CHAPTER 1: Health and Welfare Concerns

The engines and vehicles that would be subject to the proposed standards generate emissions of HC, CO, PM and air toxics that contribute to ozone and CO nonattainment as well as adverse health effects associated with ambient concentrations of PM and air toxics. Elevated emissions from those recreational vehicles that operate in national parks (e.g., snowmobiles) contribute to visibility impairment. This section summarizes the general health effects of these substances. In it, we present information about these health and environmental effects, air quality modeling results, and inventory estimates pre- and post-control.

1.1 Ozone

1.1.1 General Background

Ground-level ozone, the main ingredient in smog, is formed by complex chemical reactions of volatile organic compounds (VOC) and NOx in the presence of heat and sunlight. Ozone forms readily in the lower atmosphere, usually during hot summer weather. Volatile organic compounds are emitted from a variety of sources, including motor vehicles, chemical plants, refineries, factories, consumer and commercial products, and other industrial sources. Volatile organic compounds also are emitted by natural sources such as vegetation. Oxides of nitrogen are emitted largely from motor vehicles, off-highway equipment, power plants, and other sources of combustion. Hydrocarbons (HC) are a large subset of VOC, and to reduce mobile source VOC levels we set maximum emissions limits for hydrocarbon as well as particulate matter emissions.

The science of ozone formation, transport, and accumulation is complex. Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions involving NOx, VOC, heat, and sunlight.[1] As a result, differences in weather patterns, as well as NOx and VOC levels, contribute to daily, seasonal, and yearly differences in ozone concentrations and differences from city to city. Many of the chemical reactions that are part of the ozone-forming cycle are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up, resulting in higher ambient ozone levels than typically would occur on a single high temperature day. Further complicating matters, ozone also can be transported into an area from pollution sources found hundreds of miles upwind, resulting in elevated ozone levels even in areas with low local VOC or NOx emissions.

On the chemical level, NOx and VOC are the principal precursors to ozone formation. 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 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.

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

1.1.2 Health and Welfare Effects of Ozone and Its Precursors

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

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

children, can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during prolonged periods of moderate exertion.

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

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

Volatile organic compounds emissions are detrimental not only for their role in forming ozone, but also for their role as air toxics. Some VOCs emitted from motor vehicles are toxic compounds. At elevated concentrations and exposures, human health effects from air toxics can range from respiratory effects to cancer. Other health impacts include neurological developmental and reproductive effects. The toxicologically significant VOCs emitted in substantial quantities from the engines that are the subject of this proposal are discussed in more detail in Section 1.4, below.

1.1.3 Additional Health and Welfare Effects of NOx Emissions

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

further in Sections 1.6.4 and 1.6.5, below.

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

1.1.4 Ozone Nonattainment

The current primary and secondary ozone National Ambient Air Quality Standard (NAAQS) is 0.12 ppm daily maximum 1-hour concentration, not to be exceeded more than once per year on average. The determination that an area is at risk of exceeding the ozone standard in the future was made for all areas with current design values grater than or equal to 0.125 ppm (or within a 10 percent margin) and with modeling evidence that exceedances will persist into the future.

Ground level ozone today remains a pervasive pollution problem in the United States. In 1999, 90.8 million people (1990 census) lived in 31 areas designated nonattainment under the 1-hour ozone NAAQS.[7] This sharp decline from the 101 nonattainment areas originally identified under the Clean Air Act Amendments of 1990 demonstrates the effectiveness of the last decade's worth of emission-control programs. However, elevated ozone concentrations remain a serious public health concern throughout the nation.

Over the last decade, declines in ozone levels were found mostly in urban areas, where emissions are heavily influenced by controls on mobile sources and their fuels. Twenty-three metropolitan areas have realized a decline in ozone levels since 1989, but at the same time ozone levels in 11 metropolitan areas with 7 million people have increased.[8] Regionally, California and the Northeast have recorded significant reductions in peak ozone levels, while four other regions (the Mid-Atlantic, the Southeast, the Central and Pacific Northwest) have seen ozone levels increase.

The highest ambient concentrations are currently found in suburban areas, consistent with downwind transport of emissions from urban centers. Concentrations in rural areas have risen to the levels previously found only in cities. Particularly relevant to this proposal, ozone levels at 17 of our National Parks have increased, and in 1998, ozone levels in two parks, Shenandoah National Park and the Great Smoky Mountains National Park, were 30 to 40 percent higher than the ozone NAAQS over the last decade.[9]

To estimate future ozone levels, we refer to the modeling performed in conjunction with the final rule for our most recent heavy-duty highway engine and fuel standards.[10] We performed a series of ozone air quality modeling simulations for nearly the entire Eastern U.S. covering metropolitan areas from Texas to the Northeast.[11] This ozone air quality model was based upon

the same modeling system as was used in the Tier 2 air quality analysis, with the addition of updated inventory estimates for 2007 and 2030. The model simulations were performed for several emission scenarios, and the model outputs were combined with current air quality data to identify areas expected to exceed the ozone NAAQS in 2007, 2020, and 2030.[12] The results of this modeling are contained in Table 1.1-1. Areas presented in Table 1.1-1 have 1997-99 air quality data indicating violations of the 1-hour ozone NAAQS, or are within 10 percent of the standard, are predicted to have exceedance in 2007, 2020, or 2030. An area was considered likely to have future exceedances if exceedances were predicted by the model, and the area is currently violating the 1-hour standard, or is within 10 percent of violating the 1-hour standard. Table 1.1-1 shows that 37 areas with a 1999 population of 91 million people are at risk of exceeding the 1-hour ozone standard in 2007.

Table 1.1-1: Eastern Metropolitan Areas with Modeled Exceedances of the 1-Hour Ozone Standard in 2007, 2020, or 2030 (Includes all emission controls through HD07 standards)

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

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

With regard to future ozone levels, our photochemical ozone modeling for 2020 predicts exceedances of the 1-hour ozone standard in 32 areas with a total of 89 million people (1999 census; see Table 1.1-1). We expect that the control strategies contained in this proposal for nonroad engines will further assist state efforts already underway to attain and maintain the 1-hour ozone standard.

The inventories that underlie this predictive modeling for 2020 and 2030 include reductions from all current and committed to federal, state and local control programs, including the recently promulgated NOx and PM standards for heavy-duty vehicles and low sulfur diesel fuel. The geographic scope of these areas at risk of future exceedances underscores the need for additional, nationwide controls of ozone precursors.

It should be noted that this modeling did not attempt to examine the prospect of areas attaining or maintaining the ozone standard with possible future controls (i.e., controls beyond current or committed federal, State and local controls). Therefore, this information should be interpreted as indicating what areas are at risk of ozone violations in 2007, 2020 or 2030 without federal or state measures that may be adopted and implemented in the future. We expect many of these areas to adopt additional emission reduction programs, but we are unable to quantify or rely upon future reductions from additional State programs since they have not yet been adopted.

1.1.5 Public Health and Welfare Concerns from Prolonged and Repeated Exposures to Ozone

In addition to the health effects described above, there exists a large body of scientific literature that shows that harmful effects can occur from sustained levels of ozone exposure much lower than 0.125 ppm. Studies of prolonged exposures, those lasting about 7 hours, showed health effects from exposures to ozone concentrations as low as 0.08 ppm. Prolonged and repeated exposures to ozone at these levels are common in areas that do not attain the 1-hour NAAQS, and also occur in areas where ambient concentrations of ozone are in compliance with the 1-hour NAAQS.

Prolonged exposure to levels of ozone below the NAAQS have been reported to cause or be statistically associated with transient pulmonary function responses, transient respiratory symptoms, effects on exercise performance, increased airway responsiveness, increased susceptibility to respiratory infection, increased hospital and emergency room visits, and transient pulmonary respiratory inflamation. Such acute health effects have been observed following prolonged exposures at moderate levels of exertion at concentrations of ozone as low as 0.08 ppm, the lowest concentration tested. The effects are more pronounced as concentrations increase, affecting more subjects or having a greater effect on a given subject in terms of functional changes or symptoms. A detailed summary and discussion of the large body of ozone health effects research may be found in Chapters 6 through 9 (Volume 3) of the 1996 Criteria Document for ozone.[13] Monitoring data indicates that 333 counties in 33 states exceed these levels in 1997-99.[14]

To provide a quantitative estimate of the projected number of people anticipated to reside in areas in which ozone concentrations are predicted to exceed the 8-hour level of 0.08 to 0.12 ppm or higher for multiple days, we performed regional modeling using the variable-grid Urban Airshed Model (UAM-V).[15] UAM-V is a photochemical grid model that numerically simulates the effects of emissions, advection, diffusion, chemistry, and surface removal processes on pollutant concentrations within a 3-dimensional grid. As with the previous modeling analysis, the inventories that underlie this predictive modeling include reductions from all current and committed to federal, state and local control programs, including the recently promulgated NOx and PM standards for heavy-duty vehicles and low sulfur diesel fuel. This modeling forecast that 111 million people are predicted to live in areas that areas at risk of exceeding these moderate ozone levels for prolonged periods of time in 2020 after accounting for expected inventory reductions due to controls on light- and heavy-duty on-highway vehicles; that number is expected to increase to 125 million in 2030.[16] Prolonged and repeated ozone concentrations at these levels are common in areas throughout the country, and are found both in areas that are exceeding, and areas that are not exceeding, the 1-hour ozone standard. Areas with these high concentrations are more widespread than those in nonattainment for that 1-hour ozone standard.

