

Chapter 3: Air Quality and Resulting Health and Welfare Effects of Air Pollution from Mobile Sources

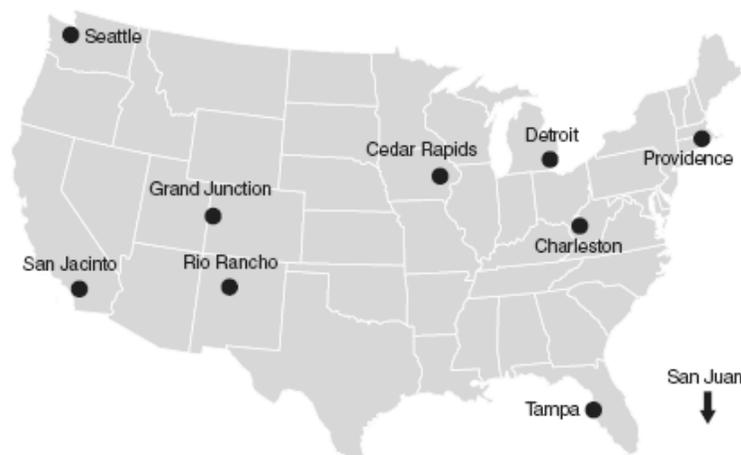
3.1 Air Quality and Exposure Measurements

3.1.1 Ambient Monitoring

Ambient air toxics data is useful for identifying pollutants of greatest concern, areas of unhealthy ambient air toxics concentrations, and air toxics trends; evaluating and improving models; and assessing the effectiveness of air toxics reduction strategies. Ambient air toxics data though has limitations for use in risk assessments. While EPA, states, tribes, and local air regulatory agencies collect monitoring data for a number of toxic air pollutants, both the chemicals monitored and the geographic coverage of the monitors vary from state to state.¹ In recent years, the US EPA and states have initiated more extensive monitoring of air toxics to assist in air pollution management through measurement and mitigation.² EPA is working with its regulatory partners to build upon the existing monitoring sites to create a national monitoring network for a number of toxic air pollutants. The goal is to ensure that those compounds that pose the greatest risk are measured. EPA also recently published a draft National Air Toxics Monitoring Strategy to advance this goal.³

The available monitoring data help air pollution control agencies track trends in toxic air pollutants in various locations around the country. EPA conducted a pilot city monitoring project in 2001 that included sampling in four urban areas and six small city/rural areas (see Figure 3.1-1). This program helped answer several important national network design questions (e.g., sampling and analysis precision, sources of variability, and minimal detection levels).

Figure 3.1-1. Map of Ten Cities in Monitoring Pilot Project

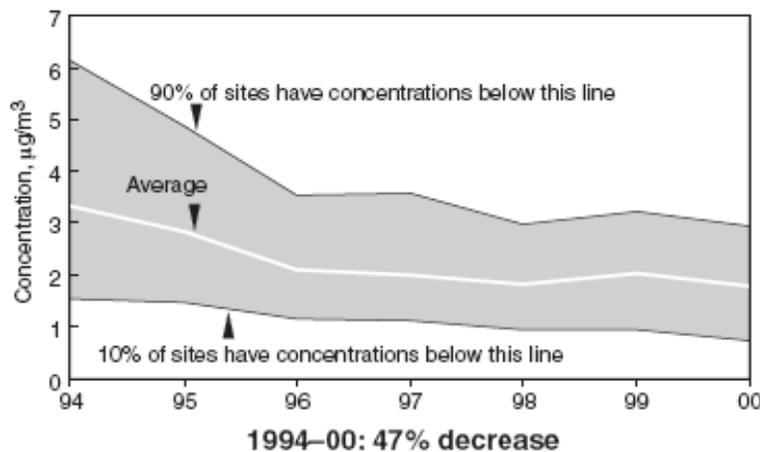


Building on the pilot program, the US EPA and states established a national air toxics

monitoring program beginning with a 10-city pilot program, which now consists of 22 national air toxics trends sites (NATTS), and numerous community-scale monitoring studies.⁴ To guide development of the monitoring program, a qualitative data analysis project was begun in 2001 and the first phase was completed in 2004. The analysis showed that typical urban concentration ranges for most VOCs are approximately an order of magnitude (or more) higher than the background concentrations. Because air toxics concentrations vary spatially, other monitoring networks are needed to provide additional, especially rural, concentrations. Extrapolation for most air toxics beyond the urban scale is not recommended without a network of rural measurements capable of capturing gradients between urban and rural areas. For the latest information on national air toxics monitoring, see www.epa.gov/ttn/amtic/airtxfil.html.

Figure 3.1-2 shows measurements of benzene taken from 95 urban monitoring sites around the country. These urban areas generally have higher levels of benzene than other areas of the country. Measurements taken at these sites show, on average, a 47% drop in benzene levels from 1994 to 2000. During this period, EPA phased in new (so-called “tier 1”) car emission standards; required many cities to begin using cleaner-burning gasoline; and set standards that required significant reductions in benzene and other pollutants emitted from oil refineries and chemical processes.

Figure 3.1-2. Ambient Benzene, Annual Average Urban Concentrations, Nationwide, 1994-2000



Following is a summary of analyses recently performed on ambient measurements of air toxics to identify pollutants and geographic areas of concern and to evaluate trends. Use of monitoring data to evaluate and improve models is discussed in Section 3.2.

New York State has a systematic program in place that has been measuring air toxics since the 1990s.⁵ The network of monitors is located throughout urban, industrial, residential and rural locations. The New York State Department of Environmental Conservation recently examined the spatial and temporal characteristics of benzene by analyzing five of the 32 total network sites across the state (see Table 3.1-1). Spatial trends show a wide range of annual

average benzene concentrations, with the lowest value at a rural site and the highest at an industrial site. The recent 3-year period of 2001-2003 was also compared with the longer 1990-2003 period. The 3-year period exhibits a decrease in mean concentration compared to the entire period, indicating that benzene concentrations are decreasing over New York State throughout this period. The mean annual rate of change in the period 1990 to 2003 was determined using linear regression of the concentration data. The analysis indicated that ambient concentration levels of benzene decreased by as much as 60% during 1990 to 2003. These decreases occurred in ozone nonattainment areas that had reformulated gasoline (RFG) requirements as well as in the rest of the state. The downward trend can be attributed to regulatory measures aimed at reducing toxic emissions from industrial sources, replacement of older higher emitting vehicles with vehicles meeting more stringent EPA standards for hydrocarbon emissions, as well as the adoption of RFG in 1995 and 1999 for the 1-hour ozone nonattainment areas in New York State. Since trends were observed for sites that were not part of the RFG program, decreases may also be attributed to the improvement in vehicle emissions technology and the state-wide adoption of the California Low Emission Vehicle program.

The downward trend in benzene concentrations reported for New York is consistent with other reported changes in ambient levels of benzene. In California, the Air Resources Board (ARB) maintains an Almanac of Emissions and Air Quality.⁶ The Almanac summarizes statewide emissions, statewide annual average concentrations (calculated as a mean of monthly means), and statewide average health risks for selected air toxics. Currently there are data available for ten air toxics in California, including benzene. The ARB network consists of 18 air quality monitoring stations. The data collected, analyzed, and reported reflect a spatial average; therefore, ambient concentrations for individual locations may be higher or lower. Estimates show that approximately 84% of the benzene emitted in California comes from motor vehicles, including evaporative leakage and unburned fuel exhaust. The predominant sources of total benzene emissions in the atmosphere are gasoline fugitive emissions and gasoline motor vehicle exhaust. Approximately 49% of the statewide benzene emissions can be attributed to on-road motor vehicles, with an additional 35% attributed to other mobile sources such as recreational boats, off-road recreational vehicles, and lawn and garden equipment. Currently, the benzene content of gasoline is less than 1%. Some of the benzene in the fuel is emitted from vehicles as unburned fuel. Benzene is also formed as a partial combustion product of larger aromatic fuel components. Industry-related stationary sources contribute 15% and area-wide sources contribute 1% of the statewide benzene emissions. The primary stationary sources of reported benzene emissions are crude petroleum and natural gas mining, petroleum refining, and electric generation. The primary area-wide sources include residential combustion of various types such as cooking and water heating. The primary natural sources are petroleum seeps that form where oil or natural gas emerge from subsurface sources to the ground or water surface. The statewide benzene levels have shown generally steady improvement since 1990. To examine the trend in benzene while minimizing the influences of weather on the trend, the statewide average benzene concentration for 1990-1992 was compared to that for 2001-2003. The result was a 72% decrease in benzene concentration. These downward trends for benzene and other air toxics are a result of many control measures implemented to reduce emissions.

Table 3.1-1. Site Descriptions of the Monitoring Stations Along with Mean Benzene Concentration from 1990-2003 and 2001-2003

Site Character	Lackawanna Industrial	Eastern District High School Urban	Troy Small Urban	Niagara Falls Urban Industrial	Whiteface Mountain Base Lodge Rural
Location Area	Buffalo	Brooklyn	Hudson Valley	Niagara	Essex
2000 Population (thousands)	950	2465	153	220	39
Annual Vehicle Miles Traveled (million miles)	8250	4246	1413	1546	577
Mean Concentration ($\mu\text{g}/\text{m}^3$) Period 1990-2003	5.09	2.85	2.31	1.80	0.86
Mean Concentration ($\mu\text{g}/\text{m}^3$) Period 2001-2003	2.26	2.05	1.68	1.08	0.54

Another recent evaluation of hazardous air pollutant (HAP) trends was conducted for selected metropolitan areas.⁷ Researchers retrieved historical concentration and emissions data from the US EPA for Boston, New York City, Philadelphia, Tampa Bay, Detroit, Dallas, St. Louis, Denver, Los Angeles, and Seattle, chosen for each of EPA's ten regions. Annual and seasonal trends were generated to evaluate reductions in HAP emissions and ambient concentrations during the time period 1990-2003. Several air toxics were targeted, including benzene. To evaluate the trends, average concentrations from 1990-1994 were compared to 2002-2003 (these time periods were chosen due to availability of data). The results showed that over 85% of the metropolitan area-HAP combinations decreased in their HAP concentrations, while less than 15% realized an increase. For example, Table 3.1-2 shows that benzene concentrations decreased in seven of the ten metropolitan areas (range 19 to 79%).

Each of these analyses consistently illustrates the significant reductions in national annual average concentrations of benzene and other air toxics. The air pollution management efforts of the US EPA and states have been effective. Additional reductions are expected with the implementation of additional regulatory measures such as this one.

Table 3.1-2. Benzene Emission (Tons Per Year) and Concentration ($\mu\text{g}/\text{m}^3$) Comparison

Metropolitan Area	1990 Emissions	2002 Emissions	% Change in Emissions	1990-1994 Average Concentration	2002-2003 Average Concentration	% Change in Concentration
Boston	6262	2229	-64.4	3.93	0.81	-79.5
New York City	16653	7512	-54.9	3.24	1.35	-58.5
Philadelphia	5961	2577	-56.8	3.60	1.26	-64.9
Tampa Bay	3103	2408	-22.4	NA	NA	NA
Detroit	6480	4388	-32.3	4.19	3.40	-18.7
Dallas	7933	2832	-64.3	1.21	0.78	-35.8
St. Louis	4358	2304	-47.1	5.16	1.43	-72.3
Denver	2800	1913	-31.7	NA	2.75	NA
Los Angeles	19762	4168	-78.9	8.97	2.34	-73.9
Seattle	5844	4315	-26.2	NA	1.39	NA

3.1.2 Population-Based (Representative) Exposure Measurements

In addition to measurements of outdoor and microenvironmental concentrations, an important component of understanding human exposure to air toxics is the body of studies that employ survey techniques to assess representative populations' exposures. Typically, these studies are designed to represent a discrete geographic area. The personal exposure concentration summaries from these studies are shown in Table 3.1-3.

The National Human EXposure Assessment Survey (NHEXAS) was a series of population-based exposure studies. The states in EPA Region 5 were the focus of one NHEXAS study, which was conducted in mid-1990.⁸ Nearly 400 personal and indoor air samples were obtained from both smokers and non-smokers, along with a smaller number of outdoor air samples in residential areas. Measurements took place over 6 days per subject. Overall, average personal exposure to benzene was $7.52 \mu\text{g}/\text{m}^3$, with indoor air concentrations averaging $7.21 \mu\text{g}/\text{m}^3$. Outdoor air concentrations averaged $3.61 \mu\text{g}/\text{m}^3$. Personal air concentrations were significantly associated with indoor air concentrations, as well as blood concentrations.

The results of the NHEXAS study in Arizona, another study area, indicate that median indoor concentrations were $1.3 \mu\text{g}/\text{m}^3$ during the mid-1990's, while outdoor concentrations were $1.0 \mu\text{g}/\text{m}^3$.⁹ Furthermore, reported results from the Arizona study indicate that fuel-related VOCs are elevated in homes with attached garages.

In another study based on a random population-based sample of an urban population, 37 non-smoking residents of South Baltimore, MD were equipped with passive monitors to assess 3-day average personal exposure to VOCs, in addition to indoor and outdoor air.¹⁰ Monitoring took place in 2000 and 2001. Modeled air quality data from the ASPEN dispersion model, employed in EPA's National Air Toxics Assessment for 1996, was also obtained for the study area. Overall, outdoor modeled concentrations of benzene and other fuel-related VOCs corresponded well with measured data in the area. Average personal exposure to benzene was $4.06 \mu\text{g}/\text{m}^3$, while 95th percentile values were $7.30 \mu\text{g}/\text{m}^3$. For indoors, the respective values were 3.70 and $8.34 \mu\text{g}/\text{m}^3$, while for outdoors the values were 1.84 and $3.14 \mu\text{g}/\text{m}^3$. Overall, the study provides evidence that modeling outdoor benzene concentrations using ASPEN, as is done in this rule, provides adequate representation of outdoor values. However, indoor and personal exposures are also influenced by other sources, as is described in the section on attached garages.

While not a population-based study, the recently-completed Relationship Between Indoor, Outdoor and Personal Air (RIOPA) study provides a depiction of indoor, outdoor, and personal concentrations of benzene and other toxics in three regions with differing source mixtures.¹¹ 100 non-smoking homes in each of Los Angeles, CA, Houston, TX, and Elizabeth, NJ were selected for sampling in areas representing locations dominated by emissions from mobile sources, stationary sources, and a mixture of sources, respectively. In the adult sample, average personal exposures to benzene were $3.64 \mu\text{g}/\text{m}^3$, with a 95th percentile of $10.7 \mu\text{g}/\text{m}^3$. Respective statistics for indoor air were $3.50 \mu\text{g}/\text{m}^3$ and $10.0 \mu\text{g}/\text{m}^3$, while outdoor statistics were 2.15 and $5.16 \mu\text{g}/\text{m}^3$. In further EPA-funded analysis of the data from Elizabeth, NJ, concentrations of benzene, toluene, ethylbenzene, and xylene isomers were found to be associated with proximity to both major roadways and gas stations, as was $\text{PM}_{2.5}$, EC, and

several PAHs.^{12,13} Section 3.1.3 provides more detail on concentrations and exposures in these types of mobile-source impacted areas.

Few studies have systematically addressed exposures among representative samples of children. Several have been done in Minnesota, with others in New York, Los Angeles, and Baltimore areas.

For the Minnesota Children's Pesticide Exposure Study (MNCPEs), conducted in urban and rural areas in the vicinity of Minneapolis-St. Paul, MN,¹⁴ all monitoring used the same 6-day monitoring duration as used in the Region 5 NHEXAS study. In the first phase of the study, a statistically representative sample of 284 homes with children underwent air monitoring for VOCs. Low-income and minority homes were over sampled to ensure representation. Indoor benzene concentrations averaged $4.6 \mu\text{g}/\text{m}^3$, with the data skewed right. The 95th percentile concentration was $12.7 \mu\text{g}/\text{m}^3$. Homes with attached garages had significantly higher concentrations of benzene indoors. In the second phase of the study, a subset of 100 children underwent intensive monitoring of personal, indoor, and outdoor air as well as activity tracking via diary. Overall personal exposures were $4.8 \mu\text{g}/\text{m}^3$, with a 95th percentile of $9.1 \mu\text{g}/\text{m}^3$. Indoor concentrations in the intensive period averaged $3.9 \mu\text{g}/\text{m}^3$ and outdoor averaged $3.3 \mu\text{g}/\text{m}^3$. Regression analysis indicated that personal exposures generally were higher than the time-weighted average of indoor and outdoor air. Furthermore, living in a home with an attached garage was associated with elevated personal exposures to both benzene and toluene.

In another study, students recruited from an inner-city school in Minneapolis, MN participated in an exposure study called SHIELD.¹⁵ Students were recruited using stratified random sampling, with a total of 153 children participating between two seasons. Home and personal samples were collected and averaged over two continuous days of sampling using passive methods. School measurements took place during school hours only, over the course of 5 days, and outdoor measurements were set up to run continuously outside the school through each week sampled (Monday through Friday). The study reported median, 10th, and 90th percentile concentrations. In personal samples, median benzene concentrations were $1.5 \mu\text{g}/\text{m}^3$ in spring and $2.1 \mu\text{g}/\text{m}^3$ in winter.¹⁶

The TEACH exposure study tracked inner-city high school students' exposures in New York, NY and Los Angeles, CA. In the New York City study, 42 students underwent personal, indoor home, and outdoor home air quality monitoring during two seasons.¹⁷ Average winter benzene personal concentrations were $4.70 \mu\text{g}/\text{m}^3$, while indoor and outdoor concentrations averaged 5.97 and $2.55 \mu\text{g}/\text{m}^3$. Summer values were 3.09 , 1.75 , and $1.31 \mu\text{g}/\text{m}^3$, respectively. The authors noted that VOC concentrations within the city tracked traffic patterns. Generally, indoor concentrations in Los Angeles were of similar magnitude, while personal exposures were not reported as of the time of this proposal. There was no substantial evidence for indoor sources of benzene.¹⁸

Overall, these studies show that personal and indoor concentrations of benzene and other VOCs are significantly higher than found outdoors. Some of the factors leading to these elevated concentrations are a result of motor vehicle impacts such as exhaust and evaporative emissions in attached garages, exposures during on-road commutes and exposures during vehicle re-fueling.

These and other factors are discussed in more detail in Section 3.1.3. This suggests that risk reductions from the controls in this proposal will be greater than can currently be estimated using national-scale modeling tools.

Table 3.1-3. Personal Exposure to Benzene from Population-Based Studies^a

Location	Year(s)	Includes Smokers	Average ($\mu\text{g}/\text{m}^3$)	“Upper Bound” ($\mu\text{g}/\text{m}^3$)	Reference
EPA Region 5	1995-1996	Yes	7.21	13.71 ^b	Clayton et al. (1999)
Baltimore, MD	2000-2001	No	4.06	7.30 ^c	Payne-Sturges et al. (2004)
NJ, TX, CA	1999-2001	No	3.64	10.7 ^c	Weisel et al. (2005)
<i>Minneapolis - St. Paul, MN</i>	<i>1997</i>	<i>Yes^e</i>	<i>4.8</i>	<i>9.1</i>	<i>Adgate et al. (2004a)</i>
<i>Minneapolis, MN</i>	<i>2000</i>	<i>Yes^e</i>	<i>2.1 Winter 1.5 Spring</i>	<i>6.5 Winter^b 4.2 Spring^b</i>	<i>Adgate et al. (2004b)</i>
<i>New York, NY</i>	<i>1999</i>	<i>No</i>	<i>4.7 Winter 3.1 Summer</i>	<i>11.4 Winter^d 7.0 Summer^d</i>	<i>Kinney et al. (2002)</i>

^a Children’s studies in italics

^b 90th percentile

^c 95th percentile

^d Mean +2 standard deviations

^e Smoking in homes

3.1.3 Elevated Concentrations and Exposures in Mobile Source-Impacted Areas

Air quality measurements near roads often identify elevated concentrations of air toxic pollutants at these locations. The concentrations of air toxic pollutants near heavily trafficked roads, as well as the pollutant composition and characteristics, differ from those measured distant from heavily trafficked roads. Thus, exposures for populations residing, working, or going to school near major roads are likely different than for other populations. Following is an overview of concentrations of air toxics and exposure to air toxics in areas experiencing elevated pollutant concentrations due to the impacts of mobile source emissions.

3.1.3.1 Concentrations Near Major Roadways

3.1.3.1.1 Particulate Matter

Mobile sources influence temporal and spatial patterns of criteria pollutants, air toxics, and PM concentrations within urban areas. Motor vehicle emissions may lead to elevated concentrations of pollutants near major roads. Since motor vehicle emissions generally occur within the breathing zone, near-road populations may be exposed to “fresh” primary emissions as well as combustion pollutants “aged” in the atmosphere. For particulate matter, these fresh

versus aged emissions can result in the presence of varying particle sizes near roadways, including ultrafine, fine, and coarse particle modes.

The range of particle sizes of concern is quite broad and is divided into smaller categories. Defining different size categories is useful since particles of different sizes behave differently in the atmosphere and in the human respiratory system. Table 3.1-4 lists the four terms for categorizing particles of different sizes as defined by the US EPA.¹⁹

Table 3.1-4. Descriptions and Particle Sizes of Each Category of Particles

Description	Particle Size, d_p (μm)
Supercoarse	$d_p > 10$
Coarse (or Thoracic Coarse Mode)	$2.5 < d_p \leq 10$
Fine (or Accumulation Mode)	$0.1 < d_p \leq 2.5$
Ultrafine (or Nuclei Mode) ^a	$d_p \leq 0.1$

^a Nuclei Mode has also been defined as $d_p \leq 0.05 \mu\text{m}$ elsewhere.

Other particle classifications of interest include total suspended particulate matter (TSP). TSP includes a broad range of particle sizes including fine, coarse, and supercoarse particles. PM_{10} is defined as particulate matter with an aerodynamic diameter of less than or equal to 10 μm . PM_{10} is regulated as a specific type of "pollutant" because this size range is considered respirable and can penetrate into the lower respiratory tract. $\text{PM}_{2.5}$ is particulate matter with an aerodynamic diameter less than or equal to 2.5 μm . $\text{PM}_{2.5}$ settles quite slowly in the atmosphere relative to coarse and supercoarse particles. Normal weather patterns can keep $\text{PM}_{2.5}$ airborne for several hours to several days and enable these particles to transport hundreds of miles. $\text{PM}_{2.5}$ can cause health problems due to widespread exposures and efficiency at reaching deep into the lungs.

The size distribution of particles can be defined as a function of number, surface area, volume, and mass.^{20,21} Typically, on a number basis, emissions from mobile sources are heavily dominated by ultrafine mode particles, which tend to be comprised of volatile carbon. On a surface area basis, the average diameter of particles emitted by mobile sources is 0.1 μm . On a volume and mass basis, the size distribution of particles emitted from mobile sources has an average particle diameter of approximately 0.2 μm .

Evidence of the large number of ultrafine mode particles emitted by motor vehicles can be found in the near-road environment. Roadside and ambient on-road measurements show that ultrafine mode particles dominate the number concentration in close proximity to the roadway, while fine mode dominates farther from the road. Particle size distributions, mass and elemental carbon concentrations have been examined near roads in Los Angeles.^{22,23} Researchers observed a four-fold increase in particle number concentrations, when comparing measurements 300 m and 20 m from LA highways. Other studies have similarly shown that ultrafine mode particles show a sharp decrease in particle number concentrations as the distance from major roadways increases.^{24,25} Evidence was recently found of increased exposures to ultrafine particles near roads when it was discovered that children living near major roads had elevated levels of particle-containing alveolar macrophages.²⁶ Additionally, roadside monitoring has shown that

particle number varies with vehicle type and vehicle operating conditions. For example, elevated ultrafine mode particle concentrations have been identified when operating speeds on the road increase as well as when the proportion of heavy-duty diesel vehicles increases.²⁷

An increase in fine particles near roads could originate from engine deterioration, brake and tire wear, and secondary aerosol formation.^{28,29,30,31} Engine deterioration is generally a function of vehicle age and maintenance condition. Brake wear emissions are highly dependent on brake pad materials.³² Secondary aerosol formation is dependent on fuel composition, emission rates, atmospheric chemistry, and meteorology. Re-entrained road dust, as well as brake and tire wear will also contribute to increased concentrations of coarse PM.

Meteorological factors can affect exposures to motor vehicle emissions near the road. Researchers have noted that particle number concentrations changed significantly with changing wind conditions, such as wind speed, near a road.³³ Wind direction also affects traffic-related air pollution mass concentrations inside and outside of schools near motorways.^{34,35} Diurnal variations in mixing layer height will also influence both near-road and regional air pollutant concentrations. Decreases in the height of the mixing layer (due to morning inversions, stable atmosphere, etc.) will lead to increased pollutant concentrations at both local and regional scales.

3.1.3.1.2 Gaseous Air Toxics

Concentrations of mobile source air toxics have been estimated by a number of different sources such as the NATA National-Scale Assessment, local-scale modeling assessments, and from air quality monitoring in locations in immediate proximity to busy roadways. Each approach offers a different level of representation of the concentrations of air toxics near roadways.

The NATA National-Scale Assessment for 1996 uses a model to predict ambient concentrations of gaseous air toxics such as benzene, as well as particle phase compounds such as metals and PAHs, and diesel particulate matter, at every census tract^A nationwide. The national average ambient benzene concentration was modeled at 1.39 $\mu\text{g}/\text{m}^3$, with considerable variation across the U.S.³⁶ This method estimates average concentrations within a census tract, but it does not differentiate between locations near roadways and those further away. Local-scale modeling can better characterize distributions of concentrations, as observed in assessments done in Houston, TX and Portland, OR. The Houston study calculated the average benzene concentration to be 2.10 $\mu\text{g}/\text{m}^3$,³⁷ using the same emissions inventory as used in the 1996 NATA National-Scale Assessment but with more refined allocation of highway vehicle emissions. In this study, spatially defined inventories placed vehicle emissions at the location of actual roadway links, thus characterizing with greater resolution the spatial distribution of ambient benzene concentrations. As a result, there was better agreement with monitor data (2.97 $\mu\text{g}/\text{m}^3$). The Portland study modeled concentrations of air toxics at the center of every census block group in the Portland, OR metropolitan area.³⁸ A subsequent analysis determined average 1,3-butadiene, benzene, and diesel PM concentrations at several distances from major roadways (0-50, 50-200, 200-400, and > 400 m). For benzene, the resulting average concentrations were

^A A census tract is a subdivision of a county that contains roughly 4000 people. In urban areas, these tracts can be very small, on the order of a city block, whereas in rural areas, they can be large.

1.29, 0.64, 0.40, and 0.12 $\mu\text{g}/\text{m}^3$, respectively, illustrating the steep concentration gradient around roadways. The overall mean benzene concentration modeled in Portland was 0.21 $\mu\text{g}/\text{m}^3$.

Air quality monitoring is another means of evaluating pollutant concentrations at locations near sources such as roadways. Several studies have found that concentrations of benzene and other mobile source air toxics are significantly elevated near busy roads compared to “urban background” concentrations measured at a fixed site.^{39,40,41,42,43,44} For example, measurements near a tollbooth in Baltimore observed mean benzene concentrations to vary by time of day from 3 to 22.3 $\mu\text{g}/\text{m}^3$ depending on traffic volume, vehicle type, and meteorology.⁴⁵ In comparison with ambient levels, Maryland’s Department of Environment reported the range of benzene annual averages measured at seven different monitoring sites in 2000 between 0.27-0.71 $\mu\text{g}/\text{m}^3$.⁴⁶ Another study measured the average benzene concentration in a relatively high traffic density (~ 16000 automobiles/day) sampling area at 9.6 $\mu\text{g}/\text{m}^3$ and in rural areas with hardly any traffic (< 50 automobiles/day) at 1.3 $\mu\text{g}/\text{m}^3$.⁴⁷ The concentration of benzene, along with several other air toxics (toluene and the isomeric xylenes), in the urban area far exceeded those in the rural area.

Ambient VOC concentrations were measured around residences in Elizabeth, NJ, as part of the Relationship among Indoor, Outdoor, and Personal Air (RIOPA) study. Data from that study was analyzed to assess the influence of proximity of known ambient emission sources on residences.⁴⁸ The ambient concentrations of benzene, toluene, ethylbenzene, and xylene isomers (BTEX) were found to be inversely associated with: distances from the sampler to interstate highways and major urban roads; distance from the sampler to gasoline stations; atmospheric stability; temperature; and wind speed. The data indicate that BTEX concentrations around homes within 200 m of roadways and gas stations are 1.5 to 4 times higher than urban background levels.

According to Gaussian dispersion theory, pollutants emitted along roadways will show highest concentrations nearest a road, and concentrations exponentially decrease with increasing distance downwind. These near-road pollutant gradients have been confirmed by measurements of both criteria pollutants and air toxics.^{49,50,51,52,53} Researchers have demonstrated exponential decreases in CO, as well as PM number, and black carbon (as measured by an aethalometer), concentration with increasing downwind distance from a freeway in Los Angeles.^{54,55} These pollutants reached background levels approximately 300 m downwind of the freeway.

3.1.3.2 Exposures Near Major Roadways

The modeling assessments and air quality monitoring studies discussed above have increased our understanding of ambient concentrations of mobile source air toxics and potential population exposures. Results from the following exposure studies reveal that populations spending time near major roadways likely experience elevated personal exposures to motor vehicle related pollutants. In addition, these populations may experience exposures to differing physical and chemical compositions of certain air toxic pollutants depending on the amount of time spent in close proximity to motor vehicle emissions. Following is a detailed discussion on exposed populations near major roadways.

3.1.3.2.1 In Vehicles

Several studies suggest that people may experience significant exposures while driving in vehicles. A recent in-vehicle monitoring study was conducted by EPA and consisted of in-vehicle air sampling throughout work shifts within ten police patrol cars used by the North Carolina State Highway Patrol (smoking not permitted inside the vehicles).⁵⁶ Troopers operated their vehicles in typical patterns, including highway and city driving and refueling. In-vehicle benzene concentrations averaged $12.8 \mu\text{g}/\text{m}^3$, while concentrations measured at an “ambient” site located outside a nearby state environmental office averaged $0.32 \mu\text{g}/\text{m}^3$. The study also found that the benzene concentrations were closely associated with other fuel-related VOCs measured.

