9.1-17.3
24 City[M]	PM_{10} (17 $\mu g/m^3$)	−2.42% (-4.30, -.0.51) FVC	22.0-28.6
12 Southern California communities[N] (all children)	PM_{10} (25 $\mu g/m^3$) Acid vapor (1.7 ppb)	−24.9 (-47.2, -2.6) FVC −24.9 (-65.08, 15.28) FVC	28.0-84.9 0.9-3.2 ppb
12 Southern California communities[N] (all children)	PM_{10} (25 $\mu g/m^3$) Acid vapor (1.7 ppb)	−32.0 (-58.9, -5.1) MMEF −7.9 (-60.43, 44.63) MMEF	28.0-84.9 0.9-3.2 ppb
12 Southern California communities[O] (4^{th} grade cohort)	PM_{10} (51.5 $\mu g/m^3$) $PM_{2.5}$ (25.9 $\mu g/m^3$) $PM_{10-2.5}$ (25.6 $\mu g/m^3$) Acid vapor (4.3 ppb)	−0.58 (-1.14, -0.02) FVC growth −0.47 (-0.94, 0.01) FVC growth −0.57 (-1.20, 0.06) FVC growth −0.57 (-1.06, -0.07) FVC growth	NR
12 Southern California communities[O] (4^{th} grade cohort)	PM_{10} (51.5 $\mu g/m^3$) $PM_{2.5}$ (25.9 $\mu g/m^3$) $PM_{10-2.5}$ (25.6 $\mu g/m^3$) Acid vapor (4.3 ppb)	−1.32 (-2.43, -0.20) MMEF growth −1.03 (-1.95, -0.09) MMEF growth −1.37 (-2.57, -0.15) MMEF growth −1.03 (-2.09, 0.05) MMEF growth	NR

Type of Health Effect and Location	Indicator	Change in Health Indicator per Increment in PM*	Range of City PM Levels ** Means ($\mu g/m^3$)
Lung Function Changes in Adults			
Southern California[P] (% predicted FEV_1, females)	PM_{10} (cutoff of 54.2 days/year >100 $\mu g/m^3$)	+0.9 % (-0.8, 2.5) FEV_1	52.7 (21.3, 80.6)
Southern California[P] (% predicted FEV_1, males)	PM_{10} (cutoff of 54.2 days/year >100 $\mu g/m^3$)	+0.3 % (-2.2, 2.8) FEV_1	54.1 (20.0, 80.6)
Southern California[P] (% predicted FEV_1, males whose parents had asthma, bronchitis, emphysema)	PM_{10} (cutoff of 54.2 days/year >100 $\mu g/m^3$)	−7.2 % (-11.5, -2.7) FEV_1	54.1 (20.0, 80.6)
Southern California[P] (% predicted FEV_1, females)	$SO_4^=$ (1.6 $\mu g/m^3$)	Not reported	7.4 (2.7, 10.1)
Southern California[P] (% predicted FEV_1, males)	$SO_4^=$ (1.6 $\mu g/m^3$)	−1.5 % (-2.9, -0.1) FEV_1	7.3 (2.0, 10.1)

*Results calculated using PM increment between the high and low levels in cities, or other PM increments given in parentheses; NS Changes = No significant changes.

**Range of mean PM levels given unless, as indicated, studies reported overall study mean (min, max), or mean (±SD); NR=not reported.

*** Results only for smoking category subgroups.

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2.1.1.6 Roadway-Related Exposure and Health Studies

A recent body of studies has suggested a link between residential proximity to heavily-trafficked roadways (where diesel engines are operated) and adverse health effects. While many of these studies did not measure PM specifically, they include potential exhaust exposures which include mobile source PM because they employ exposure indices such as roadway proximity or traffic volumes.

Based on extensive emission characterization studies and as reviewed in the EPA Diesel HAD (Health Assessment Document for Diesel Exhaust), diesel PM is found principally in the fine fraction (both primary and secondarily formed PM).[71, 72] In addition, in the Diesel HAD, we note that the particulate characteristics in the zone around nonroad diesel engines is likely to be substantially the same as published air quality measurements made along busy roadways. This conclusion supports the relevance of health effects associated with on-road diesel engine-generated PM to nonroad applications. Thus, near roadway studies are relevant to understanding potential health impacts of emissions from nonroad diesel engines.

Specifically, in a recent body of studies, scientists have examined health effects associated with living near major roads. As discussed above, a Dutch cohort study recently developed estimates of the relative risk of cardiopulmonary and all-cause mortality associated with living near a busy roadway.[73] The study found a statistically significant excess risk of cardiopulmonary mortality of 95 percent (i.e., a relative risk of 1.95, 95% CI: 1.09-3.52) associated with living near a busy road. A recent British ecological study examined mortality attributable to stroke in England and Wales.[74] After adjusting for potential confounders, the study found a significantly greater rate of mortality in men and women living within 200 meters of a busy road of 7 percent [95% CI on RR: 1.04 to 1.09] and 4 percent [95% CI on RR: 1.02-1.06], respectively. Risks decreased with increased distance from roadways. However, being an ecological study design, it is impossible to rule out confounding variables.

Other studies relate the incidence or prevalence of respiratory health outcomes to roadway proximity. Several studies have found positive associations between respiratory symptoms and residential roadway proximity or traffic volume. Most recently, a study in U.S. veterans living

in southeastern Massachusetts found significant increases in self-reported respiratory symptoms among subjects living within 50 meters of a major road.[75]

A Dutch cohort study following infants from birth found that traffic-related pollutant concentrations found positive associations with respiratory symptoms, several illnesses, and physician-diagnosed asthma, the last of which was significant for diagnoses prior to 1 year of age.[76]

In a case-control study of children under 14 years old in San Diego, CA, with asthma diagnosis was confirmed by Medicaid claims, no associations between odds of physician diagnosis of asthma and traffic was found.[77] However, a case-based analysis of the data associated traffic flows with an increased number of medical visits among children with asthma.

A case-control study of children aged 4 to 48 months diagnosed with wheezing bronchitis included exposures predicted from traffic data, dispersion models of NO_2 as a marker of mobile source emissions, and included separate exposures for home and day care.[78] Analyses found that cases had significantly elevated NO_2 exposures compared with controls, but only among girls. A significant trend with NO_2 was reported.

Two cross-sectional studies of self-reported wheezing and allergic rhinitis symptoms in German aged 12 to 15 years found increased prevalence of wheezing and allergic rhinitis based on subject-reported frequency of truck traffic.[79,80]

A cross-sectional study in the Netherlands examined self-reported respiratory diagnoses, allergies, and respiratory symptoms in association with annual truck and automobile density, living within 100 meters of a freeway, and indoor measures of air pollution (black smoke, NO_2).[81] The study found associations for truck traffic density with wheeze and asthma attacks in girls but not boys. Associations among girls but not boys were also found for homes within 100 m of a freeway and chronic cough, wheeze, and rhinitis. Physician-diagnosed asthma was not associated with traffic-related exposures. Physician-diagnosed allergy was inversely associated with NO_2 and black smoke.

A cross-sectional study in Surrey, England, compared city wards transected by freeways and those not transected by freeways.[82] Respiratory symptoms in the past year and self-reported diagnosis of asthma by a physician was not associated with any respiratory metric.

A recent review of epidemiologic studies examining associations between asthma and roadway proximity concluded that some coherence was evident in the literature, indicating that asthma, lung function decrement, respiratory symptoms, and atopic illness appear to be higher among people living near busy roads.[83] Other studies have shown children living near roads with high truck traffic density have decreased lung function and greater prevalence of lower respiratory symptoms compared with children living on other roads.[84]

Another recently published study from Los Angeles, CA, found that maternal residence near heavy traffic during pregnancy is associated with adverse birth outcomes, such as preterm birth

and low birth weight.[85] However, these studies are not specifically related to PM, but to fresh emissions from mobile sources, which includes other components as well.

Other studies have shown that living near major roads results in substantially higher exposures to ultrafine particles. A British study found that in the lungs of children living near major roads in Leicester, UK, a significantly higher proportion of the alveolar macrophages contained PM compared with children living on quiet streets.[86] All particles observed in the lungs of children were carbon particles under 0.1 um, which are known to be emitted from diesel engines and other mobile sources. This study is consistent with recent studies of ultrafine particle concentrations around major roads in Los Angeles, CA and Minnesota, which found that concentrations of the smallest particles were substantially elevated near roadways with diesel traffic.[87, 88, 89]

The particulate characteristics in the zone around nonroad diesel engines is not likely to differ substantially from published air quality measurements made along busy roadways; thus, these studies are relevant to the diesel exhaust emissions from nonroad diesel engines. While these studies do not specifically examine nonroad diesel engines, several observations may be drawn. First, nonroad diesel engine emissions are similar in their emission characteristics to on-road motor vehicles. Secondly, exposures from nonroad engines may actually negatively bias these studies, because exposures from nonroad sources are not accounted for, and therefore reduce the study's statistical power. Third, certain populations that are exposed directly to fresh nonroad diesel exhaust are exposed at greater concentrations than those found in studies among the general population. These groups include workers in the construction, timber, mining, and agriculture industries, and members of the general population that spend a large amount of time near areas where diesel engine emissions are most densely clustered, such as residents in buildings near large construction sites.

2.1.2 Attainment and Maintenance of the PM_{10} and $PM_{2.5}$ NAAQS: Current and Future Air Quality

2.1.2.1 Current PM Air Quality

There are NAAQS for both PM_{10} and $PM_{2.5}$. Violations of the annual $PM_{2.5}$ standard are much more widespread than are violations of the PM_{10} standards. Emission reductions needed to attain the $PM_{2.5}$ standards will also assist in attaining and maintaining compliance with the PM_{10} standards. Thus, since most PM emitted by nonroad diesel engines is in the fine fraction of PM, the emission controls resulting from this final rule will contribute to attainment and maintenance of the existing PM NAAQS. More broadly, the new standards will benefit public health and welfare through reductions in direct diesel PM and reductions of NOx, SOx, and HCs that contribute to secondary formation of PM. As described above, diesel particles from nonroad diesel engines are a component of both coarse and fine PM, but fall mainly in the fine (and even ultrafine) size range.

The emission reductions from this final rule will assist States as they work with EPA through implementation of local controls including the development and adoption of additional controls as needed to help their areas attain and maintain the standards.

2.1.2.1.1 PM_{10} Levels

The current NAAQS for PM_{10} were established in 1987. The primary (health-based) and secondary (public welfare based) standards for PM_{10} include both short- and long-term NAAQS. The short-term (24-hour) standard of 150 $\mu g/m^3$ is not to be exceeded more than once per year on average over three years. The long-term standard specifies an expected annual arithmetic mean not to exceed 50 $\mu g/m^3$ averaged over three years.

Currently, 29.3 million people live in PM_{10} nonattainment areas, including moderate and serious areas. There are presently 56 moderate PM_{10} nonattainment areas with a total population of 6.6 million.[90] The attainment date for the initial moderate PM_{10} nonattainment areas, designated by law on November 15, 1990, was December 31, 1994. Several additional PM_{10} nonattainment areas were designated on January 21, 1994, and the attainment date for these areas was December 31, 2000.

There are 8 serious PM_{10} nonattainment areas with a total affected population of 22.7 million. According to the Act, serious PM_{10} nonattainment areas must attain the standards no later than 10 years after designation. The initial serious PM_{10} nonattainment areas were designated January 18, 1994 and had an attainment date set by the Act of December 31, 2001. The Act provides that EPA may grant extensions of the serious area attainment dates of up to 5 years, provided that the area requesting the extension meets the requirements of Section 188(e) of the Act. Five serious PM_{10} nonattainment areas (Phoenix, Arizona; Clark County (Las Vegas), NV; Coachella Valley, South Coast (Los Angeles), and Owens Valley, California) have received extensions of the December 31, 2001 attainment date and thus have new attainment dates of December 31, 2006.

Many PM_{10} nonattainment areas continue to experience exceedances. Of the 29.3 million people living in designated PM_{10} nonattainment areas, approximately 24.5 million people are living in nonattainment areas with measured air quality violating the PM_{10} NAAQS in 2000-2002. Among these are 8 serious areas listed in Table 1.2-1 and 6 moderate areas: Nogales, AZ, Imperial Valley, CA, Mono Basin, CA, Coso Junction, CA,[B] Ft. Hall, ID, and El Paso, TX.

[B]On August 6, 2002, EPA finalized certain actions affecting the Searles Valley, California, PM_{10} nonattainment area, which is located in the rural high desert and includes portions of Inyo, Kern, and San Bernardino Counties. The action splits the Searles Valley nonattainment area into three separate areas: Coso Junction, Indian Wells Valley and Trona. EPA's action also determines that the Trona area attained the PM-10 standards by December 31, 1994. On May 7, 2003, EPA finalized approval of the Indian Wells Moderate Area and Maintenance Plan and redesignated the area from nonattainment to attainment for particulate matter (PM-10).

Source: http://www.epa.gov/region9/air/searlespm/index.html

Table 1.2-1
Serious PM_{10} Nonattainment Areas

Area	Attainment Date	2000 Population	2000-2002 Measured Violation
Owens Valley, CA	December 31, 2006	7,000	Yes
Phoenix, AZ	December 31, 2006	3,111,876	Yes
Clark County, NV (Las Vegas)	December 31, 2006	1,375,765	Yes
Coachella Valley, CA	December 31, 2006	225,000	Yes
Los Angeles South Coast Air Basin, CA	December 31, 2006	14,550,521	Yes
San Joaquin Valley, CA	2001	3,080,064	Yes
Walla Walla, WA	2001	10,000	No
Washoe County, NV (Reno)	2001	339,486	No
Total Population	22.7 million		

In addition to these designated nonattainment areas, there are 16 unclassified areas, where 6.2 million live, for which States have reported PM_{10} monitoring data for 2000-2002 period indicating a PM_{10} NAAQS violation. 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 2000-2002 in these 16 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 sources of PM_{10} emissions. Areas that remain unclassified areas 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.

2.1.2.1.2 $PM_{2.5}$ Levels

The need for reductions in the levels of $PM_{2.5}$ is widespread. Figure 2.1.1-4 below shows $PM_{2.5}$ monitoring data highlighting locations measuring concentrations above the level of the NAAQS. As can be seen from that figure, high ambient levels are widespread throughout the country. In addition, there may be counties without monitors that exceed the level of the standard. A listing of available measurements by county can be found in the air quality technical support document (AQ TSD) for the rule.

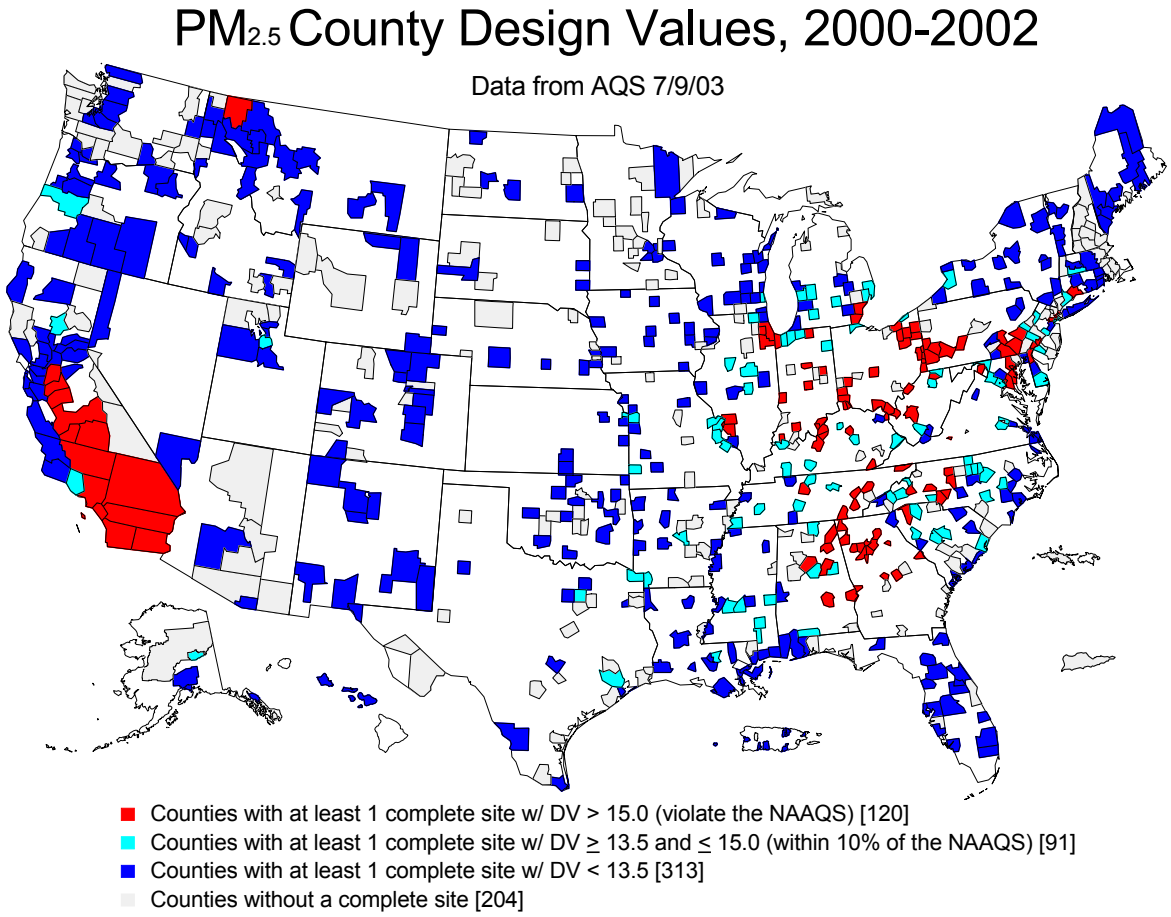
The NAAQS for $PM_{2.5}$ were established in 1997 (62 FR 38651, July 18, 1997). The short term (24-hour) standard is set at a level of 65 $\mu g/m^3$ based on the 98th percentile concentration averaged over three years. (The air quality statistic compared with the standard is referred to as

the "design value.") The long-term standard specifies an expected annual arithmetic mean not to exceed 15 $\mu g/m^3$ averaged over three years.

Current $PM_{2.5}$ monitored values for 2000-2002 indicate that 120 counties in which almost 65 million people live have annual design values that violate the $PM_{2.5}$ NAAQS. In total, this represents 23 percent of the counties and 37 percent of the population with levels above the NAAQS in the areas with monitors that met completeness criteria. An additional 32 million people live in 91 counties that have air quality measurements within 10 percent of the level of the standard. These areas, though not currently violating the standard, will also benefit from the additional reductions from this rule in order to ensure long-term maintenance. There are another 204 counties where 21 million people live that had incomplete data.

Figure 2.1.2-1 is a map of currently available $PM_{2.5}$ monitoring data, highlighting monitor locations near or above the annual $PM_{2.5}$ NAAQS. As can be seen from this figure, high ambient levels are widespread throughout the East and California.

Figure 2.1.2-1


PM2.5 County Design Values, 2000-2002
Data from AQS 7/9/03
Counties with at least 1 complete site w/ DV > 15.0 (violate the NAAQS) [120]
Counties with at least 1 complete site w/ DV ≥ 13.5 and ≤ 15.0 (within 10% of the NAAQS) [91]
Counties with at least 1 complete site w/ DV < 13.5 [313]
Counties without a complete site [204]

Further insights into the need for reductions from this rule can be gained by evaluating counties at various levels above the level of the NAAQS. As shown in Table 2.1.1-3 of the 64.9 million people currently living in counties with measurements above the NAAQS, 18.8 million live in counties above 20 $\mu g/m^3$. In Section 2.1.2.2, we discuss that absent additional controls, our modeling predicts there will continue to be large numbers of people living in counties with PM levels above the standard.

Table 2.1.1-3
2000-2002 Monitored Population[a] Living in Counties with Annual Average[b] $PM_{2.5}$ Concentrations Shown

Measured 2000-2002 Annual Average $PM_{2.5}$ Concentration (µg/m3)	Number of Counties Within The Concentration Range	2000 Population Living in Monitored Counties Within The Concentration Range (Millions, 2000 Census Data)
>25	2	3.3
>20 <=25	6	15.5
>15 <=20	112	46.1
<=15	404	110.9

[a] Monitored population estimates represent populations living in counties with monitors producing data that meet the NAAQS data completeness requirements for 2000 - 2002. This analysis excludes the 204 counties whose monitoring data do not meet the completeness criteria.
[b] Annual average represents the monitor reading with the highest average in each monitored county.
[c] The monitored population is 175.7 million (or 62 percent of the U.S. Census total county-based 2000 population for the U.S. of 281.4 million).

Chemical composition of ambient $PM_{2.5}$ also underscores the contribution of emissions from the engines subject to this rule and points to the need for reductions. Data on $PM_{2.5}$ composition are available from the EPA Speciation Trends Network and the IMPROVE Network for September 2001 to August 2002 covering both urban and rural areas in numerous regions of the United States. The relative contribution of various chemical components to $PM_{2.5}$ varies by region of the country. Figure 2.1.2-2 shows the levels and composition of ambient $PM_{2.5}$ in some urban areas. Figure 2.1.2-3 shows the levels and composition of $PM_{2.5}$ in rural areas where the total $PM_{2.5}$ levels are generally lower. These data show that carbonaceous $PM_{2.5}$ makes up the major component for $PM_{2.5}$ in both urban and rural areas in the Western United States. Carbonaceous $PM_{2.5}$ includes both elemental and organic carbon. Nonroad engines, especially nonroad diesel engines, contribute significantly to ambient $PM_{2.5}$ levels, largely through emissions of carbonaceous $PM_{2.5}$. For the Eastern and middle United States, these data show that carbonaceous $PM_{2.5}$ is a major contributor to ambient $PM_{2.5}$ both urban and rural areas. In some eastern areas, carbonaceous $PM_{2.5}$ is responsible for up to half of ambient $PM_{2.5}$ concentrations.

Figure 2.1.2-2
Annual Average $PM_{2.5}$ Species and Concentrations in Selected Urban Areas
(September 2001- August 2002)

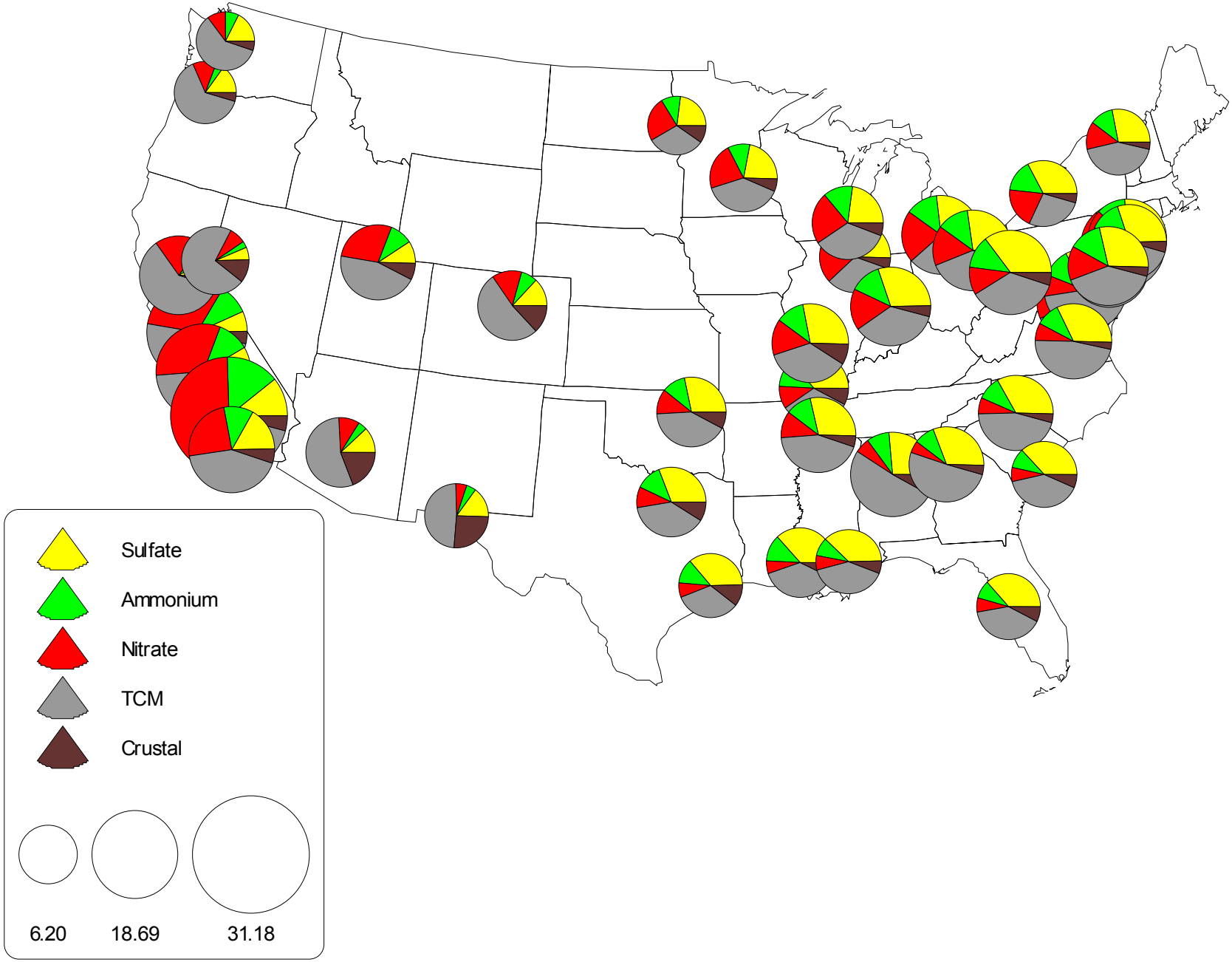
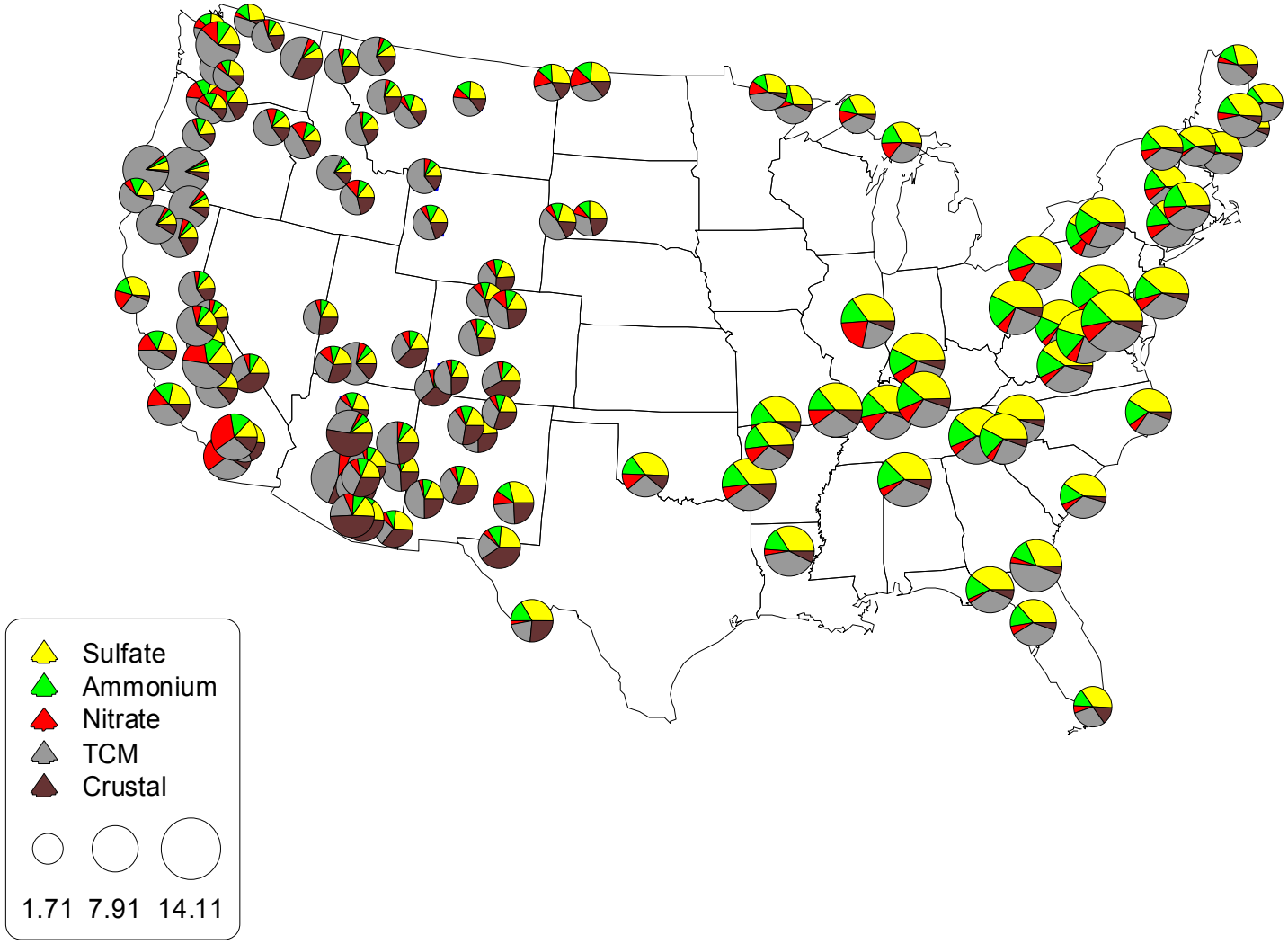


Figure 2.1.2-3
Annual Average PM2.5 Concentration and Species in Rural Areas
(September 2001 - August 2002)



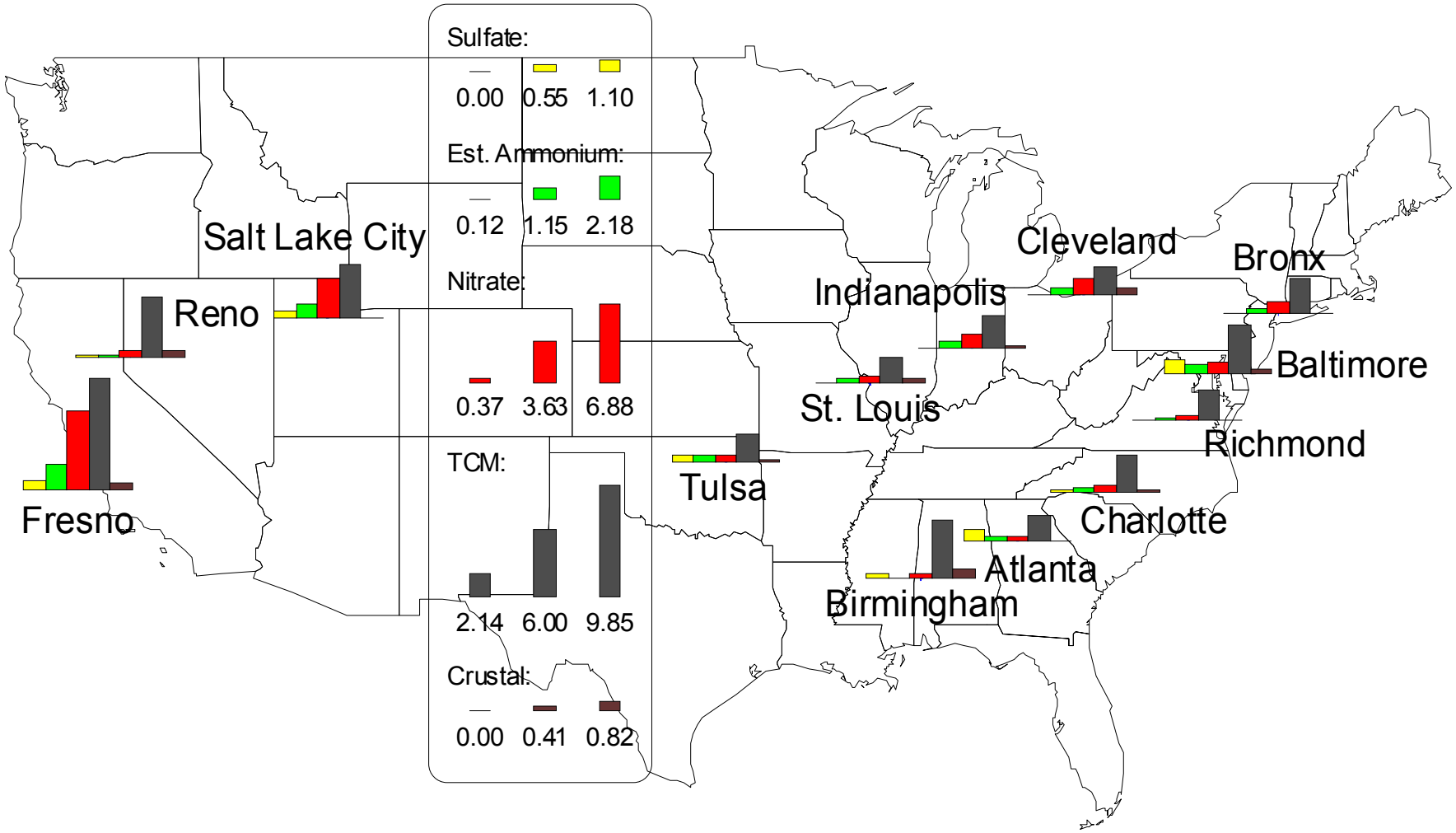
Another important component of PM in the West is nitrates, which are formed from NOx. Nitrates are especially prominent in the California area where it is responsible for about a quarter of the ambient $PM_{2.5}$ concentrations. Nonroad diesel engines also emit high levels of NOx, which reacts in the atmosphere to form secondary $PM_{2.5}$ (namely ammonium nitrate). Sulfate plays a lesser role in these western regions by mass, but it remains important to visibility impairment discussed below. Nonroad diesel engines also emit SO_2 and HC, which react in the atmosphere to form secondary $PM_{2.5}$ (namely sulfates and organic carbonaceous $PM_{2.5}$). Sulfate is also a major contributor to ambient $PM_{2.5}$ in the Eastern United States and in some areas make greater contributions than carbonaceous $PM_{2.5}$.