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

1.2 Carbon Monoxide

1.2.1 General Background

Unlike many gases, CO is odorless, colorless, tasteless, and nonirritating. Carbon monoxide results from incomplete combustion of fuel and is emitted directly from vehicle tailpipes. Incomplete combustion is most likely to occur at low air-to-fuel ratios in the engine. These conditions are common during vehicle starting when air supply is restricted ("choked"), when cars are not tuned properly, and at high altitude, where "thin" air effectively reduces the amount of oxygen available for combustion (except in cars that are designed or adjusted to compensate for altitude). 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.

1.2.2 Health Effects of CO

Carbon monoxide enters the bloodstream through the lungs and forms carboxyhemoglobin, a compound that inhibits the blood's capacity to carry oxygen to organs and tissues. 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. 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. Persons with heart disease are especially sensitive to carbon monoxide poisoning and may experience chest pain if they breathe the gas while exercising. 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. More importantly to many individuals is the frequent exposure of individuals to exhaust emissions from engines operating indoors. The Occupational Safety and Health Administration sets standards regulating the concentration of indoor pollutants, but high local CO levels are still commonplace.

Several recent epidemiological studies have shown a link between CO and premature morbidity (including angina, congestive heart failure, and other cardiovascular diseases). Several studies in the United States and Canada have also reported an association of ambient CO exposures with frequency of cardiovascular hospital admissions, especially for congestive heart failure (CHF). An association of ambient CO exposure with mortality has also been reported in epidemiological studies, though not as consistently or specifically as with CHF admissions. EPA reviewed these studies as part of the Criteria Document review process. The CO Criteria Document (EPA 600/P-99/001F) contains additional information about the health effects of CO, human exposure, and air quality. It was published as a final document and made available to the public in August 2000 (www.epa.gov/ncea/co/).

1.2.3 CO Nonattaiment

The current primary NAAQS for CO are 35 parts per million for the one-hour average and 9 parts per million for the eight-hour average. These values are not to be exceeded more than once per year. Air quality carbon monoxide value is estimated using EPA guidance for calculating design values. In 1999, 30.5 million people (1990 census) lived in 17 areas designated nonattainment under the CO NAAQS.[17]

Snowmobiles, which have relatively high per engine CO emissions, can be a significant source of ambient CO levels in CO nonattainment areas. Several states that contain CO nonattainment areas also have large populations of registered snowmobiles. This is shown in Table 1.2-1. A review of snowmobile trail maps indicates that snowmobiles are used in these CO nonattainment areas or in adjoining counties.[18] These include the Mt. Spokane and Riverside trails near the Spokane Washington CO nonattainment area; the Larimer trails near the Fort Collins, Colorado CO nonattainment area; and the Hyatt Lake, Lake of the Woods, and Cold Springs trails near the Klamath Falls and Medford, Oregon CO nonattainment area. There are

also trails in Missoula County, Montana that demonstrate snowmobile use in the Missoula, Montana CO nonattainment area. While Colorado has a large snowmobile population, the snowmobile trails are fairly distant from the Colorado Springs CO nonattainment area.[19]

Table 1.2-1
Snowmobile Use in Selected CO Nonattainment Areas

City and State	CO Nonattainment Classification	1998 State snowmobile population[a]
Fairbanks, AK	Serious	12,997
Spokane, WA	Serious	32,274
Colorado Springs, CO	Moderate	28,000
Fort Collins, CO	Moderate	
Klamath Falls, OR	Moderate	13,426
Medford, OR	Moderate	
Missoula, MT	Moderate	14,361

[a]Source: Letter from International Snowmobile Manufacturers Association to US-EPA, July 8, 1999, Docket A-2000-01, Document No. II-G-136

Exceedances of the 8-hour CO standard were recorded in three of these seven CO nonattainment areas located in the northern portion of the country over the five year period from 1994 to 1999: Fairbanks, AK; Medford, OR; and Spokane, WA.[20] Given the variability in CO ambient concentrations due to weather patterns such as inversions, the absence of recent exceedances for some of these nonattainment areas should not be viewed as eliminating the need for further reductions to consistently attain and maintain the standard. A review of CO monitor data in Fairbanks from 1986 to 1995 shows that while median concentrations have declined steadily, unusual combinations of weather and emissions have resulted in elevated ambient CO concentrations well above the 8-hour standard of 9 ppm. Specifically, a Fairbanks monitor recorded average 8-hour ambient concentrations at 16 ppm in 1988, around 9 ppm from 1990 to 1992, and then a steady increase in CO ambient concentrations at 12, 14 and 16 ppm during some extreme cases in 1993, 1994 and 1995, respectively.[21]

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

more stringent standards for certain light-duty trucks (LDT2s).[23] CO standards for passenger cars and other light-duty trucks and heavy-duty vehicles did not change as a result of other recent rulemakings. As described in Section 1.5, below, the engines subject to this rule currently account for about 7 percent of the mobile source CO inventory; this is expected to increase to 10 percent by 2020 without the emission controls proposed in this action.

The state of Alaska recently submitted draft CO attainment SIPs to the Agency for the Fairbanks CO nonattainment area. Fairbanks is located in a mountain valley with a much higher potential for air stagnation than cities within the contiguous United States. Nocturnal inversions that give rise to elevated CO concentrations can persist 24-hours a day due to the low solar elevation, particularly in December and January. These inversions typically last from 2 to 4 days (Bradley et al., 1992), and thus inversions may continue during hours of maximum CO emissions from mobile sources. Despite the fact that snowmobiles are largely banned in CO nonattainment areas by the state, the state estimated that snowmobiles contributed 0.3 tons/day in 1995 to Fairbanks' CO nonattainment area or 1.2 percent of a total inventory of 23.3 tons per day in 2001.[24] While Fairbanks has made significant progress in reducing ambient CO concentrations, existing climate conditions make achieving and maintaining attainment challenging. Fairbanks failed to attain the CO NAAQS by the applicable deadline of December 21, 2000, and EPA approved a one-year extension in May of 2001.[25]

In addition to the health effects that can result from exposure to carbon monoxide, this pollutant also can contribute to ground level ozone formation.[26] Recent studies in atmospheric chemistry in urban environments suggest CO can react with hydrogen-containing radicals, leaving fewer of these to combine with non-methane hydrocarbons and thus leading to increased levels of ozone. Few analyses have been performed that estimate these effects, but a study of an ozone episode in Atlanta, GA in 1988 found that CO accounted for about 17.5 percent of the ozone formed (compared to 82.5 percent for volatile organic compounds). While different cities may have different results, the effects of CO emissions on ground level ozone are not insignificant. The engines that are the subject of the proposed standards are contributors to these effects in urban areas, particularly because their per engine emissions are so high. For example, CO emissions from a off-highway motorcycle are high relative to a passenger car, (32 g/mi compared to 4.2 g/mi).

1.3 Particulate Matter

1.3.1 General Background

Particulate pollution is a problem affecting urban and non-urban localities in all regions of the United States. Nonroad engines and vehicles that would be subject to the proposed standards contribute to ambient particulate matter (PM) levels in two ways. First, they contribute through direct emissions of particulate matter. Second, they contribute to indirect formation of PM through their emissions of organic carbon, especially HC. Organic carbon accounts for between 27 and 36 percent of fine particle mass depending on the area of the country.

Particulate matter 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. All particles equal to and less than 10 microns are called PM_{10}. Fine particles can be generally defined as those particles with an aerodynamic diameter of 2.5 microns or less (also known as $PM_{2.5}$), and coarse fraction particles are those particles with an aerodynamic diameter greater than 2.5 microns, but equal to or less than a nominal 10 microns.

Manmade emissions that contribute to airborne particulate matter result principally from combustion sources (stationary and mobile sources) and fugitive emissions from industrial processes and non-industrial processes (such as roadway dust from paved and unpaved roads, wind erosion from cropland, construction, etc.). Human-generated sources of particles include a variety of stationary sources (including power generating plants, industrial operations, manufacturing plants, waste disposal) and mobile sources (light- and heavy-duty on-road vehicles, and off-highway vehicles such as construction, farming, industrial, locomotives, marine vessels and other sources). Natural sources also contribute to particulate matter in the atmosphere and include sources such as wind erosion of geological material, sea spray, volcanic emissions, biogenic emanation (e.g., pollen from plants, fungal spores), and wild fires.

The chemical and physical properties of PM vary greatly with time, region, meteorology, and source category. Particles may be emitted directly to the atmosphere (primary particles) or may be formed by transformations of gaseous emissions of sulfur dioxide, nitrogen oxides or volatile organic compounds (secondary particles). Secondary PM is dominated by sulfate in the eastern U.S. and nitrate in the western U.S.[27] The vast majority (>90 percent) of the direct mobile source PM emissions and their secondary formation products are in the fine PM size range. Mobile sources can reasonably be estimated to contribute to ambient secondary nitrate and sulfate PM in proportion to their contribution to total NOx and SOx emissions.

Table 1.3-1: Percent Contribution to $PM_{2.5}$ by Component, 1998

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

Source: National Air Quality and Emissions Trends Report, 1998, March, 2000, at 28. This document is available at http://www.epa.gov/oar/aqtrnd98/. Relevant pages of this report can be found in Memorandum to Air Docket A-2000-01 from Jean Marie Revelt, September 5, 2001, Document No. II-A-63.