The American Petroleum Institute funded a screening study of “high-end” exposure microenvironments as required by section 211(b) of the Clean Air Act.⁵⁷ The study included vehicle chase measurements and measurements in several vehicle-related microenvironments in several cities for benzene and other air toxics. In-vehicle microenvironments (average concentrations in parentheses) included the vehicle cabin tested on congested freeways ($17.5 \mu\text{g}/\text{m}^3$), in parking garages above-ground ($155 \mu\text{g}/\text{m}^3$) and below-ground ($61.7 \mu\text{g}/\text{m}^3$), in urban street canyons ($7.54 \mu\text{g}/\text{m}^3$), and during refueling ($46.0 \mu\text{g}/\text{m}^3$). It should be noted that sample sizes in this screening study were small, usually with only one to two samples per microenvironment.

In 1998, the California Air Resources Board published an extensive study of concentrations of in-vehicle air toxics in Los Angeles and Sacramento, CA.⁵⁸ The data set is large and included a variety of sampling conditions. On urban freeways, in-vehicle benzene concentrations ranged from 3 to $15 \mu\text{g}/\text{m}^3$ in Sacramento and 10 to $22 \mu\text{g}/\text{m}^3$ in Los Angeles. In comparison, ambient benzene concentrations ranged from 1 to $3 \mu\text{g}/\text{m}^3$ in Sacramento and 3 to $7 \mu\text{g}/\text{m}^3$ in Los Angeles.

Studies have also been conducted in diesel buses, such as the one recently conducted of LA school buses.^{59,60} In the study, five conventional diesel buses, one diesel bus equipped with a catalytic particle filter, and one natural gas bus were monitored for benzene, among other pollutants. These buses were driven on a series of real school bus routes in and around Los Angeles, CA. Average benzene concentrations in the buses were $9.5 \mu\text{g}/\text{m}^3$, compared with $1.6 \mu\text{g}/\text{m}^3$ at a background urban fixed site in west Los Angeles. Type of bus, traffic congestion levels, and encounters with other diesel vehicles contributed to high exposure variability between runs.

The same researchers additionally determined the relative importance of school bus-related microenvironments to children’s pollutant exposure.⁶¹ Real-time concentrations of black carbon (BC), particle-bound PAH, nitrogen dioxide (NO_2), particle counts (0.3-0.5 μm size range), and $\text{PM}_{2.5}$ mass were measured inside school buses during long commutes, at bus stops along the routes, at bus loading and unloading zones, and at nearby urban background sites. Across all the pollutants, mean concentrations during bus commutes were higher than in any other microenvironment. Mean exposures in bus commutes were 50 to 200 times more than for loading and unloading zones at the school, and 20 to 40 times more than for bus stops along the route, depending on the pollutant. The in-cabin exposures were dominated by the effect of

surrounding traffic when windows were open and by the bus' own exhaust when the windows were closed. The mean pollutant concentrations in the three school bus commute-related environments and background air are presented in the Table 3.1-5.

Table 3.1-5. Mean Concentrations of Black Carbon (BC), Particle Bound PAH, NO₂, Particle Count (PC), and PM_{2.5} in Three School Bus Commute Microenvironments and Background Air

	Mean Concentrations			
	Background	(Un)Loading Zone	Bus Stops	Bus Commutes ^a
BC (µg/m ³)	2 ± 0.1	2 ± 0.3	4 ± 0.4	3-19 (8)
Particle Bound -PAH (µg/m ³)	0.027 ± 0.0015	0.015 ± 0.0003	0.044 ± 0.0045	0.064-0.400 (0.134)
NO ₂ (ppb)	49 ± 1.0	35 ± 0.2	54 ± 1.9	34-110 (73)
PC (count/cm ³)	83 ± 3.1	Not collected	62 ± 1.8	77-236 (130)
PM _{2.5} (µg/m ³)	20 ± 2.4	Not collected	25 ^b	21-62 (43)

^a Ranges are associated with different bus types and window positions. Values in parenthesis are the mean for all runs.

^b Not enough data to establish a confidence interval.

In another recent study of commuter buses, concentrations of benzene and other VOCs were measured in buses on several routes in Detroit, MI.⁶² The average in-bus concentration of benzene was 4.5 µg/m³, while the average concentrations at three fixed sites taken during the study period ranged from 0.9-2.0 µg/m³. In this study, daily bus/ambient concentration ratios were reported, and ranged from 2.8-3.3 on the three reported study days. The in-bus concentrations were found to be most influenced by local traffic sources. A number of other studies similarly observe that passenger car commuters are exposed to elevated pollutant concentrations while driving on busy roads.^{63,64,65,66,67,68}

Older studies that examine in-vehicle concentrations in older model year vehicles are difficult to apply for regulatory analyses, due to the relatively rapid changes in vehicle emission controls over the last 15 years. In general, these studies indicate that concentrations in vehicles are significantly higher than ambient concentrations.^{69,70,71} The average benzene measurements of these older in-vehicle studies (Raleigh, NC and CA South Coast Air Basin) are in Table 3.1-6 along with the more recent studies for comparison.

Overall, these studies show that concentrations experienced by commuters and other roadway users are substantially higher than ambient air measured in typical urban air. As a result, the time a person spends in a vehicle will significantly affect their overall exposure.

Table 3.1-6. Benzene Concentrations ($\mu\text{g}/\text{m}^3$) Measured in Vehicles and in Ambient Air

Study	In-Vehicle		Ambient Air	
	Mean	Max	Mean	Max
Raleigh, NC (1989) ^a	11.6	42.8	1.9	8.5
CA South Coast Air Basin (1989) ^b	42.5	267.1	9.3-16.9	--
Boston, MA (1991) ^c	17.0	64.0	--	--
Los Angeles, CA (1998)	10-22	--	3-7	--
Sacramento, CA (1998)	3-15	--	1-3	--
Detroit, MI (2000) ^d	4.5	10.8	0.9-2.0	--
API Gasoline Screening (2002)	17.5	--	--	--
LA, CA School Buses (2003)	9.5	--	1.6	--
NC State Highway Patrol (2003)	12.8	43.1	0.32	1.92

^a A one-hour measurement was taken for each experimental trip.

^b The estimated sampling time period was 1.5 hours/round-trip. n=191.

^c In-vehicle measurement includes both interstate and urban driving, n=40.

^d Measurements taken from interiors of urban buses.

3.1.3.2.2 In Homes and Schools

The proximity of schools to major roads may result in elevated exposures for children due to potentially increased concentrations indoors and increased exposures during outdoor activities. Here we discuss international studies in addition to the limited number of US studies, because while fleets and fuels outside the U.S. can be much different, the spatial distribution of concentrations is relevant.

There are many sources of indoor air pollution in any home or school. These include indoor sources and outdoor sources, such as vehicle exhaust. Outdoor air enters and leaves a house by infiltration, natural ventilation, and mechanical ventilation. In infiltration, outdoor air flows into the house through openings, joints, and cracks in walls, floors, and ceilings, and around windows and doors. In natural ventilation, air moves through opened windows and doors. Air movement associated with infiltration and natural ventilation is caused by air temperature differences between indoors and outdoors and by wind. Finally, there are a number of mechanical ventilation devices, from outdoor-vented fans that intermittently remove air from a single room, such as bathrooms and kitchen, to air handling systems that use fans and duct work to continuously remove indoor air and distribute filtered and conditioned outdoor air to strategic points throughout the house. The majority of what is outdoors can therefore get indoors. A review of the literature determined that approximately 100% of gaseous compounds, such as benzene, and 80% of diesel PM can penetrate indoors.^{72,73}

In the Fresno Asthmatic Children's Environment Study (FACES), traffic-related pollutants were measured on selected days from July 2002 to February 2003 at a central site, and inside and outside of homes and outdoors at schools of asthmatic children.⁷⁴ Preliminary data indicate that PAH concentrations are higher at elementary schools located near primary roads than at elementary schools distant from primary roads (or located near primary roads with

limited access). PAH concentrations also appear to increase with increase in annual average daily traffic on nearest major collector. Remaining results regarding the variance in traffic pollutant concentrations at schools in relation to proximity to roadways and traffic density will be available in 2006.

The East Bay Children's Respiratory Health Study studied traffic-related air pollution outside of schools near busy roads in the San Francisco Bay Area in 2001.⁷⁵ Concentrations of the traffic pollutants PM₁₀, PM_{2.5}, black carbon, total NO_x, and NO₂ were measured at ten school sites in neighborhoods that spanned a busy traffic corridor during the spring and fall seasons. The school sites were selected to represent a range of locations upwind and downwind of major roads. Differences were observed in concentrations between schools nearby (< 300 m) versus those more distant (or upwind) from major roads. Investigators found spatial variability in exposure to black carbon, NO_x, NO, and (to a lesser extent) NO₂, due specifically to roads with heavy traffic within a relatively small geographic area.

An exposure assessment of PM₁₀ from a major highway interchange in East Los Angeles found that children in nearby schools were exposed to elevated pollutant levels.⁷⁶ Each of the four chosen schools was located within 500 m of a major limited-access highway, and three of them were within 150 m of the roadway. Using a computer model to calculate dispersion analysis, researchers predicted that average 24-hour (assuming 10-hour school-based exposure duration to account for time in class and at after-school programs) particle concentrations, which were dominated by road dust, would be 10.45, 14.58, 5.78, and 8.27 µg/m³, respectively, for the four schools studied. These results indicate a trend for increased emissions at school locations in closer proximity to the traffic source, with the exception of one school which was 25 m farther. These values reflect the increase in concentration over ambient exposure, not the total ambient exposure.

A study to assess children's exposure to traffic-related air pollution while attending schools near roadways was performed in the Netherlands.⁷⁷ Investigators measured PM_{2.5}, NO₂ and benzene inside and outside of 24 schools located within 400 m of roadways. The indoor average benzene concentration was 3.2 µg/m³, with a range of 0.6-8.1 µg/m³. The outdoor average benzene concentration was 2.2 µg/m³, with a range of 0.3-5.0 µg/m³. Overall results indicate that indoor pollutant concentrations are significantly correlated with traffic density and composition, percentage of time downwind, and distance from major roadways.

In another study performed in the Netherlands, investigators measured indoor concentrations of black smoke, PM₁₀, and NO₂ in twelve schools between the periods of May and August 1995.⁷⁸ The schools were located at varying distances from the motorway (35-645 m). Results indicate that black smoke and NO₂ concentrations inside the schools were significantly correlated with truck and/or car traffic intensity as well as percentage of time downwind from the motorway and distance of the school from the motorway. PM₁₀ concentrations measured in classrooms during school hours were highly variable and much higher than those measured outdoors, but they did not correlate with any of the distance or traffic parameters.

The TEACH study (Toxic Exposure Assessment – Columbia/Harvard) measured the concentrations of VOCs, PM_{2.5}, black carbon, and metals outside the homes of high school students in New York City.⁷⁹ The study was conducted during winter and summer of 1999 on 46 students and in their homes. Average winter (and summer) indoor concentrations exceeded outdoor concentrations by a factor of 2.3 (1.3). In addition, analyses of spatial and temporal patterns of MTBE concentrations, used as a tracer for motor vehicle pollution, were consistent with traffic patterns.

The RIOPA study was conducted in three cities (Los Angeles, CA, Houston, TX, and Elizabeth, NJ) during four seasons.^{80,81} The study examined 100 non-smoking homes sited in high-emissions environments, including residential areas near freeways, service stations, petroleum industrial estates, and mixed sources. The cities involved were selected to represent different sources: Los Angeles (mobile source dominated), Houston (stationary source dominated), and Elizabeth, NJ (mixture of sources). Of the polycyclic aromatic hydrocarbons (PAHs) analyzed, the presence of 5-7 ring PAHs indoors was attributed to outdoor sources which, in Los Angeles and Elizabeth, NJ, could be attributed to mobile sources.

Average benzene concentrations were determined in a recent evaluation of the exposure of urban inhabitants to atmospheric benzene in Athens, Greece.⁸² Home and personal levels of 50 non-smokers in six monitoring campaigns varied between 6.0-13.4 and 13.1-24.6 µg/m³, respectively. Urban levels varied between 15.4 and 27.9 µg/m³ with an annual mean of 20.4 µg/m³. The highest values were observed during the first two sampling periods in fall and winter, when wind speed was low. The low summer values were attributed to decreased vehicle traffic. Among home factors, only proximity to busy roads was determined to be an important influence on indoor benzene levels.

Children are exposed to elevated levels of air toxics not only in their homes, classrooms, and outside on school grounds, but also during their commute to school. See above discussion of in-vehicle (school bus and passenger car) concentrations of air toxics for one method of commuting. The discussion below also presents potential exposures to children from another commuting method.

3.1.3.2.3 Pedestrians and Bicyclists

Researchers have noted that pedestrians and cyclists along major roads experience elevated exposures to motor vehicle related pollutants. Although commuting near roadways leads to higher levels of exposure to traffic pollutants, the general consensus is that exposure levels of those commuting by walking or biking is lower than for those who travel by car or bus, (see discussion on in-vehicle exposure in previous section above). For example, investigators found that personal measurements of exposure to PM₁₀ concentrations were 16% higher inside the car than for the walker on the same route, but noted that a walker may have a larger overall exposure due to an increase in journey time.⁸³ Similarly, researchers found that traffic-related pollutant exposure concentrations of car drivers were higher than for cyclists.⁸⁴ Cyclists are typically on the border of the road or on dedicated bike paths and therefore further away from the vehicle emissions and are less delayed by traffic jams. However, after accounting for cyclists' higher ventilation, the uptake of CO, benzene, toluene, and xylenes by cyclists sometimes

approached that of car drivers, and for NO₂ it was significantly higher.

In the early 1990's, researchers studied the in-vehicle concentrations of a large number of compounds associated with motor vehicle use and the exposure to VOCs of a pedestrian on an urban sidewalk (50 m from roadways) in Raleigh, NC.⁸⁵ The mean concentration of benzene in the six pedestrian sidewalk samples was 6.8 µg/m³. This concentration was lower than the in-vehicle measurement (11.6 µg/m³), but higher than the fixed-site measurement (1.9 µg/m³) on urban roadways 100-300 m from streets.

The same researchers studied the exposure of commuters in Boston to VOCs during car driving, subway travel, walking, and biking.⁸⁶ For pedestrians, mean time-weighted concentrations of benzene, toluene, and xylenes of 10.6, 19.8, and 16.7 µg/m³, respectively, were reported. For cyclists, the time-weighted concentrations were similar to those of pedestrians, at 9.2, 16.3, and 13.0 µg/m³, respectively. In-vehicle exposure concentrations were higher as discussed above.

Numerous other studies which were conducted in Europe and Asia yield similar results. A survey of CO concentration was conducted for various transport modes along heavy traffic routes in Athens, Greece.⁸⁷ Results showed that mean CO levels for trips of 30 min were 21.4 ppm for private car, 10.4 ppm for bus, and 11.5 ppm for pedestrians. In Northampton, UK during the winter 1999, personal measurements of exposure to PM₁₀, PM_{2.5}, and PM₁ were made during walking and in-car journeys on two suburban routes.⁸⁸ In-car measurements were highest (43.16, 15.54, and 7.03 µg/m³ for PM₁₀, PM_{2.5}, and PM₁, respectively) followed by walking (38.18, 15.06, and 7.14 µg/m³, respectively). Background levels were only available for PM₁₀ (26.55 µg/m³), but were significantly lower than the walking exposure levels. Researchers found similar results for CO exposure levels of schoolchildren commuters.⁸⁹ So although personal exposures are greater for in-vehicle commutes, pedestrians and bicyclists in proximity to heavy traffic are exposed to elevated pollutant levels relative to background.

3.1.3.2.4 Measurement Uncertainties

A number of uncertainties limit our ability to fully describe the impacts of motor vehicle emissions. As described above, most people in the U.S. experience some level of exposure to emissions from motor vehicles. Thus, proper characterization of the level of these exposures is critical. However, the exposure assessment techniques used may not adequately represent the populations' true exposures to motor vehicle emissions.

Air quality measurements are expensive and therefore are limited. The high costs of measurement techniques affect the quantity of samples that can be collected and quantity of compounds that can be identified. As a result, measurements may only occur at central monitoring sites, rather than in microenvironments impacted by motor vehicle emissions. Air quality monitoring at these central sites often do not represent actual exposures, especially for populations living near roads.

Air quality samples are often integrated and therefore lack time resolution. This can result in difficulty in determining source contributions. Additionally, some compounds are hard

to measure accurately. For example, 1,3-butadiene is very reactive in the ambient atmosphere and has a short atmospheric lifetime, estimated to be only two hours.⁹⁰ Thus, this compound can easily break down before samples are analyzed. Also, a vapor pressure of 3.3 atm at 25°C makes it a very volatile compound. Secondary reactions are a confounding factor in air quality measurements and can add additional uncertainty to measured ambient concentrations.

Results from emissions studies suggest that simple methods of estimating the contribution of motor vehicle exhaust to exposure likely do not capture the substantial variability in the chemical and physical characteristics of motor vehicle exhaust. Comprehensive assessments of exposure will be a critical factor in identifying which compounds are impacting the near-road environment.

3.1.3.3 Exposure and Concentrations in Homes with Attached Garages

Residential indoor air quality is a major determinant of personal exposure, with most people spending the majority of their time indoors at home. According to the National Human Activity Pattern Survey, nationally, people spend an average of 16.68 hours per day indoors in a residence.⁹¹ The large fraction of time spent in this microenvironment implies that sources that impact indoor air are likely to have a substantial effect on personal exposure.

Indoor air quality is in large part determined by ventilation of indoor spaces. Natural ventilation occurs as a result of two factors: wind-induced pressure and the “stack effect.” The latter occurs when hot air rises in a home, causing a pressure drop in the lower part of the home, which then creates airflow into the home from higher-pressure locations outside the home. Natural ventilation can also be influenced by opening of windows and doors. Mechanical ventilation employs fans and sometimes ductwork to manage ventilation within a home.

Air can be drawn into a home from either outdoors, or in a home with an attached garage, from the garage. Air from the garage can have higher concentrations of VOCs and other pollutants as a result of the storage of vehicles, other engines and equipment, fuel (gasoline in gas cans), solvents, or cleaning products. As a result, homes with a greater fraction of airflow from the garage are more susceptible to air quality decrements from in-garage emissions.

Several studies have examined homes with attached garages to determine the fraction of residential air intake from the garage. One study from Minnesota examined homes constructed in 1994, 1998, and 2000.⁹² Homes built in 1994 had 17.4% of airflow originating in the garage. Homes built in 1998 and 2000 had 10.5% and 9.4% of airflow from the garage, respectively. In another study conducted in Ottawa, Ontario, an average of 13% of home air intake came from the garage.⁹³ That study also found that the house-garage interface area was as leaky as the rest of the building envelope. In another study from Washington, D.C., the house-garage interface was found to be 2.5 times as permeable as the rest of the house.⁹⁴ This discrepancy may indicate that homes built in colder climates are built more tightly than homes in warmer regions as a result of weather-sealing. However, there is no evidence that in regions with cold weather, colder temperatures lead to elevated indoor concentrations of VOCs.⁹⁵

Several studies have examined the influence of attached garages on indoor air and personal exposure. In the 1980's researchers identified attached garages as a major source of benzene and other VOCs in residences. The Total Exposure Assessment Methodology (TEAM) was a series of large, probability-based samples of people who underwent study of the air inside and outside their homes and in their personal breathing zones. The study took place in the 1980's, and found that a large fraction of an average nonsmoker's benzene exposure originated from sources in attached garages.⁹⁶

These early studies have highlighted the role of evaporative emissions within the garage as contributors to indoor air pollution. Since then, major changes have affected emissions from vehicles, including additional controls on evaporative emissions, on-board diagnostics, and state inspection and maintenance programs addressing evaporative emission controls. Several researchers have subsequently conducted air measurements in homes and in attached garages to evaluate the effects on indoor air.

Garage concentrations of benzene and other VOCs are generally much higher than either indoor or outdoor air, and constitute one of the highest-concentration microenvironments to which a person might typically be exposed. The garage also supplies contaminated air to the home to which it is attached, and emits the rest. One recent study from Michigan found average garage benzene concentrations of $36.6 \mu\text{g}/\text{m}^3$, with a standard deviation of $38.5 \mu\text{g}/\text{m}^3$, compared to mean and standard deviation concentrations of $0.4 \mu\text{g}/\text{m}^3$ and $0.12 \mu\text{g}/\text{m}^3$ in ambient air.⁹⁷ In Alaska, where fuel benzene levels tend to be very high and homes built very airtight, garage concentrations have been measured at even higher levels. One study measured average garage benzene concentrations of $101 \mu\text{g}/\text{m}^3$, with a standard deviation of $38 \mu\text{g}/\text{m}^3$.⁹⁸

Other studies have studied the effect of garages or the sources within them on indoor air quality. Most prominently, a group of Canadian investigators conducted source apportionment of indoor non-methane hydrocarbons (NMHC) in 16 Ontario homes in the late 1990's.⁹⁹ They also assembled source profiles from hot soak and cold start emissions, which they used to conduct source apportionment of total indoor air NMHC. All emissions samples and house testing were conducted using the same 1993 model year vehicle. Overall, while the vehicle was hot-soaking in the garage over a four hour sampling period, between 9 and 71% of the NMHC inside the house could be attributable to that vehicle's emissions. Similarly, in the two hours following a cold start event, between 13 and 85% of indoor NMHC could be attributed to the vehicle cold start. Prior to the hot soak testing, average indoor benzene concentrations were $3.77 \mu\text{g}/\text{m}^3$, while during the hot soak, concentrations averaged $13.4 \mu\text{g}/\text{m}^3$. In the garage, concentrations averaged $121 \mu\text{g}/\text{m}^3$ during the cold start. Prior to a cold start, indoor benzene concentrations averaged $6.98 \mu\text{g}/\text{m}^3$, while for the two hours following cold start, concentrations averaged $25.9 \mu\text{g}/\text{m}^3$. In the garage, concentrations averaged $422 \mu\text{g}/\text{m}^3$ over the two hours following cold start.

The study also conducted real-time monitoring of CO and total hydrocarbons (THC) within the house and garage. Overall, concentrations of CO and THC were relatively constant during hot-soaks, but following a cold start, indoor concentrations of CO and THC tended to rise sharply, and fall over the next two hours. This study provides direct evidence that a high fraction of indoor NMHC (or VOCs) are directly attributable to emission events occurring in the garage.

Other studies have examined the influence of attached garages by comparing homes with and without attached garages. In another study from Alaska, 137 Anchorage homes underwent indoor air quality monitoring for benzene and other VOCs.¹⁰⁰ Homes with attached garages had significantly higher concentrations of indoor benzene compared to homes without attached garages (70.8 $\mu\text{g}/\text{m}^3$ vs. 8.6 $\mu\text{g}/\text{m}^3$). In addition, elevated benzene indoors was also associated with the presence of a vehicle in the garage, fuel being opened in the garage, and the use of forced-air heaters.

In another Alaska study, concentrations of benzene and toluene in indoor air were found to be not significantly associated with their urinary biomarkers, but indoor concentrations were associated with the number of gasoline-powered engines stored in the garage.¹⁰¹ In a recent follow-up to the study, ventilation patterns in two homes were evaluated using perfluorocarbon tracers and a multi-zone indoor air quality model.¹⁰² In the study, average garage concentrations were consistently elevated relative to the home. Furthermore, the study calculated the “virtual” source strengths for benzene and toluene within the garage, and the garage was the only major source of benzene within the home. Median garage source strengths for benzene ranged from 14-126 mg/h.

Several population-based surveys have also found evidence of the influence of attached garages. The National Human Exposure Assessment Survey (NHEXAS) Phase I pilot study in Arizona was a representative exposure survey of the population. It found that in non-smoking homes with attached garages, distribution of toluene concentrations indoors was shifted significantly higher in homes with attached garages.¹⁰³ Homes with attached garages had median toluene levels of 24 $\mu\text{g}/\text{m}^3$, while homes without garages had median concentrations of 5 $\mu\text{g}/\text{m}^3$. The NHEXAS study in EPA Region 5 states was of similar design, but covering the states of the upper Midwest. Using multivariate statistics, investigators found that VOCs including benzene were associated with the storage of gasoline-powered equipment in an attached garage.¹⁰⁴

In one study from New Jersey, investigators evaluated the indoor air effects of a vehicle fueled with “M85” – an 85% methanol, 15% gasoline blend – parking in the garage of a single home.¹⁰⁵ Testing was undertaken with both normally-functioning and malfunctioning evaporative emissions controls, as well as with the HVAC system on and off. Garage benzene concentrations exceeded indoor concentration by approximately 10-fold. Furthermore, the room adjacent to the garage had substantially higher concentrations than a room on the opposite side of the house. This study provides evidence that the garage is a major source of benzene inside the house.

EPA undertook an investigation of the effect of attached garages on indoor air under various scenarios.¹⁰⁶ This study was undertaken to evaluate the magnitude of exposure underestimation using the national-scale exposure modeling techniques discussed above. Using a mass balance model, steady-state concentrations of benzene were calculated as a function of the concentration of air in the garage, the concentration of outdoor air, and the fraction of house air intake from a garage. Data were obtained from studies discussed above. Overall, using in-garage concentration data from Michigan, average indoor concentrations increase by approximately 4.2 $\mu\text{g}/\text{m}^3$, relative to concentrations estimated without an attached garage term.

Using data from Alaska, average indoor concentrations increase by $11.6 \mu\text{g}/\text{m}^3$, and using New Jersey data, by $9.2 \mu\text{g}/\text{m}^3$. As noted above, the National Human Activity Pattern Survey (NHAPS) estimates that the average person spends 16.68 hours per day indoors in a residence. Taking that into account, overall modeled exposures would be expected to increase by at least $2.9 \mu\text{g}/\text{m}^3$, using the Michigan data. These calculations imply that predicted exposures would more than double if attached garages were treated systematically in a national exposure model.

3.1.3.4 Exposure and Concentrations in Parking Garages

Relatively limited air quality data for parking garages is available in the literature. The following are results of air quality studies performed in parking garages, all of which indicate that air toxics and criteria pollutants measured in these environments are significantly higher than found in outdoor air.

In November 1990, a study of microenvironments, partially funded by the US EPA, evaluated the potential range in concentrations of selected air toxics.¹⁰⁷ Ten parking garages, along with gasoline stations and office buildings, were randomly chosen for sampling since they were among the least studied of the potentially important exposure microenvironments. The principal air contaminants monitored were benzene, formaldehyde, and CO. Additional compounds included toluene, xylenes, 1,2-dichloroethane, chloroform, carbon tetrachloride, perchloroethylene, 1,1,1-trichloroethane, 1,3-butadiene, and trichloroethylene. The majority of the compounds measured were significantly higher inside the garage compared to the ambient sample. For example, the median 5-minute concentration of benzene was $67.1 \mu\text{g}/\text{m}^3$ in the parking garage and $12.8 \mu\text{g}/\text{m}^3$ in ambient air. CO was 11000 ppb in the parking garage and 2000 ppb in ambient air. The researchers identified elevated levels of selected air toxics in parking garages and pointed out the potential contribution from cold starts at the end of the work day.

A more recent 2002 study was funded by The American Petroleum Institute to screen “high-end” exposure microenvironments as required by section 211(b) of the Clean Air Act.¹⁰⁸ The study included measurements at underground parking garages and surface parking lots in several cities. Air toxics quantified included hydrocarbons (HCs), carbonyl compounds, BTEX, total VOC, and CO. When sampling at parking lot exits, spikes in pollutant concentrations were observed when vehicles accelerated out of the parking lot, while presumably prior to full catalyst warm-up. In underground garages, the levels of BTEX and other compounds of interest varied with traffic level and reached concentrations that were significantly higher than ambient levels outside the garage.

A comparative study of indoor air quality in Hong Kong showed that the levels of CO, NO_x, and nonmethane hydrocarbons (NMHC) detected in a local park garage were the highest among 13 other indoor sampling locations.¹⁰⁹ The study did not specify the type or size of the chosen parking garage, but indicated that it was located in an urban commercial area. High indoor/outdoor ratios indicated that the air quality was mainly affected by indoor sources, namely the vehicle exhaust. They also concluded that the pollution generated might cause health hazards to the users and workers using such an environment.

Another assessment of the air quality in indoor park garages was performed in Hong Kong in August through December 2000.¹¹⁰ Air samples were collected in two different garages (an enclosed and semi-enclosed parking garage) as well as outdoors (within 10 m of each parking garage) and analyzed for one hundred different C3-C12 VOCs. Other compounds measured included CO, CO₂, PM₁₀, and PM_{2.5}. The CO levels in the enclosed garage were more than in the semi-enclosed garage, and double the levels of the outdoor air. The PM₁₀ and PM_{2.5} concentrations were also found to be higher in the parking garage environments than outdoors. High mass fractions of aliphatic and aromatic compounds detected in the enclosed garage showed that fuel evaporation and motor vehicular exhaust were the major contributors to the VOCs. The total concentrations of NMHC in the enclosed and semi-enclosed garages ranged from 580 to 4610 µg/m³ and 43.1 to 175 µg/m³, respectively. The mean concentration of NMHC measured in the enclosed garage (1910 µg/m³) was about 17 times higher than in the semi-enclosed garage (94.6 µg/m³), and 3 times higher than measured at the outdoor sites. Not only was the level of VOCs higher in the enclosed garage, but also the abundance of species identified. The most abundant species in similar ranking order for both garages was toluene, 2-methylbutane, *m/p*-xylenes, *n*-pentane, 2-methylpentane, *n*-hexane, and *n*-butane. Other major gasoline components such as benzene, xylenes, and C4-C7 saturated HCs were also very high in the enclosed garage. The difference between the two sites could be associated with the ventilation and location, since the occupancy rates and fleet mixes were similar. They also noted that the absence of sunlight in the enclosed garage would result in a slower or negligible photochemical depletion rate of unsaturated hydrocarbons, and consequently an increased abundance of the species observed.

In another study of multi-level parking garages in an Athens urban area, CO levels were characterized in autumn 1999.¹¹¹ Samples were collected at the exit sites (ramp where the flow of vehicles was concentrated), the indoor site (first underground level where the majority of cars parked), and immediately outside of each garage. Results indicate that CO levels varied significantly over site, time, and day of measurement. The peak 1-hour value at the indoor sites ranged from 22.9 to 109.3 ppm. At the indoor site, levels showed little variation and remained high over time. The peak 1-hour value at the exit sites ranged from 8.9 to 57.3 ppm. At the exit sites, 15-minute maximum concentrations were 5-15 times higher than the maximum recorded CO level immediately outside the garage. CO levels on Saturday were much lower than a typical weekday due to the reduced traffic, and weekday values were highest during the afternoon sampling times (12:00-16:00 hour) corresponding with peak traffic volumes.

