CHAPTER 2: Air Quality, Health, and Welfare Effects

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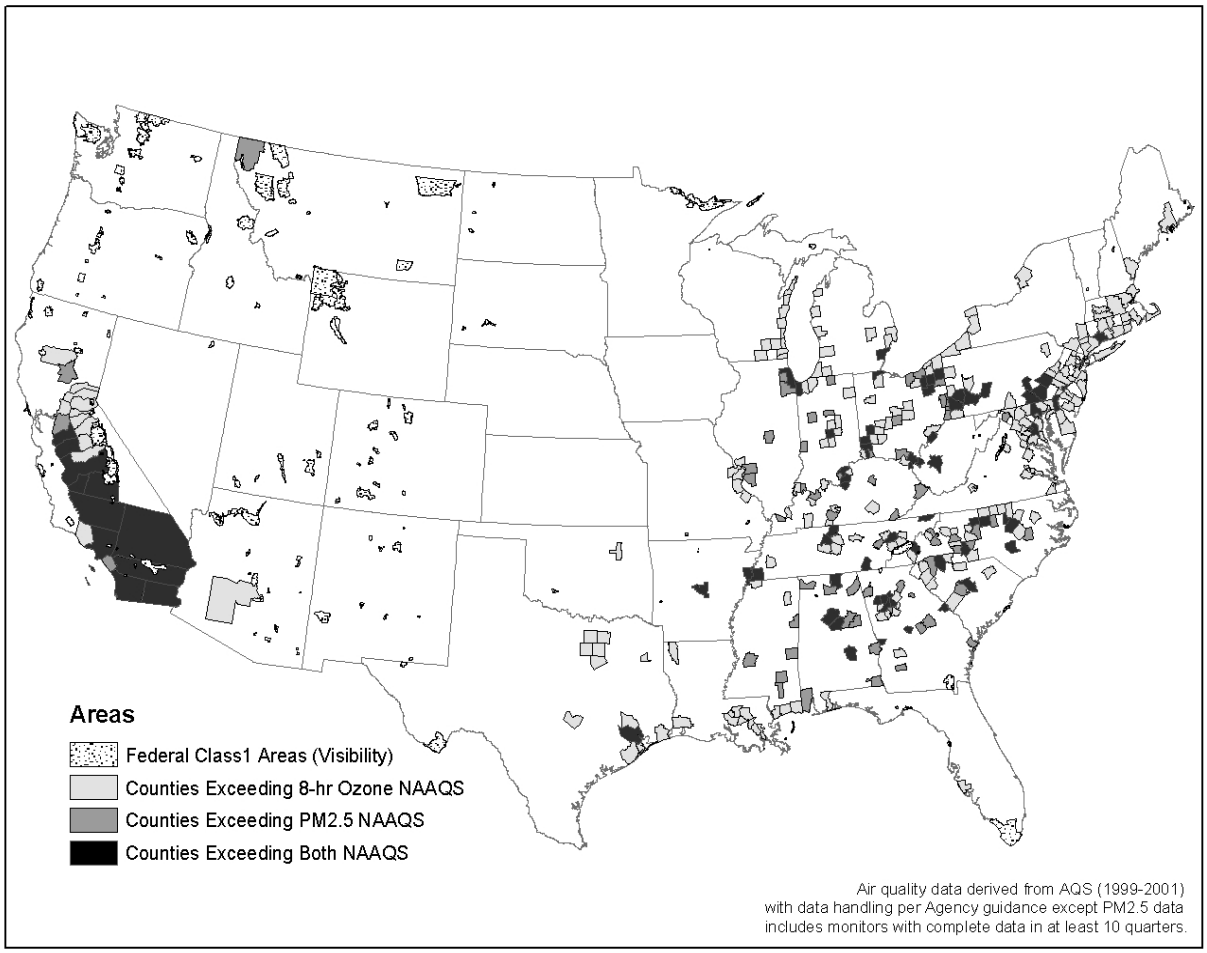
With today's proposal, EPA is acting to extend highway types of emission controls to another major source of diesel engine emissions: nonroad diesel engines. These emissions are significant contributors to atmospheric pollution of particulate matter (PM), ozone and a variety of toxic air pollutants among other pollutants. In our most recent nationwide inventory used for this proposal (1996), the nonroad diesels affected by this proposal contribute over 43 percent of diesel PM emissions from mobile sources, up to 18 percent of total $PM_{2.5}$ emissions in urban areas, and up to 14 percent of NOx emissions in urban areas.

Without further control beyond those standards we have already adopted, by the year 2020, these engines will emit 61 percent of diesel PM from mobile sources, up to 19 percent of all direct $PM_{2.5}$ emissions in urban areas, and up to 20 percent of NOx.emissions in urban areas. When fully implemented, today's proposal would reduce nonroad diesel $PM_{2.5}$ and NOx emissions by more than 90 percent. It will also virtually eliminate nonroad diesel SOx emissions, which amounted to nearly 300,000 tons in 1996, and would otherwise grow to approximately 380,000 tons by 2020.

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 other environmental effects such as visibility. Based on the most recent monitoring data available for this rule (1999-2001), such problems are widespread in the United States. There are over 70 million people living in counties with PM2.5 levels exceeding the PM2.5 NAAQS, and 111 million people living in counties exceeding the 8-hour ozone NAAQS. Figure 2.-1 illustrates the widespread nature of these problems. Shown in this figure are counties exceeding either or both of the two NAAQS plus mandatory Federal Class I areas, which have particular needs for reductions in haze.

Figure 2-1
Nonroad Diesel-related Air Quality Problems are Widespread



As we will describe later in Chapter 9, the air quality improvements expected from this proposal would produce major benefits to human health and welfare, with a combined value in excess of half a trillion dollars between 2007 and 2030. By the year 2030, this proposed rule would be expected to prevent approximately 9,600 deaths per year from premature mortality, and 16,000 nonfatal heart attacks per year. By 2030, it would also prevent 14,000 annual acute bronchitis attacks in children, 260,000 respiratory symptoms in children, nearly 1 million lost work days among adults because of their own symptoms, and 6 million days where adults have to restrict their activities due to symptoms in 2030.

In this chapter we will describe in more detail the air pollution problems associated with emissions from nonroad diesel engines and air quality benefits we expect to realize from the fuel and engine controls in this proposal. The emissions from nonroad diesel engines that are being directly controlled by this rulemaking are NOx, PM and NMHC, and to a lesser extent, CO.

Gaseous air toxics from nonroad diesel engines will also be reduced as a consequence of the proposed standards. In addition, there will be a substantial reduction in SOx emissions resulting from the proposed reduction in sulfur level in diesel fuel. SOx is transformed in the atmosphere to form PM (sulfate).

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 and 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 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 (> 10 micrometers) tend to be removed by the respiratory clearance mechanisms whereas smaller particles are deposited deeper in the lungs. Also, particulate scatters light obstructing visibility.

The emission sources, formation processes, chemical composition, atmospheric residence times, transport distances and other parameters of fine and coarse particles are distinct. Fine

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

particles are directly emitted from combustion sources and are formed secondarily from gaseous precursors such as sulfur dioxide, oxides of nitrogen, or organic compounds. Fine particles are generally composed of sulfate, nitrate, chloride, ammonium compounds, organic carbon, elemental carbon, and metals. Nonroad diesels currently emit high levels of NOx which react in the atmosphere to form secondary $PM_{2.5}$ (namely ammonium nitrate). 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 $PM_{2.5}$). Combustion of coal, oil, diesel, gasoline, and wood, as well as high temperature process sources such as smelters and steel mills, produce emissions that contribute to fine particle formation. In contrast, coarse particles typically result from mechanical crushing or grinding in both natural and anthropogenic sources. They include resuspended dusts, plant material, and crustal material from paved roads, unpaved roads, construction, farming, and mining activities. Fine particles can remain in the atmosphere for days to weeks and travel through the atmosphere hundreds to thousands of kilometers, while coarse particles 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 (which is attributable to a number of sources including diesel) contributes to a series of adverse health effects. These health effects are discussed in detail in the EPA Air Quality Criteria Document for PM as well as the draft updates of this document released in the past year.[1] In addition, EPA recently released its final "Health Assessment Document for Diesel Engine Exhaust," which also reviews health effects information related to diesel exhaust as a whole including diesel PM, which is one component of ambient PM.[2]

As detailed in these documents, health effects associated with short-term variation in ambient particulate matter (PM) have been indicated by 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]

Two major cohort studies, the Harvard Six Cities and the ACS studies suggest an association between exposure to ambient PM and premature mortality from cardiorespiratory causes.[25, 26] These are two prospective cohort studies that 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 $PM_{2.5}$.[27] This most recent finding is of special interest in this proposal, because of the association of diesel exhaust and lung cancer in occupational studies of varying design.

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.[28, 29, 30, 31, 32, 33] 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.[34, 35, 36, 37]

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 (white blood cell - WBC) 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.[38, 39, 40, 41] 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.[42, 43, 44, 45] These blood indices have been associated with coronary heart disease and cardiac events such as heart attack.[46, 47] 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.[48, 49]

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.[50, 51] 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.

Recently, the Health Effects Institute (HEI) reported findings by health researchers at Johns Hopkins University and others that have raised concerns about aspects of the statistical methods used in a number of recent time-series studies of short-term exposures to air pollution and health effects.[52] The 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 employing generalized linear models or other parametric methods, as well as case-crossover studies, are not affected. As discussed in HEI materials provided to EPA and to CASAC, researchers working on the NMMAPS found problems in the default "convergence criteria" used in Generalized Additive Models (GAM) and a separate issue first identified by Canadian investigators about the potential to underestimate standard errors in the same statistical package. These and other scientists have begun to reanalyze the results of several important time series studies with alternative approaches that address these issues and have found a downward revision of some results. For example, the mortality risk estimates for short-term exposure to PM_{10} from NMMAPS were overestimated (this study was not used in this benefits analysis of fine particle effects). However, both the relative magnitude and the direction of bias introduced by the convergence issue is case-specific. In most cases, the concentration-response relationship may be overestimated; in other cases, it may be underestimated. The preliminary reanalyses of the mortality and morbidity components of NMMAPS suggest that analyses reporting the lowest relative risks appear to be affected more greatly by this error than studies reporting higher relative risks.[53, 54]

During the compilation of the draft Air Quality Criteria Document, examination of the original studies used in our 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. While resolution of these issues is likely to take some time, the preliminary results from ongoing reanalyses of some of the studies (Dominici et al, 2002; Schwartz and Zanobetti, 2002; Schwartz, personal communication 2002) suggest a more modest effect of the S-plus error than reported for the NMMAPS PM_{10} mortality study.[55] In December 2002, a number of researchers submitted reanalysis reports, and the HEI is currently coordinating review of these reports by a peer review panel. The final report on these reanalyses is expected by the end of April 2003, and the results will be incorporated in the fourth external review draft of the Criteria Document that will be released in summer 2003. While we wait for further clarification from the scientific community, we are not presenting the tables of short-term exposure effects from the draft Air Quality Criteria Document. EPA will continue to monitor the progress of this concern, and make appropriate adjustments as further information is made available.

The long-term exposure health effects of PM are summarized in Table 2.1.1-1 which is taken directly from the draft Air Quality Criteria Document referenced earlier that was released in 2002. This document is continuing to undergo expert and public review.

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[a]	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 $\mu g/m^3$) (SSI)	1.02 (0.99-1.04)	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} (50 $\mu g/m^3$)	1.242 (0.955-1.616) (males)	51 (±17)
	PM_{10} (cutoff = 30 days/year >100 $\mu g/m^3$)	1.082 (1.008-1.162) (males)	
	PM_{10} (50 $\mu g/m^3$)	0.879 (0.713-1.085) (females)	51 (±17)
	PM_{10} (cutoff = 30 days/year >100 $\mu g/m^3$)	0.958 (0.899-1.021) (females)	
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

Table 2.1.1-1 (continued)
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[a]	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} (25 $\mu g/m^3$)	1.06 (0.93, 1.21)	28.0-84.9
	Acid vapor (1.7 ppb)	1.13 (0.92, 1.38)	0.9-3.2 ppb
12 Southern California communities[K] (children with asthma)	PM_{10} (19 $\mu g/m^3$)	1.1 (0.0.8, 1.7)	13.0-70.7
	$PM_{2.5}$ (15 $\mu g/m^3$)	1.3 (0.7, 2.4)	6.7-31.5
	Acid vapor (1.8 ppb)	1.4 (0.9, 2.1)	1.0-5.0 ppb
Increased Obstruction in Adults			
Southern California[L]	PM_{10} (cutoff of 42 days/year >100 $\mu g/m^3$)	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 $\mu g/m^3$)	-24.9 (-47.2, -2.6) FVC	28.0-84.9
	Acid vapor (1.7 ppb)	-24.9 (-65.08, 15.28) FVC	0.9-3.2 ppb
12 Southern California communities[N] (all children)	PM_{10} (25 $\mu g/m^3$)	-32.0 (-58.9, -5.1) MMEF	28.0-84.9
	Acid vapor (1.7 ppb)	-7.9 (-60.43, 44.63) MMEF	0.9-3.2 ppb
12 Southern California communities[O] (4^{th} grade cohort)	PM_{10} (51.5 $\mu g/m^3$)	-0.58 (-1.14, -0.02) FVC growth	NR
	$PM_{2.5}$ (25.9 $\mu g/m^3$)	-0.47 (-0.94, 0.01) FVC growth	
	$PM_{10-2.5}$ (25.6 $\mu g/m^3$)	-0.57 (-1.20, 0.06) FVC growth	
	Acid vapor (4.3 ppb)	-0.57 (-1.06, -0.07) FVC growth	
12 Southern California communities[O] (4^{th} grade cohort)	PM_{10} (51.5 $\mu g/m^3$)	-1.32 (-2.43, -0.20) MMEF growth	NR
	$PM_{2.5}$ (25.9 $\mu g/m^3$)	-1.03 (-1.95, -0.09) MMEF growth	
	$PM_{10-2.5}$ (25.6 $\mu g/m^3$)	-1.37 (-2.57, -0.15) MMEF growth	
	Acid vapor (4.3 ppb)	-1.03 (-2.09, 0.05) MMEF growth	

Table 2.1.1-1 (continued)
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[a]	Range of City PM Levels * Means ($\mu g/m^3$)
Decreased Lung Function 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)

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

a Schwartz, J.; Dockery, D. W.; Neas, L. M. (1996) Is daily mortality associated specifically with fine particles? J. Air Waste Manage. Assoc. 46: 927-939.

b Ostro, B. D.; Broadwin, R.; Lipsett, M. J. (2000) Coarse and fine particles and daily mortality in the Coachella Valley, California: a follow-up study. J. Exposure Anal. Environ. Epidemiol. 10: 412-419.

c Lippmann, M.; Ito, K.; Nádas, A.; Burnett, R. T. (2000) Association of particulate matter components with daily mortality and morbidity in urban populations. Cambridge, MA: Health Effects Institute; research report no. 95.

d Lipfert, F. W.; Morris, S. C.; Wyzga, R. E. (2000) Daily mortality in the Philadelphia metropolitan area and size-classified particulate matter. J. Air Waste Manage. Assoc.: 1501-1513.

e Mar, T. F.; Norris, G. A.; Koenig, J. Q.; Larson, T. V. (2000) Associations between air pollution and mortality in Phoenix, 1995-1997. Environ. Health Perspect. 108: 347-353.

f Smith, R. L.; Spitzner, D.; Kim, Y.; Fuentes, M. (2000) Threshold dependence of mortality effects for fine and coarse particles in Phoenix, Arizona. J. Air Waste Manage. Assoc. 50: 1367-1379.

g Fairley, D. (1999) Daily mortality and air pollution in Santa Clara County, California: 1989-1996. Environ. Health Perspect. 107: 637-641.

h Burnett, R. T.; Brook, J.; Dann, T.; Delocla, C.; Philips, O.; Cakmak, S.; Vincent, R.; Goldberg, M. S.; Krewski, D. (2000) Association between particulate- and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. In: Grant, L. D., ed. PM2000: particulate matter and health. Inhalation Toxicol. 12(suppl. 4): 15-39.

i Burnett, R. T.; Cakmak, S.; Brook, J. R.; Krewski, D. (1997) The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. Environ. Health Perspect. 105: 614-620.

j Burnett, R. T.; Smith-Doiron, M.; Stieb, D.; Cakmak, S.; Brook, J. R. (1999) Effects of particulate and gaseous air pollution on cardiorespiratory hospitalizations. Arch. Environ. Health 54: 130-139.

k Tolbert, P. E.; Klein, M.; Metzger, K. B.; Peel, J.; Flanders, W. D.; Todd, K.; Mulholland, J. A.; Ryan, P. B.; Frumkin, H. (2000) Interim results of the study of particulates and health in Atlanta (SOPHIA). J. Exposure Anal. Environ. Epidemiol. 10: 446-460.

l Sheppard, L.; Levy, D.; Norris, G.; Larson, T. V.; Koenig, J. Q. (1999) Effects of ambient air pollution on nonelderly asthma hospital admissions in Seattle, Washington, 1987-1994. Epidemiology 10: 23-30.

m Schwartz, J.; Neas, L. M. (2000) Fine particles are more strongly associated than coarse particles with acute respiratory health effects in schoolchildren. Epidemiology. 11: 6-10.

n Naeher, L. P.; Holford, T. R.; Beckett, W. S.; Belanger, K.; Triche, E. W.; Bracken, M. B.; Leaderer, B. P. (1999) Healthy women's PEF variations with ambient summer concentrations of PM_{10}, $PN_{2.5}$, SO_{42-}, H^+, and O_3. Am. J. Respir. Crit. Care Med. 160: 117-125.

o Zhang, H.; Triche, E.; Leaderer, B. (2000) Model for the analysis of binary time series of respiratory symptoms. Am. J. Epidemiol. 151: 1206-1215.

p Neas, L. M.; Schwartz, J.; Dockery, D. (1999) A case-crossover analysis of air pollution and mortality in Philadelphia. Environ. Health Perspect. 107: 629-631.

q Moolgavkar, S. H. (2000) Air pollution and hospital admissions for chronic obstructive pulmonary disease in three metropolitan areas in the United States. In: Grant, L. D., ed. PM2000: particulate matter and health. Inhalation Toxicol. 12(suppl. 4): 75-90.