1.3.2 Health and Welfare Effects of PM

Particulate matter can adversely affect human health and welfare. Discussions of the health and welfare effects associated with ambient PM can be found in the Air Quality Criteria for Particulate Matter.[28]

Key EPA findings regarding the health risks posed by ambient PM are summarized as follows:

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

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

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

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

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

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

may increase individuals' susceptibility to respiratory infections.

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

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

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

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

With respect to welfare or secondary effects, fine particles have been clearly associated with the impairment of visibility over urban areas and large multi-State regions. Particles also contribute to soiling and materials damage. Components of particulate matter (e.g., sulfuric or nitric acid) also contribute to acid deposition, nitrification of surface soils and water eutrophication of surface water.

1.3.3 PM Nonattainment

The NAAQS for PM_{10} was established in 1987. According to these standards, the short term (24-hour) standard of 150 $\mu g/m^3$ is not to be exceeded more than once per year on average over three years. The long-term standard specifies an expected annual arithmetic mean not to exceed 50 $\mu g/m^3$ over three years.

The most recent PM_{10} monitoring data indicate that 14 designated PM_{10} nonattainment areas with a projected population of 23 million violated the PM_{10} NAAQS in the period 1997-1999. Table 1.3-2 lists the 14 areas, and also indicates the PM_{10} nonattainment classification, and 1999 projected population for each PM_{10} nonattainment area. The projected population in 1999 was based on 1990 population figures which were then increased by the amount of population growth in the county from 1990 to 1999.

Table 1.3-2: PM_{10} Nonattainment Areas Violating the PM_{10} NAAQS in 1997- 1999

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

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

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

In addition to the 14 PM_{10} nonattainment areas that are currently violating the PM_{10} NAAQS listed in Table 1.3-2, there are 25 unclassifiable areas that have recently recorded ambient concentrations of PM_{10} above the PM_{10} NAAQS. EPA adopted a policy in 1996 that allows areas with PM_{10} exceedances that are attributable to natural events to retain their designation as unclassifiable if the State is taking all reasonable measures to safeguard public health regardless of the sources of PM_{10} emissions. Areas that remain unclassifiable areas are not required under the Clean Air Act to submit attainment plans, but we work with each of these areas to understand the nature of the PM_{10} problem and to determine what best can be done to reduce it. With respect to the monitored violations reported in 1997-99 in the 25 areas designated as unclassifiable, we have not yet excluded the possibility that factors such as a one-time monitoring upset or natural events, which ordinarily would not result in an area being designated as nonattainment for PM_{10}, may be responsible for the problem. Emission reductions from today's action will assist these currently unclassifiable areas to achieve ambient PM_{10} concentrations below the current PM_{10} NAAQS.

Current 1999 $PM_{2.5}$ monitored values, which cover about a third of the nation's counties, indicate that at least 40 million people live in areas where long-term ambient fine particulate matter levels are at or above 16 $\mu g/m^3$ (37 percent of the population in the areas with monitors).[29] This 16 $\mu g/m^3$ threshold is the low end of the range of long term average $PM_{2.5}$ concentrations in cities where statistically significant associations were found with serious health effects, including premature mortality.[30] To estimate the number of people who live in areas where long-term ambient fine particulate matter levels are at or above 16 $\mu g/m^3$ but for which there are no

monitors, we can use modeling. According to our national modeled predictions, there were a total of 76 million people (1996 population) living in areas with modeled annual average $PM_{2.5}$ concentrations at or above 16 $\mu g/m^3$ (29 percent of the population).[31]

To estimate future $PM_{2.5}$ levels, we refer to the modeling performed in conjunction with the final rule for our most recent heavy-duty highway engine and fuel standards using EPA's Regulatory Model System for Aerosols and Deposition (REMSAD).[32] The most appropriate method of making these projections relies on the model to predict changes between current and future states. Thus, we have estimated future conditions only for the areas with current $PM_{2.5}$ monitored data (which covers about a third of the nation's counties). For these counties, REMSAD predicts the current level of 37 percent of the population living in areas where fine PM levels are at or above 16 μg/m3 to increase to 49 percent in 2030.[33]

Emissions of HCs from snowmobiles contribute to secondary formation of fine particulate matter which can cause a variety of adverse health and welfare effects, including regional haze discussed in Section 1.6 below. For 20 counties across nine states, snowmobile trails are found within or near counties that registered ambient PM 2.5 concentrations at or above 15 μg/m3, the level of the revised national ambient air quality standard for fine particles.[34] These counties are listed in Table 1.3.-3. To obtain the information about snowmobile trails contained in Table 1.3.-3, we consulted snowmobile trail maps that were supplied by various states.[35]

Table 1.3-3
Counties with Annual $PM_{2.5}$ Levels Above 16 μg/m3 and Snowmobile Trails

State	$PM_{2.5}$ Exceedance County	County with Snowmobile Trails	Proximity to $PM_{2.5}$ Exceedance County
Ohio	Mahoning	Mahoning	—
	Trumbull	Trumbull	—
	Summit	Summit	—
	Montgomery	Montgomery	—
	Portage	Portage	—
	Franklin	Delaware	Borders North
	Marshall/Ohio (WV)	Belmont	Borders West
Montana	Lincoln	Lincoln	—
California	Tulane	Tulane	—
	Butte	Butte	—
	Fresno	Fresno	—
	Kern	Kern	—
Minnesota	Washington	Washington	—
	Wright	Wright	—
Wisconsin	Waukesha	Waukesha	—
	Milwaukee	Milwaukee	—
Oregon	Jackson	Douglas	Borders NNE
	Klamath	Douglas	Borders North
Pennsylvania	Washington	Layette	Borders East
		Somerset	—
Illinois	Rock Island	Rock Island	—
		Henry	Borders East
Iowa	Rock Island (IL)	Dubuque	Borders West

1.4 Gaseous Air Toxics

In addition to the human health and welfare impacts described above, emissions from the engines covered by this proposal also contain several other substances that are known or suspected human or animal carcinogens, or have serious noncancer health effects. These include

benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and acrolein. The health effects of these air toxics are described in more detail in Chapter 1 of the Draft Regulatory Support Document for this rule. Additional information can also be found in the Technical Support Document four our final Mobile Source Air Toxics rule.[36]

1.4.1 Benzene

Benzene is an aromatic hydrocarbon which is present as a gas in both exhaust and evaporative emissions from motor vehicles. Benzene in the exhaust, expressed as a percentage of total organic gases (TOG), varies depending on control technology (e.g., type of catalyst) and the levels of benzene and other aromatics in the fuel, but is generally about three to five percent. The benzene fraction of evaporative emissions depends on control technology and fuel composition and characteristics (e.g., benzene level and the evaporation rate), and is generally about one percent.[37]

EPA has recently reconfirmed that benzene is a known human carcinogen by all routes of exposure.[38] Respiration is the major source of human exposure. Long-term respiratory exposure to high levels of ambient benzene concentrations has been shown to cause cancer of the tissues that form white blood cells. Among these are acute nonlymphocytic leukemia,[39] chronic lymphocytic leukemia and possibly multiple myeloma (primary malignant tumors in the bone marrow), although the evidence for the latter has decreased with more recent studies.[40,41] Leukemias, lymphomas, and other tumor types have been observed in experimental animals exposed to benzene by inhalation or oral administration. Exposure to benzene and/or its metabolites has also been linked with genetic changes in humans and animals[42] and increased proliferation of mouse bone marrow cells.[43] The occurrence of certain chromosomal changes in individuals with known exposure to benzene may serve as a marker for those at risk for contracting leukemia.[44]

A number of adverse noncancer health effects, blood disorders such as preleukemia and aplastic anemia, have also been associated with low-dose, long-term exposure to benzene.[45] People with long-term exposure to benzene may experience harmful effects on the blood-forming tissues, especially the bone marrow. These effects can disrupt normal blood production and cause a decrease in important blood components, such as red blood cells and blood platelets, leading to anemia (a reduction in the number of red blood cells), leukopenia (a reduction in the number of white blood cells), or thrombocytopenia (a reduction in the number of blood platelets, thus reducing the ability for blood to clot). Chronic inhalation exposure to benzene in humans and animals results in pancytopenia,[46] a condition characterized by decreased numbers of circulating erythrocytes (red blood cells), leukocytes (white blood cells), and thrombocytes (blood platelets).[47,48] Individuals that develop pancytopenia and have continued exposure to benzene may develop aplastic anemia,[49] whereas others exhibit both pancytopenia and bone marrow hyperplasia (excessive cell formation), a condition that may indicate a preleukemic state.[50 51] The most sensitive noncancer effect observed in humans is the depression of absolute lymphocyte counts in the circulating blood.[52]

1.4.2 1,3-Butadiene

1,3-Butadiene is formed in vehicle exhaust by the incomplete combustion of fuel. It is not present in vehicle evaporative emissions, because it is not present in any appreciable amount in fuel. 1,3-Butadiene accounts for 0.4 to 1.0 percent of total organic gas exhaust, depending on control technology and fuel composition.[53]

1,3-Butadiene was classified by EPA as a Group B2 (probable human) carcinogen in 1985.[54] This classification was based on evidence from two species of rodents and epidemiologic data. In the EPA1998 draft Health Risk Assessment of 1,3-Butadiene, that was reviewed by the Science Advisory Board (SAB), the EPA proposed that 1,3-butadiene is a known human carcinogen based on human epidemiologic, laboratory animal data, and supporting data such as the genotoxicity of 1,3-butadiene metabolites.[55] The Environmental Health Committee of EPA's Scientific Advisory Board (SAB) reviewed the draft document in August 1998 and recommended that 1,3-butadiene be classified as a probable human carcinogen, stating that designation of 1,3-butadiene as a known human carcinogen should be based on observational studies in humans, without regard to mechanistic or other information.[56] In applying the 1996 proposed Guidelines for Carcinogen Risk Assessment, the Agency relies on both observational studies in humans as well as experimental evidence demonstrating causality, and therefore the designation of 1,3-butadiene as a known human carcinogen remains applicable.[57] The Agency has revised the draft Health Risk Assessment of 1,3-Butadiene based on the SAB and public comments. The draft Health Risk Assessment of 1,3-Butadiene will undergo the Agency consensus review, during which time additional changes may be made prior to its public release and placement on the Integrated Risk Information System (IRIS).