In Mumbai, India, ambient levels of benzene were determined during different seasons at several different locations, including two parking areas.¹¹² Parameters of the parking areas at Liberty Cinema and Natraj Cinema were not specified, but 24-hour geometric means of benzene measured 117.4 and 74.2 µg/m³ during the summer, 94.5 and 75.4 µg/m³ during the monsoon, and 148.0 and 703.0 µg/m³ during the winter seasons, respectively. These values were considerably higher in comparison to less heavily trafficked residential locations. The mean benzene concentrations of four different residential locations ranged from 4.7 to 32.9 µg/m³, 1.9 to 33.5 µg/m³, and 4.7 to 18.8 µg/m³, respectively, for the summer, monsoon, and winter seasons. The high concentrations in parking areas were attributed to cold start-up emissions of engines.

A study in the UK of twelve underground parking garages identified high pollutant levels of NO_x, CO, CO₂, BTEX, and PM.¹¹³ The parking garages selected covered a cross-section of sizes (1 to 8 decks), ventilation system (natural and mechanical), designs (50 to 690 spaces), and usages (business, shopping, and/or residential). Monitoring sites were located inside and at the exit of the parking garage. The highest 15-minute average CO levels were measured at the exit of parking garages, but a number of the parking garages had CO levels consistently higher inside than at their exit. The NO₂ measurements showed similar trends. Weekday benzene concentration measurements averaged over one hour inside the parking garage and at the exit ranged from 60 to 870 µg/m³ and 10 to 350 µg/m³, respectively.

In Madrid, Spain, atmospheric pollution produced by vehicles in parking garages was studied.¹¹⁴ Two parking garages of different design were chosen for measurements of PM₁₀, lead, 12 PAHs, and CO. In both garages, CO, NO, TSP, and lead concentrations directly correlated with vehicle traffic flow into and out of the garage. Also, higher values were observed on the weekdays than during the weekend, for CO, NO, PAHs, and TSP in both garages. For example, in one garage, the average daily TSP concentrations were 78-122 µg/m³ on the weekdays versus 39 µg/m³ on the weekend, which was similar to outdoor city average measurement (50 µg/m³). The researchers conclude that maximum concentrations for NO were observed during maximum parking garage exits and therefore due to vehicle cold-starts. They also conclude that the mechanical ventilation used in both garages was not sufficient to disperse the pollutants emitted by the vehicles.

3.1.3.5 Exposure and Concentrations at Service Stations

Although there is relatively limited air quality data for service stations available currently in the literature, the general consensus is that exposures to air toxics at service stations significantly exceed ambient background levels. The studies below measure personal exposures and concentrations during refueling either inside or outside of vehicles throughout the United States. Several studies conducted outside of the United States chronicle similar results but are not presented here due to differences in fuels and control technologies.

The Total Exposure Assessment Methodology (TEAM) Study was planned in 1979 and completed in 1985.¹¹⁵ The goal of this study was to develop methods to measure individual total exposure (through air, food and water) and resulting body burden to toxic and carcinogenic chemicals, and then to apply these methods with a probability-based sampling framework to estimate the exposures and body burdens of urban populations in several U.S. cities. The study measured personal exposures of 600 people to a number of air toxics. The subjects were selected to represent residents of cities in New Jersey, North Carolina, North Dakota, and California. One of the major findings was that pumping gas as well as exposure to auto exhaust was a specific and major source of benzene exposure. People who filled their tanks with gasoline had twice as much benzene in their breath as people who did not. Estimated concentrations at the breathing zone could then exceed 1000 µg/m³ (100 times the ambient level), based on the median breath benzene value measured (n=67) for those who had worked at or been in a service station during the past 24 hours. But since then, implementation of fuel controls and Stage II vapor recovery have changed emission and concentration levels as discussed in Section 3.1.1.

In March 1990, another study randomly sampled 100 self-service filling stations throughout Southern California along with samples at 10 parking garages and 10 offices nearby those garages.¹¹⁶ The study took five-minute samples of 13 motor vehicle air pollutants (CO, formaldehyde, and VOCs) in each microenvironment and in the ambient environment. The median benzene concentration measured at the service stations was 28.8 $\mu\text{g}/\text{m}^3$ with the maximum reported value of 323 $\mu\text{g}/\text{m}^3$. The median benzene concentration in ambient air was significantly lower at 12.8 $\mu\text{g}/\text{m}^3$.

A 1993 National Institute for Occupational Safety and Health (NIOSH) study assessed benzene and MTBE concentrations and service station attendant exposures at service stations with and without Stage II vapor recovery in Cincinnati, Phoenix, and Los Angeles.¹¹⁷ The mean (and maximum) benzene exposure measurements were 96 (927), 160 (1662), and 192 (607) $\mu\text{g}/\text{m}^3$, respectively. The study found that Stage II vapor recovery did not significantly reduce exposure to benzene during refueling. However, the efficiency of Stage II vapor recovery has improved over the years. Northeast States for Coordinated Air Use Management (NESCAUM) has suggested that Stage II vapor recovery systems are greater than 90% effective at capturing MTBE and benzene vapors during refueling.¹¹⁸ These systems would therefore be expected to reduce exposure beyond that shown in the NIOSH exposure assessment.

In March 1996 to July 1997, concentrations of MTBE, benzene, and toluene were determined inside automobile cabins during fueling.¹¹⁹ Air samples were collected at service stations in New Jersey, and the mean benzene in-cabin concentration was 54.3 $\mu\text{g}/\text{m}^3$ (n=46). The background concentration at the pump island measured 9.6 $\mu\text{g}/\text{m}^3$ (n=36). The highest in-cabin concentrations for all three pollutants occurred in a car that had a malfunctioning vapor recovery system and in a series of cars sampled on an unusually warm, calm winter day when the fuel volatility was high, the evaporation maximal, and the wind dispersion minimal. The in-cabin concentrations were also typically higher when the car window was opened during the entire fueling process.

Most recently, as discussed in the section on in-vehicle and parking garage exposure and concentrations, a screening study of “high-end” exposure microenvironments was performed by the American Petroleum Institute.⁵⁷ The study included several vehicle-related microenvironments in Houston and Atlanta during summer 2002. Among the various microenvironments examined, the highest short-term concentrations occurred during refueling. The in-vehicle average concentration of benzene measured during refueling was 46.0 $\mu\text{g}/\text{m}^3$.

3.1.3.6 Occupational Exposure

Occupational settings can be considered a microenvironment in which exposure to benzene and other air toxics can occur. Occupational exposures to benzene from mobile sources or fuels can be several orders of magnitude greater than typical exposures in the non-occupationally exposed population. Several key occupational groups are discussed below.

Occupations that involve fuel distribution, storage, and tank remediation lead to elevated exposure to mobile-source related air toxics. Researchers published a review of benzene and total hydrocarbon exposures in the downstream petroleum industry, including exposure data

from the past two decades among workers in the following categories: refinery, pipeline, marine, rail, bulk terminals, tank truck drivers, service stations, underground storage tanks, tank cleaning, and site remediation.¹²⁰ The studies reviewed indicate that benzene exposure can range from <1 to more than 10 mg/m³, which is approximately three orders of magnitude higher than typical non-occupational exposures (although there are occurrences of high benzene exposures in non-occupational settings as well). This review is relevant because of the potential for mobile source benzene emission reductions to reduce their exposures as well. This statement is echoed by researchers in the occupational literature.¹²¹

Handheld and non-handheld equipment operators are also exposed to elevated concentrations of fuel-related air toxics. Several studies were conducted in work categories employing small engine equipment, such as lawn and garden workers, workers in construction/demolition, and others. Many of these occupations require the use of personal protective equipment to prevent high exposures to carbon monoxide or other species. At present, there are no representative samples of exposures among these categories. Non-occupational exposures from these equipment types may also be important contributors to overall exposure. EPA recently conducted a study of occupational exposures among lawn and garden workers using riding tractors, walk-behind lawn mowers, string trimmers, and chainsaws.¹²² Results demonstrated that equipment operators can experience highly variable exposures, with short-term personal concentrations of CO and PM_{2.5} ranging over two orders of magnitude. Air toxics data will be available later this year. This study illustrated the role of operator's activity in affecting exposure levels to fuel-related air toxics.

Another study provides some insight into the possible range of benzene exposures in workers who operate gasoline-powered engines, particularly those with 2-stroke engine cycles.¹²³ A study of snowmobile rider exposures in Sweden found benzene concentrations ranging from under 10 µg/m³ to 2.5 mg/m³, a range of at least two orders of magnitude. Exposures measured on riders on the back of the vehicle ranged from 0.7-0.8 mg/m³. These measurements illustrate the potential for relatively high exposures when operating 2-stroke equipment, as used in this study. Yellowstone National Park commissioned a study in 2002 to examine occupational exposures of park employees to benzene, other VOCs, PM₁₀, and CO.¹²⁴ Work shift benzene concentrations at a snowmobile entry gate 176.7 µg/m³, while snowmobile-bound mobile patrol officers' exposure concentrations averaged 137.20 µg/m³. The highest observed work shift concentration in the study was 514.1 µg/m³. At major sites of tourist interest where snowmobiles parked, such as the Old Faithful geyser, concentrations averaged 41.3 to 48.8 µg/m³. 15-minute "peak" samples of workers' personal air ranged from 46.8 µg/m³ to 842.8 µg/m³. This study provides an indication of the variability of occupational benzene exposure concentrations with time, and highlights the potential for elevated work shift exposures over several hours.

A preliminary report published by the Northeast States for Coordinated Air Use Management further illustrates the occupational impact of nonroad heavy-duty diesel equipment.¹²⁵ In-cabin and work site perimeter measurements were collected for diesel equipment emissions from the agricultural, construction (building and roadway), and lumber industries in the Northeast. Initial results indicate that PM_{2.5} concentrations were 1-16 times greater than the average ambient concentrations in each monitoring area. In-cabin exposures to

PM_{2.5} for operators ranged from 2 µg/m³ to over 660 µg/m³. Additionally, measured concentrations of acetaldehyde, benzene, and formaldehyde were found to be significantly elevated, although concentrations were not presented.

In addition, some occupations require that workers spend considerable time in vehicles, which increases the time they spend in a higher-concentration microenvironment. In-vehicle concentrations are discussed in Section 3.1.3.2.1 above.

3.2 Modeled Air Quality, Exposures, and Risks for Air Toxics

3.2.1 National-Scale Modeled Air Quality, Exposure, and Risk for Air Toxics

EPA assesses human health impacts from outdoor, inhalation, chronic exposures to air toxics in the National-Scale Air Toxics Assessment. It assesses lifetime risks assuming continuous exposure to levels of air toxics estimated for a particular point in time. The most recent National-Scale Air Toxics Assessment was done for the year 1999.¹²⁶ It has four steps:

- 1) Compiling a national emissions inventory of air toxics emissions from outdoor sources. The 1999 National Emissions Inventory is the underlying basis for the emissions information in the 1999 assessment.
- 2) Estimating ambient concentrations based on emissions as input to an air dispersion model (the Assessment System for Population Exposure Nationwide, or ASPEN model).¹²⁷
- 3) Estimating population exposures based on a screening-level inhalation exposure model (Hazardous Air Pollutant Exposure Model, version 5, or HAPEM5) and the estimated ambient concentrations (from the ASPEN model) as input to the exposure model.¹²⁸
- 4) Characterizing 1999 potential public health risks due to inhalation of air toxics. This includes cancer and noncancer effects, using available information on air toxics health effects, current EPA risk assessment and risk characterization guidelines, and estimated population exposures.¹²⁹

For this rule, we have conducted air quality, exposure and risk modeling for the years 2015, 2020, and 2030, using the same tools and methods as the 1999 National-Scale Air Toxics Assessment. Thus our results are comparable to the 1999 Assessment, other than in the few situations in which risk values were re-computed resulting from stationary source inventory errors which were determined to impact a tract or county-level risk estimate. For the reference case, which includes all control programs currently planned by EPA in regulations, we modeled all the pollutants in Table 2.2-1. Note that the modeling did not include diesel PM and diesel exhaust organic gases. For the fuel benzene control case, we modeled the following pollutants: benzene, 1,3-butadiene, formaldehyde, acetaldehyde, and acrolein. This modeling work is discussed in more detail in an EPA technical report, “National Scale Modeling of Air Toxics for the Mobile Source Air Toxics Rule; Technical Support Document,” Report Number EPA-454/R-06-002. EPA has previously done future year projections of the mobile source contribution to air toxics concentrations, exposure, and risk for selected air toxics,^{130, 131, 132, 133} but has never done a comprehensive assessment that includes projections for all mobile source air toxics, as well as the stationary source contribution for those pollutants.

The National-Scale Air Toxics Assessment modeling framework has a number of limitations which prevent its use as the sole basis for setting regulatory standards. Even so, this modeling framework is very useful in identifying air toxic pollutants and sources of greatest concern, setting regulatory priorities, and informing the decision making process. Among the significant limitations of the framework is that it cannot be used to identify “hot spots,” such as areas in immediate proximity to major roads, where the air concentration, exposure and/or risk might be significantly higher within a census tract or county. This limitation may result in underestimates of exposure due to the design of ASPEN. In addition, this kind of modeling assessment cannot address the kinds of questions an epidemiology study might allow, such as the relationship between asthma or cancer risk, and proximity of residences to point sources, roadways and other sources of air toxics emissions. The framework also does not account for risk from sources of air toxics originating indoors, such as stoves or out-gassing from building materials or evaporative benzene emissions from cars in attached garages. The ASPEN model performs well for some pollutants, but has also been shown to systematically underestimate pollutant concentrations relative to measured levels for certain pollutants such as metals. The cancer unit risk estimates for many pollutants are “upper bound,” meaning they probably lead to overestimates of risk. It should be noted, however, that the unit risk estimate for benzene is a maximum likelihood estimate, which is a best scientific estimate. The above limitations are discussed in detail in Section 3.2.1.4.

Another tool which has been used by EPA to assess distributions of concentrations of air toxics at the national scale is the Community Multiscale Air Quality Model (CMAQ).¹³⁴ In general, predicted concentrations of air toxics from CMAQ were within a factor of 2 of measured values, with a tendency to underpredict measured ambient concentrations.¹³⁵ CMAQ underpredicts monitored benzene levels more than ASPEN, although it better calculates the contribution of transport, and more accurately model the effect of benzene decay. CMAQ has sophisticated photochemistry, but does not yet have the spatial resolution of dispersion models such as ASPEN, and thus accounts for less of the total variability in levels of air toxics with localized effects, such as benzene.¹³⁶ Finally, CMAQ requires more computational resources, which makes it more difficult to use for evaluating trends in a large number of air toxics over many years or impacts of control scenarios.

Details of the methods used, and presentation of key results are discussed in the following sections. Results do not account for significant sources of inhalation exposure, such as benzene emissions from sources in attached garages (such as vehicles, snowblowers, lawnmowers and gas cans). Furthermore, the modeling underestimates the contribution of hydrocarbon and particulate matter emissions at cold temperatures, based on results of recent test data discussed in Chapter 2.

3.2.1.1 Air Quality Modeling

3.2.1.1.1 Methods

Prior to performing air quality modeling on the projected emissions, the emissions from the stationary and mobile inventories (discussed in Chapter 2) are processed in the Emissions

Modeling System for Hazardous Air Pollutants (EMS-HAP) Version 3 to create the emissions input files used by ASPEN to calculate air quality concentrations.¹³⁷ In addition to projecting stationary and area source emissions to future years for some source categories, EMS-HAP spatially allocates emissions inventoried at the county level to the census tract level, and temporally allocates them to eight three-hour time periods throughout the day. Once the emissions were processed, they were input into ASPEN to calculate air quality concentrations. In addition to the emissions, ASPEN uses meteorological parameters, and census tract centroid locations for concentration calculations. ASPEN estimates do not account for day-of-week or seasonal variations in emissions. The ASPEN model takes into account important determinants of pollutant concentrations, such as: rate of release, location of release, the height from which the pollutants are released, wind speeds and directions from the meteorological stations nearest to the release, breakdown of the pollutants in the atmosphere after being released (i.e., reactive decay), settling of pollutants out of the atmosphere (i.e., deposition), and transformation of one pollutant into another. The model first estimates concentrations at receptors arranged in rings around emission sources up to 50 km away. The model then interpolates concentrations to census tract centroids. For 1999 NATA, meteorological conditions in 1999 and 2000 census tract data were used.

In using ASPEN to estimate concentrations for emissions projected to years 2015, 2020, and 2030, the same meteorology and census tract locations were used, as for the 1999 NATA. Details of how ASPEN processed emissions data are provided in the technical document, “National-Scale Modeling of Mobile Source Air Toxic Emissions, Air Quality, Exposure and Risk for the Mobile Source Air Toxics Rule.” ASPEN only accounts for sources within a 50 kilometer radius of each source when calculating ambient concentrations. Thus, the contribution to ambient levels of air toxics from sources further away than 50 kilometers, as well as the contribution of uninventoried sources is addressed through the addition of a “background” term.¹³⁸ Mobile source pollutants which include a background component are 1,3-butadiene, acetaldehyde, benzene, formaldehyde, and xylenes. Each of the three projection years used the same 1999-based background. However, background levels are likely to change with emissions. Thus, a sensitivity analysis was done to evaluate the potential impact of not changing the background concentration (see Section 3.2.1.4).

We estimated the contributions to ambient concentrations for the following source sectors: major, area and other, onroad gasoline, onroad diesel, nonroad gasoline, remaining nonroad (diesel and compressed natural gas), and background.^B

^B Major and “area and other” are stationary source emission sectors. Major sources, as defined by the Clean Air Act, are those stationary facilities that emit or have the potential to emit 10 tons of any one toxic air pollutant or 25 tons of more than one toxic air pollutant per year. Area and other sources include sources that generally have smaller emissions on an individual basis than “major sources” and are often too small or ubiquitous in nature to be inventoried as individual sources. “Area sources” include facilities that have air toxics emissions below the major source threshold as defined in the air toxics sections of the Clean Air Act and thus emit less than 10 tons of a single toxic air pollutant or less than 25 tons of multiple toxic air pollutants in any one year. Area sources include smaller facilities, such as dry cleaners. “Other sources” include sources such as wildfires and prescribed burnings that may be more appropriately addressed by other programs rather than through regulations developed under certain air toxics provisions (section 112 or 129) in the Clean Air Act. For example, wildfires and prescribed burning are being addressed through the burning policy agreed to by the Interim Federal Wildland Policy. “Background” includes emissions from transport and uninventoried sources.

3.2.1.1.2 Air Quality Trends for Air Toxics

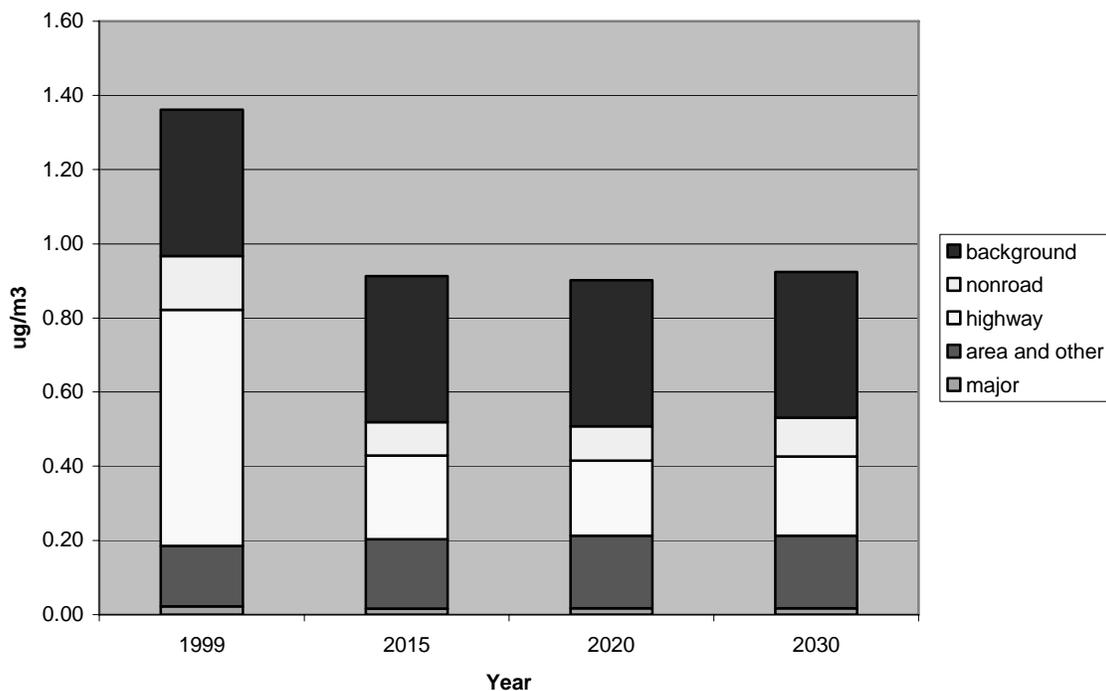
Table 3.2-1 summarizes nationwide mean census tract ambient concentrations of mobile source air toxics in 1999 and projection years for the following source sectors: major sources, area and other sources, highway vehicles, nonroad sources, and background. The behavior of benzene is typical of the projected trends. Over 90% of the mobile source contribution to ambient benzene levels is attributable to gasoline vehicles and engines. Figure 3.2-1 depicts the trend in nationwide average census tract concentrations of benzene over this time period. The mobile source contribution to ambient benzene concentrations is projected to decrease 60% by 2015, with a decrease in ambient benzene concentration from all sources of over 30%. Summary tables providing data by State, and for reformulated and non-reformulated gasoline areas, can be found in the docket for this rule. Due to greater population and vehicle activity, the average ambient benzene concentration in 1999 is much higher for counties in reformulated gasoline areas than nonreformulated gasoline areas – about $1.9 \mu\text{g}/\text{m}^3$ versus $1.1 \mu\text{g}/\text{m}^3$. However the percent reduction in average ambient concentration is similar regardless of fuel type – 29% for non-reformulated gasoline counties versus 35% for reformulated gasoline counties.

Table 3.2-1. Mean Ambient Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030

Pollutant	background ($\mu\text{g m}^{-3}$)	1999 average concentrations ($\mu\text{g m}^{-3}$)				2015 annual average concentrations ($\mu\text{g m}^{-3}$)					
		major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	5.10E-02	1.97E-03	2.05E-02	5.27E-02	1.59E-02	1.42E-01	2.17E-03	2.05E-02	1.46E-02	9.81E-03	9.81E-02
2,2,4-Trimethylpentane	0.00E+00	2.15E-02	3.10E-02	6.45E-01	1.27E-01	8.24E-01	1.07E-02	3.12E-02	2.17E-01	6.88E-02	3.28E-01
Acetaldehyde	5.17E-01	2.94E-02	5.49E-02	6.66E-01	1.62E-01	1.43E+00	2.97E-02	5.71E-02	2.49E-01	1.13E-01	9.66E-01
Acrolein	0.00E+00	3.21E-03	2.93E-02	5.87E-02	2.24E-02	1.14E-01	3.53E-03	2.62E-02	1.61E-02	1.80E-02	6.38E-02
Benzene	3.94E-01	2.24E-02	1.63E-01	6.36E-01	1.46E-01	1.36E+00	1.60E-02	1.88E-01	2.25E-01	9.05E-02	9.13E-01
Chromium III	0.00E+00	8.22E-04	4.53E-04	9.43E-05	6.34E-05	1.43E-03	1.04E-03	6.16E-04	1.43E-04	6.80E-05	1.87E-03
Chromium VI	0.00E+00	1.07E-04	1.98E-04	2.07E-05	1.39E-05	3.40E-04	1.36E-04	2.72E-04	3.15E-05	1.49E-05	4.54E-04
Ethyl Benzene	0.00E+00	1.84E-02	8.55E-02	2.62E-01	7.93E-02	4.45E-01	1.24E-02	1.12E-01	8.75E-02	4.59E-02	2.58E-01
Formaldehyde	7.62E-01	3.99E-02	8.77E-02	4.68E-01	2.42E-01	1.60E+00	4.98E-02	9.82E-02	1.41E-01	1.72E-01	1.22E+00
Hexane	0.00E+00	6.69E-02	4.48E-01	2.57E-01	5.18E-02	8.23E-01	5.95E-02	5.31E-01	1.03E-01	3.16E-02	7.26E-01
Manganese	0.00E+00	2.71E-03	2.22E-03	1.05E-04	1.60E-05	5.05E-03	3.23E-03	2.92E-03	1.66E-04	1.90E-05	6.33E-03
MTBE	0.00E+00	1.13E-02	6.43E-02	6.01E-01	8.92E-02	7.66E-01	1.14E-02	5.90E-02	9.63E-02	3.00E-02	1.97E-01
Naphthalene	0.00E+00	4.57E-03	4.76E-02	1.43E-02	4.44E-03	7.09E-02	3.99E-03	5.75E-02	7.80E-03	4.59E-03	7.39E-02
Nickel	0.00E+00	7.76E-04	1.42E-03	8.30E-05	1.08E-04	2.38E-03	8.87E-04	1.62E-03	1.26E-04	1.24E-04	2.75E-03
POM	0.00E+00	4.93E-03	1.63E-02	1.45E-03	8.64E-04	2.36E-02	3.79E-03	1.87E-02	6.99E-04	7.70E-04	2.40E-02
Propionaldehyde	0.00E+00	1.01E-02	2.33E-02	1.66E-01	4.05E-02	2.40E-01	9.31E-03	2.39E-02	6.51E-02	2.69E-02	1.25E-01
Styrene	0.00E+00	2.52E-02	1.42E-02	2.67E-02	6.86E-03	7.30E-02	3.00E-02	1.90E-02	8.23E-03	4.05E-03	6.13E-02
Toluene	0.00E+00	2.03E-01	8.04E-01	1.68E+00	3.06E-01	2.99E+00	1.43E-01	1.05E+00	5.45E-01	1.79E-01	1.91E+00
Xylenes	1.70E-01	9.99E-02	5.84E-01	1.00E+00	3.66E-01	2.22E+00	8.22E-02	7.80E-01	3.23E-01	1.99E-01	1.55E+00

Table 3.2-1 (cont'd). Mean Ambient Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030

Pollutant	background ($\mu\text{g m}^{-3}$)	2020 annual average concentrations ($\mu\text{g m}^{-3}$)					2030 annual average concentrations ($\mu\text{g m}^{-3}$)				
		major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	5.10E-02	2.34E-03	2.05E-02	1.35E-02	1.03E-02	9.77E-02	2.34E-03	2.05E-02	1.45E-02	1.18E-02	1.00E-01
2,2,4-Trimethylpentane	0.00E+00	1.15E-02	3.28E-02	1.92E-01	6.69E-02	3.03E-01	1.15E-02	3.28E-02	2.02E-01	7.24E-02	3.19E-01
Acetaldehyde	5.17E-01	3.10E-02	5.83E-02	2.16E-01	1.13E-01	9.36E-01	3.10E-02	5.83E-02	2.26E-01	1.24E-01	9.56E-01
Acrolein	0.00E+00	3.96E-03	2.54E-02	1.48E-02	1.89E-02	6.30E-02	3.96E-03	2.54E-02	1.60E-02	2.14E-02	6.67E-02
Benzene	3.94E-01	1.75E-02	1.95E-01	2.02E-01	9.30E-02	9.02E-01	1.75E-02	1.95E-01	2.13E-01	1.04E-01	9.24E-01
Chromium III	0.00E+00	1.17E-03	6.96E-04	1.61E-04	6.93E-05	2.09E-03	1.17E-03	6.96E-04	2.01E-04	7.20E-05	2.14E-03
Chromium VI	0.00E+00	1.54E-04	3.07E-04	3.53E-05	1.52E-05	5.12E-04	1.54E-04	3.07E-04	4.42E-05	1.58E-05	5.21E-04
Ethyl Benzene	0.00E+00	1.39E-02	1.23E-01	7.71E-02	4.63E-02	2.60E-01	1.39E-02	1.23E-01	8.09E-02	5.15E-02	2.69E-01
Formaldehyde	7.62E-01	5.65E-02	1.03E-01	1.29E-01	1.73E-01	1.22E+00	5.65E-02	1.03E-01	1.41E-01	1.90E-01	1.25E+00
Hexane	0.00E+00	6.54E-02	5.73E-01	8.57E-02	3.19E-02	7.56E-01	6.54E-02	5.73E-01	8.77E-02	3.55E-02	7.62E-01
Manganese	0.00E+00	3.59E-03	3.21E-03	1.88E-04	2.03E-05	7.00E-03	3.59E-03	3.21E-03	2.38E-04	2.31E-05	7.06E-03
MTBE	0.00E+00	1.28E-02	6.13E-02	7.39E-02	3.08E-02	1.79E-01	1.28E-02	6.13E-02	7.07E-02	3.47E-02	1.80E-01
Naphthalene	0.00E+00	4.47E-03	6.12E-02	7.76E-03	4.87E-03	7.83E-02	4.47E-03	6.12E-02	9.01E-03	5.56E-03	8.02E-02
Nickel	0.00E+00	9.61E-04	1.78E-03	1.41E-04	1.30E-04	3.01E-03	9.61E-04	1.78E-03	1.76E-04	1.42E-04	3.06E-03
POM	0.00E+00	4.21E-03	1.92E-02	7.20E-04	7.77E-04	2.49E-02	4.21E-03	1.92E-02	8.44E-04	8.65E-04	2.51E-02
Propionaldehyde	0.00E+00	9.35E-03	2.45E-02	5.52E-02	2.69E-02	1.16E-01	9.35E-03	2.45E-02	5.69E-02	2.93E-02	1.20E-01
Styrene	0.00E+00	3.44E-02	2.10E-02	7.60E-03	4.22E-03	6.71E-02	3.44E-02	2.10E-02	8.15E-03	4.79E-03	6.83E-02
Toluene	0.00E+00	1.60E-01	1.15E+00	4.85E-01	1.78E-01	1.97E+00	1.60E-01	1.15E+00	5.12E-01	1.96E-01	2.01E+00
Xylenes	1.70E-01	9.29E-02	8.59E-01	2.86E-01	2.00E-01	1.61E+00	9.29E-02	8.59E-01	3.02E-01	2.22E-01	1.66E+00

Figure 3.2-1. Nationwide Average Benzene Concentration, 1999-2030

3.2.1.1.3 Distributions of Air Toxic Concentrations across the U. S.

Table 3.2-2 gives the distribution of census tract concentrations, summed across all source sectors and background, for mobile source air toxics across the nation in 2020. Distributions for other years are similar. Summary tables providing distributions for other years, as well as distributions by State and for reformulated and non-reformulated gasoline areas, can be found in the docket for this rule. From this table, it can be seen that 95th percentile of average census tract concentrations for mobile-source dominated pollutants such as benzene and 1,3-butadiene are typically two to five times higher than the median of census tract concentrations, even though mobile source emissions are widely dispersed. For pollutants with large major source contributions (e.g., manganese), the 95th percentile of census tract averages can be much higher than the median. In addition, average census tract concentrations can span one to several orders of magnitude. Thus, there is considerable variation in average concentrations across the U.S.