From Figures 2.1.2-2 and 2.1.2-3, one can compare the levels and composition of $PM_{2.5}$ in various urban areas and a corresponding rural area. This comparison, in Figure 2.1.2-4, shows that much of the excess $PM_{2.5}$ in urban areas (annual average concentration at urban monitor minus annual average concentration at corresponding rural monitor) is indeed from carbonaceous PM.[91, 92] See the AQ TSD for details.

The ambient PM monitoring networks account for both directly emitted PM as well as secondarily formed PM. Emission inventories, which account for directly emitted PM and PM precursors separately, also show that mobile source PM emissions, including that from nonroad diesel engines, is a major contributor to total PM emissions. Nationally, this final rule will significantly reduce emissions of carbonaceous PM. NOx emissions, a prerequisite for formation of secondary nitrate aerosols, will also be reduced. Nonroad diesel engines are major contributors to both of these pollutants. The new requirements in this rule will also reduce SOx and HC. Nonroad diesel engines emissions also contribute to national SOx and HC emission inventories, but to a lesser degree than for PM and NOx. The emission inventories are discussed in detail in Chapter 3.

As discussed in Sections 2.2.2.6 and 2.1, diesel PM also contains small quantities of numerous mutagenic and carcinogenic compounds associated with the particles (and also organic gases). In addition, while toxic trace metals emitted by nonroad diesel engines represent a very small portion of the national emissions of metals (less than one percent) and a small portion of diesel PM (generally less than one percent of diesel PM), we note that several trace metals of potential toxicological significance and persistence in the environment are emitted by diesel engines. These trace metals include chromium, manganese, mercury and nickel. In addition, small amounts of dioxins have been measured in highway engine diesel exhaust, some of which may partition into the particulate phase; dioxins are a major health concern but diesel engines are a minor contributor to overall dioxin emissions. Diesel engines also emit polycyclic organic matter (POM), including polycyclic aromatic hydrocarbons (PAH), which can be present in both gas and particle phases of diesel exhaust. Many PAH compounds are classified by EPA as probable human carcinogens.

Figure 2.1.2-4
Composition of Urban Excess PM2.5 at Selected Sites (September 2001 - August 2002)
(Source: U.S. EPA (2004) AQ TSD; Rao and Frank (2003))



2.1.2.2 Risk of Future Violations

2.1.2.2.1 PM Air Quality Modeling and Methods

In conjunction with this rulemaking, we performed a series of PM air quality modeling simulations for the continental U.S. The model simulations were performed for five emission scenarios: a 1996 baseline projection, a 2020 baseline projection and a 2020 projection with nonroad controls, a 2030 baseline projection and a 2030 projection with nonroad controls. Further discussion of this modeling, including evaluations of model performance relative to predicted future air quality, is provided in the AQ Modeling TSD.

The model outputs from the 1996, 2020 and 2030 baselines, combined with current air quality data, were used to identify areas expected to exceed the $PM_{2.5}$ NAAQS in 2020 and 2030. These areas became candidates for being determined to be residual exceedance areas that will require additional emission reductions to attain and maintain the $PM_{2.5}$ NAAQS. The impacts of the nonroad controls were determined by comparing the model results in the future year control runs against the baseline simulations of the same year. We note that there are significant SO_2 benefits from sulfur reductions in home heating oil fuel that are not accounted for in our modeling. This modeling supports the conclusion that there is a broad set of areas with predicted $PM_{2.5}$ concentrations at or above 15 $\mu g/m^3$ between 1996 and 2030 in the baseline scenarios without additional emission reductions.

The air quality modeling performed for this rule was based upon an improved version of the modeling system used in the HD Engine/Diesel Fuel rule (to address peer-review comments) with the addition of updated inventory estimates for 1996, 2020 and 2030.

A national-scale version of the REgional Model System for Aerosols and Deposition (REMSAD) was utilized to estimate base and future-year PM concentrations over the contiguous United States for the various emission scenarios. Version 7 of REMSAD was used for this rulemaking. REMSAD was designed to calculate the concentrations of both inert and chemically reactive pollutants in the atmosphere that affect annual particulate concentrations and deposition over large spatial scales.[C] Because it accounts for spatial and temporal variations as well as differences in the reactivity of emissions, REMSAD is useful for evaluating the impacts of the final rule on PM concentrations in the United States. The following sections provide an overview of the PM modeling completed as part of this rulemaking. More detailed information is included in the AQ Modeling TSD, which is located in the docket for this rule.

[C] Given the potential impact of the final rule on secondarily formed particles it is important to employ a Eulerian model such as REMSAD. The impact of secondarily formed pollutants typically involves primary precursor emissions from a multitude of widely dispersed sources, and chemical and physical processes of pollutants that are best addressed using an air quality model that employs an Eulerian grid model design. Thus, comments from industry that EPA's methodology form computing benefits over time is based on unsupportable assumptions such as that there will be no interactions between precursors and directly emitted PM in the formation of secondary PM and that EPA excludes consideration of non-linearities in its air quality modeling are incorrect. This air quality modeling for 2020 and 2030 does incorporate the nonlinear interactions between NOx, SO_2, and direct PM.

The PM air quality analyses employed the modeling domain used previously in support of Clear Skies air quality assessment. The domain encompasses the lower 48 States and extends from 126 degrees to 66 degrees west longitude and from 24 degrees to 52 degrees north latitude. The model contains horizontal grid-cells across the model domain of roughly 36 km by 36 km. There are 12 vertical layers of atmospheric conditions with the top of the modeling domain at 16,200 meters.

The simulation periods modeled by REMSAD included separate full-year application for each of the five emission scenarios (1996 base year, 2020 base, 2020 control, 2030 baseline, 2030 control) using the 1996 meteorological inputs described below.

The meteorological data required for input into REMSAD (wind, temperature, surface pressure, etc.) were obtained from a previously developed 1996 annual run of the Fifth-Generation National Center for Atmospheric Research (NCAR) / Penn State Mesoscale Model (MM5). A postprocessor called MM5- REMSAD was developed to convert the MM5 data into the appropriate REMSAD grid coordinate systems and file formats. This postprocessor was used to develop the hourly average meteorological input files from the MM5 output. Documentation of the MM5REMSAD code and further details on the development of the input files is contained in Mansell (2000).[93] A more detailed description of the development of the meteorological input data is provided in the AQ Modeling TSD, which is located in the docket for this rule.

The modeling specified initial species concentrations and lateral boundary conditions to approximate background concentrations of the species; for the lateral boundaries the concentrations varied (decreased parabolically) with height. These initial conditions reflect relatively clean background concentration values. Terrain elevations and land use information was obtained from the U.S. Geological Survey database at 10 km resolution and aggregated to the roughly 36 km horizontal resolution used for this REMSAD application. The development of model inputs is discussed in greater detail in the AQ Modeling TSD, which is available in the docket for this rule.

2.1.2.2.2 Model Performance Evaluation

The purpose of the base year PM air quality modeling was to reproduce the atmospheric processes resulting in formation and dispersion of fine particulate matter across the United States. An operational model performance evaluation for $PM_{2.5}$ and its related speciated components (e.g., sulfate, nitrate, elemental carbon etc.) for 1996 was performed in order to estimate the ability of the modeling system to replicate base year concentrations.

This evaluation is comprised principally of statistical assessments of model versus observed pairs. The robustness of any evaluation is directly proportional to the amount and quality of the ambient data available for comparison. Unfortunately, for 1996 there were few $PM_{2.5}$ monitoring networks with available data for evaluation of the Nonroad PM modeling. Critical limitations of the existing databases are a lack of urban monitoring sites with speciated measurements and poor geographic representation of ambient concentration in the Eastern United States.

The largest available ambient database for 1996 comes from the IMPROVE network. IMPROVE is a cooperative visibility monitoring effort between EPA, federal land management agencies, and state air agencies. Data are collected at Class I areas across the United States mostly at national parks, national wilderness areas, and other protected pristine areas.[94] There were approximately 60 IMPROVE sites that had complete annual $PM_{2.5}$ mass and/or $PM_{2.5}$ species data for 1996. Using the 100th meridian to divide the Eastern and Western United States, 42 sites were located in the West and 18 sites were in the East.

The observed IMPROVE data used for the performance evaluation consisted of $PM_{2.5}$ total mass, sulfate ion, nitrate ion, elemental carbon, organic aerosols, and crustal material (soils). The REMSAD model output species were postprocessed in order to achieve compatibility with the observation species.

The principal evaluation statistic used to evaluate REMSAD performance is the "ratio of the means." It is defined as the ratio of the average predicted values over the average observed values. The annual average ratio of the means was calculated for five individual $PM_{2.5}$ species as well as for total $PM_{2.5}$ mass. The metrics were calculated for all IMPROVE sites across the country as well as for the East and West individually. Table 2.1.2-1 shows the ratio of the annual means. Numbers greater than 1 indicate overpredictions compared with ambient observations (e.g. 1.23 is a 23 percent overprediction). Numbers less than 1 indicate underpredictions.

Table 2.1.2-1
Model Performance Statistics for REMSAD $PM_{2.5}$ Species Predictions: 1996 Base Case

IMPROVE PM Species	Ratio of the Means (annual average concentrations)		
	Nationwide	Eastern U.S.	Western U.S.
$PM_{2.5}$, total mass	0.68	0.85	0.51
Sulfate ion	0.81	0.9	0.61
Nitrate ion	1.05	1.82	0.45
Elemental carbon	1.01	1.23	0.8
Organic aerosols	0.55	0.58	0.53
Soil/Other	1.38	2.25	0.88

Note: The dividing line between the West and East was defined as the 100th meridian.

When considering annual average statistics (e.g., predicted versus observed), which are computed and aggregated over all sites and all days, REMSAD underpredicts fine particulate mass ($PM_{2.5}$) by roughly 30 percent. $PM_{2.5}$ in the Eastern United States is slightly underpredicted, while $PM_{2.5}$ in the West is underpredicted by about 50 percent. Eastern sulfate is slightly underpredicted, elemental carbon is slightly overpredicted, while nitrate and crustal are

largely overpredicted. This is balanced by an underprediction in organic aerosols. Overall the $PM_{2.5}$ performance in the East is relatively unbiased due to the dominance of sulfate in the observations. Western predictions of sulfate, nitrate, elemental carbon, and organic aerosols are all underpredicted.

REMSAD performance is relatively good in the East. The model is overpredicting nitrate, but less so than in previous model applications. The overpredictions in soil/other concentrations in the East can largely be attributed to overestimates of fugitive dust emissions. The model is performing well for sulfate, which is the dominant $PM_{2.5}$ species in most of the East. Organic aerosols are underpredicted in both the East and West. There is a large uncertainty in the current primary organic inventory as well as the modeled production of secondary organic aerosols.

REMSAD is underpredicting all species in the West. The dominant species in the West is organic aerosols. Secondary formation of sulfate, nitrate, and organics appears to be underestimated in the West. Additionally, the current modeling inventory does not contain wildfires, which may be a significant source of primary organic carbon in the West.

It should be noted that $PM_{2.5}$ modeling is an evolving science. There have been few regional or national scale model applications for primary and secondary PM. Unlike ozone modeling, there is essentially no database of past performance statistics against which to measure the performance of this modeling. Given the state of the science relative to PM modeling, it is inappropriate to judge PM model performance using criteria derived for other pollutants, like ozone. Still, the performance of this air quality modeling is encouraging, especially considering that the results are limited by our current knowledge of PM science and chemistry, and by the emission inventories for primary PM and secondary PM precursor pollutants. EPA and others are only beginning to understand the limitations and uncertainties in the current inventories and modeling tools. Improvements to the tools are being made on a continuing basis.

2.1.2.2.3 Results with Areas at Risk of Future $PM_{2.5}$ Violations

Our air quality modeling performed for this rulemaking also indicates that the present widespread number of counties with annual averages above 15 $\mu g/m^3$ are likely to persist in the future in the absence of additional controls. For example, in 2020 based on emission controls currently adopted or expected to be in place, we project that 66 million people will live in 79 counties with average $PM_{2.5}$ levels at and above 15 $\mu g/m^3$. In 2030, the number of people projected to live in areas exceeding the $PM_{2.5}$ standard is expected to increase to 85 million in 107 counties. An additional 24 million people are projected to live in counties with annual averages within 10 percent of the standard in 2020, and 17 million people are projected to live in counties with annual averages within 10 percent of the standard in 2030. The AQ Modeling TSD lists the specifics.

Our modeling also indicates that the reductions from this final rule will make a substantial

contribution to reducing these potential exposures.[D] In 2020, we estimate that the number of people living in counties with $PM_{2.5}$ levels above the NAAQS will be reduced from 66 million to 60 million living in 67 counties. That is a reduction of 9 percent in potentially exposed population and 15 percent of the number of counties. In 2030, there will be an estimated reduction from 85 million people to 71 million living in 84 counties. This represents an even greater improvement than projected for 2020 because of the fleet turnover and corresponds to a 16 percent reduction in potentially exposed population and a 21 percent of the number of counties. Furthermore, our modeling also shows that the emission reductions will assist areas with future maintenance of the standards.

Table 2.1.2-2 lists the counties with 2020 and 2030 projected annual $PM_{2.5}$ design values that violate the annual standard. Counties are marked with an "V" in the table if their projected design values are greater than or equal to 15.05 $\mu g/m^3$. The current 3-year average design values of these counties are also listed. Recall that we project future design values only for counties that have current design values, so this list is limited to those counties with 1999-2001 ambient monitoring data sufficient to calculate current 3-year design values.

[D]The results illustrate the type of PM changes for the preliminary control option, as discussed in Section 3.6. The analysis differs from the modeled control case based on public comment and updated information; however, we believe that the net results would approximate future emissions, though we anticipate the PM reductions might be smaller. We also note that our modeling does not account for substantial reductions in SO_2 associated with sulfur reductions in home heating oil.

Table 2.1.2-2
Counties with 2020 and 2030 Projected Annual PM2.5
Design Values in Violation of the Annual PM2.5 Standard.[a, b]

State	County	1999 - 2001 Design Value (ug/m^3)[b]	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
AL	De Kalb	16.8			V	V	64,452
AL	Houston	16.3	V		V	V	88,787
AL	Jefferson	21.6	V	V	V	V	662,047
AL	Mobile	15.3			V	V	399,843
AL	Montgomery	16.8	V	V	V	V	223,510
AL	Morgan	19.1	V	V	V	V	111,064
AL	Russell	18.4	V	V	V	V	49,756
AL	Shelby	17.2	V	V	V	V	143,293
AL	Talladega	17.8	V	V	V	V	80,321
CA	Fresno	24	V	V	V	V	799,407
CA	Imperial	15.7			V		142,361
CA	Kern	23.7	V	V	V	V	661,645
CA	Los Angeles	25.9	V	V	V	V	9,519,338
CA	Merced	18.9	V	V	V	V	210,554
CA	Orange	22.4	V	V	V	V	2,846,289
CA	Riverside	29.8	V	V	V	V	1,545,387
CA	San Bernardino	25.8	V	V	V	V	1,709,434
CA	San Diego	17.1	V	V	V	V	2,813,833
CA	San Joaquin	16.4			V		563,598
CA	Stanislaus	19.7	V	V	V	V	446,997
CA	Tulare	24.7	V	V	V	V	368,021
CT	New Haven	16.8	V	V	V	V	824,008
DE	New Castle	16.6	V	V	V	V	500,265
DC	Washington	16.6	V	V	V	V	572,059
GA	Bibb	17.6	V	V	V	V	153,887
GA	Chatham	16.5	V	V	V	V	232,048
GA	Clarke	18.6	V	V	V	V	101,489
GA	Clayton	19.2	V	V	V	V	236,517
GA	Cobb	18.6	V	V	V	V	607,751
GA	De Kalb	19.6	V	V	V	V	665,865
GA	Dougherty	16.6	V	V	V	V	96,065
GA	Floyd	18.5	V	V	V	V	90,565
GA	Fulton	21.2	V	V	V	V	816,006
GA	Hall	17.2	V		V	V	139,277
GA	Muscogee	18	V	V	V	V	186,291
GA	Paulding	16.8	V	V	V	V	81,678
GA	Richmond	17.4	V	V	V	V	199,775

State	County	1999 - 2001 Design Value (ug/m^3)[b]	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
GA	Washington	16.5	V	V	V	V	21,176
GA	Wilkinson	18.1	V	V	V	V	10,220
IL	Cook	18.8	V	V	V	V	5,376,741
IL	Du Page	15.4			V		904,161
IL	Madison	17.3	V	V	V	V	258,941
IL	St Clair	17.4	V	V	V	V	256,082
IL	Will	15.9	V		V	V	502,266
IN	Clark	17.3	V	V	V	V	96,472
IN	Lake	16.3	V	V	V	V	484,564
IN	Marion	17	V		V	V	860,454
IN	Vanderburgh	16.9			V		171,922
KY	Jefferson	17.1	V	V	V	V	693,604
KY	Kenton	15.9			V		151,464
LA	East Baton Rouge	14.6			V	V	412,852
LA	West Baton Rouge	14.1			V		21,601
MD	Baltimore	16			V		754,292
MD	Prince Georges	17.3	V	V	V	V	801,515
MD	Baltimore City	17.8	V	V	V	V	651,154
MA	Suffolk	16.1	V		V		689,807
MI	Wayne	18.9	V	V	V	V	2,061,162
MS	Jones	16.6	V		V	V	64,958
MO	St Louis City	16.3	V		V	V	348,189
MT	Lincoln	16.4	V	V	V	V	18,837
NJ	Hudson	17.5	V	V	V	V	608,975
NJ	Union	16.3			V	V	522,541
NY	Bronx	16.4	V		V	V	1,332,650
NY	New York	17.8	V	V	V	V	1,537,195
NC	Catawba	17.1	V		V	V	141,685
NC	Davidson	17.3	V	V	V	V	147,246
NC	Durham	15.3			V		223,314
NC	Forsyth	16.2			V	V	306,067
NC	Gaston	15.3			V		190,365
NC	Guilford	16.3	V		V	V	421,048
NC	McDowell	16.2			V		42,151
NC	Mecklenburg	16.8	V	V	V	V	695,454
NC	Wake	15.3			V		627,846
OH	Butler	17.4	V		V	V	332,807
OH	Cuyahoga	20.3	V	V	V	V	1,393,978
OH	Franklin	18.1	V	V	V	V	1,068,978
OH	Hamilton	19.3	V	V	V	V	845,303
OH	Jefferson	18.9	V	V	V	V	73,894
OH	Lawrence	17.4	V	V	V	V	62,319
OH	Lucas	16.7	V	V	V	V	455,054

State	County	1999 - 2001 Design Value (ug/m^3)[b]	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
OH	Mahoning	16.4			V		257,555
OH	Montgomery	17.6	V	V	V	V	559,062
OH	Scioto	20	V	V	V	V	79,195
OH	Stark	18.3	V	V	V	V	378,098
OH	Summit	17.3	V	V	V	V	542,899
OH	Trumbull	16.2			V		225,116
PA	Allegheny	21	V	V	V	V	1,281,666
PA	Delaware	15			V		550,864
PA	Philadelphia	16.6	V	V	V	V	1,517,550
PA	York	16.3			V		381,751
SC	Greenville	17	V	V	V	V	379,616
SC	Lexington	15.6			V		216,014
TN	Davidson	17			V	V	569,891
TN	Hamilton	18.9	V	V	V	V	307,896
TN	Knox	20.4	V	V	V	V	382,032
TN	Shelby	15.6			V		897,472
TN	Sullivan	17			V		153,048
TX	Dallas	14.4			V		2,218,899
TX	Harris	15.1	V	V	V	V	3,400,578
UT	Salt Lake	13.6			V		898,387
VA	Richmond City	14.9			V		197,790
WV	Brooke	17.4	V	V	V	V	25,447
WV	Cabell	17.8	V	V	V	V	96,784
WV	Hancock	17.4	V	V	V	V	32,667
WV	Kanawha	18.4	V	V	V	V	200,073
WV	Wood	17.6	V		V	V	87,986
WI	Milwaukee	14.5			V		940,164
Number of Violating Counties [b]			79	67	107	84	
Population of Violating Counties[c]			65,821,000	60,453,500	85,525,600	71,375,600	

[a] As described in Chapter 3, the final control case differs from the modeled control case based on public comment and updated information; however, we believe that the net results would approximate future emissions, although we anticipate the design value improvements would be smaller. In our modeling, we do not account for SO2 reductions related to sulfur reductions in home heating oil.

[b] Projections are made only for counties with monitored design values for 1999-2001. These were the most current data at the time the analyses were performed. Counties with insufficient data or lacking monitors are excluded.

[c] Populations are based on 2020 and 2030 estimates rounded to nearest hundred. See the AQ Modeling TSD for details.

Table 2.1.2-3 lists the counties with 2020 or 2030 projected annual $PM_{2.5}$ design values that do not violate the annual standard, but are within 10 percent of it. Counties are marked with an "X" in the table if their projected design values are greater than or equal to13.55 $\mu g/m^3$, but less than 15.05 $\mu g/m^3$. Counties are marked with an "V" in the table if their projected design values are greater than or equal to 15.05 $\mu g/m^3$. The 1999-2001 design values of these counties are also listed. These are counties that are not projected to violate the standard, but to be close to it, so the final rule will help ensure that these counties continue to meet the standard in either the base or control case for at least one of the years analyzed.

Table 2.1.2-3
Counties with 2020 and 2030 Projected Annual PM2.5 Design Values
within Ten Percent of the Annual PM2.5 Standard.[a, b]

State	County	1999 - 2001 Design Value (ug/m^3)[b]	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
AL	Alabama	15.5	X	X	X	X	14,254
AL	De Kalb	16.8	X	X	V	V	64,452
AL	Houston	16.3	V	X	V	V	88,787
AL	Madison	15.5			X		276,700
AL	Mobile	15.3	X	X	V	V	399,843
AR	Crittenden	15.3	X	X	X	X	50,866
AR	Pulaski	15.9	X	X	X	X	361,474
CA	Butte	15.4			X	X	203,171
CA	Imperial	15.7	X	X	V	X	142,361
CA	Kings	16.6	X		X	X	129,461
CA	San Joaquin	16.4	X	X	V	X	563,598
CA	Ventura	14.5	X	X	X	X	753,197
CT	Fairfield	13.6			X		882,567
DE	Sussex	14.5			X		156,638
GA	Hall	17.2	V	X	V	V	139,277
IL	Du Page	15.4	X	X	V	X	904,161
IL	Macon	15.4	X	X	X	X	114,706
IL	Will	15.9	V	X	V	V	502,266
IN	Elkhart	15.1	X		X	X	182,791
IN	Floyd	15.6	X	X	X	X	70,823
IN	Howard	15.4	X		X	X	84,964
IN	Marion	17	V	X	V	V	860,454
IN	Porter	13.9			X		146,798
IN	Tippecanoe	15.4	X		X	X	148,955
IN	Vanderburgh	16.9	X	X	V	X	171,922
KY	Bell	16.8	X	X	X	X	30,060
KY	Boyd	15.5	X	X	X	X	49,752
KY	Bullitt	16			X		61,236
KY	Campbell	15.5	X		X	X	88,616
KY	Daviess	15.8	X		X	X	91,545
KY	Fayette	16.8	X	X	X	X	260,512
KY	Kenton	15.9	X	X	V	X	151,464
KY	Pike	16.1	X	X	X	X	68,736
LA	Caddo	13.7			X	X	252,161
LA	Calcasieu	12.7			X		183,577
LA	East Baton Rouge	14.6	X	X	V	V	412,852
LA	Iberville	13.9	X		X	X	33,320

State	County	1999 - 2001 Design Value (ug/m^3)[b]	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
LA	Jefferson	13.6			X	X	455,466
LA	Orleans	14.1	X		X	X	484,674
LA	West Baton Rouge	14.1	X	X	V	X	21,601
MD	Baltimore	16	X	X	V	X	754,292
MA	Hampden	14.1			X		456,228
MA	Suffolk	16.1	V	X	V	X	689,807
MI	Kalamazoo	15	X		X	X	238,603
MS	Forrest	15.2	X	X	X	X	72,604
MS	Hinds	15.1	X		X	X	250,800
MS	Jackson	13.8			X	X	131,420
MS	Jones	16.6	V	X	V	V	64,958
MS	Lauderdale	15.3	X	X	X	X	78,161
MO	Jackson	13.9			X		654,880
MO	Jefferson	15	X	X	X	X	198,099
MO	St Charles	14.6	X		X	X	283,883
MO	St Louis	14.1			X		1,016,315
MO	St Louis City	16.3	V	X	V	V	348,189
NJ	Mercer	14.3	X		X	X	350,761
NJ	Union	16.3	X	X	V	V	522,541
NY	Bronx	16.4	V	X	V	V	1,332,650
NC	Alamance	15.3	X	X	X	X	130,800
NC	Cabarrus	15.7	X	X	X	X	131,063
NC	Catawba	17.1	V	X	V	V	141,685
NC	Cumberland	15.4	X		X	X	302,963
NC	Durham	15.3	X	X	V	X	223,314
NC	Forsyth	16.2	X	X	V	V	306,067
NC	Gaston	15.3	X	X	V	X	190,365
NC	Guilford	16.3	V	X	V	V	421,048
NC	Haywood	15.4	X		X	X	54,033
NC	McDowell	16.2	X	X	V	X	42,151
NC	Mitchell	15.5	X		X	X	15,687
NC	Orange	14.3			X		118,227
NC	Wake	15.3	X	X	V	X	627,846
NC	Wayne	15.3			X		113,329
OH	Butler	17.4	V	X	V	V	332,807
OH	Lorain	15.1	X		X	X	284,664
OH	Mahoning	16.4	X	X	V	X	257,555
OH	Portage	15.3	X	X	X	X	152,061
OH	Trumbull	16.2	X	X	V	X	225,116
PA	Berks	15.6	X	X	X	X	373,638
PA	Cambria	15.3			X		152,598
PA	Dauphin	15.5	X		X	X	251,798
PA	Delaware	15	X	X	V	X	550,864

State	County	1999 - 2001 Design Value (ug/m^3)[b]	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
PA	Lancaster	16.9	X	X	X	X	470,658
PA	Washington	15.5			X		202,897
PA	York	16.3	X	X	V	X	381,751
SC	Georgetown	13.9			X		55,797
SC	Lexington	15.6	X	X	V	X	216,014
SC	Richland	15.4	X	X	X	X	320,677
SC	Spartanburg	15.4	X	X	X	X	253,791
TN	Davidson	17	X	X	V	V	569,891
TN	Roane	17	X	X	X	X	51,910
TN	Shelby	15.6	X	X	V	X	897,472
TN	Sullivan	17	X	X	V	X	153,048
TN	Sumner	15.7	X		X	X	130,449
TX	Dallas	14.4	X	X	V	X	2,218,899
UT	Salt Lake	13.6	X		V	X	898,387
VA	Bristol City	16			X	X	17,367
VA	Richmond City	14.9	X	X	V	X	197,790
VA	Roanoke City	15.2			X		94,911
VA	Virginia Beach Cit	13.2			X		425,257
WV	Berkeley	16	X	X	X	X	75,905
WV	Marshall	16.5	X	X	X	X	35,519
WV	Ohio	15.7	X		X	X	47,427
WV	Wood	17.6	V	X	V	V	87,986
WI	Milwaukee	14.5	X	X	V	X	940,164
WI	Waukesha	14.1			X		360,767
Number of Counties within 10%[b]			70	62	64	70	
Population of Counties within 10%[c]			23,836,400	24,151,800	16,870,300	24,839,600	

[a] As described in Chapter 3, the final control case differs from the modeled control case based on public comment and updated information; however, we believe that the net results would approximate future emissions, although we anticipate the design value improvements would be smaller. In our modeling, we do not account for SO2 reductions related to sulfur reductions in home heating oil.

[b] Projections are made only for counties with monitored design values for 1999-2001. These were the most current data at the time the analyses were performed. Counties with insufficient data or lacking monitors are excluded.

[c] Populations are based on 2020 and 2030 estimates rounded to nearest hundred. See the AQ Modeling TSD for details.

We estimate that the reduction of this final rule will produce nationwide air quality improvements in PM levels. On a population-weighted basis, the average change in future-year annual averages is projected to decrease by 0.42 $\mu g/m^3$ in 2020, and 0.59 $\mu g/m^3$ in 2030.

While the final implementation process for bringing the nation's air into attainment with the $PM_{2.5}$ NAAQS is still being completed in a separate rulemaking action, the basic framework is well defined by the statute. EPA has requested that States and Tribes submit their recommendations by February 15, 2004. EPA's current plans call for designating $PM_{2.5}$ attainement and nonattainment areas in December 2004. Following designation, Section 172(b) of the Clean Air Act allows states up to 3 years to submit a revision to their state implementation

plan (SIP) that provides for the attainment of the $PM_{2.5}$ standard. Based on this provision, states could submit these SIPs in late-2007. Section 172(a)(2) of the Clean Air Act requires that these SIP revisions demonstrate that the nonattainment areas will attain the $PM_{2.5}$ standard as expeditiously as practicable but no later than 5 years from the date that the area was designated nonattainment. However, based on the severity of the air quality problem and the availability and feasibility of control measures, the Administrator may extend the attainment date "for a period of no greater than 10 years from the date of designation as nonattainment." Based on section 172(a) provisions in the Act, we expect that areas will need to attain the $PM_{2.5}$ NAAQS in the 2010 (based on 2007 - 2009 air quality data) to 2015 (based on 2012 to 2014 air quality data) time frame, and then be required to maintain the NAAQS thereafter.

Since the emission reductions from this final rule will begin in this same time frame, the projected reductions in nonroad emissions will be used by states in meeting the $PM_{2.5}$ NAAQS. States and state organizations have told EPA that they need nonroad diesel engine reductions in order to be able to meet and maintain the $PM_{2.5}$ NAAQS as well as visibility regulations, especially in light of the otherwise increasing emissions from nonroad sources without more stringent standards.[95, 96, 97] The following are sample comments from states and state associations on the proposed rule, which corroborate that this rule is a critical element in States' NAAQS attainment efforts. Fuller information can be found in the Summary and Analysis of Comments.

- "Unless emissions from nonroad diesels are sharply reduced, it is very likely that many areas of the country will be unable to attain and maintain health-based NAAQS for ozone and PM." (STAPPA/ALAPCO)
- "Adoption of the proposed regulation ... is necessary for the protection of public health in California and to comply with air quality standards." (California Air Resources Board)
- "The EPA's proposed regulation is necessary if the West is to make reasonable progress towards improving visibility in our nation's Class I areas." (Western Regional Air Partnership (WRAP))
- "Attainment of the NAAQS for ozone and $PM_{2.5}$ is of immediate concern to the states in the northeast region....Thus, programs ... such as the proposed rule for nonroad diesel engines are essential." (NESCAUM)

Furthermore, this rule ensures that nonroad diesel emissions will continue to decrease as the fleet turns over in the years beyond 2014; these reductions will be important for maintenance of the NAAQS following attainment. The future reductions are also important to achieve visibility goals, as discussed below.

2.1.3 Environmental Effects of Particulate Matter

In this section, we discuss public welfare effects of PM and its precursors including visibility impairment, acid deposition, eutrophication and nitrification, POM deposition, materials damage, and soiling.

2.1.3.1 Visibility Degradation

Visibility can be defined as the degree to which the atmosphere is transparent to visible light.[98] Visibility impairment has been considered the "best understood and most easily measured effect of air pollution."[99] Fine particles are the major cause of reduced visibility in parts of the United States. Haze obscures the clarity, color, texture, and form of what we see. Visibility is an important effect because it has direct significance to people's enjoyment of daily activities in all parts of the country. Visibility is also highly valued in significant natural areas such as national parks and wilderness areas, because of the special emphasis given to protecting these lands now and for future generations.