1,3-Butadiene also causes a variety of noncancer reproductive and developmental effects in mice and rats (no human data) when exposed to long-term, low doses of butadiene.[58] The most sensitive effect was reduced litter size at birth and at weaning. These effects were observed in studies in which male mice exposed to 1,3-butadiene were mated with unexposed females. In humans, such an effect might manifest itself as an increased risk of spontaneous abortions, miscarriages, still births, or very early deaths. Long-term exposures to 1,3-butadiene should be kept below its reference concentration of 4.0 microgram/m^3 to avoid appreciable risks of these reproductive and developmental effects.[59] EPA has developed a draft chronic, subchronic, and acute RfC values for 1,3-butadiene exposure as part of the draft risk characterization mentioned above. The RfC values will be reported on IRIS.

1.4.3 Formaldehyde

Formaldehyde is the most prevalent aldehyde in vehicle exhaust. It is formed from incomplete combustion of both gasoline and diesel fuel and accounts for one to four percent of total organic gaseous emissions, depending on control technology and fuel composition. It is not found in evaporative emissions.

Formaldehyde exhibits extremely complex atmospheric behavior.[60] It is formed by the

atmospheric oxidation of virtually all organic species, including biogenic (produced by a living organism) hydrocarbons. Mobile sources contribute both primary formaldehyde (emitted directly from motor vehicles) and secondary formaldehyde (formed from photooxidation of other VOCs emitted from vehicles).

EPA has classified formaldehyde as a probable human carcinogen based on limited evidence for carcinogenicity in humans and sufficient evidence of carcinogenicity in animal studies, rats, mice, hamsters, and monkeys.[61] Epidemiological studies in occupationally exposed workers suggest that long-term inhalation of formaldehyde may be associated with tumors of the nasopharyngeal cavity (generally the area at the back of the mouth near the nose), nasal cavity, and sinus. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to formaldehyde causes an increase in the incidence of squamous (epithelial) cell carcinomas (tumors) of the nasal cavity. The distribution of nasal tumors in rats suggests that not only regional exposure but also local tissue susceptibility may be important for the distribution of formaldehyde-induced tumors.[62] Research has demonstrated that formaldehyde produces mutagenic activity in cell cultures.[63]

Formaldehyde exposure also causes a range of noncancer health effects. At low concentrations (0.05-2.0 ppm), irritation of the eyes (tearing of the eyes and increased blinking) and mucous membranes is the principal effect observed in humans. At exposure to 1-11 ppm, other human upper respiratory effects associated with acute formaldehyde exposure include a dry or sore throat, and a tingling sensation of the nose. Sensitive individuals may experience these effects at lower concentrations. Forty percent of formaldehyde-producing factory workers reported nasal symptoms such as rhinitis (inflammation of the nasal membrane), nasal obstruction, and nasal discharge following chronic exposure.[64] In persons with bronchial asthma, the upper respiratory irritation caused by formaldehyde can precipitate an acute asthmatic attack, sometimes at concentrations below 5 ppm.[65] Formaldehyde exposure may also cause bronchial asthma-like symptoms in non-asthmatics.[66 67]

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

1.4.4 Acetaldehyde

Acetaldehyde is a saturated aldehyde that is found in vehicle exhaust and is formed as a result of incomplete combustion of both gasoline and diesel fuel. It is not a component of evaporative emissions. Acetaldehyde comprises 0.4 to 1.0 percent of total organic gas exhaust, depending on control technology and fuel composition.[68]

The atmospheric chemistry of acetaldehyde is similar in many respects to that of formaldehyde.[69] Like formaldehyde, it is produced and destroyed by atmospheric chemical transformation. Mobile sources contribute to ambient acetaldehyde levels both by their primary emissions and by secondary formation resulting from their VOC emissions. Acetaldehyde emissions are classified as a probable human carcinogen. Studies in experimental animals provide sufficient evidence that long-term inhalation exposure to acetaldehyde causes an increase in the incidence of nasal squamous cell carcinomas (epithelial tissue) and adenocarcinomas (glandular tissue).[70] [71]

Noncancer effects in studies with rats and mice showed acetaldehyde to be moderately toxic by the inhalation, oral, and intravenous routes.[72] [73] [74] The primary acute effect of exposure to acetaldehyde vapors is irritation of the eyes, skin, and respiratory tract. At high concentrations, irritation and pulmonary effects can occur, which could facilitate the uptake of other contaminants. Little research exists that addresses the effects of inhalation of acetaldehyde on reproductive and developmental effects. The *in vitro* and *in vivo* studies provide evidence to suggest that acetaldehyde may be the causative factor in birth defects observed in fetal alcohol syndrome, though evidence is very limited linking these effects to inhalation exposure. Long-term exposures should be kept below the reference concentration of 9 $\mu g/m^3$ to avoid appreciable risk of these noncancer health effects.[75]

1.4.5 Acrolein

Acrolein is extremely toxic to humans from the inhalation route of exposure, with acute exposure resulting in upper respiratory tract irritation and congestion. Although no information is available on its carcinogenic effects in humans, based on laboratory animal data, EPA considers acrolein a possible human carcinogen.[76]

1.5 Inventory Contributions

1.5.1 Inventory Contribution

The contribution of emissions from the nonroad engines and vehicles that would be subject to the proposed standards to the national inventories of pollutants that are associated with the health and public welfare effects described in this chapter are considerable. To estimate nonroad engine and vehicle emission contributions, we used the latest version of our NONROAD emissions model. This model computes nationwide, state, and county emission levels for a wide variety of nonroad engines, and uses information on emission rates, operating data, and population to determine annual emission levels of various pollutants. A more detailed description of the model and our estimation methodology can be found in the Chapter 6 of this document.

Baseline emission inventory estimates for the year 2000 for the categories of engines and vehicles covered by this proposal are summarized in Table 1.5-1. This table show the relative

contributions of the different mobile-source categories to the overall national mobile-source inventory. Of the total emissions from mobile sources, the categories of engines and vehicles covered by this proposal contribute about 13 percent, 3 percent, 6 percent, and 1 percent of HC, NOx, CO, and PM emissions, respectively, in the year 2000. The results for large SI engines indicate they contribute approximately 3 percent to HC, NOx, and CO emissions from mobile sources. The results for land-based recreational engines reflect the impact of the significantly different emissions characteristics of two-stroke engines. These engines are estimated to contribute 10 percent of HC emissions and 3 percent of CO from mobile sources. Recreational CI marine contribute less than 1 percent to NOx mobile source inventories. When only nonroad emissions are considered, the engines and vehicles that would be subject to the proposed standards would account for a larger share.

Our draft emission projections for 2020 for the nonroad engines and vehicles subject to this proposal show that emissions from these categories are expected to increase over time if left uncontrolled. The projections for 2020 are summarized in Table 1.5-2 and indicate that the categories of engines and vehicles covered by this proposal are expected to contribute 33 percent, 9 percent, 9 percent, and 2 percent of HC, NOx, CO, and PM emissions in the year 2020. Population growth and the effects of other regulatory control programs are factored into these projections. The relative importance of uncontrolled nonroad engines is higher than the projections for 2000 because there are already emission control programs in place for the other categories of mobile sources which are expected to reduce their emission levels. The effectiveness of all control programs is offset by the anticipated growth in engine populations.