Most diesel PM is smaller than 2.5 microns based on extensive emissions characterization studies and as reviewed in the recently release Diesel HAD (Health Assessment Document for Diesel Exhaust).[56, 57] Since there are other sources of PM between the 2.5 to 10 micron range (such as earth crustal material), diesel PM constitutes a smaller fraction of PM_{10} than it does of $PM_{2.5}$. EPA is also evaluating the health effects of PM between 2.5 and 10 microns in the draft revised Air Quality Criteria Document.

In addition to the information in the draft revised Air Quality Criteria Document, further conclusions about health effects associated with mobile source PM on-road diesel engine-generated PM being relevant to nonroad application is supported by the observation in the Diesel HAD that the particulate characteristics in the zone around nonroad diesel engines is likely to be substantially the same as the characteristics of diesel particles in general (such as those found along heavily traveled roadways).

Another body of studies have examined health effects associated with living near a major roads. 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.[58] 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.[59] 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 to children living on other roads.[60] Another recently published study from Los Angeles found that maternal residence near heavy traffic during pregnancy is associated with adverse birth outcomes, such as preterm birth and low birth weight.[61] However, these studies are not specifically related to PM, but to fresh emissions from mobile sources, which includes other components as well.

Another recent cohort study examined the association between mortality and residential proximity to major roads in the Netherlands. Examining a cohort of 55 to 69 year-olds from 1986 to1994, the study indicated that long-term residence near major roads, an index of exposure to primary mobile source emissions (including diesel exhaust), was significantly associated with increased cardiopulmonary mortality.[62]

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 (WBCs) contained PM compared with children living on quiet streets.[63] 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.[64, 65, 66]

The particle characteristics in the zone around nonroad diesel engines is not likely to differ substantially from published air quality measurements made along busy roadways. 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 of exposure misclassification in these studies. 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 diesel nonroad engines is fine PM, the emission controls proposed today should contribute to attainment and maintenance of the existing PM NAAQS. More broadly, the proposed standards will benefit public health and welfare through reductions in direct diesel PM and reductions of NOx, SOx, and HCs which 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 reductions from today's proposed rules 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 first 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 ug/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 ug/m^3 averaged over three years.

Currently, 29.5 million people live in PM_{10} nonattainment areas, including moderate and serious areas. There are presently 58 moderate PM_{10} nonattainment areas with a total population of 6.8 million. The attainment date for the initial moderate PM_{10} nonattainment areas, designated by operation of 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. Four serious PM_{10} nonattainment areas (Phoenix, Arizona; 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.[B] While all of these areas are expected to be in attainment before the emission reductions from this proposed rule are expected to occur, these reductions will be important to assist these areas in maintaining the standards.

Many PM_{10} nonattainment areas continue to experience exceedances. Of the 29.5 million people living in designated PM_{10} nonattainment areas, approximately 25 million people are living in nonattainment areas with measured air quality violating the PM_{10} NAAQS in 1999-2001.

[B]EPA has also proposed to grant Las Vegas, Nevada, an extension until December 31, 2006.

Among these are the seven serious areas listed in Table 2.1.1-2 and 4 moderate areas: Nogales, AZ, Imperial Valley, CA, Mono Basin, CA, and El Paso, TX.

Table 2.1.1-2
Serious PM_{10} Nonattainment Areas

Area	Attainment Date	2000 Population	1999-2001 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)	Proposed 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	Yes
Total Population	22.7 million		

In addition to these designated nonattainment areas, there are 19 unclassified areas, where 8.7 million live, for which States have reported PM_{10} monitoring data for 1999-2001 period indicating a PM_{10} NAAQS violation. Although we do not believe that we are limited to considering only designated nonattainment areas a part of this rulemaking, we have focused on the designated areas in the case of PM_{10}. An official designation of PM_{10} nonattainment indicates the existence of a confirmed PM_{10} problem that is more than a result of a one-time monitoring upset or a result of PM_{10} exceedances attributable to natural events. We have not yet excluded the possibility that one or the other of these is responsible for the monitored violations in 1999-2001 in these 19 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. The emission reductions from today's proposal would help States improve their PM_{10} air quality levels and maintain the PM_{10} NAAQS.

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 Fed. Reg., 38651, July 18, 1997). The short term (24-hour) standard is set at a level of 65 $\mu g/m^3$ based on the 98^{th} percentile concentration averaged over three years. (The air quality statistic compared to the standard is referred to as the "design value.") The long-term standard specifies an expected annual arithmetic mean not to exceed 15 ug/m^3 averaged over three years.

Current $PM_{2.5}$ monitored values for 1999-2001, which cover counties having about 75 percent of the country's population, indicate that at least 65 million people in 129 counties live in areas where annual design values of ambient fine PM violate the $PM_{2.5}$ NAAQS. There are an additional 9 million people in 20 counties where levels above the NAAQS are being measured, but there are insufficient data at this time to calculate a design value in accordance with the standard, and thus determine whether these areas are violating the $PM_{2.5}$ NAAQS. In total, this represents 37 percent of the counties and 64 percent of the population in the areas with monitors with levels above the NAAQS. Furthermore, an additional 14 million people live in 40 counties that have air quality measurements within 10 percent of the level of the standard. These areas, although not currently violating the standard, will also benefit from the additional reductions from this rule in order to ensure long term maintenance.

Figure 2.1.1-4 is a map of currently available $PM_{2.5}$ monitoring data, highlighting monitor locations near or above the annual PM2.5 NAAQS. As can be seen from this figure, high ambient levels are widespread throughout the East and California.

Figure 2.1.1-5 graphically presents the numbers of people currently exposed to various unhealthy levels of $PM_{2.5}$.[67] As shown in Table 2.1.1-3 of the 74 million people currently living in counties with measurements above the NAAQS, 22 million live in counties above 20 ug/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
1999/2001 Monitored Population[a] Living in Counties with Annual Average[b] $PM_{2.5}$ Concentrations Shown (70 Percent of Total U.S. Population)

Measured 1999/2000 Annual Average $PM_{2.5}$ Concentration (μg/m3) (A)	Number of Counties Within The Concentration Range	2000 Population Living in Monitored Counties Within The Concentration Range (Millions, 2000 Census Data) (B)	Cumulative Percent of 2000 Monitored Population Living in Counties Within The Concentration Range[c] (C)
>25	3	12.8	7
>20 <=25	10	9.2	5
>15 <=20	136	52.3	27
<=15	402	115.6	61

[a] Monitored population estimates represent populations living in monitored counties (with community based monitors) based on monitors with at least 10 quarter with at least 11 samples per quarter between 1999 and 2001.
[b] Annual average represents the monitor reading with the highest average in each monitored county.
[c] The monitored population is 189.2 million (as reflected in column C, where C=B/Monitored Population). Total monitored population is 191 million; the Census total county-based 2000 population is 272.7 million.

Figure 2.1.1-4
Current Fine PM Monitoring Data

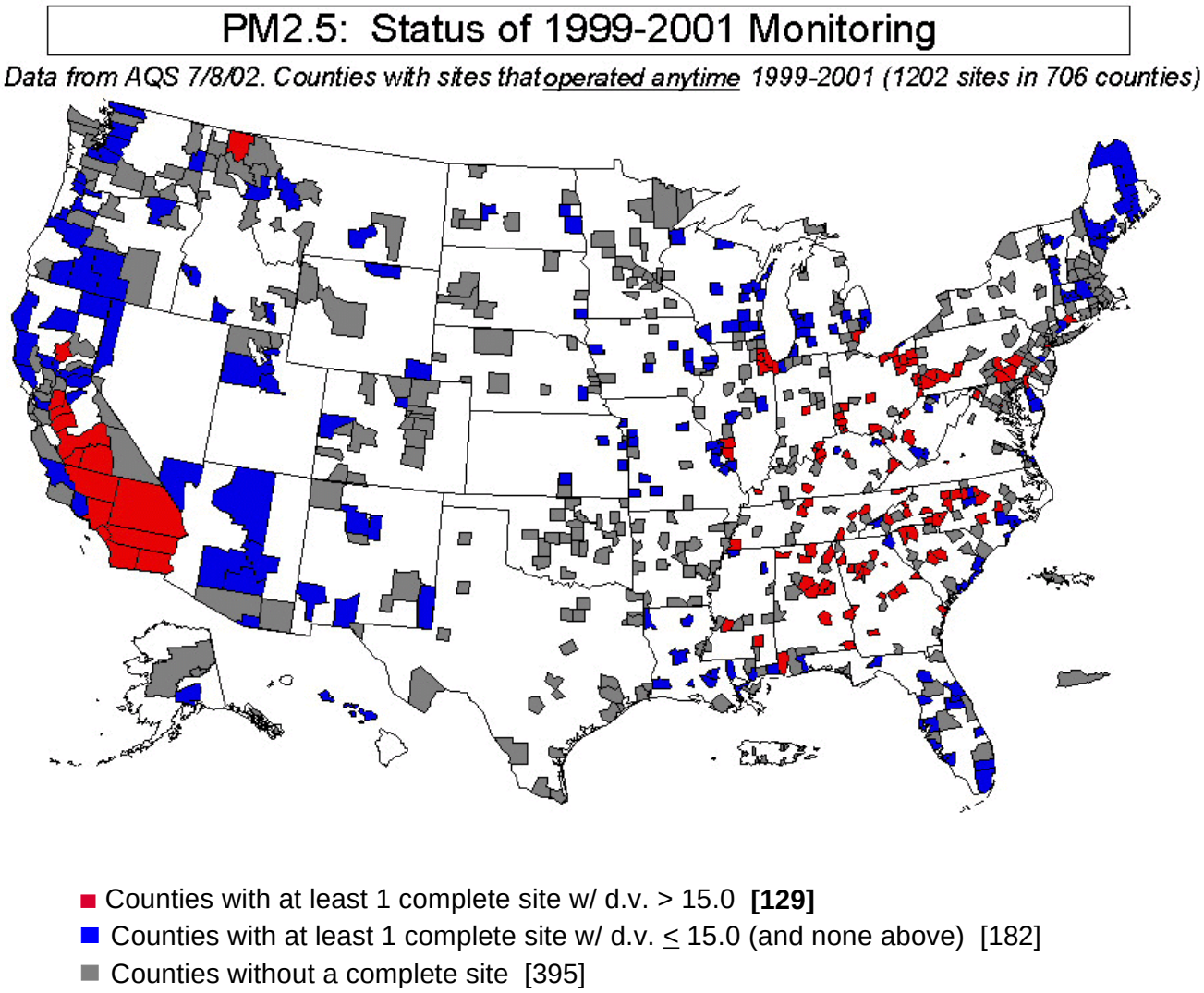
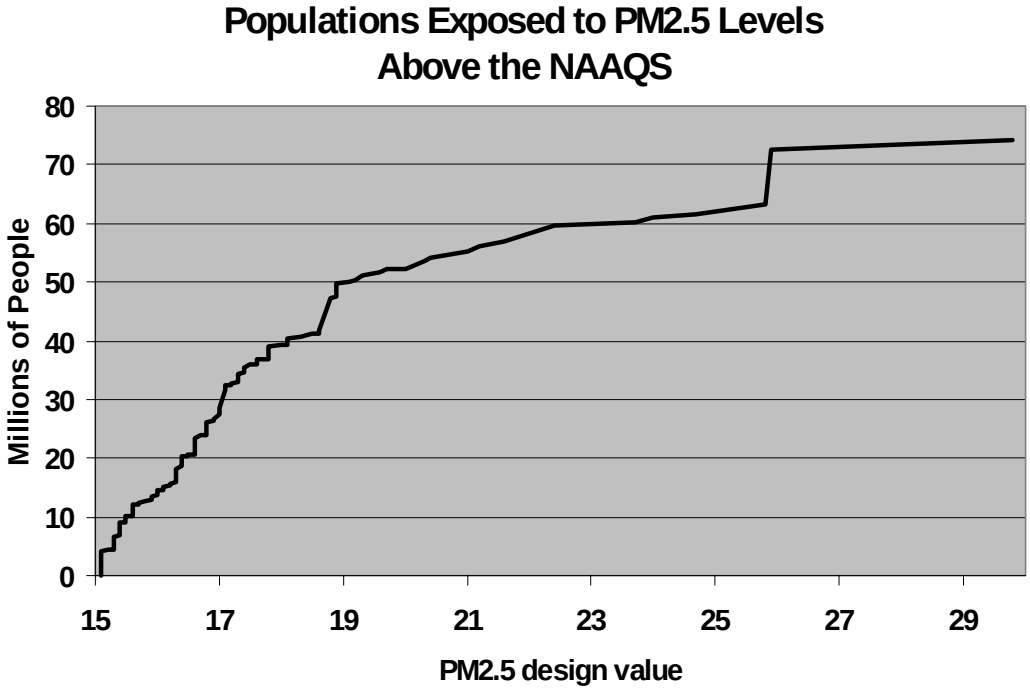


Figure 2.1.1-5



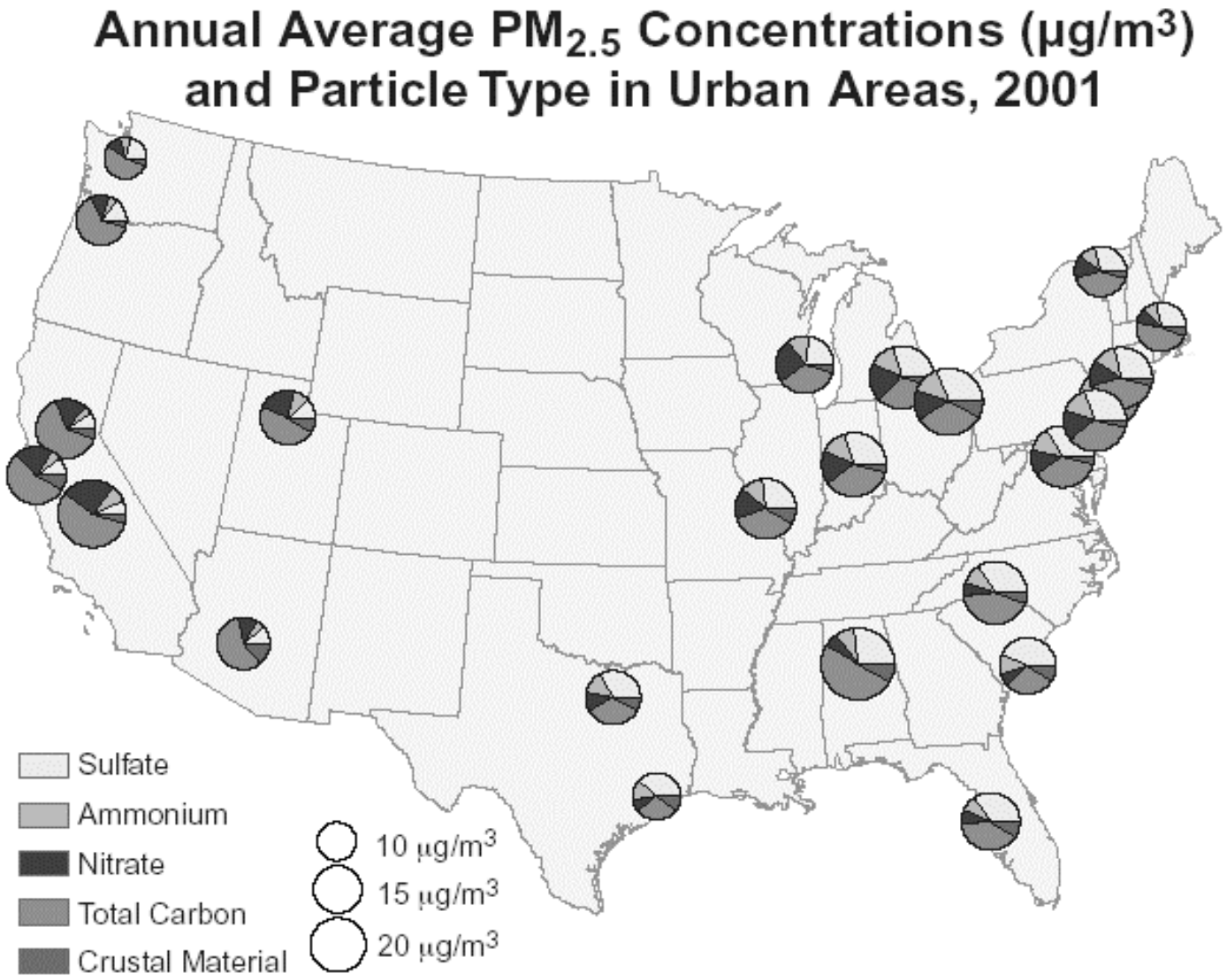
The relative contribution of various chemical components to $PM_{2.5}$ varies by region of the country. Data on $PM_{2.5}$ composition are available from the EPA Speciation Trends Network in 2001 and the IMPROVE Network in 1999 covering both urban and rural areas in numerous regions of the U. S. These 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 U.S. Carbonaceous $PM_{2.5}$ includes both elemental and organic carbon. Nitrates formed from NOx also plays a major role in the western U.S., especially in the California area where it is responsible for about a quarter of the ambient $PM_{2.5}$ concentrations. Sulfate plays a lesser role in these regions by mass, but it remains important to visibility impairment discussed below. For the Eastern and mid U.S., these data show that both sulfates and carbonaceous $PM_{2.5}$ are major contributors 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. Sulfate is also a major contributor to ambient $PM_{2.5}$ in the Eastern U.S. and in some areas make greater contributions than carbonaceous $PM_{2.5}$.