Scattering and absorption by both gases and particles decrease light transmittance. Size and chemical composition of particles strongly affects their ability to scatter or absorb light. The same particles (sulfates, nitrates, organic carbon, smoke, and soil dust) comprising $PM_{2.5}$, which are linked to serious health effects and environmental effects (e.g., ecosystem damage), can also significantly degrade visual air quality. (For data on chemical composition of particles in slected urban and rural areas, see Figures 2.1.2-2 and 2.1.2-3 above.) Sulfates contribute to visibility impairment especially on the haziest days, accounting in the rural Eastern United States for more than 60 percent of annual average light extinction on the best days and up to 86 percent of average light extinction on the haziest days. Nitrates and elemental carbon each typically contribute 1 to 6 percent of average light extinction on haziest days in rural locations in the Eastern United States.[100]

To quantify changes in visibility, the analysis presented in this chapter computes a light-extinction coefficient, based on the work of Sisler, which shows the total fraction of light that is decreased per unit distance.[101] This coefficient accounts for the scattering and absorption of light by both particles and gases, and accounts for the higher extinction efficiency of fine particles compared with coarse particles. Visibility can be described in terms of visual range, light extinction or deciview.[E] Visibility impairment also has a temporal dimension in that impairment might relate to a short-term excursion or to longer periods (e.g., worst 20 percent of days or annual average levels). More detailed discussions of visibility effects are contained in the EPA Criteria Document for PM.[102]

Visibility effects are manifest in two principal ways: (1) as local impairment (e.g., localized hazes and plumes) and (2) as regional haze. The emissions from engines covered by this rule contribute to both types of visibility impairment.

[E]Visual range can be defined as the maximum distance at which one can identify a black object against the horizon sky. It is typically described in miles or kilometers. Light extinction is the sum of light scattering and absorption by particles and gases in the atmosphere. It is typically expressed in terms of inverse megameters (Mm^{-1}), with larger values representing worse visibility. The deciview metric describes perceived visual changes in a linear fashion over its entire range, analogous to the decibel scale for sound. A deciview of 0 represents pristine conditions. The higher the deciview value, the worse the visibility, and an improvement in visibility is a decrease in deciview value.

Local-scale visibility degradation is commonly in the form of either a plume resulting from the emissions of a specific source or small group of sources, or it is in the form of a localized haze such as an urban "brown cloud." Plumes are comprised of smoke, dust, or colored gas that obscure the sky or horizon relatively near sources. Impairment caused by a specific source or small group of sources has been generally termed as "reasonably attributable."

The second type of impairment, regional haze, results from pollutant emissions from a multitude of sources located across a broad geographic region. It impairs visibility in every direction over a large area, in some cases over multi-state regions. Regional haze masks objects on the horizon and reduces the color and contrast of nearby objects.[103]

On an annual average basis, the concentrations of non-anthropogenic fine PM are generally small when compared with concentrations of fine particles from anthropogenic sources.[104] Anthropogenic contributions account for about one-third of the average extinction coefficient in the rural West and more than 80 percent in the rural East.[105] In the Eastern United States, reduced visibility is mainly attributable to secondarily formed particles, particularly those less than a few micrometers in diameter (e.g., sulfates). While secondarily formed particles still account for a significant amount in the West, primary emissions contribute a larger percentage of the total particulate load than in the East. Because of significant differences related to visibility conditions in the Eastern and Western United States, we present information about visibility by region. Furthermore, it is important to note that even in those areas with relatively low concentrations of anthropogenic fine particles, such as the Colorado plateau, small increases in anthropogenic fine particle concentrations can lead to significant decreases in visual range. This is one of the reasons mandatory Federal Class I areas have been given special consideration under the Clean Air Act. The 156 mandatory Federal Class I areas are displayed on the map in Figure 2-1 above.

EPA determined that emissions from nonroad engines significantly contribute to air pollution that may be reasonably anticipated to endanger public health and welfare for visibility effects in particular (67 FR 68242, November 8, 2002). The primary and PM-precursor emissions from nonroad diesel engines subject to this rule contribute to these effects. To demonstrate this, in addition to the inventory information in Chapter 3, we present information about both general visibility impairment related to ambient PM levels across the country, and we also analyze visibility conditions in mandatory Federal Class I areas. Accordingly, in this section, for both the nation and for mandatory Federal Class I areas, we discuss the types of effects, current and future visibility conditions absent the projected emission reductions, and the changes we anticipate from the projected emission reductions. We conclude that the projected emission reductions will improve visibility conditions across the country and in particular in mandatory Federal Class I areas.

2.1.3.1.1 Visibility Impairment Where People Live, Work and Recreate

Good visibility is valued by people throughout the country - in the places they live, work, and enjoy recreational activities. However, unacceptable visibility impairment occurs in many areas throughout the country. In this section, in order to estimate the magnitude of the visibility

problem, we use monitored $PM_{2.5}$ data and modeled air quality accounting for projected emissions from nonroad diesel engines absent additional controls. The air quality modeling is discussed in Section 2.1.2 above and in the AQ Modeling TSD.[106] The engines covered by this rule contribute to $PM_{2.5}$ levels in areas across the country with significant visibility impairment.

The secondary PM NAAQS is designed to protect against adverse welfare effects such as visibility impairment. In 1997, the secondary PM NAAQS was set as equal to the primary (health-based) PM NAAQS (62 Federal Register No. 138, July 18, 1997). EPA concluded that PM can and does produce adverse effects on visibility in various locations, depending on PM concentrations and factors such as chemical composition and average relative humidity. In 1997, EPA demonstrated that visibility impairment is an important effect on public welfare and that visibility impairment is experienced throughout the United States, in multi-state regions, urban areas, and remote Federal Class I areas.

The updated monitored data and air quality modeling presented below confirm that the visibility situation identified during the NAAQS review in 1997 is still likely to exist. Specifically, there will still likely be a broad number of areas that are above the annual $PM_{2.5}$ NAAQS in the Northeast, Midwest, Southeast and California , such that the determination in the NAAQS rulemaking about broad visibility impairment and related benefits from NAAQS compliance are still relevant. Thus, levels above the fine PM NAAQS cause adverse welfare impacts, such as visibility impairment (both regional and localized impairment). EPA recently confirmed this in our determination about nonroad engines significant contribution to unacceptable visibility impairment (67 FR 68251, November 8, 2002).

In addition, in setting the PM NAAQS, EPA acknowledged that levels of fine particles below the NAAQS may also contribute to unacceptable visibility impairment and regional haze problems in some areas, and Clean Air Act Section 169 provides additional authorities to remedy existing impairment and prevent future impairment in the 156 national parks, forests and wilderness areas labeled as mandatory Federal Class I areas (62 FR at 38680-81, July 18, 1997).

In making determinations about the level of protection afforded by the secondary PM NAAQS, EPA considered how the Section 169 regional haze program and the secondary NAAQS would function together.[107] Regional strategies, such as this rule, are expected to improve visibility in many urban and non-Class I areas as well. Visibility impairment in mandatory Federal Class I areas is discussed in Section 2.1.4.

2.1.3.1.1.1 Current Areas Affected by Visibility Impairment: Monitored Data

The need for reductions in the levels of $PM_{2.5}$ is widespread, as discussed above and shown in Figure 2-1. Currently, high ambient $PM_{2.5}$ levels are measured throughout the country. Fine particles may remain suspended for days or weeks and travel hundreds to thousands of kilometers, and thus fine particles emitted or created in one county may contribute to ambient concentrations in a neighboring region.[108]

Without the effects of pollution, a natural visual range is approximately 120 to 180 miles

(200 to 300 kilometers) in the West and 45 to 90 miles (75 to 150 kilometers) in the East.[109] However, over the years, in many parts of the United States, fine particles have significantly reduced the range that people can see. In the West, the visibility range is 33 to 90 miles (53 to 144 kilometers), and in the East, the current range is only 14 to 24 miles (22 to 38 kilometers).[110]

Current $PM_{2.5}$ monitored values for 2000-2002 indicate that almost 65 million people in 120 counties live in areas where design values of $PM_{2.5}$ annual levels are at or above the $PM_{2.5}$ NAAQS. This represents 23 percent of the counties and 37 percent of the population in the areas with monitoring data that met completeness requirements and had levels above the NAAQS. Thus, at least these populations (plus others who travel to these areas) would likely be experiencing visibility impairment that is unacceptable. Emissions of PM and its precursors from nonroad diesel engines contribute to this unacceptable impairment.

An additional 32 million people live in 91 counties that have air quality measurements for 2000-2002 within 10 percent of the level of the PM standard. These areas, though not currently violating the standard, will also benefit from the additional reductions from this final rule to ensure long-term maintenance of the standard and to prevent deterioration in visibility conditions.

Although we present the annual average to represent national visibility conditions, visibility impairment can also occur on certain days or other shorter periods. As discussed below, the Regional Haze program targets the worst 20 percent of days in a year. The projected emission reductions from this rule are also needed to improve visibility on the worst days.

2.1.3.1.1.2 Areas Affected by Future Visibility Impairment

Because the chemical composition of PM and other atmospheric conditions affect visibility impairment, we used the REMSAD air quality model to project visibility conditions in 2020 and 2030 to estimate visibility impairment directly as changes in deciview. One of the inputs to the PM modeling described above is a projection of future emissions from nonroad diesel engines absent additional controls. Thus, we are able to demonstrate that the nonroad diesel emissions contribute to the projected visibility impairment and that there continues to be a need for reductions from those engines.

As described above, based on this modeling and absent additional controls, we predicted that in 2020, there will be 79 counties with a population of 66 million where annual $PM_{2.5}$ levels are above 15 µg/m3.[111] In 2030, this number will rise to 107 counties with a population of 85 million in the absence of additional controls. Section 2.1.2 and the AQ Modeling TSD provides additional details.

Based upon the light-extinction coefficient, we also calculated a unitless visibility index or deciview. As shown in Table 2.1.3-1, in 2030 we estimate visibility in the East to be about 20.54 deciviews (or visual range of 50 kilometers) on average, with poorer visibility in urban areas, compared with the visibility conditions without man-made pollution of 9.5 deciviews (or visual range of 150 kilometers). Likewise, we estimate visibility in the West to be about 8.83

deciviews (or visual range of 162 kilometers) in 2030, compared with the visibility conditions without anthropogenic pollution of 5.3 deciviews (or visual range of 230 kilometers). Thus, in the future, a substantial percent of the population may experience unacceptable visibility impairment in areas where they live, work and recreate.

Table 2.1.3-1
Summary of Future National (48 state) Baseline Visibility Conditions Absent Additional Controls (Deciviews)

Regions[a]	Predicted 2020 Visibility (annual average)	Predicted 2030 Visibility (annual average)	Natural Background Visibility
Eastern U.S.	20.27	20.54	9.5
Urban	21.61	21.94	
Rural	19.73	19.98	
Western U.S.	8.69	8.83	5.3
Urban	9.55	9.78	
Rural	8.5	8.61	

[a] Eastern and Western Regions are separated by 100 degrees north longitude. Background visibility conditions differ by region.

The emissions from nonroad diesel engines contribute to this visibility impairment as discussed in Chapter 3. Nonroad diesel engines emissions contribute a large portion of the total PM emissions from mobile sources and anthropogenic sources, in general. These emissions occur in and around areas with PM levels above the annual $PM_{2.5}$ NAAQS. The nonroad engines subject to this rule contribute to these effects as well as localized visibility impairment. Thus, the emissions from these sources contribute to the unacceptable current and anticipated visibility impairment.

2.1.3.1.1.3 Future Improvements in Visibility from the Projected Emission Reductions

For this rule, we also modeled a preliminary control scenario that illustrates the likely emission reductions. As public comment and additional data regarding technical feasibility and other factors became available, our judgment about the controls that are feasible has evolved. Thus, the preliminary control option differs from what we are proposing, as summarized in Section 3.6. It is important to note that these changes would not affect our estimates of the baseline conditions without additional controls described above. In our air quality modeling, we did not account for SO_2 reductions from reductions in sulfur levels in home heating oil. We anticipate that the nonroad diesel emission reductions from this final rule together with other strategies would improve the projected visibility impairment, and we conclude that there continues to be a need for reductions from those engines.

Based on our modeling, we predict that in 2020, there will be 12 counties with a population of 6 million that come into attainment with the annual $PM_{2.5}$ because of the improvements in air quality from the emission reductions resulting from this final rule. In 2030, an estimated total of 24 counties (12 additional counties) with a population of 14 million (8 million additional people) will come into attainment with the annual $PM_{2.5}$ because of the improvements in air quality from this final rule. There will also be emission reductions in counties with levels close to the air quality standards that will improve visibility conditions and help them maintain the standards. All of these areas and their populations will experience improvements in visibility as well as health effects, as described earlier.

We estimate that the emission reductions resulting from this final rule will produce nationwide air quality improvements in PM levels. On a population-weighted basis, the average change in future-year annual averages will be a decrease of 0.33 $\mu g/m^3$ in 2020, and 0.46 $\mu g/m^3$ in 2030. These reductions are discussed in more detail in Section 2.1.2 above.

We can also calculate these improvement in visibility as decreases in deciview value. As shown in Table 2.1.3-2, in 2030 we estimate visibility in the East to be about 20.54 deciviews (or visual range of 50 kilometers) on average, with poorer visibility in urban areas. Emission reductions from this final rule in 2030 will improve visibility by an estimated 0.33 deciviews. Likewise, we estimate visibility in the West to be about 8.83 deciviews (or visual range of 162 kilometers) in 2030, and we estimate that emission reductions from this final rule in 2030 will improve visibility by 0.25 deciviews. These improvements are needed in conjunction with other sulfur reduction strategies in the East and a combination of strategies in the West to make reasonable progress toward visibility goals.[112] Thus, this final rule is an important part of strategies to improve visibility in areas where they live, work and recreate.

Table 2.1.3-2
Summary of Future National Visibility Improvements
from Nonroad Diesel Emission Reductions (Annual Average Deciviews)

Regions[a]	2020		2030	
	Predicted Baseline 2020 Visibility	Predicted 2020 Control Visibility[b]	Predicted Baseline 2030 Visibility	Predicted 2030 Control Visibility[b]
Eastern U.S.	20.27	20.03	20.54	20.21
Urban	21.61	21.37	21.94	21.61
Rural	19.73	19.49	19.98	19.65
Western U.S.	8.69	8.51	8.83	8.58
Urban	9.55	9.3	9.78	9.43
Rural	8.5	8.33	8.61	8.38

[a] Eastern and Western Regions are separated by 100 degrees north longitude. Background visibility conditions differ by region.

[b] The results illustrate the type of visibility improvements for the preliminary control option, as discussed in Section 3.6. The analysis in Chapter 3 differs based on updated information; however, we believe that the net results would approximate future PM emissions, although we anticipate the annual average visibility improvements would be smaller.

2.1.3.1.2 Visibility Impairment in Mandatory Federal Class I Areas

Achieving the annual $PM_{2.5}$ NAAQS will help improve visibility across the country, but it will not be sufficient to meet the statutory goal of no manmade impairment in the mandatory Federal Class I areas (64 FR 35722, July 1, 1999 and 62 FR 38680, July 18, 1997). In setting the NAAQS, EPA discussed how the NAAQS in combination with the regional haze program, is deemed to improve visibility consistent with the goals of the Act.[113] In the East, there are and will continue to be sizable areas above 15 $\mu g/m^3$ and where light extinction is significantly above natural background. Thus, large areas of the Eastern United States have air pollution that is causing and will continue to cause unacceptable visibility problems. In the West, scenic vistas are especially important to public welfare. Although the annual $PM_{2.5}$ NAAQS is met in most areas outside of California, virtually the entire West is in close proximity to a scenic mandatory Federal Class I area protected by 169A and 169B of the Act.

The 156 Mandatory Federal Class I areas are displayed on the map in Figure 2-1 above. These areas include many of our best known and most treasured natural areas, such as the Grand Canyon, Yosemite, Yellowstone, Mount Rainier, Shenandoah, the Great Smokies, Acadia, and the Everglades. More than 280 million visitors come to enjoy the scenic vistas and unique natural features including the night sky in these and other park and wilderness areas each year.

In the 1990 Clean Air Act amendments, Congress provided additional emphasis on regional haze issues (see section 169B). In 1999 EPA finalized a rule that calls for States to establish goals and emission reduction strategies for improving visibility in all 156 mandatory Class I national parks and wilderness areas. In this rule, EPA established a "natural visibility" goal.[114] In that rule, EPA also encouraged the States to work together in developing and implementing their air quality plans. The regional haze program is focused on long-term emissions decreases from the entire regional emission inventory comprised of major and minor stationary sources, area sources and mobile sources. The regional haze program is designed to improve visibility and air quality in our most treasured natural areas so that these areas may be preserved and enjoyed by current and future generations. At the same time, control strategies designed to improve visibility in the national parks and wilderness areas will improve visibility over broad geographic areas, including other recreational sites, our cities and residences. In the PM NAAQS rulemaking, EPA also anticipated the need in addition to the NAAQS and Section 169 regional haze program to continue to address localized impairment that may relate to unique circumstances in some Western areas. For mobile sources, there may also be a need for a Federal role in reduction of those emissions, in particular, because mobile source engines are regulated primarily at the Federal level.

The regional haze program calls for states to establish goals for improving visibility in national parks and wilderness areas to improve visibility on the haziest 20 percent of days and to ensure that no degradation occurs on the clearest 20 percent of days (64 FR 35722. July 1, 1999). The rule requires states to develop long-term strategies including enforceable measures designed to meet reasonable progress goals toward natural visibility conditions. Under the regional haze program, States can take credit for improvements in air quality achieved as a result of other Clean Air Act programs, including national mobile-source programs.[F]

2.1.3.1.2.1 Current Mandatory Federal Class I Areas Affected by Visibility Impairment: Monitored Data

Detailed information about current and historical visibility conditions in mandatory Federal Class I areas is summarized in the EPA Report to Congress and the recent EPA Trends Report.[115] The conclusions draw upon the Interagency Monitoring of Protected Visual Environments (IMPROVE) network data.[116] The National Park Service report also describes the state of national park visibility conditions and discusses the need for improvement.[117]

As described in the EPA Trends Report 1999, most of the IMPROVE sites in the intermountain West and Colorado Plateau have annual average impairment of 12 deciviews or

[F] Although a recent court case, American Corn Growers Association v. EPA, 291F.3d 1(D.C .Cir 2002), vacated the Best Available Retrofit Technology (BART) provisions of the Regional Haze rule, the court denied industry's challenge to EPA's requirement that state's SIPS provide for reasonable progress towards achieving natural visibility conditions in national parks and wilderness areas and the "no degradation" requirement. Industry did not challenge requirements to improve visibility on the haziest 20 percent of days. The court recognized that mobile source emission reductions would need to be a part of a long-term emission strategy for reducing regional haze. A copy of this decision can be found in Docket A-2000-01, Document IV- A-113.

less, with the worst days ranging up to 17 deciviews (compared with 5.3 deciviews of natural background visibility).[118] Several other western IMPROVE sites in the Northwest and California experience levels on the order of 16 to 23 deciviews on the haziest 20 percent of days. Many rural locations in the East have annual average values exceeding 21 deciviews, with average visibility levels on the haziest days up to 32 deciviews.

Although there have been general trends toward improved visibility, progress is still needed on the haziest days. Specifically, as discussed in the EPA Trends Report, in the 10 Class I areas in the Eastern United States, visibility on the haziest 20 percent of days remains significantly impaired with a mean visual range of 23 kilometers for 1999, as compared with 84 kilometers for the clearest days in 1999. In the 26 Class I reported areas in the Western United States, the conditions for the haziest 20 percent of days degraded between 1997 and 1999 by 17 percent. However, visibility on the haziest 20 percent of days in the West remains relatively unchanged over the 1990s with the mean visual range for 1990 (80 kilometers) nearly the same as the 1990 level (86 kilometers).

2.1.3.1.2.2 Mandatory Federal Class I Areas Affected by Future Visibility Impairment

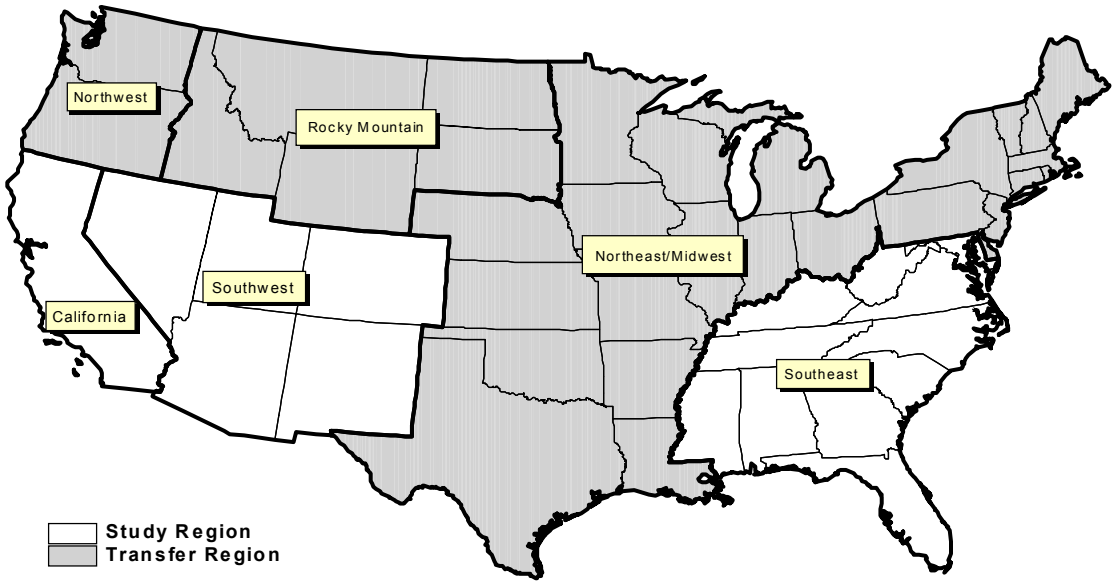
As part of the PM air quality modeling described above, we modeled future visibility conditions in the mandatory Federal Class I areas absent additional controls. The results by region are summarized in Table 2.1.3-3. In Figure 2.1.3-1, we define the regions used in this analysis.[119] These air quality results show that visibility is impaired in most mandatory Federal Class I areas and additional reductions from engines subject to this rule are needed to achieve the goals of the Clean Air Act of preserving natural conditions in mandatory Federal Class I areas.

Table 2.1.3-3
Summary of Future Baseline Visibility Conditions in Mandatory Federal Class I Areas Absent Additional Emission Reductions (Annual Average Deciview)

Class I Regions [a]	Predicted 2020 Visibility	Predicted 2030 Visibility	Natural Background Visibility
Eastern	19.72	20.01	9.5
Southeast	21.31	21.62	
Northeast/Midwest	18.30	18.56	
Western	8.80	8.96	5.3
Southwest	6.87	7.03	
California	9.33	9.56	
Rocky Mountain	8.46	8.55	
Northwest	12.05	12.18	
National Class I Area Average	11.61	11.80	

[a] Regions are depicted in Figure 1-5.1. Background visibility conditions differ by region based on differences in relative humidity and other factors: Eastern natural background is 9.5 deciviews (or visual range of 150 kilometers) and in the West natural background is 5.3 deciviews (or visual range of 230 kilometers).

Figure 2.1.3-1
Visibility Regions for the Continental United States



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

2.1.3.1.2.3 Future Improvements in Mandatory Federal Class I Visibility from the Projected Emission Reductions

The overall goal of the regional haze program is to prevent future and remedy existing visibility impairment in mandatory Federal Class I areas. As shown by the future deciview estimates in Table 2.1.3-4, additional emission reductions will be needed from the broad set of sources that contribute, including the emissions from engines subject to this rule. The table also presents the results from our modeling of a preliminary control scenario that illustrates the likely reductions from the final rule. Emission reductions from nonroad diesel engines are needed to achieve the goals of the Act of preserving natural conditions in mandatory Federal Class I areas. These reductions are a part of the overall strategy to achieve the visibility goals of the Act and the regional haze program.

Table 2.1.3-4
Summary of Future Visibility Improvements[b] in Mandatory Federal Class I Areas from Nonroad Diesel Emission Reductions (Annual Average Deciviews)

Mandatory Federal Class I Regions[a]	2020		2030	
	Predicted Baseline 2020 Average Visibility	Predicted 2020 Control Average Visibility[b]	Predicted Baseline 2030 Average Visibility	Predicted 2030 Control Average Visibility[b]
Eastern	19.72	19.54	20.01	19.77
Southeast	21.31	21.13	21.62	21.38
Northeast/Midwest	18.30	18.12	18.56	18.32
Western	8.80	8.62	8.96	8.72
Southwest	6.87	6.71	7.03	6.82
California	9.33	9.12	9.56	9.26
Rocky Mountain	8.46	8.31	8.55	8.34
Northwest	12.05	11.87	12.18	11.94
National Class I Area Average	11.61	11.43	11.80	11.56

[a] Regions are presented in Figure 2.1.3-1 based on Chestnut and Rowe (1990a, 1990b) study regions.
[b] The results illustrate the type of visibility improvements for the preliminary control option, as discussed in Section 3.6. The final control scenario described in Chapter 3 differs from the modeled scenario based on public comment and updated information; however, we believe that the net results would approximate future PM emissions, although we anticipate the annual average visibility improvements would be smaller.

2.1.3.2 Other Effects

2.1.3.2.1 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.[120] 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 near $80 million per year when applied to all new cars and trucks sold in the United States each year.

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.

A study of emission trends and acidity of water bodies in the Eastern United States by the General Accounting Office (GAO) found that from 1992 to 1999 sulfates declined in 92 percent of a representative sample of lakes, and nitrate levels increased in 48 percent of the lakes sampled.[121] The decrease in sulfates is consistent with emission trends, but the increase in nitrates is inconsistent with the stable levels of nitrogen emissions and deposition. The study suggests that the vegetation and land surrounding these lakes have lost some of their previous capacity to use nitrogen, thus allowing more of the nitrogen to flow into the lakes and increase their acidity. Recovery of acidified lakes is expected to take a number of years, even where soil and vegetation have not been "nitrogen saturated," as EPA called the phenomenon in a 1995 study.[122] This situation places a premium on reductions of SOx and especially NOx from all sources, including nonroad diesel engines, in order to reduce the extent and severity of nitrogen saturation and acidification of lakes in the Adirondacks and throughout the United States.

The SOx and NOx reductions from this rule will 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 will also help reduce stress on forests, thereby accelerating reforestation efforts and improving timber production. Deterioration of our historic buildings and monuments, and of buildings, vehicles, and other structures exposed to acid rain and dry acid deposition also will be reduced, and the costs borne to prevent acid-related damage may also decline. While the reduction in sulfur and nitrogen acid deposition will be roughly proportional to the reduction in SOx and NOx emissions, respectively, the precise impact of this rule will differ across different areas.

2.1.3.2.2 Eutrophication and Nitrification

Eutrophication is the accelerated production of organic matter, particularly algae, in a water body. 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.

In 1999, the National Oceanic and Atmospheric Administration (NOAA) published the results of a five year national assessment of the severity and extent of estuarine eutrophication. An estuary is defined as the inland arm of the sea that meets the mouth of a river. The 138 estuaries characterized in the study represent more than 90 percent of total estuarine water surface area and the total number of U.S. estuaries. The study found that estuaries with moderate to high eutrophication conditions represented 65 percent of the estuarine surface area. Eutrophication 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.

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

In recent decades, human activities have greatly accelerated nutrient inputs, such as nitrogen and phosphorous, causing excessive growth of algae and leading to degraded water quality and associated impairments of freshwater and estuarine resources for human uses.[123] Since 1970,

eutrophic conditions worsened in 48 estuaries and improved in 14. In 26 systems, there was no trend in overall eutrophication conditions since 1970.[124] 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. 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.

Between 1992 and 1997, experts surveyed by National Oceanic and Atmospheric Administration (NOAA) most frequently recommended that control strategies be developed for agriculture, wastewater treatment, urban runoff, and atmospheric deposition.[125] In its Third Report to Congress on the Great Waters, EPA reported that atmospheric deposition contributes from 2 to 38 percent of the nitrogen load to certain coastal waters.[126] A review of peer reviewed literature in 1995 on the subject of air deposition suggests a typical contribution of 20 percent or higher.[127] Human-caused nitrogen loading to the Long Island Sound from the atmosphere was estimated at 14 percent by a collaboration of federal and state air and water agencies in 1997.[128] The National Exposure Research Laboratory, U.S. EPA, estimated based on prior studies that 20 to 35 percent of the nitrogen loading to the Chesapeake Bay is attributable to atmospheric deposition.[129] The mobile source portion of atmospheric NOx contribution to the Chesapeake Bay was modeled at about 30 percent of total air deposition.[130]

Deposition of nitrogen from nonroad diesel engines contributes to elevated nitrogen levels in waterbodies. The new emission standards for nonroad diesel engines will reduce total NOx emissions by 738,000 tons in 2030. The NOx reductions will reduce the airborne nitrogen deposition that contributes to eutrophication of watersheds, particularly in aquatic systems where atmospheric deposition of nitrogen represents a significant portion of total nitrogen loadings.

2.1.3.2.3 Polycyclic Organic Matter (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.[131] 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 seven most toxic POM is at least 62 tons/year and represents only those POM that are adhered to mobile source particulate emissions.[132] The majority of these emissions are produced by diesel engines.

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

Evidence for potential human health effects associated with POM comes from studies in animals (fish, amphibians, rats) and in human cells culture assays. Reproductive, developmental, immunological, and endocrine (hormone) effects have been documented in these systems. Many of the compounds included in the class of compounds known as POM are classified by EPA as probable human carcinogens based on animal data.

The new emission standards will reduce not only the PM emissions from land-based nonroad diesel engines, but also the deposition of the POM adhering to the particles, thereby reducing health effects of POM in lakes and streams, accelerating the recovery of affected lakes and streams, and reviving adversely affected ecosystems.

2.1.3.2.4 Materials Damage and Soiling

The deposition of airborne particles can also reduce the aesthetic appeal of buildings and culturally important articles through soiling, and can contribute directly (or in conjunction with other pollutants) to structural damage by means of corrosion or erosion. Particles affect materials principally by promoting and accelerating the corrosion of metals, by degrading paints, and by deteriorating building materials such as concrete and limestone. Particles contribute to these effects because of their electrolytic, hygroscopic, and acidic properties, and their ability to sorb corrosive gases (principally sulfur dioxide). The rate of metal corrosion depends on a number of factors, including the deposition rate and nature of the pollutant; the influence of the metal protective corrosion film; the amount of moisture present; variability in the electrochemical reactions; the presence and concentration of other surface electrolytes; and the orientation of the metal surface.

Paints undergo natural weathering processes from exposure to environmental factors such as sunlight, moisture, fungi, and varying temperatures. In addition to the natural environmental factors, studies show particulate matter exposure may give painted surfaces a dirty appearance. Several studies also suggest that particles serve as carriers of other more corrosive pollutants, allowing the pollutants to reach the underlying surface or serve as concentration sites for other pollutants. A number of studies have shown some correlation between particulate matter and damage to automobile finishes. A number of studies also support the conclusion that gaseous pollutants contribute to the erosion rates of exterior paints.

Damage to calcareous stones (i.e., limestone, marble and carbonated cemented stone) has been attributed to deposition of acidic particles. Moisture and salts are considered the most important factors in building material damage. However, many other factors (such as normal weathering and microorganism damage) also seem to play a part in the deterioration of inorganic building materials. The relative importance of biological, chemical, and physical mechanisms has not been studied to date. Thus, the relative contribution of ambient pollutants to the damage observed in various building stone is not well quantified. Under high wind conditions, particulates result in slow erosion of the surfaces, similar to sandblasting.

Soiling is the accumulation of particles on the surface of an exposed material resulting in the degradation of its appearance. When such accumulation produces sufficient changes in

reflection from opaque surfaces and reduces light transmission through transparent materials, the surface will become perceptibly dirty to the human observer. Soiling can be remedied by cleaning or washing, and depending on the soiled material, repainting.

2.2 Air Toxics

2.2.1 Diesel Exhaust PM

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

2.2.1.1 Potential Cancer Effects of Diesel Exhaust

In addition to its contribution to ambient PM inventories, diesel exhaust is of specific concern because it has been judged to pose a lung cancer hazard for humans as well as a hazard from noncancer respiratory effects such as pulmonary inflammation.

In 2001, EPA completed a rulemaking on mobile source air toxics with a determination that diesel particulate matter and diesel exhaust organic gases be identified as a Mobile Source Air Toxic (MSAT).[133] This determination was based on a draft of the Diesel HAD on which the Clean Air Scientific Advisory Committee (CASAC) of the Science Advisory Board had reached closure. Including both diesel PM and diesel exhaust organic gases in the determination was made in order to be precise about the components of diesel exhaust expected to contribute to the observed cancer and non-cancer health effects. Currently available science, while suggesting an important role for the particulate phase component of diesel exhaust, does not attribute the likely cancer and noncancer health effects independently to diesel particulate matter as distinct from the gas phase components (EPA, 2001). The purpose of the MSAT list is to provide a screening tool that identifies compounds emitted from motor vehicles or their fuels for which further evaluation of emission controls is appropriate.