Table 1.5-1
Modeled Annual Emission Levels for
Mobile-Source Categories in 2000 (thousand short tons)

Category	NOx		HC		CO		PM	
	tons	percent of mobile source	tons	percent of mobile source	tons	percent of mobile source	tons	percent of mobile source
Total for engines subject to proposed standards	**343**	**3%**	**985**	**13%**	**4,870**	**6%**	**8.3**	**1.2%**
Nonroad Large SI > 19 kW	306	2%	247	3%	2,294	3%	1.6	0.2%
Recreational SI	13.0	0.10%	737	10%	2,572	3%	5.7	0.9%
Recreation Marine CI	24	0.2%	1	0%	4	0%	1	0%
Highway Motorcycles	8	0%	84	1%	329	0%	0.4	0.1%
Marine SI Evap	0	0.0%	89	1%	0	0%	0	0%
Marine SI Exhaust	32	0.2%	708	9%	2,144	3%	38	5%
Nonroad SI < 19 kW	106	0.8%	1,460	19%	18,359	24%	50	7%
Nonroad CI	2,625	20%	316	4%	1,217	2%	253	36%
Commercial Marine CI	977	7%	30	0%	129	0.2%	41	6%
Locomotive	1,192	9%	47	1%	119	0.2%	30	4%
Total Nonroad	5,275	39%	3,635	48%	26,838	35%	420	60%
Total Highway	7,981	59%	3,811	50%	49,811	64%	240	36%
Aircraft	178	1%	183	2%	1,017	1%	39	6%
Total Mobile Sources	13,434	100%	7,629	100%	77,666	100%	660	100%
Total Man-Made Sources	24,538	--	18,575	--	99,745	--	3,095	--
Mobile Source percent of Total Man-Made Sources	55%	--	41%	--	78%	--	23%	–

Table 1.5-2
Modeled Annual Emission Levels for
Mobile-Source Categories in 2020 (thousand short tons)

Category	NOx		HC		CO		PM	
	tons	percent of mobile source	tons	percent of mobile source	tons	percent of mobile source	tons	percent of mobile source
Total for engines subject to proposed standards	**552**	**9%**	**2,055**	**33%**	**8,404**	**9%**	**11.9**	**1.9%**
Nonroad Large SI >19 kW	486	8%	348	6%	2,991	3%	2.4	0.4%
Recreational SI	27.0	0.40%	1,706	28%	5,407	3%	7.5	1.2%
Recreation Marine CI	39	0.6%	1	0%	6	0%	2	0%
Highway Motorcycles	14	0%	144	2%	569	1%	0.8	0.1%
Marine SI Evap	0	0.0%	102	1%	0	0%	0	0%
Marine SI Exhaust	58	0.9%	284	5%	1,985	2%	28	4%
Nonroad SI < 19 kW	106	1.7%	986	16%	27,352	31%	77	12%
Nonroad CI	1,791	29%	142	2%	1,462	2%	261	41%
Commercial Marine CI	819	13%	35	1%	160	0.2%	46	7%
Locomotive	611	10%	35	1%	119	0.1%	21	3%
Total Nonroad	3,937	63%	3,639	59%	39,482	44%	444	70%
Total Highway	2,050	33%	2,278	37%	48,903	54%	145	23%
Aircraft	232	4%	238	4%	1,387	2%	43	7%
Total Mobile Sources	6,219	100%	6,155	100%	89,772	100%	632	100%
Total Man-Made Sources	16,195	--	16,215	--	113,440	--	3,016	--
Mobile Source percent of Total Man-Made Sources	38%	--	38%	--	79%	--	21%	–

1.5.2 Inventory Impacts on a Per Vehicle Basis

In addition to the general inventory contributions described above, the engines that would be subject to the proposed standards are more potent polluters than their highway counterparts in that they have much higher emissions on a per vehicle basis. This is illustrated in Table 1.5-3, which equates the emissions produced in one hour of operation from the different categories of equipment covered by the proposal to the equivalent miles of operation it would take for a car produced today to emit the same amount of emissions.

Table 1.5-3: Per-Vehicle Emissions Comparison

Equipment Category	Emission Comparison	Miles a Current Passenger Car Would Need to Drive to Emit the Same Amount of Pollution as the Equipment Category Emits in One Hour of Operation
Recreational Marine CI	HC+NOx	2,400
Large SI	HC+NOx	1,470
Snowmobiles	HC	24,300
Snowmobiles	CO	1,520
2-Stroke ATVs & off-road motorcycles	HC	14,850
4-Stroke ATVs & off-road motorcycles	HC	590

The per engine emissions are important because they mean that operators of these engines and vehicles, as well as those who work in their vicinity, are exposed to high levels of emissions, many of which are air toxics. These effects are of particular concern for people who operate forklifts in enclosed areas and for snowmobile riders. These effects are described in more detail in the next section.

1.6 Other Adverse Public Health and Welfare Effects Associated with Nonroad Engines and Vehicles

The previous section describes national-scale adverse public health effects associated with the nonroad engines and vehicles covered by this proposal. This section describes significant adverse health and welfare effects arising from the usage patterns of snowmobiles, large SI engines, and gasoline marine engines on the regional and local scale. Studies suggest that emissions from these engines can be concentrated in specific areas, leading to elevated ambient concentrations of particular pollutants and associated elevated personal exposures to operators and by-standers. Recreational vehicles, and particularly snowmobiles, are typically operating in rural areas such as national parks and wilderness areas, and emissions from these vehicles contribute to ambient particulate matter which is a leading component of visibility impairment. This section describes these effects. We end this section by describing two other environmental effects of nonroad emissions: acid deposition and water eutrophication and nitrification

1.6.1 Snowmobiles

In this section, we describe more localized human health and welfare effects associated with snowmobile emissions: visibility impairment and personal exposure to air toxics and CO. We describe the contribution of snowmobile HC emissions to secondary formation of fine particles, which are the leading component of visibility impairment and adverse health effects related to ambient PM2.5 concentrations greater than 16 μg/m3. We also discuss personal

exposure to CO emissions and air toxics. Gaseous air toxics are components of hydrocarbons, and CO personal exposure measurements suggest that snowmobile riders and bystanders are exposed to unhealthy levels of gaseous air toxics (e.g., benzene) and CO.

1.6.1.1 Nonroad Engines and Regional Haze

The Clean Air Act established special goals for improving visibility in many national parks, wilderness areas, and international parks. In the 1977 amendments to the Clean Air Act, Congress set as a national goal for visibility the "prevention of any future, and the remedying of any existing, impairment of visibility in mandatory class I Federal areas which impairment results from manmade air pollution" (CAA section 169A(a)(1)). The Amendments called for EPA to issue regulations requiring States to develop implementation plans that assure "reasonable progress" toward meeting the national goal (CAA Section 169A(a)(4)). EPA issued regulations in 1980 to address visibility problems that are "reasonably attributable" to a single source or small group of sources, but deferred action on regulations related to regional haze, a type of visibility impairment that is caused by the emission of air pollutants by numerous emission sources located across a broad geographic region. At that time, EPA acknowledged that the regulations were only the first phase for addressing visibility impairment. Regulations dealing with regional haze were deferred until improved techniques were developed for monitoring, for air quality modeling, and for understanding the specific pollutants contributing to regional haze.

In the 1990 Clean Air Act amendments, Congress provided additional emphasis on regional haze issues (see CAA section 169B). In 1999 EPA finalized a rule that calls for States to establish goals and emission reduction strategies for improving visibility in all 156 mandatory Class I national parks and wilderness areas. In that rule, EPA also encouraged the States to work together in developing and implementing their air quality plans. The regional haze program is designed to improve visibility and air quality in our most treasured natural areas. At the same time, control strategies designed to improve visibility in the national parks and wilderness areas will improve visibility over broad geographic areas.

Regional haze is caused by the emission from numerous sources located over a wide geographic area. Such sources include, but are not limited to, major and minor stationary sources, mobile sources, and area sources. Visibility impairment is caused by pollutants (mostly fine particles and precursor gases) directly emitted to the atmosphere by a number of activities (such as electric power generation, various industry and manufacturing processes, truck and auto emissions, construction activities, etc.). These gases and particles scatter and absorb light, removing it from the sight path and creating a hazy condition.

Some fine particles are formed when gases emitted to the air form particles as they are carried downwind (examples include sulfates, formed from sulfur dioxide, and nitrates, formed from nitrogen oxides). These activities generally span broad geographic areas and fine particles can be transported great distances, sometimes hundreds or thousands of miles. Consequently, visibility impairment is a national problem. Without the effects of pollution a natural visual

range is approximately 140 miles in the West and 90 miles in the East. However, fine particles have significantly reduced the range that people can see and in the West the current range is 33-90 miles and in the East it is only 14-24 miles.

Because of evidence that fine particles are frequently transported hundreds of miles, all 50 states, including those that do not have Class I areas, will have to participate in planning, analysis and, in many cases, emission control programs under the regional haze regulations. Even though a given State may not have any Class I areas, pollution that occurs in that State may contribute to impairment in Class I areas elsewhere. The rule encourages states to work together to determine whether or how much emissions from sources in a given state affect visibility in a downwind Class I area.

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

Nonroad engines (including construction equipment, farm tractors, boats, planes, locomotives, recreational vehicles, and marine engines) contribute significantly to regional haze. This is because there are nonroad engines in all of the states, and their emissions contain precursors of fine PM and organic carbon that are transported and contribute to the formation of regional haze throughout the country and in Class I areas specifically. As illustrated in Table 1.6-1, nonroad engines are expected to contribute 15 percent of national VOC emissions, 23 percent of national NOx emissions, 6 percent of national SOx emissions, and 14 percent of national PM10 emissions. Snowmobiles alone are estimated to emit 208,926 tons of total hydrocarbons (THC), 1,461 tons of NOx, 2,145 tons of SOx, and 5,082 tons of PM in 2007.

Table 1.6-1
National Emissions of Various Pollutants - 2007
(Thousands Short Tons)

Source	VOC		NOx		SOx		PM_{10}	
	Tons	Percent	Tons	Percent	Tons	Percent	Tons	Percent
Heavy-Duty Highway	413	3%	2,969	14%	24	0	115	4%
Light-Duty Highway	2,596	18%	2,948	14%	24	0	82	3%
Nonroad	2,115	15%	4,710	23%	1,027	6%	407	14%
Electric Gen.	35	0	4,254	21%	10,780	63%	328	12%
Point	1,639	11%	3,147	15%	3,796	22%	1,007	36 %
Area	7,466	52%	2,487	12%	1,368	8%	874	31%
TOTAL	14,265		20,516		17,019		2,814	

1.6.1.2 Snowmobiles and Visibility Impairment

As noted above, EPA issued regulations in 1980 to address Class I area visibility impairment that is "reasonably attributable" to a single source or small group of sources. In 40 CFR Part 51.301 of the visibility regulations, visibility impairment is defined as "any humanly perceptible change in visibility (light extinction, visual range, contrast, coloration) from that which would have existed under natural conditions." States are required to develop implementation plans that include long-term strategies for improving visibility in each class I area. The long-term strategies under the 1980 regulations should consist of measures to reduce impacts from local sources and groups of sources that contribute to poor air quality days in the class I area. Types of impairment covered by these regulations includes layered hazes and visible plumes. While these kinds of visibility impairment can be caused by the same pollutants and processes as those that cause regional haze, they generally are attributed to a smaller number of sources located across a smaller area. The Clean Air Act and associated regulations call for protection of visibility impairment in class I areas from localized impacts as well as broader impacts associated with regional haze.