Nonroad engines, especially nonroad diesel engines, contribute significantly to ambient $PM_{2.5}$ levels, largely through emissions of carbonaceous $PM_{2.5}$. Carbonaceous $PM_{2.5}$ is a major portion of ambient $PM_{2.5}$, especially in populous urban areas. Much of the total carbon PM excess is organic carbon. Nonroad diesels also emit high levels of NOx which react in the atmosphere to form secondary $PM_{2.5}$ (namely ammonium nitrate). 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 $PM_{2.5}$). Figure 2.1.1-1 shows the levels and composition of ambient $PM_{2.5}$ in some

urban areas.

Figure 2.1.1-2 shows the levels and composition of $PM_{2.5}$ in rural areas where the total $PM_{2.5}$ levels are generally lower. From Figures 2.1.1-1 and 2.1.1-2, one can compare the levels and composition of $PM_{2.5}$ in various urban areas and a corresponding rural area. This comparison, in Figure 2.1.1-3, 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.[68, 69] See the AQ TSD for details.

Figure 2.1.1-1



Source: EPA Speciation Network, 2001.

Figure 2.1.1-2

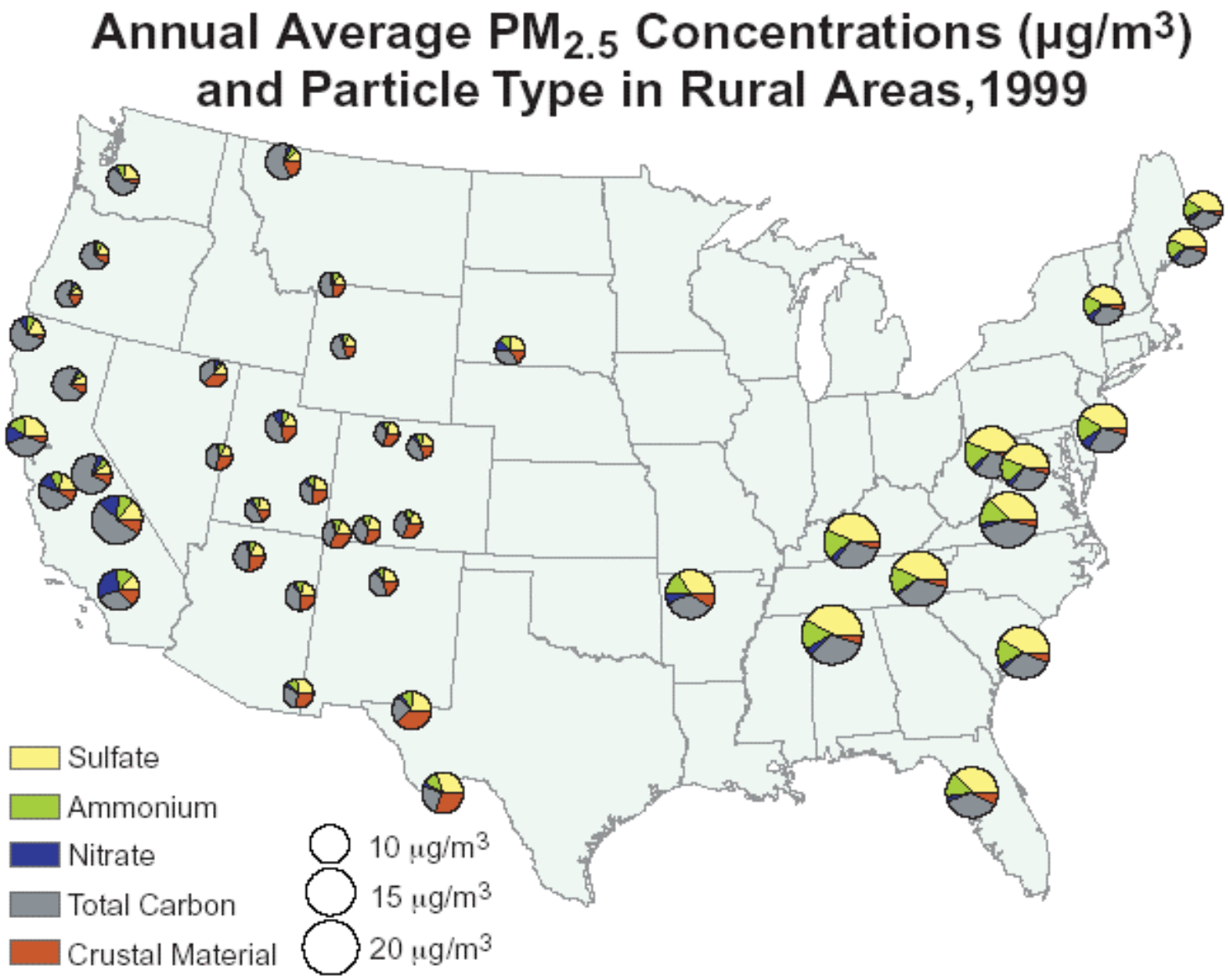
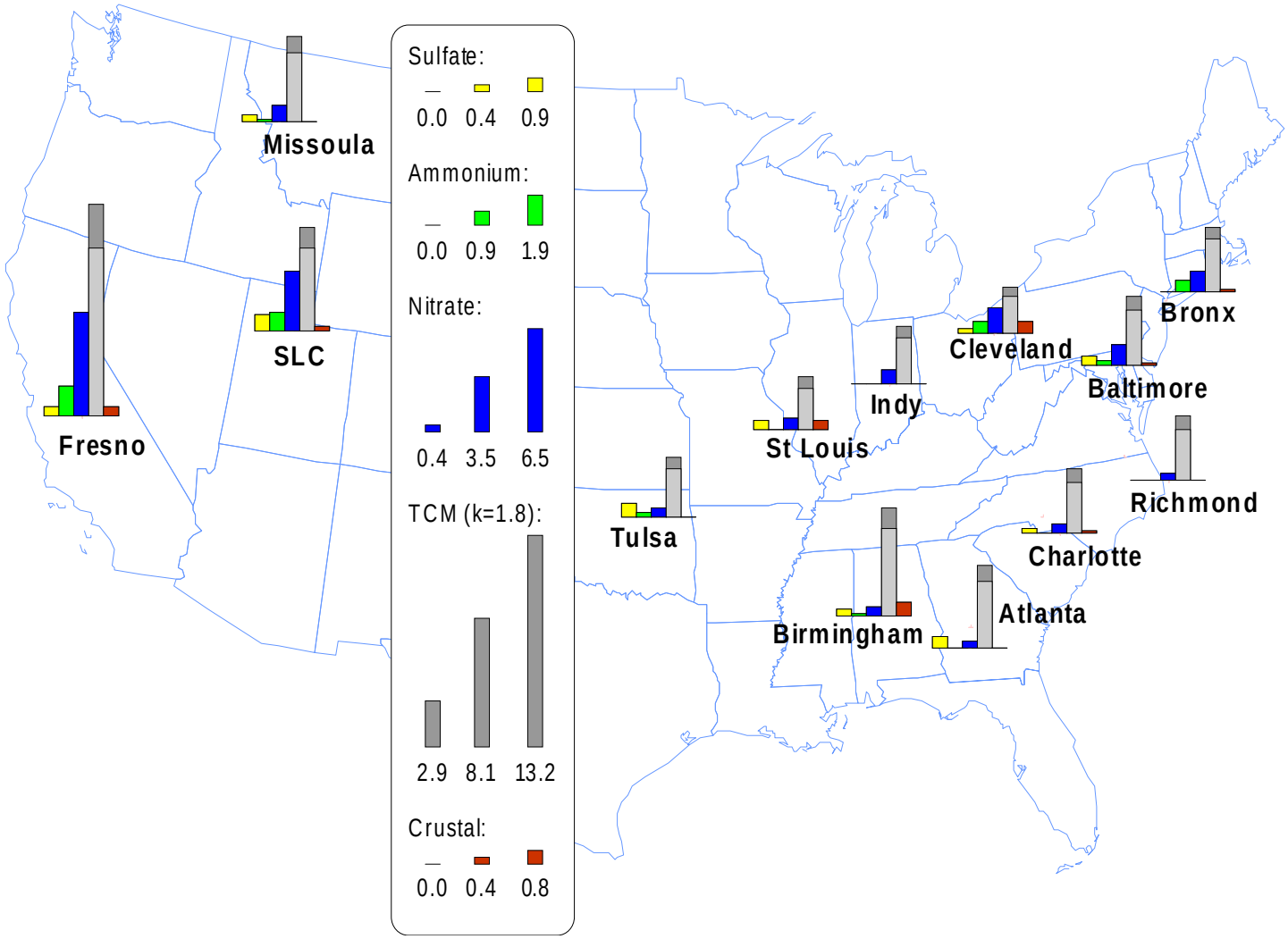

Annual Average PM2.5 Concentrations (µg/m3)
and Particle Type in Rural Areas,1999
Sulfate
Ammonium
Nitrate
Total Carbon
Crustal Material
10 µg/m3
15 µg/m3
20 µg/m3

Figure 2.1.1-3
Composition of Urban Excess $PM_{2.5}$ at Selected Sites, 1999
(Source: U.S. EPA (2003) AQ TSD;Roa and Frank 2003)



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, the proposed standards would 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 proposed standards will also reduce SOx and VOC. Nonroad diesel engines emissions also contribute to national SOx and VOC emissions 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.

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 emissions 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 which 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. This modeling supports the conclusion that there is a broad set of areas with predicted $PM_{2.5}$ concentrations at or above 15 ug/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 U.S. for the various emissions scenarios. Version 7 of REMSAD was used for this proposed rule. 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 proposed rule on U.S. PM concentrations. 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.

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 emissions 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).[70] 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

[C] Given the potential impact of the porposed 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.

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 U.S. 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, there are 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 U.S.

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 U.S. mostly at national parks, national wilderness areas, and other protected pristine areas.[71] 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 100^{th} meridian to divide the Eastern and Western U.S., 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 to 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 U.S. 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 emissions 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 proposal also indicates that the present widespread number of counties with annual averages above 15 ug/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 ug/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 within 10 percent of the standard in 2020, which will increase to 64 million people in 2030. The AQ Modeling TSD lists the specifics.

Our modeling also indicates that the reductions we are expecting from today's proposal will make a substantial contribution to reducing these exposures.[D] In 2020, the number of people living in counties with $PM_{2.5}$ levels above the NAAQS would be reduced from 66 million to 60 million living in 67 counties. That is a reduction of 9 percent in exposed population and 15 percent of the number of counties. In 2030, there would be a 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 exposed population and a 21 percent of the number of counties. Furthermore, our modeling also shows that the emission reductions would 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 ug/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 ambient monitoring data sufficient to calculate current 3-year design values.

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]

[D]The results illustrate the type of PM changes for the preliminary control option, as discussed in the Draft RIA in Section 3.6. The proposal differs from the modeled control case based on updated information; however, we believe that the net results would approximate future emissions, although we anticipate the PM reductions might be slightly smaller.

State	County	1999 - 2001 Design Value (ug/m^3)	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
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

State	County	1999 - 2001 Design Value (ug/m^3)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
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
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

State	County	1999 - 2001 Design Value (ug/m^3)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
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			79	67	107	84	
Population of Violating Counties[b]			65,821,078	60,453,470	85,525,624	71,375,639	

[a] The proposal differs based on updated information; however, we believe that the net results would approximate future emissions, although we anticipate the design value improvements would be slightly smaller.
[b] Populations are based on 2020 and 2030 estimates. See the AQ Modeling TSD for details.

Table 2.1.2-3 lists the counties with 2020 and 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 ug/m^3, but less than 15.05 ug/m^3. Counties are marked with an "V" in the table if their projected design values are greater than or equal to 15.05 ug/m^3. The current 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 proposed rule will help assure that these counties continue to meet the standard.

Table 2.1.3-3
Counties with 2020 and 2030 Projected Annual PM2.5 Design Values
within Ten Percent of the Annual PM2.5 Standard.[a]

State	County	1999 - 2001 Design Value (ug/m^3)	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
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

State	County	1999 - 2001 Design Value (ug/m^3)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
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
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

State	County	1999 - 2001 Design Value (ug/m^3)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
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%			70	62	64	70	
Population of Counties within 10%[b]			23,836,367	24,151,782	16,870,324	24,839,565	

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

[b] Populations are based on 2020 and 2030 estimates. See the AQ Modeling TSD for details.

We estimate that the reduction of this proposed rule would produce nationwide air quality improvements in PM levels. On a population weighted basis, the average change in future year annual averages would be a decrease of 0.33 ug/m^3 in 2020, and 0.46 ug/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's current plans call for designating $PM_{2.5}$ nonattainment areas in late-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." Therefore, based on this information, we expect that most or all areas will need to attain the $PM_{2.5}$ NAAQS in the 2009 to 2014 time frame, and then be required to maintain the NAAQS thereafter.

Since the emission reductions expected from today's proposal would begin in this same time frame, the projected reductions in nonroad emissions would 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.[72, 73, 74] Furthermore, this action would ensure 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 later.

2.1.3 Welfare 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.[75] Visibility impairment has been considered the "best understood and most easily measured effect of air pollution."[76] Visibility degradation is often directly proportional to decreases in light transmittal in the atmosphere. Scattering and absorption by both gases and particles decrease light transmittance. Haze obscures the clarity, color, texture, and form of what we see. Fine particles are the major cause of reduced visibility in parts of the U.S. 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.

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. Sulfates contribute to visibility impairment especially on the haziest days across the U.S., accounting in the rural Eastern U.S. 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 Eastern U.S. locations.[77]

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.[78] 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 to 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.[79]

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.

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.[80]

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.[81] 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.[82] In the Eastern U.S., 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 U.S., 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

[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.

