Control of Emissions from Marine SI and Small SI Engines, Vessels, and Equipment

Draft Regulatory Impact Analysis

Chapter 2
Air Quality, Health, and Welfare Concerns

Assessment and Standards Division
Office of Transportation and Air Quality
U.S. Environmental Protection Agency

United States
Environmental Protection
Agency

EPA420-D-07-004
April 2007

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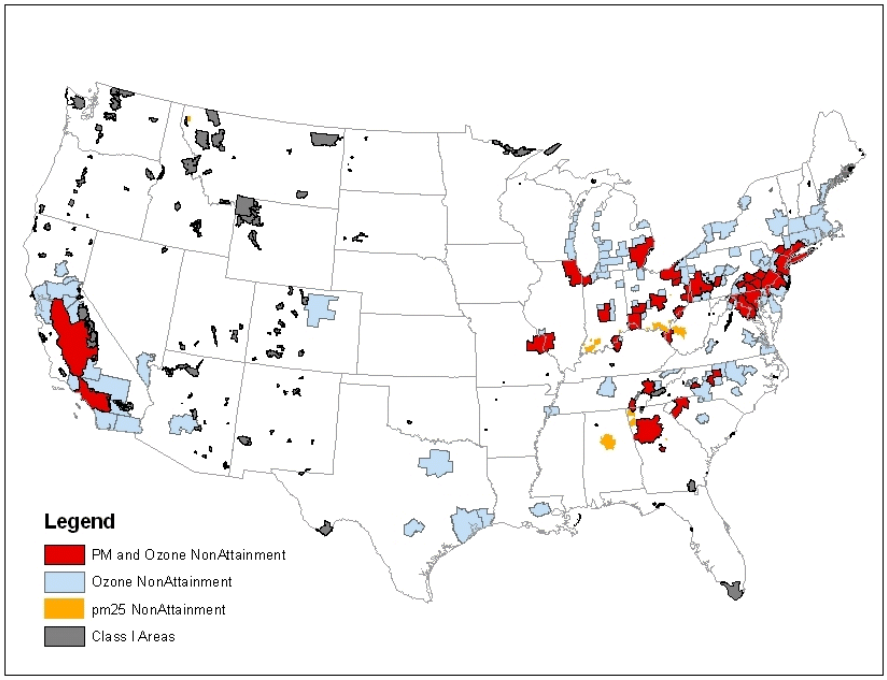
The proposed standards would reduce emissions of hydrocarbons (HC), oxides of nitrogen (NOx), carbon monoxide (CO) and air toxics from the engines, vessels and equipment subject to this proposal. These pollutants contribute to ozone, PM and CO nonattainment and to adverse health effects associated with air toxics. The emissions from these engines, vessels and equipment can also impact health through personal exposure and contribute to adverse environmental effects including visibility impairment both in mandatory class I federal areas and in areas where people live, work and recreate.

The health and environmental effects associated with emissions from Small SI engines and equipment and Marine SI engines and vessels are a classic example of a negative externality (an activity that imposes uncompensated costs on others). With a negative externality, a activity's social cost (the cost on society imposed as a result of the activity taking place) exceeds its private cost (the cost to those directly engaged in the activity). In this case, as described in this chapter, emissions from Small SI engines and equipment and Marine SI engines and vessels impose public health and environmental costs on society. The market system itself cannot correct this externality. The end users of the equipment and vessels are often unaware of the environmental impacts of their use for lawn care or recreation. Because of this, consumers fail to send the market a signal to provide cleaner equipment and vessels. In addition, producers of these engines, equipment, and vessels are rewarded for emphasizing other aspects of these products (e.g., total power). To correct this market failure and reduce the negative externality, it is necessary to give producers social cost signals. The standards EPA is proposing will accomplish this by mandating that Small SI engines and equipment and Marine SI engines and vessels reduce their emissions to a technologically feasible limit. In other words, with this proposed rule the costs of the services provided by these engines and equipment will account for social costs more fully.

In this Chapter we will discuss the impacts of the pollutants emitted by Small SI engines and equipment and Marine SI engines and vessels on health and welfare, National Ambient Air Quality Standard (NAAQS) attainment and personal exposure. Air quality modeling and monitoring data presented in this chapter indicate that a large number of our citizens continue to be affected by these emissions. Figure 2-1 illustrates the widespread nature of these problems. Shown in this figure are counties designated as nonattainment for either or both of the 8-hour ozone or $PM_{2.5}$ NAAQS, also depicted are the mandatory class I federal areas. The emission standards proposed in this rule would help reduce HC, NOx, air toxic and CO emissions and their associated health and environmental effects.

Figure 2-1: 8-Hour Ozone and $PM_{2.5}$ Nonattainment Areas and Mandatory Class I Federal Areas



2.1 Ozone

In this section we review the health and welfare effects of ozone. We also describe the air quality monitoring and modeling data which indicates that people in many areas across the country continue to be exposed to high levels of ambient ozone and will continue to be into the future. Emissions of volatile organic compounds (VOCs) and NOx from the engines, vessels and equipment subject to this proposed rule contribute to these ozone concentrations. Information on air quality was gathered from a variety of sources, including monitored ozone concentrations, air quality modeling forecasts conducted for this rulemaking, and other state and local air quality information.

2.1.1 Science of Ozone Formation

Ground-level ozone pollution is formed by the reaction of VOCs, of which HC are the major subset, and 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 such as highway and nonroad motor vehicles (including those subject to this proposed rule), power plants, chemical plants, refineries, makers of consumer and commercial products, industrial facilities, and smaller area sources.

The science of ozone formation, transport, and accumulation is complex.[1] Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions, many of which are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up and result in more ozone than typically would occur on a single high-temperature day. 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.

The highest levels of ozone are produced when both VOC and NOx emissions are present in significant quantities on clear summer days. 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.

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 (NO) with ozone, forming nitrogen dioxide (NO_2); as the air moves downwind and the cycle continues, the NO_2 forms additional ozone. The importance of this reaction depends, in part, on the relative concentrations of NOx, VOC, and ozone, all of which change with time and location.

2.1.2 Health Effects of Ozone Pollution

Exposure to ambient ozone contributes to a wide range of adverse health effects.[1] These health effects are well documented and are critically assessed in the EPA ozone air quality criteria document (ozone AQCD) and EPA staff paper.[2,3] We are relying on the data and conclusions in the ozone AQCD and staff paper, regarding the health effects associated with ozone exposure.

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

Ozone-related health effects include lung function decrements, respiratory symptoms, aggravation of asthma, increased hospital and emergency room visits, increased asthma medication usage, inflammation of the lungs, and a variety of other respiratory effects. There is also evidence that ozone may contribute to cardiovascular health effects. People who are more susceptible to effects associated with exposure to ozone include children, asthmatics and the elderly. There is also suggestive evidence that certain people may have greater genetic susceptibility. Those with greater exposures to ozone, for instance due to time spent outdoors (e.g., outdoor workers) are also of concern.

Based on a large number of scientific studies, EPA has identified several key health effects associated with exposure to levels of ozone found today in many areas of the country. Short-term (1 to 3 hours) and prolonged exposures (6 to 8 hours) to higher ambient ozone concentrations have been linked to lung function decrements, respiratory symptoms, increased hospital admissions and emergency room visits for respiratory problems.[4, 5, 6, 7, 8, 9] Repeated exposure to ozone can increase susceptibility to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.[10, 11, 12, 13, 14] Repeated exposure to sufficient concentrations of ozone can also cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure, which over time could lead to premature aging of the lungs and/or chronic respiratory illnesses, such as emphysema and chronic bronchitis.[15, 16, 17, 18]

Children and adults who are outdoors and active during the summer months, such as construction workers and other outdoor workers, are among those most at risk of elevated ozone exposures.[19] Children and outdoor workers tend to have higher ozone exposures because they typically are active outside, working, playing and exercising, during times of day and seasons (e.g., the summer) when ozone levels are highest.[20] For example, summer camp studies in the Eastern United States and Southeastern Canada have reported significant reductions in lung function in children who are active outdoors.[21, 22, 23, 24, 25, 26, 27, 28] Further, children are more at risk of experiencing health effects from ozone exposure than adults because their respiratory systems are still developing. These individuals (as well as people with respiratory illnesses such as asthma, especially asthmatic children) can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during prolonged periods of moderate exertion.[29, 30, 31, 32]

EPA typically quantifies ozone-related health impacts in its regulatory impact analyses (RIAs) when possible. In the analysis of past air quality regulations, ozone-related benefits have included morbidity endpoints and welfare effects such as damage to commercial crops. EPA has not recently included a separate and additive mortality effect for ozone, independent of the effect associated with fine particulate matter. For a number of reasons, including 1) advice from the Science Advisory Board (SAB) Health and Ecological Effects Subcommittee (HEES) that EPA consider the plausibility and viability of including an estimate of premature mortality associated with short-term ozone exposure in its benefits analyses and 2) conclusions regarding the scientific support for such relationships in EPA's 2006 Air Quality Criteria for Ozone and Related Photochemical Oxidants (the CD), EPA is in the process of determining how to appropriately characterize ozone-related mortality benefits within the context of benefits

analyses for air quality regulations. As part of this process, we are seeking advice from the National Academy of Sciences (NAS) regarding how the ozone-mortality literature should be used to quantify the reduction in premature mortality due to diminished exposure to ozone, the amount of life expectancy to be added and the monetary value of this increased life expectancy in the context of health benefits analyses associated with regulatory assessments.

Since the NAS effort is not expected to conclude until 2008, the agency is currently deliberating how best to characterize ozone-related mortality benefits in its rulemaking analyses in the interim. For the analysis of the proposed small engine standards, we do not quantify an ozone mortality benefit. So that we do not provide an incomplete picture of all of the benefits associated with reductions in emissions of ozone precursors, we have chosen not to include an estimate of total ozone benefits in the proposed RIA. By omitting ozone benefits in this proposal, we acknowledge that this analysis underestimates the benefits associated with the proposed standards. For more information regarding the quantified benefits included in this analysis, please refer to Chapter 8.

2.1.3 Current and Projected Ozone Levels

The Clean Air Act (CAA) requires EPA to set NAAQS for wide-spread pollutants from diverse sources considered harmful to public health and the environment. The CAA established two types of NAAQS: primary standards to protect public health, secondary standards to protect public welfare. The primary and secondary ozone NAAQS are identical. The 8-hour ozone standard is met when the 3-year average of the annual 4th highest daily maximum 8-hour ozone concentration is less than 0.08 ppm (62 FR 38855, July 18, 1997).

The proposed emission reductions from this rule would assist 8-hour ozone nonattainment and maintenance areas in reaching the standard by each area's respective attainment date, and maintaining the 8-hour ozone standard in the future. The emission reductions would also help continue to lower ambient ozone levels and resulting health impacts into the future. In this section we present information on current and projected future 8-hour ozone levels.

2.1.3.1 Current 8-Hour Ozone Levels

A nonattainment area is defined in the CAA as an area that is violating a NAAQS or is contributing to a nearby area that is violating the NAAQS. EPA designated nonattainment areas for the 8-hour ozone NAAQS in June 2004. The final rule on Air Quality Designations and Classifications for the 8-hour Ozone NAAQS (69 FR 23858, April 30, 2004) lays out the factors that EPA considered in making the 8-hour ozone nonattainment designations, including 2001-2003 measured data, air quality in adjacent areas, and other factors.[2]

[2]An ozone design value is the concentration that determines whether a monitoring site meets the NAAQS for ozone. Because of the way they are defined, design values are determined based on three consecutive-year monitoring periods. 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

As of October 2006, approximately 157 million people live in the 116 areas that are designated as nonattainment for either failing to meet the 8-hour ozone NAAQS or for contributing to poor air quality in a nearby area.[3] There are 461 full or partial counties that make up the 116 8-hour ozone nonattainment areas, as shown in Figure 2-1.

Counties designated as ozone nonattainment were categorized, on the basis of their one-hour ozone design value, as Subpart 1 or Subpart 2. Areas categorized as Subpart 2 were then further classified, on the basis of their 8-hour ozone design value, as marginal, moderate, serious, severe or extreme. The maximum attainment date assigned to an ozone nonattainment area is based on the area's classification.

States with 8-hour ozone nonattainment areas are required to take action to bring those areas into compliance prior to the ozone season in the attainment year. Based on the final rule designating and classifying 8-hour ozone nonattainment areas, most 8-hour ozone nonattainment areas will be required to attain the 8-hour ozone NAAQS in the 2007 to 2014 time frame and then be required to maintain the 8-hour ozone NAAQS thereafter.[4] The emission standards being proposed in this action would become effective between 2008 and 2013. Thus, the expected ozone precursor emission inventory reductions from the standards proposed in this action would be useful to states in attaining and/or maintaining the 8-hour ozone NAAQS.

EPA's review of the ozone NAAQS is currently underway and a proposed decision in this review is scheduled for June 2007 with a final rule scheduled for March 2008. If the ozone NAAQS is revised then new nonattainment areas could be designated. While EPA is not relying on it for purposes of justifying this rule, the emission reductions from this proposal would also be helpful to states if there is an ozone NAAQS revision.