Figure 3.2-2 depicts the geographic distribution of county median concentrations of benzene in 2020. Relatively high levels are seen in the Northeast, Southern California, Florida, parts of Texas, and the Great Lakes Region, where there is high population density and thus high vehicle and nonroad equipment activity. Relatively high levels are also seen in the Pacific Northwest, parts of Alaska, and the upper Great Lakes region. Analysis of fuel survey data indicate higher than average fuel benzene levels in these areas. Higher benzene levels in Idaho are not due to fuel benzene levels, but are primarily due to wildfire emission estimates, which were determined to be an error in the 1999 National Emissions Inventory and the subsequent projections.

Table 3.2-2. National Distribution of Census Tract Concentrations for Mobile Source Air Toxics in 2020

Pollutant	2020 concentration ($\mu\text{g m}^{-3}$) distribution						
	5th percentile	10th percentile	25th percentile	Median	75th percentile	90th percentile	95th percentile
1,3-Butadiene	2.24E-03	4.29E-03	2.70E-02	7.65E-02	1.16E-01	1.71E-01	2.85E-01
2,2,4-Trimethylpentane	2.43E-02	4.30E-02	1.05E-01	2.32E-01	4.02E-01	6.39E-01	8.15E-01
Acetaldehyde	5.33E-01	5.61E-01	6.51E-01	8.31E-01	1.07E+00	1.46E+00	1.78E+00
Acrolein	4.41E-03	7.44E-03	1.64E-02	3.44E-02	7.11E-02	1.55E-01	2.49E-01
Benzene	3.25E-01	3.87E-01	5.67E-01	7.97E-01	1.06E+00	1.48E+00	1.84E+00
Chromium III	7.72E-06	1.99E-05	8.12E-05	3.14E-04	1.03E-03	2.66E-03	5.06E-03
Chromium VI	2.45E-06	6.56E-06	2.97E-05	1.16E-04	3.30E-04	9.80E-04	1.63E-03
Ethyl Benzene	1.50E-02	2.73E-02	7.64E-02	1.78E-01	3.09E-01	5.00E-01	7.17E-01
Formaldehyde	3.99E-01	5.16E-01	7.80E-01	1.10E+00	1.44E+00	2.03E+00	2.53E+00
Hexane	2.75E-02	5.18E-02	1.68E-01	4.29E-01	8.21E-01	1.65E+00	2.81E+00
MTBE	2.16E-03	5.19E-03	1.61E-02	4.96E-02	1.95E-01	5.32E-01	7.62E-01
Manganese	1.54E-05	4.66E-05	2.06E-04	8.72E-04	3.56E-03	1.53E-02	2.26E-02
Naphthalene	2.98E-03	6.06E-03	1.91E-02	4.63E-02	9.19E-02	1.81E-01	2.80E-01
Nickel	1.41E-05	3.91E-05	1.69E-04	6.80E-04	2.11E-03	5.13E-03	8.75E-03
POM	1.72E-03	2.95E-03	5.70E-03	1.19E-02	2.06E-02	3.59E-02	5.76E-02
Propionaldehyde	9.97E-03	1.68E-02	3.73E-02	8.19E-02	1.50E-01	2.65E-01	3.53E-01
Styrene	2.06E-03	3.95E-03	9.58E-03	2.11E-02	4.45E-02	9.45E-02	1.62E-01
Toluene	1.17E-01	2.13E-01	5.64E-01	1.28E+00	2.29E+00	4.11E+00	5.88E+00
Xylenes	2.44E-01	3.04E-01	5.44E-01	1.04E+00	1.74E+00	3.15E+00	4.90E+00

Similar benzene median county concentration maps for 1999, 2015, and 2030 can be found in the docket for this rule, along with maps for other mobile source air toxics and tables of concentration distributions.

3.2.1.1.4 Impacts of Proposed Fuel Benzene Controls on Ambient Concentrations

The fuel benzene standard proposed in this rule will substantially reduce ambient concentrations of benzene across the United States. Table 3.2-3 shows that in 2015, 2020, and 2030, the highway vehicle portion of ambient concentrations will be reduced on average 8 to 9% across the U.S., the nonroad equipment contribution will be reduced about 7%, and the area source contribution about 4%. The reduction for area sources is due to the impacts of fuel benzene control on gasoline distribution emissions. Reductions in non-Federal reformulated gasoline areas (i.e., conventional gasoline areas) are even larger. It should be noted that the estimated total reductions are probably significantly underestimated, since we could not account for the impacts of controls on background levels, which includes transport of emissions from these sources. The fuel benzene control proposed does not significantly affect ambient concentrations of other air toxics. Figure 3.2-3 presents the distribution of percent reductions in median ambient benzene concentrations for U. S. counties with the proposed fuel control in 2020. Summary tables providing data by State, as well as maps of benzene concentrations with fuel controls and percent reductions with controls, can be found in the docket for the rule.

Similar data are also available for 1,3-butadiene, formaldehyde and acetaldehyde, even though concentrations were not significantly affected.

Figure 3.2-2. Geographic Distribution of County Median Concentrations ($\mu\text{g}/\text{m}^3$) of Benzene in 2020

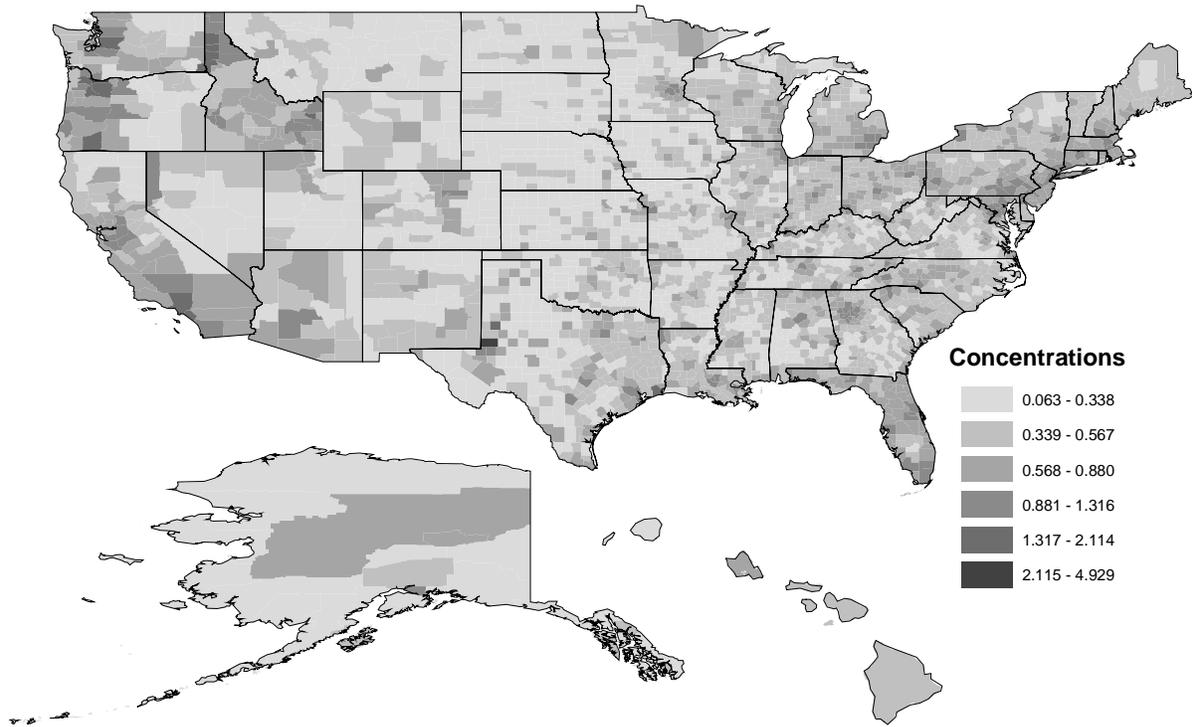
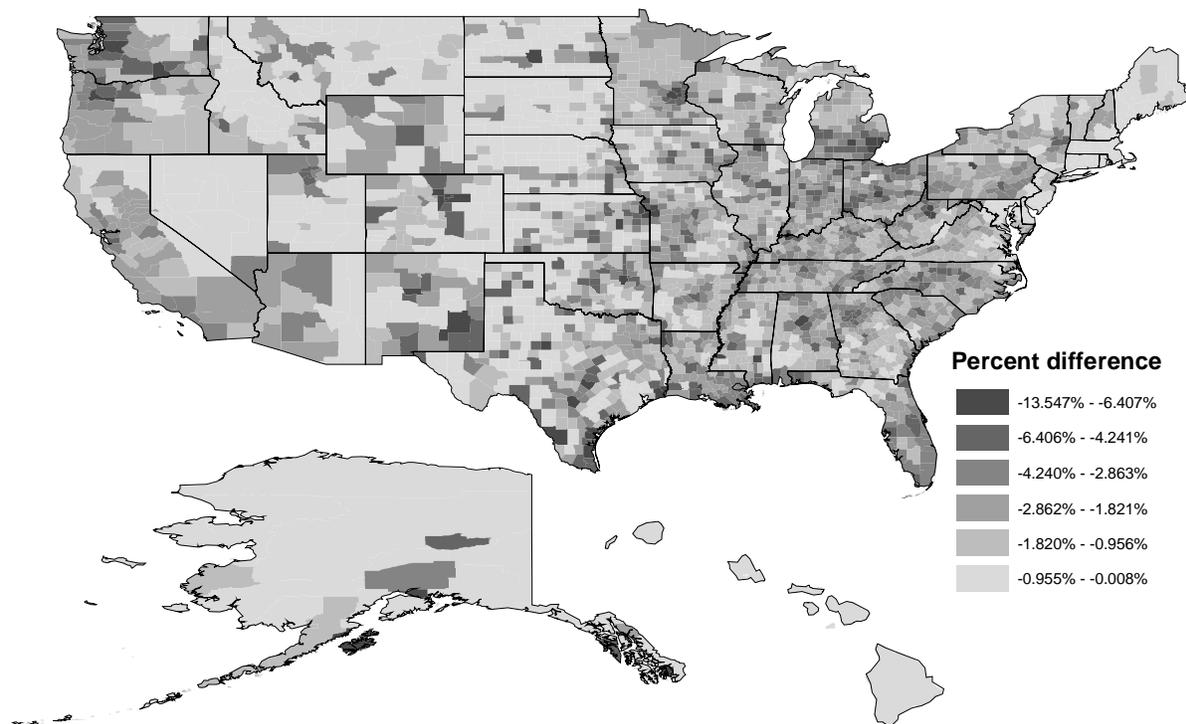


Table 3.2-3. Contributions of Source Sectors to Nationwide Average Census Tract Concentrations of Benzene, with and without Proposed Fuel Benzene Standard, 2015, 2020, and 2030

	2015 annual average concentrations (µg m ⁻³)					2020 annual average concentrations (µg m ⁻³)					2030 annual average concentrations (µg m ⁻³)				
	major	area & other	highway vehicles	nonroad	total (including background)	major	area & other	highway vehicles	nonroad	total (including background)	major	area & other	highway vehicles	nonroad	total (including background)
Reference	0.02	0.19	0.23	0.09	0.91	0.02	0.20	0.20	0.09	0.90	0.02	0.20	0.21	0.10	0.92
Control	0.02	0.18	0.20	0.08	0.88	0.02	0.19	0.18	0.09	0.87	0.02	0.19	0.20	0.10	0.89
% Difference	-1	-4	-9	-7	-4	-1	-4	-9	-7	-4	-1	-4	-8	-7	-4
Average Nationwide Difference in Ambient Benzene Concentration -- Non RFG Areas															
Reference	0.01	0.15	0.18	0.06	0.77	0.01	0.16	0.16	0.07	0.76	0.01	0.16	0.16	0.07	0.77
Control	0.01	0.15	0.15	0.06	0.73	0.01	0.15	0.14	0.06	0.72	0.01	0.15	0.14	0.06	0.73
% Difference	-1	-4	-14	-12	-5	-1	-4	-13	-12	-5	-1	-4	-13	-13	-5
Average Nationwide Difference in Ambient Benzene Concentration -- RFG Areas															
Reference	0.02	0.25	0.31	0.14	1.18	0.03	0.26	0.28	0.14	1.16	0.03	0.26	0.31	0.16	1.20
Control	0.02	0.24	0.30	0.13	1.15	0.03	0.25	0.27	0.14	1.14	0.03	0.25	0.29	0.15	1.18
% Difference	-1	-3	-4	-3	-2	-1	-3	-4	-3	-2	-1	-3	-4	-3	-2

Figure 3.2-3. Distribution of Percent Reductions in Median Ambient Benzene Concentrations, 2020, for U. S. Counties with the Proposed Fuel Control



3.2.1.2 Exposure and Risk Modeling

3.2.1.2.1 Methods

The HAPEM5 exposure model used in this assessment is the most recent version in a series of models that the EPA has used to model population exposures and risks at the urban and national scale in a number of assessments.^{139, 140, 141} HAPEM5 is designed to assess average long-term inhalation exposures of the general population, or a specific sub-population, over spatial scales ranging from urban to national. HAPEM5 uses the general approach of tracking representatives of specified demographic groups as they move among indoor and outdoor microenvironments and among geographic locations. The estimated pollutant concentrations in each microenvironment visited are combined into a time weighted average concentration, which is assigned to members of the demographic group.

HAPEM5 uses four primary sources of information: population data from the US Census, population activity data, air quality data, and microenvironmental data. The population data used is obtained from the US census. Two kinds of activity data are used: activity pattern data and commuting pattern data. The activity pattern data quantify the amount of time individuals spend in a variety of microenvironments and come from EPA's Consolidated Human Activity Database (CHAD).¹⁴² The commuting data contained in the HAPEM5 default file were derived from a special 1990 US Census study that specifies the number of residents of each tract that work in that tract and every other US Census tract. The air quality data come from ASPEN (after

background has been added). The microenvironmental data consist of factors that estimate air toxic concentrations in specific microenvironments, based on penetration of outdoor air into the microenvironment, proximity of the microenvironment to the emission source, and emission sources within the microenvironment. These factors vary among pollutants.¹⁴³

HAPEM5 has a number of technical improvements over the previous version of HAPEM. These improvements, along with other details of the model, are described in the HAPEM5 User's Guide.¹⁴⁴ The projection year HAPEM runs used year 2000 census data and 1990 commuting pattern data. Average lifetime exposure for an individual in a census tract was calculated from data for individual demographic groups using a post-processing routine. We estimated the contributions to ambient concentrations for the following source sectors: major, area and other, onroad gasoline, onroad diesel, nonroad gasoline, remaining nonroad (diesel and compressed natural gas), and background.

Once HAPEM runs were completed, cancer risk and non-cancer risk calculations were made for each of the mobile source air toxic pollutants. Table 3.2-4 lists the pollutants with their respective Unit Risk Estimates (UREs) for cancer calculations and reference concentrations (RfCs) for non-cancer calculations. These are the same values used in the 1999 National-Scale Air Toxics Assessment, and more detailed information on how dose-response values were selected is provided at the website for that assessment. Also listed are the cancer weight of evidence classifications and target organ system(s) for non-cancer calculations. The weight of evidence classifications provided in this table were developed under EPA's 1986 risk assessment guidelines where:

A = Known human carcinogen

B1 = Probable human carcinogen, based on incomplete human data

B2 = Probable human carcinogen, based on adequate animal data

C = Possible human carcinogen

Dose-response values were selected using the following hierarchy:

- 1) Externally peer reviewed draft EPA Integrated Risk Information System (IRIS) assessments (not yet finalized).
- 2) EPA IRIS assessments.
- 3) Agency for Toxic Substances and Disease Registry (ATSDR) minimum risk levels (MRLs) for non-cancer effects – used as RfC.
- 4) California Office of Environmental Health Hazard Assessment (OEHHA) values.

Table 3.2-4. Dose Response Values Use in Risk Modeling (Concentrations in $\mu\text{g}/\text{m}^3$)

HAP	Carcinogen Class	URE (per $\mu\text{g}/\text{m}^3$)	Source	Organ Systems	RfC (mg/ m^3)	Source
1,3-Butadiene	A	3.0×10^{-5}	IRIS	Reproductive	2.0×10^{-3}	
2,2,4-Trimethylpentane	N/A	N/A		N/A	N/A	
Acetaldehyde	B2	2.2×10^{-6}	IRIS	Respiratory	9.0×10^{-3}	IRIS
Acrolein		0		Respiratory	2.0×10^{-5}	IRIS
Benzene	A	7.8×10^{-6}	IRIS	Immune	3.0×10^{-2}	IRIS
Chromium III	N/A	N/A		N/A	N/A	
Chromium VI	A	1.2×10^{-2}	IRIS	Respiratory	1.0×10^{-4}	IRIS
Ethyl Benzene		0		Developmental	1.0	IRIS
Formaldehyde	B	5.5×10^{-9}	CIIT	Respiratory	9.8×10^{-3}	ATSDR
Hexane		N/A		Respiratory, Neurological	2.0×10^{-1}	IRIS
Manganese		N/A		Neurological	5.0×10^{-5}	IRIS
MTBE		N/A		Liver, Kidney, Ocular	3.0	IRIS
Naphthalene	C	3.4×10^{-5}	CAL	Respiratory	3.0×10^{-3}	IRIS
Nickel	A	1.6×10^{-4}	EPA/OAQ PS	Respiratory, Immune	6.5×10^{-5}	CAL
Propionaldehyde	N/A	N/A		N/A	N/A	
POM1	B2	5.5×10^{-5}	OAQPS		N/A	
POM2	B2	5.5×10^{-5}	OAQPS		N/A	
POM3	B2	1.0×10^{-1}	OAQPS		N/A	
POM4	B2	1.0×10^{-2}	OAQPS		N/A	
POM5	B2	1.0×10^{-3}	OAQPS		N/A	
POM6	B2	1.0×10^{-4}	OAQPS		N/A	
POM7	B2	1.0×10^{-5}	OAQPS		N/A	
POM8	B2	2.0×10^{-4}	OAQPS		N/A	
Styrene		N/A		Neurological	1.0	IRIS
Toluene		N/A		Respiratory, Neurological	4.0×10^{-1}	IRIS
Xylenes		N/A		Neurological	1.0×10^{-1}	IRIS

There are a number of exceptions to this hierarchy:

- 1) Formaldehyde -- EPA no longer considers the formaldehyde URE reported in IRIS, which is based on a 1987 study, to represent the best available science in the peer-reviewed literature. Accordingly, the 1999 risk estimates for formaldehyde are based on a dose-response value developed by the CIIT Centers for Health Research (formerly the Chemical Industry Institute of Toxicology) and published in 1999. This issue is discussed in Chapter 1 of the RIA.
- 2) Nickel -- The IRIS URE for nickel inhalation shown in Table 3.2.-4 was derived from evidence of the carcinogenic effects of insoluble nickel compounds in crystalline form. Soluble nickel species, and insoluble species in amorphous form, do not appear to produce genotoxic effects by the same toxic mode of action as insoluble crystalline nickel. Nickel speciation information for some of the largest nickel-emitting sources (including oil combustion, coal combustion, and others) suggests that at least 35% of total nickel emissions may be soluble compounds. The remaining insoluble nickel

emissions are not well-characterized, however. Consistent with this limited information, this analysis has conservatively assumed that 65% of emitted nickel is insoluble, and that all insoluble nickel is crystalline. On this basis, the nickel URE (based on nickel subsulfide, and representative of pure insoluble crystalline nickel) was adjusted to reflect an assumption that 65% of the total mass of nickel may be carcinogenic. The ATSDR MRL in Table 2 was not adjusted, however, because the noncancer effects of nickel are not thought to be limited to the crystalline, insoluble form.

3) POM -- POM was divided into eight toxicity categories to cover the range of unit risks of the individual POM species and POM groups contained in the 1999 NEI. The unit risks for those eight categories were based on the midpoint of the range of unit risks defining the toxicity category. More details on the development of these unit risks can be found on the website for the 1999 National Scale Assessment and in Appendix H of the 2001 EPA draft report to the Science Advisory Board on the 1996 National-Scale Assessment.¹⁴⁵

Cancer risk estimates (the product of unit risk estimates and exposure levels) for various pollutants were assumed to be additive, since there was no evidence of non-additive interactions for any of the pollutants. Most of the estimates are based on the statistical upper confidence limit (UCL) of the fitted dose-response curve, but the estimates for hexavalent chromium, nickel, and benzene are based on the statistical best fit ("maximum likelihood estimate," or MLE).

To express chronic noncancer hazards, we used the RfC as part of a calculation called the hazard quotient (HQ), which is the ratio between the concentration to which a person is exposed and the RfC. A value of the HQ less than one indicates that the exposure is lower than the RfC and that no adverse health effects would be expected. A value of the HQ greater than one indicates that the exposure is higher than the RfC. However, because many RfCs incorporate protective assumptions in the face of uncertainty, an HQ greater than one does not necessarily suggest a likelihood of adverse effects. Furthermore, the HQ cannot be translated to a probability that adverse effects will occur and is not likely to be proportional to risk. A HQ greater than one can best be described as indicating that a potential exists for adverse health effects. Following the approach used in the 1999 National-Scale Assessment, combined noncancer hazards were calculated using the hazard index (HI), defined as the sum of hazard quotients for individual air toxics compounds that affect the same organ or organ system. The HI is only an approximation of the combined effect, because some of the substances may affect the target organs in different (i.e., non-additive) ways. As with the HQ, a value of the HI below 1.0 will likely not result in adverse effects over a lifetime of exposure. However, a value of the HI greater than 1.0 does not necessarily suggest a likelihood of adverse effects. Furthermore, the HI cannot be translated to a probability that adverse effects will occur and is not likely to be proportional to risk. A HI greater than one can be best described as indicating that a potential may exist for adverse health effects.

3.2.1.2.2 Exposure and Risk Trends for Air Toxics

Table 3.2-5 summarizes nationwide average census tract exposure concentrations of mobile source air toxics in 1999, 2015, 2020, and 2030. It should be noted that all the other non-inventoried sources, as well as the contribution from transport, contribute to background levels.

Overall, exposure concentrations tend to be less than ambient concentrations because penetration rates to indoor microenvironments are typically less than one. However, highway vehicles make a larger contribution to overall average population exposures than they do to ambient levels. This is largely because of elevated exposures experienced inside vehicles.

Table 3.2-6 summarizes national average population cancer risk across census tracts for these years by pollutant, as well as total cancer risk across pollutants. The total cancer risk from mobile source air toxics (including the stationary source contribution) was about 23 in a million in 1999. This compares to an overall nationwide average population cancer risk from all air toxics in the 1999 National-Scale Assessment of 48 in a million. About twenty-two percent of this risk in the 1999 National Scale Assessment is attributable to benzene. If an “upper bound” unit risk were used for benzene, this contribution would be even larger.

In all projection years, benzene emissions are by far the largest contributor to cancer risk from mobile sources (see Figure 3.2-4). Furthermore, about 90% of the mobile source risk from all air toxics is due to gasoline vehicles and engines, and about 95% of the benzene risk from mobile sources is from gasoline vehicles and engines. Other significant contributors to cancer risk from mobile source air toxics include 1,3-butadiene, acetaldehyde, naphthalene, and hexavalent chromium. If the cancer unit risk for formaldehyde currently used in IRIS had been used in this assessment, it would have been a major contributor as well.

Despite significant reductions in risk from mobile source air toxics, average inhalation cancer risks for these pollutants, accounting for both mobile and stationary source contributions, remain well above 1 in 100,000 (Figure 3.2-5). In addition, average risk from exposure to benzene remains above 5 in 1,000,000.

Table 3.2-5. Mean Population Exposure Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030

Pollutant	background ($\mu\text{g m}^{-3}$)	1999 average exposure concentrations ($\mu\text{g m}^{-3}$)					2015 annual average exposure concentrations ($\mu\text{g m}^{-3}$)				
		major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	3.82E-02	1.61E-03	1.72E-02	6.53E-02	1.51E-02	1.37E-01	1.78E-03	1.73E-02	1.84E-02	9.27E-03	8.58E-02
2,2,4-Trimethylpentane	0.00E+00	1.80E-02	2.67E-02	7.57E-01	1.12E-01	9.14E-01	9.01E-03	2.71E-02	2.56E-01	6.04E-02	3.52E-01
Acetaldehyde	3.97E-01	2.51E-02	4.64E-02	7.60E-01	1.41E-01	1.37E+00	2.54E-02	4.86E-02	2.86E-01	9.77E-02	8.58E-01
Acrolein	0.00E+00	2.72E-03	2.48E-02	6.72E-02	1.95E-02	1.14E-01	3.01E-03	2.23E-02	1.86E-02	1.54E-02	5.94E-02
Benzene	2.98E-01	1.88E-02	1.42E-01	7.58E-01	1.33E-01	1.35E+00	1.35E-02	1.64E-01	2.71E-01	8.28E-02	8.33E-01
Chromium III	0.00E+00	3.28E-04	1.86E-04	6.10E-05	2.67E-05	6.01E-04	4.17E-04	2.52E-04	9.27E-05	2.84E-05	7.90E-04
Chromium VI	0.00E+00	4.39E-05	8.19E-05	1.36E-05	5.89E-06	1.45E-04	5.58E-05	1.12E-04	2.07E-05	6.25E-06	1.95E-04
Ethyl Benzene	0.00E+00	1.55E-02	7.52E-02	3.08E-01	7.13E-02	4.70E-01	1.05E-02	9.77E-02	1.03E-01	4.10E-02	2.52E-01
Formaldehyde	5.85E-01	3.47E-02	7.43E-02	5.79E-01	2.14E-01	1.49E+00	4.32E-02	8.43E-02	1.76E-01	1.49E-01	1.04E+00
Hexane	0.00E+00	5.68E-02	3.89E-01	3.03E-01	4.64E-02	7.95E-01	5.04E-02	4.82E-01	1.22E-01	2.82E-02	6.63E-01
Manganese	0.00E+00	9.45E-03	5.38E-02	7.03E-01	7.95E-02	8.45E-01	9.57E-03	4.99E-02	1.14E-01	2.59E-02	2.00E-01
MTBE	0.00E+00	1.07E-03	9.12E-04	7.17E-05	7.60E-06	2.06E-03	1.28E-03	1.20E-03	1.14E-04	8.84E-06	2.60E-03
Naphthalene	0.00E+00	3.94E-03	4.04E-02	1.73E-02	4.01E-03	6.57E-02	3.46E-03	4.90E-02	9.44E-03	4.00E-03	6.59E-02
Nickel	0.00E+00	3.12E-04	5.94E-04	5.48E-05	4.76E-05	1.01E-03	3.58E-04	6.68E-04	8.36E-05	5.19E-05	1.16E-03
POM	0.00E+00	2.89E-03	1.03E-02	1.28E-03	5.69E-04	1.50E-02	2.28E-03	1.17E-02	6.16E-04	5.02E-04	1.51E-02
Propionaldehyde	0.00E+00	8.52E-03	1.95E-02	1.87E-01	3.50E-02	2.50E-01	7.87E-03	2.02E-02	7.36E-02	2.32E-02	1.25E-01
Styrene	0.00E+00	2.11E-02	1.20E-02	3.24E-02	6.32E-03	7.18E-02	2.48E-02	1.61E-02	9.92E-03	3.67E-03	5.45E-02
Toluene	0.00E+00	1.72E-01	6.97E-01	1.98E+00	2.74E-01	3.13E+00	1.22E-01	9.12E-01	6.46E-01	1.60E-01	1.84E+00
Xylenes	1.27E-01	8.53E-02	5.11E-01	1.18E+00	3.30E-01	2.23E+00	7.04E-02	6.82E-01	3.82E-01	1.80E-01	1.44E+00

Table 3.2-5 (cont'd). Mean Population Exposure Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030