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 which 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 proposed 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 mandatory Federal Class I areas, we discuss the types of effects, current and future visibility conditions absent the proposed reductions, and the changes we anticipate from the proposed reductions in emissions from nonroad diesels. We conclude that the proposed 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.[83] 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 U.S., 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.[84] Regional strategies 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.[85]

Without the effects of pollution, a natural visual range is approximately 140 miles (230 kilometers) in the West and 90 miles (150 kilometers) in the East. However, over the years, in many parts of the U.S., fine particles have significantly reduced the range that people can see. In the West, the current 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).[86]

Current $PM_{2.5}$ monitored values for 1999-2001 indicate that at least 65 million people in 129 counties live in areas where design values of $PM_{2.5}$ annual levels are at or above the $PM_{2.5}$ NAAQS. There are an additional 9 million people in 20 counties where levels exceeding the NAAQS are being measured, but there are insufficient data at this time to make a complete comparison with the NAAQS. In total, this represents 37 percent of the counties and 64 percent of the population in the areas with monitors with levels above the NAAQS. Taken together, these data indicate that a total of 74 million people live in areas where long-term ambient fine particulate matter levels are at or above 15 $\mu g/m^3$.[87] 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 14 million people live in 41 counties that have air quality measurements for 1999-2001 within 10 percent of the level of the PM standard. These areas, although not currently violating the standard, would also benefit from the additional reductions from this proposed rule in order 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 reductions from this proposed 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.[88] In 2030, this number will rise to 107 counties with a population of 71 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 to 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 to 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 proposed rule contribute to these effects. 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 Proposed Reductions

For this proposal, we also modeled a preliminary control scenario which illustrates the likely reductions from our proposal. 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 process based on our best judgement at that time. As additional data regarding technical feasibility and other factors became available, our judgement 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 below. It is important to note that these changes would not affect our estimates of the baseline conditions without additional controls described above. For the final rule, considering public comment, we plan to model the final control scenario. We anticipate that the proposed nonroad diesel emissions reductions would improve to the projected visibility impairment, and that there continues to be a need for reductions from those engines.

Based on our modeling, we predict that in 2020, there would be 12 counties with a population of 6 million that would come into attainment with the annual $PM_{2.5}$ because of the improvements in air quality from the proposed emissions reductions. In 2030, a total of 24 counties (12 additional counties) with a population of 14 million (8 million additional people) would come

into attainment with the annual $PM_{2.5}$ because of the improvements in air quality from the proposed emissions reductions. There would also be reductions in counties with levels close to the standard that would improve visibility conditions and help them maintain the standards. All of these areas and their populations would experience improvements in visibility as well as health, described earlier.

We estimate that the reduction of this proposed rule would produce nationwide air quality improvements in PM levels. On a population weighted basis, the average change in future year annual averages would be a decrease of 0.33 ug/m^3 in 2020, and 0.46 ug/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 proposed rule in 2030 would improve visibility by 0.33 deciview. Likewise, we estimate visibility in the West to be about 8.83 deciviews (or visual range of 162 kilometers) in 2030, and we estimate emission reductions from this proposed rule in 2030 would improve visibility by 0.25 deciview. These improvements are needed in conjunction with other sulfur strategies in the East and a combination of strategies in the West to make reasonable progress toward visibility goals.[89] Thus, in the future, a substantial percent of the population may experience improvements visibility in areas where they live, work and recreate because of the proposed nonroad emission reductions.

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 proposal differs based on updated information; however, we believe that the net results would approximate future PM emissions, although we anticipate the visibility improvements would be slightly 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.[90] In the East, there are and will continue to be sizable areas above 15 ug/m^3 and where light extinction is significantly above natural background. Thus, large areas of the Eastern U.S. 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 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.[91] 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 emissions 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.[92] The conclusions draw upon the Interagency Monitoring of Protected Visual Environments (IMPROVE) network data.[93]

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

[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.

worst days ranging up to 17 deciviews (compared to 5.3 deciviews of natural background visibility).[94] 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 Eastern U.S. Class I areas trend sites, visibility on the haziest 20 percent of days remains significantly impaired with a mean visual range of 23 kilometers for 1999 as compared to 84 kilometers for the clearest days in 1999. In the 26 Western U.S. Class I areas trends sites, 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

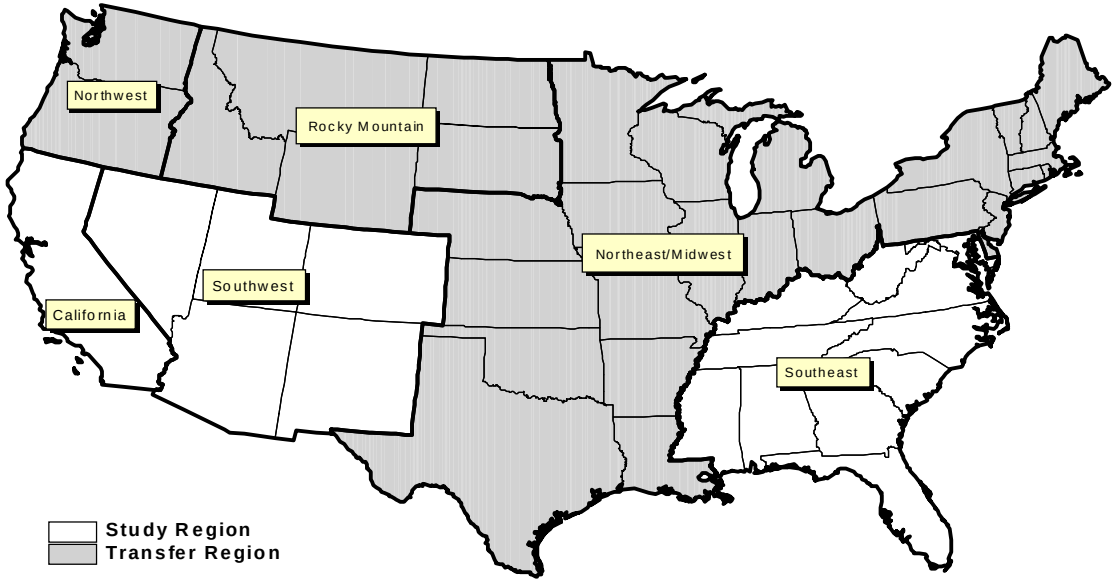
As part of the PM air quality modeling described above, we modeled future visibility conditions in the mandatory Federal Class I areas absent additional controls. The results by region are summarized in Table 2.1.3-3. In Figure 2.1.3-1, we define the regions used in this analysis.[95] These air quality 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 Emissions 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 Continental U.S.



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 Proposed 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 emissions 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 which illustrates the likely reductions from our proposal. 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 proposal differs based on updated information; however, we believe that the net results would approximate future PM emissions, although we anticipate the visibility improvements would be slightly 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.[96] 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 U.S. 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 emissions trends and acidity of water bodies in the Eastern U.S. 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.[97] The decrease in sulfates is consistent with emissions 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.[98] 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 U.S.

The SOx and NOx reductions from today's action 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 today's action 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 US 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.[99] 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. [100] 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 U.S., 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.[101] 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.[102] A review of peer reviewed literature in 1995 on the subject of air deposition suggests a typical contribution of 20 percent or higher.[103] 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.[104] The National Exposure Research Laboratory, US EPA, estimated based on prior studies that 20 to 35 percent of the nitrogen loading to the Chesapeake Bay is attributable to atmospheric deposition.[105] The mobile source portion of atmospheric NOx contribution to the Chesapeake Bay was modeled at about 30 percent of total air deposition.[106]

Deposition of nitrogen from nonroad diesel engines contributes to elevated nitrogen levels in waterbodies. The proposed standards for nonroad diesel engines will reduce total NOx emissions by 831,000 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.[107] One of these 15 compounds, a group known as polycyclic organic matter (POM), are compounds that are mainly adhered to the particles emitted by mobile sources and later fall to earth in the form of precipitation or dry deposition of particles. The mobile source contribution of the 7 most toxic POM is at least 62 tons/year[108] and represents only those POM that are adhered to mobile source particulate emissions. The majority of these emissions are produced by diesel engines.

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

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

The PM reductions from today's proposed action will help reduce not only the PM emissions from land-based nonroad diesel engines but also the deposition of the POM adhering to the particles, thereby helping to reduce health effects of POM in lakes and streams, accelerate the recovery of affected lakes and streams, and revive the ecosystems adversely affected.

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 which are generally high molecular weight hydrocarbon types but not the more volatile gaseous hydrocarbon compounds) is generally used as a surrogate measure for diesel exhaust.

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).[109] 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 emissions controls is appropriate.

EPA recently 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.[110] In accordance with earlier EPA guidelines, diesel exhaust would be similarly classified as a probable human carcinogen (Group B1).[111, 112] 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 US Department of Health and Human Services) have made similar classifications.[113,114,115,116,117] The Health Effects Institute has also made numerous studies and

report on the potential carcinogenicity of diesel exhaust.[118, 119, 120] Numerous animal and bioassay/genotoxic tests have been done on diesel exhaust.[121, 122] Also, case-control and cohort studies have been conducted on railroad engine exposures[123,124,125] in addition to studies on truck workers. [126, 127,128] Also, there are numerous other epidemiologic studies including some studying mine workers and fire fighters.[129, 130]

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 cleaner 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 onroad and nonroad engines. The EPA Diesel HAD acknowledges that the studies were done on engines with older technologies generally for onroad and that "there have been changes in the physical and chemical composition of some DE [diesel exhaust] emissions (onroad vehicle emissions) over time, though there is no definitive information to show that the emission changes portend significant toxicological changes." 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 onroad engines which have been subject to PM standards since 1988.

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.[131] Another cohort study of operators of heavy construction equipment also showed increased lung cancer incidence for these workers.[132]

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.[133,134] That is, these two studies show an overall increase in lung cancer for the exposed groups of 35 percent and 47 percent compared to 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 ug/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 to 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 ug./m^3 for diesel PM from on-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 ug/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 judgement. 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 appropriateness of extrapolation from occupational risk to ambient environmental exposure levels should be more confidently judged to be appropriate.

EPA also recently 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.[135] This national assessment estimates average population inhalation exposures to diesel PM in 1996 for nonroad as well as onroad 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 an onroad 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.

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 has been designed a likely human carcinogen due to inhalation at environmental exposure. Other organizations have made similar determinations.
2. The entire 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 US 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 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.[136],[137],[138],[139] The diesel RfC is based on a "no observable adverse effect" level of 144 ug/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 ug/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 is presently lacking to derive an RfC.

While there have been relatively few human 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 is relevant to diesel PM is 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.[140,141,142,143] 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 ug/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 ug/m^3 provides adequate public health protection for ambient $PM_{2.5}$. In fact, there may be benefits to reducing diesel PM below 5 ug/m^3 since diesel PM is a major contributor to ambient $PM_{2.5}$.[G]

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 diesels in

[G]It should again be noted that recent epidemiologic studies (such as by Schwartz, Laden, and Zanobetti) of ambient $PM_{2.5}$ do not indicate a threshold of effects at low concentrations.

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 and others) 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. In a review discussed earlier of studies (by R. Delfino) of the respiratory health of people living near roadways, another publication indicated that the risk of asthma and related respiratory disease appeared elevated in people living near heavy traffic. These studies offer evidence that people exposed most directly to emissions from mobile sources including those from diesels 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.

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 which are 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 on-highway diesel PM.

2.2.1.3.1 Toxics Modeling and Methods

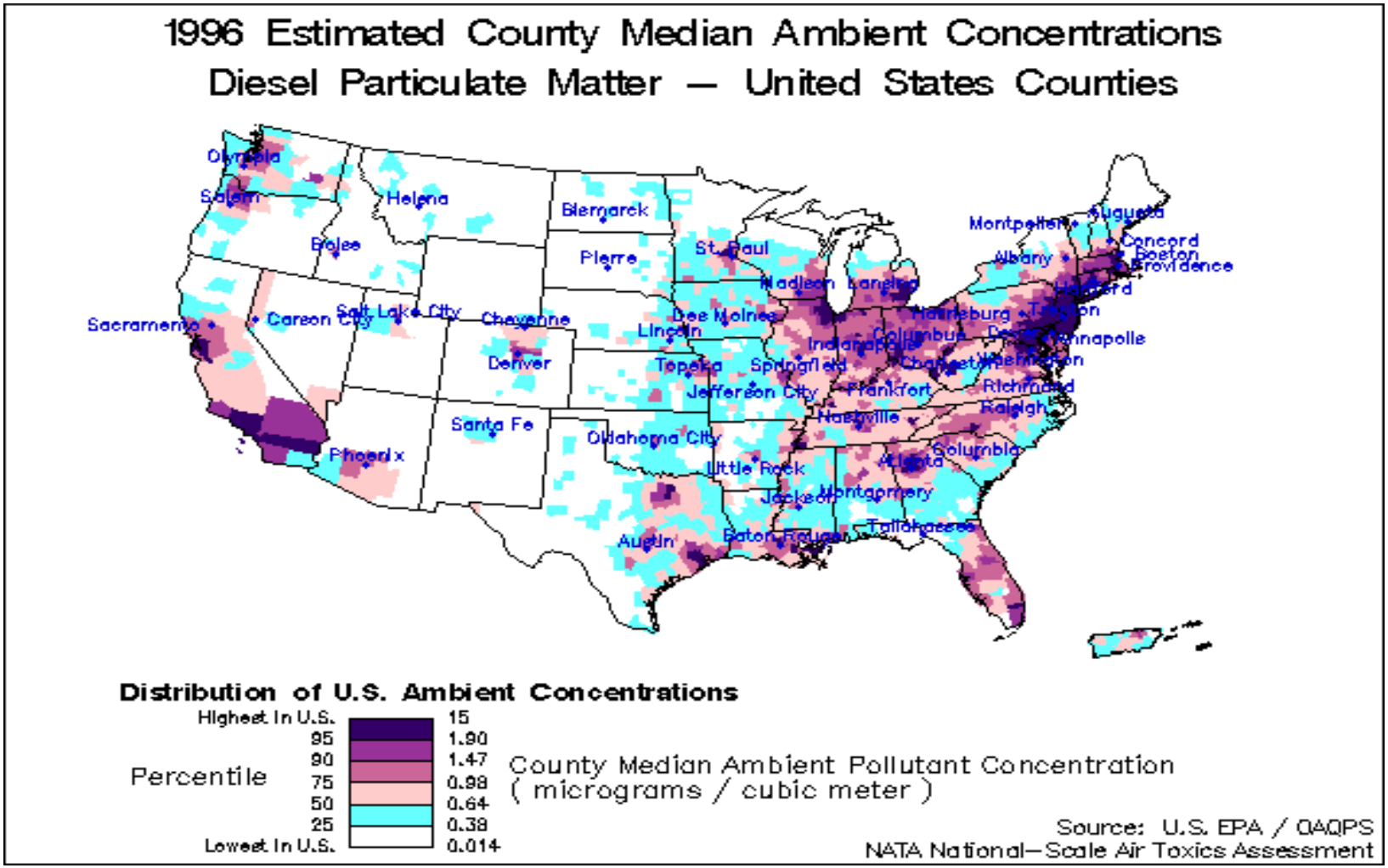
In addition to the general ambient PM modeling conducted for this proposal, diesel PM concentrations for 1996 were recently estimated as part of the National-Scale Air Toxics Assessment (NATA; EPA, 2002). In this assessment, the PM inventory developed for the recent regulation promulgating 2007 heavy 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 which 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 diesels. A map of county median concentrations is provided in Figure 2.2.1-1. While the high median concentrations are clustered in the Northeast, Great Lake States and California, areas of high median concentrations are distributed throughout the U.S.

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 ($\mu g/m^3$)
5^{th} Percentile	0.33	0.51	0.15
25^{th} Percentile	0.85	1.17	0.42
Average	2.06	2.41	0.74
75^{th} Percentile	2.45	2.7	0.97
95^{th} 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

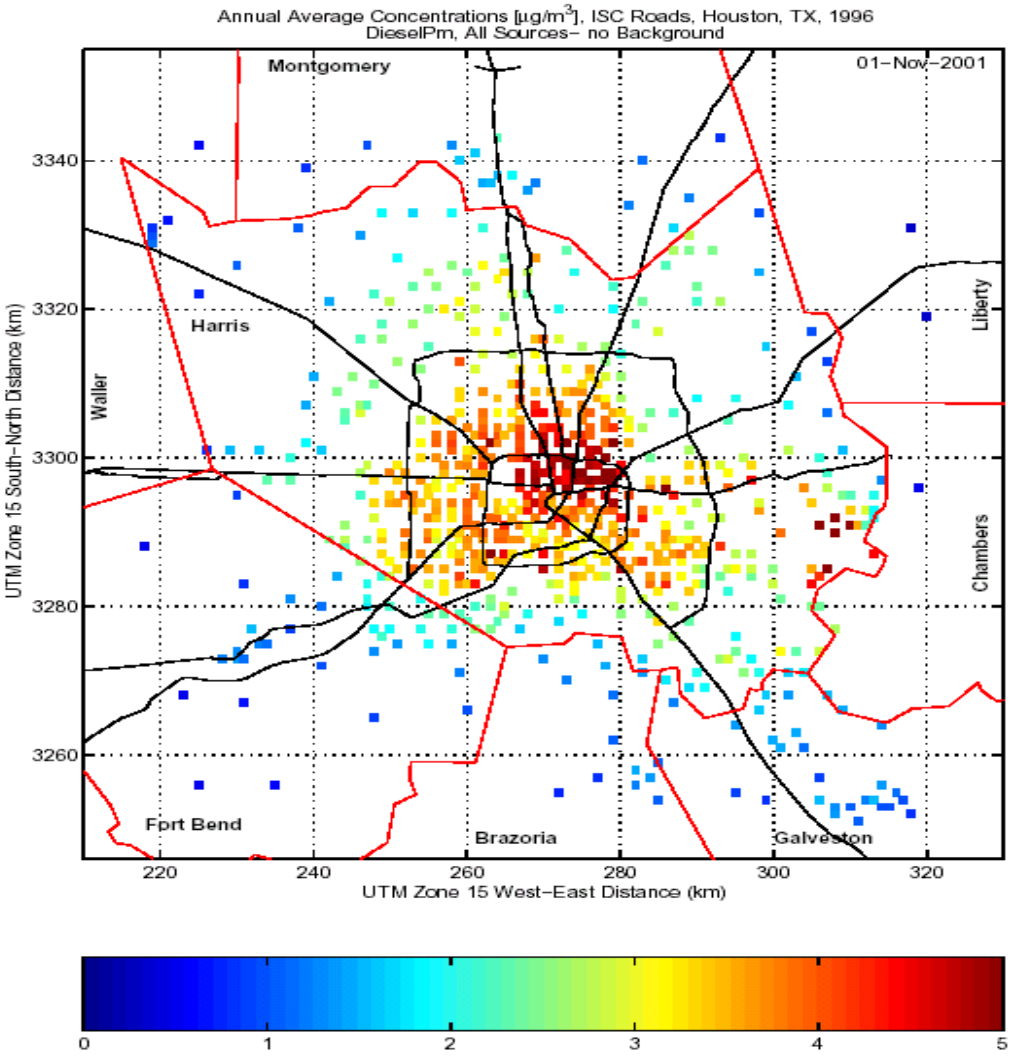
Figure 2.2.1-1
Estimated County Median Concentrations of Diesel Particulate Matter



Source: EPA National-Scale Air Toxics Assessment for 1996.