EPA released its final "Health Assessment Document for Diesel Engine Exhaust" (the EPA Diesel HAD), referenced earlier. There, diesel exhaust was classified as likely to be carcinogenic to humans by inhalation at environmental exposures, in accordance with the revised draft 1996/1999 EPA cancer guidelines.[134] In accordance with earlier EPA guidelines, diesel exhaust would be similarly classified as a probable human carcinogen (Group B1).[135, 136] A number of other agencies (National Institute for Occupational Safety and Health, the International Agency for Research on Cancer, the World Health Organization, California EPA, and the U.S. Department of Health and Human Services) have made similar classifications.[137,138,139,140,141] The Health Effects Institute has also made numerous studies and report on the potential carcinogenicity of diesel exhaust.[142, 143, 144] Numerous animal and

bioassay/genotoxic tests have been done on diesel exhaust.[145, 146] Also, case-control and cohort studies have been conducted on railroad engine exposures[147,148,149] in addition to studies on truck workers.[150, 151,152] Also, there are numerous other epidemiologic studies including some studying mine workers and fire fighters.[153, 154]

It should be noted that the conclusions in the EPA Diesel HAD were based on diesel engines currently in use, including nonroad diesel engines such as those found in bulldozers, graders, excavators, farm tractor drivers and heavy construction equipment. As new diesel engines with significantly less PM exhaust emissions replace existing engines, the conclusions of the EPA Diesel HAD will need to be reevaluated.

More specifically, the EPA Diesel HAD states that the conclusions of the document apply to diesel exhaust in use today including both highway and nonroad engines. The EPA Diesel HAD acknowledges that the studies were done on engines with older technologies generally for highway applications and that "there have been changes in the physical and chemical composition of some DE [diesel exhaust] emissions (highway vehicle emissions) over time, though there is no definitive information to show that the emission changes portend significant toxicological changes." The EPA Diesel HAD further concludes that "taken together, these considerations have led to a judgment that the hazards identified from older-technology-based exposures are applicable to current-day exposures." The diesel technology used for nonroad diesel engines typically lags that used for highway engines, which have been subject to PM standards since 1988. Thus, the conclusions from the EPA Diesel HAD continue to be relevant to current nonroad diesel engine emissions.

Some of the epidemiologic studies discussed in the EPA Diesel HAD were conducted specifically on nonroad diesel engine emissions. In particular, one recent study examined bulldozer operators, graders, excavators, and full-time farm tractor drivers finding increased odds of lung cancer.[155] Another cohort study of operators of heavy construction equipment also showed increased lung cancer incidence for these workers.[156]

For the EPA Diesel HAD, EPA reviewed 22 epidemiologic studies in detail, finding increased lung cancer risk in 8 out of 10 cohort studies and 10 out of 12 case-control studies. Relative risk for lung cancer associated with exposure range from 1.2 to 2.6. In addition, two meta-analyses of occupational studies of diesel exhaust and lung cancer have estimated the smoking-adjusted relative risk of 1.35 and 1.47, examining 23 and 30 studies, respectively.[157,158] That is, these two studies show an overall increase in lung cancer for the exposed groups of 35 percent and 47 percent compared with the groups not exposed to diesel exhaust. In the EPA Diesel HAD, EPA selected 1.4 as a reasonable estimate of occupational relative risk for further analysis.

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

and the absence of quantitative exposure characterization in retrospective studies.

However, in the absence of a cancer unit risk, the EPA Diesel HAD sought to provide additional insight into the possible ranges of risk that might be present in the population. Such insights, while not confident or definitive, nevertheless contribute to an understanding of the possible public health significance of the lung cancer hazard. The possible risk range analysis was developed by comparing a typical environmental exposure level to a selected range of occupational exposure levels and then proportionally scaling the occupationally observed risks according to the exposure ratio's to obtain an estimate of the possible environmental risk. If the occupational and environmental exposures are similar, the environmental risk would approach the risk seen in the occupational studies whereas a much higher occupational exposure indicates that the environmental risk is lower than the occupational risk. A comparison of environmental and occupational exposures showed that for certain occupations the exposures are similar to environmental exposures while, for others, they differ by a factor of about 200 or more.

The first step in this process is to note that the occupational relative risk of 1.4, or a 40 percent from increased risk compared with the typical 5 percent lung cancer risk in the U.S. population, translates to an increased risk of 2 percent (or 10^{-2}) for these diesel exhaust exposed workers. The Diesel HAD derived a typical nationwide average environmental exposure level of 0.8 $\mu g./m^3$ for diesel PM from highway sources for 1996. This estimate was based on national exposure modeling; the derivation of this exposure is discussed in detail in the EPA Diesel HAD. Diesel PM is a surrogate for diesel exhaust and, as mentioned above, has been classified as a carcinogen by some agencies.

The possible environmental risk range was estimated by taking the relative risks in the occupational setting, EPA selected 1.4 and converting this to absolute risk of 2% and then ratioing this risk by differences in the occupational vs environmental exposures of interest. A number of calculations are needed to accomplish this, and these can be seen in the EPA Diesel HAD. The outcome was that environmental risks from diesel exhaust using higher estimates of occupational exposure could range from a low of 10^{-4} to 10^{-5} or be as high as 10^{-3} if lower estimates of occupational exposure were used. Note that the environmental exposure of interest (0.8 $\mu g/m^3$) remains constant in this analysis, while the occupational exposure is a variable. The range of possible environmental risk is a reflection of the range of occupational exposures that could be associated with the relative and related absolute risk levels observed in the occupational studies.

While these risk estimates are exploratory and not intended to provide a definitive characterization of cancer risk, they are useful in gauging the possible range of risk based on reasonable judgment. It is important to note that the possible risks could also be higher or lower and a zero risk cannot be ruled out. Some individuals in the population may have a high tolerance to exposure from diesel exhaust and low cancer susceptibility. Also, one cannot rule out the possibility of a threshold of exposure below which there is no cancer risk, although evidence has not been seen or substantiated on this point.

Also, as discussed in the Diesel HAD, there is a relatively small difference between some

occupational settings where increased lung cancer risk is reported and ambient environmental exposures. The potential for small exposure differences underscores the concerns about the potential public hazard, since small differences suggest that environmental risk levels may be close to those observed in the occupational setting.

EPA also assessed air toxic emissions and their associated risk (the National-Scale Air Toxics Assessment or NATA for 1996), and we concluded that diesel exhaust ranks with other substances that the national-scale assessment suggests pose the greatest relative risk.[159] This national assessment estimates average population inhalation exposures to diesel PM in 1996 for nonroad as well as highway sources. These are the sum of ambient levels in various locations weighted by the amount of time people spend in each of the locations. This analysis shows a somewhat higher diesel exposure level than the 0.8 $\mu g/m^3$ used to develop the risk perspective in the Diesel HAD. The average nationwide NATA mobile exposure levels are 1.44 $\mu g/m^3$ total with highway source contribution of 0.46 $\mu g/m^3$ and a nonroad source contribution of 0.98 $\mu g/m^3$. The average urban exposure was 1.64 $\mu g/m^3$ and the average rural exposure was 0.55 $\mu g/m^3$. In five percent of urban census tracts across the United States, average exposures were above 4.33 $\mu g/m^3$. The EPA Diesel HAD states that use of the NATA exposure estimates instead of the 0.8 $\mu g/m^3$ estimate results in a similar risk perspective.[G]

In summary, even though EPA does not have a specific carcinogenic potency with which to accurately estimate the carcinogenic impact of diesel exhaust, the likely hazard to humans together with the potential for significant environmental risks leads us to conclude that diesel exhaust emissions need to be reduced from nonroad engines in order to protect public health. The following factors lead to our determination.

1. EPA has officially designated diesel exhaust as a likely human carcinogen due to inhalation at environmental exposure. Other organizations have made similar determinations.
2. The entire U.S. population is exposed to various levels of diesel exhaust. The higher exposures at environmental levels is comparable to some occupational exposure levels, so that environmental risk could be the same as, or approach, the risk magnitudes observed in the occupational epidemiologic studies.
3. The possible range of risk for the general U.S. population due to exposure to diesel exhaust is 10^{-3} to 10^{-5} although the risk could be lower and a zero risk cannot be ruled out.

Thus, the concern for a carcinogenicity hazard resulting from diesel exhaust exposures is longstanding based on studies done over many years. This hazard may be widespread due to the

[G]It should be note that, as with any modeling assessment, there are a number of significant limitations and uncertainties in NATA. These uncertainties and limitations include use of default values to model local conditions, limitations in emissions data, uncertainties in locating emissions spatially and temporally, and accounting for atmospheric processes. NATA limitations and uncertainties are discussed at the following website: http://www.epa.gov/ttn/atw/nata/natsalim2.html

ubiquitous nature of exposure to diesel exhaust.

2.2.1.2 Other Health Effects of Diesel Exhaust

The acute and chronic exposure-related effects of diesel exhaust emissions are also of concern to the Agency. The Diesel HAD established an inhalation Reference Concentration (RfC) specifically based on animal studies of diesel exhaust. An RfC is defined by EPA as "an estimate of a continuous inhalation exposure to the human population, including sensitive subgroups, with uncertainty spanning perhaps an order of magnitude, that is likely to be without appreciable risks of deleterious noncancer effects during a lifetime." EPA derived the RfC from consideration of four well-conducted chronic rat inhalation studies showing adverse pulmonary effects.[160, 161, 162, 163] The diesel RfC is based on a "no observable adverse effect" level of 144 $\mu g/m^3$ that is further reduced by applying uncertainty factors of 3 for interspecies extrapolation and 10 for human variations in sensitivity. The resulting RfC derived in the Diesel HAD is 5 $\mu g/m^3$ for diesel exhaust as measured by diesel PM. This RfC does not consider allergenic effects such as those associated with asthma or immunologic effects. There is growing evidence that diesel exhaust can exacerbate these effects, but the exposure-response data are presently lacking to derive an RfC.

While there have been relatively few human controlled exposure studies associated specifically with the noncancer impact of diesel PM alone, diesel PM is frequently part of the ambient particles studied in numerous epidemiologic studies. Conclusions that health effects associated with ambient PM in general are relevant to diesel PM are supported by studies that specifically associate observable human noncancer health effects with exposure to diesel PM. As described in the Diesel HAD, these studies include some of the same health effects reported for ambient PM, such as respiratory symptoms (cough, labored breathing, chest tightness, wheezing), and chronic respiratory disease (cough, phlegm, chronic bronchitis and suggestive evidence for decreases in pulmonary function). Symptoms of immunological effects such as wheezing and increased allergenicity are also seen. Studies in rodents, especially rats, show the potential for human inflammatory effects in the lung and consequential lung tissue damage from chronic diesel exhaust inhalation exposure. The Diesel HAD notes that acute or short-term exposure to diesel exhaust can cause acute irritation (e.g., eye, throat, bronchial), neurophysiological symptoms (e.g., lightheadedness, nausea), and respiratory symptoms (cough, phlegm). There is also evidence for an immunologic effect such as the exacerbation of allergenic responses to known allergens and asthma-like symptoms.[164,165,166,167] The Diesel HAD lists numerous other studies as well. Also, as discussed in more detail previously, in addition to its contribution to ambient PM inventories, diesel PM is of special concern because it has been associated with an increased risk of lung cancer.

The Diesel HAD also briefly summarizes health effects associated with ambient PM and the EPA's annual NAAQS of 15 $\mu g/m^3$. There is a much more extensive body of human data showing a wide spectrum of adverse health effects associated with exposure to ambient PM, of which diesel exhaust is an important component. The RfC is not meant to say that 5 $\mu g/m^3$ provides adequate public health protection for ambient $PM_{2.5}$. In fact, there may be benefits to

reducing diesel PM below 5 $\mu g/m^3$ since diesel PM is a major contributor to ambient $PM_{2.5}$.[H]

Also, as mentioned earlier in the health effects discussion for $PM_{2.5}$, there are a number of other health effects associated with PM in general—and motor vehicle exhaust, including that from diesel engines in particular—that provide additional evidence for the need for significant emission reductions from nonroad diesel sources.

As indicated earlier, a number of recent studies have associated living near roadways with adverse health effects. Two of the studies cited earlier will be mentioned again here as examples of the type of work that has been done. A Dutch study (discussed earlier by G. Hoek et al., 2002) of a population of people 55-69 years old found that there was an elevated risk of heart and lung related mortality among populations living near high traffic roads. A review discussed earlier of studies (by R. Delfino et al., 2002) of the respiratory health of people living near roadways included a publication indicating that the risk of asthma and related respiratory disease appeared elevated in people living near heavy traffic.[168] These studies offer evidence that people exposed most directly to emissions from mobile sources, including those from diesel engines, face an elevated risk of illness or death.

All of these health effects plus the designation of diesel exhaust as a likely human carcinogen provide ample health justification for control.

Public comments from the Building and Construction Trades Department, AFL-CIO, and International Union of Operating Engineers supported the need to adopt the nonroad rule noting that exposure to diesel emissions from nonroad diesel engine poses a great risk to workers in the construction industry and other occupations, but are highest among construction workers because they work in close proximity to the exposure source, and are exposed daily to the hazards of nonroad diesel pollution. In their comments, BCTD noted that construction workers may be exposed to hazards generated from work performed by other trades employed by other contractors because sources of diesel exposure are scattered throughout the site. They noted further that in an exposure study, railway workers, heavy equipment operators and miners had

[H]It should again be noted that recent epidemiologic studies of ambient $PM_{2.5}$ do not indicate a threshold of effects at low concentrations. For example, the authors of the Pope reanalysis note that, for the range of exposures considered in their reanalysis, the slope of the concentration-response function appears to be monotonic and nearly linear, although they cannot exclude the potential for a leveling off or steepening at higher exposure levels. The EPA Science Advisory Board's Advisory Council for Clean Air Compliance, which provides advice and review of EPA's methods for assessing the benefits and costs of the Clean Air Act under Section 812 of the Act, has advised that there is currently no scientific basis for assuming any specific threshold for the PM-related health effects considered in typical benefits analyses (EPA-SAB-Council-ADV-99-012, 1999). Also, the National Research Council, in its own review of EPA's approach to benefits analyses, has agreed with this advice. This advice is supported by the recent literature on health effects of PM exposure (Daniels et al., 2000; Pope, 2000; Pope et al., 2002, Rossi et al., 1999; Schwartz, 2000, Schwartz, Laden, and Zanobetti 2002 [Schwarz, J.; Laden, F.; and Zanobetti, A. (2002) The Concentration-Response Relation between PM2.5 and Daily Deaths. Environ Health Perspect 110(10): 1025-1029]) which generally finds no evidence of a non-linear concentration-response relationship and, in particular, no evidence of a distinct threshold for health effects. The most recent draft of the EPA Air Quality Criteria for Particulate Matter (U.S. EPA, 2002) reports only one study, analyzing data from Phoenix, AZ, that reported even limited evidence suggestive of a possible threshold for $PM_{2.5}$ (Smith et al., 2000).

higher mortality rates from lung cancer and all causes than workers without diesel exposure. Heavy equipment operators and miners had comparable relative risk for lung cancer, both of which were over 2.5 times that of non-exposed workers (Boffetta, 1988).

2.2.1.3 Diesel Exhaust PM Ambient Levels

Because diesel PM is part of overall ambient PM and cannot be easily distinguished from overall PM, we do not have direct measurements of diesel PM in the ambient air. Diesel PM concentrations are estimated instead using one of three approaches: 1) ambient air quality modeling based on diesel PM emission inventories; 2) using elemental carbon concentrations in monitored data as surrogates; or 3) using the chemical mass balance (CMB) model in conjunction with ambient PM measurements. (Also, in addition to CMB, UNMIX/PMF have also been used). Estimates using these three approaches are described below. In addition, estimates developed using the first two approaches above are subjected to a statistical comparison to evaluate overall reasonableness of estimated concentrations from ambient air quality modeling. It is important to note that, while there are inconsistencies in some of these studies on the relative importance of gasoline and diesel PM, the studies discussed in the Diesel HAD all show that diesel PM is a significant contributor to overall ambient PM. Some of the studies differentiate nonroad from highway diesel PM.

2.2.1.3.1 Toxics Modeling and Methods

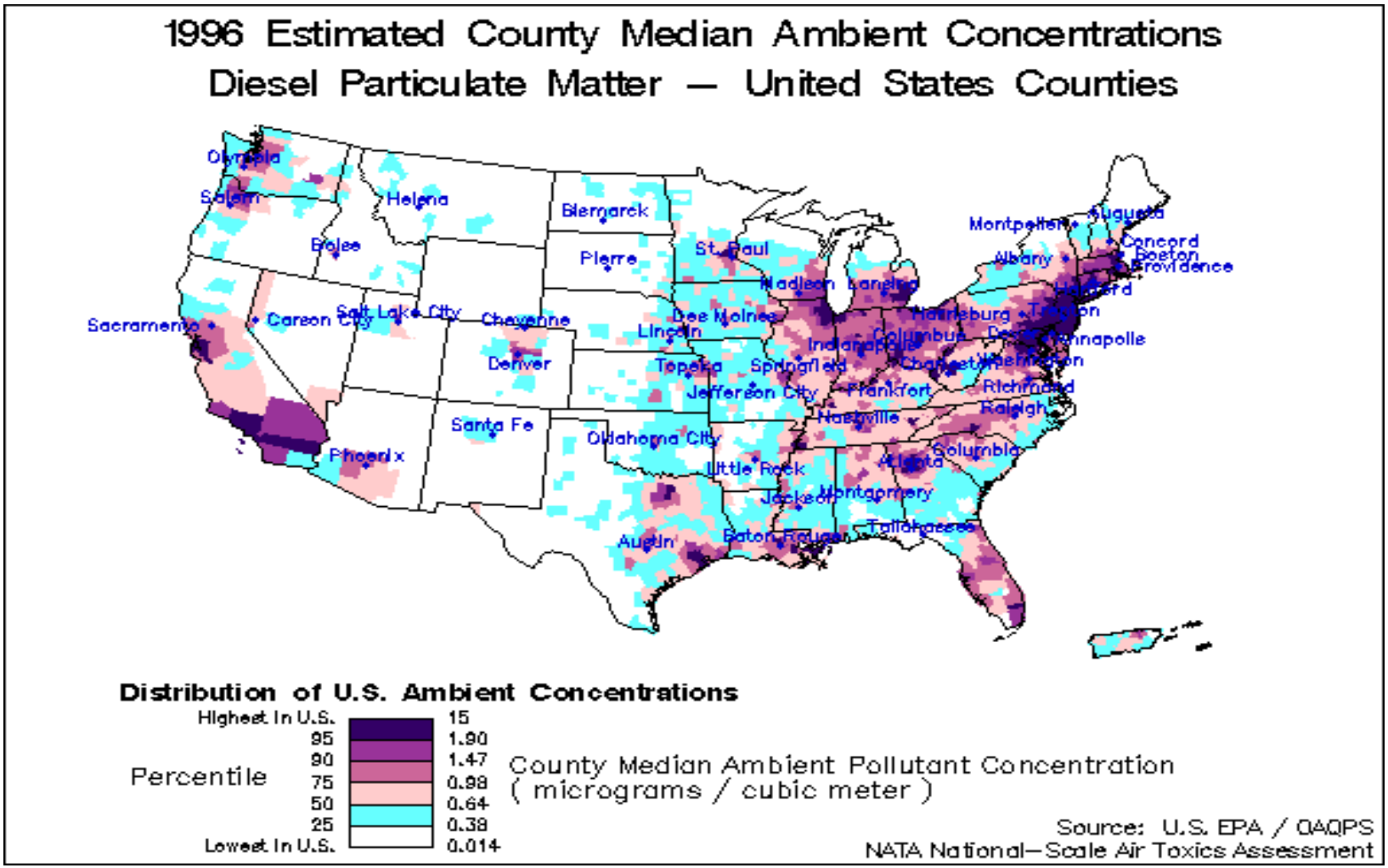
In addition to the general ambient PM modeling conducted for this rulemaking, diesel PM concentrations for 1996 were estimated as part of the National-Scale Air Toxics Assessment (NATA; EPA, 2002). In this assessment, the PM inventory developed for the recent regulation promulgating 2007 heavy duty vehicle standards was used (EPA, 2000). Note that the nonroad inventory used in this modeling was based on an older version of the draft NONROAD Model that showed higher diesel PM than the current version, so the ambient concentrations may be biased high. Ambient impacts of mobile source emissions were predicted using the Assessment System for Population Exposure Nationwide (ASPEN) dispersion model.

From the NATA 1996 modeling, overall mean annual national ambient diesel PM levels of 2.06 $\mu g/m^3$ were calculated with a mean of 2.41 in urban counties and 0.74 in rural counties. Table 2.2.1-1 below summarizes the distribution of average ambient concentrations to diesel PM at the national scale. Over half of the diesel PM can be attributed to nonroad diesel engines. A map of county median concentrations (median of census tract concentrations) from highway and nonroad sources is provided in Figure 2.2.1-1. We have not generated a map depicting the estimated geographic distribution of nonroad diesel PM alone. While the high median concentrations are clustered in the Northeast, Great Lake States and California, areas of high median concentrations are distributed throughout the United States.

Table 2.2.1-1
Distribution of Average Ambient Concentrations of
Diesel PM at the National Scale in the 1996 NATA Assessment.

	Nationwide ($\mu g/m^3$)	Urban ($\mu g/m^3$)	Rural ($\mu g/m^3$)
5th Percentile	0.33	0.51	0.15
25th Percentile	0.85	1.17	0.42
Average	2.06	2.41	0.74
75th Percentile	2.45	2.7	0.97
95th Percentile	5.37	6.06	1.56
Onroad Contribution to Average	0.63	0.72	0.27
Nonroad Contribution to Average	1.43	1.69	0.47

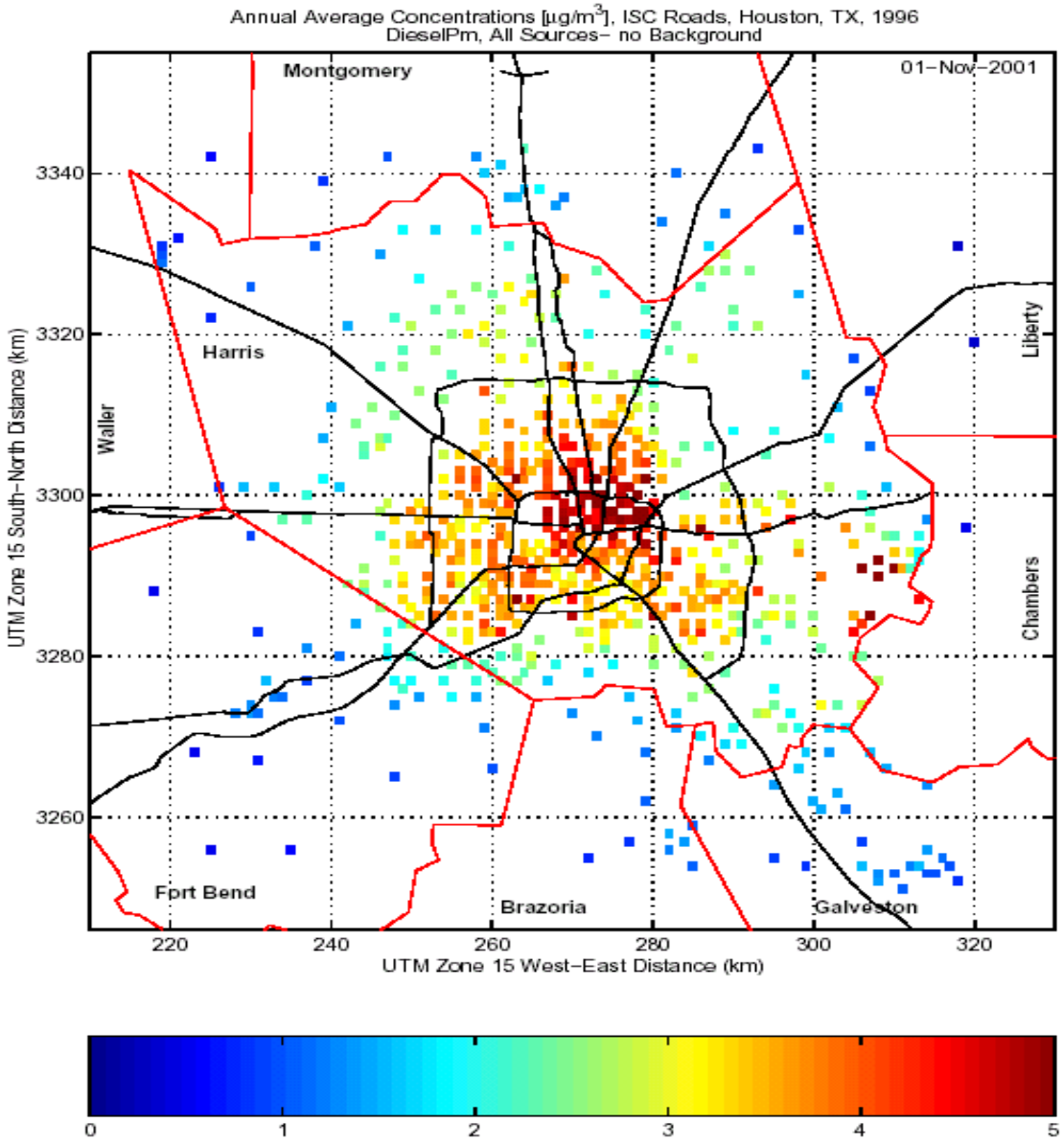
Figure 2.2.1-1
Estimated County Median Concentrations of Diesel Particulate Matter



Source: EPA National-Scale Air Toxics Assessment for 1996. Results should not be used to draw conclusions about local concentrations. Results are most meaningful at the Regional or National level.

Diesel PM concentrations were also recently modeled across a representative urban area, Houston, Texas, for 1996, using the Industrial Source Complex Short Term (ISCST3) model.[169] The methodology used to model diesel PM concentrations is the same as the methodology used for benzene and other hazardous air pollutants, as described in a recent EPA technical report.[170] For Harris County, which has the highest traffic density in Houston area, link-based diesel PM emissions were estimated for highway mobile sources, using diesel PM emission rates developed for the recent EPA 2007 heavy duty engine and highway diesel fuel sulfur control rule.[171] This link-based modeling approach is designed to specifically account for local traffic patterns within the urban center, including diesel truck traffic along specific roadways. For other counties in the Houston metropolitan area, county level emission estimates from highway vehicles were allocated to one kilometer grid cells based on total roadway miles. Nonroad diesel emissions for Houston area counties were obtained from the inventory done for the 2007 heavy duty rule, and allocated to one kilometer grid cells using activity surrogates. The modeling in Houston suggests strong spatial gradients (on the order of a factor of 2-3 across a modeling domain) for diesel PM and indicates that "hotspot" concentrations can be very high. Values as high as 8 $\mu g/m^3$ at were estimated at a receptor versus a 3 $\mu g/m^3$ average in Houston. Such "hot spot" concentrations suggest both a high localized exposure plus higher estimated average annual exposure levels for urban centers than what has been estimated in assessments such as NATA 1996, which are designed to focus on regional and national scale averages. Figure 2.2.1-2 depicts the spatial distribution of diesel PM concentrations in Houston.

Figure 2.2.1-2
Annual Average Ambient Concentrations of Diesel PM in Houston, 1996, based on Dispersion Modeling Using Industrial Source Complex Short Term (ISCST3) model.



2.2.1.3.2 Elemental Carbon Measurements

As shown in Figures 2.1.1-1 to 3, the carbonaceous component is significant in ambient PM. The carbonaceous component consists of organic carbon and elemental carbon. Monitoring data on elemental carbon concentrations can be used as a surrogate to determine ambient diesel PM concentrations. Elemental carbon is a major component of diesel exhaust, contributing to

approximately 60-80 percent of diesel particulate mass, depending on engine technology, fuel type, duty cycle, lube oil consumption, and state of engine maintenance. In most areas, diesel engine emissions are 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 areas, ambient diesel PM concentrations can be bounded using elemental carbon measurements.

The measured mass of elemental carbon at a given site varies depending on the measurement technique used. Moreover, to estimate diesel PM concentration based on elemental carbon level, one must first estimate the percentage of PM attributable to diesel engines and the percentage of elemental carbon in diesel PM. Thus, there are significant uncertainties in estimating diesel PM concentrations using an elemental carbon surrogate. Also, there are issues with the measurement methods used for elemental carbon. Many studies used thermal optimal transmission (TOT), the NIOSH method developed at Sunset laboratories. Other studies used thermal optical reflectance (TOR), a method developed by Desert Research Institute. EPA has developed multiplicative conversion factors to estimate diesel PM concentrations based on elemental carbon levels.[172] Results from several source apportionment studies were used to develop these factors.[173, 174, 175, 176, 177, 178, 179] Average conversion factors were compiled together with lower and upper bound values. Conversion factors (CFs) were calculated by dividing the diesel $PM_{2.5}$ concentration reported in these studies by the total organic carbon or elemental carbon concentrations also reported in the studies. Table 2.2.1-2 presents the minimum, maximum, and average EC conversion factors as a function of:

- Measurement technique
- Eastern or Western United States
- Season
- Urban or rural

The reported minimum, maximum, and average values in Table 2.2.1-2 are the minima, maxima, and arithmetic means of the EC conversion factors across all sites (and seasons, where applicable) in the given site subset. For the TOT data collected in the East, the minimum, maximum, and average conversion factors are all equal. This is because these values were based only on one study where the data were averaged over sites, by season.[180] Depending on the measurement technique used, and assumptions made in converting elemental carbon concentration to diesel PM concentration, average nationwide concentrations for current years of diesel PM estimated from elemental carbon data range from about 1.2 to 2.2 $\mu g/m^3$. EPA has compared these estimates based on elemental carbon measurements with modeled concentrations in the NATA for 1996. Results of comparisons of mean percentage differences are presented in Table 2.2.1-3. These results show that the two sets of data agree reasonably well, with estimates for the majority of sites within a factor of 2, regardless of the measurement technique or methodology for converting elemental carbon to diesel PM concentration. Agreement was better when modeled concentrations were adjusted to reflect recent changes in the nonroad inventory. The best model performance based on the fraction of modeled values within 100 % of the monitored value is for the DPM-maximum value, which reflects changes to the nonroad

inventory model. The corresponding fractions of modeled values within 100 % of the monitored value are 73 % for TOR sites, 80 % for TOT sites, and 92 % for TORX sites. All in all, this performance compares favorably with the model to monitor results for other pollutants assessed in NATA, with the exception of benzene, for which the performance of the NATA modeling was better.

2.2.1.3.3 Chemical Mass Balance Receptor Modeling and Source Apportionment

The third approach for estimating ambient diesel PM concentrations uses the chemical mass balance (CMB) model for source apportionment in conjunction with ambient PM measurements and chemical source "fingerprints" to estimate ambient diesel PM concentrations. The CMB model uses a statistical fitting technique to determine how much mass from each source would be required to reproduce the chemical fingerprint of each speciated ambient monitor. Inputs to the CMB model applied to ambient $PM_{2.5}$ include measurements made at an air monitoring site and measurements made of each of the source types suspected to affect the site. The CMB model uses a statistical fitting technique ("effective variance weighted least squares") to determine how much mass from each source would be required to reproduce the chemical fingerprint of each speciated ambient monitor. This calculation is based on optimizing the sum of sources, so that the difference between the ambient monitor and the sum of sources is minimized. The optimization technique employs "fitting species" that are related to the sources. The model assumes that source profiles are constant over time, that the sources do not interact or react in the atmosphere, that uncertainties in the source fingerprints are well-represented, and that all sources are represented in the model.

This source apportionment technique presently does not distinguish between highway and nonroad but, instead, gives diesel PM as a whole. One can allocate the diesel PM numbers based on the inventory split between highway and nonroad diesel, although this allocation was not done in the studies published to date. This source apportionment technique can though distinguish between diesel and gasoline PM. Caution in interpreting CMB results is warranted, as the use of fitting species that are not specific to the sources modeled can lead to misestimation of source contributions. Ambient concentrations using this approach are generally about 1 $\mu g/m^3$ annual average. UNMIX/PMF models show similar results.

Table 2.2.1-2
Summary of Calculated Elemental Carbon (EC) Conversion Factors
(Conversion factors to convert total EC to diesel $PM_{2.5}$ concentration)

Ambient Measurement Technique: TOT or TOR	East or West	Season	Location Type General	MIN[a]	MAX[a]	AVERAGE[a]	Recommended Conversion Factors	
							EAST	WEST
TOT	East	Fall (Q4)	Mixed	2.3	2.3	2.3	X	
	East	Spring (Q2)	Mixed	2.4	2.4	2.4	X	
	East	Summer (Q3)	Mixed	2.1	2.1	2.1	X	
	East	Winter (Q1)	Mixed	2.2	2.2	2.2	X	
	West	Unknown	Urban	1.2	2.4	1.6		X
TOT Total				1.2	2.4	2.0		
TOR		Winter	Rural	0.6	1.0	0.8	X	X
		Winter	Urban	0.5	1.0	0.7	X	X
	Winter Total			0.5	1.0	0.8		
TOR Total				0.5	1.0	0.8		
Grand Total				0.5	2.4	1.3		

Source: ICF Consulting for EPA, 2002, Office of Transportation and Air Quality. Report No. EPA420-D-02-004.
[a] Minimum, maximum, or average value across all sites of the estimated conversion factors.