Pollutant	background ($\mu\text{g m}^{-3}$)	2020 annual average exposure concentrations ($\mu\text{g m}^{-3}$)					2030 annual average exposure concentrations ($\mu\text{g m}^{-3}$)				
		major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	3.82E-02	1.92E-03	1.73E-02	1.70E-02	9.80E-03	8.50E-02	1.91E-03	1.73E-02	1.82E-02	1.12E-02	8.76E-02
2,2,4-Trimethylpentane	0.00E+00	9.70E-03	2.86E-02	2.27E-01	5.93E-02	3.24E-01	9.69E-03	2.86E-02	2.38E-01	6.45E-02	3.41E-01
Acetaldehyde	3.97E-01	2.65E-02	4.97E-02	2.49E-01	9.80E-02	8.24E-01	2.65E-02	4.97E-02	2.60E-01	1.07E-01	8.44E-01
Acrolein	0.00E+00	3.38E-03	2.16E-02	1.71E-02	1.62E-02	5.83E-02	3.38E-03	2.16E-02	1.85E-02	1.83E-02	6.18E-02
Benzene	2.98E-01	1.48E-02	1.71E-01	2.43E-01	8.55E-02	8.16E-01	1.48E-02	1.71E-01	2.57E-01	9.61E-02	8.40E-01
Chromium III	0.00E+00	4.66E-04	2.84E-04	1.04E-04	2.90E-05	8.84E-04	4.66E-04	2.84E-04	1.30E-04	3.03E-05	9.11E-04
Chromium VI	0.00E+00	6.36E-05	1.26E-04	2.33E-05	6.38E-06	2.20E-04	6.36E-05	1.26E-04	2.90E-05	6.67E-06	2.26E-04
Ethyl Benzene	0.00E+00	1.17E-02	1.07E-01	9.05E-02	4.16E-02	2.51E-01	1.17E-02	1.07E-01	9.49E-02	4.65E-02	2.60E-01
Formaldehyde	5.85E-01	4.90E-02	8.86E-02	1.62E-01	1.50E-01	1.04E+00	4.90E-02	8.86E-02	1.75E-01	1.65E-01	1.07E+00
Hexane	0.00E+00	5.54E-02	4.98E-01	1.02E-01	2.86E-02	6.83E-01	5.54E-02	4.98E-01	1.04E-01	3.20E-02	6.89E-01
Manganese	0.00E+00	1.07E-02	5.19E-02	8.78E-02	2.67E-02	1.77E-01	1.07E-02	5.19E-02	8.40E-02	3.02E-02	1.77E-01
MTBE	0.00E+00	1.42E-03	1.32E-03	1.29E-04	9.47E-06	2.88E-03	1.42E-03	1.32E-03	1.63E-04	1.08E-05	2.91E-03
Naphthalene	0.00E+00	3.88E-03	5.22E-02	9.38E-03	4.25E-03	6.97E-02	3.88E-03	5.22E-02	1.09E-02	4.84E-03	7.18E-02
Nickel	0.00E+00	3.89E-04	7.34E-04	9.38E-05	5.43E-05	1.27E-03	3.89E-04	7.34E-04	1.17E-04	5.96E-05	1.30E-03
POM	0.00E+00	2.53E-03	1.20E-02	6.34E-04	5.07E-04	1.57E-02	2.53E-03	1.20E-02	7.41E-04	5.64E-04	1.59E-02
Propionaldehyde	0.00E+00	7.91E-03	2.07E-02	6.26E-02	2.32E-02	1.14E-01	7.91E-03	2.07E-02	6.46E-02	2.52E-02	1.18E-01
Styrene	0.00E+00	2.83E-02	1.78E-02	9.15E-03	3.84E-03	5.91E-02	2.83E-02	1.78E-02	9.81E-03	4.36E-03	6.03E-02
Toluene	0.00E+00	1.36E-01	1.00E+00	5.75E-01	1.60E-01	1.87E+00	1.36E-01	1.00E+00	6.06E-01	1.77E-01	1.92E+00
Xylenes	1.27E-01	7.95E-02	7.51E-01	3.39E-01	1.82E-01	1.48E+00	7.94E-02	7.51E-01	3.56E-01	2.03E-01	1.52E+00

Table 3.2-6. National Average Cancer Risk Across Census Tracts for 1999, 2015, 2020, and 2030 by Pollutant

Pollutant	1999 average cancer risk					2015 annual average cancer risk					
	background	major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
Total Risk: All MSATs	4.35E-06	1.14E-06	5.19E-06	1.04E-05	2.05E-06	2.31E-05	1.20E-06	6.19E-06	3.92E-06	1.39E-06	1.71E-05
POM	0.00E+00	1.75E-07	1.01E-06	8.42E-08	3.70E-08	1.31E-06	1.40E-07	1.17E-06	3.97E-08	3.32E-08	1.38E-06
Nickel	0.00E+00	4.99E-08	9.50E-08	8.78E-09	7.61E-09	1.61E-07	5.73E-08	1.07E-07	1.34E-08	8.30E-09	1.86E-07
Naphthalene	0.00E+00	1.34E-07	1.38E-06	5.87E-07	1.36E-07	2.23E-06	1.18E-07	1.67E-06	3.21E-07	1.36E-07	2.24E-06
Formaldehyde	3.22E-09	1.91E-10	4.08E-10	3.18E-09	1.18E-09	8.18E-09	2.37E-10	4.64E-10	9.68E-10	8.22E-10	5.74E-09
Chromium VI	0.00E+00	5.26E-07	9.83E-07	1.63E-07	7.07E-08	1.74E-06	6.70E-07	1.34E-06	2.48E-07	7.50E-08	2.34E-06
Benzene	2.32E-06	1.47E-07	1.11E-06	5.91E-06	1.04E-06	1.05E-05	1.05E-07	1.28E-06	2.12E-06	6.46E-07	6.49E-06
Acetaldehyde	8.74E-07	5.51E-08	1.02E-07	1.67E-06	3.11E-07	3.01E-06	5.59E-08	1.07E-07	6.30E-07	2.15E-07	1.89E-06
1,3-Butadiene	1.15E-06	4.82E-08	5.15E-07	1.96E-06	4.52E-07	4.12E-06	5.33E-08	5.20E-07	5.52E-07	2.78E-07	2.57E-06

Pollutant	2020 annual average cancer risk					2030 annual average cancer risk					
	background	major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
Total Risk: All MSATs	4.35E-06	1.34E-06	6.57E-06	3.61E-06	1.44E-06	1.74E-05	1.34E-06	6.57E-06	3.91E-06	1.61E-06	1.78E-05
POM	0.00E+00	1.54E-07	1.20E-06	4.07E-08	3.37E-08	1.43E-06	1.54E-07	1.20E-06	4.75E-08	3.76E-08	1.44E-06
Nickel	0.00E+00	6.22E-08	1.18E-07	1.50E-08	8.69E-09	2.03E-07	6.22E-08	1.18E-07	1.88E-08	9.53E-09	2.08E-07
Naphthalene	0.00E+00	1.32E-07	1.77E-06	3.19E-07	1.44E-07	2.37E-06	1.32E-07	1.77E-06	3.70E-07	1.65E-07	2.44E-06
Formaldehyde	3.22E-09	2.70E-10	4.87E-10	8.90E-10	8.27E-10	5.73E-09	2.70E-10	4.87E-10	9.65E-10	9.06E-10	5.88E-09
Chromium VI	0.00E+00	7.63E-07	1.52E-06	2.79E-07	7.66E-08	2.64E-06	7.63E-07	1.52E-06	3.49E-07	8.00E-08	2.71E-06
Benzene	2.32E-06	1.15E-07	1.33E-06	1.90E-06	6.67E-07	6.36E-06	1.15E-07	1.33E-06	2.00E-06	7.49E-07	6.55E-06
Acetaldehyde	8.74E-07	5.84E-08	1.09E-07	5.48E-07	2.16E-07	1.81E-06	5.84E-08	1.09E-07	5.73E-07	2.36E-07	1.86E-06
1,3-Butadiene	1.15E-06	5.75E-08	5.19E-07	5.09E-07	2.94E-07	2.55E-06	5.74E-08	5.18E-07	5.47E-07	3.35E-07	2.63E-06

Figure 3.2-4. Contributions to Inhalation Cancer Risk from Air Toxics Emitted by Mobile Sources, 2020 (Not Including Diesel PM and Diesel Exhaust Organic Gases)

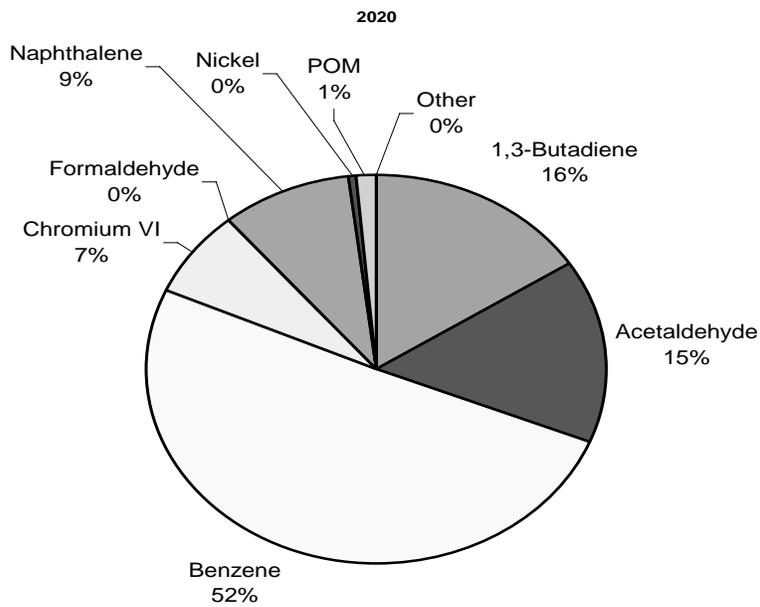
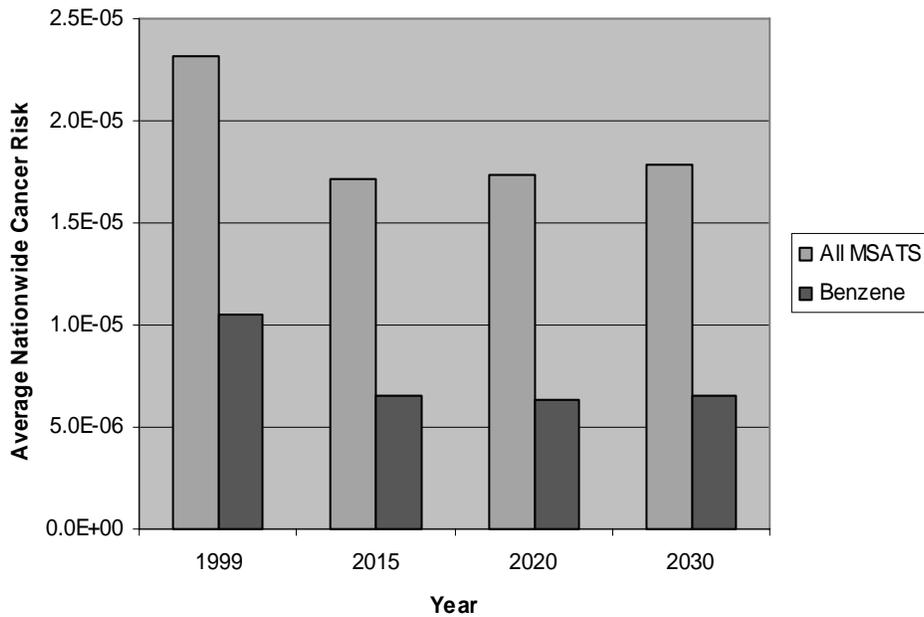
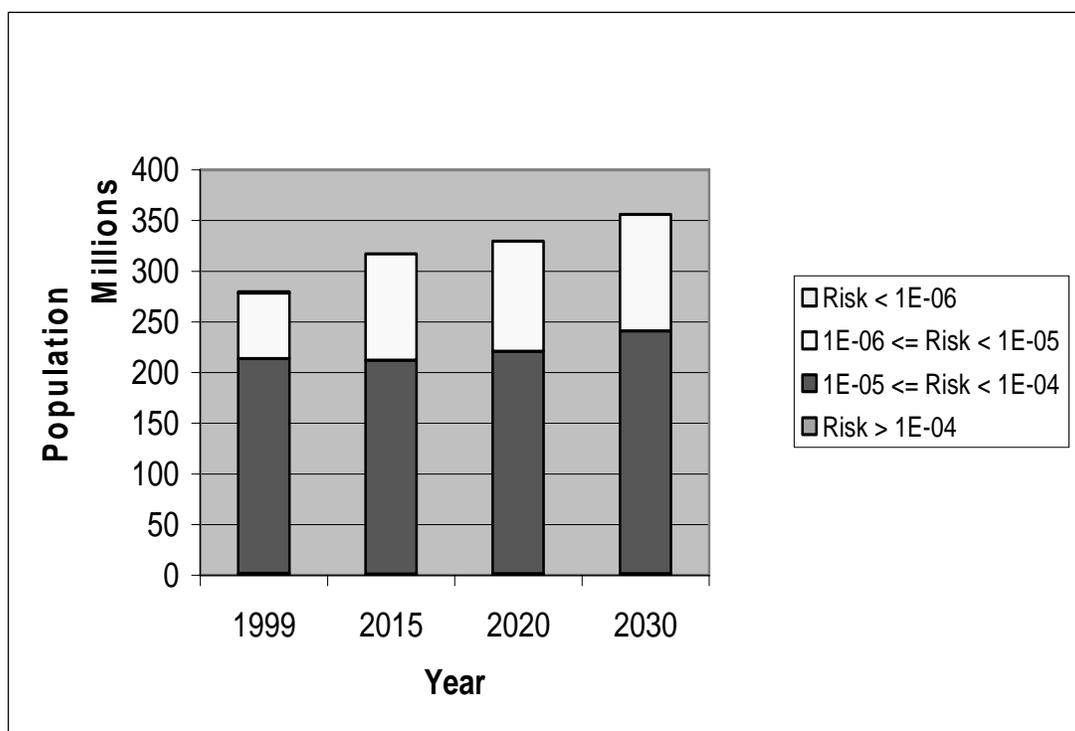


Figure 3.2-5. Average Nationwide Cancer Risk from Emissions of Mobile Source Air Toxics from both Mobile and Stationary Sources across Census Tracts, 1999 to 2030 (Not Including Diesel PM and Diesel Exhaust Organic Gases)



It should also be noted that because of population growth projected to occur in the United States, the number of Americans above cancer risk benchmarks is will increase. Figure 3.2-6 depicts the U. S. population at various risk benchmarks for mobile source air toxics in 1999, 2015, 2020, and 2030, using population projections from EPA’s BenMAP model, a tool the EPA uses to estimate benefits of air pollution control strategies, and average census tract exposures. (BenMAP was recently used for EPA’s Clean Air Interstate Air Quality Rule (CAIR).¹⁴⁶). These statistics do not include populations in Alaska and Hawaii. More details on the methodology used to project the U. S. population above various cancer risk benchmarks are provided in the document “National-Scale Modeling of Mobile Source Air Toxic Emissions, Air Quality, Exposure and Risk for the Mobile Source Air Toxics Rule.” From this figure it can be seen that, based on average census tract risks, the vast majority of the population experiences risks between one in a million (1×10^{-6}) and one in ten thousand (1×10^{-4}). However, the number of people experiencing risks above one in a hundred thousand (1×10^{-5}) increases from 214 million in 1999 to 240 million in 2030.

Figure 3.2-6. U. S. Population at Various Cancer Risk Benchmarks due to Exposure to Mobile Source Air Toxics, 1999 – 2030



Tables 3.2-7 and 3.2-8 summarize national average population hazard quotient for chronic non-cancer effects across census tracts for these years by pollutant, as well as the respiratory hazard index across pollutants. The respiratory system is the only target organ system where the hazard index exceeds one. Although the average respiratory hazard index for mobile source air toxics decreases by almost 50% between 1999 and 2030 (Figure 3.2-7), it is still over 3 in 2030, indicating a potential for adverse health effects. In addition, about 95% of this non-cancer risk is attributable to acrolein in all projection years. About 25% of primary

acrolein emissions are from mobile sources, and about 70% of ambient concentrations of acrolein (and about 75% of exposure) is attributable to mobile sources. The mobile source contribution to concentrations and exposure is largely attributable to the contribution from mobile source 1,3-butadiene, which is transformed to acrolein in the atmosphere. Moreover, projected growth in the U. S. population will increase the number of Americans with a respiratory hazard index for mobile source air toxics above one, from 250 million in 1999 to 273 million in 2030 (Figure 3.2-8).

Detailed summary tables presenting cancer risk, hazard quotients and hazard indices by State, and for reformulated and non-reformulated (i.e., conventional) gasoline areas, can be found in the docket for this rule, along with statistics on number of individuals above various cancer and non-cancer benchmarks, by source sector.

3.2.1.2.3 Distributions of Air Toxics Risk across the U. S.

Table 3.2-9 gives the distribution of nationwide average cancer risks for mobile source air toxics in 2020. Summary tables providing distributions for other years, as well as distributions by State and for reformulated and non-reformulated gasoline areas, can be found in the docket for this rule. Risk distributions are broader than the distributions of ambient concentrations in Table 3.2-2. For instance, while the 95th percentile benzene concentration is about twice the median value, the 95th percentile cancer risk is roughly six times the median risk. A key reason for this is the variability in activity patterns, concentrations among microenvironments, and commuting patterns. Figures 3.2-9 through 3.2-12 depict the geographic distributions of median county cancer risks in 2020 for all mobile source air toxics, benzene, acetaldehyde and 1,3-butadiene. These geographic distributions closely track distributions of ambient concentrations, with the highest risks in major population centers of the country where mobile source activity is the greatest. Relatively high benzene risks are also seen in areas of the country where fuel benzene levels are higher, such as the Pacific Northwest, parts of Alaska, and the upper Great Lakes region, since higher fuel benzene levels lead to higher benzene emissions and higher exposures.

Table 3.2-10 gives the distribution of nationwide average census tract hazard quotients for acrolein, and hazard indices for the respiratory target system in 2020. Patterns for other years are similar. The average respiratory hazard index at the 95th percentile is almost 40 times that at the 5th percentile, and about six times the median. Thus, some populations are experiencing much higher hazard indices than others. Figure 3.2-13 depicts the geographic distribution of median county respiratory hazard indices in 2020. The high hazard indices in Idaho are the result of high inventory estimates for wildfires and reflect a known error in the Idaho inventory for this source.

Table 3.2-7. National Average Population Hazard Quotient for Chronic Non-Cancer Effects across Census Tracts

Pollutant	Target System	background	1999 average Hazard Quotient					2015 average Hazard Quotient				
			major	area & other	onroad	Nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	Reproductive	1.91E-02	8.04E-04	8.58E-03	3.27E-02	7.54E-03	6.87E-02	8.88E-04	8.66E-03	9.19E-03	4.63E-03	4.29E-02
Acetaldehyde	Respiratory	4.41E-02	2.78E-03	5.15E-03	8.44E-02	1.57E-02	1.52E-01	2.82E-03	5.40E-03	3.18E-02	1.09E-02	9.54E-02
Acrolein	Respiratory	0.00E+00	1.36E-01	1.24E+00	3.36E+00	9.77E-01	5.72E+00	1.50E-01	1.12E+00	9.32E-01	7.71E-01	2.97E+00
Benzene	Immunological	9.93E-03	6.27E-04	4.72E-03	2.53E-02	4.42E-03	4.50E-02	4.49E-04	5.47E-03	9.04E-03	2.76E-03	2.78E-02
Chromium VI	Respiratory	0.00E+00	4.39E-04	8.19E-04	1.36E-04	5.89E-05	1.45E-03	5.58E-04	1.12E-03	2.07E-04	6.25E-05	1.95E-03
Ethyl Benzene	Developmental	0.00E+00	1.55E-05	7.52E-05	3.08E-04	7.13E-05	4.70E-04	1.05E-05	9.77E-05	1.03E-04	4.10E-05	2.52E-04
Formaldehyde	Respiratory	5.97E-02	3.55E-03	7.58E-03	5.91E-02	2.19E-02	1.52E-01	4.41E-03	8.60E-03	1.80E-02	1.52E-02	1.07E-01
Hexane	Neurological, Respiratory	0.00E+00	2.84E-04	1.94E-03	1.52E-03	2.32E-04	3.98E-03	2.52E-04	2.31E-03	6.11E-04	1.41E-04	3.31E-03
MTBE	Liver, Kidney, Ocular	0.00E+00	3.15E-06	1.79E-05	2.34E-04	2.65E-05	2.82E-04	3.19E-06	1.66E-05	3.80E-05	8.62E-06	6.65E-05
Manganese	Neurological	0.00E+00	2.14E-02	1.82E-02	1.43E-03	1.52E-04	4.13E-02	2.55E-02	2.40E-02	2.28E-03	1.77E-04	5.20E-02
Naphthalene	Respiratory	0.00E+00	1.31E-03	1.35E-02	5.76E-03	1.34E-03	2.19E-02	1.15E-03	1.63E-02	3.15E-03	1.33E-03	2.20E-02
Nickel	Respiratory, Immunological	0.00E+00	4.79E-03	9.14E-03	8.44E-04	7.32E-04	1.55E-02	5.50E-03	1.03E-02	1.29E-03	7.98E-04	1.79E-02
Styrene	Neurological	0.00E+00	2.11E-05	1.20E-05	3.24E-05	6.32E-06	7.18E-05	2.48E-05	1.61E-05	9.92E-06	3.67E-06	5.45E-05
Toluene	Respiratory, Neurological	0.00E+00	4.29E-04	1.74E-03	4.96E-03	6.85E-04	7.81E-03	3.05E-04	2.28E-03	1.61E-03	4.00E-04	4.60E-03
Xylenes	Neurological	1.27E-03	8.53E-04	5.11E-03	1.18E-02	3.30E-03	2.23E-02	7.04E-04	6.82E-03	3.82E-03	1.80E-03	1.44E-02

Table 3.2-7 (cont'd). National Average Population Hazard Quotient for Chronic Non-Cancer Effects across Census Tracts

Pollutant	Target System	background	2020 average Hazard Quotient					2030 average Hazard Quotient				
			major	area & other	onroad	Nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	Reproductive	1.91E-02	9.58E-04	8.64E-03	8.48E-03	4.90E-03	4.25E-02	9.57E-04	8.64E-03	9.12E-03	5.59E-03	4.38E-02
Acetaldehyde	Respiratory	4.41E-02	2.95E-03	5.52E-03	2.77E-02	1.09E-02	9.15E-02	2.95E-03	5.52E-03	2.89E-02	1.19E-02	9.38E-02
Acrolein	Respiratory	0.00E+00	1.69E-01	1.08E+00	8.57E-01	8.09E-01	2.91E+00	1.69E-01	1.08E+00	9.25E-01	9.16E-01	3.09E+00
Benzene	Immunological	9.93E-03	4.93E-04	5.70E-03	8.12E-03	2.85E-03	2.72E-02	4.93E-04	5.69E-03	8.57E-03	3.20E-03	2.80E-02
Chromium VI	Respiratory	0.00E+00	6.36E-04	1.26E-03	2.33E-04	6.38E-05	2.20E-03	6.36E-04	1.26E-03	2.90E-04	6.67E-05	2.26E-03
Ethyl Benzene	Developmental	0.00E+00	1.17E-05	1.07E-04	9.05E-05	4.16E-05	2.51E-04	1.17E-05	1.07E-04	9.49E-05	4.65E-05	2.60E-04
Formaldehyde	Respiratory	5.97E-02	5.00E-03	9.04E-03	1.65E-02	1.53E-02	1.06E-01	5.00E-03	9.04E-03	1.79E-02	1.68E-02	1.09E-01
Hexane	Neurological, Respiratory	0.00E+00	2.77E-04	2.49E-03	5.08E-04	1.43E-04	3.42E-03	2.77E-04	2.49E-03	5.20E-04	1.60E-04	3.44E-03
MTBE	Liver, Kidney, Ocular	0.00E+00	3.58E-06	1.73E-05	2.93E-05	8.91E-06	5.91E-05	3.58E-06	1.73E-05	2.80E-05	1.01E-05	5.89E-05
Manganese	Neurological	0.00E+00	2.84E-02	2.63E-02	2.58E-03	1.89E-04	5.75E-02	2.84E-02	2.63E-02	3.26E-03	2.15E-04	5.82E-02
Naphthalene	Respiratory	0.00E+00	1.29E-03	1.74E-02	3.13E-03	1.42E-03	2.32E-02	1.29E-03	1.74E-02	3.63E-03	1.61E-03	2.39E-02
Nickel	Respiratory, Immunological	0.00E+00	5.98E-03	1.13E-02	1.44E-03	8.36E-04	1.96E-02	5.98E-03	1.13E-02	1.80E-03	9.17E-04	2.00E-02
Styrene	Neurological	0.00E+00	2.83E-05	1.78E-05	9.15E-06	3.84E-06	5.91E-05	2.83E-05	1.78E-05	9.81E-06	4.36E-06	6.03E-05
Toluene	Respiratory, Neurological	0.00E+00	3.40E-04	2.50E-03	1.44E-03	4.00E-04	4.68E-03	3.40E-04	2.50E-03	1.52E-03	4.42E-04	4.80E-03
Xylenes	Neurological	1.27E-03	7.95E-04	7.51E-03	3.39E-03	1.82E-03	1.48E-02	7.94E-04	7.51E-03	3.56E-03	2.03E-03	1.52E-02

Table 3.2-8. National Respiratory Hazard Index for Chronic Non-Cancer Effects across Census Tracts

Respiratory System Average Hazard Index						
Year	background	major	area & other	onroad	nonroad	total (including background)
1999	1.04E-01	1.49E-01	1.28E+00	3.52E+00	1.02E+00	6.07E+00
2015	1.04E-01	1.65E-01	1.16E+00	9.88E-01	7.99E-01	3.22E+00
2020	1.04E-01	1.85E-01	1.13E+00	9.08E-01	8.38E-01	3.17E+00
2030	1.04E-01	1.85E-01	1.13E+00	9.79E-01	9.48E-01	3.35E+00

Figure 3.2-7. Average Respiratory Hazard Index for U.S. Population (Aggregate of Hazard Quotients for Individual Pollutants)

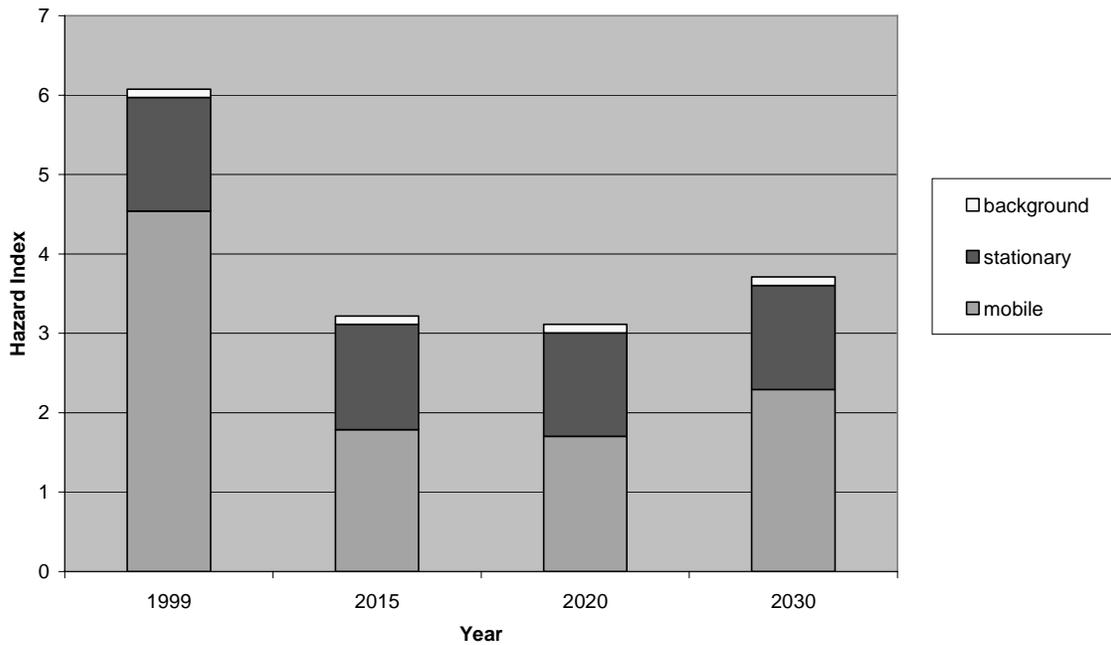


Figure 3.2-8. U. S. Population at Various Non-Cancer Hazard Benchmarks due to Exposure to Mobile Source Air Toxics, 1999 – 2030

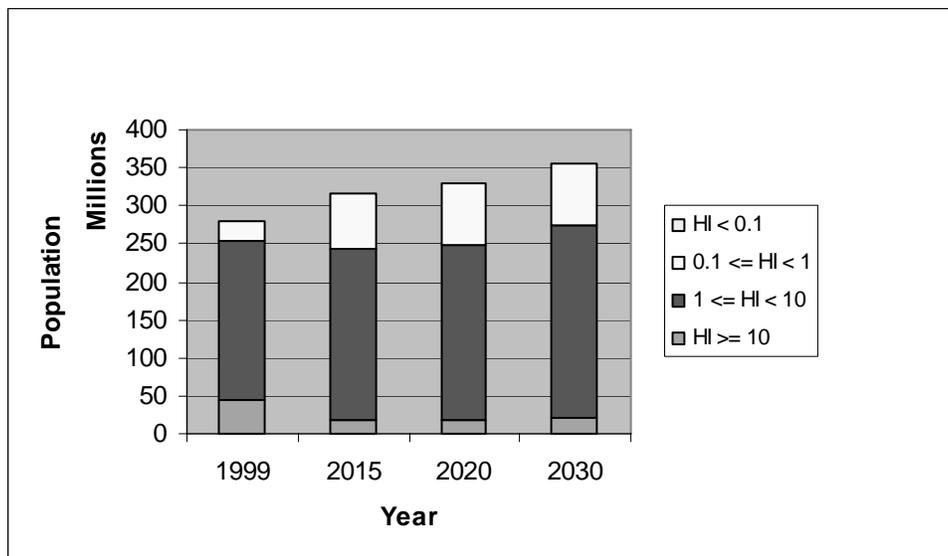


Table 3.2-9. Distribution of Average Census Tract Cancer Risks for Mobile Source Air Toxics in 2020

Pollutant	2020 risk distribution						
	5th percentile	10th percentile	25th percentile	Median	75th percentile	90th percentile	95th percentile
Total Risk: All HAPs	3.59E-06	4.61E-06	8.04E-06	1.34E-05	2.02E-05	3.34E-05	4.39E-05
POM	7.48E-08	1.40E-07	7.38E-07	1.99E-06	3.05E-06	4.48E-06	7.47E-06
Nickel	9.32E-07	9.98E-07	1.20E-06	1.60E-06	2.13E-06	2.94E-06	3.64E-06
Naphthalene	2.08E-06	2.54E-06	3.87E-06	5.61E-06	7.63E-06	1.07E-05	1.35E-05
Formaldehyde	1.65E-08	4.33E-08	1.82E-07	6.73E-07	1.83E-06	5.32E-06	8.58E-06
Chromium VI	1.75E-09	2.29E-09	3.53E-09	5.12E-09	6.80E-09	9.63E-09	1.23E-08
Benzene	1.02E-07	2.09E-07	6.20E-07	1.44E-06	2.79E-06	5.38E-06	8.47E-06
Acetaldehyde	1.27E-09	3.38E-09	1.35E-08	5.04E-08	1.53E-07	3.62E-07	6.15E-07
1,3-Butadiene	9.64E-08	1.71E-07	3.32E-07	6.81E-07	1.15E-06	1.93E-06	3.11E-06