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.[144] 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.[145] 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.[146] 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.[147] Results from several source apportionment studies were used to develop these factors.[148, 149, 150, 151, 152, 153, 154] 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
- East or West US
- 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.[155] 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 to 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 onroad and nonroad but, instead, gives diesel PM as a whole. One can allocate the diesel PM numbers based on the inventory split between onroad 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 on-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.[156] 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 $\mu g/m^3$. It should be noted 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).[157] 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 onroad vehicles.

The CMB model was applied in California's San Joaquin Valley during winter 1995-1996.[158] 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 U.S. 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 Pensacola, FL 1.91-3.07 $\mu g/m^3$ which 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.[159] 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 onroad 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.[160, 161, 162, 163] 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) is presently measuring 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. These data, tentatively scheduled to be available in about a year, will be useful in quantifying high localized exposure levels in the vicinity of nonroad equipment.[164] Some initial results are expected in late 2003.

2.2.1.4.2 Ambient Exposures in the General Population

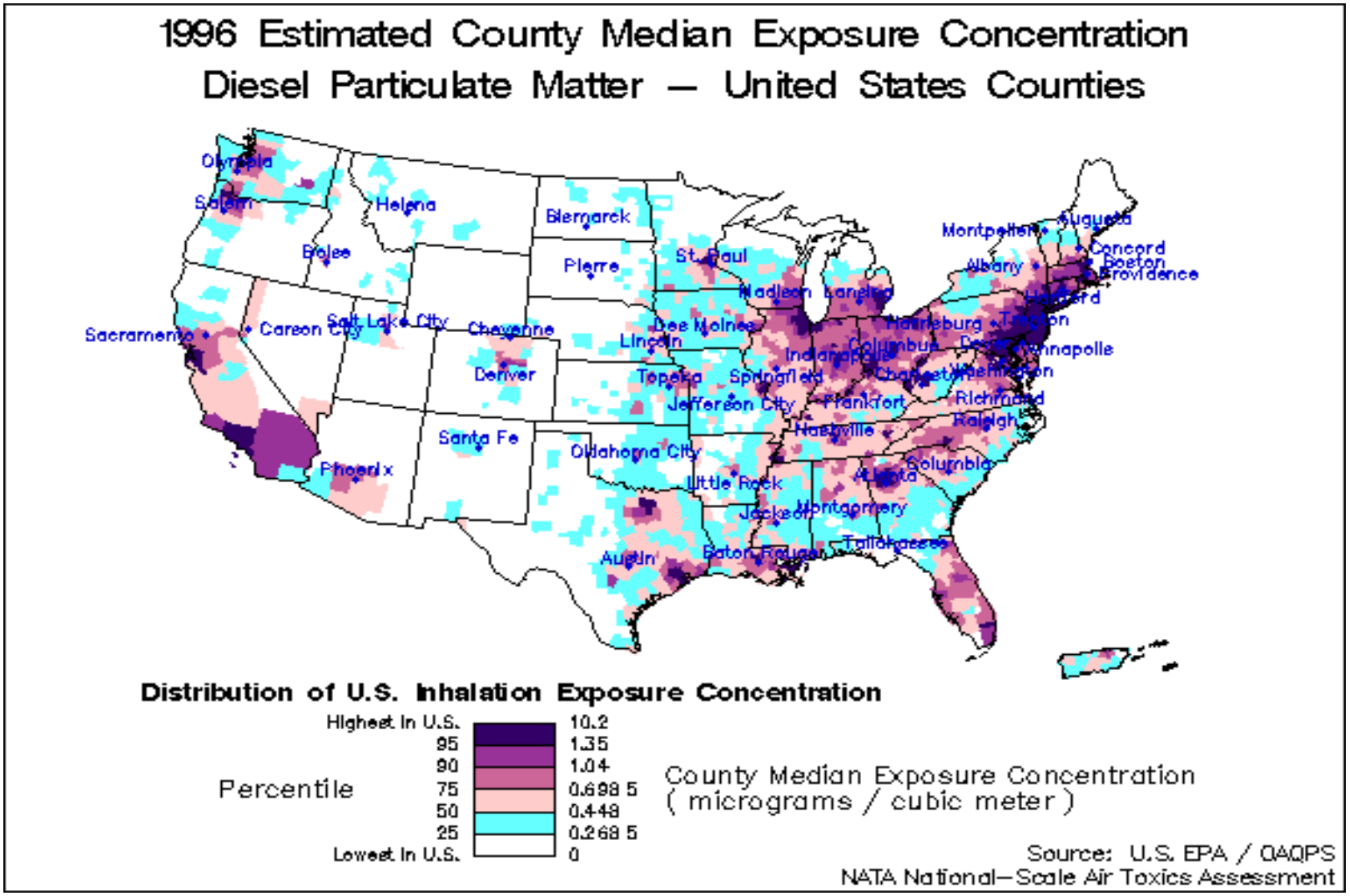
Currently, personal exposure monitors for PM cannot differentiate diesel from other PM. Thus, we use modeling to estimate exposures. Specifically, exposures for the general population are estimated by first conducting dispersion modeling of both on-highway and nonroad diesel emissions, described above, 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.[165] 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.

As explained earlier, the fact that these levels are below the 5 ug/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. In other words, all of the health effects cited in the implementation of the $PM_{2.5}$ NAAQS apply to diesel PM.

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 onroad rather than nonroad diesels, there are many similarities between on-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 (CARB) has measured elemental carbon near the Long Beach Freeway in 1993.[166] Levels measured ranged from 0.4 to 4.0 $\mu g/m^3$ (with one value as high as 7.5 ug/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 diesels.

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). For some of these pollutants, nonroad diesel engine emissions are believed to account for a significant proportion of total nationwide emissions. 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. Mobile sources contribute significantly to total emissions of these air toxics. As discussed later in this section, this proposed rulemaking will result in significant reductions of these emissions.

Nonroad engines are major contributors to nationwide cancer risk from air toxic pollutants, as indicated by the NATA 1996.[167] In fact, this study and the National Toxics Inventory (NTI) for 1996 are used throughout this section for toxics inventory information for nonroad sources.[168] Also, a supplemental paper provides more detail on nonroad diesel.[169] In addition, a paper published by the Society of Automotive Engineers gives future projections to 2007 for these air toxics.[170] 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.[171] 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.[172] The EPA proposed rulemaking will result in reductions of 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

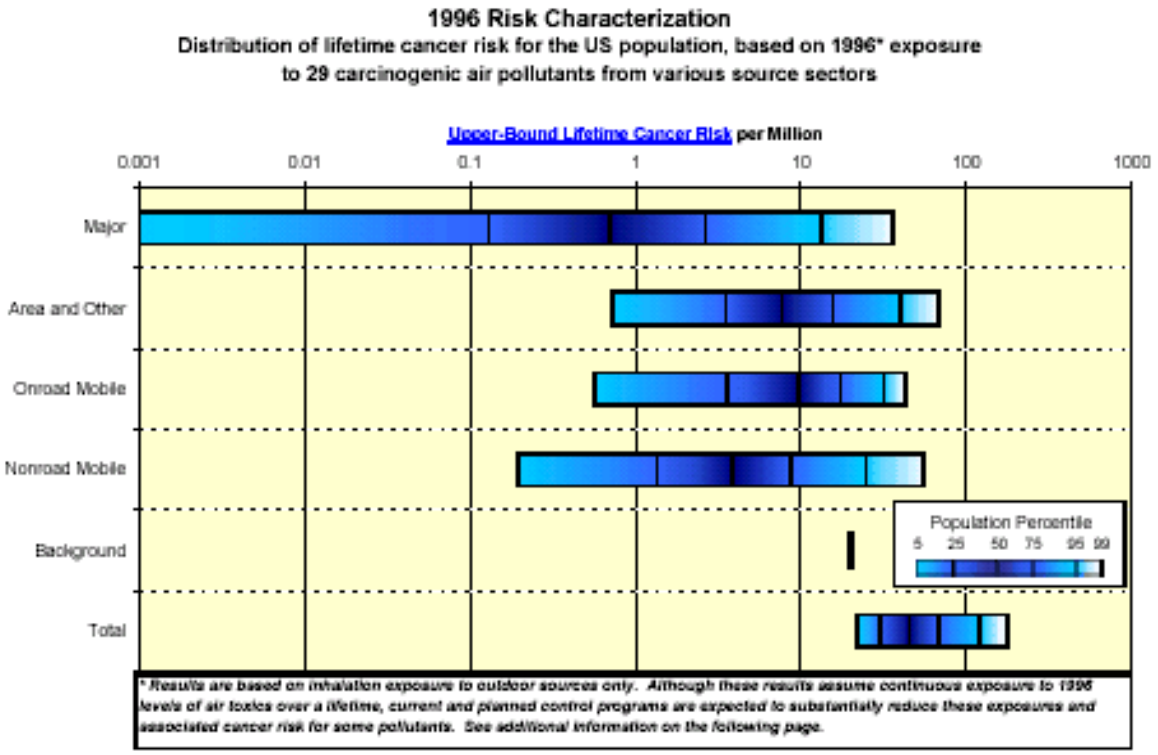
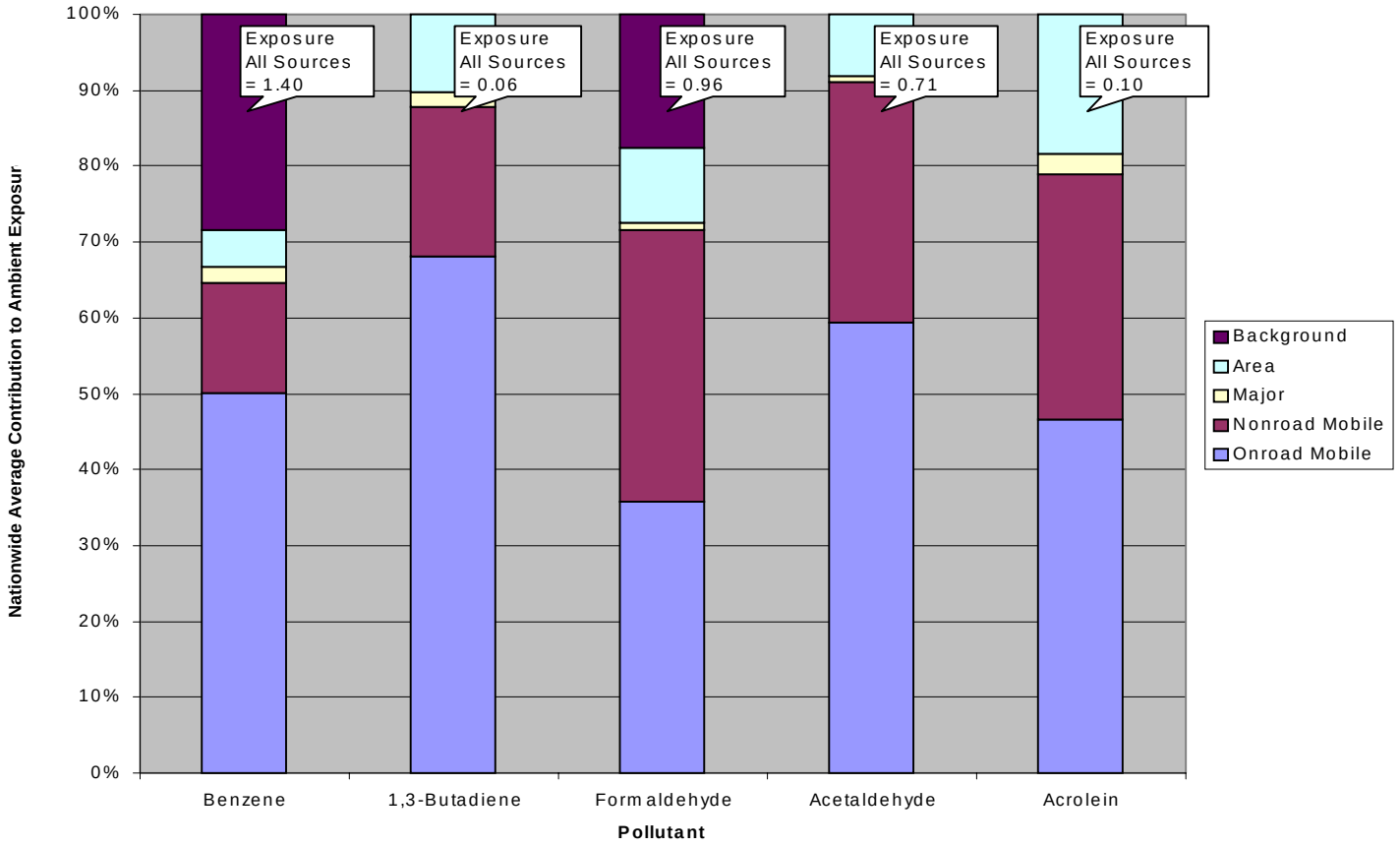

1996 Risk Characterization
Distribution of lifetime cancer risk for the US population, based on 1996* exposure
to 29 carcinogenic air pollutants from various source sectors
Upper-Bound Lifetime Cancer Risk per Million
0.001
0.01
0.1
1
10
100
1000
Major
Area and Other
Onroad Mobile
Nonroad Mobile
Background
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



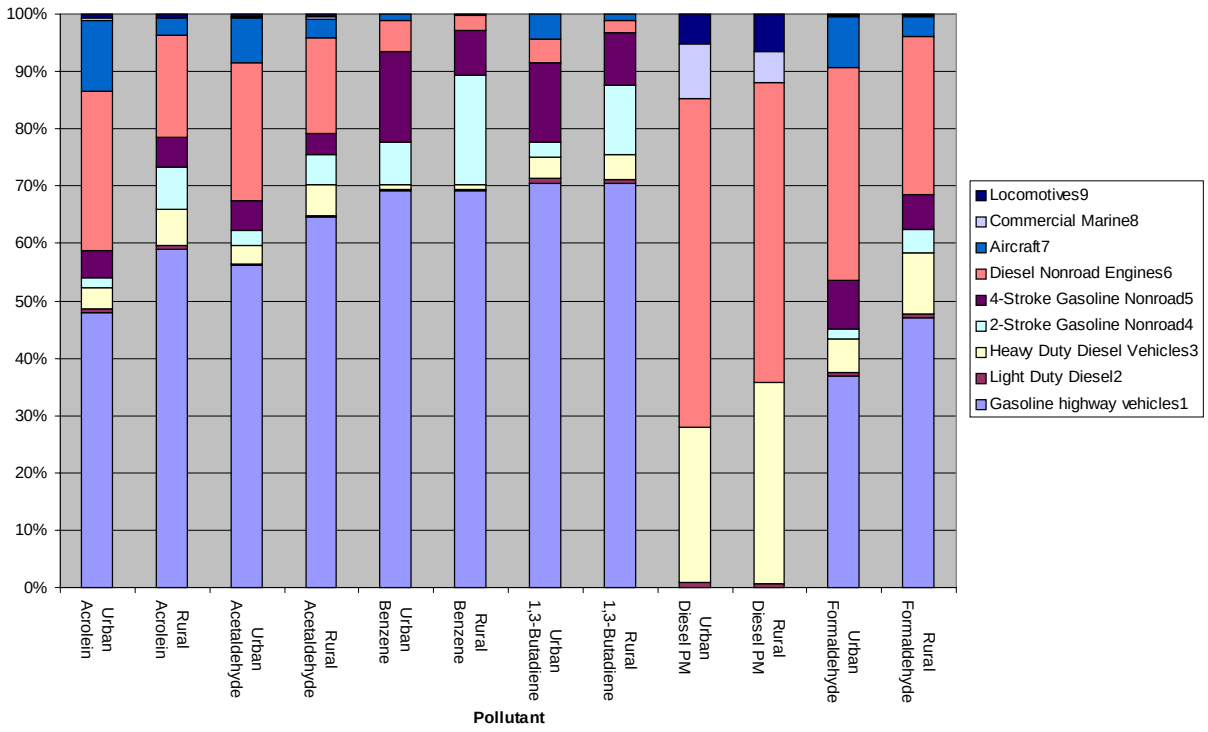
Source: National Scale Air Toxics Assessment.