2.1.3.2 Projected 8-Hour Ozone Levels

Air quality modeling analyses completed for this proposed rule included assessing ambient ozone concentrations with and without the proposed emission controls. The air quality modeling predicts that without additional local, regional or national controls there will continue

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(including accounting for missing values and other complexities) are given in Appendices H and I of 40 CFR Part 50. 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 or 85 parts per billion (ppb). 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. However, readers should note that ozone design values generally represent air quality across a broad area and that absence of a design value does not imply that the county is in compliance with the ozone NAAQS. Therefore, our analysis may underestimate the number of counties with design values above the level of NAAQS.

[3]The 8-hour ozone nonattainment areas are listed in a Memo to the Docket titled "Nonattainment Areas and Mandatory Class I Federal Areas" and contained in Docket EPA-HQ-OAR-2004-0008.

[4] The Los Angeles Southcoast Air Basin 8-hour ozone nonattainment area will have to attain before June 15, 2021.

to be a need for reductions in 8-hour ozone concentrations in some areas in the future.

We performed a series of ozone air quality modeling simulations for the Eastern United States using the Comprehensive Air Quality Model with Extension (CAMx). The air quality modeling performed for this proposed rule was based upon the same modeling system as was used in the Clean Air Interstate rule (CAIR) and Clean Air Nonroad Diesel (CAND) legislation. The model simulations were performed for five emission scenarios: a 2001 baseline projection, a 2020 baseline projection and a 2020 projection with controls, a 2030 baseline projection and a 2030 projection with controls.

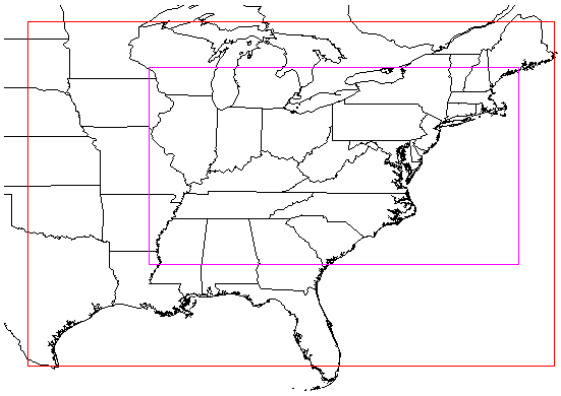
The impacts of the proposed emission standards were determined by comparing the model results in the future year control runs against the baseline simulations of the same year. This modeling supports the conclusion that the proposed controls would help reduce ambient ozone concentrations across the country.

2.1.3.2.1 Ozone Modeling Methodology

CAMx was utilized to estimate base and future-year ozone concentrations over the Eastern United States for various emission scenarios. CAMx simulates the numerous physical and chemical processes involved in the formation, transport, and destruction of ozone. CAMx is a photochemical grid model that numerically simulates the effects of emissions, advection, diffusion, chemistry, and surface removal processes on pollutant concentrations within a three-dimensional grid. This model is commonly used in developing attainment demonstration State Implementation Plans (SIPs) 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 air quality modeling technical support document (TSD), which is located in the docket for this rule.

The modeling domain used for this analysis and in the recent CAIR includes 37 states in the Eastern U.S., see Figure 2.1-2. 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.

Figure 2.1-2: Map of CAIR Modeling Domain



Note: The inner area represents fine grid modeling at 12 km resolution. The outer area represents the coarse grid modeling at 36 km resolution.

The simulation periods modeled by CAMx included several multi-day periods when ambient measurements were representative of ozone episodes over the Eastern 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. 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 emission scenarios were simulated for the selected episodes.

The meteorological data required for input into CAMx (wind, temperature, vertical mixing, etc.) was developed by a separate meteorological model. 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. The 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 air quality 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 CAIR. Additionally, initial conditions were assumed to be relatively clean as well. Given the ramp-up days and the expansive domains, it is expected that these assumptions will not affect the modeling results, except in areas near the boundary (e.g., Dallas-Fort Worth TX). The other non-emission CAMx inputs (land use, photolysis rates, etc.) were developed using procedures employed in the highway light duty Tier 2/OTAG regional modeling. The development of model inputs is discussed in greater detail in the air quality modeling TSD.

Future-year estimates of 8-hour ozone design values were calculated based on relative reduction factors (RRF) between the future simulations, the 2001 base year simulation and 2001-2003 8-hour ozone design values. The procedures for determining the RRFs are similar to those in EPA's guidance for modeling for an 8-hour ozone standard.[33] Hourly model predictions were processed to determine daily maximum 8-hour concentrations for each grid cell for each 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. 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. For more information see the air quality modeling TSD.

The inventories that underlie the ozone modeling conducted for this rulemaking included emission reductions from all current or committed federal, State, and local controls including the recent CAIR and, for the control case, including this proposed rulemaking.

Finally, it should be noted that the emission control scenarios used as input for the air quality and benefits modeling are slightly different than the emission control program being proposed. The proposed levels of the standards have changed, in response to new information on the emission control technologies under consideration and other factors, since we performed the air quality modeling for this proposed rule. Additional detail is provided in Section 3.6.

2.1.3.2.2 Areas at Risk of Future 8-Hour Ozone Violations

This section summarizes the results of recent ozone air quality modeling from the CAIR analysis. 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 the 8-hour ozone NAAQS or to be within 10 percent of the 8-hour ozone NAAQS in the future.

The determination that an area is at risk of exceeding the 8-hour ozone standard in the future was made for all areas with current design values greater than or equal to 85 ppb (or within a 10 percent margin) and with modeling evidence that concentrations at and above this level will persist into the future. Those interested in greater detail should review the CAIR air quality modeling TSD.

Based upon our CAIR air quality modeling, we anticipate that without emission reductions beyond those that were already required under promulgated regulation and approved SIPs, ozone nonattainment will likely persist into the future. With reductions from programs already in place (but excluding the emission reductions from this rule), the number of Eastern counties with projected 8-hour ozone design values at or above 85 ppb in 2010 is expected to be 37 counties where 24 million people are projected to live, see Table 2.1-1. In addition, in 2010, 148 Eastern counties where 61 million people are projected to live, will be within 10 percent of

violating the 8-hour ozone NAAQS.

Table 2.1.3.2.2-1. Eastern Counties with 2010 projected 8-hour Ozone Concentrations Above and Within 10% of the 8-hour Ozone Standard

State	County	2010 Projected 8-hour Ozone Concentration (ppb)[a]	2000 pop[b]	2010 pop[c]
Arkansas	Crittenden Co	80.8	50,866	52,889
Connecticut	Fairfield Co	**92.2**	882,567	891,694
Connecticut	Hartford Co	80.1	857,183	859,080
Connecticut	Middlesex Co	**90.6**	155,071	164,202
Connecticut	New Haven Co	**91.3**	824,008	829,181
Connecticut	New London Co	83.4	259,088	267,199
Connecticut	Tolland Co	82.7	136,364	142,988
D.C.	Washington Co	**85**	572,058	554,474
Delaware	Kent Co	78.7	126,697	139,376
Delaware	New Castle Co	84.7	500,264	534,631
Delaware	Sussex Co	80.3	156,638	181,962
Georgia	Bibb Co	80	153,887	158,291
Georgia	Cobb Co	79.4	607,750	744,488
Georgia	Coweta Co	76.6	89,215	111,522
Georgia	De Kalb Co	81.9	665,864	698,335
Georgia	Douglas Co	78.7	92,174	114,380
Georgia	Fayette Co	76.7	91,263	117,580
Georgia	Fulton Co	**85.1**	816,005	855,826
Georgia	Henry Co	80.3	119,341	153,957
Georgia	Rockdale Co	80.4	70,111	87,977
Illinois	Cook Co	81.8	5,376,739	5,363,464
Illinois	Jersey Co	77	21,668	22,905
Illinois	Lake Co	76.8	644,356	731,690
Illinois	McHenry Co	76.6	260,077	307,400
Indiana	Boone Co	78.1	46,107	54,035
Indiana	Clark Co	78.4	96,472	107,096
Indiana	Hamilton Co	81.7	182,740	230,565
Indiana	Hancock Co	80.4	55,391	65,282
Indiana	La Porte Co	81.8	110,106	111,566
Indiana	Lake Co	82.8	484,563	489,220
Indiana	Madison Co	78.6	133,358	137,710
Indiana	Marion Co	79.6	860,453	879,932
Indiana	Porter Co	81.1	146,798	165,350
Indiana	Shelby Co	81.6	43,445	46,565
Indiana	St Joseph Co	77.8	265,559	275,031
Kentucky	Campbell Co	81.5	88,616	92,109
Louisiana	Bossier Parish	77	98,310	110,838
Louisiana	East Baton Rouge Parish	80.6	412,852	465,411
Louisiana	Iberville Parish	79.4	33,320	33,089
Louisiana	Jefferson Parish	78.6	455,466	493,359
Louisiana	Livingston Parish	77.8	91,814	124,895
Louisiana	West Baton Rouge Parish	78.8	21,601	22,672
Maine	Hancock Co	80.5	51,791	53,886
Maine	York Co	80.2	186,742	201,082
Maryland	Anne Arundel Co	**88.6**	489,656	543,785
Maryland	Baltimore Co	83.7	754,292	792,284
Maryland	Carroll Co	80	150,897	179,918
Maryland	Cecil Co	**89.5**	85,951	96,574
Maryland	Charles Co	78.7	120,546	145,763

Maryland	Frederick Co	78.1	195,277	234,304
Maryland	Harford Co	**92.8**	218,590	268,207
Maryland	Kent Co	**85.8**	19,197	20,233
Maryland	Montgomery Co	79.3	873,341	940,126
Maryland	Prince Georges Co	84.2	801,515	842,221
Massachusetts	Barnstable Co	83.6	222,230	249,495
Massachusetts	Bristol Co	83	534,678	558,460
Massachusetts	Essex Co	81.7	723,419	747,556
Massachusetts	Hampden Co	80.2	456,228	452,718
Massachusetts	Hampshire Co	78	152,251	158,130
Massachusetts	Middlesex Co	79.1	1,465,396	1,486,428
Massachusetts	Suffolk Co	78.1	689,807	674,179
Michigan	Allegan Co	82.1	105,665	121,415
Michigan	Benzie Co	77.9	15,998	17,849
Michigan	Berrien Co	78.1	162,453	164,727
Michigan	Cass Co	78.2	51,104	53,544
Michigan	Genesee Co	76.7	436,141	441,196
Michigan	Macomb Co	**85.4**	788,149	838,353
Michigan	Mason Co	78.9	28,274	30,667
Michigan	Muskegon Co	82	170,200	175,901
Michigan	Oakland Co	80.7	1,194,155	1,299,592
Michigan	Ottawa Co	76.6	238,314	277,400
Michigan	St Clair Co	80.6	164,235	178,391
Michigan	Washtenaw Co	81	322,895	344,398
Michigan	Wayne Co	84.7	2,061,161	1,964,209
Missouri	Clay Co	76.5	184,006	213,643
Missouri	Jefferson Co	76.7	198,099	230,539
Missouri	St Charles Co	80.5	283,883	341,686
Missouri	St Louis City	79.4	348,188	324,156
Missouri	St Louis Co	80.5	1,016,315	1,024,964
New Hampshire	Hillsborough Co	76.6	380,841	412,071
New Jersey	Atlantic Co	80.4	252,552	269,754
New Jersey	Bergen Co	**86**	884,118	898,450
New Jersey	Camden Co	**91.6**	508,932	509,912
New Jersey	Cumberland Co	84.4	146,438	149,595
New Jersey	Gloucester Co	**91.3**	254,673	278,612
New Jersey	Hudson Co	84.3	608,975	607,256
New Jersey	Hunterdon Co	**88.6**	121,989	139,641
New Jersey	Mercer Co	**95.2**	350,761	359,912
New Jersey	Middlesex Co	**92.1**	750,162	805,537
New Jersey	Monmouth Co	**86.4**	615,301	670,971
New Jersey	Morris Co	**85.5**	470,212	500,033
New Jersey	Ocean Co	**100.3**	510,916	572,364
New Jersey	Passaic Co	79.7	489,049	495,610
New York	Bronx Co	79.7	1,332,649	1,298,206
New York	Chautauqua Co	81.8	139,750	139,909
New York	Dutchess Co	81	280,150	291,098
New York	Erie Co	**86.9**	950,265	953,085
New York	Essex Co	77.6	38,851	39,545
New York	Jefferson Co	80.5	111,738	113,075
New York	Monroe Co	76.9	735,343	745,350
New York	Niagara Co	82.3	219,846	220,407
New York	Orange Co	77.1	341,367	371,434
New York	Putnam Co	82.3	95,745	107,967
New York	Queens Co	78.3	2,229,379	2,239,026
New York	Richmond Co	**87.1**	443,728	488,728
New York	Suffolk Co	**90.8**	1,419,369	1,472,127
New York	Westchester Co	84.7	923,459	944,535