Figure 3.2-9. 2020 County Median Cancer Risk for All Mobile Source Air Toxics

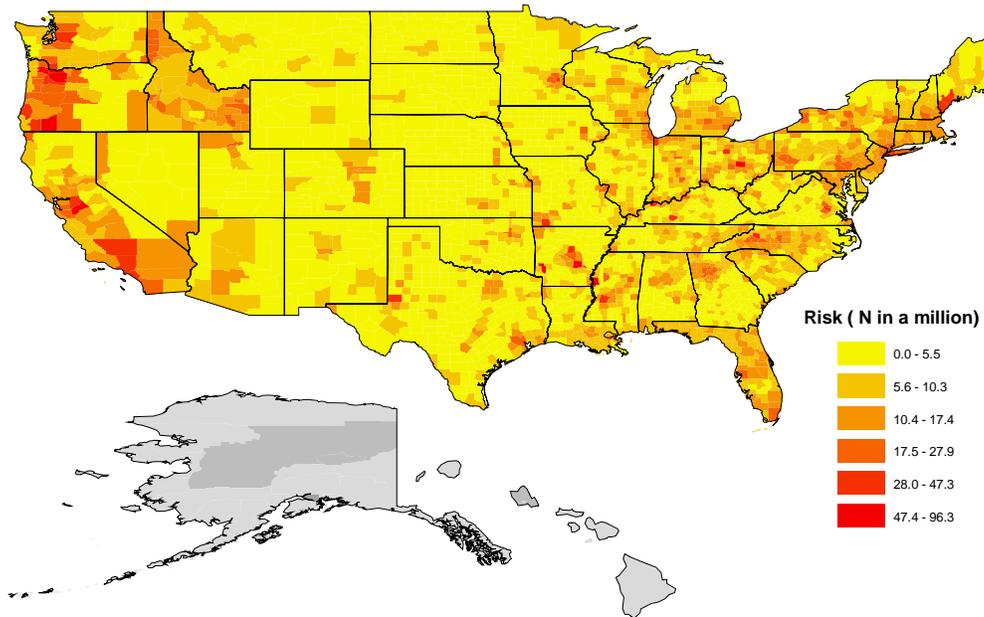


Figure 3.2-10. 2020 County Median Cancer Risk for Benzene

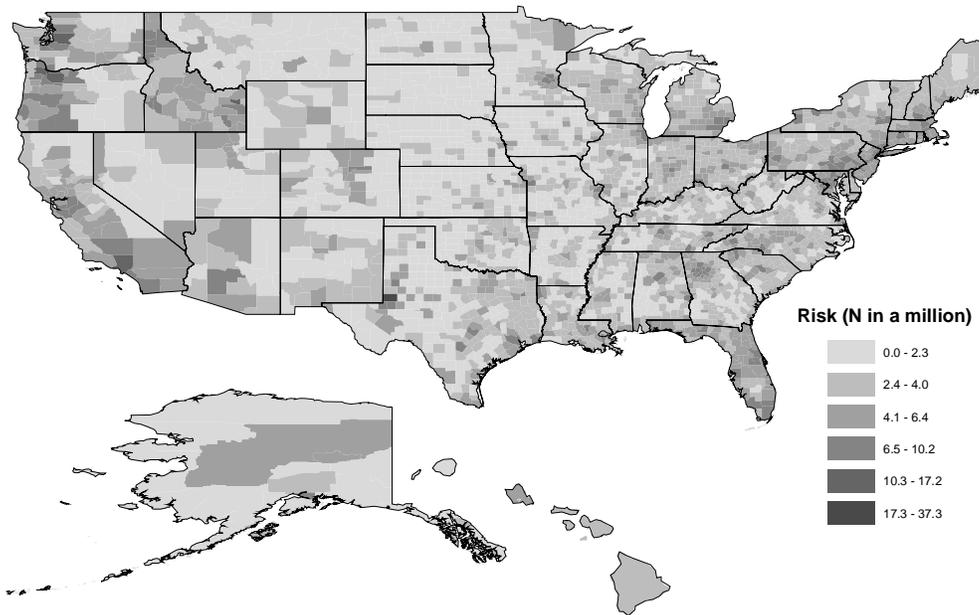


Figure 3.2-11. 2020 County Median Cancer Risk for Acetaldehyde

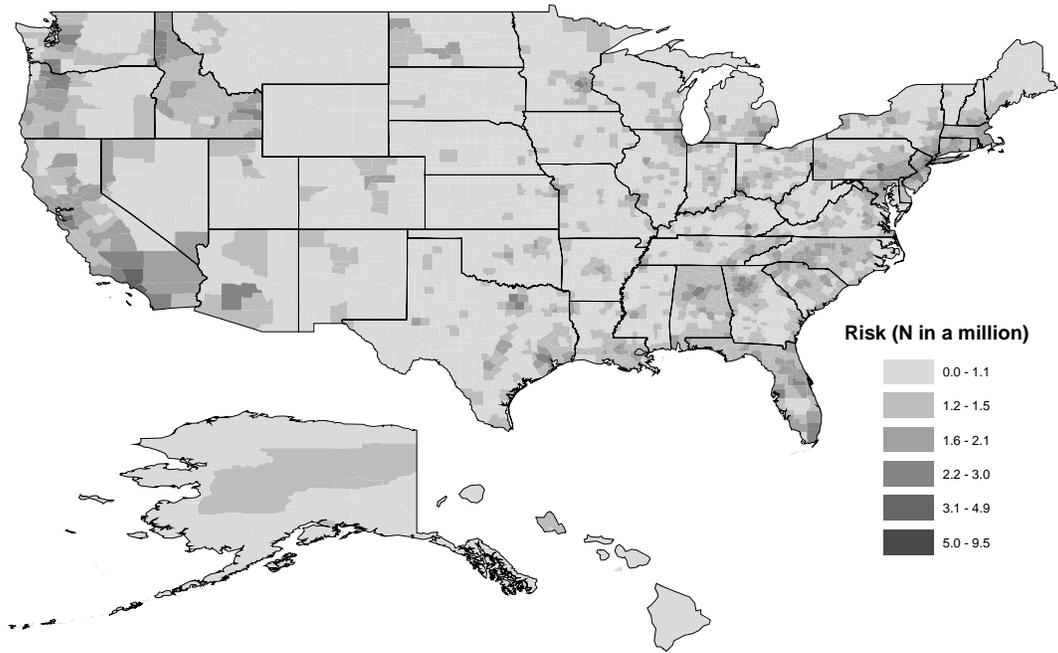


Figure 3.2-12. 2020 County Median Cancer Risk for 1,3-Butadiene

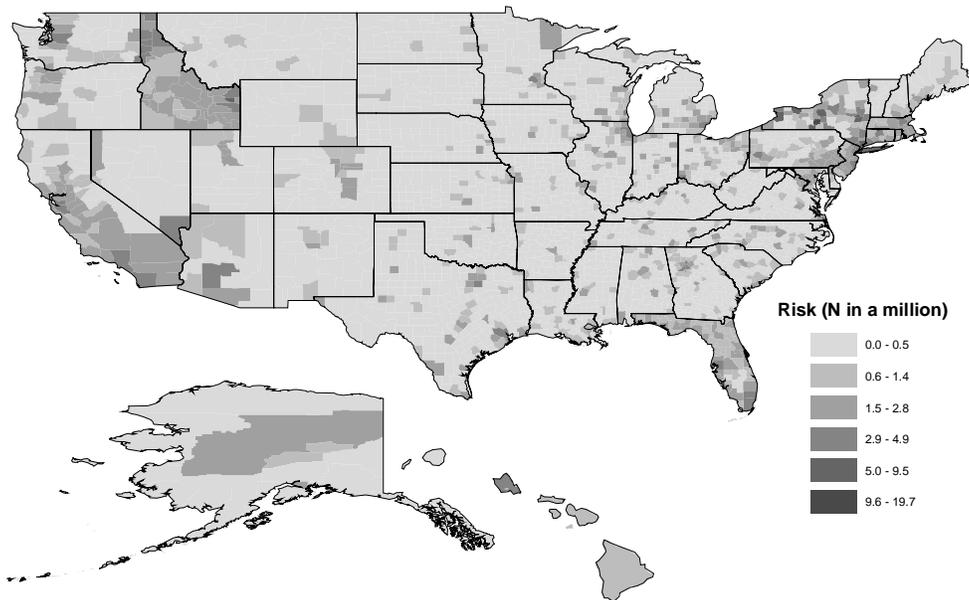
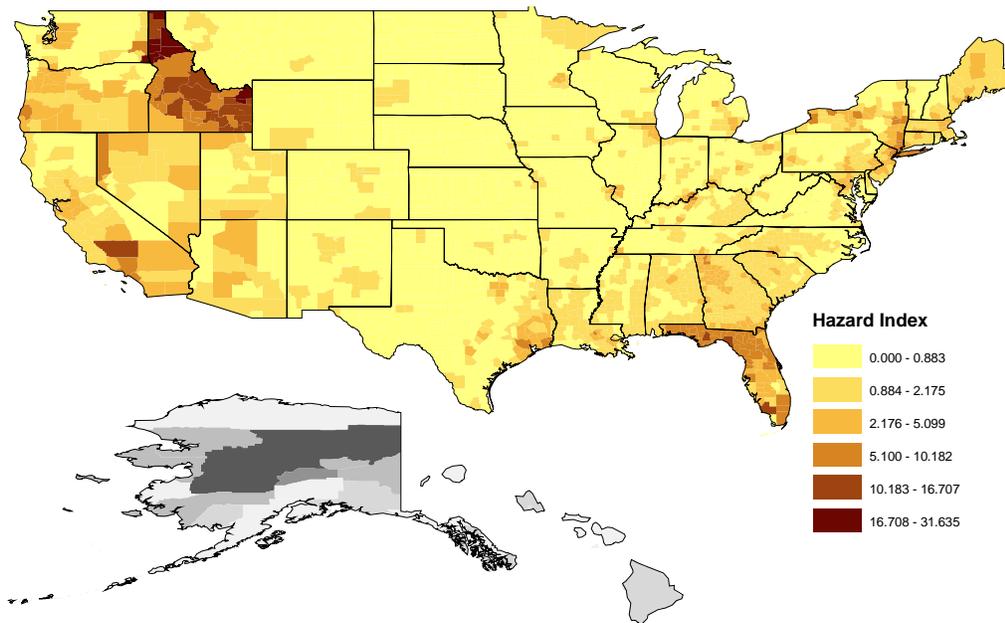


Table 3.2-10. Distribution of Average Census Tract Hazard Quotients/Hazard Indices for Mobile Source Air Toxics (from both Mobile and Stationary Sources) in 2020

Pollutant	2020 average Hazard Quotient or Hazard Index						95th percentile
	5th percentile	10th percentile	25th percentile	Median	75th percentile	90th percentile	
Acrolein	2.13E-01	3.65E-01	8.08E-01	1.69E+00	3.36E+00	6.99E+00	1.11E+01
Respiratory System	3.06E-01	4.79E-01	9.61E-01	1.91E+00	3.67E+00	7.39E+00	1.17E+01

Figure 3.2-13. 2020 County Median Non-Cancer Hazard Index Respiratory Mobile Source Air Toxics



3.2.1.2.4 Impacts of Proposed Fuel Benzene Controls on Average Inhalation Cancer Risk

The fuel benzene standard proposed in this rule will substantially reduce inhalation cancer risk from exposure to benzene emitted by mobile sources across the United States. Table 3.2-11 shows that in 2015, 2020, and 2030, the highway vehicle contribution to benzene cancer risk will be reduced on average 8 to 9 percent across the U.S., the nonroad equipment contribution will be reduced about 7 percent, and the area source contribution about 4 percent. Reductions in conventional gasoline areas (i.e., areas not subject to reformulated gasoline) are almost 13 percent. In States with high fuel benzene levels, such as Minnesota and Washington, the risk reduction exceeds 17 percent (Table 3.2-12). Figure 3.2-14 depicts the impact on the mobile source contribution to nationwide average population cancer risk from benzene in 2020. Figure 3.2-15 presents the distribution of percent reductions in average benzene cancer risk for U. S. counties with the proposed control in 2020. Patterns are similar for other years. Summary tables providing exposure and risk data by State, as well as maps of benzene cancer risks with fuel controls and percent reductions with controls, can be found in the docket for the rule. Similar data are also available for 1,3-butadiene, formaldehyde and acetaldehyde, even though cancer risks were not significantly affected. Data are also available for noncancer risks, which are also not significantly affected.

It should be noted that the estimated total reductions are significant underestimates, since we could not account for further reductions in emissions from transport, i.e., background sources. In Section 3.2.1.4, we provide a quantitative estimate of the expected reductions in background concentrations in future years.

Table 3.2-11. Contributions of Source Sectors to Nationwide Average Benzene Cancer Risk, with and without Proposed Fuel Benzene Standard, 2015, 2020, and 2030

	2015 Average Risks					2020 Average Risks					2030 Average Risks				
	major	area & other	highway vehicles	nonroad	total (including background)	major	area & other	highway vehicles	nonroad	total (including background)	major	area & other	highway vehicles	nonroad	total (including background)
Reference	1.05E-07	1.28E-06	2.12E-06	6.46E-07	6.49E-06	1.15E-07	1.33E-06	1.90E-06	6.67E-07	6.36E-06	1.15E-07	1.33E-06	2.00E-06	7.49E-07	6.55E-06
Control	1.04E-07	1.23E-06	1.92E-06	6.00E-07	6.21E-06	1.14E-07	1.28E-06	1.73E-06	6.19E-07	6.10E-06	1.14E-07	1.28E-06	1.84E-06	6.95E-07	6.28E-06
% Difference	0.8	3.6	9.0	7.0	4.3	0.8	3.7	8.7	7.1	4.1	0.8	3.7	8.3	4.1	
Average Nationwide Difference in Risk -- Non RFG Areas															
Reference	7.24E-08	1.05E-06	1.66E-06	4.57E-07	5.40E-06	8.06E-08	1.08E-06	1.49E-06	4.74E-07	5.29E-06	8.05E-08	1.08E-06	1.54E-06	5.35E-07	5.40E-06
Control	7.19E-08	1.00E-06	1.44E-06	4.02E-07	5.08E-06	7.99E-08	1.04E-06	1.30E-06	4.15E-07	5.00E-06	7.99E-08	1.03E-06	1.34E-06	4.68E-07	5.09E-06
% Difference	0.8	4.2	13.6	12.2	6.0	0.8	4.2	13.1	12.4	5.6	0.8	4.2	12.9	12.5	5.7
Average Nationwide Difference in Risk -- RFG Areas															
Reference	1.64E-07	1.70E-06	2.93E-06	9.84E-07	8.46E-06	1.78E-07	1.79E-06	2.63E-06	1.01E-06	8.28E-06	1.78E-07	1.79E-06	2.84E-06	1.13E-06	8.62E-06
Control	1.63E-07	1.65E-06	2.80E-06	9.57E-07	8.25E-06	1.77E-07	1.73E-06	2.52E-06	9.85E-07	8.09E-06	1.76E-07	1.73E-06	2.73E-06	1.10E-06	8.42E-06
% Difference	0.7	3.0	4.4	2.7	2.4	0.8	3.1	4.1	2.8	2.3	0.8	3.1	3.9	2.8	2.3

Table 3.2-12. States with Highest Reductions in Average Benzene Cancer Risk Resulting from Mobile Source Emissions, 2020

State	Average Risk – Reference Case	Average Risk – 0.62% Benzene Standard	Percent Difference
Alaska	1.22×10^{-6}	8.36×10^{-7}	-31%
Washington	3.21×10^{-6}	2.64×10^{-6}	-18%
Minnesota	2.60×10^{-6}	2.14×10^{-6}	-18%
New Mexico	1.45×10^{-6}	1.19×10^{-6}	-18%
Oregon	2.97×10^{-6}	2.47×10^{-6}	-17%

Figure 3.2-14. Contribution to Nationwide Average Population Cancer Risk in 2020 Resulting from Proposed Fuel Benzene Controls

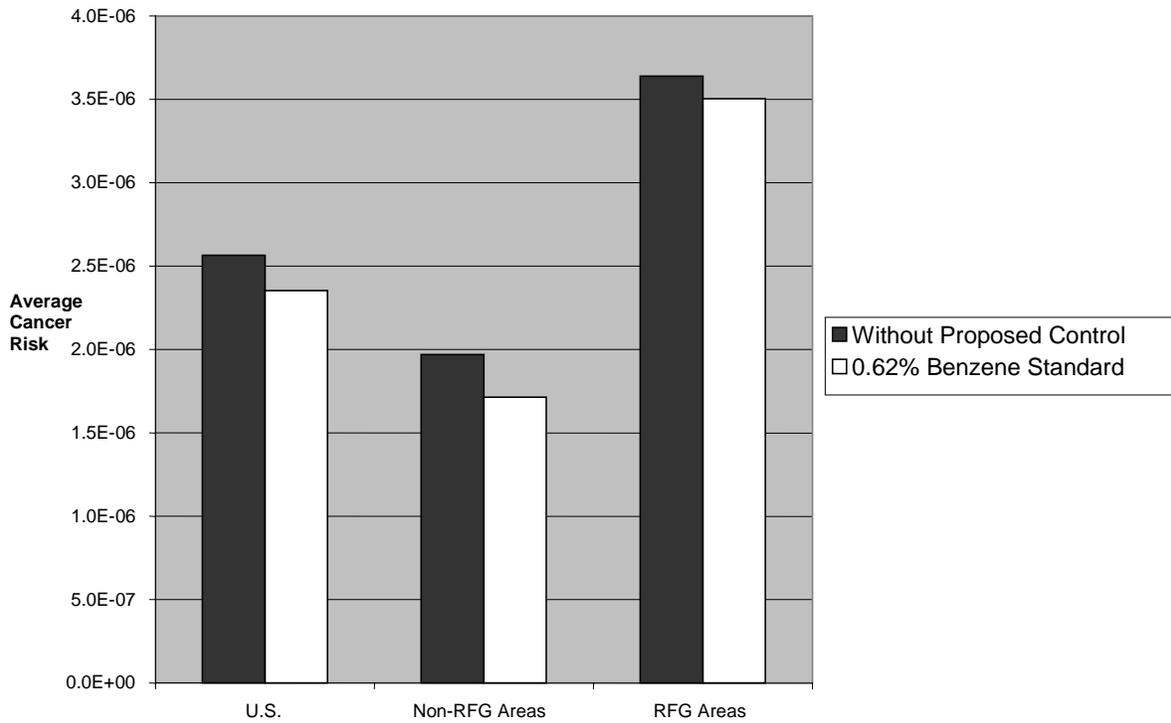
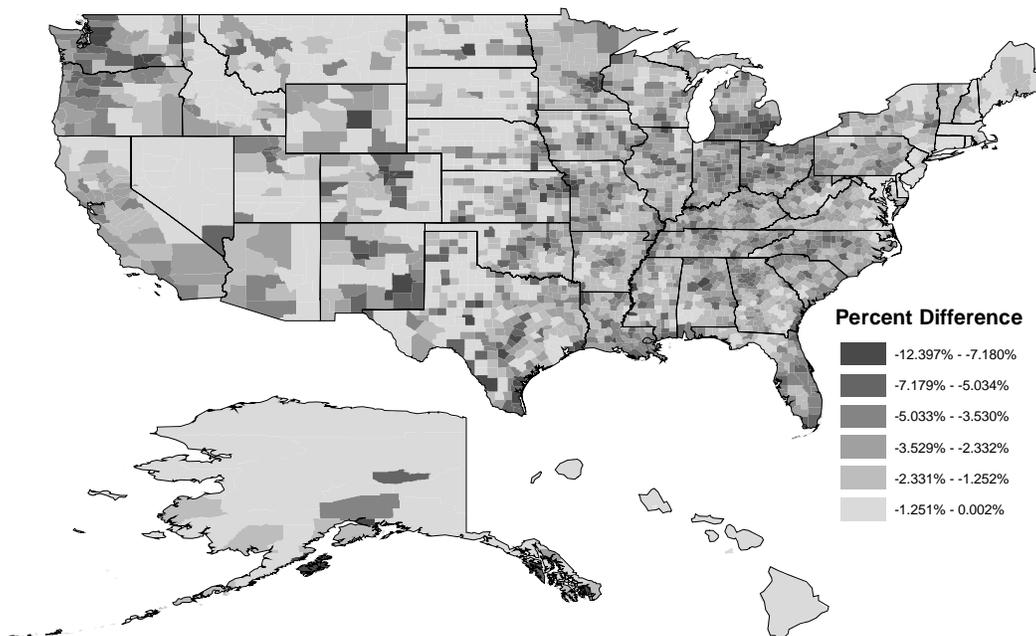


Figure 3.2-15. Distribution of Percent Reductions in Median Benzene Cancer Risk, 2020, for U.S. Counties with the Proposed Control



As a result of the proposed fuel benzene control, the number of people above the 1 in 100,000 cancer risk level due to exposure to all mobile source air toxics from all sources will decrease by over 3 million in 2020 and by about 3.5 million in 2030. The number of people above the 1 in 100,000 increased cancer risk level from exposure to benzene from all sources decreases by over 4 million in 2020 and 5 million in 2030 (Table 3.2-13).

Table 3.2-13. Decrease in Number of People with Inhalation Exposure above the 1 in 100,000 Cancer Risk Level due to Inhalation Exposure from Ambient Sources, with Proposed Fuel Benzene Control

Year	Benzene	All Mobile Source Air Toxics
2015	4,976,000	3,226,000
2020	4,150,000	3,077,000
2030	5,253,000	3,477,000

The proposed standard will have little impact on the number of people above various respiratory hazard index levels, since this potential non-cancer risk is dominated by exposure to acrolein. Population statistics on number of individuals above various cancer and non-cancer benchmarks, by source sector, with fuel benzene control are available in the docket for this rule.

3.2.1.3 Impacts of Near Roadway Microenvironment on Modeled Exposures to Benzene

3.2.1.3.1 Assessment Methods

In HAPEM5, if only a single outdoor concentration is provided for each census tract, as is typical, this concentration is assumed to uniformly apply to the entire census tract. EPA has recently developed a new version of the model, HAPEM6, which refines the model to account for the spatial variability of outdoor concentrations within a tract due to higher outdoor concentrations of onroad mobile source pollutants at locations near major roadways.^C More information on development of the model can be found in technical reports in the docket of this proposed rulemaking. The new version of HAPEM more accurately reflects the average and range of exposure concentrations within each census tract by accounting for some of the spatial variability in the outdoor concentrations within the tract, and by extension some of the spatial variability in indoor concentrations within the tract. At this time, HAPEM6 only accounts for near-roadway effects for benzene.

The new version of HAPEM was developed using the following three steps.

1) *Estimating the fraction of the population living near major roadways in each census tract by demographic group.*

First, the “zone of influence” of transportation facilities needed to be determined – that is, the width of the area around major roads within which concentrations of benzene are elevated. Second, population data of sufficient geographic specificity was needed. Using geographic information systems, we conducted a study of the populations in three states, Colorado, Georgia, and New York¹⁴⁷. In Colorado, 22% live within 75 meters of a major road, while an additional 33% live between 75 and 200 meters of major roads. In Georgia, the respective percentages are 17% living within 75 meters and an additional 24% living between 75 and 200 meters. In New York, the percentages are 31% and 36%.

This was done by overlaying extracts from the ESRI StreetMap US roadway geographic database on a geographic database of US Census blocks.

2) *Estimating the increase near major roadways of air toxic pollutant concentrations from onroad motor vehicle emissions relative to concentrations at other outdoor locations.*

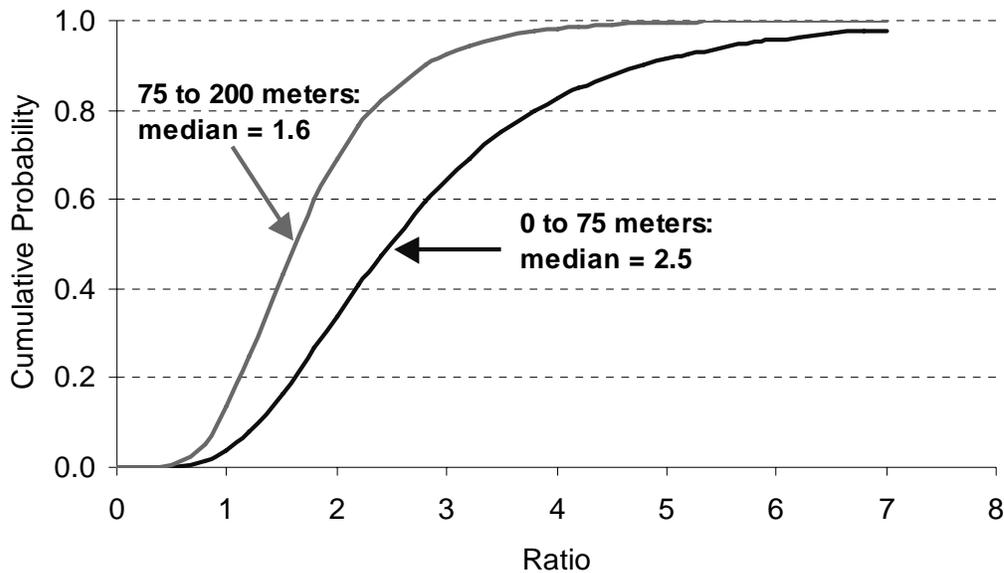
In this step, data on spatial gradients of pollutants near roads from several sources were analyzed.^{148, 149, 150, 151, 152, 153, 154} Data were analyzed for their suitability to estimate concentration distributions within 75 meters of a major roadway, or between 75 meters and 200 meters from such a road. All the data sources analyzed were from monitoring studies, except for one, which was a modeling study in Portland using the CALPUFF dispersion model to estimate concentrations at receptors located at census block centroids (Cohen et al., 2005). The monitoring data were consistent with the spatial gradients characterized using CALPUFF, but had limitations which precluded their use in quantifying concentration distributions. Among the limitations were that measured concentrations did not span various distances near the road needed to develop a model, monitors were all downwind, or measurements were taken at limited

^C The term “major roadway” will be used to describe a “Limited Access Highway”, “Highway”, “Major Road” or “Ramp”, as defined by the Census Feature Class Codes (CFCC).

times of year, making it difficult to extrapolate to annual averages. Modeling output from Portland included receptor locations at many distances from major roadways and calculated annual averages of benzene concentration in ambient air at those receptors. Thus the Portland modeling data was used to develop concentration ratios via regression analysis.

One way of comparing the concentrations for near-road and other locations is to examine the distribution of ratios between concentrations at multiple distances from a major road. Figure 3.2-16 presents a distribution of the concentration ratios between locations “near” a major roadway (within 75 meters) and locations “far” from a roadway (>200 meters distant). Also shown is a distribution of concentration ratios between locations at “intermediate” locations (between 75 meters and 200 meters) and those “far” from a roadway. These data were derived from the Portland modeling.

Figure 3.2-16. Distribution of Ratios of Near Roadway to Remote Concentrations



3) Modification of the HAPEM model

HAPEM6 models exposure for a simulated, demographically representative population within each census tract. For each simulated individual, HAPEM6 randomly selects for each home tract indoor microenvironment whether it is within D1 (75) meters of a major roadway, from D1 to D2 (75 to 200) meters from a major roadway, or greater than D2 meters from a major roadway, according to the database developed in the first step described above.

If the simulated person is a commuter, HAPEM6 randomly selects for each work tract indoor microenvironment whether it is within D1 meters of a major roadway, from D1 to D2 meters from a major roadway, or greater than D2 meters from a major roadway, according to the fractions of the populations living near major roadways in Step 1.

For each microenvironment, HAPEM6 selects a near-roadway ratio multiplier distribution for the proper distance according to the probabilities specified in the Step 2; and selects a ratio multiplier from that distribution.

HAPEM6 calculates the ambient concentration for locations more than D2 meters from a major roadway according to the equation:

$$A_{\text{far}} * C_{\text{far}} + A_{\text{D1-D2}} * C_{\text{D1-D2}} = C_{\text{ASPEN}} \quad \text{or}$$

$$A_{\text{far}} * C_{\text{far}} + A_{\text{D1-D2}} * R_{\text{D1-D2}} * C_{\text{far}} = C_{\text{ASPEN}} \quad \text{or}$$

$$C_{\text{far}} = C_{\text{ASPEN}} / (A_{\text{far}} + A_{\text{D1-D2}} * R_{\text{D1-D2}})$$

Where:

C_{ASPEN} is the ASPEN concentration prediction for the tract

$C_{\text{D1-D2}}$ is the ambient concentration in the area between D1 and D2 meters from a major roadway (i.e., ASPEN concentration estimate x mean of the ratio multiplier distribution)

C_{far} is ambient concentration in the area more than D2 meters from a major roadway

$A_{\text{D1-D2}}$ is the fraction of the tract area that is between D1 and D2 meters from a major roadway,

A_{far} is the fraction of the tract area that is more than D2 meters from a major roadway, and

$R_{\text{D1-D2}}$ is the near roadway ratio multiplier selected for the D1 meters to D2 meters distance range.

The implicit assumption for this step is that the ASPEN estimate for the average census tract concentration represents the spatial average over the tract excluding the area within D1 meters of a major roadway. This is a reasonable assumption given the way that the ASPEN concentration estimate is generated.

The ASPEN estimate for the census tract average concentration is an aggregate of the contributions from all sources within 50 km of the tract. For sources located outside of the tract the concentration contribution is estimated at the geographic centroid of the tract and assumed to be uniform throughout the tract. For sources within the tract, which we expect to be the dominant contributors, the concentration contribution is calculated as a weighted average of the concentrations at all the modeling receptors that fall within the tract.

HAPEM6 calculates the "ambient" concentrations in at different distances from major roads by applying the relevant ambient concentration ratio. If located within 75 meters of a major road, the concentration ratio for that area is applied to the C_{far} concentration, shown above. Indoor microenvironmental concentrations are calculated based on this ambient concentration. Likewise if located between 75 and 200 meters of a major road, the concentration ratio for that area is used to calculate ambient and indoor concentrations at that point. If located more that 200 meters away from a major road, the C_{far} concentration is used for ambient air and for calculating indoor microenvironmental concentrations.

3.2.1.3.2 Results

The revised model was run for three geographic areas representing different parts of the

country. For these initial runs, benzene was the only pollutant modeled. ASPEN output for calendar year 1999 were used as inputs. We studied the states of Colorado, Georgia, and New York. These areas are intended to represent different geographies, development patterns, and housing densities.

Within a given census tract, the HAPEM model predicts 30 lifetime exposure concentrations depicting the variation in potential individual exposures within the tract. Such variation can result from differences in human activity patterns and as is the case for the simulation with HAPEM6, proximity of populations to roadways. Table 3.2-14 depicts the results of a comparison between HAPEM5 (does not include near roadway residents) and HAPEM6 (includes near roadway residents). The Table shows the distribution of individual exposure concentrations both within a given tract as well as in tracts across the state. When applied to each of these states, the greatest change in exposures resulting from the use of HAPEM6 occurred for the individuals at the upper end of the exposure distribution within each tract. Further, this effect was most pronounced at the tracts with the highest exposures within a state. In summary, the models show that including the effects of residence locations can result in exposures to some individuals that are up to 50% higher than those predicted by HAPEM5 (as was applied in the 1999 NATA).