Visibility and particle monitoring data are available for 8 Class I areas where snowmobiles are commonly used. These are: Acadia, Boundary Waters, Denali, Mount Rainier, Rocky Mountain, Sequoia and Kings Canyon, Voyageurs, and Yellowstone.[77] Visibility and fine particle data for these parks are set out in Table 1.6-2. This table shows the number of monitored days in the winter that fell within the 20-percent haziest days for each of these eight parks.

Monitors collect data two days a week for a total of about 104 days of monitored values. Thus, for a particular site, a maximum of 21 worst possible days of these 104 days with monitored values constitute the set of 20-percent haziest days during a year which are tracked as the primary focus of regulatory efforts.[78] With the exception of Denali in Alaska, we defined the snowmobile season as January 1 through March 15 and December 15 through December 31 of the same calendar year, consistent with the methodology used in the Regional Haze Rule, which is calendar-year based. For Denali in Alaska, the snowmobile season is October 1 to April 30.

Table 1.6-2
Winter Days That Fall Within the 20 Percent Haziest Days
At National Parks Used by Snowmobiles

NPS Unit	State(s)	Number of Sampled Wintertime Days Within 20 Percent Haziest Days (maximum of 21 sampled days)			
		1996	1997	1998	1999
Acadia NP	ME	4	4	2	1
Denali NP and Preserve	AK	10	10	12	9
Mount Rainier NP	WA	1	3	1	1
Rocky Mountain NP	CO	2	1	2	1
Sequoia and Kings Canyon NP	CA	4	9	1	8
Voyageurs NP (1989-1992)	MN	1989: 3	1990: 4	1991: 6	1992: 8
-- Boundary Waters USFS Wilderness Area (close to Voyaguers with recent data)	MN	2	5	1	5
Yellowstone NP	ID, MT, WY	0	2	0	0

Source: Letter from Debra C. Miller, Data Analyst, National Park Service, to Drew Kodjak, August 22, 2001. Docket No. A-2000-01, Document Number. II-B-28.

The information presented in Table 1.6-2 shows that visibility data supports a conclusion that there are at least eight Class I Areas (7 in National Parks and one in a Wilderness Area) frequented by snowmobiles with one or more wintertime days within the 20-percent haziest days of the year. For example, Rocky Mountain National Park in Colorado was frequented by about 27,000 snowmobiles during the 1998-1999 winter. Of the monitored days characterized as within the 20-percent haziest monitored days, two (2) of those days occurred during the wintertime when snowmobile emissions such as hydrocarbons contributed to visibility impairment. According to the National Park Service, "[s]ignificant differences in haziness occur at all eight sites between the averages of the clearest and haziest days. Differences in mean standard visual range on the clearest and haziest days fall in the approximate range of 115-170 km."[79]

Ambient concentrations of fine particles are the primary pollutant responsible for visibility impairment. Five pollutants are largely responsible for the chemical composition of fine particles: sulfates, nitrates, organic carbon particles, elemental carbon, and crustal material. Hydrocarbon emissions from automobiles, trucks, snowmobiles, and other industrial processes are common sources of organic carbon. The organic carbon fraction of fine particles ranges from 47 percent in Western areas such as Denali National Park, to 28 percent in Rocky Mountain National Park, to 13 percent in Acadia National Park.[80]

The contribution of snowmobiles to elemental carbon and nitrates is small. Their contribution to sulfates is a function of fuel sulfur and is small and will decrease even more as the sulfur content of their fuel decreases due to our recently finalized fuel sulfur requirements. In the winter months, however, hydrocarbon emissions from snowmobiles can be significant, as indicated in Table 1.6-3, and these HC emissions can contribute significantly to the organic carbon fraction of fine particles which are largely responsible for visibility impairment. This is because they are typically powered by two-stroke engines that emit large amounts of hydrocarbons. In Yellowstone, a park with high snowmobile usage during the winter months, snowmobile hydrocarbon emissions can exceed 500 tons per year, as much as several large stationary sources. Other parks with less snowmobile traffic are less impacted by these hydrocarbon emissions.[81]

Table 1.6-3 shows modeled tons of four pollutants during the winter season in five Class I national parks for which we have estimates of snowmobile use. The national park areas outside of Denali in Alaska are open to snowmobile operation in accordance with special regulations (36 CFR Part 7). Denali National Park permits snowmobile operation by local rural residents engaged in subsistence uses (36 CFR Part 13). Emission calculations are based on an assumed 2 hours of use per snowmobile visit at 16 hp with the exception of Yellowstone where 4 hours of use at 16 hp was assumed. The emission factors used to estimate these emissions are identical to those used by the NONROAD model. Two-stroke snowmobile emission factors are: 111 g/hp-hr HC, 296 g/hp-hr CO, 0.86 g/hp-hr NOx, and 2.7 g/hp-hr PM. These emission factors are based on a number of engine tests performed by the International Snowmobile Manufacturers Association (ISMA) and the Southwest Research Institute (SwRI). These emission factors are still under review, and the emissions estimates may change pending the outcome of that review.

Table 1.6-3
Winter Season Snowmobile Emissions (tons; 1999 Winter Season)

NPS Unit	HC	CO	NOx	PM
Denali NP and Preserve	>9.8	>26.1	>0.08	>0.24
Grand Teton NP	13.7	36.6	0.1	0.3
Rocky Mountain NP	106.7	284.7	0.8	2.6
Voyageurs NP	138.5	369.4	1.1	3.4
Yellowstone NP	492.0	1,311.9	3.8	12.0

Source: Letter from Aaron J. Worstell, Environmental Engineer, National Park Service, Air Resources Division, to Drew Kodjak, August 21, 2001, particularly Table 1. Docket No. A-2000-01, Document No. II-G-178.

Inventory analysis performed by the National Park Service for Yellowstone National Park suggests that snowmobile emissions can be a significant source of total annual mobile source emissions for the park year round. Table 1.6-4 shows that in the 1998 winter season snowmobiles contributed 64 percent, 39 percent, and 30 percent of HC, CO, and PM emissions.[82] It should be noted that the snowmobile emission factors used to estimate these contributions are currently under review, and the snowmobile emissions may be revised down. However, when the emission factors used by EPA in its NONROAD model are used, the contribution of snowmobiles to total emissions in Yellowstone is still high: 59 percent, 33 percent, and 45 percent of HC, CO and PM emissions. The University of Denver used remote-sensing equipment to estimate snowmobile HC emissions at Yellowstone during the winter of 1998-1999, and estimated that snowmobiles contribute 77% of annual hydrocarbon emissions at the park.[83] The portion of wintertime emissions attributable to snowmobiles is even higher, since all snowmobile emissions occur during the winter months.

Table 1.6-4
1998 Annual HC Emissions (tpy), Yellowstone National Park

Source	HC		CO		NOx		PM	
Coaches	2.69	0%	24.29	1%	0.42	0%	0.01	0%
Autos	307.17	33%	2,242.12	54%	285.51	88%	12.20	60%
RVs	15.37	2%	269.61	6%	24.33	7%	0.90	4%
Snowmobiles	596.22	64%	1,636.44	39%	1.79	1%	6.07	30%
Buses	4.96	1%	18.00	0%	13.03	4%	1.07	5%
TOTAL	926.4		4190.46		325.08		20.25	

Source: National Park Service, February 2000. Air Quality Concerns Related to Snowmobile Usage in National Parks. Air Docket A-2000-01, Document No. II-A-44.

The information presented in this discussion indicates that snowmobiles are significant emitters of pollutants that are known to contribute to visibility impairment in some Class I areas. Annual and particularly wintertime hydrocarbon emissions from snowmobiles are high in the five parks considered in Table 1.6-4, with two parks having HC emissions nearly as high as Yellowstone (Rocky Mountain and Voyageurs). The proportion of snowmobile emissions to emissions from other sources affecting air quality in these parks is likely to be similar to that in Yellowstone.

1.6.1.3 Individual Air Toxics and CO Exposure

In addition to their contribution to ozone formation and CO concentrations generally, snowmobile emissions are of concern because of their potential impacts on riders and on park attendants, as well as other groups of people who are in contact with these vehicles for extended periods of time.