North Carolina	Mecklenburg Co	81.4	695,453	814,088
North Carolina	Rowan Co	80.1	130,340	143,729
North Carolina	Wake Co	77.2	627,846	787,707
Ohio	Allen Co	76.8	108,473	106,900
Ohio	Ashtabula Co	83.5	102,728	104,850
Ohio	Butler Co	78	332,806	384,410
Ohio	Clermont Co	78	177,977	205,365
Ohio	Clinton Co	81.4	40,543	47,137
Ohio	Cuyahoga Co	77.3	1,393,977	1,348,313
Ohio	Delaware Co	77.3	109,989	136,125
Ohio	Franklin Co	81.9	1,068,977	1,142,894
Ohio	Geauga Co	**86.6**	90,895	102,083
Ohio	Hamilton Co	78.6	845,302	843,226
Ohio	Knox Co	76.5	54,500	59,435
Ohio	Lake Co	82.2	227,511	237,161
Ohio	Lorain Co	78.5	284,664	292,040
Ohio	Lucas Co	80	455,053	447,302
Ohio	Medina Co	76.5	151,095	173,985
Ohio	Portage Co	79.8	152,061	162,685
Ohio	Summit Co	82.4	542,898	552,567
Ohio	Trumbull Co	79.7	225,116	226,157
Ohio	Warren Co	80	158,383	186,219
Ohio	Wood Co	77.4	121,065	129,124
Oklahoma	Tulsa Co	79.2	563,299	610,536
Pennsylvania	Allegheny Co	81.9	1,281,665	1,259,040
Pennsylvania	Armstrong Co	79.7	72,392	72,829
Pennsylvania	Beaver Co	79.6	181,412	183,693
Pennsylvania	Berks Co	81.7	373,637	388,194
Pennsylvania	Bucks Co	**94.3**	597,635	648,796
Pennsylvania	Cambria Co	76.9	152,598	146,811
Pennsylvania	Chester Co	**85.4**	433,501	478,460
Pennsylvania	Dauphin Co	80.8	251,798	265,019
Pennsylvania	Delaware Co	84	550,863	543,169
Pennsylvania	Erie Co	79.1	280,843	284,835
Pennsylvania	Franklin Co	80.2	129,313	135,088
Pennsylvania	Lancaster Co	83.6	470,657	513,684
Pennsylvania	Lehigh Co	82.1	312,090	323,215
Pennsylvania	Mercer Co	78.1	120,293	122,546
Pennsylvania	Montgomery Co	**87.6**	750,097	772,849
Pennsylvania	Northampton Co	81.8	267,066	279,797
Pennsylvania	Philadelphia Co	**89.9**	1,517,549	1,420,803
Pennsylvania	Washington Co	77.3	202,897	205,153
Pennsylvania	Westmoreland Co	76.7	369,993	372,941
Pennsylvania	York Co	79.4	381,750	404,807
Rhode Island	Kent Co	**86.2**	167,090	174,126
Rhode Island	Providence Co	81.2	621,602	621,355
Rhode Island	Washington Co	84.2	123,546	137,756
South Carolina	Richland Co	76.9	320,677	349,826
Tennessee	Sevier Co	76.5	71,170	96,097
Tennessee	Shelby Co	76.7	897,471	958,501
Texas	Brazoria Co	84.1	241,767	281,960
Texas	Collin Co	82.5	491,675	677,868
Texas	Dallas Co	82.2	2,218,899	2,382,657
Texas	Denton Co	**86.8**	432,976	554,033
Texas	Galveston Co	84.6	250,158	283,963
Texas	Gregg Co	79.1	111,379	121,241
Texas	Harris Co	**97.4**	3,400,577	3,770,129
Texas	Jefferson Co	**85**	252,051	260,847

Texas	Johnson Co	78.2	126,811	157,545
Texas	Montgomery Co	81.2	293,768	413,048
Texas	Tarrant Co	**87.2**	1,446,219	1,710,920
Virginia	Alexandria City	80.9	128,283	130,422
Virginia	Arlington Co	**86**	189,453	193,370
Virginia	Charles City Co	77.7	6,926	7,382
Virginia	Fairfax Co	**85.4**	969,749	1,085,483
Virginia	Hampton City	78.7	146,437	153,246
Virginia	Hanover Co	80.9	86,320	98,586
Virginia	Henrico Co	78.2	262,300	294,174
Virginia	Loudoun Co	78.6	169,599	214,469
Virginia	Suffolk City	77.5	63,677	69,003
Wisconsin	Door Co	82.1	27,961	30,508
Wisconsin	Kenosha Co	**91**	149,577	166,359
Wisconsin	Kewaunee Co	79.9	20,187	20,538
Wisconsin	Manitowoc Co	80	82,887	83,516
Wisconsin	Milwaukee Co	82.1	940,164	922,943
Wisconsin	Ozaukee Co	**85.8**	82,317	95,549
Wisconsin	Racine Co	83.9	188,831	199,178
Wisconsin	Sheboygan Co	**87.7**	112,646	118,866
Number of Violating Counties		37		
Population of Violating Counties			22,724,010	24,264,574
Number of Counties within 10%		148		
Population of Counties within 10%			58,453,962	61,409,062

a) Bolded concentrations indicate levels above the 8-hour ozone standard.
b) Populations are based on 2000 census data.
c) Populations are based on 2000 census projections.

The CAMx model also contains a source apportionment tool which can be used to estimate how emissions from individual source areas and regions impact modeled ozone concentrations. Small SI and Marine SI sector contributions were calculated for the areas which the CAIR modeling projected to have design values at or above 85 ppb in 2020. In those areas, Small SI and Marine SI emissions were estimated to be responsible for between one and seven percent of the ozone concentrations above 85 ppb. Additional information on the source apportionment tool and analysis can be found in the air quality modeling TSD for this proposal.

We have described the current nonattainment with the 8-hour ozone NAAQS and that absent additional controls, modeling predicts that there will continue to be people living in counties with 8-hour ozone levels above the NAAQS in the future. In addition, we have described how in the future, in areas which are projected to have ozone levels greater than 85 ppb, Small SI engines and equipment and Marine SI engines and vessels are projected to contribute to these ozone concentrations.

These analyses demonstrate the need for reductions in emissions from this proposed rule. As shown earlier in Figure 2-1, unhealthy ozone concentrations occur over wide geographic areas and the engines, vessels and equipment covered in this proposed rule contribute to the ozone precursors in and near these areas. Thus, reductions in ozone precursors from Small SI engines and equipment and Marine SI engines and vessels are needed to assist States in attaining and maintaining the 8-hour ozone NAAQS and reducing ozone exposures.

2.1.3.2.3 Modeling Projections of ozone with the proposed controls

This section summarizes the results of our modeling of ozone air quality impacts in the future due to the reductions in Small SI engine and equipment and Marine SI engine and vessel emissions proposed in this action. Specifically, we compare baseline scenarios to scenarios with the proposed controls. Our modeling indicates that the reductions from this proposed rule would contribute to reducing ambient ozone concentrations and potential exposures in future years.

On a population-weighted basis, the average change in future year design values for the eastern U.S. would be a decrease of 0.7 ppb in 2020 and 0.8 ppb in 2030. In areas with larger design values, greater than 85 ppb, the population-weighted average decrease would be somewhat higher, 0.8 ppb in 2020 and 1.0 ppb in 2030.

Table 2.1-2 shows the average change in future year eight-hour ozone design values. Average changes are shown 1) for all counties with 2001-2003 8-hour ozone design values, 2) for counties with design values that did not meet the standard in 2001-2003 ("violating" counties), and 3) for counties that met the standard, but were within 10 percent of it in 2001-2003. 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 2.1-2 demonstrates a broad improvement in ozone air quality. The average across violating counties shows that the proposed rule would help bring these counties into attainment. Since some of the VOC and NOx emission reductions expected from this proposed rule would go into effect during the period when areas will need to attain the 8-hour ozone NAAQS, the projected reductions in emissions are expected to assist States and local agencies in their effort to attain and maintain the 8-hour ozone standard. The average over counties within ten percent of the standard shows that the proposed rule would also help those counties to maintain the standard. All of these metrics show a decrease in 2020 and a larger decrease in 2030, indicating in four different ways the overall improvement in ozone air quality.

Table 2.1-2: Average Change in Projected Future Year 8-hour Ozone Design Value

Average[a]	Number of Eastern Counties	change in 2020 design value[b] (ppb)	change in 2030 design value[b] (ppb)
All	525	-0.5	-0.7
All, population-weighted	525	-0.7	-0.8
Violating counties[c]	270	-0.6	-0.8
Violating counties[c], population-weighted	270	-0.8	-1.0
Counties within 10 percent of the standard[d]	185	-0.4	-0.5
Counties within 10 percent of the standard[d], population-weighted	185	-0.5	-0.7

[a] averages are over counties with 2001 modeled design values
[b] assuming the nominal modeled control scenario
[c] counties whose 2001 design values exceeded the 8-hour ozone standard (>= 85 ppb)
[d] counties whose 2001 design values were less than but within 10 percent of the 8-hour ozone standard (between 77 and 85 ppb)

The impact of the proposed reductions has also been analyzed with respect to those areas that have the highest projected design values. We project that there will be 13 Eastern counties with design values at or above 85 ppb in 2030. After implementation of this proposed action, we project that 7 of these 13 counties would be at least 40% closer to a design value of less than 85 ppb, and on average all 13 counties would be 35% closer to a design value of less than 85 ppb.

2.1.4 Environmental Effects of Ozone Pollution

There are a number of public welfare effects associated with the presence of ozone in the ambient air.[34] In this section we discuss the impact of ozone on plants, including trees, agronomic crops and urban ornamentals.

The Air Quality Criteria Document for Ozone and related Photochemical Oxidants notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant".[35] 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.[36] Once sufficient levels of ozone, a highly reactive substance, (or its reaction products) reaches the interior of plant cells, it can inhibit or damage essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns.[37, 38] 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.[39] 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. Furthermore, there is some 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.[40]

Ozone can produce both acute and chronic injury in sensitive species depending on the concentration level and the duration of the exposure. Ozone effects also tend to accumulate over the growing season of the plant, so that even lower concentrations experienced for a longer duration have the potential to create chronic stress on sensitive vegetation. Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of O_3 uptake through closure of stomata).[41, 42, 43] 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.[44] Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and response to ozone, it is not possible to identify threshold values above which ozone is consistently toxic for all plants. The next few paragraphs present additional information on ozone damage to trees, ecosystems, agronomic crops and urban ornamentals.

Ozone also has been conclusively shown to cause discernible injury to forest trees.[45, 46] In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts.[47] Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function.[48,49]

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 impacts at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors.[50] In most instances, responses to chronic or recurrent exposure in forest exosystems are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems.[51, 52, 53] It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge

of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States.

Laboratory and field experiments have also shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the Unites States."[54] In addition, economic studies have shown reduced economic benefits as a result of predicted reductions in crop yields associated with observed ozone levels.[55, 56, 57]

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. 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.[58] This is therefore a potentially costly environmental effect. However, in the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative analysis has been conducted. 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.

2.2 Particulate Matter

In this section we review the health and welfare effects of PM. We also describe air quality monitoring and modeling data that indicate many areas across the country continue to be exposed to levels of ambient PM above the NAAQS. Emissions of HCs and NOx from the engines, vessels and equipment subject to this proposed rule contribute to these PM concentrations. Information on air quality was gathered from a variety of sources, including monitored PM concentrations, air quality modeling done for recent EPA rulemakings and other state and local air quality information.

2.2.1 Science of PM Formation

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 is further described by breaking it down into size fractions. PM_{10} refers to particles generally less than or equal to 10 micrometers (µm) in diameter. $PM_{2.5}$ refers to fine particles, those particles generally less than or equal to 2.5 µm in diameter. Inhalable (or "thoracic") coarse particles refer to those particles generally greater than 2.5 µm but less than or equal to 10 µm in diameter. Ultrafine PM refers to particles with diameters generally less than 100 nanometers (0.1 µm). Larger particles (>10 µm) tend to be removed by the respiratory clearance mechanisms, whereas smaller particles are deposited deeper in the lungs.

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

The engines, vessels and equipment that would be covered by the proposed standards contribute to ambient PM levels through primary (direct) and secondary (indirect) PM. Primary PM is directly emitted into the air, and secondary PM forms in the atmosphere from gases emitted by fuel combustion and other sources. Along with primary PM, the engines, vessels and equipment controlled in this action emit HC and NOx, which react in the atmosphere to form secondary $PM_{2.5}$. Both types of directly and indirectly formed particles from Small SI engines and equipment and Marine SI engines and vessels are found principally in the fine fraction.