2.2.2.1 Benzene

Benzene is an aromatic hydrocarbon which 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.[173, 174] 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.[175]

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).

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



The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia at high, prolonged air exposures) by all routes of exposure.[176] It is associated with additional health effects including genetic changes in humans and animals and increased proliferation of bone marrow cells in mice.[177, 178] EPA states in its IRIS database that the data indicate a causal relationship between benzene exposure and acute lymphocytic leukemia and suggest a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. Respiration is the major source of human exposure and at least half of this exposure is attributable to gasoline vapors and automotive emissions. A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with low-dose, long-term exposure to benzene.

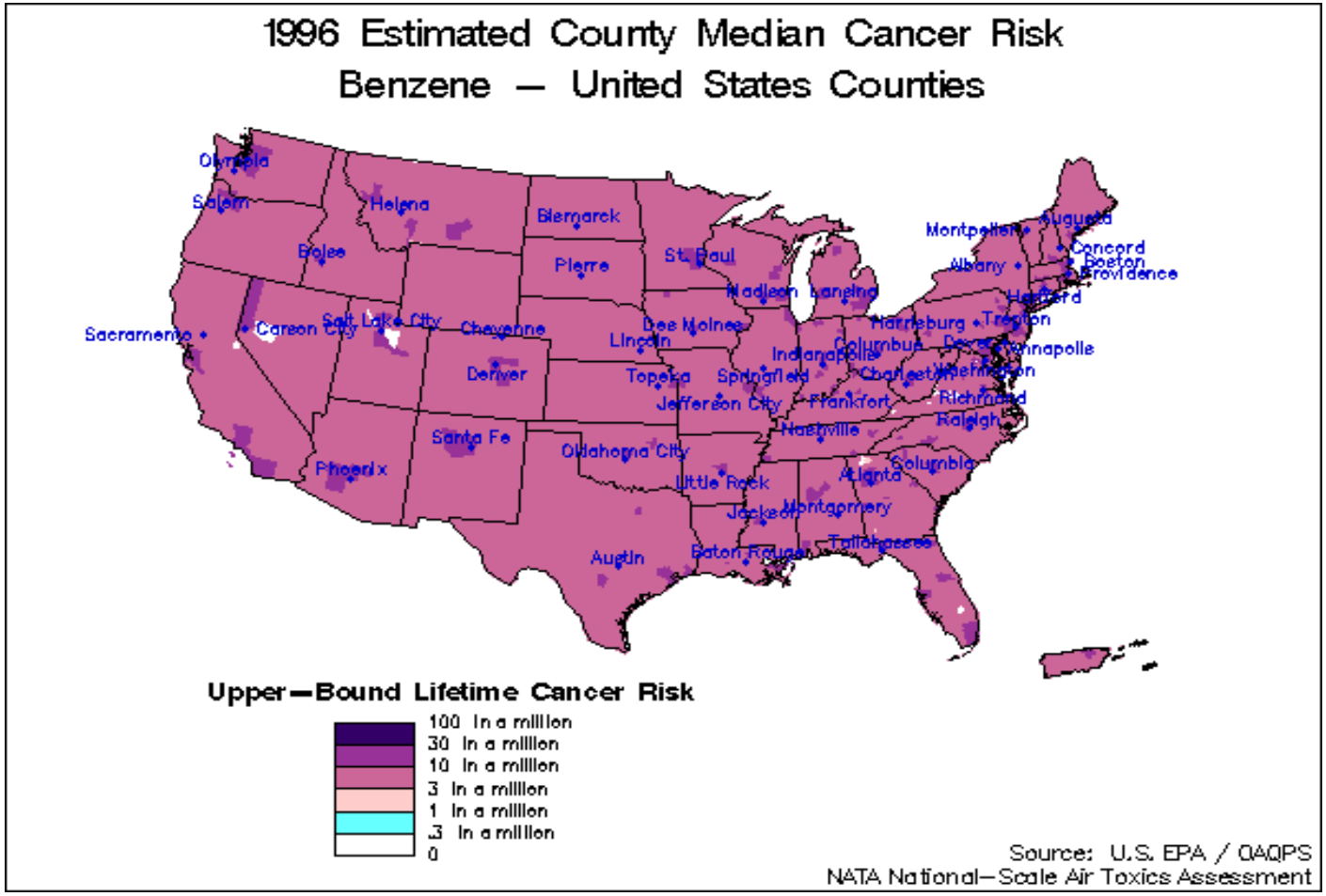
Respiration is the major source of human exposure to benzene. Long-term respiratory exposure to high levels of ambient benzene concentrations has been shown to cause cancer of the

tissues that form white blood cells. Among these are acute nonlymphocytic leukemia,[H] chronic lymphocytic leukemia and possibly multiple myeloma (primary malignant tumors in the bone marrow), although the evidence for the latter has decreased with more recent studies.[179,180] Leukemias, lymphomas, and other tumor types have been observed in experimental animals exposed to benzene by inhalation or oral administration. Exposure to benzene and/or its metabolites has also been linked with genetic changes in humans and animals[181] and increased proliferation of mouse bone marrow cells.[182] The occurrence of certain chromosomal changes in individuals with known exposure to benzene may serve as a marker for those at risk for contracting leukemia.[183]

The latest assessment by EPA places the excess risk of developing acute nonlymphocytic leukemia at 2.2×10^{-6} to $7.7 \times 10^{-6}/\mu g/m^3$. In other words, there is a risk of about two to eight excess acute nonlymphocytic leukemia cases in one million people exposed to 1 $\mu g/m^3$ over a lifetime (70 years).[184] This range of unit risk represents the maximum likelihood estimate of risk. Figure 2.2.2-4 depicts the distribution of upper bound lifetime cancer risk from inhalation of benzene from ambient sources, based on average population exposure, from the 1996 NATA Assessment. Upper bound cancer risk is above 10 in a million across the entire U.S. EPA projects a median nationwide reduction in ambient concentrations of benzene from mobile sources of about 46percent 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.

[H]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 2.2.2-4
Distribution of Upper Bound Lifetime Cancer Risk from Inhalation of
Benzene from Ambient Sources, Based on Average Population Exposure



Source: 1996 NATA Assessment.

A number of adverse noncancer health effects, blood disorders such as preleukemia and aplastic anemia, have also been associated with low-dose, long-term exposure to benzene.[185, 186] People with long-term exposure to benzene may experience harmful effects on the blood-forming tissues, especially the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components, such as red blood cells and blood platelets, leading to anemia (a reduction in the number of red blood cells), leukopenia (a reduction in the number of white blood cells), or thrombocytopenia (a reduction in the number of blood platelets, thus reducing the ability for blood to clot). Chronic inhalation exposure to benzene in humans and animals results in pancytopenia,[I] a condition characterized by decreased numbers of circulating erythrocytes (red blood cells), leukocytes (white blood cells), and thrombocytes (blood platelets).[187,188] Individuals that develop pancytopenia and have continued exposure to benzene may develop aplastic anemia,[J] whereas others exhibit both pancytopenia and bone marrow hyperplasia (excessive cell formation), a condition that may indicate a preleukemic state.[189 190] The most sensitive noncancer effect observed in humans is the depression of absolute lymphocyte counts in the circulating blood.[191]

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 U.S. Of ambient butadiene levels due to mobile

[I]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.

[J]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).

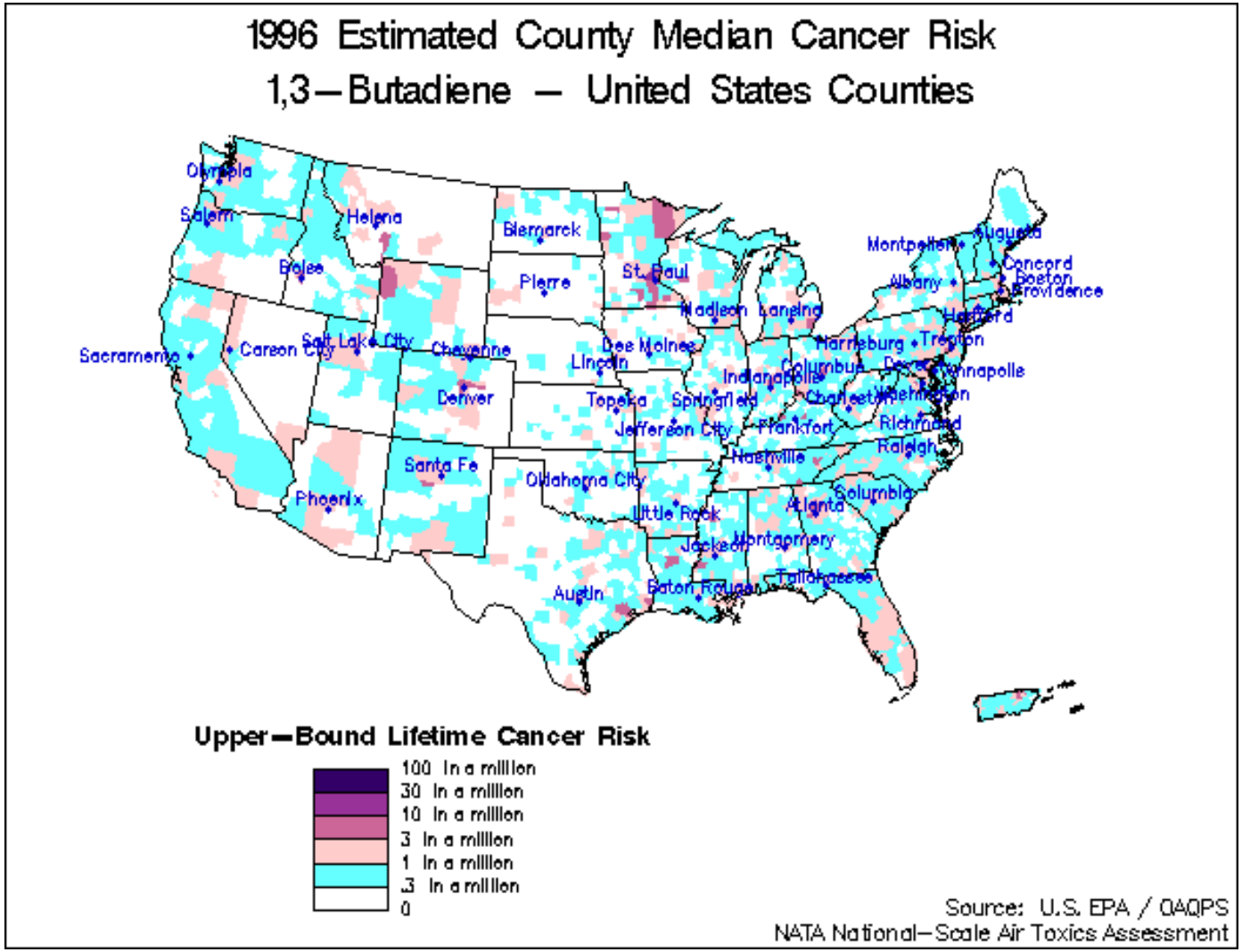
sources, 4 percent in urban and 2 percent in rural areas come from nonroad diesel (see Figure 2.2.2-3).

EPA earlier identified 1,3-butadiene as a probable human carcinogen in its IRIS database.[192] Recently EPA redesignated 1,3-butadiene as a known human carcinogen.[193,194,195] The specific mechanisms of 1,3-butadiene-induced carcinogenesis are unknown. However, it is virtually certain 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 unit cancer risk estimate is 0.08/ppm or 3x10-5 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 $\mu g/m^3$ 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.[196] A twofold adjustment to the epidemiologic-based unit cancer risk was 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. Figure 2.2.2-5 depicts the distribution of upper bound lifetime cancer risk from inhalation of 1,3-butadiene from ambient sources, based on average population exposure, from the 1996 NATA Assessment. Upper bound cancer risk is above 10 in a million across the entire U.S. 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.

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.[197] Based on this critical effect and the benchmark concentration methodology, an RfC (i.e., a chronic exposure level presumed to be "without appreciable risk" for noncancer effects) was calculated. This RfC for chronic health effects was 0.9 ppb.

Figure 2.2.2-5
Distribution of Upper Bound Lifetime Cancer Risk from
Inhalation of 1,3-Butadiene from Ambient Sources, Based on Average Population Exposure



Source: 1996 NATA Assessment.

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 which 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.[198] 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 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.[199, 200] 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.[201] 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.[202, 203, 204] 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.[205] Research has demonstrated that formaldehyde produces mutagenic activity in cell cultures.[206]

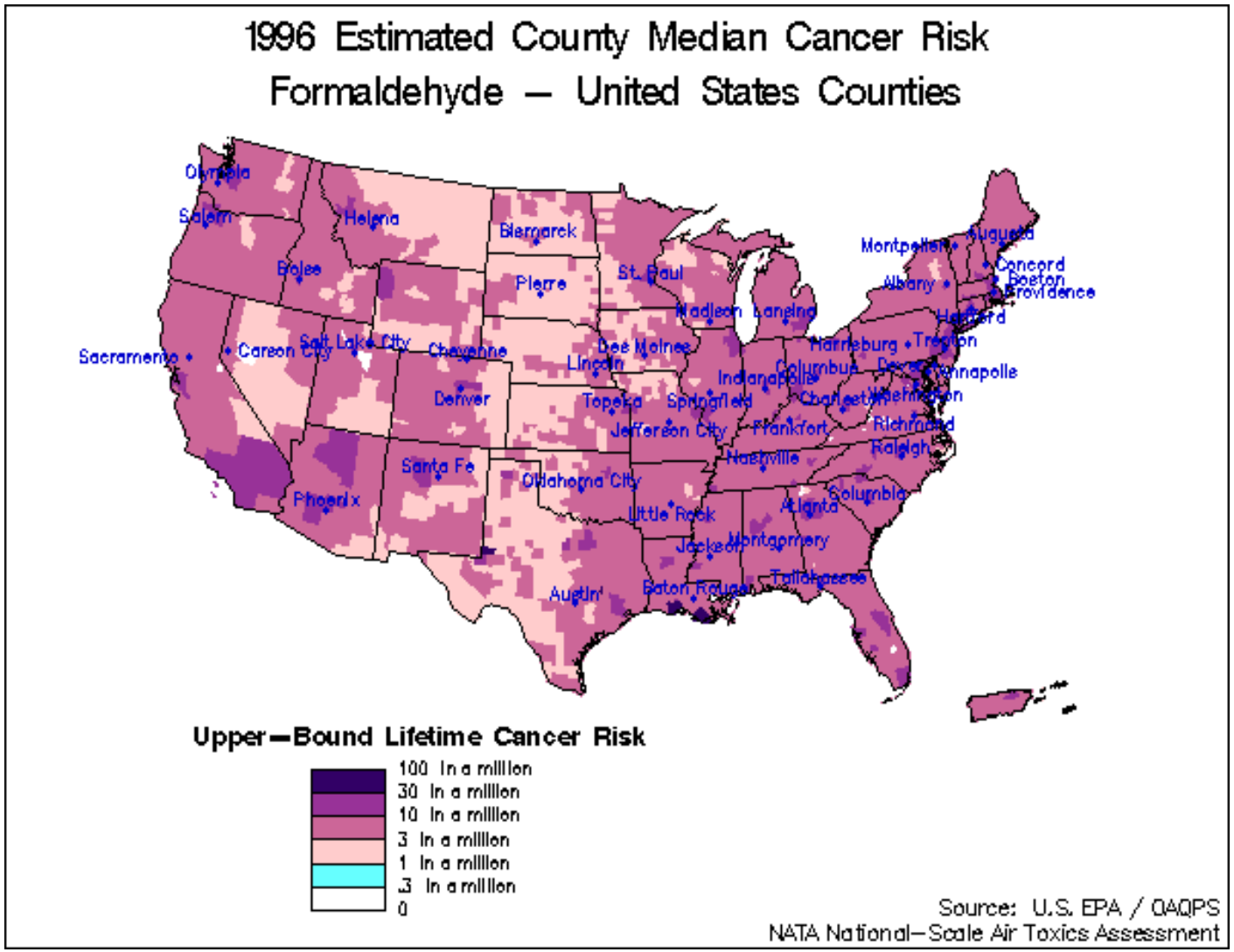
The upper confidence limit estimate of a lifetime extra cancer risk from continuous formaldehyde exposure is about $1.3 \times 10^{-5}/\mu g/m^3$. In other words, it is estimated that approximately 10 persons in one million exposed to 1 $\mu g/m^3$ formaldehyde continuously for their lifetime (70 years) would develop cancer as a result of this exposure. The agency is currently conducting a reassessment of risk from inhalation exposure to formaldehyde based on new information including a study by the Chemistry Industry Institute of Toxicology.[207, 208] Figure 2.2.2-6 depicts the distribution of upper bound lifetime cancer risk from inhalation of formaldehyde from ambient sources, based on the current unit risk and average population

exposure from the 1996 NATA Assessment. Upper bound cancer risk is above 10 in a million for more than one hundred million Americans. EPA projects a median nationwide reduction in ambient concentrations of benzene from mobile sources of about 43 percent between 1996 and 2007, as a result of current and planned control programs (Cook et al., 2002).