Table 3.2-14. Comparison Predicted Exposure Concentrations from HAPEM5 and HAPEM6 Results for Georgia, Colorado, and New York

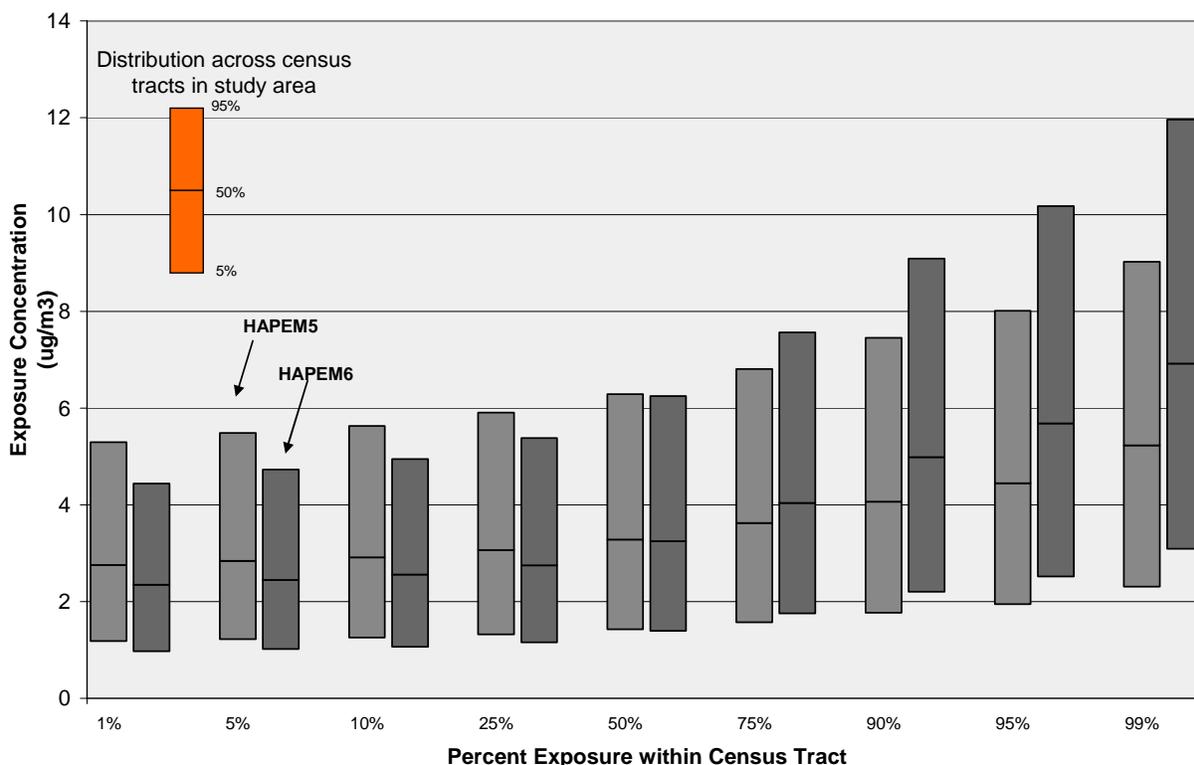
Modeled State	Model Version	Percentile of Tracts Across State	Percentile of Exposure within Census Tract (ug/m3)								
			1%	5%	10%	25%	50%	75%	90%	95%	99%
Georgia	HAPEM6	5% tract	0.54	0.57	0.60	0.64	0.73	0.92	1.15	1.31	1.55
		50% tract	0.83	0.87	0.89	0.95	1.06	1.28	1.57	1.76	2.07
		95% tract	1.29	1.34	1.39	1.53	1.86	2.45	3.11	3.59	4.43
	HAPEM5	5% tract	0.63	0.66	0.68	0.71	0.77	0.85	0.95	1.00	1.11
		50% tract	0.91	0.94	0.96	1.01	1.08	1.19	1.32	1.40	1.57
		95% tract	1.65	1.70	1.75	1.84	2.01	2.19	2.40	2.59	2.76
Colorado	HAPEM6	5% tract	0.49	0.52	0.53	0.58	0.67	0.80	1.01	1.10	1.27
		50% tract	0.71	0.74	0.76	0.81	0.92	1.08	1.30	1.42	1.61
		95% tract	0.66	0.71	0.76	0.84	0.99	1.24	1.50	1.70	2.18
	HAPEM5	5% tract	0.56	0.58	0.59	0.63	0.69	0.77	0.87	0.92	1.04
		50% tract	0.76	0.78	0.81	0.85	0.92	1.02	1.13	1.20	1.34
		95% tract	0.89	0.93	0.96	1.01	1.09	1.17	1.24	1.29	1.40
New York	HAPEM6	5% tract	0.97	1.02	1.07	1.16	1.39	1.76	2.20	2.52	3.09
		50% tract	1.37	1.43	1.49	1.59	1.85	2.27	2.79	3.16	3.82
		95% tract	2.10	2.28	2.39	2.63	3.00	3.53	4.10	4.49	5.05
	HAPEM5	5% tract	1.19	1.22	1.26	1.32	1.42	1.57	1.77	1.95	2.31
		50% tract	1.57	1.62	1.66	1.74	1.86	2.05	2.29	2.50	2.92
		95% tract	2.54	2.65	2.71	2.85	3.00	3.18	3.39	3.56	3.79

The results indicate that by accounting for within-tract variability in concentrations, HAPEM6 substantially increases overall variability in exposure to benzene. Demonstrating

these trends, the results of this modeling exercise for the state of New York are shown in Figure 3.2-17. In the graph, the horizontal axis shows percentiles of exposure within census tracts, while the range of each bar represents the 50th, 5th, and 95th percentiles of exposure concentration across census tracts within the state.

Overall, these study results indicate that proximity to major roads can significantly increase personal exposure for populations living near major roads. These models will be extended to a national scale for the final rulemaking.

Figure 3.2-17. Changes in Predicted Benzene Exposure Patterns between HAPEM5 (no near-roadway adjustment) and HAPEM6 (with near-roadway adjustment) for New York



3.2.1.4 Impacts of Attached Garage Emissions on National-scale Levels of Benzene Inhalation Cancer Risk

As discussed in Section 3.1.3.3, a major source of exposure to benzene is from penetration of emissions from sources in attached garages into the home. These emissions can result from the storage of vehicles, other engines and equipment, fuel (gasoline in gas cans), solvents, or other cleaning products. EPA analyses indicate that nationwide predicted inhalation risks from benzene could more than double if this source of exposure were accounted for. The standards proposed in this rule will dramatically reduce this source of exposure by reducing cold start emissions of vehicles, reducing the amount of benzene in gasoline, and decrease emissions of gasoline stored in portable fuel containers.

3.2.1.5 Strengths and Limitations

Air quality, exposure, and risk were assessed using the best available suite of tools for national-scale analysis of air toxics. In addition, the modeling done to support this rule was consistent with NATA for 1999, making direct comparisons of results possible. The first NATA, done for calendar year 1996, was reviewed by EPA's Science Advisory Board, and the analyses done for 1999 incorporate several changes in response to comments made in this peer review. Among the improvements were:

- Improved emission inventory with detailed characterization of source categories within the onroad and nonroad source sectors and more speciated data for some pollutant groups (POM) within particular source categories.
- Speciation of chromium to hexavalent form based on emission sources rather than a single number applied across all sources
- Improved surrogates for spatial allocation in EMS-HAP.
- Improved estimation of "background" concentrations for many pollutants. These background levels were previously uniform across the country. Now, for many pollutants, background levels are based on recent monitor data and spatially vary depending on county population density.¹⁵⁵
- Improved version of HAPEM, which includes more recent census data, commuting algorithms and better characterization of exposure distributions through improvements in modeling long-term activity patterns and variability in concentration levels in microenvironments.

The SAB expressed their belief that due to the limitations inherent in the analysis, the 1996 NATA should not be used to support regulatory action. However, the use of the improved analyses does provide useful insight on the nature of the mobile source air toxics problem and the possible public health improvements associated with this rule.

In addition to the strengths listed above, there are limitations due to uncertainty. The inventory uncertainties discussed in Chapter 2. There are a number of additional significant uncertainties associated with the air quality, exposure and risk modeling. These uncertainties result from a number of parameters including: development of county-level estimates from broader geographic data (i.e., state, regional or national), surrogates used to allocate emissions to census tracts, parameters used to characterize photochemical processes, long range transport, terrain effects, deposition rates, human activity pattern parameters, assumptions about relationships between ambient levels in different microenvironments, and dose-response parameters. The modeling also has certain key limitations: results are most accurate for large geographic areas and cannot be used to identify "hot spots," such as the near road microenvironment, exposure modeling does not fully reflect variation among individuals, non-inhalation exposure pathways and indoor sources are not accounted for; and for some pollutants, the ASPEN dispersion model may underestimate concentrations. Also, the 1999 NATA does not include default adjustments for early life exposures recently recommended in the Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens.¹⁵⁶ Incorporation of such adjustments would lead to higher estimates of lifetime risk. EPA will

determine as part of the IRIS assessment process which substances meet the criteria for making adjustments, and future assessments will reflect them.

As part of the 1999 NATA, EPA compared ASPEN-modeled concentrations with available, but geographically limited, ambient air quality monitoring data for 1999. For each monitor-pollutant combination, EPA compared the annual average concentration estimated by the ASPEN model at the exact geographical coordinates of the monitor location with the annual average monitored value to get a point-to-point comparison between the model and monitor concentrations. The agreement between model and monitor values for benzene was very good, with a median model to monitor ratio of 0.95, and 74% of sites within a factor of 2. Agreement for acetaldehyde was almost as good as benzene, but data suggest that ASPEN could be underpredicting for other mobile source air toxics (see Table 3.2-15).

More detailed discussion of modeling limitations and uncertainties can be found on the 1999 NATA website.

Table 3.2-15. Agreement of 1999 Model and Monitors by Pollutant on a Point-to-Point Basis Pollutants listed were Monitored in at least 30 Sites and in a Broad Geographical Area (Several States)

Pollutant	No. of Sites	Median of Ratios	Within Factor of 2	Within 30%	Underestimated
Acetaldehyde	68	0.92	74%	44%	56%
Benzene	115	0.95	72%	43%	52%
Formaldehyde	68	0.64	60%	28%	76%
Chromium	42	0.29	26%	5%	95%
Manganese	34	0.4	44%	15%	91%
Nickel	40	0.53	48%	18%	75%

In addition to the limitations and uncertainties associated with modeling the 1999 base year, there are additional ones in the projection year modeling. For instance, the modeling is not accounting for impacts of demographic shifts that are likely to occur in the future. A key limitation is using 1999 “background” levels to account for mid-range to long-range transport. However, since background is related to emissions far away from receptors, these levels should decrease as those emissions decrease. We performed a sensitivity analysis for benzene, formaldehyde, acetaldehyde and 1,3-butadiene to evaluate the potential bias introduced by this assumption. We used background estimates scaled by the change in the inventory for a future year relative to 1999. The scaling factors applied to the background level for an individual county were based on emissions for counties within 300 kilometers of that county’s centroid. Our analysis indicated that using a scaled background reduced benzene concentrations about 15% on average across the U. S in 2015, 2020, and 2030. Table 3.2-16 compares national average total concentrations using 1999 versus scaled backgrounds. More details are provided in the technical document previously referenced.

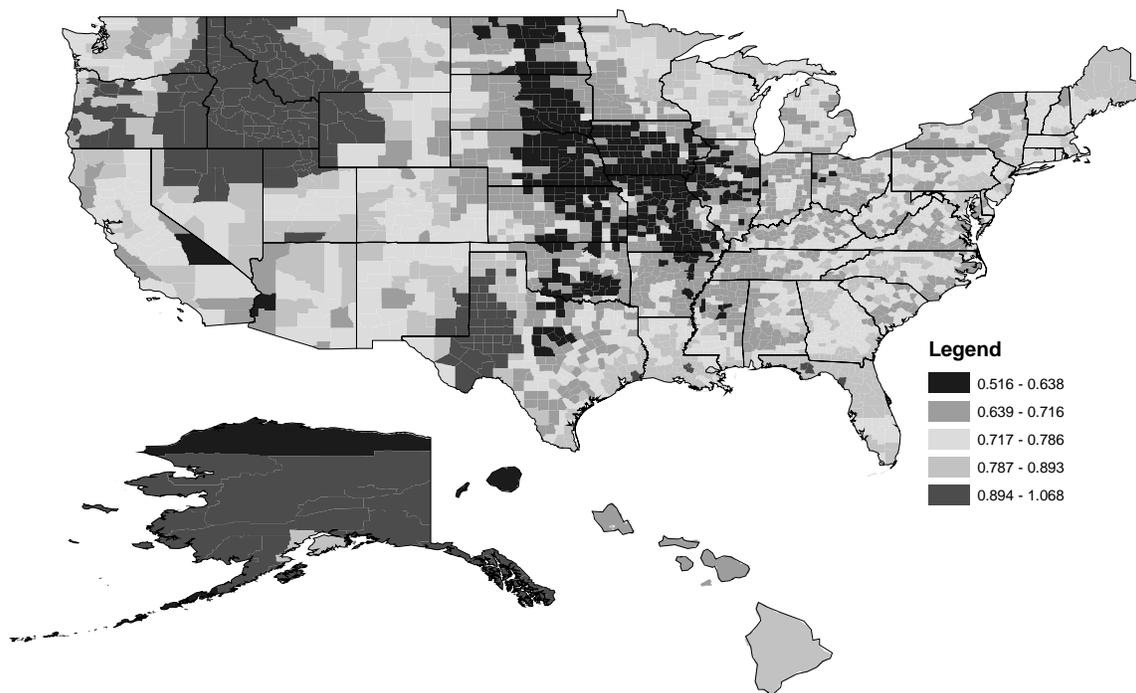
Table 3.2-16. National Average Total Concentrations (All Sources and Background) for 2015, 2020, and 2030 using both the 1999 Background and the Scaled Backgrounds

HAP	Total Concentrations ($\mu\text{g m}^{-3}$) using 1999 Background			Total Concentrations ($\mu\text{g m}^{-3}$) using Scaled Concentrations		
	2015	2020	2030	2015	2020	2030
1,3-Butadiene	9.81×10^{-2}	9.77×10^{-2}	1.00×10^{-1}	7.57×10^{-2}	7.50×10^{-2}	7.86×10^{-2}
Acetaldehyde	9.66×10^{-1}	9.36×10^{-1}	9.56×10^{-1}	7.77×10^{-1}	7.47×10^{-1}	7.78×10^{-1}
Benzene	9.13×10^{-1}	9.02×10^{-1}	9.24×10^{-1}	7.57×10^{-1}	7.40×10^{-1}	7.71×10^{-1}
Formaldehyde	1.22	1.22	1.25	9.56×10^{-1}	9.68×10^{-1}	1.01
Xylenes	1.55	1.61	1.65	1.50	1.56	1.60

The largest impacts were in the Midwest as can be seen in Figure 3.2-18, which depicts ratios of the ASPEN-modeled ambient benzene concentrations with an adjusted background versus the 1999 background in 2020. Data tables with results of the sensitivity comparison by U. S. County, along with maps of pollutant concentrations with and without an adjusted background can be found in the docket for the rule.

While accounting for impacts of emission reductions on background levels would reduce estimated population risks, it would increase estimated reductions in risk of control strategies in a given year, since background levels would be reduced. Also, if the modeling accounted for: (1) near road impacts; (2) impacts of emissions from vehicles, equipment and fuels in attached garages; (3) increased risks from early lifetime exposures; and (4) properly estimated cold start emissions, estimated risks and risk reductions from fuel benzene control would be larger.

Figure 3.2-18. Ratios of Benzene Concentrations with and without an Adjusted Background, 2020



3.2.2 Local-Scale Modeling

Modeling at the national or regional scale, such the modeling done for the NATA National-Scale Assessment described in Section 3.2.1, is designed to identify and prioritize air toxics, emission source types and locations which are of greatest potential concern in terms of contributing to population risk. Such assessments also help elucidate patterns of exposure and risk across broad geographic areas, and can help characterize trends in air toxics risk and potential impacts of controls at a broad geographic scale, as demonstrated above. However, more localized assessments are needed to characterize and compare risks at local levels, and identify potential “hotspots.”

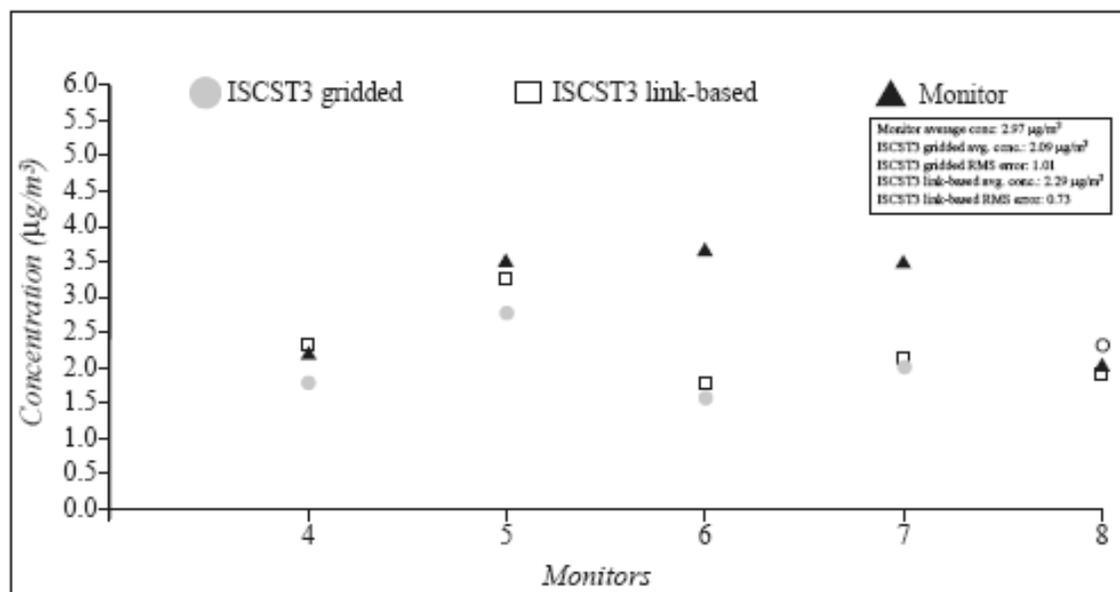
National or regional-scale assessments typically rely on a “top down” approach to estimate emissions. Under a “top down” approach, emissions are estimated at the county level, typically starting from more aggregated information (e.g., state or national level) on activity. Spatial surrogates are then used to allocate emissions to grid cells or census tracts for modeling. Use of more local data can greatly improve the characterization of the magnitude and distribution of air toxic emissions. Air quality modeling can also be conducted with better spatial resolution than is computationally feasible in a regional or national-scale assessment. As a result, spatial gradients of air toxic concentrations and locations where the highest risks are likely to occur can be more accurately identified.

Local-scale modeling is typically done using steady-state plume dispersion models, such as the Integrated Source Complex (ISC) Model, the newly promulgated AERMOD (AMS/EPA Regulatory Model), or non-steady-state puff models such as CALPUFF. These models have a limited ability to simulate chemical reactions in the atmosphere. As discussed in Section 3.2.1, grid-based models, such as CMAQ, which better simulate chemical processes, do not yet have the spatial resolution of dispersion models. Significant advances are being made, however, in combining features of grid-based models and plume/puff models. These advances are described in a recent paper.¹⁵⁷ A case study of diesel exhaust particulate matter in Wilmington, CA was recently conducting employing some of these advances.¹⁵⁸ The researchers combined Gaussian and regional photochemical grid models. They found that local data, when modeled, provided a much more refined picture of the magnitude and distribution of possible community “hot spots” than more traditional, regional data, which rely on more default assumptions. An evaluation of the approach determined that spatial allocation and emission rates contribute most to uncertainty in model results, and this uncertainty could be substantially reduced through the collection and integration of site specific information about the location of emission sources, and the activity and emission rates of key sources affecting model concentrations. They conclude that for neighborhood assessments, incorporating site-specific data can lead to improvement in modeled estimates of concentrations, especially where site-specific data are lacking in regulatory databases.

The Wilmington study discussed above also allocated motor vehicle emissions to individual road “links,” rather than using spatial surrogates to allocate county level vehicle emissions to grid cells. In using spatial surrogates to allocate emissions, high local

concentrations may not be captured for environments near major roadways, which are often clustered in urban centers. One local-scale assessment done in the Minneapolis-St. Paul area of Minnesota, using such an inventory with the ISC model, found that the model tended to overpredict at low monitored benzene concentrations and underpredict at high monitored concentrations.¹⁵⁹ Local-scale modeling using activity data for individual road links can better characterize distributions of concentrations, and differentiate between locations near roadways and those further away, as observed in the following studies.

Researchers at US EPA developed a methodology which utilized a Geographic Information System (GIS) to allocate benzene emissions in Houston to major road segments in an urban area and model the segments as elongated area sources.¹⁶⁰ The Industrial Source Complex Short Term (ISCST) dispersion model used both gridded and link-based emissions to evaluate the effect of improved spatial allocation of emissions on ambient modeled benzene concentrations. Allocating onroad mobile emissions to road segments improved the agreement between modeled concentrations when compared with monitor observations, and also resulted in higher estimated concentrations in the urban center where the density of neighborhood streets is greater and the largest amount of traffic found. The calculated annual average benzene model concentrations at monitor sites are compared to the observed annual average concentrations in Figure 3.2-18. Most of the gridded model emissions show lower benzene concentrations than both the link-based and observed monitor concentrations. Allocating the onroad mobile emissions to road segments resulted in an increase in the average benzene concentration, resulting in values that more closely match concentrations reported by monitors.

Figure 3.2-19. Model to Monitor Comparisons of Houston Benzene Concentrations

Recent air quality modeling in Portland, OR using the CALPUFF dispersion model assigned emissions to specific roadway links.¹⁶¹ The resulting data were used to develop a regression model to approximate the CALPUFF predicted concentrations, determine the impacts of roadway proximity on ambient concentration of three hazardous air pollutants (1,3-butadiene, benzene, and diesel PM), and to estimate the zone of influence around roadways. Concentrations were modeled at several distances from major roadways (0-50, 5-200, 200-400, and > 400 meters). For benzene, the resulting average concentrations were 1.29, 0.64, 0.40, and 0.12 µg/m³, respectively, illustrating the steep concentration gradient along roadways. There was a zone of influence between 200 and 400 meters, with concentrations falling to urban background levels beyond this distance. The overall mean motor vehicle benzene concentration modeled in Portland was about 0.21 µg/m³, with concentrations increasing to 1.29 µg/m³ at model receptor sites within 50 meters of a road. The results indicate that in order to capture localized impacts of hazardous air pollutants in a dispersion model, there is a need to include individual roadway links.

A recent review of local-scale modeling studies concluded that:¹⁶²

- 1) Significant variations in air toxic concentrations occurred across the cities, with highest concentrations occurring near the highest emitting sources, illustrating the need for modeling on a local scale.
- 2) Increasing the receptor density near high emission sources changes the location of maximum concentrations, illustrating the concentration gradients that can occur near high emission sources and the importance of receptor placement and density for model performance.
- 3) Allocating on-road mobile emissions to road segments improved the agreement between modeled concentrations when compared with the observations, and also resulted in higher estimated concentrations in the urban center.
- 4) It is important to refine the national emissions inventory for input into local air quality

model applications.

In another US EPA study, researchers provide a comparison of “top down” and “bottom up” approaches to developing a motor vehicle emissions inventory for one urban area, Philadelphia, in calendar year 1999.¹⁶³ Under the “top down” approach, emissions were estimated at the county level, typically starting from more aggregated information. Data on vehicle miles traveled (VMT) in the metropolitan statistical area were allocated to counties using population information. Default national model inputs (e.g. fleet characteristics, vehicle speeds) rather than local data were also used. The “bottom up” approach utilizes vehicle activity data from a travel demand model (TDM), and this “bottom up” approach estimates emission rates using more local input data to better estimate levels and spatial distribution of onroad motor vehicle emissions. TDM data can include information on the spatial distribution of vehicle activity, speeds along those roads (which can have a large impact on emissions), and the distribution of the VMT among vehicle classes for different speed ranges. These data can be used to more accurately estimate the magnitude of toxic emissions at the local scale and where they occur. Both the spatial distribution of emissions and the total county emissions in Philadelphia differed significantly between the top-down and the bottom-up methodologies as shown in Table 3.2-17.

Table 3.2-17. Comparison of Annual 1999 Benzene Emissions from Two Approaches in Philadelphia Area Counties

County	Local (TDM) Based	National (NEI)	Percent Difference
Camden	165	210	-27%
Delaware	162	160	1%
Gloucester	110	104	6%
Montgomery	333	209	59%
Philadelphia	255	467	-45%
Total	1,025	1,150	-12%

In the case of Philadelphia, using local registration distribution data resulted in significantly lower air toxics emission factors and resultant emissions.

Local-scale modeling could also be improved by using local data on nonroad equipment activity for lawn and garden, recreational, construction and other sectors. EPA’s county-level inventories used in NATA and other modeling are developed using activity allocated from the national or state level using surrogates.

The use of more spatially refined emission inventories, in conjunction with other refined air quality modeling techniques, improve the performance of air quality models. They also enable better characterization of the magnitude and distribution of air toxic emissions, exposure and risk in urban areas, including risks associated with locations heavily impacted by mobile sources.

In conclusion, local scale modeling studies indicated higher concentrations of air toxics than predicted by National scale analysis, particularly in near-source microenvironments such as near roads. Thus, National scale analyses such as 1999 NATA are likely underestimating high end exposures and risks.

3.3 Ozone

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

3.3.1 Science of Ozone Formation

Ground-level ozone, the main ingredient in smog, is formed by the reaction of VOCs and nitrogen oxides (NO_x) in the atmosphere in the presence of heat and sunlight. These pollutants, often referred to as ozone precursors, are emitted by many types of pollution sources such as highway and nonroad motor vehicles, gas cans, power plants, chemical plants, refineries, makers of consumer and commercial products, industrial facilities, and smaller “area” sources. VOCs can also be emitted by natural sources such as vegetation.

The science of ozone formation, transport, and accumulation is complex.¹⁶⁴ Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions, many of which are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up and result in more ozone than typically would occur on a single high-temperature day. Further complicating matters, ozone also can be transported into an area from pollution sources found hundreds of miles upwind, resulting in elevated ozone levels even in areas with low VOC or NO_x emissions. As a result, spatial and temporal differences in VOC and NO_x emissions and weather patterns contribute to daily, seasonal, and yearly differences in ozone concentrations across different locations.

The highest levels of ozone are produced when both VOC and NO_x emissions are present in significant quantities on clear summer days. Relatively small amounts of NO_x enable ozone to form rapidly when VOC levels are relatively high, but ozone production is quickly limited by removal of the NO_x. Under these conditions NO_x reductions are highly effective in reducing ozone while VOC reductions have little effect. Such conditions are called “NO_x-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 NO_x-limited.

When NO_x levels are relatively high and VOC levels relatively low, NO_x forms inorganic

nitrates (i.e., particles) but relatively little ozone. Such conditions are called “VOC-limited.” Under these conditions, VOC reductions are effective in reducing ozone, but NO_x reductions can actually increase local ozone under certain circumstances. Even in VOC-limited urban areas, NO_x reductions are not expected to increase ozone levels if the NO_x reductions are sufficiently large.

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

Ozone concentrations in an area also can be lowered by the reaction of nitric oxide with ozone, forming nitrogen dioxide (NO₂); as the air moves downwind and the cycle continues, the NO₂ forms additional ozone. The importance of this reaction depends, in part, on the relative concentrations of NO_x, VOC, and ozone, all of which change with time and location.

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

3.3.2 Health Effects of Ozone

Exposure to ambient ozone contributes to a wide range of adverse health effects. We are relying on the data and conclusions in the 1996 ozone criteria document (CD) and ozone staff paper, which reflect EPA’s analysis of policy-relevant science from the ozone CD, regarding the health effects associated with ozone.^{165,166} In August 2005, the EPA released the second external review draft of a new ozone CD which is scheduled to be released in final form in February 2006.¹⁶⁷ The new ozone criteria document summarizes the findings of the 1996 ozone CD and critically assesses relevant new scientific information that has emerged in the past decade. In all, the new epidemiological studies that have become available since the 1996 ozone CD continue to demonstrate the harmful effects of ozone on public health, and the need to attain and maintain the ozone NAAQS.

Ozone-related health effects include lung function decrements, respiratory symptoms, aggravation of asthma, increased hospital and emergency room visits, increased medication usage, inflammation of lung tissues, as well as a variety of other respiratory effects. People who are particularly at risk for high ozone exposures include healthy children and adults who are active outdoors. Susceptible subgroups include children, people with respiratory disease, such as asthma, and people with unusual sensitivity to ozone.^{168,169,170,171,172}

Based on a large number of scientific studies, EPA has identified several key health effects caused when people are exposed to levels of ozone found today in many areas of the country. Short-term (1 to 3 hours) and prolonged exposures (6 to 8 hours) to higher ambient

ozone concentrations have been linked to lung function decrements, respiratory symptoms, increased hospital admissions and emergency room visits for respiratory problems.^{173,174,175,176,177,178,179,180,181,182,183,184,185,186} Repeated exposure to ozone can make people more susceptible to lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.^{187,188,189,190,191,192,193,194,195} Repeated exposure to ozone can also cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure.^{196,197,198,199,200,201}

Children and adults who are outdoors and active during the summer months, such as construction workers and other outdoor workers, are among those most at risk of elevated ozone exposures.^{202,203,204,205,206} It may be that children and outdoor workers are most at risk from ozone exposure because they typically are active outside, working, playing and exercising, during the summer when ozone levels are highest.^{207,208,209,210} 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 pain on deep inspiration and cough, when exposed to relatively low ozone levels during prolonged periods of moderate exertion.²¹¹ For example, summer camp studies in the Eastern United States and Southeastern Canada have reported significant reductions in lung function in children who are active outdoors.^{212,213,214,215,216,217,218,219,220} Further, children are more at risk of experiencing health effects from ozone exposure than adults because their respiratory systems are still developing.