EPA has recently amended the PM NAAQS (71 FR 61144, October 17, 2006). The final rule, signed on September 21, 2006 and published on October 17, 2006, addressed revisions to the primary and secondary NAAQS for PM to provide increased protection of public health and welfare, respectively. The primary $PM_{2.5}$ NAAQS include a short-term (24-hour) and a long-term (annual) standard. The level of the 24-hour $PM_{2.5}$ NAAQS has been revised from 65 µg/m3 to 35 µg/m3 to provide increased protection against health effects associated with short-term exposures to fine particles. The current form of the 24-hour $PM_{2.5}$ standard was retained (e.g., based on the 98th percentile concentration averaged over three years). The level of the annual $PM_{2.5}$ NAAQS was retained at 15 µg/m3, continuing protection against health effects associated with long-term exposures. The current form of the annual $PM_{2.5}$ standard was retained as an annual arithmetic mean averaged over three years, however, the following two aspects of the spatial averaging criteria were narrowed: (1) the annual mean concentration at each site shall be within 10 percent of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair shall yield a correlation coefficient of at least 0.9 for each calendar quarter. With regard to the primary PM_{10} standards, the 24-hour PM_{10} NAAQS was retained at a level of 150 $\mu g/m^3$ not to be exceeded more than once per year on average over a three-year period. Given that the available evidence does not suggest an association between long-term exposure to coarse particles at current ambient levels and health effects, EPA has revoked the annual PM_{10} standard.

With regard to the secondary PM standards, EPA has revised these standards to be identical in all respects to the revised primary standards. Specifically, EPA has revised the current 24-hour $PM_{2.5}$ secondary standard by making it identical to the revised 24-hour $PM_{2.5}$ primary standard, retained the annual $PM_{2.5}$ and 24-hour PM_{10} secondary standards, and revoked the annual PM_{10} secondary standards. This suite of secondary PM standards is intended to provide protection against PM-related public welfare effects, including visibility impairment, effects on vegetation and ecosystems, and material damage and soiling.

2.2.2 Health Effects of PM

As stated in the EPA Particulate Matter Air Quality Criteria Document (PM AQCD), available scientific findings "demonstrate well that human health outcomes are associated with ambient PM."[5] We are relying primarily on the data and conclusions in the PM AQCD and PM staff paper, which reflects EPA's analysis of policy-relevant science from the PM AQCD, regarding the health effects associated with particulate matter.[59,60] We also present additional recent studies published after the cut-off date for the PM AQCD.[6, 61] Taken together this information supports the conclusion that PM-related emissions from Small SI engines and equipment and Marine SI engines and vessels are associated with adverse health effects.

2.2.2.1 Short-term Exposure Mortality and Morbidity Studies

As discussed in the PM AQCD, short-term exposure to $PM_{2.5}$ is associated with mortality from cardiopulmonary diseases (PM AQCD, p. 8-305), hospitalization and emergency department visits for cardiopulmonary diseases (PM AQCD, p. 9-93), increased respiratory symptoms (PM AQCD, p. 9-46), decreased lung function (PM AQCD Table 8-34) and physiological changes or biomarkers for cardiac changes (PM AQCD, Section 8.3.1.3.4). In addition, the PM AQCD describes a limited body of new evidence from epidemiologic studies for potential relationships between short-term exposure to PM and health endpoints such as low birth weight, preterm birth, and neonatal and infant mortality. (PM AQCD, Section 8.3.4).

Among the studies of effects from short-term exposure to $PM_{2.5}$, several studies specifically address the contribution of mobile sources to short-term $PM_{2.5}$ effects on daily mortality. These studies indicate that there are statistically significant associations between mortality and PM related to mobile source emissions (PM AQCD, p. 8-85). The analyses incorporate source apportionment tools into daily mortality studies and are briefly mentioned here. Analyses incorporating source apportionment by factor analysis with daily time-series studies of daily death established a specific influence of mobile source-related $PM_{2.5}$ on daily mortality[62] and a concentration-response function for mobile source-associated $PM_{2.5}$ and daily mortality.[63] Another recent study in 14 U.S. cities examined the effect of PM_{10} exposures on daily hospital admissions for cardiovascular disease. 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

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

[6]These additional studies are included in the 2006 Provisional Assessment of Recent Studies on Health Effects of Particulate Matter Exposure. The provisional assessment did not and could not (given a very short timeframe) undergo the extensive critical review by EPA, CASAC, and the public, as did the PM AQCD. The provisional assessment found that the "new" studies expand the scientific information and provide important insights on the relationship between PM exposure and health effects of PM. The provisional assessment also found that "new" studies generally strengthen the evidence that acute and chronic exposure to fine particles and acute exposure to thoracic coarse particles are associated with health effects.

PM_{10} when compared with other sources.[64] These studies provide evidence that PM-related emissions, specifically from mobile sources, are associated with adverse health effects.

2.2.2.2 Long-term Exposure Mortality and Morbidity Studies

Long-term exposure to elevated ambient $PM_{2.5}$ is associated with mortality from cardiopulmonary diseases and lung cancer (PM AQCD, p. 8-307), and effects on the respiratory system such as decreased lung function or the development of chronic respiratory disease (PM AQCD, pp. 8-313, 8-314). Of specific importance to this proposal, the PM AQCD also notes that the PM components of gasoline and diesel engine exhaust represent one class of hypothesized likely important contributors to the observed ambient PM-related increases in lung cancer incidence and mortality (PM AQCD, p. 8-318).

The PM AQCD and PM Staff Paper emphasize the results of two long-term studies, the Six Cities and American Cancer Society (ACS) prospective cohort studies, based on several factors - the inclusion of measured PM data, the fact that the study populations were similar to the general population, and the fact that these studies have undergone extensive reanalysis (PM AQCD, p. 8-306, Staff Paper, p.3-18).[65,66,67] These studies indicate that there are significant associations for all-cause, cardiopulmonary, and lung cancer mortality with long-term exposure to $PM_{2.5}$. A variety of studies have been published since the completion of the PM AQCD. One such study, an analysis of a subset of the ACS cohort data, which was published after the PM AQCD was finalized but in time for the 2006 Provisional Assessment, found a larger association than had previously been reported between long-term $PM_{2.5}$ exposure and mortality in the Los Angeles area using a new exposure estimation method that accounted for variations in concentration within the city.[68] EPA is assessing the significance of this study within the context of the broader literature.

As discussed in the PM AQCD, the morbidity studies that combine the features of cross-sectional and cohort studies provide the best evidence for chronic exposure effects. Long-term studies evaluating the effect of ambient PM on children's development have shown some evidence indicating effects of $PM_{2.5}$ and/or PM_{10} on reduced lung function growth (PM AQCD, Section 8.3.3.2.3). One such study, which was summarized in the 2006 Provisional Assessment, reported the results of a cross-sectional study of outdoor $PM_{2.5}$ and measures of atherosclerosis in the Los Angeles basin.[69] The study found significant associations between ambient residential $PM_{2.5}$ and carotid intima-media thickness (CIMT), an indicator of subclinical atherosclerosis, an underlying factor in cardiovascular disease. EPA is assessing the significance of this study within the context of the broader literature.

2.2.2.3 Roadway-Related Exposure and Health Studies

A recent body of studies reinforces the findings of these PM morbidity and mortality effects by looking at traffic-related exposures, PM measured along roadways, or time spent in traffic and adverse health effects. While many of these studies did not measure PM specifically, they include potential exhaust exposures which include mobile source PM because they employ indices such as roadway proximity or traffic volumes. One study with specific relevance to

$PM_{2.5}$ health effects is a study that was done in North Carolina looking at concentrations of $PM_{2.5}$ inside police cars and corresponding physiological changes in the police personnel driving the cars. The authors report significant elevations in markers of cardiac risk associated with concentrations of $PM_{2.5}$ inside police cars on North Carolina state highways.[70] A number of studies of traffic-related pollution have shown associations between fine particles and adverse respiratory outcomes in children who live near major roadways. [71,72,73] Additional information on near-roadway health effects is included in the recent Mobile Source Air Toxics rule (72 FR 8428, February 26, 2007).

2.2.3 Current and Projected PM Levels

The proposed emission reductions from this rule would assist PM nonattainment areas in reaching the standard by each area's respective attainment date and assist PM maintenance areas in maintaining the PM standards in the future. The emission reductions would also help continue to lower ambient PM levels and resulting health impacts into the future. In this section we present information on current and future attainment of the PM standards.

2.2.3.1 Current $PM_{2.5}$ Levels

A nonattainment area is defined in the Clean Air Act (CAA) as an area that is violating an ambient standard or is contributing to a nearby area that is violating the standard. In 2005, EPA designated 39 nonattainment areas for the 1997 $PM_{2.5}$ NAAQS based on air quality design values (using 2001-2003 or 2002-2004 measurements) and a number of other factors.[7](70 FR 943, January 5, 2005; 70 FR 19844, April 14, 2005) These areas are comprised of 208 full or partial counties with a total population exceeding 88 million.[8] As mentioned in Section 2.2.1, the 1997 $PM_{2.5}$ NAAQS were recently revised and the 2006 $PM_{2.5}$ NAAQS became effective on December 18, 2006. Nonattainment areas will be designated with respect to the new 2006 PM NAAQS in early 2010. Table 2.2-1 presents the number of counties in areas currently designated as nonattainment for the 1997 $PM_{2.5}$ NAAQS as well as the number of additional counties which have monitored data that is violating the 2006 $PM_{2.5}$ NAAQS.

[7] The full details involved in calculating a $PM_{2.5}$ design value are given in Appendix N of 40 CFR Part 50.

[8]The $PM_{2.5}$ nonattainment areas are listed in a Memo to the Docket titled "Nonattainment Areas and Mandatory Class I Federal Areas" and contained in Docket EPA-HQ-OAR-2004-0008.

Table 2.2-1. Fine Particle Standards: Current Nonattainment Areas and Other Violating Counties

	Number of Counties	Population[1]
1997 $PM_{2.5}$ Standards: 39 areas currently designated	208	88,394,000
2006 $PM_{2.5}$ Standards: Counties with violating monitors[2]	49	18,198,676
Total	257	106,592,676

[1] Population numbers are from 2000 census data.

[2] This table provides an estimate of the counties violating the 2006 $PM_{2.5}$ NAAQS based on 2003-05 air quality data. The areas designated as nonattainment for the 2006 $PM_{2.5}$ NAAQS will be based on 3 years of air quality data from later years. Also, the county numbers in the summary table includes only the counties with monitors violating the 2006 $PM_{2.5}$ NAAQS. The monitored county violations may be an underestimate of the number of counties and populations that will eventually be included in areas with multiple counties designated nonattainment.

States with $PM_{2.5}$ nonattainment areas will be required to take action to bring those areas into compliance in the future. Most $PM_{2.5}$ nonattainment areas will be required to attain the 1997 $PM_{2.5}$ NAAQS in the 2010 to 2015 time frame and then be required to maintain the 1997 $PM_{2.5}$ NAAQS thereafter.[9] The attainment dates associated with the potential nonattainment areas based on the 2006 $PM_{2.5}$ NAAQS would likely be in the 2015 to 2020 timeframe. The emission standards being proposed in this action would become effective between 2008 and 2013. The expected $PM_{2.5}$ inventory reductions from the standards proposed in this action would be useful to states in attaining or maintaining the $PM_{2.5}$ NAAQS.

2.2.3.2 Current PM_{10} Levels

EPA designated PM_{10} nonattainment areas in 1990.[10] As of October 2006, approximately 28 million people live in the 46 areas that are designated as PM_{10} nonattainment, for either failing to meet the PM_{10} NAAQS or for contributing to poor air quality in a nearby area. There are 46 full or partial counties that make up the PM_{10} nonattainment areas.[11]

[9]The EPA finalized $PM_{2.5}$ attainment and nonattainment areas in April 2005. The EPA finalized the PM Implementation rule in March 2007.

[10]A PM_{10} design value is the concentration that determines whether a monitoring site meets the NAAQS for PM_{10}. The full details involved in calculating a PM_{10} design value are given in Appendices H and I of 40 CFR Part 50.

[11]The PM_{10} nonattainment areas are listed in a Memo to the Docket titled "Nonattainment Areas and Mandatory Class I Federal Areas" and contained in Docket EPA-HQ-OAR-2004-0008.

2.2.3.3 Projected $PM_{2.5}$ Levels

Recent air quality modeling predicts that without additional controls there will continue to be a need for reductions in PM concentrations in the future. In the following sections we describe the recent PM air quality modeling and results of the modeling.

2.2.3.3.1 PM Modeling Methodology

Recently PM air quality analyses were performed for the PM NAAQS final rule, which was promulgated by EPA in 2006. The Community Multiscale Air Quality (CMAQ) model was used as the tool for simulating base and future year concentrations of PM, visibility and deposition in support of the PM NAAQS air quality assessments. The PM NAAQS analysis included all federal rules up to and including the Clean Air Interstate Rule (CAIR) and all final mobile source rule controls as of October 2006. Details on the PM air quality modeling are provided in the RIA for the final PM NAAQS rule, included in the docket for this proposed rule.

2.2.3.3.2 Areas at Risk of Future $PM_{2.5}$ Violations

Air quality modeling performed for the final PM NAAQS indicates that in the absence of additional local, regional or national controls, there will likely continue to be counties that will not attain some combination of the annual 2006 $PM_{2.5}$ standard (15 $\mu g/m^3$) and the daily 2006 $PM_{2.5}$ standard (35 $\mu g/m^3$). The PM NAAQS analysis provides estimates of future $PM_{2.5}$ levels across the country. For example, in 2015 based on emission controls currently adopted or expected to be in place[12], we project that 53 million people will live in 52 counties with projected $PM_{2.5}$ design values at and above the 2006 standard, see Table 2.2-2.[13] The proposed rule would provide emission reductions that will help areas to attain the $PM_{2.5}$ NAAQS. Table 2.2-2 also lists the 54 counties, where 27 million people are projected to live, with 2015 projected design values that do not violate the $PM_{2.5}$ NAAQS but are within ten percent of it. The proposed rule may help ensure that these counties continue to maintain their attainment status.