Formaldehyde exposure also causes a range of noncancer health effects. At low concentrations (e.g. 0.05-2.0 ppm), irritation of the eyes (tearing of the eyes and increased blinking) and mucous membranes is the principal effect observed in humans. At exposure to 1-11 ppm, other human upper respiratory effects associated with acute formaldehyde exposure include a dry or sore throat, and a tingling sensation of the nose. Sensitive individuals may experience these effects at lower concentrations. Forty percent of formaldehyde-producing factory workers reported nasal symptoms such as rhinitis (inflammation of the nasal membrane), nasal obstruction, and nasal discharge following chronic exposure.[209] In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 5 ppm.[210] Formaldehyde exposure may also cause bronchial asthma-like symptoms in non-asthmatics.[211] [212]

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.

Figure 2.2.2-6
Distribution of Upper Bound Lifetime Cancer Risk from Inhalation
of 1,3-Butadiene from Ambient Sources, Based on Average Population Exposure



Source: 1996 NATA Assessment.

2.2.2.4 Acetaldehyde

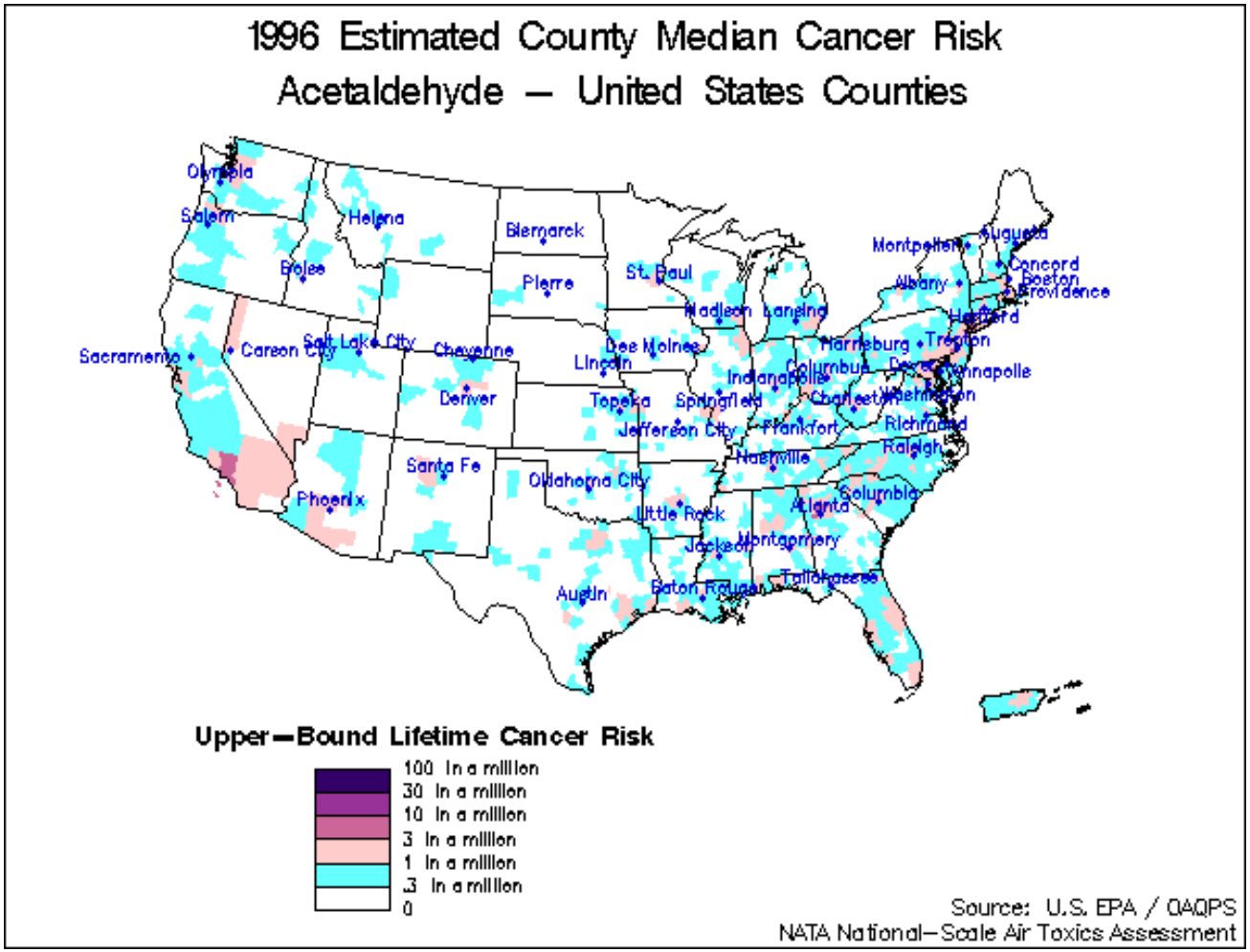
Acetaldehyde is a saturated aldehyde that is found in engine exhaust and is formed as a result of incomplete combustion of both gasoline and diesel fuel. In a recent test program which measured toxic emissions from several nonroad diesel engines, ranging from 50 to 480 horsepower, acetaldehyde consistently 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 form 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.

Acetaldehyde is classified as a probable human carcinogen. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to acetaldehyde causes an increase in the incidence of nasal squamous cell carcinomas (epithelial tissue) and adenocarcinomas (glandular tissue).[213, 214, 215, 216, 217] The upper confidence limit estimate of a lifetime extra cancer risk from continuous acetaldehyde exposure is about 2.2×10^{-6} /$\mu g/m^3$. 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. Figure 2.2.2-7 depicts the distribution of upper bound lifetime cancer risk from inhalation of formaldehyde from ambient sources, based on the current unit risk and average population exposure from the 1996 NATA. Upper bound cancer risk is above one in a million for more than one hundred million Americans. EPA projects a median nationwide reduction in ambient concentrations of benzene 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.[218] 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

Figure 2.2.2-7
Distribution of Upper Bound Lifetime Cancer Risk from Inhalation of
Acetaldehyde from Ambient Sources, Based on Average Population Exposure



Source: 1996 NATA Assessment.

acetaldehyde on reproductive and developmental effects. The in vitro and in vivo studies provide evidence to suggest that acetaldehyde may be the causative factor in birth defects observed in fetal alcohol syndrome, though evidence is very limited linking these effects to inhalation exposure. Long-term exposures should be kept below the reference concentration of 9 $\mu g/m^3$ to avoid appreciable risk of these noncancer health effects (EPA, 1988).

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.[219]

2.2.2.5 Acrolein

In a recent test program which 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.

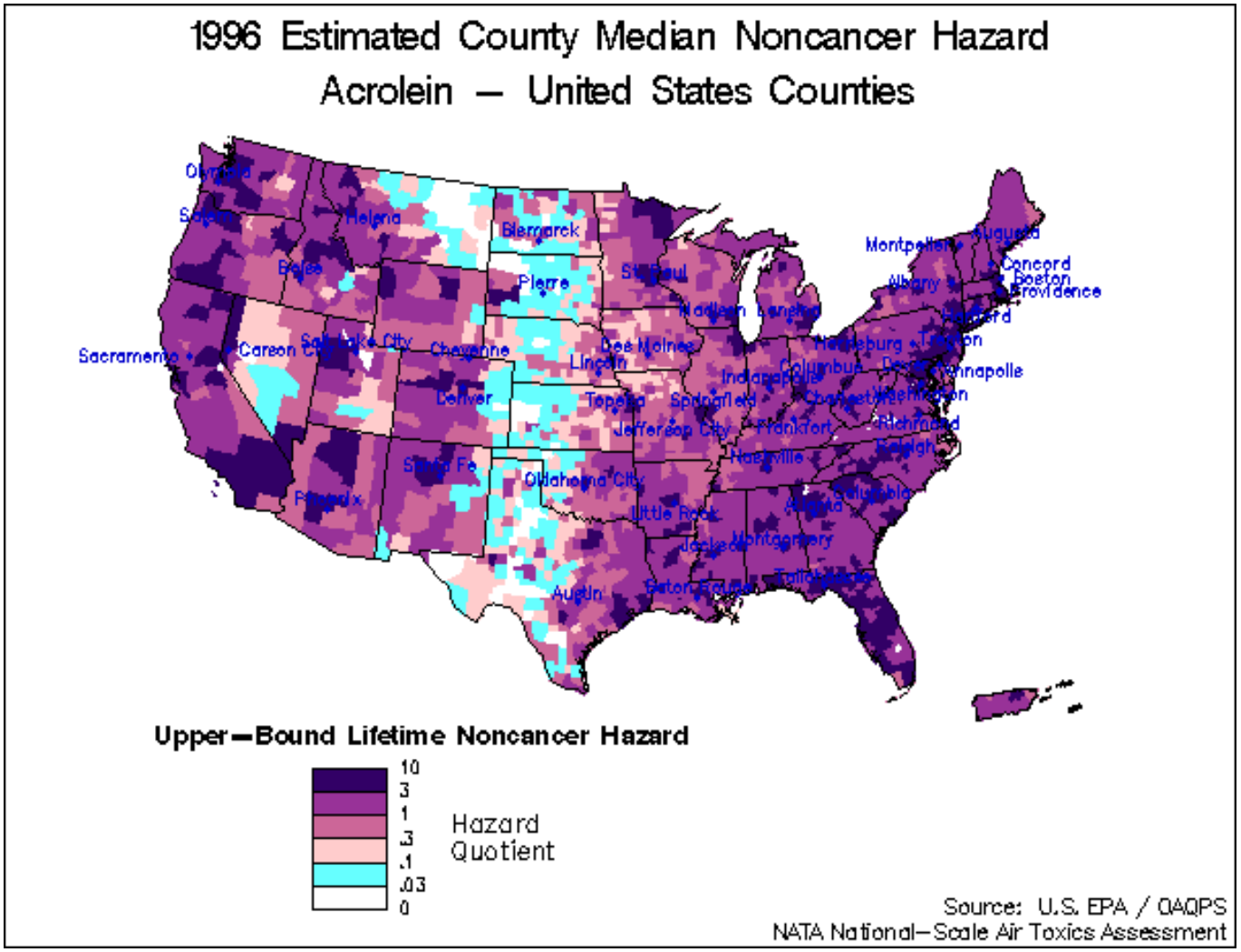
Nonroad engines account for 25 percent of nationwide emissions of acetaldehyde in 1996 with nonroad diesel accounting for about 17.5 percent based on NATA, NTI, and the supplemental information Mobile sources as a whole account for 43 percent of the total acrolein emissions in the nation. Of ambient 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 extremely toxic to humans from the inhalation route of exposure, with acute exposure resulting in upper respiratory tract irritation and congestion. The Agency developed a reference concentration for inhalation (RfC) of acrolein of 0.02 $\mu g/m^3$ in 1993. Figure 2.2.2-8 depicts the distribution of hazard quotients for acrolein across the U.S.[K] The hazard quotient is greater than one for most of the U.S. population, indicating a potential for adverse noncancer health effects.

Although no information is available on its carcinogenic effects in humans, based on laboratory animal data, EPA considers acrolein a possible human carcinogen.[220]

[K]The hazard quotient is the ratio of average ambient exposure over the reference concentration (level below which adverse health effects are not expected to occur). A hazard quotient above one indicates the potential for adverse health effects, but does not necessarily mean adverse health effects will occur.

Figure 2.2.2-8
Distribution of Noncancer Hazard Quotients for Inhalation
of Acrolein from Ambient Sources, Based on Average Population Exposure



Source: 1996 NATA Assessment.

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 on only 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.[221]

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.[222]

2.2.2.7 Dioxins

Recent studies have confirmed that dioxins are formed by and emitted from diesels (both heavy-duty diesel trucks and non-road diesels although in very small amounts) and are estimated to account for about 1 percent of total dioxin emissions in 1995. Recently EPA issued a draft assessment designating one dioxin compound, 2,3,7,8-tetrachlorodibenzo-p-dioxin as a human carcinogen and the complex mixtures of dioxin-like compounds as likely to be carcinogenic to humans using the draft 1996 carcinogen risk assessment guidelines. EPA is working on its final assessment for dioxin.[223] An interagency review group is evaluating EPA's designation of dioxin as a likely human carcinogen. These nonroad rules will have minimal impact on overall dioxin 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 on-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.[224] 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.[225, 226, 227, 228, 229, 230] Repeated exposure to ozone can make people more susceptible to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.[231, 232, 233, 234, 235] 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.[236, 237, 238, 239]

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.[240] 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.[241, 242] For example, summer camp studies in the Eastern U.S. and southeastern Canada have reported significant reductions in lung function in children who are active outdoors.[243, 244, 245, 246, 247, 248, 249, 250] 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.[251, 252, 253, 254]

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.[255] 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.[256] 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.[257, 258] 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).[259] 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.[260] 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.[261] 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

[L]In communities with high ozone (i.e., mean 8-hour 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.

conducted in Nevada in kindergarten to 6th grade [262] and in Southern California in grades 4 to 6.[263] 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 which may predispose elderly and other sensitive individuals to become more susceptible to the adverse health effects of other air pollutants, such as PM.[264, 265] 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.[266] Similarly, Thurston and Ito (1999) have reported associations between ozone and mortality.[267] 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).[268]

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 recent time-series studies of short-term exposures to air pollution and health effects.[269]

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 level of the 8-hour standard which is requiste 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 U.S. and large areas of California. Nonroad engines contribute a substantial fraction of ozone precursors in metropolitan areas.

In presenting these values, we examine concentrations in counties as well as calculating design values. An ozone design value is the concentration that determines whether a monitoring site meets the NAAQS for ozone. Because of the way they are defined, design values are determined based on 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. Thus, we follow this convention in these analyses.

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 defining the current design value of a given area, the 1999-2001 design values were chosen to provide the most recent set of air quality data for identifying areas likely to have an ozone problem in the future. The1999-2001 design values are listed in the AQ TSD, which is available in the docket to this rule.

2.3.2.1 1-Hour Ozone Nonattainment Areas and Concentrations

Currently, there are 116 million people living in 56 1-hour ozone nonattainment areas covering 233 counties. Of these, there are 1 extreme and 10 severe 1-hour ozone nonattainment areas with a total affected population of 86.5 million as shown in Table 2.3-1. We focus on these designated areas because the timing of their attainment dates relates to the timing of the proposed reductions. Five severe 1-hour ozone nonattainment areas have attainment dates of December 31, 2007. While all of these areas are expected to be in attainment before the emission reductions from this proposed rule are expected to occur, these reductions will be important to assist these areas in maintaining the standards. The Los Angeles South Coast Air Basin is designated as an extreme nonattainment area and has a compliance date of December 31, 2010. The reductions from this rule will be an important part of their overall strategy to attain and maintain the standard.

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

Nonattainment Area	Attainment Date	2000 Population (millions)	1999-2001 Measured Violation?
Los Angeles South Coast Air Basin, CA[a]	December 31, 2010[a]	14.6	Yes
Chicago-Gary-Lake County, IL-IN	December 31, 2007	8.9	No
Houston-Galveston-Brazoria, TX	December 31, 2007	4.5	Yes
Milwaukee-Racine, WI	December 31, 2007	1.7	No
New York-New Jersey-Long Island, NY-NJ-CT	December 31, 2007	20.2	Yes
Southeast Desert Modified AQMA, CA	December 31, 2007	0.5	Yes
Baltimore, MD	2005	0.8	Yes
Philadelphia-Wilmington-Trenton, PA-NJ-DE-MD	2005	6.0	Yes
Sacramento, CA	2005	1.2	Yes
San Joaquin Valley, CA	2005	7.8	Yes
Ventura County, CA	2005	0.1	Yes
Total Population	86.5 million		

[a] Extreme 1-Hour nonattainment areas. All other areas are severe nonattainment areas.