TOT = thermal optimal transmission, the NIOSH method developed at Sunset laboratories.
TOR = thermal optical reflectance, a method developed by Desert Research Institute.

Table 2.2.1-3
Summary of Differences Between the Nearest Modeled Concentration
of Diesel Pm from the National Scale Air Toxics Assessment and Monitored Values
Based on Elemental Carbon Measurements (Diesel PM model-to-measurement comparison)

Modeled Variable[a]	Monitored Variable[b]	N	Mean Modeled Value	Mean Monitored Value	Mean Difference	Mean % Difference	Fraction of Modeled Values Within			
							10%	25%	50%	100%
concnear	TOR	15	1.56	0.94	0.63	100	0.07	0.13	0.53	0.53
concnear2	TOR	15	1.20	0.94	0.26	56	0.07	0.13	0.47	0.60
concnear	TORH	15	1.56	1.16	0.40	62	0.00	0.07	0.40	0.60
concnear2	TORH	15	1.20	1.16	0.04	26	0.00	0.07	0.33	0.73
concnear	TORL	15	1.56	0.64	0.92	190	0.13	0.40	0.47	0.53
concnear2	TORL	15	1.20	0.64	0.55	126	0.07	0.33	0.47	0.53
concnear	TOT	95	2.61	1.73	0.88	80	0.12	0.21	0.45	0.68
concnear2	TOT	95	2.05	1.73	0.32	42	0.11	0.37	0.53	0.77
concnear	TOTH	95	2.61	2.10	0.52	61	0.11	0.22	0.46	0.74
concnear2	TOTH	95	2.05	2.10	-0.05	27	0.11	0.35	0.53	0.80
concnear	TOTL	95	2.61	1.52	1.09	101	0.09	0.17	0.43	0.63
concnear2	TOTL	95	2.05	1.52	0.52	58	0.09	0.32	0.52	0.72
concnear	TORX	88	2.31	1.70	0.61	47	0.10	0.30	0.59	0.78
concnear2	TORX	88	1.81	1.70	0.11	15	0.17	0.30	0.59	0.85
concnear	TORXH	88	2.31	2.23	0.08	13	0.11	0.26	0.60	0.84
concnear2	TORXH	88	1.81	2.23	-0.42	-12	0.08	0.22	0.52	0.92
concnear	TORXL	88	2.31	1.19	1.12	110	0.10	0.26	0.41	0.65
concnear2	TORXL	88	1.81	1.19	0.62	65	0.14	0.31	0.52	0.74

Source: ICF Consulting for EPA, 2002, Office of Transportation and Air Quality. Report No. EPA420-D-02-004.

[a] Modeled variable:
- concnear Nearest modeled DPM concentration from the 1996 NATA
- concnear2 Nearest modeled DPM concentration with NATA concentrations adjusted to be consistent with changes to the nonroad inventory model

[b] Monitored variable:
- TOR EC value multiplied by TOR average correction factor
- TORH EC value multiplied by TOR maximum correction factor
- TORL EC value multiplied by TOR minimum correction factor
- TOT EC value multiplied by TOT average correction factor
- TOTH EC value multiplied by TOT maximum correction factor
- TOTL EC value multiplied by TOR minimum correction factor
- TORX TOR values plus the TOR equivalent values multiplied by TOR average correction factor
- TORXH TOR values plus the TOR equivalent values multiplied by TOR maximum correction factor
- TORXL TOR values plus the TOR equivalent values multiplied by TOR minimum correction factor

Because of the correlation of diesel and gasoline exhaust PM emissions in time and space, chemical molecular species that provide markers for separation of these sources have been sought. 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. As mentioned previously, however, caution in interpreting CMB results is warranted. Markers that have been used in CMB receptor modeling have included elemental carbon, polycyclic aromatic hydrocarbons (PAHs), organic acids, hopanes, and steranes.

It should be noted that since receptor modeling is based on the application of source profiles to ambient measurements, this estimate of diesel PM concentrations includes the contribution from on-highway and nonroad sources of diesel PM, although no study to date has included source profiles from nonroad engines. Engine operations, fuel properties, regulations, and other factors may distinguish nonroad diesel engines from their highway counterparts.

In addition, this model accounts for primary emissions of diesel PM only; the contribution of secondary aerosols is not included. The role of secondarily formed organic PM in urban $PM_{2.5}$ concentrations is not known, particularly from diesel engines.

The first major application of organic tracer species in applying the CMB model evaluated ambient $PM_{2.0}$ in Los Angeles, CA sampled in 1982.[181] This study was the first to distinguish gasoline and diesel exhaust. CMB model application at four sites in the Los Angeles area estimated ambient diesel $PM_{2.0}$ concentrations to be 1.02-2.72 $\mu g/m^3$. Note that diesel PM estimates are derived from source profiles measured on in-use diesel trucks.

Another major study examining diesel exhaust separately from gasoline exhaust and other sources is the Northern Front Range Air Quality Study (NFRAQS).[182] This study was conducted in the metropolitan Denver, CO area during 1996-1997. The NFRAQS study employed a different set of chemical species, including PAHs and other organics to produce source profiles for a diverse range of mobile sources, including "normal emitting" gasoline vehicles, cold start gasoline vehicles, high emitting gasoline vehicles, and diesel vehicles. Average source contributions from diesel engines in NFRAQS were estimated to be 1.7 $\mu g/m^3$ in an urban area, and 1.2 $\mu g/m^3$ in a rural area. Source profiles in this study were based on highway vehicles.

The CMB model was applied in California's San Joaquin Valley during winter 1995-1996.[183] The study employed similar source tracers as the earlier study of Los Angeles PM2.0, in addition to other more specific markers. Diesel PM source contribution estimates in Bakersfield, CA were 3.92 and 5.32 during different measurement periods. Corresponding estimates in Fresno, CA were 9.68 and 5.15 $\mu g/m^3$. In the Kern Wildlife Refuge, diesel PM source contribution estimates were 1.32 and 1.75 $\mu g/m^3$ during the two periods.

The CMB model was applied in the Southeastern United States on data collected during the Southeastern Aerosol Research and Characterization (SEARCH) study (Zheng et al., 2002). Modeling was conducted on data collected during April, July, and October 1999 and January 2000. Examining ambient monitors in urban, suburban, and rural areas, the modeled annual average contribution of primary diesel emissions to ambient $PM_{2.5}$ was 3.20-7.30 $\mu g/m^3$ in N. Birmingham, AL, 1.02-2.43 $\mu g/m^3$ in Gulfport, MS, 3.29-5.56 $\mu g/m^3$ in Atlanta, GA, and 1.91-3.07 $\mu g/m^3$ in Pensacola, FL, which together represented the urban sites in the study. Suburban sites in the study were located outside Pensacola, FL (1.08-1.73 $\mu g/m^3$). Rural sites were located in Centreville, AL (0.79-1.67 $\mu g/m^3$), Oak Grove, MS (1.05-1.59 $\mu g/m^3$), and Yorkville, GA (1.07-2.02 $\mu g/m^3$).

The CMB model was applied to ambient $PM_{2.5}$ data collected during a severe photochemical smog event during 1993 in Los Angeles using organic tracers.[184] Modeled concentrations of

diesel contributions to $PM_{2.5}$ during this episode were conducted for Long Beach (8.33 $\mu g/m^3$), downtown Los Angeles (17.9 $\mu g/m^3$), Azusa (14.9 $\mu g/m^3$), and Claremont, CA (7.63 $\mu g/m^3$).

While these studies provide an indication that diesel exhaust is a substantial contributor to ambient $PM_{2.5}$ mass, they should still be viewed with caution. CMB modeling depends on ensuring the use of highly specific tracer species. If sources, such as nonroad diesel engines, are chemically different from other sources, including highway diesel trucks, the CMB model can misestimate source contributions. Nevertheless, these studies provide information that is complementary to source-oriented air quality modeling (discussed above). From these studies, it is apparent that diesel exhaust is a substantial contributor to ambient $PM_{2.5}$, even in remote and rural areas.

2.2.1.4 Diesel Exhaust PM Exposures

Exposure of people to diesel exhaust depends on their various activities, the time spent in those activities, the locations where these activities occur, and the levels of diesel exhaust pollutants (such as PM) in those locations. While ambient levels are specific for a particular location, exposure levels account for such factors as a person moving from location to location, proximity to the emission source, and whether the exposure occurs in an enclosed environment.

2.2.1.4.1 Occupational Exposures

Diesel particulate exposures have been measured for a number of occupational groups over various years but generally for more recent years (1980s and later) rather than earlier years. Occupational exposures had a wide range varying from 2 to 1,280 $\mu g/m^3$ for a variety of occupational groups including miners, railroad workers, firefighters, air port crew, public transit workers, truck mechanics, utility linemen, utility winch truck operators, fork lift operators, construction workers, truck dock workers, short-haul truck drivers, and long-haul truck drivers. These individual studies are discussed in the Diesel HAD.

The highest exposure to diesel PM is for workers in coal mines and noncoal mines, which are as high a 1,280 $\mu g/m^3$, as discussed in the Diesel HAD. The National Institute of Occupational Safety and Health (NIOSH) has estimated a total of 1,400,000 workers are occupationally exposed to diesel exhaust from on-road and nonroad equipment.

Many measured or estimated occupational exposures are for on-road diesel engines and some are for school buses.[185, 186, 187,188] Also, some (especially the higher ones) are for occupational groups (fork lift operator, construction workers, or mine workers) who would be exposed to nonroad diesel exhaust. Sometimes, as is the case for the nonroad engines, there are only estimates of exposure based on the length of employment or similar factors rather than a $\mu g/m^3$ level. Estimates for exposures to diesel PM for diesel fork lift operators have been made that range from 7 to 403 $\mu g/m^3$ as reported in the Diesel HAD. In addition, the Northeast States for Coordinated Air Use Management (NESCAUM) measured occupational exposures to particulate and elemental carbon near the operation of various diesel non-road equipment. Exposure groups include agricultural farm operators, grounds maintenance personnel (lawn and garden

equipment), heavy equipment operators conducting multiple job tasks at a construction site, and a saw mill crew at a lumber yard. Samples will be obtained in the breathing zone of workers. In a recently released interim report on occupational health risks from diesel engine exposure, pollution inside the cabs of heavy diesel equipment were shown to be up to 16 times higher than federal health recommendations. The diesel PM was estimated to exist at levels that pose risk of chronic inflammation and lung damage in exposed individuals (NESCAUM, 2003).

In public comments from the Building and Trade Department, AFL-CIO, they note their research center, the Center to Protect Workers' Rights, has sponsored research conducted by the Construction Occupational Health Program (COHP) at University of Massachusetts at Lowell which documents diesel emissions exposure among a number of trades employed on a major highway project underway in Boston, MA. Over 260 personal samples of diesel exposure were collected among laborers (116); operating engineers (113) and other trades including ironworkers (15), carpenters (9), piledrivers (5), boilermakers (1), plumbers (1) and surveyors (1). Exposures associated with specific work processes were also documented. Using the American Conference of Governmental Industrial Hygienists Threshold Limit Value (TLV) for diesel exhaust as elemental carbon of 20 ug/m^3 as proposed in 2002, the percentage of samples exceeding the TLV overall was 14 percent (Woskie, 2002; ACGIH, 2002). It should be noted that much of this project involves construction of underground tunnels. However, work in enclosed and/or poorly ventilated work areas is common in construction.

One recent study found that construction workers in Ontario are exposed to elevated concentrations of elemental carbon (EC) measured by thermal-optical transmission (TOT), which the authors used as a surrogate for diesel exhaust.[189] Task-based exposure measurements were made corresponding to engine use. Demolition laborers were exposed to between 4.9 to146 ug/m3 of EC-TOT while operating compressors, performing excavation and cleanup, and in tearing down structures. Construction equipment operating engineers were exposed to 4.3 to 7.8 ug/m3 EC-TOT while operating their machinery. Painters in new commercial construction were exposed to between 3.6 to 9.0 ug/m3 EC-TOT, as a result of operating mixers. While these concentrations are substantially higher than those seen in typical urban air, it is difficult to assign these EC-TOT measurements to diesel engines, and the study authors did not indicate the fuel source of the equipment used. However, it is likely that many of the engines in this study were diesel engines.

2.2.1.4.2 Ambient Exposures in the General Population

Currently, personal exposure monitors for PM cannot differentiate diesel from other PM. Thus, we use modeling to estimate exposures. Specifically, exposures for the general population are estimated by first conducting dispersion modeling of both highway and nonroad diesel emissions, described above, and then by conducting exposure modeling. The most comprehensive modeling for cumulative on-road and non-road exposures to diesel PM is the NATA. This assessment calculates exposures of the national population as a whole to a variety of air toxics, including diesel PM. As discussed previously, the ambient levels are calculated using the ASPEN dispersion model. As discussed above, the preponderance of modeled diesel PM concentrations are within a factor of 2 of diesel PM concentrations estimated from elemental

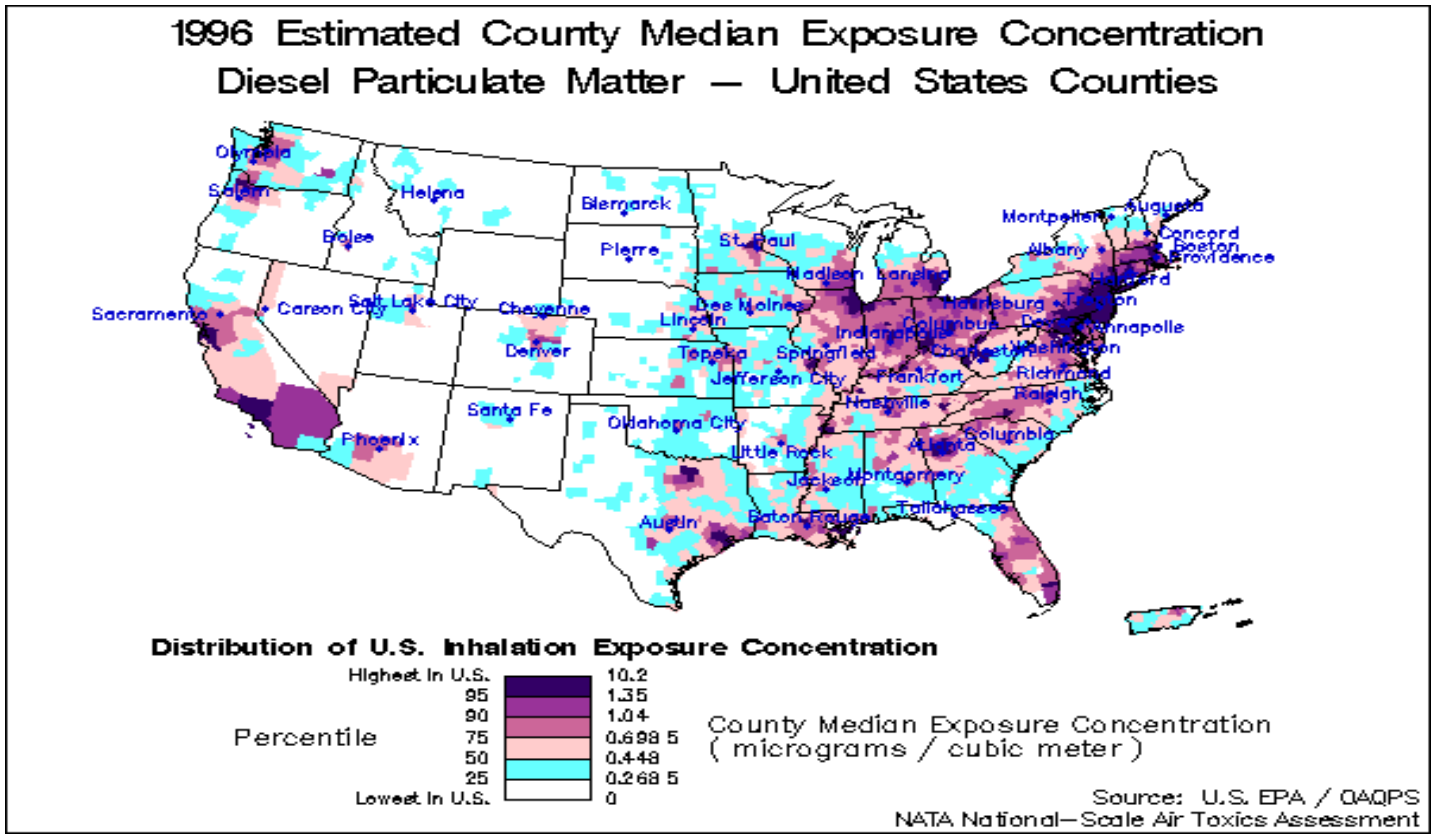
carbon measurements.[190] This comparison adds credence to the modeled ASPEN results and associated exposure assessment.

The modeled concentrations for calendar year 1996 are used as inputs into an exposure model called the Hazardous Air Pollution Exposure Model (HAPEM4) to calculate exposure levels. Average exposures calculated nationwide are 1.44 $\mu g/m^3$ with levels of 1.64 $\mu g/m^3$ for urban counties and 0.55 $\mu g/m^3$ for rural counties. Again, nonroad diesel emissions account for over half of the this exposure. Table 2.2.1-4 summarizes the distribution of average exposure concentrations to diesel PM at the national scale in the 1996 NATA assessment. Figure 2.2.1-3 presents a map of the distribution of median exposure concentrations for U.S. counties.

Table 2.2.1-4
Distribution of Average Exposure Concentrations to
Diesel PM at the National Scale in the 1996 NATA Assessment.

	Nationwide ($\mu g/m^3$)	Urban ($\mu g/m^3$)	Rural ($\mu g/m^3$)
5th Percentile	0.16	0.29	0.07
25th Percentile	0.58	0.81	0.29
Average	1.44	1.64	0.55
75th Percentile	1.73	1.91	0.67
95th Percentile	3.68	4.33	1.08
Onroad Contribution to Average	0.46	0.52	0.21
Nonroad Contribution to Average	0.98	1.12	0.34

Figure 2.2.1-3
Estimated County Median Exposure Concentrations of Diesel Particulate Matter



Source: EPA National-Scale Air Toxics Assessment for 1996. Results should not be used to draw conclusions about local exposure concentrations. Results are most meaningful at the Regional or National level.

As explained earlier, the fact that these levels are below the 5 $\mu g/m^3$ RfC (which is based on limited animal studies on diesel PM) does not necessarily mean that there are no adverse health implications from overall $PM_{2.5}$ exposure. The health studies for the $PM_{2.5}$ NAAQS are far more encompassing than the limited animal studies used to develop the RfC for diesel exhaust, and, also, the NAAQS applies to $PM_{2.5}$ regardless of its composition.

2.2.1.4.3 Ambient Exposures to Diesel Exhaust PM in Microenvironments

One common microenvironment for ambient exposures to diesel exhaust PM is beside freeways. Although freeway locations are associated mostly with highway rather than nonroad diesel enignes, there are many similarities between highway and nonroad diesel emissions, as discussed in the Diesel HAD. Also, similar spatial gradients in concentrations would be expected where nonroad equipment is used. The California Air Resources Board (California ARB) has measured elemental carbon near the Long Beach Freeway in 1993.[191] Levels measured ranged from 0.4 to 4.0 $\mu g/m^3$ (with one value as high as 7.5 $\mu g/m^3$) above background levels. Microenvironments associated with nonroad engines would include construction zones. PM and elemental carbon samples are being collected by NESCAUM in the immediate area of the nonroad engine operations (such as at the edge or fence line of the construction zone). Besides PM and elemental carbon levels, various toxics such as benzene, 1,3-butadiene, formaldehyde, and acetaldehyde will be sampled. The results should be especially useful since they focus on microenvironments affected by nonroad diesel engines.

Also, EPA is funding research in Fresno, California to measure indoor and outdoor PM component concentrations in the homes of over 100 asthmatic children. Some of these homes are located near agricultural, construction, and utility nonroad equipment operations. This work will measure infiltration of elemental carbon and other PM components to indoor environments. The project also evaluates lung function changes in the asthmatic children during fluctuations in exposure concentrations and compositions. This information may allow an evaluation of adverse health effects associated with exposures to elemental carbon and other PM components from on-road and nonroad sources.

2.2.2 Gaseous Air Toxics

Nonroad diesel engine emissions contain several substances known or suspected as human or animal carcinogens, or have noncancer health effects. These other compounds include benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, dioxin, and polycyclic organic matter (POM). Mobile sources, including nonroad diesel engines, contribute significantly to total emissions of these air toxics. All of these compounds were identified as national or regional "risk" drivers in the 1996 NATA. That is, these compounds pose a significant portion of the total inhalation cancer risk to a significant portion of the population. As discussed later in this section, this final rule will significantly reduce these emissions.

Nonroad engines are major contributors to nationwide cancer risk from air toxic pollutants, as indicated by the NATA 1996.[192] In fact, this study and the National Toxics Inventory (NTI) for 1996 are used throughout this section for toxics inventory information for nonroad sources.[193]

Also, a supplemental paper provides more detail on nonroad diesel exhaust.[194] In addition, a paper published by the Society of Automotive Engineers gives future projections to 2007 for these air toxics.[195] These references form the basis for much of what will be discussed in this section.

Figure 2.2.2-1 summarizes the contribution of nonroad engines to average nationwide lifetime upper bound cancer risk from outdoor sources in the 1996 NATA. These data do not include the cancer risk from diesel PM since EPA does not presently have a potency for diesel particulate/exhaust. Figure 2.2.2-2 depicts the nonroad engine contribution to average nationwide inhalation exposure for benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and acrolein. These compounds are all known or suspected human carcinogens, except for acrolein, which has serious noncancer health effects. All of these compounds were identified as national or regional risk drivers in the 1996 NATA, and mobile sources contribute significantly to total emissions in NATA. As indicated previously, NATA exposure and risk estimates are based on air dispersion modeling using the ASPEN model. Comparisons of the predicted concentrations from the model to monitor data indicate good agreement for benzene, where the ratio of median modeled concentrations to monitor values is 0.92, and results are within a factor of two at almost 90 percent of monitors.[196] Comparisons with aldehydes indicate significantly lower modeled concentrations than monitor values. Comparisons with 1,3-butadiene have not been done. Previously, extensive work was done on gaseous air toxic emissions including those from nonroad diesel and reported in EPA's 1993 Motor Vehicle-Related Air Toxics Study.[197] This final rule will reduce these emissions. Dioxin and some POM compounds have also been identified as probable human carcinogens and are emitted by mobile sources, although nonroad sources are less than 1% of total emissions for these compounds.

Figure 2.2.2-1

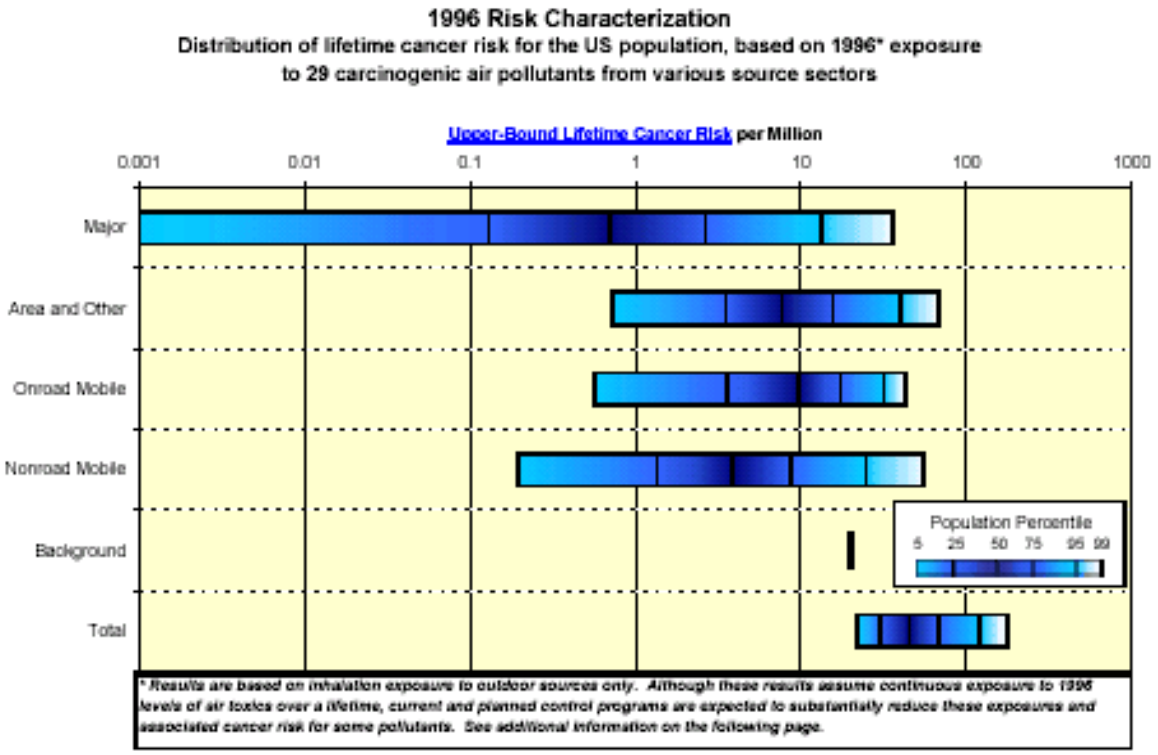
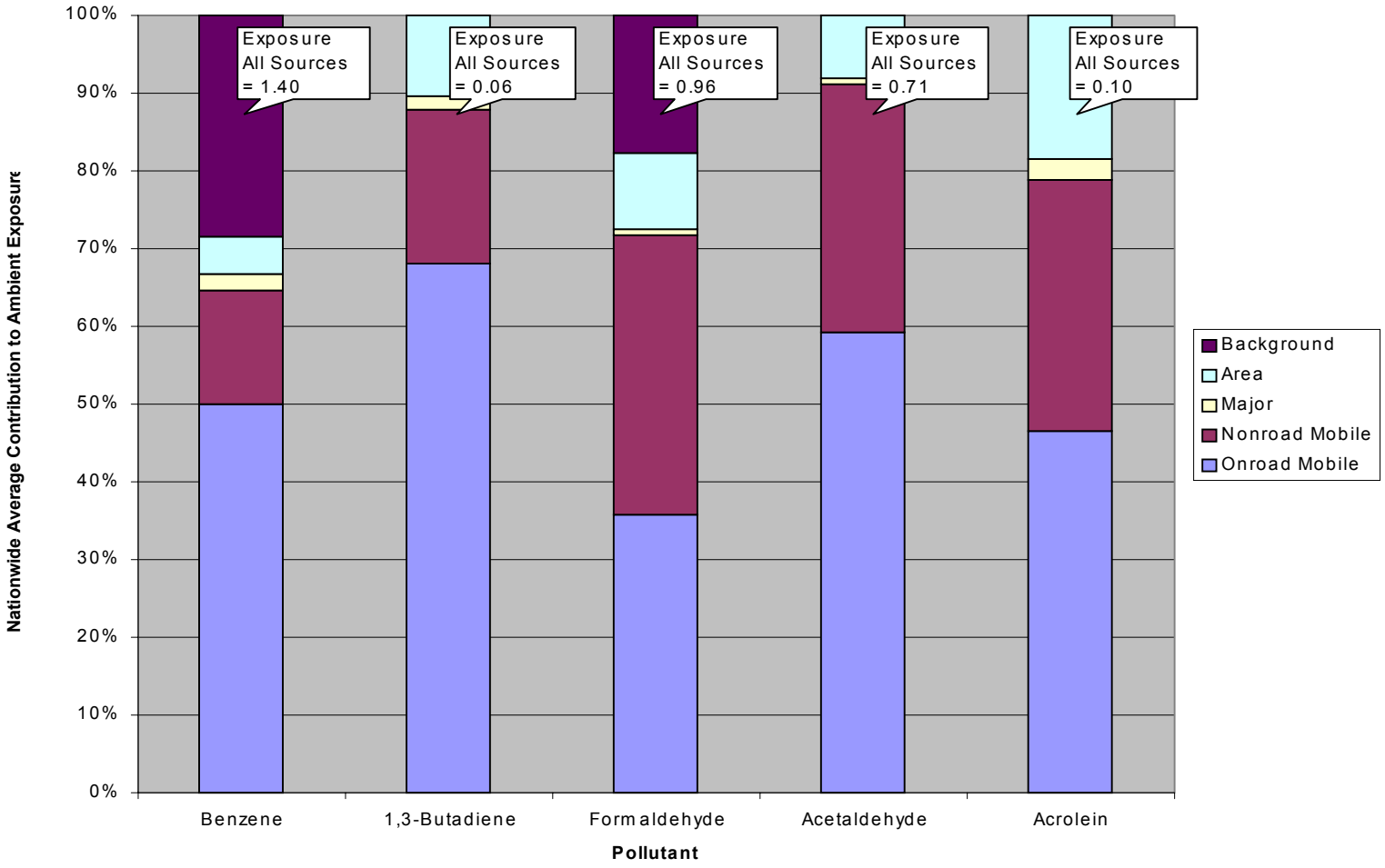

1996 Risk Characterization
Distribution of lifetime cancer risk for the US population, based on 1996* exposure
to 29 carcinogenic air pollutants from various source sectors
Upper-Bound Lifetime Cancer Risk per Million
0.001
0.01
0.1
1
10
100
1000
Major
Area and Other
Onroad Mobile
Nonroad Mobile
Background
Total
Population Percentile
5 25 50 75 95 99
* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated cancer risk for some pollutants. See additional information on the following page.

Figure 2.2.2-2
Contribution of Source Sectors to Average Annual Nationwide Inhalation Exposure to Air Toxics in 1996



Source: National Scale Air Toxics Assessment.

2.2.2.1 Benzene

Benzene is an aromatic hydrocarbon that is present as a gas in both exhaust and evaporative emissions from mobile sources. Benzene accounts for one to two percent of the exhaust hydrocarbons, expressed as a percentage of total organic gases (TOG), in diesel engines.[198, 199] For gasoline-powered highway vehicles, the benzene fraction of 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 higher than for diesel engines, about three to five percent. The benzene fraction of evaporative emissions from gasoline vehicles depends on control technology and fuel composition and characteristics (e.g., benzene level and the evaporation rate) and is generally about one percent.[200]

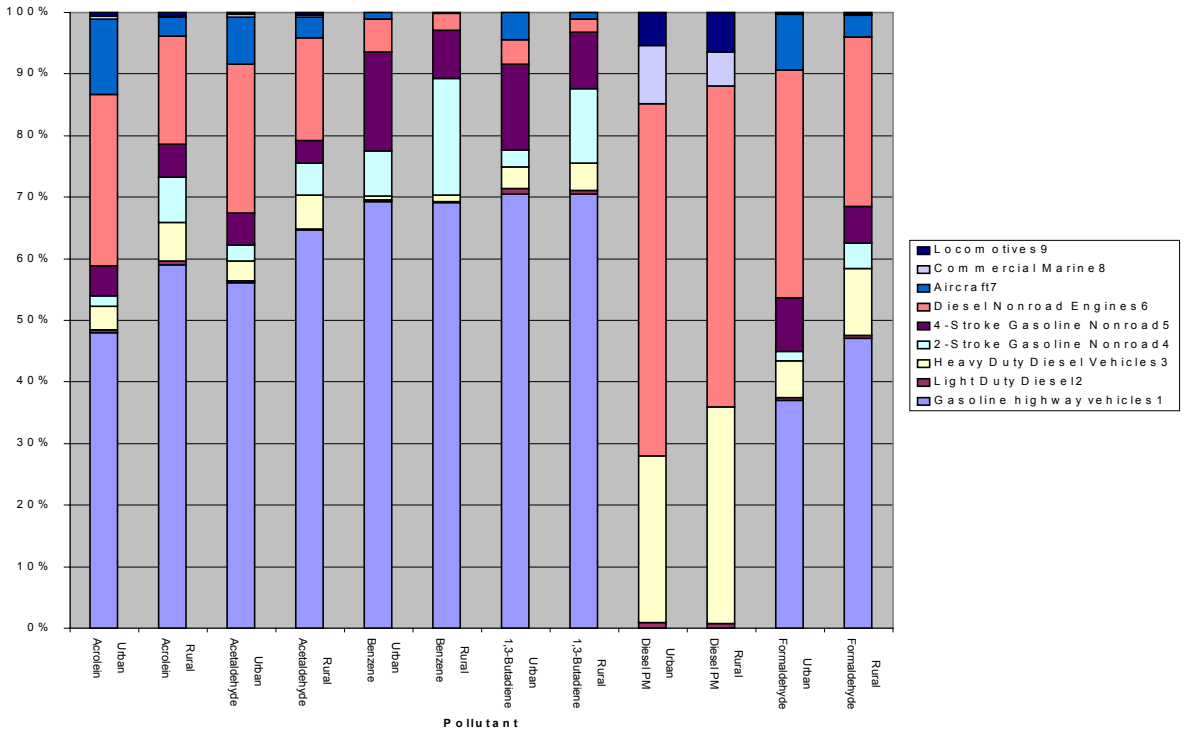
Nonroad engines account for 28 percent of nationwide emissions of benzene with nonroad diesel accounting for about 3 percent in 1996. Mobile sources as a whole account for 78 percent of the total benzene emissions in the nation. Nonroad sources as a whole account for an average of about 17 percent of ambient benzene in urban areas and about 9 percent of ambient benzene in rural areas across the U.S, in the 1996 NATA assessment. Of ambient benzene levels due to mobile sources, 5 percent in urban and 3 percent in rural areas come from nonroad diesel engines (see Figure 2.2.2-3).