Table 2.2-2 Counties with 2015 Projected $PM_{2.5}$ Design Values Above and within 10% of the 2006 $PM_{2.5}$ Standard

State	County	2015 Projected Annual $PM_{2.5}$ Design Value ($\mu g/m^3$)[a]	2015 Projected Daily $PM_{2.5}$ Design Value ($\mu g/m^3$)[a]	2015 Population[b]
Alabama	Jefferson Co	**15.9**	**36.9**	669,850
California	Alameda Co	**13.3**	**59.4**	1,628,698
California	Butte Co	**13.4**	**50.7**	242,166

[12]Counties forecast to remain in nonattainment may need to adopt additional local or regional controls to attain the standards by dates set pursuant to the Clean Air Act. The emissions reductions associated with this proposed rule would help these areas attain the PM standards by their statutory date.

[13]Note that this analysis identifies only counties projected to have a violating monitor; the number of counties to be designated and the associated population would likely exceed these estimates.

California	Colusa Co	9.5	33.5	23,066
California	Contra Costa Co	**12.6**	**61.3**	1,155,323
California	Fresno Co	**20.1**	**73.0**	960,934
California	Imperial Co	**14.8**	**45.7**	173,482
California	Inyo Co	**6.1**	**38.1**	19,349
California	Kern Co	**21.3**	**81.4**	804,940
California	Kings Co	**17.2**	**70.6**	161,607
California	Los Angeles Co	**23.7**	**62.2**	9,910,805
California	Merced Co	**15.8**	**54.4**	250,152
California	Orange Co	**20.0**	**41.1**	3,467,120
California	Placer Co	**11.4**	**38.1**	403,624
California	Riverside Co	**27.8**	**73.5**	2,015,955
California	Sacramento Co	**12.2**	**49.8**	1,488,456
California	San Bernardino Co	**24.6**	**65.7**	2,157,926
California	San Diego Co	**15.8**	**40.7**	3,489,368
California	San Francisco Co	**11.3**	**52.5**	765,846
California	San Joaquin Co	**15.4**	**51.1**	675,362
California	San Luis Obispo Co	**9.4**	**35.8**	304,079
California	San Mateo Co	**10.5**	**41.9**	785,949
California	Santa Clara Co	**10.7**	**48.5**	1,899,727
California	Solano Co	**11.7**	**57.7**	529,784
California	Sonoma Co	**10.0**	**38.9**	569,486
California	Stanislaus Co	**16.6**	**61.9**	547,041
California	Sutter Co	**11.2**	**39.3**	99,716
California	Tulare Co	**21.2**	**77.2**	441,185
California	Ventura Co	**14.1**	**38.8**	923,205
California	Yolo Co	10.2	33.0	206,388
Connecticut	Fairfield Co	11.0	31.6	893,629
Georgia	Bibb Co	13.7	27.0	160,468
Georgia	Clayton Co	13.9	28.7	280,476
Georgia	DeKalb Co	13.6	31.5	715,947
Georgia	Floyd Co	14.0	30.9	97,674
Georgia	Fulton Co	**15.5**	**32.2**	877,365
Georgia	Muscogee Co	13.4	34.2	197,634
Georgia	Wilkinson Co	13.6	29.3	11,259
Idaho	Ada Co	8.9	32.2	397,456
Idaho	Bannock Co	**9.1**	**40.2**	88,033
Idaho	Canyon Co	9.2	32.6	154,137
Idaho	Power Co	**10.5**	**36.6**	8,932
Idaho	Shoshone Co	**12.4**	**36.2**	15,646
Illinois	Cook Co	**15.5**	**37.1**	5,362,931
Illinois	Madison Co	**15.2**	**35.5**	271,854
Illinois	St. Clair Co	14.6	30.4	251,612
Illinois	Will Co	13.2	32.0	634,068
Indiana	Clark Co	13.6	31.1	112,523
Indiana	Lake Co	**13.4**	**40.8**	490,795
Indiana	Marion Co	13.5	33.1	889,645
Kentucky	Jefferson Co	13.8	33.4	710,231
Maryland	Anne Arundel Co	11.1	33.2	574,322
Maryland	Baltimore city	13.0	35.5	596,076
Maryland	Baltimore Co	11.3	32.6	810,172
Massachusetts	Hampden Co	11.6	32.9	452,055
Michigan	Kalamazoo Co	12.8	32.7	257,817
Michigan	Kent Co	12.0	31.9	654,449

Michigan	Oakland Co	13.0	33.2	1,355,670
Michigan	St. Clair Co	12.5	32.5	185,970
Michigan	Wayne Co	**17.4**	**39.0**	1,921,253
Montana	Lincoln Co	**15.0**	**42.4**	19,875
Montana	Missoula Co	10.6	32.1	118,303
New Jersey	Camden Co	11.1	32.1	512,135
New Jersey	Hudson Co	12.0	32.8	604,036
New Jersey	Union Co	12.2	32.8	525,096
New York	Bronx Co	12.8	33.2	1,283,316
New York	New York Co	14.0	33.2	1,551,641
Ohio	Cuyahoga Co	**15.4**	**40.0**	1,325,507
Ohio	Franklin Co	13.7	33.5	1,181,578
Ohio	Hamilton Co	14.3	34.2	841,858
Ohio	Jefferson Co	14.2	34.2	68,909
Ohio	Lucas Co	12.5	32.2	443,230
Ohio	Scioto Co	**15.6**	**34.3**	81,013
Ohio	Trumbull Co	12.1	34.2	227,546
Oregon	Jackson Co	**10.9**	**37.6**	250,169
Oregon	Klamath Co	**10.1**	**39.1**	69,423
Oregon	Lane Co	**12.9**	**53.6**	387,237
Oregon	Washington Co	9.0	32.0	639,839
Pennsylvania	Allegheny Co	**16.5**	**53.4**	1,245,917
Pennsylvania	Beaver Co	12.1	33.2	184,648
Pennsylvania	Berks Co	**12.0**	**35.5**	396,410
Pennsylvania	Dauphin Co	11.0	33.3	272,748
Pennsylvania	Lancaster Co	12.2	33.7	535,622
Pennsylvania	Lehigh Co	10.5	34.7	328,523
Pennsylvania	Mercer Co	11.0	31.6	123,577
Pennsylvania	Northampton Co	10.9	35.0	286,838
Pennsylvania	Philadelphia Co	13.3	35.2	1,372,037
Pennsylvania	York Co	**12.3**	**35.9**	417,408
Tennessee	Knox Co	13.6	29.6	448,931
Utah	Box Elder Co	**8.6**	**39.0**	49,878
Utah	Cache Co	**12.5**	**51.9**	114,729
Utah	Salt Lake Co	**12.6**	**49.3**	1,133,410
Utah	Utah Co	**9.3**	**36.7**	508,106
Utah	Weber Co	**9.1**	**36.2**	229,807
Washington	Clark Co	9.2	34.3	479,002
Washington	King Co	10.8	34.0	2,013,808
Washington	Pierce Co	**11.1**	**43.0**	879,363
Washington	Snohomish Co	**11.3**	**40.1**	782,319
Washington	Thurston Co	8.9	34.9	264,364
Washington	Yakima Co	9.6	34.9	261,452
West Virginia	Berkeley Co	12.0	32.7	99,349
West Virginia	Hancock Co	13.4	32.7	30,857
West Virginia	Kanawha Co	13.9	28.9	196,498
Wisconsin	Milwaukee Co	12.1	32.1	908,336
Wisconsin	Waukesha Co	11.8	32.4	441,482
Wyoming	Sheridan Co	10.5	31.8	28,623
Number of Violating Counties		52		
Population of Violating Counties				53,468,515
Number of Counties within 10%		54		
Population of Counties within 10%				26,896,926

[a] Bolded concentrations indicate levels above the annual $PM_{2.5}$ standard.

[b] Populations are based on 2000 census projections.

2.2.4 Environmental Effects of PM Pollution

In this section we discuss some of the public welfare effects of PM and its precursors, including NOx, such as visibility impairment, acid deposition, eutrophication, nitrification and fertilization, materials damage, and deposition of PM.

2.2.4.1 Visibility Impairment

Visibility can be defined as the degree to which the atmosphere is transparent to visible light.[74] Visibility impairment manifests in two principal ways: as local visibility impairment and as regional haze.[75] Local visibility impairment may take the form of a localized plume, a band or layer of discoloration appearing well above the terrain as a result from complex local meteorological conditions. Alternatively, local visibility impairment may manifest as an urban haze, sometimes referred to as a "brown cloud." This urban haze is largely caused by emissions from multiple sources in the urban areas and is not typically attributable to only one nearby source or to long-range transport. The second type of visibility impairment, regional haze, usually results from multiple pollution sources spread over a large geographic region. Regional haze can impair visibility over large regions and across states.

Visibility is important because it directly affects people's enjoyment of daily activities in all parts of the country. Individuals value good visibility for the well-being it provides them directly, both in where they live and work, and in places where they enjoy recreational opportunities. Visibility is also highly valued in significant natural areas such as national parks and wilderness areas, and special emphasis is given to protecting visibility in these areas. For more information on visibility see the 2004 PM AQCD as well as the 2005 PM Staff Paper.[76,77]

Fine particles are the major cause of reduced visibility in parts of the United States. To address the welfare effects of PM on visibility, EPA set secondary $PM_{2.5}$ standards which would act in conjunction with the establishment of a regional haze program. In setting this secondary standard, EPA concluded that $PM_{2.5}$ causes adverse effects on visibility in various locations, depending on PM concentrations and factors such as chemical composition and average relative humidity. The secondary (welfare-based) $PM_{2.5}$ NAAQS was established as equal to the suite of primary (health-based) NAAQS. Furthermore, section 169 of the Act provides additional authority to remedy existing visibility impairment and prevent future visibility impairment in the 156 national parks, forests and wilderness areas labeled as mandatory class I federal areas (62 FR 38680-81, July 18, 1997).[1415] In July 1999 the regional haze rule (64 FR 35714) was put in place to protect the visibility in mandatory class I federal areas. Visibility can be said to be

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

[15]The mandatory class I federal areas are listed in a Memo to the Docket titled "Nonattainment Areas and Mandatory Class I Federal Areas" and contained in Docket EPA-HQ-OAR-2004-0008.

impaired in both $PM_{2.5}$ nonattainment areas and mandatory class I federal areas.

EPA has determined that emissions from nonroad engines significantly contribute to air pollution that may be reasonably anticipated to endanger public health and welfare for visibility effects in particular (67 FR 68242, November 8, 2002). The hydrocarbon emissions from the Small SI engines and equipment subject to this proposed rule are PM-precursors and contribute to these visibility effects. This is evident in the PM and visibility modeling recently completed for the PM NAAQS and the CAIR. Small SI engines and equipment and Marine SI engines and vessels were included in the PM NAAQS and CAIR PM and visibility modeling which projected visibility problems persisting in the future.[78,79] In this section we present current information and projected estimates about both visibility impairment related to ambient $PM_{2.5}$ levels across the country and visibility impairment in mandatory class I federal areas. We conclude that visibility will continue to be impaired in the future and the projected emission reductions from this proposed action would help improve visibility conditions across the country and in mandatory class I federal areas.

2.2.4.1.1 Current Visibility Impairment

The need for reductions in the levels of $PM_{2.5}$ is widespread. 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.[80]

As mentioned above, the secondary $PM_{2.5}$ standards were set as equal to the suite of primary $PM_{2.5}$ standards. Recently designated $PM_{2.5}$ nonattainment areas indicate that, as of October 2006, almost 90 million people live in 208 counties that are in nonattainment for the $PM_{2.5}$ NAAQS. Thus, at least these populations (plus others who travel to these areas) would likely be experiencing visibility impairment. Emissions of PM precursors, such as hydrocarbons, from Small SI engines and equipment and Marine SI engines and vessels contribute to this impairment.

2.2.4.1.2 Current Visibility Impairment at Mandatory Class I Federal Areas

Detailed information about current and historical visibility conditions in mandatory class I federal areas is summarized in the EPA Report to Congress and the 2002 EPA Trends Report.[81,82] The conclusions draw upon the Interagency Monitoring of Protected Visual Environments (IMPROVE) network data. One of the objectives of the IMPROVE monitoring network program is to provide regional haze monitoring representing all mandatory class I federal areas where practical. The National Park Service report also describes the state of national park visibility conditions and discusses the need for improvement.[83]

The regional haze rule requires states to establish goals for each affected mandatory class I federal area to improve visibility on the haziest days (20% most impaired days) and ensure no degradation occurs on the cleanest days (20% least impaired days). Although there have been general trends toward improved visibility, progress is still needed on the haziest days.