The extreme nonattainment area will need additional reductions to attain the ozone standard and will also be able to rely on additional reductions from today's proposed action in order to maintain the standard. The severe areas will be able to rely on the reductions from today's proposed action in order to maintain the standard.

The emission reductions from this proposed rule would also help these areas reach attainment at lower overall cost, with less impact on small businesses, as discussed in other chapters of this document. Following implementation of controls for regional NOx reductions, States will have already adopted emission reduction requirements for most large sources of NOx for which cost-effective control technologies are known and for which they have authority to control. Those that must adopt measures to complete their attainment demonstrations and maintenance plans, therefore, will have to consider their remaining alternatives. Many of the alternatives that areas may consider could be more costly, and the NOx emissions impact from each additional emissions source subjected to new emissions controls could be considerably smaller than the emissions impact of the standards being proposed today. Therefore, the emission reductions from the standards we are finalizing today will ease the need for States to find first-time

reductions from the mostly smaller sources that have not yet been controlled, including area sources that are closely connected with individual and small business activities. The emission reductions from nonroad diesel engines also reduce the need for States to seek even deeper reductions from large and small sources already subject to emission controls.

Each of the areas in Table 2.3-1 is adopting additional measures to address specific emission reduction shortfalls in attainment SIPs submitted for New York, Houston, the South Coast Basin, Philadelphia, and Baltimore based on the local ozone modeling and other evidence. The San Joaquin Valley will need additional reductions to attain and maintain the standards. There is some risk that New York will fail to attain the standard by 2007, and thus a transferred risk that Connecticut will also fail. A similar situation exists in Southern California, where attainment of the South Coast is a precondition of the ability of downwind to reach attainment by their respective attainment dates. Additional reductions from this rule will assist New York and Greater Connecticut, and the South Coast and its downwind nonattainment areas, in reaching the standard by each areas' respective attainment dates and maintaining the standard in the future.

The Los Angeles (South Coast Air Basin) ozone attainment demonstration is fully approved, but it is based in part on reductions from new technology measures that have yet to be identified (as allowed under CAA Section 182(e)(5)). Thus, additional reductions would be helpful to this area, as discussed in the draft plan.[270] The 2007 attainment demonstration for the Southeast Desert area is also approved. However, a transport situation exists between the Southeast Desert areas and the South Coast Air Basin, such that attainment in the Southeast Desert depends on progress in reducing ozone levels in the South Coast Air Basin.

Even if the SIPs were approved and all shortfalls were filled in an area, there would still be a risk that ozone levels in such an area could exceed the NAAQS. EPA's approval of an attainment demonstration generally indicates our belief that a nonattainment area is reasonably likely to attain by the applicable attainment date with the emission controls in the SIP. However, such approval does not indicate that attainment is certain. Moreover, no ozone forecasting is 100 percent certain, so attainment by these deadlines is not certain, even though we believe it is more likely than not. There are significant uncertainties inherent in predicting future air quality, such as unexpected economic growth, unexpected vehicle miles traveled (VMT) growth, the year-to-year variability of meteorological conditions conducive to ozone formation, and modeling approximations. There is at least some risk in each of these areas that even assuming all shortfalls are filled, attainment will not be reached by the applicable dates without further emission reductions. The Agency's mid-course review in the SIP process—as well as the Clean Air Act's provisions for contingency measures—is part of our strategy for dealing with some of these uncertainties, but does not ensure successful attainment.

Many 1-hour ozone nonattainment areas continue to experience exceedances. Approximately 51 million people are living in counties with measured air quality violating the 1-hour NAAQS

in 1999-2001.[M] See the AQ TSD for more details about the counties and populations experiencing various levels of measured 1-hour ozone concentrations.

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 proposed 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 today's proposed action will help areas to attain and maintain the 1-hour standards.

2.3.2.2 8-Hour Ozone Levels: Current and Future Concentrations

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.[271] The 8-hour standard greatly limits ozone exposures of concern for the general population and sensitive populations. This section describes the current measured 8-hour concentrations and describes our modeling to predict future 8-hour ozone concentrations.

2.3.2.2.1 Current 8-Hour Ozone Concentrations

Based upon the measured data from years 1999 - 2001, there are 291 counties with measured values that violate the 8-hour ozone NAAQS, with a population totaling 111 million, as shown in Figure 2-1. Of these, 61 million people live in counties that meet the 1-hour standard but violate the 8-hour standard. There may be additional areas above the level of the NAAQS for which no monitoring data are available.

An additional 37 million people live in 155 counties that have air quality measurements within 10 percent of the level of the standard. These areas, though currently not violating the standard, will also benefit from the emission reductions from this proposed rule.

Approximately 48 million people lived in counties with at least a week (7 days) of 8-hour ozone concentrations measurements at or above 0.085 ppm in 2000. Approximately 8 million people lived in counties experiencing 20 days and 4 million experienced 40 days of 8-hour ozone concentrations at or above 0.085 ppm in 2000. See the AQ TSD for more details about the

[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 are experiencing recent exceedances, as indicated in the table. Further, the analysis is limited to areas with monitors.

counties and populations experiencing various levels of measured 8-hour ozone concentrations.

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 proposal, we modeled a preliminary control scenario which illustrates the likely reductions from our proposal. 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 process based on our best judgement at that time. As additional data regarding technical feasibility and other factors became available, our judgement 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 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. For the final rule, considering public comment, we plan to model the final control scenario. This proposed 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 U.S. using Comprehensive Air Quality Model with Extension (CAMx). The model simulations were performed for five emissions 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 which will require additional emission reductions to attain and maintain the ozone NAAQS. The impacts of the proposed controls were determined by comparing the model results in the future year control

[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 emissions changes relate to what was modeled.

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.

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 U.S. for the various emissions 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/non-attainment as well as estimating the ozone reductions expected to occur from a reduction in emitted pollutants. The following sections provide an overview of the ozone modeling completed as part of this rulemaking. More detailed information is included in the 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 on-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 U.S., 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 U.S. 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 U.S.: June 12-24, July 5-15, and August 7-21. Two multi-day meteorogical scenarios during the summer of 1996 were used in the model simulations over the western U.S.: 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 emissions 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 U.S., 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 U.S., 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 on-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 which adequately replicates observed pollutant concentrations in the base year can be used to assess the effects of future year emissions 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 U.S. as shown in Table 2.3-2. When all hourly observed ozone values greater than a 60 ppb threshold are compared to 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 2.3-2.
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 proposal 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/economic benefits due to the proposed emissions reductions.

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

This next section summarizes the results of our modeling of ozone air quality impact 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, which are both 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 emissions 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.[272] 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 273] 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 proposal, 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 proposed nonroad diesel reductions), 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.[274] 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 without the nonroad diesel emissions reductions proposed today 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

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

attainment with the ozone 8-hour NAAQS. EPA's current plans call for designating ozone 8-hour nonattainment areas in April 2004. EPA is planning to propose that States submit SIPs that address how areas will attain the 8-hour ozone standard within three years after nonattainment designation regardless of their classification. EPA is also planning to propose that certain SIP components, such as those related to reasonably available control technology (RACT) and reasonable further progress (RFP) be submitted within 2 years after designation. We therefore anticipate that States will submit their attainment demonstration SIPs by April 2007. Section 172(a)(2) of the Clean Air Act requires that SIP revisions for areas that may be covered only under subpart 1 of part D, Title I of the Act demonstrate that the nonattainment areas will attain the ozone 8-hour standard as expeditiously as practicable but no later than five 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 these provisions, we expect that most or all areas covered under subpart 1 will attain the ozone standard in the 2007 to 2014 time frame. For areas covered under subpart 2, the maximum attainment dates will range from 3 to 20 years after designation, depending on an area's classification. Thus, we anticipate that areas covered by subpart 2 will attain in the 2007 to 2014 time period.

Furthermore, the inventories that underlie the ozone modeling conducted for this rulemaking included reductions from all current or committed federal, State and local controls and, for the control case, the proposed nonroad diesel program itself. It did not did not attempt to examine the prospects of areas attaining or maintaining the ozone standard with possible future controls (i.e., controls beyond current or committed federal, State and local controls). Therefore, Tables 2.2-3 and 2.2-4 below should be interpreted as indicating what areas 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 2.2-3 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 today's proposal would begin in the same time period in which areas will need reductions to attain by their attainment dates, the projected reductions in nonroad emissions would be extremely important to States in meeting the new NAAQS. It is our expectation that States will be relying on such nonroad reductions in order 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 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 non-attaining 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 proposal, but they could substantially change the count 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 today's proposal might change the actual attainment status of very few counties. Bearing this limitation in mind, our modeling indicates that the nonroad diesel emissions reductions would decrease the net number of nonattainment counties by 2 in 2020 and by 4 in 2030, without consideration of new state programs.

Areas presented in Table 2.3-3 and 2.3-4 have monitored 1999-2001 air quality data indicating violations of the 8-hour ozone NAAQS, or are within 10 percent of the standard, and are predicted to have exceedances in 2020 or 2030 without the reductions from this rule. Table 2.3-3 lists those counties with predicted exceedances of the 8-hour ozone standard in 2020 or 2030 without emission reductions from this rule (i.e., base cases). These areas are listed in columns with a "b" after the year (e.g., 2020b). Table 2.3-2 also lists those counties with predicted exceedances of the 8-hour ozone standard in 2020 and 2030, with emission reductions from this rule (i.e., control case). These areas are listed in columns with a "c" after the year (e.g., 2020c). An area was considered likely to have future exceedances if exceedances were predicted by the model, and the area is currently violating the 8-hour ozone standard, or is within 10 percent of violating the 8-hour ozone standard.

In Table 2.3-3 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 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 ambient monitoring data sufficient to calculate current design values.

[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 the proposed rule is positive.

Table 2.3-3: 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 proposed emission reductions differs 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 2.3-4 we present the counties with 2020 and 2030 projected 8-hour ozone 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 to 77 ppb, but less 85 ppb. Counties are marked with a "V" in the table if their projected design values are greater than or equal to 85 ppb. The current 3-year average 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 proposed rule will help assure that these counties continue to meet the standard.

Table 2.3-4
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 Rou	91	X	X	X	X	412,852
LA	Iberville	86	X		X		33,320
LA	Jefferson	89	X	X	X	X	455,466
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

State	County	1999 - 2001 Design Value (ppb)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
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
PA	Philadelphia	88	X	X	X	X	1,517,550
RI	Kent	94	X	X	X		167,090

State	County	1999 - 2001 Design Value (ppb)	2020		2030		Population in 2000
			Base	Control[a]	Base	Control[a]	
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 proposed emission reductions differs 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.

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 0.085 to 0.12 ppm 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.

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 0.085 ppm. We then grouped the counties which had days with ozone in this range according to the number of days this was predicted to happen, and summed their projected populations.

In the base case (i.e., before the application of emission reductions resulting from this rule),

we estimated that in 2020 53 million people are predicted to live in counties with at least 2 days with 8-hour average concentrations of 0.085 ppm 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 0.085 ppm or higher. About 30 million people live in counties with at least 7 days of 8-hour ozone concentrations at or above 0.085 ppm 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 0.085 ppm in 2020 and 2030 without additional controls. 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 Emissions Reductions

While the proposed rule would 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 emissions 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.[275]

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.[276, 277] There are other hypotheses for the cause of the "weekend effect."[278] 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 emissions 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.

EPA maintains that the best available approach for determining the value of a particular emissions 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[279]. Model scenarios of 2020 and 2030 with and without the proposed controls are compared to determine the expected changes in future pollutant levels resulting from the proposed rule. There are several factors related to the air quality modeling and inputs which should be considered regarding the disbenefit issue. First, our future year modeling conducted does not contain any local governmental actions beyond the controls proposed 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 proposal (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 CARB 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 the assessment of the proposed emissions reductions. Three of the most important assessments are: 1) the effect of the proposed rule on projected future-year ozone violations, 2) the effect of the proposed 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 today's 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 the proposed controls is projected to be strongly positive due to the expected reductions in fine PM.

The net impact of the proposed rule on projected 8-hour ozone violations in 2020 is that three counties would no longer violate the NAAQS[280]. 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. 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 ($\geq$ 85 ppb).
[c] Counties whose present-day design values were less than but within 10 percent of the 8-hour standard ($77 \leq DV < 85$ ppb).
[d] Counties whose present-day design values exceeded the 1-hour standard ($\geq$ 125 ppb).
[e] Counties whose present-day design values were less than but within 10 percent of the 1-hour standard ($112 \leq DV < 125$ ppb) in 2001.
[f] The proposal 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 proposal 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 which are 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/national ozone levels[1]. Consistent with that fact, the photochemical modeling completed for this rule indicates that the emissions reductions proposed today 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 emissions reductions that States, local governments, and Tribes need to reach their clean air goals. It is expected that future 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 support the rule because they believe the resulting NOx reductions would reduce both ozone and PM[281]. EPA believes that a balanced air

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

Another category of potential effects that may change in response to ozone reduction strategies results from the shielding provided by ozone against the harmful effects of ultraviolet radiation (UV-B) derived from the sun. The great majority of this shielding results from naturally occurring ozone in the stratosphere, but the 10 percent of total "column"ozone present in the troposphere also contributes.[282] A variable portion of this tropospheric fraction of UV-B shielding is derived from ground level ozone related to anthropogenic air pollution. Therefore, strategies that reduce ground level ozone could, in some small measure, increase exposure to UV-B from the sun.

While it is possible to provide quantitative estimates of benefits associated with globally based strategies to restore the far larger and more spatially uniform stratospheric ozone layer, the changes in UV-B exposures associated with ground level ozone reduction strategies are much more complicated and uncertain. Comparatively smaller changes in ground-level ozone (compared to the total ozone in the troposphere) and UV-B are not likely to measurably change long-term risks of adverse effects.

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.[283] Because the proposed rule would reduce ground-level ozone and nitrogen deposition, benefits are expected to accrue to the welfare effects categories described in the paragraphs (subsections) below.

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."[284] 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.[285] 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.[286] [287] 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.[288] 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.[289]

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).[290] [291] [292] 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.[293] 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.[294] [295] In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts.[296] Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function.[297] [298] [299]

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.[300] 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.[301] [302] [303] 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,[304] welfare benefits are also expected to accrue as a result of reductions in ambient ozone concentrations in the U.S. is the economic value the public receives from reduced aesthetic injury to forests.[305] 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 U.S."[306] In addition, economic studies have shown a relationship between observed ozone levels and crop yields.[307] [308] [309]

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 economic benefits 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.[310] This is therefore a potentially important welfare effects category. However, information and valuation 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. The proposed rule, by reducing NO_X emissions, 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 deposition of nitrogen could have negative effects on forest and vegetation growth in ecosystems where nitrogen is a limiting factor.[311]

On the other hand, there is evidence that forest ecosystems in some areas of the United States are already or are becoming nitrogen saturated.[312] 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.[313]

The reductions in ground-level ozone and nitrogen deposition that would result from the proposed rule would be 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, would be reduced.

2.4 Carbon Monoxide

The standards being proposed today would also help 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 November 4, 2002, there were 24 areas designated as non-attainment with the SO2 standard, and 14 designated CO non-attainment 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 non-attainment area (65 FR 76790, December 7, 2000). In that action EPA found that engines subject to this proposed rule contribute to CO non-attainment in areas such as Los Angeles, Phoenix, Spokane, Anchorage, and Las Vegas. Nonroad land-based diesel engines emitted 927,500 tons of CO in 1996 (1 percent of mobile source CO). Thus, nonroad diesel engines contribute to CO non-attainment 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 non-attainment areas. Thus, the emissions benefits from this proposed 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.[314, 315] 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.[316] The subsequent hypoxia in brain tissue then produces behavioral effects, including decrements in continuous performance and reaction time.[317]

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.[318] Persons with heart disease are especially sensitive to carbon monoxide poisoning and may experience chest pain if they breathe the gas while exercising.[319] 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.[320]

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 U.S. 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.[321]

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 22 million people currently live in the 13 non-attainment 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.[322] Over the next decade, we would expect there to be a minor decreasing trend from the highway segment due primarily to the more stringent standards for certain light-duty trucks.[323] 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 explained in Chapter 9, EPA currently does not have appropriate tools for modeling changes in ambient concentrations of CO or air toxics for input into a national benefits analysis. As noted above, CO has been linked to numerous health effects; however, we are unable to quantify the CO-related health or welfare benefits of the Nonroad Diesel Engine rule at this time. However, nonroad diesel engines do contribute to nonattainment in some areas. Thus, the emissions benefits from this proposed rule would help areas to attain and maintain the CO NAAQS.

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