Snowmobile users can be exposed to high air toxic and CO emissions, both because they sit very close to the vehicle's exhaust port and because it is common for them to ride their vehicles on groomed trails where they travel fairly close behind other snowmobiles. Because of these riding patterns, snowmobilers breathe exhaust emissions from their own vehicle, the vehicle directly in front as well as those farther up the trail. This can lead to relatively high personal exposure levels of harmful pollutants. A study of snowmobile rider CO exposure conducted at Grand Teton National Park showed that a snowmobiler riding at distances of 25 to 125 feet behind another snowmobiler and traveling at speeds from 10 to 40 mph can be exposed to average CO levels ranging from 0.5 to 23 ppm, depending on speed and distance. The highest CO level measured in this study was 45 ppm, as compared to the current 1-hour NAAQS for CO of 35 ppm.[84] While exposure levels can be less if a snowmobile drives 15 feet off the centerline of the lead snowmobile, the exposure levels are still of concern. This study led to the development of an empirical model for predicting CO exposures from riding behind snowmobiles.

Hydrocarbon speciation for snowmobile emissions was performed for the State of Montana in a 1997 report.[85] Using the empirical model for CO from the Grand Teton exposure study with benzene emission rates from the State of Montana's emission study, benzene exposures for riders driving behind a single snowmobile were predicted to range from 1.2E+02 to 1.4E+03 μg/m3. Using the same model to predict exposures when riding at the end of a line of six snowmobiles spaced 25 feet apart yielded exposure predictions of 3.5E+03, 1.9E+03, 1.3E+03, and 1.2E+03 μg/m3 benzene. at 10, 20, 30, and 40 mph, respectively.

The cancer risk posed to those exposed to benzene emissions from snowmobiles must be viewed within the broader context of expected lifetime benzene exposure. Observed monitoring data and predicted modeled values demonstrate that a significant cancer risk already exists from ambient concentrations of benzene for a large portion of the US population. The Agency's 1996 National-Scale Air Toxics Assessment of personal exposure to ambient concentrations of air toxic compounds emitted by outside sources (e.g. cars and trucks, power plants) found that

benzene was among the five air toxics appear to pose the greatest risk to people nationwide. This national assessment found that for approximately 50% of the US population in 1996, the inhalation cancer risks associated with benzene exceeded 10 in one million. Modeled predictions for ambient benzene from this assessment correlated well with observed monitored concentrations of benzene ambient concentrations.

Specifically, the draft National-Scale Assessment predicted nationwide annual average benzene exposures from outdoor sources to be 1.4 μg/m3.[86] In comparison, snowmobile riders and those directly exposed to snowmobile exhaust emissions had predicted benzene levels two to three orders of magnitude greater than the 1996 national average benzene concentrations.[87] These elevated levels are also known as air toxic "hot spots," which are of particular concern to the Agency. Thus, total annual average exposures to typical ambient benzene concentrations combined with elevated short-term exposures to benzene from snowmobiles may pose a significant risk of adverse public health effects to snowmobile riders and those exposed to exhaust benzene emissions from snowmobiles.

Since snowmobile riders often travel in large groups, the riders towards the back of the group are exposed to the accumulated exhaust of those riding ahead. These exposure levels can continue for hours at a time. An additional consideration is that the risk to health from CO exposure increases with altitude, especially for unacclimated individuals. Therefore, a park visitor who lives at sea level and then rides his or her snowmobile on trails at high-altitude is more susceptible to the effects of CO than local residents.

In addition to snowmobilers themselves, people who are active in proximity to the areas where snowmobilers congregate may also be exposed to high CO levels. An OSHA industrial hygiene survey reported a peak CO exposure of 268 ppm for a Yellowstone employee working at an entrance kiosk where snowmobiles enter the park. This level is greater than the NIOSH peak recommended exposure limit of 200 ppm. OSHA's survey also measured employees' exposures to several air toxics. Benzene exposures in Yellowstone employees ranged from 67-600 μg/m3, with the same individual experiencing highest CO and benzene exposures. The highest benzene exposure concentrations exceeded the NIOSH Recommended Exposure Limit of 0.1 ppm for 8-hour exposures.[88]

1.6.2 Large SI Engines

Exhaust emissions from applications with significant indoor use can expose individual operators or bystanders to dangerous levels of pollution. Forklifts, ice-surfacing machines, sweepers, and carpet cleaning equipment are examples of large industrial spark-ignition engines that often operate indoors or in other confined spaces. Forklifts alone account for over half of the engines in this category. Indoor use may include extensive operation in a temperature-controlled environment where ventilation is kept to a minimum (for example, for storing, processing, and shipping produce).

The principal concern for human exposure relates to CO emissions. One study showed

several forklifts with measured CO emissions ranging from 10,000 to 90,000 ppm (1 to 9 percent).[89] The threshold limit value for a time-weighted average 8-hour workplace exposure set by the American Conference of Governmental Industrial Hygienists is 25 ppm.

One example of a facility that addressed exposure problems with new technology is in the apple-processing field.[90] Trout Apples in Washington added three-way catalysts to about 60 LPG-fueled forklifts to address multiple reports of employee health complaints related to CO exposure. The emission standards proposed in this document are based on the same technologies installed on these in-use engines.

Additional exposure concerns occur at ice rinks. Numerous papers have identified ice-surfacing machines with spark-ignition engines as the source of dangerous levels of CO and NO_2, both for skaters and for spectators.[91] This is especially problematic for skaters, who breathe air in the area where pollutant concentration is highest, with higher respiration rates resulting from their high level of physical activity. This problem has received significant attention from the medical community.

In addition to CO emissions, HC emissions from these engines can also lead to increased exposure to harmful pollutants, particularly air toxics. Since many gasoline or dual-fuel engines are in forklifts that operate indoors, reducing evaporative emissions could have direct health benefits to operators and other personnel. Fuel vapors can also cause odor problems.

1.6.3 Acid Deposition

Acid deposition, or acid rain as it is commonly known, occurs when SO_2 and NOx react in the atmosphere with water, oxygen, and oxidants to form various acidic compounds that later fall to earth in the form of precipitation or dry deposition of acidic particles.[92] It contributes to damage of trees at high elevations and in extreme cases may cause lakes and streams to become so acidic that they cannot support aquatic life. In addition, acid deposition accelerates the decay of building materials and paints, including irreplaceable buildings, statues, and sculptures that are part of our nation's cultural heritage. To reduce damage to automotive paint caused by acid rain and acidic dry deposition, some manufacturers use acid-resistant paints, at an average cost of $5 per vehicle--a total of $61 million per year if applied to all new cars and trucks sold in the U.S.

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

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

1.6.4 Eutrophication and Nitrification

Nitrogen deposition into bodies of water can cause problems beyond those associated with acid rain. The Ecological Society of America has included discussion of the contribution of air emissions to increasing nitrogen levels in surface waters in a recent major review of causes and consequences of human alteration of the global nitrogen cycle in its Issues in Ecology series.[93] Long-term monitoring in the United States, Europe, and other developed regions of the world shows a substantial rise of nitrogen levels in surface waters, which are highly correlated with human-generated inputs of nitrogen to their watersheds. These nitrogen inputs are dominated by fertilizers and atmospheric deposition.

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

Collectively, these effects are referred to as eutrophication, which the National Research Council recently identified as the most serious pollution problem facing the estuarine waters of the United States (NRC, 1993). Nitrogen is the primary cause of eutrophication in most coastal waters and estuaries.[94] On the New England coast, for example, the number of red and browntides and shellfish problems from nuisance and toxic plankton blooms have increased over the past two decades, a development thought to be linked to increased nitrogen loadings in coastal waters. We believe that airborne NOx contributes from 12 to 44 percent of the total nitrogen loadings to United States coastal water bodies. For example, some estimates assert that approximately one-quarter of the nitrogen in the Chesapeake Bay comes from atmospheric deposition.

Excessive fertilization with nitrogen-containing compounds can also affect terrestrial ecosystems.[95] Research suggests that nitrogen fertilization can alter growth patterns and change the balance of species in an ecosystem, providing beneficial nutrients to plant growth in areas

that do not suffer from nitrogen over-saturation. In extreme cases, this process can result in nitrogen saturation when additions of nitrogen to soil over time exceed the capacity of the plants and microorganisms to utilize and retain the nitrogen. This phenomenon has already occurred in some areas of the U.S.

Notes to Chapter 1

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

2.U.S. EPA, 1996, Review of National Ambient Air Quality Standards for Ozone, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-96-007. A copy of this document can be obtained from Air Docket A-99-06, Document No. II-A-22.

3.U.S. EPA, 1996, Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA/600/P-93/004aF. The document is available on the internet at http://www.epa.gov/ncea/ozone.htm. A copy can also be obtained from Air Docket No. A-99-06, Documents Nos. II-A-15, II-A-16, II-A-17.

4.U.S. EPA, 1995, Review of National Ambient Air Quality Standards for Nitrogen Dioxide, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA-452/R-95-005.

5.U.S.EPA, 1993, Air Quality Criteria for Oxides of Nitrogen, EPA/600/8-91/049aF.

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

7.National Air Quality and Emissions Trends Report, 1999, EPA, 2001, at Table A-19. This document is available at http://www.epa.gov/oar/aqtrnd99/. The data from the Trends report are the most recent EPA air quality data that has been quality assured. A copy of this table can also be found in Docket No. A-2000-01, Document No. II-A-64.

8.National Air Quality and Emissions Trends Report, 1998, March, 2000, at 28. This document is available at http://www.epa.gov/oar/aqtrnd98/. Relevant pages of this report can be found in Memorandum to Air Docket A-2000-01 from Jean Marie Revelt, September 5, 2001, Document No. II-A-63.

9.National Air Quality and Emissions Trends Report, 1998, March, 2000, at 32. This document is available at http://www.epa.gov/oar/aqtrnd98/. Relevant pages of this report can be found in Memorandum to Air Docket A-2000-01 from Jean Marie Revelt, September 5, 2001, Document No. II-A-63.