Specifically, as discussed in the 2002 EPA Trends Report,without the effects of pollution a natural visual range in the United States is approximately 75 to 150 km in the East and 200 to 300 km in the West. In 2001, the mean visual range for the worst days was 29 km in the East and 98 km in the West.[84]

2.2.4.1.3 Future Visibility Impairment

Recent modeling for the final PM NAAQS rule was used to project $PM_{2.5}$ levels in the U.S. in 2015. The results suggest that $PM_{2.5}$ levels above the 2006 NAAQS will persist in the future. We predicted that in 2015, there will be 52 counties with a population of 53 million where $PM_{2.5}$ levels will exceed the 2006 $PM_{2.5}$ NAAQS. Thus, in the future, a percentage of the population may continue to experience visibility impairment in areas where they live, work and recreate.

The emissions from Small SI engines and equipment and Marine SI engines and vessels contribute to visibility impairment. These emissions occur in and around areas with $PM_{2.5}$ levels above the $PM_{2.5}$ NAAQS. Thus, the emissions from these sources contribute to the current and anticipated visibility impairment and the proposed emission reductions would help improve future visibility impairment.

2.2.4.1.4 Future Visibility Impairment at Mandatory Class I Federal Areas

Achieving the $PM_{2.5}$ 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 class I federal 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.[85] In the East, there are and will continue to be areas with $PM_{2.5}$ concentrations above the $PM_{2.5}$ NAAQS and where light extinction is significantly above natural background. Thus, large areas of the Eastern United States have air pollution that is causing and will continue to cause visibility problems. In the West, scenic vistas are especially important to public welfare. Although the $PM_{2.5}$ NAAQS is met in most areas outside of California, virtually the entire West is in close proximity to a scenic mandatory class I federal area protected by 169A and 169B of the CAA.

Recent modeling for the CAIR was used to project visibility conditions in mandatory class I federal areas across the country in 2015. The results for the mandatory class I federal areas suggest that these areas are predicted to continue to have visibility impairment above background on the 20% worst days in the future.

The overall goal of the regional haze program is to prevent future visibility impairment and remedy existing visibility impairment in mandatory class I federal areas. As shown by the future visibility estimates in Table 2.2-3, it is projected that there will continue to be mandatory class I federal areas with visibility levels above background in 2015. Additional emission reductions will be needed from the broad set of sources that contribute, including the engines, vessels and equipment subject to this proposed rule.[86] The reductions proposed in this action are

a part of the overall strategy to achieve the visibility goals of the Act and the regional haze program.

Table 2.2-3: Current (1998-2002) Visibility, Projected (2015) Visibility, and Natural Background Levels for the 20% Worst Days at 116 IMPROVE Sites

Class I Area Name[a]	State	1998-2002 Baseline Visibility (deciviews)[b]	2015 CAIR Control Case Visibility[c] (deciviews)	Natural Background (deciviews)
Acadia	ME	22.7	21.0	11.5
Agua Tibia	CA	23.2	23.2	7.2
Alpine Lakes	WA	18.0	17.4	7.9
Anaconda - Pintler	MT	12.3	12.2	7.3
Arches	UT	12.0	12.1	7.0
Badlands	SD	17.3	16.8	7.3
Bandelier	NM	13.2	13.2	7.0
Big Bend	TX	18.4	18.3	6.9
Black Canyon of the Gunnison	CO	11.6	11.4	7.1
Bob Marshall	MT	14.2	14.0	7.4
Boundary Waters Canoe Area	MN	20.0	19.0	11.2
Bridger	WY	11.5	11.3	7.1
Brigantine	NJ	27.6	25.4	11.3
Bryce Canyon	UT	12.0	11.9	7.0
Cabinet Mountains	MT	13.8	13.4	7.4
Caney Creek	AR	25.9	24.1	11.3
Canyonlands	UT	12.0	12.0	7.0
Cape Romain	SC	25.9	23.9	11.4
Caribou	CA	14.8	14.6	7.3
Carlsbad Caverns	NM	17.6	17.9	7.0
Chassahowitzka	FL	25.7	23.0	11.5
Chiricahua NM	AZ	13.9	13.9	6.9
Chiricahua W	AZ	13.9	13.9	6.9
Craters of the Moon	ID	14.7	14.7	7.1
Desolation	CA	12.9	12.8	7.1
Dolly Sods	WV	27.6	23.9	11.3
Dome Land	CA	20.3	19.9	7.1
Eagle Cap	OR	19.6	19.0	7.3
Eagles Nest	CO	11.3	11.4	7.1
Emigrant	CA	17.6	17.4	7.1
Everglades	FL	20.3	19.2	11.2
Fitzpatrick	WY	11.5	11.3	7.1
Flat Tops	CO	11.3	11.4	7.1
Galiuro	AZ	13.9	14.1	6.9
Gates of the Mountains	MT	11.2	10.8	7.2
Gila	NM	13.5	13.5	7.0
Glacier	MT	19.5	19.1	7.6
Glacier Peak	WA	14.0	13.8	7.8
Grand Teton	WY	12.1	12.0	7.1
Great Gulf	NH	23.2	21.2	11.3
Great Sand Dunes	CO	13.1	13.0	7.1
Great Smoky Mountains	TN	29.5	26.1	11.4
Guadalupe Mountains	TX	17.6	17.5	7.0
Hells Canyon	OR	18.1	18.0	7.3
Isle Royale	MI	21.1	20.1	11.2
James River Face	VA	28.5	25.1	11.2

Jarbidge	NV	12.6	12.8	7.1
Joshua Tree	CA	19.5	20.3	7.1
Joyce Kilmer - Slickrock	NC	29.5	26.1	11.5
Kalmiopsis	OR	14.8	14.4	7.7
Kings Canyon	CA	23.5	24.1	7.1
La Garita	CO	11.6	11.5	7.1
Lassen Volcanic	CA	14.8	14.6	7.3
Lava Beds	CA	16.6	16.5	7.5
Linville Gorge	NC	27.9	24.6	11.4
Lostwood	ND	19.6	18.7	7.3
Lye Brook	VT	23.9	21.1	11.3
Mammoth Cave	KY	30.2	27.0	11.5
Marble Mountain	CA	17.1	16.8	7.7
Maroon Bells - Snowmass	CO	11.3	11.3	7.1
Mazatzal	AZ	13.1	13.5	6.9
Medicine Lake	MT	17.7	17.1	7.3
Mesa Verde	CO	12.8	12.8	7.1
Mingo	MO	27.5	25.9	11.3
Mission Mountains	MT	14.2	14.0	7.4
Mokelumne	CA	12.9	12.8	7.1
Moosehorn	ME	21.4	20.3	11.4
Mount Hood	OR	14.0	13.7	7.8
Mount Jefferson	OR	15.7	15.2	7.8
Mount Rainier	WA	18.9	19.4	7.9
Mount Washington	OR	15.7	15.2	7.9
Mount Zirkel	CO	11.7	11.8	7.1
North Cascades	WA	14.0	14.0	7.8
Okefenokee	GA	26.4	24.7	11.5
Otter Creek	WV	27.6	24.0	11.3
Pasayten	WA	14.7	14.5	7.8
Petrified Forest	AZ	13.5	13.8	7.0
Pine Mountain	AZ	13.1	13.4	6.9
Presidential Range - Dry	NH	23.2	20.9	11.3
Rawah	CO	11.7	11.7	7.1
Red Rock Lakes	WY	12.1	12.1	7.1
Redwood	CA	16.5	16.5	7.8
Rocky Mountain	CO	14.1	14.1	7.1
Roosevelt Campobello	ME	21.4	20.1	11.4
Salt Creek	NM	17.7	17.3	7.0
San Gorgonio	CA	21.5	22.1	7.1
San Jacinto	CA	21.5	21.4	7.1
San Pedro Parks	NM	11.4	11.4	7.0
Sawtooth	ID	13.6	13.5	7.2
Scapegoat	MT	14.2	14.1	7.3
Selway - Bitterroot	MT	12.3	12.1	7.3
Seney	MI	23.8	22.6	11.4
Sequoia	CA	23.5	24.1	7.1
Shenandoah	VA	27.6	23.4	11.3
Sierra Ancha	AZ	13.4	13.7	6.9
Sipsey	AL	28.7	26.1	11.4
South Warner	CA	16.6	16.5	7.3
Strawberry Mountain	OR	19.6	19.2	7.5
Superstition	AZ	14.7	15.0	6.9
Swanquarter	NC	24.6	21.9	11.2

Sycamore Canyon	AZ	16.1	16.6	7.0
Teton	WY	12.1	12.1	7.1
Theodore Roosevelt	ND	17.6	16.8	7.3
Thousand Lakes	CA	14.8	14.6	7.3
Three Sisters	OR	15.7	15.2	7.9
UL Bend	MT	14.7	14.1	7.2
Upper Buffalo	AR	25.5	24.3	11.3
Voyageurs	MN	18.4	17.6	11.1
Weminuche	CO	11.6	11.4	7.1
West Elk	CO	11.3	11.3	7.1
Wind Cave	SD	16.0	15.4	7.2
Wolf Island	GA	26.4	24.9	11.4
Yellowstone	WY	12.1	12.1	7.1
Yolla Bolly - Middle Eel	CA	17.1	16.9	7.4
Yosemite	CA	17.6	17.4	7.1
Zion	UT	13.5	13.3	7.0

[a] 116 IMPROVE sites represent 155 of the 156 Mandatory Class I Federal Areas. One isolated Mandatory Class I Federal Area (Bering Sea, an uninhabited and infrequently visited island 200 miles from the coast of Alaska), was considered to be so remote from electrical power and people that it would be impractical to collect routine aerosol samples.[87]

[b] 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.

[c] The 2015 modeling projections are based on the Clear Air Interstate Rule analyses (EPA, 2005).

2.2.4.2 Atmospheric Deposition

Wet and dry deposition of ambient particulate matter delivers a complex mixture of metals (e.g., mercury, zinc, lead, nickel, aluminum, cadmium), organic compounds (e.g., POM, dioxins, furans) and inorganic compounds (e.g., nitrate, sulfate) to terrestrial and aquatic ecosystems. The chemical form of the compounds deposited is impacted by a variety of factors including ambient conditions (e.g., temperature, humidity, oxidant levels) and the sources of the material. Chemical and physical transformations of the particulate compounds occur in the atmosphere as well as the media onto which they deposit. These transformations in turn influence the fate, bioavailability and potential toxicity of these compounds. Atmospheric deposition has been identified as a key component of the environmental and human health hazard posed by several pollutants including mercury, dioxin and PCBs.[88]

Adverse impacts on water quality can occur when atmospheric contaminants deposit to the water surface or when material deposited on the land enters a waterbody through runoff. Potential impacts of atmospheric deposition to waterbodies include those related to both nutrient and toxic inputs. Adverse effects to human health and welfare can occur from the addition of excess particulate nitrate nutrient enrichment which contributes to toxic algae blooms and zones of depleted oxygen, which can lead to fish kills, frequently in coastal waters. Particles contaminated with heavy metals or other toxins may lead to the ingestion of contaminated fish, ingestion of contaminated water, damage to the marine ecology, and limited recreational uses. Several studies have been conducted in U.S. coastal waters and in the Great Lakes Region in which the role of ambient PM deposition and runoff is investigated.

Adverse impacts on soil chemistry and plant life have been observed for areas heavily impacted by atmospheric deposition of nutrients, metals and acid species, resulting in species shifts, loss of biodiversity, forest decline and damage to forest productivity. Potential impacts also include adverse effects to human health through ingestion of contaminated vegetation or livestock (as in the case for dioxin deposition), reduction in crop yield, and limited use of land due to contamination.

2.2.4.2.1 Acid Deposition

Acid deposition, or acid rain as it is commonly known, occurs when NOx and SO_2 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.[94] 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.

Acid deposition primarily affects bodies of water that rest atop soil with a limited ability to neutralize acidic compounds. The National Surface Water Survey (NSWS) investigated the effects of acidic deposition in over 1,000 lakes larger than 10 acres and in thousands of miles of streams. It found that acid deposition was the primary cause of acidity in 75 percent of the acidic lakes and about 50 percent of the acidic streams, and that the areas most sensitive to acid rain were the Adirondacks, the mid-Appalachian highlands, the upper Midwest and the high elevation West. The NSWS found that approximately 580 streams in the Mid-Atlantic Coastal Plain are acidic primarily due to acidic deposition. Hundreds of the lakes in the Adirondacks surveyed in the NSWS have acidity levels incompatible with the survival of sensitive fish species. Many of the over 1,350 acidic streams in the Mid-Atlantic Highlands (mid-Appalachia) region have already experienced trout losses due to increased stream acidity. Emissions from U.S. sources contribute to acidic deposition in Eastern Canada, where the Canadian government has estimated that 14,000 lakes are acidic. Acid deposition also has been implicated in contributing to degradation of high-elevation spruce forests that populate the ridges of the Appalachian Mountains from Maine to Georgia. This area includes national parks such as the Shenandoah and Great Smoky Mountain National Parks.