The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia) by all routes of exposure.[201] It is associated with additional health effects including chromosomal changes in human and animal cells and increased proliferation of bone marrow cells in mice.[202, 203] A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with long-term occupational exposure to benzene.

Inhalation is the major source of human exposure to benzene in the occupational and non-occupational setting. At least half of this exposure is attributable to gasoline vapors and automotive emissions. Long-term inhalation occupational exposure to benzene has been shown to cause cancer of the hematopoetic (blood cell) system. Among these are acute nonlymphocytic leukemia,[I] chronic lymphocytic leukemia and possibly multiple myeloma

[I]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.

Figure 2.2.2-3
Contribution of Source Sectors to Total Average
Nationwide Mobile Source Ambient Concentrations in 1996



(primary malignant tumors in the bone marrow), although the evidence for the latter has decreased with more recent studies.[204,205] 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 chromosomal changes in humans and animals[206] and increased proliferation of mouse bone marrow cells.[207]

The latest assessment by EPA places the excess risk of developing acute nonlymphocytic leukemia at 2.2×10^{-6} to 7.8×10^{-6} per $\mu g/m^3$. In other words, there is a risk of about two to eight excess leukemia cases in one million people exposed to 1 $\mu g/m^3$ over a lifetime (70 years).[208] This range of unit risks are the maximum likelihood estimate (MLE) calculated from different exposure assumptions and dose-response models that are linear at low doses. It should be noted that not enough information is known to determine the slope of the dose-response curve at environmental levels of exposure and to provide a sound scientific basis to choose any particular extrapolation model to estimate human cancer risk at low doses. In fact, recent data[209] suggest that because genetic abnormalities occur at low exposure in humans, and the formation of toxic metabolites plateaus above 25 ppm (80,000 $\mu g/m^3$), the dose-response curve could be supralinear below 25 ppm. Thus, EPA believes the use of a linear extrapolation model as a default approach is appropriate.

Based on average population exposures in the 1996 NATA Assessment, upper bound cancer risk (using the upper end of the MLE range) from inhalation of benzene from ambient sources is above 10 in a million across the entire United States. These results are best interpreted as upper estimates of risks to typical individuals (provided exposure estimates are not underestimated). Thus most individuals are likely to have risks that are equal to or lower than these estimates, but some individuals may have risks which are higher. EPA projects a median nationwide reduction in ambient concentrations of benzene from mobile sources of about 46 percent between 1996 and 2007, as a result of current and planned control programs based on the analysis referenced earlier examining these pollutants in the 1996 to 2007 time frame based on the analysis of hazardous air pollutants in the 1996 to 2007 time frame referenced earlier.

A number of adverse noncancer health effects, blood disorders such as preleukemia and aplastic anemia, have also been associated with long-term exposure to benzene.[210, 211] People with long-term occupational exposure to benzene have experienced harmful effects on the blood-forming tissues, especially in bone marrow. These effects can disrupt normal blood production and suppress the production of important blood components, such as red and white 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 of blood to clot). Chronic inhalation exposure to benzene in humans and animals results in pancytopenia,[J] a condition characterized by decreased numbers of circulating erythrocytes (red blood cells), leukocytes (white blood cells), and thrombocytes (blood platelets).[212,213] Individuals that develop pancytopenia and have continued exposure to benzene may develop aplastic anemia,[K] whereas others exhibit both pancytopenia and bone marrow hyperplasia (excessive cell formation), a condition that may indicate a preleukemic state.[214, 215] It should be noted that these health effects occur in human and animal studies at concentrations well above those typically found in the ambient environment. The most sensitive noncancer effect observed in humans, based on current data, is the depression of the absolute lymphocyte count in blood.[216] EPA's inhalation reference concentration (RfC, i.e., a chronic exposure level presumed to be "without appreciable risk" for noncancer effects) for

[J]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.

[K]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 leukemias known as preleukemia. The aplastic anemia can progress to AML (acute mylogenous leukemia).

benzene is 30 μg/m^3, based on suppressed absolute lymphocyte counts as seen in humans under occupational exposure conditions.

The average inhalation exposure concentration to benzene from ambient sources in the 1996 NATA assessment is 1.4 μg/m^3, and the 95th percentile exposure concentration is about twice as high (U. S. EPA, 2002). However, the assessment does not account for localized hotspots. In these hot spots, such as in close proximity to roadways, inhalation exposures from ambient sources are likely to be much higher.[217, 218, 219, 220, 221, 222] As mentioned above, nonroad diesel engines are small but significant contributors to the ambient concentrations resulting in these exposures.

2.2.2.2 1,3-Butadiene

1,3-Butadiene is formed in engine exhaust by the incomplete combustion of fuel. It is not present in engine evaporative emissions, because it is not present in any appreciable amount in fuel. 1,3-butadiene accounts for less than one percent of total organic gas exhaust from mobile sources.

Nonroad engines account for 18 percent of nationwide emissions of 1,3-butadiene in 1996 with nonroad diesel accounting for about 1.5 percent based on the NATA, NTI, and supplemental information already discussed in the previous section. Mobile sources account for 63 percent of the total 1,3-butadiene emissions in the nation as a whole. Nonroad sources as a whole account for an average of about 21 percent of ambient butadiene in urban areas and about 13 percent of ambient 1,3-butadiene in rural areas across the United States. Of ambient butadiene levels due to mobile sources, 4 percent in urban and 2 percent in rural areas come from nonroad diesel (see Figure 2.2.2-3).

EPA earlier identified 1,3-butadiene as a probable human carcinogen in its IRIS database.[223] EPA characterized 1,3-butadiene as carcinogenic to humans by inhalation.[224,225,226] The specific mechanisms of 1,3-butadiene-induced carcinogenesis are not fully characterized. However, the data strongly suggest that the carcinogenic effects are mediated by genotoxic metabolites of 1,3-butadiene. Animal data suggest that females may be more sensitive than males for cancer effects; but more data are needed before reaching definitive conclusions on potentially sensitive subpopulations.

The cancer unit risk estimate is 0.08/ppm or 3×10-5 per μg/m3 (based primarily on linear modeling and extrapolation of human data). In other words, it is estimated that approximately 30 persons in one million exposed to 1 μg/m^3 1,3-butadiene continuously for their lifetime (70 years) would develop cancer as a result of this exposure. The human incremental lifetime unit cancer risk (incidence) estimate is based on extrapolation from leukemias observed in an occupational epidemiologic study.[227] This estimate includes a twofold adjustment to the epidemiologic-based unit cancer risk applied to reflect evidence from the rodent bioassays suggesting that the epidemiologic-based estimate may underestimate total cancer risk from 1,3-butadiene exposure in the general population. Based on average population exposure from the 1996 NATA Assessment, upper bound lifetime cancer risk from inhalation of 1,3-butadiene

is above 10 in a million across the entire United States. Most individuals are likely to have risks that are equal to or lower than these estimates, but some individuals may have risks which are higher. EPA projects a median nationwide reduction in ambient concentrations of butadiene from mobile sources of about 46 percent between 1996 and 2007, as a result of current and planned control programs.

1,3-Butadiene also causes a variety of reproductive and developmental effects in mice; no human data on these effects are available. The most sensitive effect was ovarian atrophy observed in a lifetime bioassay of female mice.[228] Based on this critical effect and the benchmark concentration methodology, an RfC was calculated. This RfC for chronic health effects was 0.9 ppb, or about 2 $\mu g/m^3$. The average inhalation exposure from outdoor sources in the 1996 NATA assessment was 0.08 $\mu g/m^3$, with a 95th percentile concentration of 0.2 $\mu g/m^3$ (U. S. EPA, 2002). As is the case with benzene, in some hot spots, such as in close proximity to roadways, inhalation exposures from ambient sources are likely to be much higher. As mentioned above, nonroad diesel engines are small but significant contributors to the ambient concentrations resulting in these exposures.

2.2.2.3 Formaldehyde

Formaldehyde is the most prevalent aldehyde in engine exhaust. It is formed from incomplete combustion of both gasoline and diesel fuel. In a recent test program that measured toxic emissions from several nonroad diesel engines, ranging from 50 to 480 horsepower, formaldehyde consistently accounted for well over 10 percent of total exhaust hydrocarbon emissions.[229] Formaldehyde accounts for far less of total exhaust hydrocarbon emissions from gasoline engines, although the amount can vary substantially by duty cycle, emission control system, and fuel composition. It is not found in evaporative emissions.

Nonroad engines account for 29 percent of nationwide emissions of formaldehyde in 1996, with nonroad diesel accounting for about 22 percent based on the NATA, NTI, and supplemental information already discussed. Mobile sources as a whole account for 56 percent of the total formaldehyde emissions in the nation. Of ambient formaldehyde levels due to mobile sources, 37 percent in urban and 27 percent in rural areas come from nonroad diesel. Nonroad sources as a whole account for an average of about 41 percent of ambient formaldehyde in urban areas and about 10 percent of ambient formaldehyde in rural areas across the U.S, in the 1996 NATA assessment. These figures are for tailpipe emissions of formaldehyde. Formaldehyde in the ambient air comes not only from tailpipe (of direct) emissions but is also formed from photochemical reactions of hydrocarbons. Mobile sources are responsible for well over 50 percent of total formaldehyde including both the direct emissions and photochemically formed formaldehyde in the ambient air, according to the NATA for 1996. EPA projects a median nationwide reduction in ambient concentrations of formaldehyde from mobile sources of about 43 percent between 1996 and 2007, as a result of current and planned control programs (Cook et al., 2002).

EPA has classified formaldehyde as a probable human carcinogen based on limited evidence for carcinogenicity in humans and sufficient evidence of carcinogenicity in animal studies, rats,

mice, hamsters, and monkeys.[230, 231] 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.[232] 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.[233, 234, 235] 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.[236] Research has demonstrated that formaldehyde produces mutagenic activity in cell cultures.[237]

The agency is currently conducting a reassessment of risk from inhalation exposure to formaldehyde based on new information including a study by the CIIT Centers for Health Research.[238, 239] The CIIT information and other recent information, including recently published epidemiological studies, are being reviewed and considered in the reassessment of the formaldehyde unit risk estimate. The epidemiological studies examine the potential for formaldehyde to cause cancer in organs other than those addressed by the CIIT model. We plan to bring this reassessment to the Science Advisory Board in the summer of 2004.

Formaldehyde exposure also causes a range of noncancer health effects. At low concentrations (e.g. 60 – 2500 $\mu g/m^3$), irritation of the eyes (tearing of the eyes and increased blinking) and mucous membranes is the principal effect observed in humans. At exposure to 1200-14,000 $\mu g/m^3$, 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.[240] In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 6200 $\mu g/m^3$.[241] Formaldehyde exposure may also cause bronchial asthma-like symptoms in non-asthmatics.[242, 243]

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

Average inhalation exposure from outdoor sources in the 1996 NATA assessment was 0.9 $\mu g/m^3$, with a 95^{th} percentile concentration of 2.3 $\mu g/m^3$.

2.2.2.4 Acetaldehyde

Acetaldehyde is a saturated aldehyde that is found in engine exhaust and is formed as a result of incomplete combustion of both gasoline and diesel fuel. In a recent test program that measured toxic emissions from several nonroad diesel engines, ranging from 50 to 480 horsepower, acetaldehyde consistently accounted for over 5 percent of total exhaust hydrocarbon emissions (Southwest Research, 2002). Acetaldehyde accounts for far less of total exhaust hydrocarbon emissions from gasoline engines, although the amount can vary substantially by duty cycle, emission control system, and fuel composition. It is not a component of evaporative emissions.

Nonroad engines account for 43 percent of nationwide emissions of acetaldehyde with nonroad diesel accounting for about 34 percent based on the NATA, NTI, and supplemental information. Mobile sources as a whole account for 73 percent of the total acetaldehyde emissions in the nation. Nonroad sources as a whole account for an average of about 36 percent of ambient acetaldehyde in urban areas and about 21 percent of ambient acetaldehyde in rural areas across the U.S, in the 1996 NATA assessment. Of ambient acetaldehyde levels due to mobile sources, 24 percent in urban and 17 percent in rural areas come from nonroad diesel.. Also, acetaldehyde can be formed photochemically in the atmosphere. Counting both direct emissions and photochemically formed acetaldehyde, mobile sources are responsible for the major portion of acetaldehyde in the ambient air according to the NATA for 1996.

Based primarily on nonhuman animal model studies, acetaldehyde is classified by EPA as a probable human carcinogen. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to acetaldehyde causes an increase in the incidence of nasal squamous cell carcinomas (epithelial tissue) and adenocarcinomas (glandular tissue).[244, 245, 246, 247, 248] The upper confidence limit estimate of a lifetime extra cancer risk from continuous acetaldehyde exposure is about 2.2×10^{-6} per $\mu g/m^3$. In other words, it is estimated that about 2 persons in one million exposed to 1 $\mu g/m^3$ acetaldehyde continuously for their lifetime (70 years) would develop cancer as a result of their exposure. The Agency is currently conducting a reassessment of risk from inhalation exposure to acetaldehyde. Based on the current unit risk and average population exposure from the 1996 NATA Assessment, upper bound cancer risk from inhalation of acetaldehyde from ambient sources is above one in a million for more than one hundred million Americans. Most individuals are likely to have risks that are equal to or lower than these estimates, but some individuals may have risks which are higher. EPA projects a median nationwide reduction in ambient concentrations of acetaldehyde from mobile sources of about 36 percent between 1996 and 2007, as a result of current and planned control programs

EPA's IRIS database states that noncancer effects in studies with rats and mice showed acetaldehyde to be moderately toxic by the inhalation, oral, and intravenous routes (EPA, 1988). Similar conclusions have been made by the California Air Resources Board.[249] 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. Long-term exposures should be kept below the reference concentration of 9 $\mu g/m^3$ to avoid appreciable risk of these noncancer health effects (EPA, 1988). The average inhalation exposure from outdoor sources in the 1996 NATA assessment was 0.7 $\mu g/m^3$, with a 95th percentile concentration of 1.8 $\mu g/m^3$ (U. S. EPA, 2002). As is the case with other air toxic compounds emitted by mobile sources, in some hot spots, such as in close proximity to roadways, inhalation exposures are likely to be much higher. As mentioned above, nonroad diesel engines are significant contributors to the ambient concentrations resulting in these exposures.

Acetaldehyde has been associated with lung function decrements in asthmatics. In one study, aerosolized acetaldehyde caused reductions in lung function and bronchoconstriction in asthmatic subjects.[250]

2.2.2.5 Acrolein

In a recent test program that measured toxic emissions from several nonroad diesel engines, ranging from 50 to 480 horsepower, acrolein accounted for about 0.5 to 2 percent of total exhaust hydrocarbon emissions (Southwest Research, 2002). Acrolein accounts for far less of total exhaust hydrocarbon emissions from gasoline engines, although the amount can vary substantially by duty cycle, emission control system, and fuel composition. It is not a component of evaporative emissions.

Nonroad engines account for 25 percent of nationwide emissions of acrolein in 1996 with nonroad diesel accounting for about 17.5 percent based on NATA, NTI, and the supplemental information Mobile sources as a whole account for 43 percent of the total acrolein emissions in the nation. Of ambient acrolein levels due to mobile sources, 28 percent in urban and 18 percent in rural areas come form nonroad diesel according to NATA.

Acrolein is intensely irritating to humans when inhaled, with acute exposure resulting in substantial discomfort and sensory irritancy, mucus hypersecretion, and congestion. These effects have been noted at acrolein levels ranging from 390 $\mu g/m^3$ to 990 $\mu g/m^3$.[251] The intense irritancy of this carbonyl has been demonstrated during controlled tests in human subjects who suffer intolerable eye and nasal mucosal sensory reactions within minutes of exposure.[252] The irritant nature of acrolein provides the basis for the OSHA Permissible Exposure Limit (PEL) for the workplace of 0.1 ppm (230 $\mu g/m^3$) for an 8-hour exposure period. Acrolein has an odor threshold of about 0.16 ppm (370 $\mu g/m^3$),[253] and acute inhalation exposure of humans to 10 ppm (23,000 $\mu g/m^3$) may result in death over a short period of time.[254]

Acrolein is an extremely volatile vapor, and it possesses considerable water solubility.[255] As such, it readily absorbs into airway fluids in the respiratory tract when inhaled. Lesions to the lungs and upper respiratory tract of rats, rabbits, and hamsters exposed to acrolein formed the basis of the reference concentrations for inhalation (RfC) developed in 2003.[256] The RfC of acrolein is 0.02 $\mu g/m^3$. Average population inhalation exposures from the 1996 NATA assessment are between 0.02 $\mu g/m^3$ and 0.2 $\mu g/m^3$. Thus, the hazard quotient (inhalation exposure divided by the RfC) is greater than one for most of the U.S. population, indicating a

potential for adverse noncancer health effects.

The toxicological data base demonstrating the highly irritating nature of this vapor has been consistent regardless of test species. Animal inhalation studies revealed early on that acrolein induces damage throughout the respiratory tract at 0.7 ppm (1600 $\mu g/m^3$) [257] in concordance with data showing similar vapor uptake along isolated upper and lower lung regions of animals.[258] At levels that humans may encounter incidentally, acrolein has been shown to alter breathing mechanics[259, 260] and airway structure in animals[261] as well as to interfere with macrophage function and to alter microbial infectivity.[262, 263, 264] As with many other irritants, acrolein has the potential to induce adaptation to its own irritancy with repeated exposures to low concentrations (1260 $\mu g/m^3$)[265] -- a phenomenon consistent with the apparent human adaptation to the high spikes of acrolein emanating in mainstream smoke from cigarettes.[266] Hence, sensory awareness of exposure to low levels of acrolein may diminish the apparent acute discomfort, while exposure and the potential for longer term impacts persist. Prolonged exposure to acrolein has been shown in animals to have an impact on pulmonary structure and function that can be quantified.[267] Over the range of 0.4 to 4.0 ppm (920 to 9200 $\mu g/m^3$) acrolein, distinct dose-dependent changes in the degree of injury/disease are apparent, which have lung function consequences. There are clear changes in the cell lining of the airways, including mucus cell hyperplasia, as well as changes in the underlying supportive matrix of the airways. These changes parallel changes in airway hyperreactivity (sometimes referred to as "twitchiness"). Such changes are similar to those observed with asthma. The structural changes in the larger airways, likewise, are reminiscent of those associated with chronic exposure to tobacco smoke.

Irritant effects in humans can be seen at levels encountered industrially that are below the odor threshold and thus may be erroneously thought to be safe. Over time, these same occupational levels of exposure in rats appear to alter airway structure and function. As those in the workplace generally do not reflect the more sensitive groups of the public, the potential for persistent, low level exposures eliciting health outcomes among susceptible groups, including asthmatics who have sensitive airways is a concern.[268]

EPA has concluded that the potential for carcinogenicity of acrolein cannot be determined either for oral or inhalation routes of exposure.[269]

2.2.2.6 Polycyclic Organic Matter

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 degrees C. Polycyclic aromatic hydrocarbons (PAHs) are a chemical class that is a subset of POM. POM are naturally occurring substances that are byproducts of the incomplete combustion of fossil fuels and plant and animal biomass (e.g., forest fires). They occur as byproducts from steel and coke productions and waste incineration. They also are a component of diesel PM emissions. As mentioned in Section 2.1.2.1.2, 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. In particular, EPA obtained data on 7 of the POM compounds, which we analyzed separately as a class in the NATA for 1996. Nonroad engines account for only 1 percent of these 7 POM compounds with total mobile sources responsible for only 4 percent of the total; most of the 7 POMs come from area sources. For total POM compounds, mobile sources as a whole are responsible for only 1 percent. The mobile source emission numbers used to derive these

inventories are based only on particulate-phase POM and do not include the semi-volatile phase POM levels. Were those additional POMs included (which is now being done in the NATA for 1999), these inventory numbers would be substantially higher. A study of indoor PAH found that concentrations of indoor PAHs followed the a similar trend as outdoor motor traffic, and that motor vehicle traffic was the largest outdoor source of PAH.[270]

A recent study found that maternal exposures to polycyclic aromatic hydrocarbons (PAHs) in a multiethnic population of pregnant women were associated with adverse birth outcomes, including low birth weight, low birth length, and reduced head circumference.[271]

2.2.2.7 Dioxins

Exposure to dioxins are recognized by several authoritative bodies, including the International Agency for Research on Cancer, the National Institute of Environmental Health Sciences, the Agency for Toxic Substances and Disease Registry, EPA and some State health and environmental agencies, to present a human health hazard for cancer and non-cancer effects. Recent studies have confirmed that very small amounts of dioxins are formed by and emitted from diesel engines (both heavy-duty diesel trucks and nonroad diesel engines). In an inventory for dioxin sources in 1995, such emissions accounted for only about 1 percent of total dioxin emissions. These nonroad rules will have minimal impact on overall dioxin emissions since these are a very small part of total emissions.

2.3 Ozone

This section reviews health and welfare effects of ozone and describes the air quality information that forms the basis of our conclusion that ozone concentrations in many areas across the country face a significant risk of exceeding the ozone standard into the year 2030. Information on air quality was gathered from a variety of sources, including monitored ozone concentrations from 1999-2001, air quality modeling forecasts conducted for this rulemaking and other state and local air quality information.

Ground-level ozone, the main ingredient in smog, is formed by the reaction of volatile organic compounds (VOCs) and nitrogen oxides (NOx) in the atmosphere in the presence of heat and sunlight. These pollutants, often referred to as ozone precursors, are emitted by many types of pollution sources, including highway and nonroad motor vehicles and engines, power plants, chemical plants, refineries, makers of consumer and commercial products, industrial facilities, and smaller "area" sources. VOCs are also emitted by natural sources such as vegetation. Oxides of nitrogen are emitted largely from motor vehicles, off-highway equipment, power plants, and other sources of combustion.

The science of ozone formation, transport, and accumulation is complex. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions involving NOx, VOC, heat, and sunlight. 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. 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.

These complexities also have implications for programs to reduce ozone. For example, relatively small amounts of NOx enable ozone to form rapidly when VOC levels are relatively high, but ozone production is quickly limited by removal of the NOx. Under these conditions, NOx reductions are highly effective in reducing ozone while VOC reductions have little effect. Such conditions are called "NOx-limited." Because the contribution of VOC emissions from biogenic (natural) sources to local ambient ozone concentrations can be significant, even some areas where man-made VOC emissions are relatively low can be NOx-limited.

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

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

Ozone concentrations in an area also can be lowered by the reaction of nitric oxide with ozone, forming nitrogen dioxide (NO_2); as the air moves downwind and the cycle continues, the NO_2 forms additional ozone. The importance of this reaction depends, in part, on the relative concentrations of NOx, VOC, and ozone, all of which change with time and location.

2.3.1 Health Effects of Ozone

Exposure to ambient ozone contributes to a wide range of adverse health effects, which are discussed in detail in the EPA Air Quality Criteria Document for Ozone.[272] Effects include lung function decrements, respiratory symptoms, aggravation of asthma, increased hospital and emergency room visits, increased medication usage, inflammation of the lungs, as well as a variety of other respiratory effects. People who are particularly at risk for high ozone exposures inclue healthy children and adults who are active outdoors. Susceptible subgroups include children, people with respiratory disease, such as asthma, and people with unusual sensitivity to ozone. More information on health effects of ozone is also available at http://www.epa.gov/ttn/naaqs/standards/ozone/s_03_index.html.

Based on a large number of scientific 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. Short-term (1 to3 hours) and prolonged exposures (6 to 8 hours) to higher ambient ozone concentrations have been linked to lung function decrements, respiratory symptoms, increased hospital admissions and emergency room visits for respiratory problems.[273, 274, 275, 276, 277, 278] Repeated exposure to ozone can make people more susceptible to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.[279, 280, 281, 282, 283] It also can cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure, which over time could lead to premature aging of the lungs and/or chronic respiratory illnesses, such as emphysema and chronic bronchitis.[284, 285, 286, 287]

Adults who are outdoors and active during the summer months, such as construction workers and other outdoor workers, also are among those most at risk of elevated exposures.[288] Thus, it may be that children and outdoor workers are most at risk from ozone exposure because they typically are active outside, playing and exercising, during the summer when ozone levels are highest.[289, 290] For example, summer camp studies in the Eastern United States and Southeastern Canada have reported significant reductions in lung function in children who are active outdoors.[291, 292, 293, 294, 295, 296, 297, 298] Further, children are more at risk of experiencing health effects than adults from ozone exposure because their respiratory systems are still developing. These individuals, as well as people with respiratory illnesses such as asthma, especially asthmatic children, can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during prolonged periods of moderate exertion.[299, 300, 301, 302]

The 8-hour NAAQS is based on well-documented science demonstrating that more people are experiencing adverse health effects at lower levels of exertion, over longer periods, and at lower ozone concentrations than addressed by the 1-hour ozone standard.[303] Attaining the 8-hour standard greatly limits ozone exposures of concern for the general population and populations most at risk, including children active outdoors, outdoor workers, and individuals with pre-existing respiratory disease, such as asthma.

There has been new research that suggests additional serious health effects beyond those that had been know when the 8-hour ozone standard was set. Since 1997, over 1,700 new health and welfare studies have been published in peer-reviewed journals.[304] Many of these studies have investigated the impact of ozone exposure on such health effects as changes in lung structure and biochemistry, inflammation of the lungs, exacerbation and causation of asthma, respiratory illness-related school absence, hospital and emergency room visits for asthma and other respiratory causes, and premature mortality. EPA is currently in the process of evaluating these and other studies as part of the ongoing review of the air quality criteria and NAAQS for ozone. A revised Air Quality Criteria Document for Ozone and Other Photochemical Oxidants will be prepared in consultation with the EPA's Clean Air Scientific Advisory Committee (CASAC).

Key new health information falls into four general areas: development of new-onset asthma, hospital admissions for young children, school absence rate, and premature mortality. Examples

of new studies in these areas are briefly discussed below.

Aggravation of existing asthma resulting from short-term ambient ozone exposure was reported prior to the 1997 decision and has been observed in studies published since.[305, 306] More recent studies now suggest a relationship between long-term ambient ozone concentrations and the incidence of new-onset asthma. In particular, such a relationship in adult males (but not in females) was reported by McDonnell et al. (1999).[307] Subsequently, McConnell et al. (2002) reported that incidence of new diagnoses of asthma in children is associated with heavy exercise in communities with high concentrations (i.e., mean 8-hour concentration of 59.6 ppb) of ozone.[308] This relationship was documented in children who played 3 or more sports and was not statistically significant for those children who played one or two sports.[L] The larger effect of high activity sports than low activity sports and an independent effect of time spent outdoors also in the higher ozone communities strengthened the inference that exposure to ozone may modify the effect of sports on the development of asthma in some children.

Previous studies have shown relationships between ozone and hospital admissions in the general population. A new study in Toronto reported a significant relationship between 1-hour maximum ozone concentrations and respiratory hospital admissions in children under two.[309] Given the relative vulnerability of children in this age category, we are particularly concerned about the findings from the literature on ozone and hospital admissions.

Increased respiratory disease that are serious enough to cause school absences has been associated with 1-hour daily maximum and 8-hour average ozone concentrations in studies conducted in Nevada in kindergarten to 6th grade[310] and in Southern California in grades 4 to 6.[311] These studies suggest that higher ambient ozone levels may result in increased school absenteeism.

The ambient air pollutant most clearly associated with premature mortality is PM, with dozens of studies reporting such an association. However, repeated ozone exposure may be a contributing factor for premature mortality, causing an inflammatory response in the lungs that may predispose elderly and other sensitive individuals to become more susceptible to the adverse health effects of other air pollutants, such as PM.[312, 313] Although the findings in the past have been mixed, the findings of three recent analyses suggests that ozone exposure is associated with increased mortality. Although the National Morbidity, Mortality, and Air Pollution Study (NMMAPS) did not find an effect of ozone on total mortality across the full year, Samet et al. (2000), who conducted the NMMAPS study, did report an effect after limiting the analysis to summer when ozone levels are highest.[314] Similarly, Thurston and Ito (1999) have reported associations between ozone and mortality.[315] Toulomi et al., (1997) reported that 1-hour maximum ozone levels were associated with daily numbers of deaths in 4 cities (London, Athens, Barcelona, and Paris), and a quantitatively similar effect was found in a group of 4 additional cities (Amsterdam, Basel, Geneva, and Zurich).[316]

[L] In communities with mean 8-hour ozone concentration of 59.6 ppb, the relative risk of developing asthma in children playing three or more sports was 3.3. (95% CI 1.9 - 5.8) compared with children playing no sports.

As discussed in Section 2.1 with respect to PM studies, the Health Effects Institute (HEI) reported findings by health researchers that have raised concerns about aspects of the statistical methodology used in a number of older time-series studies of short-term exposures to air pollution and health effects.[317]

2.3.2 Attainment and Maintenance of the 1-Hour and 8-Hour Ozone NAAQS

As shown earlier in Figure 2-1, unhealthy ozone concentrations (i.e., those exceeding the 8-hour standard, which is requisite to protect public health with an adequate margin of safety) occur over wide geographic areas, including most of the nation's major population centers. These areas include much of the eastern half of the United States and large areas of California. Nonroad engines contribute a substantial fraction of ozone precursors in metropolitan areas.

Emission reductions from this rule will assist nonattainment and maintenance areas in reaching the standard by each area's respective attainment date and help maintaining the standard in the future. We discuss both the 1-hour and the 8-hour NAAQS, which are based on air quality measurements, called design values and other factors.

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 3 consecutive-year monitoring periods. For example, an 8-hour design value is the fourth highest daily maximum 8-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 (0.08 parts per million (ppm) for the 8-hour), a violation of the 8-hour standard is defined as a design value greater than or equal to 0.085 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. Thus, our analysis may underestimate the number of counties with design values above the level of NAAQS. For the purposes of identifying areas likely to have an ozone problem in the future, we used the 1999-2001 because these data were the most current at the time we performed the modeling (i.e, 2003 data were not yet available). In the recent designations, the 2001-2003 data were used. The 1999-2001, the 2000-2002, and the 2001-2003 sets of design values are listed in the AQ TSD, which is available in the docket to this rule.

A number of States and local areas in their public comments discussed their need for the rule to reduce ozone levels. The California Air Resources Board noted, "Adoption of the proposed regulations outlined in the NPRM by US EPA is necessary for the protection of public health in California to comply with air quality standards." In addition, the South Coast Air Quality Management District (SCAQMD) requested more federal reductions, citing their need: "In 2010, federal sources including non-road engines, ships, trains, aircraft, and 49-state vehicles would contribute to 34% of the NOx emissions in the South Coast Air Basin (Basin). Of this amount,

non-road engines account for 14% or 108 tons per day of NOx in the Basin. ... without aggressive regulations which would achieve substantial reductions by 2010 for non-road engines, as well as other sources under federal jurisdiction, attainment of the federal 1-hour ozone and PM2.5 standards could be seriously jeopardized. ...Where EPA has exclusive or nearly exclusive jurisdiction, EPA must achieve the maximum feasible reductions to enable states to attain federal standards. Therefore, it is incumbent upon EPA to craft its proposed regulation in a manner that would provide maximum emissions benefit in the near term as well as on a long-term basis."

The City of Houston commented that as the largest city with a severe 1-hour ozone nonattainment area and a near-nonattainment area for PM that they had a need for "huge emission reductions from all sectors in the 8-county area to reach attainment... While diesel engines constitute less than 25% of the city's vehicle fleet, they account for over 40 percent of our mobile source emissions and almost 35% of our overall emissions. The non-road portion of our fleet alone produces 26% of our mobile source, and 21% of the city's overall emissions."

Comments from Illinois Lieutenant Governor comments supported the need for reductions in ozone: "Working to relieve the affects of asthma is of particular importance in Illinois where the mortality rate is the highest in the country and is the number one reason for children missing school."

Similarly, New York State Department of Environmental Conservation "strongly supports EPA's proposed rule to control emissions of air pollution from nonroad diesel engines and fuels. We believe that these regulations, when fully implemented, will provide substantial environmental and public health benefits. ..Nonroad diesel equipment is a major source of NOx, SOx and PM emissions and this proposal will help the state of New York attain and maintain the NAAQS for ozone and PM."