10.Additional information about this modeling can be found in our Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements, document EPA420-R-00-026, December 2000. Docket No. 1-2000-01, Document No. II-A-13. This document is also available at http://www.epa.gov/otaq/diesel.htm#documents.

11.We also performed ozone air quality modeling for the western United States but, as described further in the air quality technical support document, model predictions were well below corresponding ambient concentrations for out heavy-duty engine standards and fuel sulfur control rulemaking. Because of poor model performance for this region of the country, the results of the Western ozone modeling were not relied on for that rule.

12.Additional information about these studies can be found in Chapter 2 of "Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements," December 2000, EPA420-R-00-026. Docket No. A-2000-01, Document Number II-A-13. This document is also available at http://www.epa.gov/otaq/diesel.htm#documents.

13.Air Quality Criteria Document for Ozone and Related Photochemical Oxidants, EPA National Center for Environmental Assessment, July 1996, Report No. EPA/600/P-93/004cF. The document is available on the internet at http://www.epa.gov/ncea/ozone.htm. A copy can also be obtained from Air Docket No. A-99-06, Documents Nos. II-A-15, II-A-16, II-A-17.

14.A copy of this data can be found in Air Docket A-2000-01, Document No.II-A-80.

15.Memorandum to Docket A-99-06 from Eric Ginsburg, EPA, "Summary of Model-Adjusted Ambient Concentrations for Certain Levels of Ground-Level Ozone over Prolonged Periods," November 22, 2000. Docket A-2000-01, Document Number II-B-13.

16.Memorandum to Docket A-99-06 from Eric Ginsburg, EPA, "Summary of Model-Adjusted Ambient Concentrations for Certain Levels of Ground-Level Ozone over Prolonged Periods," November 22, 2000, at Table C, Control Scenario – 2020 Populations in Eastern Metropolitan Counties with Predicted Daily 8-Hour Ozone greater than or equal to 0.080 ppm. Docket A-2000-01, Document Number II-B-13.

17.National Air Quality and Emissions Trends Report, 1999, EPA, 2001, at Table A-19. This document is available at http://www.epa.gov/oar/aqtrnd99/. The data from the Trends report are the most recent EPA air quality data that has been quality assured. A copy of this table can also be found in Docket No. A-2000-01, Document No. II-A-64.

18.St. Paul, Minnesota was recently reclassified as being in attainment but is still considered a maintenance area. There is also a significant population of snowmobiles in Minnesota, with snowmobile trails in Washington County.

19.The trail maps consulted for this proposal can be found in Docket No. A-2000-01, Document No. II-A-65.

20. Technical Memorandum to Docket A-2000-01 from Drew Kodjak, Attorney-Advisor, Office of Transportation and Air Quality, "Air Quality Information for Selected CO Nonattainment Areas," July 27, 2001, Docket Number A-2000-01, Document Number II-B-18.

21. Air Quality Criteria for Carbon Monoxide, US EPA, EPA 600/P-99/001F, June 2000, at 3-38, Figure 3-32 (Federal Bldg, AIRS Site 020900002). Air Docket A-2000-01, Document Number II-A-29. This document is also available at http://www.epa.gov/ncea/coabstract.htm.

22. National Air Quality and Emissions Trends Report, 1998, March, 2000; this document is available at http://www.epa.gov/oar/aqtrnd98/. National Air Pollutant Emission Trends, 1900-1998 (EPA-454/R-00-002), March, 2000. These documents are available at Docket No. A-2000-01, Document No. II-A-72. See also Air Quality Criteria for Carbon Monoxide, US EPA, EPA 600/P-99/001F, June 2000, at 3-10. Air Docket A-2000-01, Document Number II-A-29. This document is also available at http://www.epa.gov/ncea/coabstract.htm.

23. LDTs are light-duty trucks greater than 3750 lbs. loaded vehicle weight, up through 6000 gross vehicle weight rating.

24. Draft Anchorage Carbon Monoxide Emission Inventory and Year 2000 Attainment Projections, Air Quality Program, May 2001, Docket Number A-2000-01, Document II-A-40; Draft Fairbanks 1995-2001 Carbon Monoxide Emissions Inventory, June 1, 2001, Docket Number A-2000-01, Document II-A-39.

25.66 FR 28836, May 25, 2001. Clean Air Act Promulgation of Attainment Date Extension for the Fairbanks North Star Borough Carbon Monoxide Nonattainment Area, AK, Direct Final Rule.

26.U.S. EPA, Air Quality Criteria for Carbon Monoxide, EPA 600/P-99.001F, June 2000, Section 3.2.3. Air Docket A-2000-01, Document Number II-A-29. This document is also available at http://www.epa.gov/ncea/coabstract.htm.

27. Air Quality and Emissions Trends Report, 1998, March, 2000. This document is available at http://www.epa.gov/oar/aqtrnd98/. Relevant pages of this report can be found in Memorandum to Air Docket A-2000-01 from Jean Marie Revelt, September 5, 2001, Document No. II-A-63.

28.EPA (1996) Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information OAQPS Staff Paper. EPA-452/R-96-013. Docket Number A-99-06, Documents Nos. II-A-18, 19, 20, and 23. The particulate matter air quality criteria documents are also available at http://www.epa.gov/ncea/partmatt.htm.

29.Memorandum to Docket A-99-06 from Eric O. Ginsburg, Senior Program Advisor, "Summary of 1999 Ambient Concentrations of Fine Particulate Matter," November 15, 2000. This memo is also available in the docket for this rule. Docket A-2000-01, Document Number II-B-12.

30.EPA (1996) Review of the National Ambient Air Quality Standards for Particulate Matter: Policy Assessment of Scientific and Technical Information OAQPS Staff Paper. EPA-452/R-96-013. Docket Number A-99-06, Documents Nos. II-A-18, 19, 20, and 23. The particulate matter air quality criteria documents are also available at http://www.epa.gov/ncea/partmatt.htm. .

31.Memorandum to Docket A-99-06 from Eric O. Ginsburg, Senior Program Advisor, "Summary of Absolute Modeled and Model-Adjusted Estimates of Fine Particulate Matter for Selected Years," December 6, 2000. This memo is also available in the docket for this rule. Docket A-2000-01, Document Number II-B-14.

32.Additional information about the Regulatory Model System for Aerosols and Deposition (REMSAD) and our modeling protocols can be found in our Regulatory Impact Analysis: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements, document EPA420-R-00-026, December 2000. Docket No. A-2000-01, Document No. A-II-13. This document is also available at http://www.epa.gov/otaq/disel.htm#documents.

33. Technical Memorandum, EPA Air Docket A-99-06, Eric O. Ginsburg, Senior Program Advisor, Emissions Monitoring and Analysis Division, OAQPS, Summary of Absolute Modeled and Model-Adjusted Estimates of Fine Particulate Matter for Selected Years, December 6, 2000, Table P-2. Docket Number 2000-01, Document Number II-B-14.

34. Memo to file from Terence Fitz-Simons, OAQPS, Scott Mathias, OAQPS, Mike Rizzo, Region 5, "Analyses of 1999 PM Data for the PM NAAQS Review," November 17, 2000, with attachment B, 1999 PM2.5 Annual Mean and 98th Percentile 24-Hour Average Concentrations. Docket No. A-2000-01, Document No. II-B-17.

35. The trail maps consulted for this proposal can be found in Docket No. A-2000-01, Document No. II-A-65.

36.See our Mobile Source Air Toxics final rulemaking, 66 FR 17230, March 29, 2001, and the Technical Support Document for that rulemaking. Docket No. A-2000-01, Documents Nos. II-A-42 and II-A-30.

37.U.S. EPA. (1999) Analysis of the Impacts of Control Programs on Motor Vehicle Toxic Emissions and Exposure in Urban Areas and Nationwide: Volume I. Prepared for EPA by Sierra Research, Inc. and Radian International Corporation/Eastern Research Group, November 30, 1999. Report No. EPA420-R-99-029. http://www.epa.gov/otaq/toxics.htm.

38.U.S. EPA (1998) Environmental Protection Agency, Carcinogenic Effects of Benzene: An Update, National Center for Environmental Assessment, Washington, DC. 1998. EPA/600/P-97/001F. http://www.epa.gov/ncepihom/Catalog/EPA600P97001F.html.

39.Leukemia is a blood disease in which the white blood cells are abnormal in type or number. Leukemia may be divided into nonlymphocytic (granulocytic) leukemias and lymphocytic leukemias. Nonlymphocytic leukemia generally involves the types of white blood cells (leukocytes) that are involved in engulfing, killing, and digesting bacteria and other parasites (phagocytosis) as well as releasing chemicals involved in allergic and immune responses. This type of leukemia may also involve erythroblastic cell types (immature red blood cells). Lymphocytic leukemia involves the lymphocyte type of white bloods cell that are responsible for the immune responses. Both nonlymphocytic and lymphocytic leukemia may, in turn, be

separated into acute (rapid and fatal) and chronic (lingering, lasting) forms. For example; in acute myeloid leukemia (AML) there is diminished production of normal red blood cells (erythrocytes), granulocytes, and platelets (control clotting) which leads to death by anemia, infection, or hemorrhage. These events can be rapid. In chronic myeloid leukemia (CML) the leukemic cells retain the ability to differentiate (i.e., be responsive to stimulatory factors) and perform function; later there is a loss of the ability to respond.

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