A study of emission trends and acidity of water bodies in the Eastern United States by the General Accounting Office (GAO) found that from 1992 to 1999 sulfates declined in 92 percent of a representative sample of lakes, and nitrate levels increased in 48 percent of the lakes sampled.[95] The decrease in sulfates is consistent with emission trends, but the increase in nitrates is inconsistent with the stable levels of nitrogen emissions and deposition. The study suggests that the vegetation and land surrounding these lakes have lost some of their previous capacity to use nitrogen, thus allowing more of the nitrogen to flow into the lakes and increase their acidity. Recovery of acidified lakes is expected to take a number of years, even where soil and vegetation have not been "nitrogen saturated," as EPA called the phenomenon in a 1995 study.[96] This situation places a premium on reductions of NOx from all sources, including Small SI and Marine SI engines, vessels and equipment in order to reduce the extent and severity of

nitrogen saturation and acidification of lakes in the Adirondacks and throughout the United States.

The NOx reductions from this rule would help reduce acid rain and acid deposition, thereby helping to reduce acidity levels in lakes and streams throughout the country and helping 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 nitrogen acid deposition will be roughly proportional to the reduction in NOx emissions, respectively, the precise impact of this proposed rule will differ across different areas.

2.2.4.2.2 Eutrophication, Nitrification and Fertilization

In recent decades, human activities have greatly accelerated nutrient impacts, such as nitrogen deposition in both aquatic and terrestrial systems. Nitrogen deposition in aquatic systems can cause excessive growth of algae and lead to degraded water quality and associated impairment of fresh water and estuarine resources for human uses.[97] Nitrogen deposition on terrestrial systems can cause fertilization and lead to ecosystem stress and species shift.

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 adversely affect fish and shellfish populations.

Deposition of nitrogen contributes to elevated nitrogen levels in waterbodies. The NO_x reductions from today's promulgated standards will help 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.

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.[98]

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.[99] A

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

In U.S. terrestrial systems, the nutrient whose supply most often sets the limit of possible plant based productivity at a given site is nitrogen. By increasing available nitrogen, overall ecosystem productivity may be expected to increase for a time, and then decline as nitrogen saturation is reached. However, because not all vegetation, organisms, or ecosystems react in the same manner to increased nitrogen fertilization, those plants or organisms that are predisposed to capitalize on any increases in nitrogen availability gain an advantage over those that are not as responsive to added nutrients, leading to a change in plant community composition and diversity. Changes to plant community composition and structure within an ecosystem are of concern because plants in large part determine the food supply and habitat types available for use by other organisms. Further, in terrestrial systems, plants serve as the integrators between above-ground and below-ground environments and influence nutrient, energy and water cycles. Because of these linkages, chronic excess nutrient nitrogen additions can lead to complex, dramatic, and severe ecosystem level responses such as changes in habitat suitability, genetic diversity, community dynamics and composition, nutrient status, energy and nutrient cycling, and frequency and intensity of natural disturbance regimes such as fire.

These types of effects have been observed both experimentally and in the field. For example, experimental additions of nitrogen to a Minnesota grassland dominated by native warm-season grasses produced a shift to low-diversity mixtures dominated by coolseason grasses over a 12 year period at all but the lowest rate of nitrogen addition.[104] Similarly, the coastal sage scrub (CSS) community in California has been declining in land area and in drought deciduous shrub density over the past 60 years, and is being replaced in many areas by the more nitrogen responsive Mediterranean annual grasses. Some 25 plant species are already extinct in California, most of them annual and perennial forbs that occurred in sites now experiencing conversion to annual grassland. As CSS converts more extensively to annual grassland dominated by invasive species, loss of additional rare species may be inevitable. Though invasive species are often identified as the main threat to rare species, it is more likely that invasive species combine with other factors, such as excess N deposition, to promote increased productivity of invasive species and resulting species shifts.

Deposition of nitrogen from the engines covered in this proposal contributes to elevated nitrogen levels in bodies of water and on land. The NOx reductions proposed in this action will reduce the airborne nitrogen deposition that contributes to eutrophication of watersheds and nitrogen saturation on land.

2.2.4.2.3 Heavy Metals

Heavy metals, including cadmium, copper, lead, chromium, mercury, nickel and zinc, have the greatest potential for influencing forest growth (PM AQCD, p. 4-87).[105] Investigation of trace metals near roadways and industrial facilities indicate that a substantial burden of heavy metals can accumulate on vegetative surfaces. Copper, zinc, and nickel have been documented to cause direct toxicity to vegetation under field conditions (PM AQCD, p. 4-75). Little research has been conducted on the effects associated with mixtures of contaminants found in ambient PM. While metals typically exhibit low solubility, limiting their bioavailability and direct toxicity, chemical transformations of metal compounds occur in the environment, particularly in the presence of acidic or other oxidizing species. These chemical changes influence the mobility and toxicity of metals in the environment. Once taken up into plant tissue, a metal compound can undergo chemical changes, accumulate and be passed along to herbivores or can re-enter the soil and further cycle in the environment.

Although there has been no direct evidence of a physiological association between tree injury and heavy metal exposures, heavy metals have been implicated because of similarities between metal deposition patterns and forest decline (PM AQCD, p. 4-76).[106] Contamination of plant leaves by heavy metals can lead to elevated soil levels. Some trace metals absorbed into the plant and can bind to the leaf tissue (PM AQCD, p. 4-75). When these leaves fall and decompose, the heavy metals are transferred into the soil.[107,108]

The environmental sources and cycling of mercury are currently of particular concern due to the bioaccumulation and biomagnification of this metal in aquatic ecosystems and the potent toxic nature of mercury in the forms in which is it ingested by people and other animals. Mercury is unusual compared with other metals in that it largely partitions into the gas phase (in elemental form), and therefore has a longer residence time in the atmosphere than a metal found predominantly in the particle phase. This property enables a portion of emitted mercury to travel far from the primary source before being deposited and accumulating in the aquatic ecosystem. Localized or regional impacts are also observed for mercury emitted from combustion sources. The major source of mercury in the Great Lakes is from atmospheric deposition, accounting for approximately eighty percent of the mercury in Lake Michigan.[109,110] Over fifty percent of the mercury in the Chesapeake Bay has been attributed to atmospheric deposition.[111] Overall, the National Science and Technology Council (NSTC, 1999) identifies atmospheric deposition as the primary source of mercury to aquatic systems. Forty-four states have issued health advisories for the consumption of fish contaminated by mercury; however, most of these advisories are issued in areas without a mercury point source.

Elevated levels of zinc and lead have been identified in streambed sediments, and these elevated levels have been correlated with population density and motor vehicle use.[112,113] Zinc and nickel have also been identified in urban water and soils. In addition, platinum, palladium, and rhodium, metals found in the catalysts of modern motor vehicles, have been measured at elevated levels along roadsides.[114] Plant uptake of platinum has been observed at these locations.

2.2.4.2.4 Polycyclic Organic Matter

Polycyclic organic matter (POM) is a byproduct of incomplete combustion and consists of organic compounds with more than one benzene ring and a boiling point greater than or equal to 100 degrees centigrade.[115] Polycyclic aromatic hydrocarbons (PAHs) are a class of POM that contains compounds which are known or suspected carcinogens.

Major sources of PAHs include mobile sources. PAHs in the environment may be present as a gas or adsorbed onto airborne particulate matter. Since the majority of PAHs are adsorbed onto particles less than 1.0 μm in diameter, long range transport is possible. However, studies have shown that PAH compounds adsorbed onto diesel exhaust particulate and exposed to ozone have half lives of 0.5 to 1.0 hours.[116]

Since PAHs are insoluble, the compounds generally are particle reactive and accumulate in sediments. Atmospheric deposition of particles is believed to be the major source of PAHs to the sediments of Lake Michigan.[117,118] Analyses of PAH deposition to Chesapeake and Galveston Bay indicate that dry deposition and gas exchange from the atmosphere to the surface water predominate.[119,120] Sediment concentrations of PAHs are high enough in some segments of Tampa Bay to pose an environmental health threat. EPA funded a study to better characterize the sources and loading rates for PAHs into Tampa Bay.[121] PAHs that enter a waterbody through gas exchange likely partition into organic rich particles and be biologically recycled, while dry deposition of aerosols containing PAHs tends to be more resistant to biological recycling.[122] Thus, dry deposition is likely the main pathway for PAH concentrations in sediments while gas/water exchange at the surface may lead to PAH distribution into the food web, leading to increased health risk concerns.

Trends in PAH deposition levels are difficult to discern because of highly variable ambient air concentrations, lack of consistency in monitoring methods, and the significant influence of local sources on deposition levels.[123] Van Metre et al. (2000) noted PAH concentrations in urban reservoir sediments have increased by 200-300% over the last forty years and correlates with increases in automobile use.[124]

Cousins et al. (1999) estimates that greater than ninety percent of semi-volatile organic compound (SVOC) emissions in the United Kingdom deposit on soil.[125] An analysis of polycyclic aromatic hydrocarbon (PAH) concentrations near a Czechoslovakian roadway indicated that concentrations were thirty times greater than background.[126]

2.2.4.2.5 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.[127] 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 absorb 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.

2.3 Gaseous Air Toxics

Small SI and Marine SI emissions contribute to ambient levels of gaseous air toxics known or suspected as human or animal carcinogens, or that have non-cancer health effects. These compounds include benzene, 1,3-butadiene, formaldehyde, acetaldehyde, acrolein, polycyclic organic matter (POM), and naphthalene. All of these compounds, except acetaldehyde, were identified as national or regional risk drivers in the 1999 National-Scale Air Toxics Assessment (NATA) and have significant inventory contributions from mobile sources. The reductions in Small SI and Marine SI emissions proposed in this rulemaking would help reduce exposure to these harmful substances.

Air toxics can cause a variety of cancer and noncancer health effects. A number of the mobile source air toxic pollutants described in this section are known or likely to pose a cancer hazard in humans. Many of these compounds also cause adverse noncancer health effects resulting from chronic,[16] subchronic,[17] or acute[18] inhalation exposures. These include neurological, cardiovascular, liver, kidney, and respiratory effects as well as effects on the immune and reproductive systems.

Benzene: The EPA's IRIS database lists benzene as a known human carcinogen (causing leukemia) by all routes of exposure, and that exposure is associated with additional health effects, including genetic changes in both humans and animals and increased proliferation of bone marrow cells in mice.[128, 129, 130] EPA states in its IRIS database that data indicate a causal relationship between benzene exposure and acute lymphocytic leukemia and suggests a relationship between benzene exposure and chronic non-lymphocytic leukemia and chronic lymphocytic leukemia. A number of adverse noncancer health effects including blood disorders, such as preleukemia and aplastic anemia, have also been associated with long-term exposure to benzene.[131, 132] The most sensitive noncancer effect observed in humans, based on current data, is the depression of the absolute lymphocyte count in blood.[133, 134] In addition, recent work, including studies sponsored by the Health Effects Institute (HEI), provides evidence that biochemical responses are occurring at lower levels of benzene exposure than previously known.[135, 136, 137, 138] EPA's IRIS program has not yet evaluated these new data.

[16]Chronic exposure is defined in the glossary of the Integrated Risk Information (IRIS) database (http://www.epa.gov/iris) as repeated exposure by the oral, dermal, or inhalation route for more than approximately 10 of the life span in humans (more than approximately 90 days to 2 years in typically used laboratory animal species).

[17]Defined in the IRIS database as exposure to a substance spanning approximately 10 of the lifetime of an organism.

[18]Defined in the IRIS database as exposure by the oral, dermal, or inhalation route for 24 hours or less.

1,3-Butadiene: EPA has characterized 1,3-butadiene as carcinogenic to humans by inhalation.[139, 140] 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; while there are insufficient data in humans from which to draw conclusions about sensitive subpopulations. 1,3-Butadiene also causes a variety of reproductive and developmental effects in mice; no human data on these effects are available. The most sensitive effect was ovarian atrophy observed in a lifetime bioassay of female mice.[141]

Formaldehyde: Since 1987, EPA has classified formaldehyde as a probable human carcinogen based on evidence in humans and in rats, mice, hamsters, and monkeys.[142] EPA's current IRIS summary provides an upper bound cancer unit risk estimate of $1.3x10^{-5}$ per $\mu g/m^3$. In other words, there is an estimated risk of about thirteen excess cancer cases in one million people exposed to 1 $\mu g/m^3$ of formaldehyde over a lifetime. EPA is currently reviewing recently published epidemiological data. For instance, research conducted by the National Cancer Institute (NCI) found an increased risk of nasopharyngeal cancer and lymphohematopoietic malignancies such as leukemia among workers exposed to formaldehyde.[143, 144] NCI is currently performing an update of these studies. A recent National Institute of Occupational Safety and Health (NIOSH) study of garment workers also found increased risk of death due to leukemia among workers exposed to formaldehyde.[145] In 2004, the working group of the International Agency for Research on Cancer (IARC) concluded that formaldehyde is carcinogenic to humans (Group 1), on the basis of sufficient evidence in humans and sufficient evidence in experimental animals—a higher classification than previous IARC evaluations. The agency is currently conducting a reassessment of the human hazard and dose-response associated with formaldehyde.