2.3.2 Attainment and Maintenance of the 1-Hour and 8-Hour Ozone NAAQS

As shown earlier in Figure 2-1, nonattainment with the ozone NAAQS occur over wide geographic areas, including most of the nation's major population centers. These areas include much of the eastern half of the United States, industrial midwest, and large areas of California. Nonroad diesel engines contribute a substantial fraction of ozone precursors in metropolitan areas.

Emission reductions from this rule will assist nonattainment and maintenance areas in reaching the standard by each area's respective attainment date and help maintaining the standard in the future. We discuss both the 1-hour, an exceedance-based standard, and the 8-hour NAAQS, which is based on air quality measurements, called design values, as well as other factors.

An ozone design value is a calculated ozone concentration that is used in determining whether a monitoring site meets the NAAQS. Because of the way they are defined, design values are determined based on 3 consecutive-year monitoring periods. For example, an 8-hour

ozone design value is the average of the annual fourth highest daily maximum 8-hour average ozone concentrations measured over a three-year period at a given monitor. Determination of whether an area attains the 1-hour NAAQS is based on the number of "exceedances" of the standard over a three year period. 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 (0.08 parts per million (ppm) for the 8-hour), a violation of the 8-hour standard is defined as a design value greater than or equal to 0.085 ppm.

For a county, the design value is the highest design value from among all the monitors with valid design values within that county. A nonattainment area may contain counties both with and without monitors. The highest design value of any county monitor representing the nonattainment area would determine the design value for that nonattainment county. For the purposes of identifying areas likely to have an ozone problem in the future, we performed modeling and used the 1999-2001 air quality data as described below because these data were the most current at the time we performed the modeling (i.e, 2003 data were not yet available). In the 8-hour designations and classifications, we used the 2001-2003 data in addition to considering other factors. The 1999-2001, the 2000-2002, and the 2001-2003 sets of design values are listed in the AQ TSD, which is available in the docket to this rule.

A number of States and local areas in their public comments discussed their need for the rule to reduce ozone levels. For example, the California Air Resources Board noted, "Adoption of the proposed regulations outlined in the NPRM by US EPA is necessary for the protection of public health in California to comply with air quality standards." In addition, the South Coast Air Quality Management District (SCAQMD) requested more federal reductions, citing their need: "In 2010, federal sources including non-road engines, ships, trains, aircraft, and 49-state vehicles would contribute to 34% of the NOx emissions in the South Coast Air Basin (Basin). Of this amount, non-road engines account for 14% or 108 tons per day of NOx in the Basin. ... without aggressive regulations which would achieve substantial reductions by 2010 for non-road engines, as well as other sources under federal jurisdiction, attainment of the federal 1-hour ozone and $PM_{2.5}$ standards could be seriously jeopardized. ...Where EPA has exclusive or nearly exclusive jurisdiction, EPA must achieve the maximum feasible reductions to enable states to attain federal standards. Therefore, it is incumbent upon EPA to craft its proposed regulation in a manner that would provide maximum emissions benefit in the near term as well as on a long-term basis."

The City of Houston commented that as the largest city with a severe 1-hour ozone nonattainment area and a near-nonattainment area for PM that they had a need for "huge emission reductions from all sectors in the 8-county area to reach attainment... While diesel engines constitute less than 25% of the city's vehicle fleet, they account for over 40 percent of our mobile source emissions and almost 35% of our overall emissions. The non-road portion of our fleet alone produces 26% of our mobile source, and 21% of the city's overall emissions."

Comments from Illinois Lieutenant Governor comments supported the need for reductions in ozone: "Working to relieve the effects of asthma is of particular importance in Illinois where the mortality rate is the highest in the country and is the number one reason for children missing school."

Similarly, New York State Department of Environmental Conservation "strongly supports EPA's proposed rule to control emissions of air pollution from nonroad diesel engines and fuels. We believe that these regulations, when fully implemented, will provide substantial environmental and public health benefits. ..Nonroad diesel equipment is a major source of NOx, SOx and PM emissions and this proposal will help the state of New York attain and maintain the NAAQS for ozone and PM."

2.3.2.1 1-Hour Ozone Nonattainment and Maintenance Areas and Concentrations

Currently, there are 110 million people living in 53 1-hour ozone nonattainment areas covering 219 counties.[318] Of these areas, there are one extreme and 13 severe 1-hour ozone nonattainment areas with a total affected population of 74 million as shown in Table 2.3-1. We focus on these classifications of designated areas because the timing of their attainment dates relates to the timing of the new emission standards. Five severe 1-hour ozone nonattainment areas have attainment dates of November 15, 2007. The Los Angeles South Coast Air Basin is designated as an extreme nonattainment area and has a compliance date of November 15, 2010. While all of these areas are expected to be in attainment before the emission reductions from this rule are fully realized, these reductions will be important to assist these areas in achieving the health and welfare protections of the standards and maintaining compliance with air quality standards.

Table 2.3-1
1-Hour Ozone Extreme and Severe Nonattainment Areas

Nonattainment Area	Attainment Date	2000 Population (millions)	2000-2002 Measured Violation?
Los Angeles South Coast Air Basin, CA[a]	November 15, 2010[a]	14.6	Yes
Chicago-Gary-Lake County, IL-IN	November 15, 2007	8.8	Yes
Houston-Galveston-Brazoria, TX	November 15, 2007	4.7	Yes
Milwaukee-Racine, WI	November 15, 2007	1.8	Yes
New York-New Jersey-Long Island, NY-NJ-CT	November 15, 2007	19.2	Yes
Southeast Desert Modified AQMA, CA	November 15, 2007	1.0	Yes
Atlanta, GA	2005	3.7	Yes
Baltimore, MD	2005	0.8	Yes
Baton Rouge, LA	2005	0.6	Yes
Philadelphia-Wilmington-Trenton, PA-NJ-DE-MD	2005	6.3	Yes
Sacramento, CA	2005	2.0	Yes
San Joaquin Valley, CA	2005	3.2	Yes
Ventura County, CA	2005	0.7	No
Washington, DC-MD-VA	2005	4.5	Yes
Total Population	74million		

[a] Extreme 1-Hour nonattainment areas. All other areas are severe nonattainment areas.
Source: US EPA, Air Quality TSD 2004

Many 1-hour ozone nonattainment areas continue to experience exceedances. Approximately 53 million people are living in 73 counties with measured air quality violating the 1-hour NAAQS in 2000-2002.[M] See the AQ TSD for more details about the counties and populations experiencing various levels of measured 1-hour ozone concentrations.

[M] Typically, county design values (and thus exceedances) are consolidated where possible into design values for consolidated metropolitan statistical areas (CMSA) or metropolitan statistical areas (MSA). Accordingly, the design value for a metropolitan area is the highest design value among the included counties, and counties that are not in metropolitan areas would be treated separately. However, for this section, we examined data on a county basis, not consolidating into CMSA or MSA. Designated nonattainment areas may contain more than one county, and some of these counties have experienced recent exceedances, as indicated in the table. Further, the analysis is limited to areas with ozone monitors.

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. Recurrent nonattainment is especially problematic for areas where high population growth rates lead to significant annual increases in vehicle trips and VMT. Moreover, ozone modeling conducted for this rule predicted exceedances in 2020 and 2030 (without additional controls), which adds to the Agency's uncertainty about the prospect of continued attainment for these areas. The reductions from this final rule will help areas attain and maintain the 1-hour standards.

2.3.2.2 8-Hour Ozone Levels: Current Nonattainment and Future Concentrations

EPA has recently designated nonattainment areas for the 8-hour NAAQS by calculating air quality design values (using 2001-2003 measurements) and considering other factors (www.epa.gov/ozonedesignations).

As described above in Section 2.3.1, the 8-hour NAAQS is based on well-documented science demonstrating that more people are experiencing adverse health effects at lower levels of exertion, over longer periods, and at lower ozone concentrations than addressed by the 1-hour ozone standard.[319] The 8-hour standard greatly limits ozone exposures of concern for the general population and sensitive populations. This section describes the current nonattainment with the 8-hour ozone NAAQS and describes our modeling to predict future 8-hour ozone concentrations, which demonstrate a need for reductions in emissions from this final rule.

2.3.2.2.1 Current 8-Hour Ozone Nonattainment

All or part of 474 counties are in nonattainment, as shown in Figure 2-1, for either failing to meet the 8-hour ozone NAAQS or for contributing to poor air quality in a nearby area. About 159 million people live in the 126 areas that do not meet the 8-hour NAAQS. Based upon the measured data from years 2001-2003 and other factors, these areas were recently designated and classified by EPA.). The nonattainment areas covered under subpart 1 will be required to attain the standard no later than 5 years after designation and, in limited circumstances, they may apply for an additional extension of up to 5 years (e.g., 2009 to 2014). The areas classified under subpart 2 have attainment dates ranging from up to 3 years for marginal areas (2007) to up to 20 years for extreme areas (2024). .

Table 2.3-2 presents the areas, their design values for the 8-hour and 1-hour standards and their category or classification. The reductions from this rule will contribute to these areas' overall strategy to attain and maintain the standards.

Table 2.3-2. 8-Hour Ozone Nonattainment Areas

EPA Region	Area Name	Design Value ppb (2001-2003 data) 8-Hr	1-Hr	Category/Classification
2	Albany-Schenectady-Troy, NY	87	115	Subpart 1
5	Allegan Co, MI	97	115	Subpart 1
3	Allentown-Bethlehem-Easton, PA	91	114	Subpart 1
3	Altoona, PA	85	107	Subpart 1
9	Amador and Calaveras, CA(Central Mtn Co)	91	117	Subpart 1
4	Atlanta, GA	91	125	Subpart 2 Marginal
3	Baltimore, MD	103	143	Subpart 2 Moderate
6	Baton Rouge, LA	86	131	Subpart 2 Marginal
6	Beaumont-Port Arthur, TX	91	129	Subpart 2 Marginal
5	Benton Harbor, MI	91	117	Subpart 1
5	Benzie Co, MI	88	116	Subpart 1
3	Berkeley and Jefferson Counties, WV	86	105	EAC Subpart 1
4	Birmingham, AL	87	113	Subpart 1
1	Boston-Lawrence-Worcester (E. MA), MA	95	124	Subpart 2 Moderate
1	Boston-Manchester-Portsmouth(SE),NH*	95	124	Subpart 2 Moderate
2	Buffalo-Niagara Falls, NY	99	116	Subpart 1
5	Canton-Massillon, OH	90	109	Subpart 1
5	Cass Co, MI	93	124	Subpart 2 Moderate
3	Charleston, WV	86	107	Subpart 1
4	Charlotte-Gastonia-Rock Hill, NC-SC	100	129	Subpart 2 Moderate
4	Chattanooga, TN-GA	88	113	Subpart 1
5	Chicago-Gary-Lake County, IL-IN	101	134	Subpart 2 Moderate
9	Chico, CA	89	102	Subpart 1
5,4	Cincinnati-Hamilton, OH-KY-IN	96	118	Subpart 1
4	Clarksville-Hopkinsville, TN-KY	85	99	Subpart 1
3	Clearfield and Indiana Cos, PA	90	106	Subpart 1
5	Cleveland-Akron-Lorain, OH	103	128	Subpart 2 Moderate
4	Columbia, SC	89	108	EAC Subpart 1
5	Columbus, OH	95	117	Subpart 1
6	Dallas-Fort Worth, TX	100	135	Subpart 2 Moderate
5	Dayton-Springfield, OH	90	117	Subpart 1
8	Denver-Boulder-Greeley-Ft Collins-Love., CO	87	114	EAC Subpart 1
5	Detroit-Ann Arbor, MI	97	127	Subpart 2 Moderate
5	Door Co, WI	94	113	Subpart 1
3	Erie, PA	92	114	Subpart 1
2	Essex Co (Whiteface Mtn) NY	91	113	Subpart 1
5	Evansville, IN	85	106	Subpart 1
4	Fayetteville, NC	87	108	EAC Subpart 1
5	Flint, MI	90	103	Subpart 1

EPA Region	Area Name	Design Value ppb (2001-2003 data) 8-Hr	1-Hr	Category/Classification
5	Fort Wayne, IN	88	106	Subpart 1
3	Franklin Co, PA	93	114	Subpart 1
3	Frederick Co, VA	85	106	EAC Subpart 1
3	Fredericksburg, VA*	99	140	Subpart 2 Moderate
5	Grand Rapids, MI	89	110	Subpart 1
1	Greater Connecticut, CT	95	139	Subpart 2 Moderate
5	Greene Co, IN	88	102	Subpart 1
3	Greene Co, PA	89	107	Subpart 1
4	Greensboro-Winston Salem-High Point, NC	93	121	EAC Subpart 2 Moderate
4	Greenville-Spartanburg-Anderson, SC	87	114	EAC Subpart 1
1	Hancock, Knox, Lincoln and Waldo Cos, ME	94	120	Subpart 1
3	Harrisburg-Lebanon-Carlisle, PA	88	111	Subpart 1
4	Haywood and Swain (Great Smoky NP), NC	85	104	Subpart 1
4	Hickory-Morganton-Lenoir, NC	88	105	EAC Subpart 1
6	Houston-Galveston-Brazoria, TX	102	175	Subpart 2 Moderate
3,4	Huntington-Ashland, WV-KY	91	115	Subpart 1
5	Huron Co, MI	87	109	Subpart 1
9	Imperial Co, CA	87	142	Subpart 2 Marginal
5	Indianapolis, IN	96	119	Subpart 1
5	Jackson Co, IN	85	100	Subpart 1
2	Jamestown, NY	94	115	Subpart 1
2	Jefferson Co, NY	97	121	Subpart 2 Moderate
4	Johnson City-Kingsport-Bristol, TN	86	110	EAC Subpart 1
3	Johnstown, PA	87	106	Subpart 1
5	Kalamazoo-Battle Creek, MI	86	102	Subpart 1
3	Kent and Queen Anne's Co, MD	95	122	Subpart 2 Moderate
9	Kern Co (Eastern Kern), CA	98	118	Subpart 1
5	Kewaunee Co, WI	93	110	Subpart 1
4	Knoxville, TN	92	114	Subpart 1
5	La Porte Co, IN	93	135	Subpart 2 Moderate
3	Lancaster, PA	92	124	Subpart 2 Moderate
5	Lansing-East Lansing, MI	86	102	Subpart 1
9	Las Vegas, NV	86	107	Subpart 1
5	Lima, OH	89	108	Subpart 1
9	Los Angeles South Coast Air Basin, CA	131	180	Subpart 2 Severe 17
9	Los Angeles-San Bernardino (W Mojave),CA	106	138	Subpart 2 Moderate
4,5	Louisville, KY-IN	92	120	Subpart 1
4	Macon, GA	86	113	Subpart 1
3	Madison and Page Cos (Shenandoah NP), VA	87	104	Subpart 1
5	Manitowoc Co, WI	90	110	Subpart 1
9	Mariposa and Tuolumne Cos, CA (S. Mtn Cos)	91	113	Subpart 1

EPA Region	Area Name	Design Value ppb (2001-2003 data) 8-Hr	1-Hr	Category/Classification
5	Mason Co, MI		89	114 Subpart 1
4,6	Memphis, TN-AR		92	126 Subpart 2 Moderate
5	Milwaukee-Racine, WI		101	134 Subpart 2 Moderate
5	Muncie, IN	88	104	Subpart 1
4	Murray Co (Chattahoochee Nat Forest), GA		85	103 Subpart 1
5	Muskegon, MI		95	121 Subpart 2 Moderate
4	Nashville, TN	86	107	EAC Subpart 1
9	Nevada Co, CA (Western Portion)		98	116 Subpart 1
2,1	New York-N. N -Long Island,NY-NJ-CT		102	146 Subpart 2 Moderate
3	Norfolk-Virginia Beach-Newport News,VA		90	121 Subpart 2 Marginal
3,5	Parkersburg-Marietta, WV-OH		87	113 Subpart 1
3,2	Philadelphia-Wilmin-Atl.City,PA-NJ-MD-DE		106	133 Subpart 2 Moderate
9	Phoenix-Mesa, AZ		87	111 Subpart 1
3	Pittsburgh-Beaver Valley, PA		94	120 Subpart 1
1	Portland, ME		91	126 Subpart 2 Marginal
2	Poughkeepsie, NY		94	126 Subpart 2 Moderate
1	Providence (All RI), RI		95	130 Subpart 2 Moderate
4	Raleigh-Durham-Chapel Hill, NC		94	118 Subpart 1
3	Reading, PA		91	116 Subpart 1
3	Richmond-Petersburg, VA		94	131 Subpart 2 Moderate
9	Riverside Co, (Coachella Valley), CA		108	133 Subpart 2 Serious
3	Roanoke, VA		85	107 EAC Subpart 1
2	Rochester, NY		88	110 Subpart 1
4	Rocky Mount, NC		89	106 Subpart 1
9	Sacramento Metro, CA		107	143 Subpart 2 Serious
6	San Antonio, TX		89	119 EAC Subpart 1
9	San Diego, CA		93	118 Subpart 1
9	San Francisco Bay Area, CA		86	123 Subpart 2 Marginal
9	San Joaquin Valley, CA		115	151 Subpart 2 Serious
3	Scranton-Wilkes-Barre, PA		86	108 Subpart 1
5	Sheboygan, WI		100	124 Subpart 2 Moderate
5	South Bend-Elkhart, IN		93	116 Subpart 1
1	Springfield (Western MA), MA		94	132 Subpart 2 Moderate
7,5	St Louis, MO-IL		92	122 Subpart 2 Moderate
3	State College, PA		88	109 Subpart 1
5,3	Steubenville-Weirton, OH-WV		86	113 Subpart 1
9	Sutter Co, CA (Sutter Buttes)		88	113 Subpart 1
5	Terre Haute, IN		87	108 Subpart 1
3	Tioga Co, PA		86	102 Subpart 1
5	Toledo, OH		93	112 Subpart 1
9	Ventura Co, CA		95	124 Subpart 2 Moderate

EPA Region	Area Name	Design Value ppb (2001-2003 data) 8-Hr	1-Hr	Category/Classification
3	Washington Co (Hagerstown), MD	86	109	EAC Subpart 1
3	Washington, DC-MD-VA	99	140	Subpart 2 Moderate
3,5	Wheeling, WV-OH	87	111	Subpart 1
3	York, PA	89	114	Subpart 1
5,3	Youngstown-Warren-Sharon, OH-PA	95	118	Subpart 1

Boston-Manchester-Portsmouth(SE),NH has the same classification as Boston-Lawrence-Worcester (E. MA), MA. Fredericksburg, VA has the same classification as Washington, DC-MD-VA.

The level of the 8-hour ozone (O_3) National Ambient Air Quality Standards (NAAQS) is 0.08 parts per million (ppm). The air quality design value for the 8-hour O_3 NAAQS is the 3-year average of the annual 4th highest daily maximum 8-hour average O_3 concentration. The 8-hour O_3 NAAQS is not met when the 8-hour ozone design value is greater than 0.08 ppm (85 parts per billion [ppb] rounds up). Therefore, an area with a design value of 85 ppb does not meet the NAAQS.

An area with a 1-hour design value of 120 ppb or lower is in a Subpart 1 category and must attain the standard by up to 5 years after designation and they may apply for an extension of up to 5 years.

Areas classified under Subpart 2 must attain the standards by the following attainment dates:

- Marginal up to 3 years,
- Moderate up to 6 years,
- Serious up to 9 years,
- Severe up to 15 or 17 years,
- Extreme up to 20 years.

2.3.2.2.2 Risk of Future 8-Hour Ozone Violations

Our air quality modeling shows that there will continue to be a need for reductions in ozone concentrations in the future without additional controls. In this section we describe the air quality modeling including the non-emission inventory inputs. (See Chapter 3.6 summarizes the emission inventory inputs.) We then discuss the results of the modeling for baseline conditions absent additional control of nonroad diesel engines.

We have also used our air quality modeling to estimate the change in future ozone levels that would result from reductions in emissions from nonroad diesel engines. For this propose rule we modeled a preliminary control scenario that illustrates the likely emission reductions. Because of the substantial lead time to prepare the complex air quality modeling analyses, it was necessary to develop a control options early in the proposal process based on our best judgment at that time. Based on public comment and as additional data regarding technical feasibility and other factors became available, our judgment about the controls that are feasible has evolved. Thus, the preliminary control option differs from what we are finalizing, as summarized in Section 3.6 below.[N] It is important to note that these changes would not affect our estimates of the baseline conditions without additional controls from nonroad diesel engines. This final rule would produce nationwide air quality improvements in ozone levels, and we present the modeled improvements in this section. Those interested in greater detail should review the AQ Modeling TSD, which is available in the docket to this rule.

2.3.2.2.3 Ozone Modeling Methodology, Domains and Simulation Periods

In conjunction with this rulemaking, we performed a series of ozone air quality modeling simulations for the Eastern and Western United States using Comprehensive Air Quality Model with Extension (CAMx). The model simulations were performed for five emission scenarios: a 1996 baseline projection, a 2020 baseline projection and a 2020 projection with nonroad controls, a 2030 baseline projection and a 2030 projection with nonroad controls.

The model outputs from the 1996, 2020 and 2030 baselines, combined with current air quality data, were used to identify areas expected to exceed the ozone NAAQS in 2020 and 2030. These areas became candidates for being determined to be residual exceedance areas that will require additional emission reductions to attain and maintain the ozone NAAQS. The impacts of the new emission standards were determined by comparing the model results in the future year control runs against the baseline simulations of the same year. This modeling supports the conclusion that there is a broad set of areas with predicted ozone concentrations at or above 0.085 ppm between 1996 and 2030 in the baseline scenarios without additional emission reductions.

[N]Because of the complexities and non-linear relationships in the air quality modeling, we are not attempting to make any adjustments to the results. Instead, we are presenting the results for the preliminary control option with information about how the emission changes relate to what was modeled.

The air quality modeling performed for this rule was based upon the same modeling system as was used in the EPA's air quality assessment of the Clear Skies legislation with the addition of updated inventory estimates for 1996, 2020 and 2030. Further discussion of this modeling, including evaluations of model performance relative to predicted future air quality, is provided in the AQ Modeling TSD.

CAMx was utilized to estimate base and future-year ozone concentrations over the Eastern and Western United States for the various emission scenarios. CAMx simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone. CAMx is a photochemical grid model that numerically simulates the effects of emissions, advection, diffusion, chemistry, and surface removal processes on pollutant concentrations within a three-dimensional grid. This model is commonly used for purposes of determining attainment/nonattainment 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 AQ Modeling TSD, which is located in the docket for this rule.

The regional ozone analyses used the modeling domains used previously for OTAG and the highway passenger vehicle Tier 2 rulemaking. The Eastern modeling domain encompasses the area from the East coast to mid-Texas and consists of two grids with differing resolutions. The model resolution was 36 km over the outer portions of the domain and 12 km in the inner portion of the grids. The vertical height of the eastern modeling domain is 4,000 meters above ground level with 9 vertical layers. The western modeling domain encompasses the area west of the 99th degree longitude (which runs through North and South Dakota, Nebraska, Kansas, Oklahoma, and Texas) and also consists of two grids with differing resolutions. The vertical height of the western modeling domains is 4,800 meters above ground level with 11 vertical layers. As for the Eastern United States, the model resolution was 36 km over the outer portions of the domain and 12 km in the inner portion of the grids.

The simulation periods modeled by CAMx included several multi-day periods when ambient measurements were representative of ozone episodes over the Eastern and Western United States. A simulation period, or episode, consists of meteorological data characterized over a block of days that are used as inputs to the air quality model. Three multi-day meteorological scenarios during the summer of 1995 were used in the model simulations over the Eastern United States: June 12-24, July 5-15, and August 7-21. Two multi-day meteorological scenarios during the summer of 1996 were used in the model simulations over the Western United States: July 5-15 and July 18-31. In general, these episodes do not represent extreme ozone events but, instead, are generally representative of ozone levels near local design values. Each of the five emission scenarios (1996 base year, 2020 base, 2020 control, 2030 baseline, 2030 control) were simulated for the selected episodes.

The meteorological data required for input into CAMx (wind, temperature, vertical mixing, etc.) were developed by separate meteorological models. For the Eastern United States, the gridded meteorological data for the three historical 1995 episodes were developed using the Regional Atmospheric Modeling System (RAMS), version 3b. This model provided needed data at every grid cell on an hourly basis. For the Western United States, the gridded meteorological data for the two historical 1996 episodes were developed using the Fifth-Generation National Center for Atmospheric Research (NCAR) / Penn State Mesoscale Model (MM5). These

meteorological modeling results were evaluated against observed weather conditions before being input into CAMx and it was concluded that the model fields were adequate representations of the historical meteorology. A more detailed description of the settings and assorted input files employed in these applications is provided in the AQ Modeling TSD, which is located in the docket for this rule.

The modeling assumed background pollutant levels at the top and along the periphery of the domain as in Tier 2. 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 CAMx inputs (land use, photolysis rates, etc.) were developed using procedures employed in the highway light duty Tier 2/OTAG regional modeling. The development of model inputs is discussed in greater detail in the AQ Modeling TSD, which is available in the docket for this rule.

2.3.2.2.4 Model Performance Evaluation

The purpose of the base year photochemical ozone modeling was to reproduce the atmospheric processes resulting in the observed ozone concentrations over these domains and episodes. One of the fundamental assumptions in air quality modeling is that a model that adequately replicates observed pollutant concentrations in the base year can be used to assess the effects of future-year emission controls.

A series of performance statistics was calculated for both model domains, the four quadrants of the eastern domain, and multiple subregions in the eastern and western domains. Table 2.3-2 summarizes the performance statistics. The model performance evaluation consisted solely of comparisons against ambient surface ozone data. There was insufficient data available in terms of ozone precursors or ozone aloft to allow for a more complete assessment of model performance. Three primary statistical metrics were used to assess the overall accuracy of the base year modeling simulations.

- Mean normalized bias is defined as the average difference between the hourly model predictions and observations (paired in space and time) at each monitoring location, normalized by the magnitude of the observations.

- Mean normalized gross error is defined as the average absolute difference between the hourly model predictions and observations (paired in space and time) at each monitoring location, normalized by the magnitude of the observations.

- Average accuracy of the peak is defined as the average difference between peak daily model predictions and observations at each monitoring location, normalized by the magnitude of the observations.

In general, the model tends to underestimate observed ozone, especially in the modeling over the Western United States, as shown in Table 2.3-3. When all hourly observed ozone values greater than a 60 ppb threshold are compared with their model counterparts for the 30 episode modeling days in the eastern domain, the mean normalized bias is -1.1 percent and the mean normalized gross error is 20.5 percent. When the same statistics are calculated for the 19 episode days in the western domain, the bias is -21.4 percent and the error is 26.1 percent.

Table 2.3-3
Model Performance Statistics for the CAMx Ozone Predictions: Base Case

Region	Episode	Average Accuracy of the Peak	Mean Normalized Bias	Mean Normalized Gross Error
Eastern U.S.	June 1995	-7.3	-8.8	19.6
	July 1995	-3.3	-5.0	19.1
	August 1995	9.6	8.6	623.3
Western U.S.	July 1996	-20.5	-21.4	26.1

At present, there are no guidance criteria by which one can determine if a regional ozone modeling exercise is exhibiting adequate model performance. These base case simulations were determined to be acceptable based on comparisons to previously completed model rulemaking analyses (e.g., Ozone Transport Assessment Group (OTAG), the light-duty passenger vehicle Tier-2 standards, and on highway Heavy-Duty Diesel Engine 2007 standards). The modeling completed for this rule exhibits less bias and error than any past regional ozone modeling application done by EPA. Thus, the model is considered appropriate for use in projecting changes in future year ozone concentrations and the resultant health and economic benefits due to the anticipated emission reductions.

2.3.2.2.5 Results of Photochemical Ozone Modeling: Areas at Risk of Future 8-Hour Violations

This section summarizes the results of our modeling of ozone air quality impact in the future of reductions in nonroad diesel emissions. Specifically, it provides information on our calculations of the number of people estimated to live in counties in which ozone monitors are predicted to exceed design values or to be within 10 percent of the design value in the future. We also provide specific information about the number of people who would repeatedly experience levels of ozone of potential concern over prolonged periods, i.e., over 0.085 ppm ozone 8-hour concentrations over a number of days.

The determination that an area is at risk of exceeding the ozone standard in the future was made for all areas with current design values greater than or equal to 0.085 ppm (or within a 10 percent margin) and with modeling evidence that concentrations at and above this level will persist into the future. The following sections provide background on methods for analysis of

attainment and maintenance. Those interested in greater detail should review the AQ TSD and AQ Modeling TSD, both of which are available in the docket to this rule.

The relative reduction factor method was used for interpreting the future-year modeling results to determine where nonattainment is expected to occur in the 2020 and 2030 control cases. The CAMx simulations were completed for base cases in 1996, 2020, and 2030 considering growth and expected emission controls that will affect future air quality. The effects of the nonroad engine reductions (control cases) were modeled for the two future years. As a means of assessing the future levels of air quality with regard to the ozone NAAQS, future-year estimates of ozone design values were calculated based on relative reduction factors (RRF) between the various baselines and 1999-2001 ozone design values. The procedures for determining the RRFs are similar to those in EPA's draft guidance for modeling for an 8-hour ozone standard.[320] Hourly model predictions were processed to determine daily maximum 8-hour concentrations for each grid cell for each non-ramp-up day modeled. The RRF for a monitoring site was determined by first calculating the multi-day mean of the 8-hour daily maximum predictions in the nine grid cells surrounding the site using only those predictions greater than or equal to 70 ppb, as recommended in the guidance.[O, 321] This calculation was performed for the base year scenario and each of the future-year baselines. The RRF for a site is the ratio of the mean prediction in the future-year scenario to the mean prediction in the base year scenario. RRFs were calculated on a site-by-site basis. The future-year design value projections were then calculated by county, based on the highest resultant design values for a site within that county from the RRF application.

Based upon our air quality modeling for this rule, we anticipate that without emission reductions beyond those already required under promulgated regulation and approved SIPs, ozone nonattainment will likely persist into the future. With reductions from programs already in place (but excluding the emission reductions from this rule), the number of counties violating the ozone 8-hour standard is expected to decrease in 2020 to 30 counties where 43 million people are projected to live.[322] Thereafter, exposure to unhealthy levels of ozone is expected to increase again. In 2030 the number of counties violating the ozone 8-hour NAAQS, without considering the emission reductions from this rule, is projected to increase to 32 counties where 47 million people are projected to live.

EPA is still developing the implementation process for bringing the nation's air into attainment with the ozone 8-hour NAAQS (see proposal, 68 FR 32702, June 2, 2003, that was recently finalized www.epa.gov/ozonedesignations) as described above. Since the VOC and NOx emission reductions expected from this final rule will go into effect during the period when areas will need to attain the 8-hour ozone NAAQS, the projected reductions in nonroad diesel emissions are expected to assist States and local agencies in their effort to meet and maintain that standard. Many states mentioned this need in their public comments. The following are sample comments from states and state associations on the proposed rule, which corroborate that this rule is a critical element in States' NAAQS attainment efforts. Fuller information can be found in the Summary and Analysis of Comments.

[O]For the one-hour NAAQS we used a cut-off of 80 ppb. Please see the Highway Passenger Vehicle Tier 2 Air Quality Modeling TSD for more details (EPA 1999b).

- "Unless emissions from nonroad diesels are sharply reduced, it is very likely that many areas of the country will be unable to attain and maintain health-based NAAQS for ozone and PM." (STAPPA/ALAPCO)
- "Adoption of the proposed regulation ... is necessary for the protection of public health in California and to comply with air quality standards." (California Air Resources Board)
- "Attainment of the NAAQS for ozone and $PM_{2.5}$ is of immediate concern to the states in the northeast region....Thus, programs ... such as the proposed rule for nonroad diesel engines are essential." (NESCAUM)

Furthermore, the inventories that underlie the ozone modeling conducted for this rulemaking included emission reductions from all current or committed federal, State, and local controls and, for the control case, including this rulemaking. There was no attempt to examine the prospects of areas attaining or maintaining the ozone standard with possible future controls (i.e., controls beyond current or committed federal, State, and local controls). Tables 2.2-4 and 2.2-5 below should therefore be interpreted as indicating what counties are at risk of ozone violations in 2020 or 2030 without additional federal or State measures that may be adopted and implemented after this rulemaking is finalized. We expect many of the areas listed in Table 2.2-4 to adopt additional emission reduction programs, but we are unable to quantify or rely upon future reductions from additional State programs since they have not yet been adopted.

Since the emission reductions expected from this final rule begin in the same time period in which areas will need reductions to attain by their attainment dates, the projected reductions in nonroad emissions will be extremely important to States in meeting the new NAAQS. In public comment, many States and local agencies commented that they will be relying on such nonroad reductions to help them attain and maintain the 8-hour NAAQS. Furthermore, since the nonroad emission reductions will continue to grow in the years beyond 2014, they will also be important for maintenance of the NAAQS for areas with attainment dates of 2014 and earlier.

On a population-weighted basis, the average change in future year design values would be a decrease of 1.8 ppb in 2020, and 2.5 ppb in 2030. Within nonattainment areas, the population-weighted average decrease would be somewhat higher: 1.9 ppb in 2020 and 3 ppb in 2030.[P] In terms of modeling accuracy, the count of modeled nonattaining counties is much less certain than the average changes in air quality. For example, actions by states to meet their SIP obligations would not be expected to significantly change the overall concentration changes induced by this final rule, but they could substantially change the number of counties in or out of attainment. If state actions resulted in an increase in the number of areas that are very close to, but still above, the NAAQS, then this rule might bring many of those counties down sufficiently to change their attainment status. On the other hand, if state actions brought several counties we project to be very close to the standard in the future down sufficiently to reach attainment status, then the air quality improvements from this rule might change the actual attainment status of very few counties. Bearing this limitation in mind, our modeling indicates that the nonroad diesel emission reductions will decrease the net number of nonattainment counties by 2 in 2020 and by 4 in 2030, without consideration of new state or local programs.

[P]This is in spite of the fact that NOx reductions can at certain times in some areas cause ozone levels to increase. Such "disbenefits" are observed in our modeling, but these results make clear that the overall effect of this final rule is positive.

This air quality modeling suggests that without emission reductions beyond those already required under promulgated regulations and approved SIPs, ozone nonattainment will likely persist into the future. With reductions from programs already in place, the number of counties violating the ozone 8-hour standard is expected to decrease from today's levels to 30 counties in 2020 where 43 million people are projected to live.[323] Thereafter, exposure to unhealthy levels of ozone is expected to begin to increase again. In 2030 the number of counties violating the ozone 8-hour NAAQS is projected to increase to 32 counties where 47 million people are projected to live. In addition, in 2030, 82 counties where 44 million people are projected to live will be within 10 percent of violating the ozone 8-hour NAAQS. Specifically, counties presented in Table 2.3-3 and 2.3-4 have monitored 1999-2001 air quality data[Q] and our modeling predicts violations of the 8-hour ozone NAAQS, or predicts concentrations within 10 percent of the standard, in 2020 or 2030. The base case indicates conditions predicted without the reductions from this rule, and the control case represents a preliminary control option similar to the final rule, as described in section 3.6 of the RIA.

In Table 2.3-4 we list the counties with 2020 and 2030 projected 8-hour ozone design values (4^{th} maximum concentration) that violate the 8-hour standard. Counties are marked with an "V" in the table if their projected design values are greater than or equal to 85 ppb. The 1999-2001 average design values of these counties are also listed. Recall that we project future design values only for counties that have 1999-2001 design values, so this list is limited to those counties with ambient monitoring data sufficient to calculate these design values.

[Q]Since the air quality modeling and analyses performed at proposal used the 1999-2001 monitored data set, we present these data rather than the 2000-2002 data for consistency.

Table 2.3-4: Counties with 2020 and 2030 Projected Ozone Design Values in Violation of the 8-Hour Ozone Standard.[a]

State	County	1999 - 2001 Design Value (ppb)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
CA	Fresno	108	V	V	V	V	799,407
CA	Kern	109	V	V	V	V	661,645
CA	Los Angeles	105	V	V	V	V	9,519,338
CA	Orange	77	V	V	V	V	2,846,289
CA	Riverside	111	V	V	V	V	1,545,387
CA	San Bernardino	129	V	V	V	V	1,709,434
CA	Ventura	101	V	V	V	V	753,197
CT	Fairfield	97	V	V	V	V	882,567
CT	Middlesex	99	V	V	V	V	155,071
CT	New Haven	97	V	V	V	V	824,008
GA	Bibb	98	V		V		153,887
GA	Fulton	107	V	V	V		816,006
GA	Henry	107	V		V		119,341
IL	Cook	88	V	V	V	V	5,376,741
IN	Lake	90			V		484,564
MD	Harford	104	V		V		218,590
MI	Macomb	88			V	V	788,149
MI	Wayne	88	V	V	V	V	2,061,162
NJ	Camden	103	V	V	V	V	508,932
NJ	Gloucester	101	V	V	V	V	254,673
NJ	Hudson	93	V	V	V	V	608,975
NJ	Hunterdon	100	V	V	V	V	121,989
NJ	Mercer	105	V	V	V	V	350,761
NJ	Middlesex	103	V	V	V	V	750,162
NJ	Ocean	109	V	V	V	V	510,916
NY	Bronx	83		V		V	1,332,650
NY	Richmond	98	V	V	V	V	443,728
NY	Westchester	92	V	V	V	V	923,459
PA	Bucks	105	V	V	V	V	597,635
PA	Montgomery	100	V	V	V	V	750,097
TX	Galveston	98	V	V	V	V	250,158
TX	Harris	110	V	V	V	V	3,400,578
WI	Kenosha	95	V	V	V	V	149,577
Number of Violating Counties			30	28	32	28	
Population of Violating Counties[b]			42,930,060	43,532,490	46,998,413	46,038,489	

[a] The projected emission reductions differ based on updated information (see Chapter 3.6); however, the base results presented here would not change, but we anticipate the control case improvements would generally be smaller.

[b] Populations are based on 2020 and 2030 estimates from the U.S. Census.

In Table 2.3-5 we present the counties with 1999-2001 design values and 2020 and 2030 projected 8-hour ozone design values that are within 10 percent of it in either base or control scenarios. Counties are marked with an "X" in the table if their projected design values are greater than or equal to 77 ppb, but less than 85 ppb. Counties are marked with a "V" in the table if their projected design values are greater than or equal to 85 ppb. This list is limited to those counties with ambient monitoring data sufficient to calculate these design values, and the 1999-2001 average design values of these counties are also presented. Most of these are counties are not projected to violate the standard, but their future values are project to be close to the standard. Thus, the final rule will help ensure that these counties continue to meet the standard.

Table 2.3-5
Counties with 2020 and 2030 Projected Ozone Design Values
within Ten Percent of the 8-Hour Ozone Standard.[a]

State	County	1999 - 2001 Design Value (ppb)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
AR	Crittenden	92	X	X	X	X	50,866
AZ	Maricopa	85	X	X	X	X	3,072,149
CA	Kings	98	X	X	X	X	129,461
CA	Merced	101	X	X	X	X	210,554
CA	Tulare	104	X	X	X	X	368,021
CO	Jefferson	81	X	X	X	X	527,056
CT	New London	90	X		X		259,088
DC	Washington	94	X	X	X	X	572,059
DE	New Castle	97	X	X	X	X	500,265
GA	Bibb	98	V	X	V	X	153,887
GA	Coweta	96	X	X	X	X	89,215
GA	De Kalb	102	X	X	X	X	665,865
GA	Douglas	98	X		X		92,174
GA	Fayette	99	X		X		91,263
GA	Fulton	107	V	V	V	X	816,006
GA	Henry	107	V	X	V	X	119,341
GA	Rockdale	104	X	X	X	X	70,111
IL	McHenry	83	X		X		260,077
IN	Lake	90	X	X	V	X	484,564
IN	Porter	90	X	X	X	X	146,798
LA	Ascension	86	X	X	X	X	76,627
LA	Bossier	90	X	X	X	X	98,310
LA	Calcasieu	86	X	X	X	X	183,577
LA	East Baton Rouge	91	X	X	X	X	412,852
LA	Iberville	86	X		X		33,320
LA	Jefferson	89	X	X	X	X	455,466

State	County	1999 - 2001 Design Value (ppb)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
LA	Livingston	88	X	X	X	X	91,814
LA	St Charles	86	X	X	X	X	48,072
LA	St James	83			X		21,216
LA	St John The Ba	86	X	X	X	X	43,044
LA	West Baton Rou	88	X	X	X	X	21,601
MA	Barnstable	96	X		X		222,230
MA	Bristol	93	X		X		534,678
MD	Anne Arundel	103	X	X	X	X	489,656
MD	Baltimore	93	X	X	X	X	754,292
MD	Cecil	106	X	X	X	X	85,951
MD	Harford	104	V	X	V	X	218,590
MD	Kent	100	X		X		19,197
MD	Prince Georges	97	X	X	X		801,515
MI	Benzie	89	X		X		15,998
MI	Macomb	88	X	X	V	V	788,149
MI	Mason	91	X		X		28,274
MI	Muskegon	92	X	X	X		170,200
MI	Oakland	84	X	X	X	X	1,194,156
MI	St Clair	85			X		164,235
MO	St Charles	90			X		283,883
MO	St Louis	88			X		1,016,315
MS	Hancock	87	X		X		42,967
MS	Harrison	89	X	X	X	X	189,601
MS	Jackson	87	X	X	X	X	131,420
NJ	Cumberland	97	X		X		146,438
NJ	Monmouth	94	X	X	X	X	615,301
NJ	Morris	97	X	X	X	X	470,212
NJ	Passaic	89	X	X	X	X	489,049
NY	Bronx	83	X	V	X	V	1,332,650
NY	Erie	92	X	X	X	X	950,265
NY	Niagara	87	X		X		219,846
NY	Putnam	89	X		X		95,745
NY	Suffolk	91	X	X	X	X	1,419,369
OH	Geauga	93	X		X		90,895
OH	Lake	91	X		X		227,511
PA	Allegheny	92	X		X		1,281,666
PA	Delaware	94	X	X	X	X	550,864
PA	Lancaster	96	X		X		470,658
PA	Lehigh	96	X	X	X	X	312,090
PA	Northampton	97	X	X	X	X	267,066

State	County	1999 - 2001 Design Value (ppb)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
PA	Philadelphia	88	X	X	X	X	1,517,550
RI	Kent	94	X	X	X		167,090
RI	Washington	92	X		X		123,546
TN	Shelby	93	X	X	X	X	897,472
TX	Brazoria	91	X	X	X	X	241,767
TX	Collin	99	X	X	X	X	491,675
TX	Dallas	93	X	X	X	X	2,218,899
TX	Denton	101	X	X	X	X	432,976
TX	Jefferson	85	X	X	X	X	252,051
TX	Montgomery	91	X		X	X	293,768
TX	Tarrant	97	X	X	X	X	1,446,219
VA	Alexandria City	88			X		128,283
VA	Arlington	92	X	X	X	X	189,453
VA	Fairfax	95	X	X	X	X	969,749
WI	Door	93	X	X	X	X	27,961
WI	Kewaunee	89	X		X		20,187
WI	Manitowoc	92	X	X	X		82,887
WI	Milwaukee	89	X	X	X	X	940,164
WI	Ozaukee	95	X	X	X	X	82,317
WI	Racine	87	X		X		188,831
WI	Sheboygan	95	X	X	X	X	112,646
WI	Waukesha	86	X		X		360,767
Number of Counties within 10%			79	58	82	54	
Population of Counties within 10%[b]			40,465,492	33,888,031	44,013,587	35,631,215	

[a] The projected emission reductions differ based on updated information (see Section 3.6); however, the base results presented here would not change, but we anticipate the control case improvements would generally be smaller.
[b] Populations are based on 2020 and 2030 estimates from the U.S. Census.

Based on our modeling, we are also able to provide a quantitative prediction of the number of people anticipated to reside in counties in which ozone concentrations are predicted to for 8-hour periods in the range of 85 to 120 ppb and higher on multiple days. Our analysis relies on projected county-level population from the U.S. Department of Census for the period representing each year analyzed.[324]

For each of the counties analyzed, we determined the number of days for periods on which the highest model-adjusted 8-hour concentration at any monitor in the county was predicted, for example, to be equal to or above 85 ppb. We then grouped the counties that had days with ozone in this range according to the number of days this was predicted to happen and summed their projected populations.

In the base case (i.e., before the application of emission reductions resulting from this rule), we estimated in 2020 that 53 million people are predicted to live in counties with at least 2 days with 8-hour average concentrations of 85 ppb or higher. This baseline will increase in 2030 to 56 million people are predicted to live in counties with at least 2 days with 8-hour average concentrations of 85 ppb or higher. About 30 million people live in counties with at least 7 days of 8-hour ozone concentrations at or above 85 ppb in 2020 and 2030 without additional controls. Approximately 15 million people are predicted to live in counties with at least 20 days of 8-hour ozone concentrations at or above 85 ppb in 2020 and 2030 without additional controls.[325] Thus, reductions in ozone precursors from nonroad diesel engines are needed to assist States in meeting the ozone NAAQS and to reduce ozone exposures.

2.3.2.3 Potentially Counterproductive Impacts on Ozone Concentrations from NOx Emission Reductions

While this final rule will reduce ozone levels generally and provide significant ozone-related health benefits, this is not always the case at the local level. Due to the complex photochemistry of ozone production, NOx emissions lead to both the formation and destruction of ozone, depending on the relative quantities of NOx, VOC, and ozone catalysts such as the OH and HO_2 radicals. In areas dominated by fresh emissions of NOx, ozone catalysts are removed via the production of nitric acid, which slows the ozone formation rate. Because NOx is generally depleted more rapidly than VOC, this effect is usually short-lived and the emitted NOx can lead to ozone formation later and further downwind. The terms "NOx disbenefits" or "ozone disbenefits" refer to the ozone increases that can result from NOx emission reductions in these localized areas. According to the NARSTO Ozone Assessment, these disbenefits are generally limited to small regions within specific urban cores and are surrounded by larger regions in which NOx control is beneficial.[326]

In the context of ozone disbenefits, some have postulated that present-day weekend conditions serve as a demonstration of the effects of future NOx reduction strategies because NOx emissions decrease more than VOC emissions on weekends, due to a disproportionate decrease in the activity of heavy-duty diesel trucks and other diesel equipment. Recent research indicates that ambient ozone levels are higher in some metropolitan areas on weekends than weekdays.[327, 328] There are other hypotheses for the cause of the "weekend effect."[329] For instance, the role of ozone and ozone precursor carryover from previous days is difficult to evaluate because of limited ambient data, especially aloft. The role of the changed timing of emissions is difficult to evaluate because of limited ambient and emission inventory information. It is also important to note that in many areas with "weekend effects" (e.g., Los Angeles and San Francisco) significant ozone reductions have been observed over the past 20 years for all days of the week, during a period in which both NOx and VOC emissions have been greatly reduced.

We received some public comments that in some cities, decreased motor vehicle traffic (particularly diesels) results in a higher VOC/NOx ratio which, in airsheds that are VOC-limited, can result in higher ozone concentrations. EPA's air quality modeling predicts NOx disbenefits in the areas identified by some studies as "VOC-limited" (e.g., Los Angeles). However, these

areas represent a small minority of the area in the United States. While some empirical studies to date point to a weekend ozone effect related to NOx reduction, modeling conducted for this rule predicts that this rule will result in net gains in benefits as a result of reduced ozone and $PM_{2.5}$ related to NOx.

EPA maintains that the best available approach for determining the value of a particular emission reduction strategy is the net air quality change projected to result from the rule, evaluated on a nationwide basis and for all pollutants that are health and/or welfare concerns. The primary tool for assessing the net impacts of this rule are the air quality simulation models.[330] Model scenarios of 2020 and 2030 with and without the emission controls from this rulemaking are compared to determine the expected changes in future pollutant levels resulting from the rule. There are several factors related to the air quality modeling and inputs that should be considered regarding the disbenefit issue. First, our future year modeling does not contain any local governmental actions beyond the controls in this rule. It is possible that significant local controls of VOC and/or NOx could modify the conclusions regarding ozone changes in some areas. Second, the modeled NOx reductions are greater than those actually included in the analysis to quantify the emission reductions resulting from the final rule (see Section 3.6 for more detail). This could lead to an exaggeration of the benefits and disbenefits expected to result from the rule. Also, recent work by California ARB has indicated that model limitations and uncertainties may lead to overestimates of ozone disbenefits attributed to NOx emission reductions. While EPA maintains that the air quality simulations conducted for the rule represent state-of-the-science analyses, any changes to the underlying chemical mechanisms, grid resolution, and emissions/meteorological inputs could result in revised conclusions regarding the strength and frequency of ozone disbenefits.

A wide variety of ozone metrics were considered in assessing the emission reductions. Three of the most important assessments are: 1) the effect of the rule on projected future-year ozone violations, 2) the effect of the rule in assisting local areas in attainment and maintenance of the NAAQS, and 3) an economic assessment of the rule benefits based on existing health studies. Additional metrics for assessing the air quality effects are discussed in the TSD for the modeling.

Based only on the reductions from this rule, our modeling predicts that periodic ozone disbenefits will occur most frequently in New York City, Los Angeles, and Chicago. Smaller and less frequent disbenefits also occur in Boston, Detroit, and San Francisco. As described below, despite these localized increases, the net ozone impact of the rule nationally is positive for the majority of the analysis metrics. Even within the few metropolitan areas that experience periodic ozone increases, these disbenefits are infrequent relative to the benefits accrued at ozone levels above the NAAQS. Furthermore, and most importantly, the overall air quality impact of this final rule is projected to be strongly positive due to the expected reductions in fine PM.

The projected net impact of the rule on 8-hour ozone violations in 2020 is that three counties will no longer violate the NAAQS.[331] Conversely, one county in the NewYork City CMSA (Bronx County), which is currently not in violation of the NAAQS, is projected to violate the

standard in 2020 as a result of the rule. The net effect is a projected 1.4 percent increase in the population living in violating counties. It is important to note that ozone nonattainment designations are historically based on larger geographical areas than counties (e.g., see public comments from New York Department of Environmental Conservation requesting that EPA use metropolitan areas instead of counties for its analyses for this reason). Bronx County, NY is the only county within the New York City CMSA in which increases are detected in 8-hour violations in 2020. Considering a larger area, the modeling indicates that projected violations over the entire New York City CMSA will be reduced by 6.8 percent. Upon full turnover of the fleet in 2030, the net impact of the rule on projected 8-hour ozone violations is a 2.0 percent decrease in the population living in violating counties as two additional counties are no longer projected to violate the NAAQS. The net impact of the rule on projected 1-hour ozone violations is to eradicate projected violations from four counties (in both 2020 and 2030), resulting in a 10.5 percent decrease in the population living in violating counties.

Another way to assess the air quality impact of the rule is to calculate its effect on all projected future year design values concentrations, as opposed to just those that cross the threshold of the NAAQS. This metric helps assess the degree to which the rule will assist local areas in attaining and/or maintaining the NAAQS. Future year design values were calculated for every location for which complete ambient monitoring data existed for the period 1999-2001. These present-day design values were then projected by using the modeling projections (future base vs. future control) in a relative sense. For the 1999-2001 monitoring period, there were sites in 522 counties for which 8-hour design values could be calculated and sites in 510 counties for which 1-hour design values could be calculated.

Table 2.3.2-1 shows the average change in future year eight-hour and one-hour ozone design values. Average changes are shown 1) for all counties with design values in 2001, 2) for counties with design values that did not meet the standard in 1999-2001 ("violating" counties), and 3) for counties that met the standard, but were within 10 percent of it in 1999-2001. This last category is intended to reflect counties that meet the standard, but will likely benefit from help in maintaining that status in the face of growth. The average and population-weighted average over all counties in Table 2.3.2-1 demonstrates a broad improvement in ozone air quality. The average across violating counties shows that the rule will help bring these counties into attainment. The average over counties within ten percent of the standard shows that the rule will also help those counties to maintain the standard. All of these metrics show a decrease in 2020 and a larger decrease in 2030 (due to fleet turnover), indicating in four different ways the overall improvement in ozone air quality as measured by attainment of the NAAQS.

Table 2.3.2-1
Average Change in Projected Future-Year Ozone Design Value[f]

Design Value	Average[a]	Number of Counties	2020 Control[f] minus Base (ppb)	2030 Control[f] minus Base (ppb)
8-Hour	All	522	-1.8	-2.8
	All, population-weighted	522	-1.6	-2.6
	Violating counties[b]	289	-1.9	-3
	Counties within 10 percent of the standard[c]	130	-1.7	-2.6
1-Hour	All	510	-2.4	-3.8
	All, population-weighted	510	-2.3	-3.6
	Violating counties[d]	73	-2.9	-4.5
	Counties within 10 percent of the standard[e]	130	-2.4	-3.8

[a] Averages are over counties with 2001 design values.
[b] Counties whose present-day design values exceeded the 8-hour standard (≥ 85 ppb).
[c] Counties whose present-day design values were less than but within 10 percent of the 8-hour standard (77≤DV<85 ppb).
[d] Counties whose present-day design values exceeded the 1-hour standard (≥ 125 ppb).
[e] Counties whose present-day design values were less than but within 10 percent of the 1-hour standard (112≤DV<125 ppb) in 2001.
[f] The analysis in Chapter 3 differs based on updated information; however, we believe that the net results would approximate future emissions, although we anticipate the design value improvements would generally be slightly smaller.

Table 2.3.2-2 presents counts of the same set of counties (those with 1999-2001 design values) examined by the size and direction of their change in design value in 2020 and 2030. For the 8-hour design value, 96 percent of counties show a decrease in 2020, 97 percent in 2030. For the 1-hour design value, 97 percent of counties show a decrease in 2020, 98 percent in 2030.

Table 2.3.2-2
Numbers of Counties Projected to Be in
Different Design-Value Change Bins in 2020 and 2030 as a Result of the Rule[a]

Design value change	2020		2030	
	8-Hour	1-Hour	8-Hour	1-Hour
≥ 2ppb increase	1	1	1	1
1 ppb increase	1	5	3	2
No change	21	10	10	5
1 ppb decrease	140	69	42	22
2-3 ppb decrease	357	356	333	193
4 ppb decrease	2	69	133	287
Total	522	510	522	510

[a] The analysis in Chapter 3 differs based on updated information; however, we believe that the net results would approximate future emissions, although we anticipate the design value improvements would generally be slightly smaller.

A third way to assess the impacts of the rule is an economic consideration of the economic benefits. Benefits related to changes in ambient ozone are expected to be positive for the nation as a whole. However, for certain health endpoints associated with longer ozone-averaging times, such as minor restricted activity days related to 24-hour average ozone, the national impact may be small or even negative. This is due to the forecasted increases in ozone for certain hours of the day in some urban areas. Many of the increases occur during hours when baseline ozone levels are low, but the benefits estimates rely on the changes in ozone along the full distribution of baseline ozone levels, rather than changes occurring only above a particular threshold. As such, the benefits estimates are more sensitive to increases in ozone occurring due to the "NOx disbenefits" effect described above. For more details on the economic effects of the rule, please see Chapter 9: Public Health and Welfare Benefits.

Historically, NOx reductions have been very successful at reducing regional and national ozone levels. Consistent with that fact, the photochemical modeling completed for this rule indicates that the projected emission reductions will significantly assist in the attainment and maintenance of the ozone NAAQS at the national level. Furthermore, NOx reductions also result in reductions in PM and its associated health and welfare effects. This rule is one aspect of overall emission reductions that States, local governments, and Tribes need to reach their clean air goals. It is expected that future state, local and national controls that decrease VOC, CO, and regional ozone will mitigate any localized disbenefits. EPA will continue to rely on local attainment measures to ensure that the NAAQS are not violated in the future. Many organizations with an interest in improved air quality have supported the rule because they believe the resulting NOx reductions will reduce both ozone and PM.[332] EPA believes that a

balanced air quality management approach that includes NOx emission reductions from nonroad engines is needed as part of the nation's progress toward clean air.

2.3.3 Welfare Effects Associated with Ozone and its Precursors

There are a number of significant welfare effects associated with the presence of ozone and NO_X in the ambient air.[333] Because this rule will reduce ground-level ozone and nitrogen deposition, benefits are expected to accrue to the welfare effects categories described in the following paragraphs.

2.3.3.1 Ozone-related welfare effects.

The Ozone Criteria Document notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant."[334] Like carbon dioxide (CO_2) and other gaseous substances, ozone enters plant tissues primarily through apertures (stomata) in leaves in a process called "uptake". To a lesser extent, ozone can also diffuse directly through surface layers to the plant's interior.[335] Once ozone, a highly reactive substance, reaches the interior of plant cells, it inhibits or damages essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns.[336, 337] This damage is commonly manifested as visible foliar injury such as chlorotic or necrotic spots, increased leaf senescence (accelerated leaf aging) and/or as reduced photosynthesis. All these effects reduce a plant's capacity to form carbohydrates, which are the primary form of energy used by plants.[338] With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, more susceptible to disease, insect attack, harsh weather (e.g., drought, frost) and other environmental stresses (e.g., increasing CO_2 concentrations). Furthermore, there is considerable evidence that ozone can interfere with the formation of mycorrhiza, essential symbiotic fungi associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont.[339]

Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of O_3 uptake through closure of stomata).[340, 341, 342] Other resistance mechanisms may involve the intercellular production of detoxifying substances. Several biochemical substances capable of detoxifying ozone have been reported to occur in plants including the antioxidants ascorbate and glutathione. After injuries have occurred, plants may be capable of repairing the damage to a limited extent.[343] Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and response to ozone, it

is not possible to identify threshold values above which ozone is toxic for all plants. However, in general, the science suggests that ozone concentrations of 0.10 ppm or greater can be phytotoxic to a large number of plant species, and can produce acute foliar injury responses, crop yield loss and reduced biomass production. Ozone concentrations below 0.10 ppm (0.05 to 0.09 ppm) can produce these effects in more sensitive plant species, and have the potential over a longer duration of creating chronic stress on vegetation that can lead to effects of concern associated with reduced carbohydrate production and decreased plant vigor.

The economic value of some welfare losses due to ozone can be calculated, such as crop yield loss from both reduced seed production (e.g., soybean) and visible injury to some leaf crops (e.g., lettuce, spinach, tobacco) and visible injury to ornamental plants (i.e., grass, flowers, shrubs), while other types of welfare loss may not be fully quantifiable in economic terms (e.g., reduced aesthetic value of trees growing in Class I areas).

Forests and Ecosystems. Ozone also has been shown conclusively to cause discernible injury to forest trees.[344, 345] In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts.[346] Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function.[347, 348, 349]

Because plants are at the center of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone damages at the community and ecosystem-level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors.[350] In most instances, responses to chronic or recurrent exposure are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems.[351, 352, 353] It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States.

Given the scientific information establishing that ambient ozone levels cause visible injury to foliage of some sensitive forest species,[354] there is a corresponding loss of public welfare from reduced aesthetic properties of forests.[355] However, present analytic tools and resources preclude EPA from quantifying the benefits of improved forest aesthetics.

Agriculture. Laboratory and field experiments have shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the Unites States."[356] In addition, economic studies have shown a relationship between observed ozone levels and crop yields.[357 358 359]

Urban Ornamentals. Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to impact large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative analysis has been conducted. It is estimated that more than $20 billion (1990 dollars) are spent annually on landscaping using ornamentals, both by private property owners/tenants and by governmental units responsible for public areas.[360] This is therefore a potentially important environmental effect. However, methods are not available to allow for plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

2.3.3.2 Nitrogen (NO_X)-related welfare effects.

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

Forests and Ecosystems. Information on the effects of changes in passive nitrogen deposition on forests and other terrestrial ecosystems is very limited. The multiplicity of factors affecting forests, including other potential stressors such as ozone, and limiting factors such as moisture and other nutrients, confound assessments of marginal changes in any one stressor or nutrient in forest ecosystems. However, reductions in nitrogen deposition can have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor.[361]

On the other hand, there is evidence that forest ecosystems in some areas of the United States are already or are becoming nitrogen saturated.[362] Once saturation is reached, adverse effects of additional nitrogen begin to occur, such as soil acidification, which can lead to leaching of nutrients needed for plant growth and mobilization of harmful elements such as aluminum, leading to reductions in tree growth or forest decline. Increased soil acidification is also linked to higher amounts of acidic runoff to streams and lakes and leaching of harmful elements into aquatic ecosystems, harming fish and other aquatic life.[363]

The reductions in ground-level ozone and nitrogen deposition that will result from this rule are expected to reduce the adverse impacts described above. In particular, it is expected that

economic impacts, such as those related to reduced crop yields and forest productivity, will be reduced.

2.4 Carbon Monoxide

This final rule will reduce levels of other pollutants for which NAAQS have been established: carbon monoxide (CO), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2). Currently every area in the United States has been designated to be in attainment with the NO_2 NAAQS. As of August 27, 2003, there were 24 areas designated as nonattainment with the SO2 standard, and 11 designated CO nonattainment areas. The rest of this section describes issues related to CO.

2.4.1 General Background

Unlike many gases, CO is odorless, colorless, tasteless, and nonirritating. Carbon monoxide results from incomplete combustion of fuel and is emitted directly from vehicle tailpipes. Incomplete combustion is most likely to occur at low air-to-fuel ratios in the engine. These conditions are common during vehicle starting when air supply is restricted ("choked"), when vehicles are not tuned properly, and at high altitude, where "thin" air effectively reduces the amount of oxygen available for combustion (except in engines that are designed or adjusted to compensate for altitude). High concentrations of CO generally occur in areas with elevated mobile-source emissions. Carbon monoxide emissions increase dramatically in cold weather. This is because engines need more fuel to start at cold temperatures and because some emission control devices (such as oxygen sensors and catalytic converters) operate less efficiently when they are cold. Also, nighttime inversion conditions are more frequent in the colder months of the year. This is due to the enhanced stability in the atmospheric boundary layer, which inhibits vertical mixing of emissions from the surface.

As described in Chapter 3, nonroad diesel engines currently account for about one percent of the national mobile source CO inventory. EPA previously determined that the category of nonroad diesel engines cause or contribute to ambient CO and ozone in more than one nonattainment area (65 FR 76790, December 7, 2000). In that action, EPA found that engines subject to this final rule contribute to CO nonattainment in areas such as Los Angeles, Phoenix, Spokane, Anchorage, and Las Vegas. Nonroad land-based diesel engines emitted 1,004,600 tons of CO in 1996 (1 percent of mobile source CO). Thus, nonroad diesel engines contribute to CO nonattainment in more than one of these areas.

Although nonroad diesel engines have relatively low per-engine CO emissions, they can be a significant source of ambient CO levels in CO nonattainment areas. Thus, the emission benefits from this final rule will help areas to attain and maintain the CO NAAQS.

2.4.2 Health Effects of CO

Carbon monoxide enters the bloodstream through the lungs and forms carboxyhemoglobin (COHb), a compound that inhibits the blood's capacity to carry oxygen to organs and tissues.[364, 365] Carbon monoxide has long been known to have substantial adverse effects on human health, including toxic effects on blood and tissues, and effects on organ functions. Although there are effective compensatory increases in blood flow to the brain, at some concentrations of COHb, somewhere above 20 percent, these compensations fail to maintain sufficient oxygen delivery, and metabolism declines.[366] The subsequent hypoxia in brain tissue then produces behavioral effects, including decrements in continuous performance and reaction time.[367]

Carbon monoxide has been linked to increased risk for people with heart disease, reduced visual perception, cognitive functions and aerobic capacity, and possible fetal effects.[368] Persons with heart disease are especially sensitive to carbon monoxide poisoning and may experience chest pain if they breathe the gas while exercising.[369] Infants, elderly persons, and individuals with respiratory diseases are also particularly sensitive. Carbon monoxide can affect healthy individuals, impairing exercise capacity, visual perception, manual dexterity, learning functions, and ability to perform complex tasks.[370]

Several recent epidemiological studies have shown a link between CO and premature morbidity (including angina, congestive heart failure, and other cardiovascular diseases. Several studies in the United States and Canada have also reported an association of ambient CO exposures with frequency of cardiovascular hospital admissions, especially for congestive heart failure (CHF). An association of ambient CO exposure with mortality has also been reported in epidemiological studies, though not as consistently or specifically as with CHF admissions. EPA reviewed these studies as part of the Criteria Document review process.[371]

2.4.3 CO Nonattainment

The current primary NAAQS for CO are 35 parts per million for the one-hour average and 9 parts per million for the eight-hour average. These values are not to be exceeded more than once per year. Air quality carbon monoxide value is estimated using EPA guidance for calculating design values. Over 19 million people currently live in the 11 nonattainment areas for the CO NAAQS.

Nationally, significant progress has been made over the last decade to reduce CO emissions and ambient CO concentrations. Total CO emissions from all sources have decreased 16 percent from 1989 to 1998, and ambient CO concentrations decreased by 39 percent. During that time, while the mobile source CO contribution of the inventory remained steady at about 77 percent, the highway portion decreased from 62 percent of total CO emissions to 56 percent while the nonroad portion increased from 17 percent to 22 percent.[372] Over the next decade, we expect there to be a minor decreasing trend from the highway segment due primarily to the more stringent standards for certain light-duty trucks and gasoline nonroad engines.[373] CO standards

for passenger cars and other light-duty trucks and heavy-duty vehicles did not change as a result of other recent rulemakings.

As noted above, CO has been linked to numerous health effects; however, we are unable to quantify the CO-related health or environmental effects of the Nonroad Diesel Engine rule at this time. However, nonroad diesel engines do contribute to nonattainment in some areas. Thus, the emission benefits from this rule will help areas to attain and maintain the CO NAAQS.

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