In the past 15 years there has been substantial research on the inhalation dosimetry for formaldehyde in rodents and primates by the CIIT Centers for Health Research (formerly the Chemical Industry Institute of Toxicology), with a focus on use of rodent data for refinement of the quantitative cancer dose-response assessment.[146, 147, 148] CIIT's risk assessment of formaldehyde incorporated mechanistic and dosimetric information on formaldehyde. The risk assessment analyzed carcinogenic risk from inhaled formaldehyde using approaches that are consistent with EPA's draft guidelines for carcinogenic risk assessment. In 2001, Environment Canada relied on this cancer dose-response assessment in their assessment of formaldehyde.[149] Extended follow-up of a cohort of British chemical workers did not find evidence of an increase in nasopharyngeal or lymphohematopoetic cancers, but a continuing statistically significant excess in lung cancers was reported.[150]

Based on the developments of the last decade, in 2004, EPA also relied on this cancer unit risk estimate during the development of the plywood and composite wood products national emissions standards for hazardous air pollutants (NESHAPs).[151] In these rules, EPA concluded that the CIIT work represented the best available application of the available mechanistic and dosimetric science on the dose-response for portal of entry cancers due to formaldehyde exposures. EPA is reviewing the recent work cited above from the NCI and NIOSH, as well as the analysis by the CIIT Centers for Health Research and other studies, as part of a reassessment

of the human hazard and dose-response associated with formaldehyde.

Formaldehyde exposure also causes a range of noncancer health effects, including irritation of the eyes (tearing of the eyes and increased blinking) and mucous membranes.

Acetaldehyde: Acetaldehyde is classified in EPA's IRIS database as a probable human carcinogen, based on nasal tumors in rats, and is considered moderately toxic by the inhalation, oral, and intravenous routes.[152] The primary acute effect of exposure to acetaldehyde vapors is irritation of the eyes, skin, and respiratory tract.[153] The agency is currently conducting a reassessment of the health hazards from inhalation exposure to acetaldehyde.

Acrolein: Acrolein is intensely irritating to humans when inhaled, with acute exposure resulting in upper respiratory tract irritation and congestion. EPA determined in 2003 using the 1999 draft cancer guidelines that the human carcinogenic potential of acrolein could not be determined because the available data was inadequate. No information was available on the carcinogenic effects of acrolein in humans, and the animal data provided inadequate evidence of carcinogenicity.[154]

Polycyclic Organic Matter (POM): POM is generally defined as a large class of organic compounds which have multiple benzene rings and a boiling point greater than 100 degrees Celsius. One of these compounds, naphthalene, is discussed separately below. Polycyclic aromatic hydrocarbons (PAH) are a class of POM that contain only hydrogen and carbon atoms. A number of PAHs are known or suspected carcinogens.

Recent studies have found that maternal exposures to PAHs in a population of pregnant women were associated with several adverse birth outcomes, including low birth weight and reduced length at birth as well as impaired cognitive development at age three.[155][156] EPA has not yet evaluated these recent studies.

Naphthalene: Naphthalene is found in small quantities in gasoline and diesel fuels. Naphthalene emissions have been measured in larger quantities in both gasoline and diesel exhaust and evaporative emissions from mobile sources. EPA recently released an external review draft of a reassessment of the inhalation carcinogenicity of naphthalene based on a number of recent animal carcinogenicity studies.[157] The draft reassessment recently completed external peer review.[158] California EPA has also released a new risk assessment for naphthalene, and the IARC has reevaluated naphthalene and re-classified it as Group 2B: possibly carcinogenic to humans.[159] Naphthalene also causes a number of chronic non-cancer effects in animals, including abnormal cell changes and growth in respiratory and nasal tissues.[160]

In addition to reducing VOC, NOx, CO and $PM_{2.5}$ emissions from Small SI engines and equipment and Marine SI engines and vessels the standards being proposed today would also reduce air toxics emitted from these engines, vessels and equipment thereby helping to mitigate some of the adverse health effects associated with operation of these engines, vessels and equipment.

2.4 Carbon Monoxide

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.

2.4.1 Health Effects of CO Pollution

We are relying on the data and conclusions in the EPA Air Quality Criteria Document for CO (CO Criteria Document) regarding the health effects associated with CO exposure.[161] 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.[162, 163] 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.[164] The subsequent hypoxia in brain tissue then produces behavioral effects, including decrements in continuous performance and reaction time.[165]

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.[166] Persons with heart disease are especially sensitive to carbon monoxide poisoning and may experience chest pain if they breathe the gas while exercising.[167] 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.[168]

Several epidemiological studies have shown a link between CO and premature morbidity (including angina, congestive heart failure, and other cardiovascular diseases). Several studies in the United States and Canada have also reported an association between ambient CO exposures and frequency of cardiovascular hospital admissions, especially for congestive heart failure (CHF). An association between ambient CO exposure and 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 CO Criteria Document review process and noted the possibility that the average ambient CO levels used as exposure indices in the epidemiology

studies may be surrogates for ambient air mixes impacted by combustion sources and/or other constituent toxic components of such mixes. More research will be needed to better clarify CO's role.[169]

As noted above, CO has been linked to numerous health effects. In addition to health effects from chronic exposure to ambient CO levels, acute exposures to higher levels are also a problem. Acute exposures to CO are discussed further in Section 2.5.

2.4.2 Attainment and Maintenance of the CO NAAQS

On July 3, 1995 EPA made a finding that small land-based spark-ignition engines cause or contribute to CO nonattainment (60 FR 34581, July 3, 1995). Marine spark-ignition engines, which have relatively high per engine CO emissions, can also be a source of CO emissions in CO nonattainment areas. In the preamble for this proposed rule EPA makes a finding that recreational marine engines and vessels cause or contribute to CO nonattainment and we provide information showing CO emissions from spark-ignition marine engines and vessels in the CO nonattainment areas in 2005. Spark-ignition marine engines and vessels contribute to CO nonattainment in more than one of the CO nonattainment areas.

A nonattainment area is defined in the Clean Air Act (CAA) as an area that is violating an ambient standard or is contributing to a nearby area that is violating the standard. EPA has designated nonattainment areas for the CO NAAQS by calculating air quality design values and considering other factors.[19]

There are two CO NAAQS. The 8-hour average CO NAAQS is 9 ppm, not to be exceeded more than once per year, and the 1-hour average CO NAAQS is 35 ppm, not to be exceeded more than once per year. As of October 26, 2006, there are approximately 15 million people living in 6 areas (which include 10 counties) that are designated as nonattainment for CO, see Table 2.4-1. The emission reductions proposed in this action would help areas to attain and maintain the CO NAAQS.

Table 2.4-1: Classified Carbon Monoxide Nonattainment Areas as of October 2006[a]

Area	Classification	Population (1000s)
Las Vegas, NV	serious	479
Los Angeles South Coast Air Basin	serious	14,594
El Paso, TX	moderate <= 12.7 ppm	62
Missoula, MT	moderate <= 12.7 ppm	52
Reno, NV	moderate <= 12.7 ppm	179
Total		15,365

[a] This table does not include Salem, OR which is an unclassified CO nonattainment area.

In addition to the CO nonattainment areas, there are areas that have not been designated as nonattainment where air quality monitoring may indicate a need for CO control. For example,

[19] The full details involved in calculating a CO design value are given in 40 CFR Part 50.8.

areas like Birmingham, AL and Calexico, CA have not been designated as nonattainment although monitors in these areas have recorded multiple exceedances since 1995.[170]

There are also over 54 million people living in CO maintenance areas, see Table 2.4-2.[20] Carbon monoxide maintenance areas may remain at risk for high CO episodes especially in geographic areas with unusually challenging meteorological and topographical conditions and in areas with high population growth and increasing vehicle miles traveled.

Table 2.4-2: Carbon Monoxide Maintenance Areas as of October 2006

	Number of Areas	Number of Counties	Population (1000s)
Serious	5	11	5,902
Moderate > 12.7ppm	4	19	17,576
Moderate <= 12.7ppm	30	61	23,319
Unclassified	33	41	7,544
Total	72	132	54,341

A 2003 NAS report found that in geographical areas that have achieved attainment of the NAAQS, it might still be possible for ambient concentrations of CO to sporadically exceed the standard under unfavorable conditions such as strong winter inversions. Areas like Alaska are prone to winter inversions due to their topographic and meteorologic conditions. The report further suggests that additional reductions in CO are prudent to further reduce the risk of violations in regions with problematic topography and temporal variability in meteorology.[171] The reductions in CO emissions from this proposed rule could assist areas in maintaining the CO standard.

As discussed in the preamble, Small SI engines and equipment and Marine SI engines and vessels do contribute to CO nonattainment. The CO emission benefits from this rule would help states in their strategy to attain the CO NAAQS. Maintenance of the CO NAAQS is also challenging and many areas would be able to use the emissions reductions from this proposed rule to assist in maintaining the CO NAAQS into the future.

2.5 Acute Exposure to Air Pollutants

Emissions from Small SI engines and equipment and Marine SI engines and vessels contribute to ambient concentrations of ozone, CO, air toxics and PM and acute exposures to CO and PM. As mentioned in Section II.B.4 of the preamble for this proposal, elevated exposures to

[20]The CO nonattainment and maintenance areas are listed in a Memo to the Docket titled "Nonattainment Areas and Mandatory Class I Federal Areas" and contained in Docket EPA-HQ-OAR-2004-0008.

CO from Marine SI engines and vessels have been well documented. As mentioned in Sections II.B.2 and II.B.4 of the preamble, elevated exposures to CO and PM can occur as a result of operating Small SI engines and equipment. The standards being proposed in this action can help reduce acute exposures to CO and PM from Marine SI engines and vessels and Small SI engines and equipment.

2.5.1 Exposure to CO from Marine SI Engines and Vessels

In recent years, a substantial number of carbon monoxide (CO) poisonings and deaths have occurred on and around recreational boats across the nation. The actual number of deaths attributable to CO poisoning while boating is difficult to estimate because CO-related deaths in the water may be labeled as drowning. An interagency team consisting of the National Park Service, the U.S. Department of Interior, and the National Institute for Occupational Safety and Health maintains a record of published CO-related fatal and nonfatal poisonings.[172] Between 1984 and 2004, 113 CO-related deaths and 458 non-fatal CO poisonings have been identified based on hospital records, press accounts, and other information. Deaths have been attributed to exhaust from both onboard generators and propulsion engines. Houseboats, cabin cruisers, and ski boats are the most common types of boats associated with CO poisoning cases. These incidents have prompted other federal agencies, including the United States Coast Guard and National Park Service, to issue advisory statements and other interventions to boaters to avoid activities that could lead to excessive CO exposure.[173]

CO concentrations can be extremely elevated within several meters of the exhaust port. Engineers and industrial hygienists from CDC/NIOSH and other state and federal agencies have conducted field studies of CO concentrations on and around houseboats. In one study of houseboat concentrations, CO concentrations immediately at the point of generator exhaust discharge on one houseboat averaged 0.5% (5,000 ppm), and ranged from 0.0% to 1.28% (12,800 ppm).[174] With both propulsion and generators running, time-averaged concentrations on the swim deck were 0.2 - 169 ppm at different locations on one boat's swim platform, 17-570 ppm on another's, and 0-108 on another. Other studies also show the potential for high concentrations with extreme peaks in CO concentrations in locations where boaters and swimmers can be exposed during typical boating activities, such as standing on a swim deck or swimming near a boat.

2.5.2 Exposure to CO and PM from Small SI Engines and Equipment

A large segment of the population uses small, gasoline-powered spark-ignition (SI) lawn and garden equipment on a regular basis. Emissions from many of the Small SI engines powering this equipment may lead to elevated air pollution exposures for a number of gaseous and particulate compounds, especially for individuals such as landscapers, whose occupations require the daily use of these engines and equipment.

Emission studies with lawn and garden equipment suggest a potential for high exposures during the Small SI engine operation.[175,176] Studies investigating air pollutant exposures during small engine use did report elevated personal exposure measurements related to lawn and garden

equipment use.[177,178] Bunger et al. reported elevated CO personal measurements related to chainsaw use, with short-term concentrations exceeding 400 ppm for certain cutting activities. This study evaluated personal exposures during the use of uncontrolled chainsaws. Baldauf at al. evaluated the use of lawnmowers, chainsaws and string trimmers meeting US EPA Phase 2 standards. In this study, short-term exposures during lawnmower and chainsaw use exceeded 120 ppm of CO, while string trimmer use resulted in some short-term exposures approaching 100 ppm of CO. This study also indicated that short-term $PM_{2.5}$ exposures could exceed 100 $\mu g/m^3$. Pollutant exposures were highly dependent on the operator's orientation to the engine and wind direction, as well as the activities being conducted.

These studies indicate that emissions from some lawn and garden equipment meeting EPA's current Phase 2 standards may result in exposures to certain pollutants at levels of concern for adverse health effects. The potential for elevated exposure to CO and $PM_{2.5}$ for operators of Small SI engines and equipment would be reduced by this proposed rule.

Chapter 2 References

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