There has been new research that suggests additional serious health effects beyond those that had been known when the 1996 ozone CD was published. Since then, over 1,700 new ozone-related health and welfare studies have been published in peer-reviewed journals.²²¹ Many of these studies have investigated the impact of ozone exposure on such health effects as changes in lung structure and biochemistry, inflammation of the lungs, exacerbation and causation of asthma, respiratory illness-related school absence, hospital and emergency room visits for asthma and other respiratory causes, and premature mortality. EPA is currently in the process of evaluating these and other studies as part of the ongoing review of the criteria document and NAAQS for ozone. Key new health information falls into four general areas: development of new-onset asthma, hospital admissions for young children, school absence rate, and premature mortality. Examples of new studies in these areas are briefly discussed below.

Aggravation of existing asthma resulting from short-term ambient ozone exposure was reported prior to the 1997 ozone NAAQS revision and has been observed in studies published since then.^{222,223,224,225,226} More recent studies now suggest a relationship between long-term ambient ozone concentrations and the incidence of new-onset asthma. In particular, such a relationship in adult males (but not in females) was reported by McDonnell et al. (1999).²²⁷ Subsequently, McConnell et al. (2002) reported that incidence of new diagnoses of asthma in children is associated with heavy exercise in communities with high ambient ozone concentrations (i.e., mean 8-hour concentration of 59.6 ppb or greater) of ozone.²²⁸ This relationship was documented in children who played 3 or more sports and thus spent more time outdoors. It was not documented for those children who played one or two sports.^D The larger effect of high activity sports than low activity sports and an independent effect of time spent

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

outdoors also in the higher ozone communities strengthens the inference that exposure to ozone may modify the effect of sports on the development of asthma in some children.

Previous studies have shown relationships between ozone and hospital admissions in the general population. More recently there have been studies that report the effects of ozone on unscheduled respiratory hospital admissions of children.^{229,230,231,232} A study in Toronto reported a significant relationship between 1-hour maximum ozone concentrations and respiratory hospital admissions in children under the age of two.²³³ Given the relative vulnerability of children in this age category, there is particular concern about these findings from the literature on ozone and hospital admissions.

Increased rates of illness-related school absenteeism have been associated with 1-hour daily maximum²³⁴ and 8-hour average ozone concentrations.^{235,236} In a study by Chen and colleagues (2000), daily school absenteeism was examined in 27,793 students (kindergarten to sixth grade) from 57 elementary school students in Washoe County, NV over a two-year period.²³⁷ In models adjusting for PM₁₀ and CO, ambient ozone levels were found to be associated with school absenteeism. Ozone-related school absences were also examined in a study of 1,933 fourth grade students from 12 southern California communities participating in the Children's Health Study.²³⁸ Due to the comprehensive characterization of health outcomes, this study is valuable in assessing the effect of ozone on illness-related school absenteeism in children. The study spanned the months of January through June 1996, which captured a wide range of exposures while staying mostly below the highest levels observed in the summer season. Larger ozone effects were seen for respiratory causes than for nonrespiratory causes. Park et al. (2002) examined the association between air pollution and school absenteeism in 1,264 students, first to sixth grade, attending school in Seoul, Korea.²³⁹ The study period extended from March 1996 to December 1999, with 8-hour average ozone concentrations ranging from 3.13 ppb to 69.15 ppb (mean 22.86 ppb). Same day ozone concentrations were positively associated with illness-related absences, but inversely associated with non-illness-related absences. The results from these studies suggest that ambient ozone concentrations, on the same day as well as accumulated over two to four weeks, may be associated with school absenteeism, particularly illness-related absences.

The air pollutant most clearly associated with premature mortality is PM, with many studies reporting such an association. However, the new findings of other recent analyses provide evidence that ozone exposure is associated with increased mortality. Key findings are available from multicity time-series studies that report associations between ozone and mortality. These studies include analyses using data from 90 U.S. cities in the National Mortality, Morbidity and Air Pollution (NMMAPS) study^{240,241} and from 95 U.S. cities in an extension to the NMMAPS analyses²⁴², and further analyses using a subset of 19 U.S. cities and focusing on cause-specific mortality associations²⁴³. An additional study used case-crossover design and data from 14 U.S. cities, to further investigate the influence of adjustment for weather variables in the ozone-mortality relationship.²⁴⁴ Finally, results are available from a European study, Air Pollution and Health: a European Approach (APHEA), an analysis using data from 23 cities and 4 cities.^{245,246}

In addition, several meta-analyses have been conducted on the relationship between O₃ and mortality. As described in section 7.4.4 of the second draft CD, these analyses reported fairly consistent and positive combined effect estimates for an increase in mortality for a standardized change in O₃ (CD Figure 7-18, page 7-84).²⁴⁷ Three recent meta-analyses evaluated potential sources of heterogeneity in ozone-mortality associations.^{248,249,250} The second draft CD observes common findings across all three analyses, in that all reported that effect estimates were larger in warm season analyses, reanalysis of results using default GAM criteria did not change the effect estimates, and there was no strong evidence of confounding by PM (CD, p. 7-84). Bell et al. (2005) and Ito et al. (2005) both provided suggestive evidence of publication bias, but ozone-mortality associations remained after accounting for that potential bias. The second draft CD concludes that these studies “provide strong evidence that ozone is associated with mortality.” (CD, p. 7-84)

As discussed in the second draft ozone CD,²⁵¹ there is a substantial amount of additional experimental evidence that links ozone exposure unequivocally with respiratory effects in laboratory animals and humans. These include structural changes in the bronchiolar-alveolar transition (centriacinar) region of the lung, biochemical evidence of acute cellular/tissue injury, inflammation, increased frequency and severity of experimental bacterial infection, and temporary reductions in mechanical lung function. The data linking ozone exposure with respiratory effects have been observed with exposure to ozone at ambient or near-ambient concentrations. Thus, many of the reported epidemiologic associations of ambient ozone with respiratory health effects have considerable biological credibility. Accordingly, the new epidemiologic studies of ambient ozone discussed here are best considered in combination with information on ambient ozone concentration and exposure, and toxicological effects of ozone in animals and humans.

3.3.3 Current 8-Hour Ozone Levels

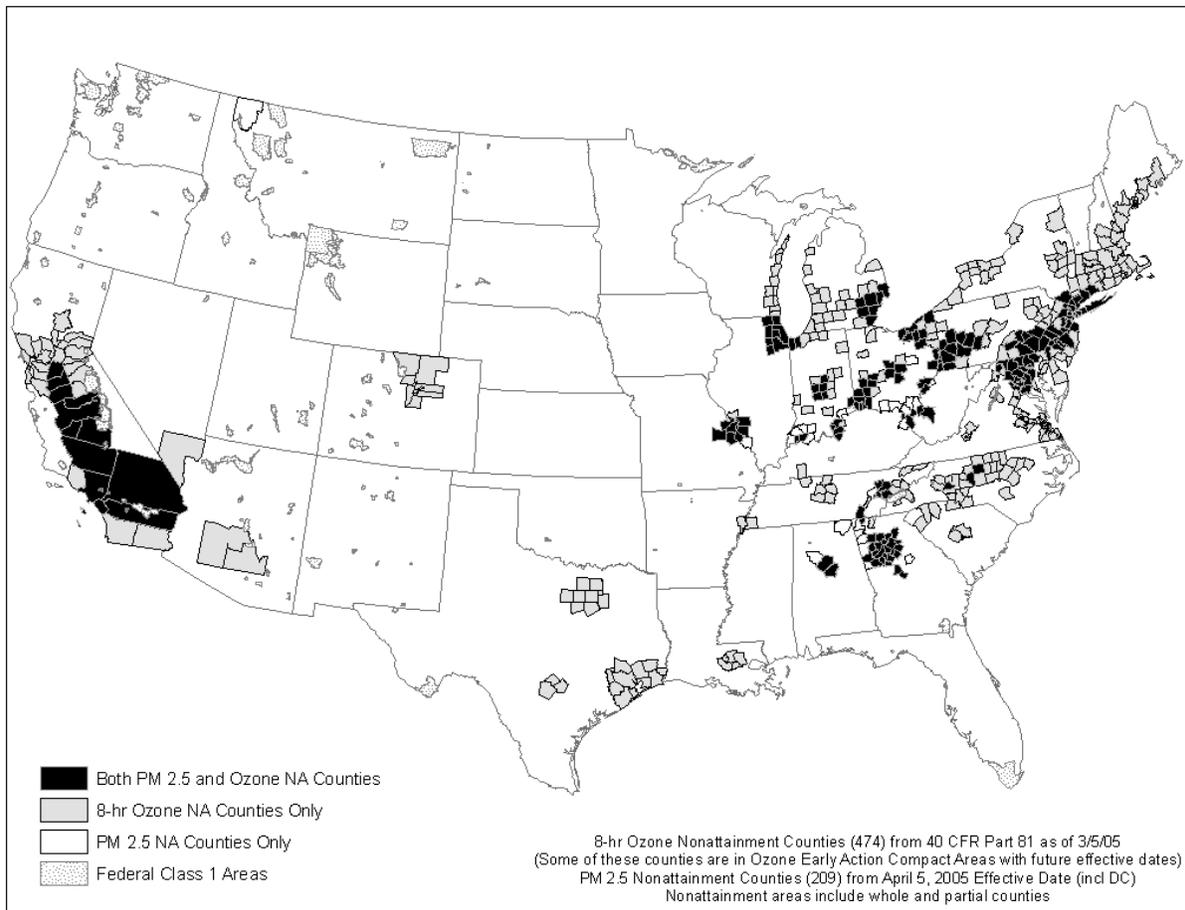
The proposed gas can emission reductions would assist 8-hour ozone nonattainment areas in reaching the standard by each area’s respective attainment date and assist 8-hour ozone maintenance areas in maintaining the 8-hour ozone standard in the future. In this section and the next section we present information on current and model-projected future 8-hour ozone levels.

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

^E An ozone design value is the concentration that determines whether a monitoring site meets the NAAQS for ozone. Because of the way they are defined, design values are determined based on three consecutive-year monitoring periods. For example, an 8-hour design value is the fourth highest daily maximum 8-hour average ozone concentration measured over a three-year period at a given monitor. The full details of these determinations (including accounting for missing values and other complexities) are given in Appendices H and I of 40 CFR Part 50. Due to the precision with which the standards are expressed (0.08 parts per million (ppm) for the 8-hour), a violation of the 8-hour standard is defined as a design value greater than or equal to 0.085 ppm or 85 parts per billion (ppb). For a county, the design value is the highest design value from among all the monitors with valid

According to EPA’s designations, as of September 29, 2005, approximately 159 million people live in the 126 areas that are currently designated as nonattainment for either failing to meet the 8-hour ozone NAAQS or for contributing to poor air quality in a nearby area. There are 474 full or partial counties that make up the 126 8-hour ozone nonattainment areas. Figure 3.3-1 illustrates the widespread nature of these problems. Shown in this figure are counties designated as nonattainment for the 8-hour ozone NAAQS, also depicted are PM_{2.5} nonattainment areas and the Mandatory Class I Federal Areas. The 8-hour ozone nonattainment areas, nonattainment counties and populations are listed in Appendix 3A to this RIA.

Figure 3.3.-1. 8-Hour Ozone and PM_{2.5} Nonattainment Areas and Mandatory Class I Federal Areas



Counties designated as 8-hour ozone nonattainment were classified, on the basis of their one-hour ozone design value, as Subpart 1 or Subpart 2 (69 FR 23951, April 30, 2004). Areas classified as Subpart 2 were then further classified, on the basis of their 8-hour ozone design value, as marginal, moderate, serious, severe or extreme. The maximum attainment date

design values within that county. If a county does not contain an ozone monitor, it does not have a design value. Thus, our analysis may underestimate the number of counties with design values above the level of NAAQS.

assigned to an ozone nonattainment area is based on the area's classification.

Table 3A-1 presents the 8-hour ozone nonattainment areas, their 8-hour design values, their category or classification and their maximum attainment date. States with 8-hour ozone nonattainment areas are required to take action to bring those areas into compliance in the future. Based on the final rule designating and classifying 8-hour ozone nonattainment areas, most 8-hour ozone nonattainment areas will be required to attain the 8-hour ozone NAAQS in the 2007 to 2014 time frame and then be required to maintain the 8-hour ozone NAAQS thereafter.^F The gas can emission standards being proposed in this action would become effective in 2009. Thus, the expected ozone precursor emission inventory reductions from the standards proposed in this action would be useful to States in attaining and/or maintaining the 8-hour ozone NAAQS.

The information presented here indicates that there is currently widespread ozone nonattainment. The proposed gas can controls would be helpful to states in moving towards attainment of the 8-hour ozone NAAQS.

3.3.4 Projected 8-Hour Ozone Levels

Recent air quality modeling predicts that without additional local, regional or national controls there will continue to be a need for reductions in 8-hour ozone concentrations in the future. In the following sections we describe recent ozone air quality modeling from the CAIR-CAMR-CAVR multi-pollutant analysis as well as results of the ozone response surface metamodel (RSM) analysis we completed to assess the potential ozone impacts resulting from the VOC emissions controls proposed for gas cans.

3.3.4.1 CAIR-CAMR-CAVR Ozone Air Quality Modeling

The most recent ozone air quality analyses were performed for an assessment of the combination of the Clean Air Interstate Rule (CAIR), the Clean Air Mercury Rule (CAMR) and the Clean Air Visibility Rule (CAVR), which were rules promulgated by EPA in 2005. The Comprehensive Air Quality Model with Extension (CAMx) model was used as the tool for simulating base and future year concentrations of ozone in support of the CAIR-CAMR-CAVR air quality assessments. Final rules included in the CAIR-CAMR-CAVR analysis include CAIR and all other federal rules that were finalized prior to CAIR. Details on the air quality modeling are provided in the Air Quality Modeling Technical Support Document for the Final Clean Air Interstate Rule, the Air Quality Modeling Technical Support Document for EPA's Multi-Pollutant Analysis and the Air Quality Modeling results for the CAIR-CAMR-CAVR analysis, included in the docket for this proposed rule.^{252,253,254}

Air quality modeling performed for CAIR-CAMR-CAVR indicates that in the absence of additional controls, counties with projected 8-hour ozone concentrations greater than or equal to 85 ppb are likely to persist in the future. The CAIR-CAMR-CAVR analysis provided estimates of future ozone levels across the country. For example, in 2020 based on emission controls currently adopted or expected to be in place, we project that 34 million people will live in 21

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

counties with 8-hour ozone concentrations at and above 85 ppb, see Table 3.3-1.^G Table 3.3-1 also lists the 73 counties, where 44 million people are projected to live, with 2020 projected design values that do not violate the 8-hour ozone NAAQS but are within ten percent of it.

Table 3.3-1. Counties with 2020 projected 8-hour Ozone Concentrations Above and within 10% of the 8-hour Ozone Standard

State	County	2020 Projected 8-hour Ozone Concentration (ppb)	2000 pop	2020 pop
Arizona	Maricopa	79.0	3,072,149	4,513,344
California	Fresno	91.0	799,407	1,010,798
California	Kern	92.0	661,645	851,039
California	Kings	78.0	129,461	171,603
California	Los Angeles	120.0	9,519,338	10,068,317
California	Merced	80.0	210,554	261,895
California	Orange	100.0	2,846,289	3,681,637
California	Riverside	106.0	1,545,387	2,176,313
California	San Bernardino	132.0	1,709,434	2,298,311
California	Tulare	80.0	368,021	461,550
California	Ventura	92.0	753,197	974,455
Colorado	Jefferson	77.0	527,056	644,914
Connecticut	Fairfield Co	90.2	882,567	900,915
Connecticut	Middlesex Co	87.6	155,071	175,771
Connecticut	New Haven Co	88.3	824,008	837,362
Connecticut	New London Co	80.3	259,088	274,769
Connecticut	Tolland Co	77.3	136,364	151,381
D.C.	Washington Co	82.0	572,059	535,936
Delaware	New Castle Co	81.1	500,265	569,214
Delaware	Sussex Co	76.7	156,638	210,515
Illinois	Cook Co	81.0	5,376,740	5,369,914
Indiana	La Porte Co	77.6	110,106	113,133
Indiana	Lake Co	78.5	484,564	492,577
Louisiana	East Baton Rouge Parish	79.4	412,852	516,961
Louisiana	Iberville Parish	78.4	33,320	33,064
Louisiana	Jefferson Parish	77.0	455,466	531,771
Louisiana	Livingston Parish	76.5	91,814	158,340
Louisiana	West Baton Rouge Parish	77.4	21,601	23,836
Maine	York Co	76.7	186,742	217,887
Maryland	Anne Arundel Co	83.9	489,656	603,613
Maryland	Baltimore Co	80.4	754,292	826,595
Maryland	Cecil Co	84.6	85,951	108,489
Maryland	Harford Co	88.8	218,590	320,395
Maryland	Kent Co	81.6	19,197	21,370
Maryland	Prince Georges Co	79.9	801,515	895,723
Massachusetts	Barnstable Co	79.0	222,230	281,619
Massachusetts	Bristol Co	78.4	534,678	583,227

State	County	2020 Projected 8-hour Ozone Concentration (ppb)	2000 pop	2020 pop
Massachusetts	Essex Co	80.2	723,419	774,701
Michigan	Macomb Co	84.1	788,149	884,628
Michigan	Oakland Co	78.3	1,194,156	1,411,792
Michigan	Washtenaw Co	76.5	322,895	369,791
Michigan	Wayne Co	84.1	2,061,162	1,879,877
New Jersey	Atlantic Co	77.2	252,552	286,405
New Jersey	Bergen Co	83.8	884,118	911,737
New Jersey	Camden Co	88.1	508,932	512,662
New Jersey	Cumberland Co	80.2	146,438	153,502
New Jersey	Gloucester Co	88.1	254,673	304,923
New Jersey	Hudson Co	81.9	608,975	603,949
New Jersey	Hunterdon Co	84.5	121,989	160,454
New Jersey	Mercer Co	91.8	350,761	369,956
New Jersey	Middlesex Co	87.9	750,162	858,721
New Jersey	Monmouth Co	82.1	615,301	731,191
New Jersey	Morris Co	80.4	470,212	535,685
New Jersey	Ocean Co	96.0	510,916	642,051
New York	Bronx Co	79.6	1,332,649	1,269,835
New York	Chautauqua Co	77.4	139,750	140,795
New York	Erie Co	82.7	950,265	951,156
New York	Jefferson Co	76.6	111,738	115,332
New York	Niagara Co	79.0	219,846	219,840
New York	Putnam Co	77.7	95,745	122,586
New York	Richmond Co	82.8	443,728	538,856
New York	Suffolk Co	88.8	1,419,369	1,531,991
New York	Westchester Co	82.8	923,459	963,790
Ohio	Ashtabula Co	78.7	102,728	107,401
Ohio	Geauga Co	80.5	90,895	113,978
Ohio	Lake Co	77.8	227,511	248,161
Pennsylvania	Allegheny Co	77.7	1,281,666	1,234,866
Pennsylvania	Bucks Co	91.5	597,635	704,253
Pennsylvania	Chester Co	81.8	433,501	528,280
Pennsylvania	Delaware Co	80.8	550,864	537,547
Pennsylvania	Lancaster Co	78.0	470,658	557,896
Pennsylvania	Lehigh Co	77.2	312,090	334,116
Pennsylvania	Montgomery Co	84.8	750,097	791,523
Pennsylvania	Northampton Co	76.9	267,066	293,668
Pennsylvania	Philadelphia Co	87.5	1,517,550	1,322,901
Rhode Island	Kent Co	82.1	167,090	182,031
Rhode Island	Providence Co	76.6	621,602	622,459
Rhode Island	Washington Co	80.0	123,546	155,633
Texas	Brazoria Co	82.7	241,767	321,123
Texas	Denton Co	78.6	432,976	678,368

State	County	2020 Projected 8-hour Ozone Concentration (ppb)	2000 pop	2020 pop
Texas	Galveston Co	83.4	250,158	315,425
Texas	Harris Co	96.5	3,400,577	4142898
Texas	Jefferson Co	84.5	252,051	265060
Texas	Montgomery Co	77.1	293,768	533560
Texas	Tarrant Co	79.6	1,446,219	1968880
Virginia	Alexandria City	78.3	128,283	131423
Virginia	Arlington Co	83.2	189,453	198100
Virginia	Fairfax Co	82.3	969,749	1210471
Virginia	Hanover Co	77.2	86,320	108636
Wisconsin	Kenosha Co	86.8	149,577	182420
Wisconsin	Milwaukee Co	76.9	940,164	899138
Wisconsin	Ozaukee Co	79.7	82,317	109255
Wisconsin	Racine Co	79.6	188,831	209777
Wisconsin	Sheboygan Co	81.2	112,646	125032
Number of Violating Counties		21		
Population of Violating Counties			29,375,085	33,868,089
Number of Counties within 10%		73		
Population of Counties within 10%			39,432,941	44,380,949

- a) Bolded concentrations indicate levels above the 8-hour ozone standard.
b) Concentrations are calculated for counties with Federal Reference Method PM_{2.5} monitoring data.
c) Populations are based on 2000 census data.
d) Populations are based on 2000 census projections.

3.3.4.2 Ozone Response Surface Metamodel Methodology

We performed ozone air quality modeling simulations for the Eastern United States using the ozone RSM. The ozone RSM is a screening-level air quality modeling tool that allows users to quickly assess the estimated air quality changes over the modeling domain. The ozone RSM is a model of a full-scale air quality model and is based on statistical relationships between model inputs and outputs obtained from the full-scale air quality model. In other words, the ozone RSM uses statistical techniques to relate a response variable to a set of factors that are of interest, e.g., emissions of precursor pollutants from particular sources and locations. The following section describes the modeling methodology, including the development of the multi-dimensional experimental design for control strategies and implementation and verification of the RSM technique. Additional detail is available in the Air Quality Modeling Technical Support Document (AQMTSD) for this proposal.²⁵⁵

The foundation for the ozone response surface metamodeling analyses was the CAMx modeling done in support of the final Clean Air Interstate Rule (CAIR). The CAIR modeling is fully described in the CAIR Air Quality Modeling Technical Support Document, but a brief description is provided below.²⁵⁶ The modeling procedures used in the CAIR analysis (e.g., domain, episodes, meteorology) have been used for several EPA rulemaking analyses over the past five years and are well-established at this point.

The ozone RSM uses the 2015 controlled CAIR emissions inventory as its baseline.²⁵⁷ This inventory does not include the gas can emissions that are being controlled in this proposed action. The controlled and uncontrolled gas can emissions have been incorporated into the base and control runs of the ozone RSM (see Section 2.1 for more detail about the gas can emissions inventory). The inventory also does not include the higher estimates of cold temperature emissions for gasoline vehicles developed for this rule; however, these emissions are not likely to have a significant impact on ozone formation. Because the base years of our air quality modeling analysis are 2020 and 2030, we extrapolate the model from 2015 to 2020 and 2030. Additional detail on how the model was extrapolated to reflect gas can emissions and various projection years is included in the AQMTSD for this proposal.²⁵⁸

The modeling simulations that comprised the metamodeling were conducted using CAMx version 3.10. It should be noted that because the ozone RSM is built from CAMx air quality model runs, it therefore has the same strengths and limitations of the underlying model and its inputs. CAMx is a non-proprietary computer model that simulates the formation and fate of photochemical oxidants including ozone for given input sets of meteorological conditions and emissions. The gridded meteorological data for three historical episodes were developed using the Regional Atmospheric Modeling System (RAMS), version 3b.²⁵⁹ In all, 30 episode days were modeled using frequently-occurring, ozone-conducive, meteorological conditions from the summer of 1995. Emissions estimates were developed for the evaluation year (1995) as well as a future year (2015).

The CAMx model applications were performed for a domain covering all, or portions of, 37 States (and the District of Columbia) in the Eastern U.S., as shown in Figure 3.3-2. The domain has nested horizontal grids of 36 km and 12 km. However, the output data from the metamodeling is provided at a 12 km resolution (i.e., cells from the outer 36 km cells populate the nine finer scale cells, as appropriate). Although the domain of the ozone RSM is the 37 Eastern states, the gas can controls are a nationwide program. Section 2.1.3 describes the nationwide inventory reductions that could be achieved by the proposed gas can controls. Section 2.1.1.2 also details the states that have their own gas can control programs and how the controls proposed here impact states which already have gas can control programs.

Figure 3.3-2. Map of the CAMx Domain used for MSAT Ozone Metamodeling

The ozone RSM used for assessing the impacts of proposed gas can emission reductions was developed broadly to look at various control strategies with respect to attaining the 8-hour ozone NAAQS. The experimental design for the ozone RSM covered three key areas: type of precursor emission (NO_x or VOC), emission source type (i.e., onroad vehicles, nonroad vehicles, area sources, electrical generating utility (EGU) sources, and non-utility point sources), and location in or out of a 2015 model-projected residual ozone nonattainment area. This resulted in a set of 14 emissions factors. Since some of the spillage emissions associated with gas cans are currently included in the NONROAD emissions model, for the purposes of the ozone RSM we have included gas can emissions as part of the nonroad factor in our air quality modeling.

The 14 emission factors were randomly varied and used as inputs to CAMx. The experimental design for these 14 factors was developed using a Maximin Latin Hypercube method. Based on a rule of thumb of 10 runs per factor, we developed an overall design with 140 runs (a base case plus 139 control runs). The range of emissions reductions considered within the metamodel ranged from 0 to 120 percent of the 2015 CAIR emissions. This experimental design resulted in a set of CAMx simulations that serve as the inputs to the ozone response surface metamodel. Because the metamodeling was going to be used to assess the impacts of the proposed gas can standards, the experimental design also included oversampling

in the range of 0 to 10 percent control for the nonroad VOC sector, as well as CAMx runs that only included VOC controls.

To develop a response surface approximation to CAMx, we used a multidimensional kriging approach, implemented through the MIXED procedure in SAS. We modeled the predicted changes in ozone in each CAMx grid cell as a function of the weighted average of the modeled responses in the experimental design. A response-surface was then fit for the ozone design value metric. Validation was performed and is summarized in the AQMTSD. The validation exercises indicated that the ozone RSM replicates CAMx response to emissions changes very well for most emissions combinations and in most locations.

The assessment of proposed gas can controls conducted for this analysis involved adjusting the nonroad mobile source VOC emissions both in and out of ozone nonattainment areas and looking at the impact on the 8-hour ozone design value metric. We created an input or adjustment factor for the nonroad mobile source VOC emission factor by adding future year gas can emission estimates to the projected CAIR emission inventory and then relating the future year emissions estimate to 2015. For this assessment the future years modeled are 2020 and 2030.

3.3.4.3 Ozone Response Surface Metamodel Results

This section summarizes the results of our modeling of ozone air quality impacts in the future with and without the proposed reductions in gas can emissions. Based upon our previous CAIR-CAMR-CAVR air quality modeling, we anticipate that without emission reductions beyond those already required under promulgated regulations and approved SIPs, ozone nonattainment will likely persist into the future.

The inventories that underlie the ozone modeling conducted for this rulemaking included emission reductions from all current or committed federal, state, and local controls, including the recent CAIR. There was no attempt to examine the prospects of areas attaining or maintaining the 8-hour ozone standard with possible additional future controls (i.e., controls beyond current or committed federal, State, and local controls).

According to the ozone response surface metamodel (RSM), the proposed gas can controls are projected to result in a very small population-weighted net improvement in future ozone. The net improvement is generally so small as to be rendered insignificant when presenting design values. The model changes are smaller than the precision with which the ozone standard is expressed (0.08 parts per million (ppm)) and to which 8-hour ozone data is reported.^H Nonetheless, there are some areas where the ozone improvement is more significant. These areas include Chicago, Milwaukee, Detroit and New York City. For example, in Chicago the projected improvement in ozone design values will bring the area approximately 8% closer to attainment. It is also important to note that the ozone RSM results indicate that the counties which are projected to experience the greatest improvement in ozone design values are generally also those that are projected to be either exceeding the NAAQS or within 20% of the NAAQS in the future (e.g. 2020). Those counties that are projected to experience an extremely small

^H Appendix I of 40 CFR Part 50.

increase in ozone design values generally have design values that are already greater than 20% below the NAAQS. The results from the metamodeling projections indicate a net overall improvement in future 8-hour ozone design values due to the proposed gas can controls, when weighted by population. The AQMTSD, contained in the docket for this proposal, includes additional detail on the ozone RSM results.

3.3.5 Environmental Effects of Ozone Pollution

There are a number of public welfare effects associated with the presence of ozone in the ambient air.²⁶⁰ In this section we discuss the impact of ozone on plants, including trees, agronomic crops and urban ornamentals.

3.3.5.1 Impacts on Vegetation

The Air Quality Criteria Document for Ozone and related Photochemical Oxidants notes that “ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant.”²⁶¹ Like carbon dioxide (CO₂) and other gaseous substances, ozone enters plant tissues primarily through apertures (stomata) in leaves in a process called “uptake.” To a lesser extent, ozone can also diffuse directly through surface layers to the plant's interior.²⁶² Once ozone, a highly reactive substance, reaches the interior of plant cells, it inhibits or damages essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns.^{263,264} This damage is commonly manifested as visible foliar injury such as chlorotic or necrotic spots, increased leaf senescence (accelerated leaf aging) and/or reduced photosynthesis. All these effects reduce a plant's capacity to form carbohydrates, which are the primary form of energy used by plants.²⁶⁵ With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, more susceptible to disease, insect attack, harsh weather (e.g., drought, frost) and other environmental stresses (e.g., increasing CO₂ concentrations). Furthermore, there is considerable evidence that ozone can interfere with the formation of mycorrhiza, essential symbiotic fungi associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont.²⁶⁶

Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of O₃ uptake through closure of stomata).^{267,268,269} Other resistance mechanisms may involve the intercellular production of detoxifying substances. Several biochemical substances capable of detoxifying ozone have been reported to occur in plants including the antioxidants ascorbate and glutathione. After injuries have occurred, plants may be capable of repairing the damage to a limited extent.²⁷⁰ Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that numerous other environmental factors modify plant uptake and

response to ozone, it is not possible to identify threshold values above which ozone is toxic for all plants. However, in general, the science suggests that ozone concentrations of 100 ppb or greater can be phytotoxic to a large number of plant species, and can produce acute foliar injury responses, crop yield loss and reduced biomass production. Ozone concentrations below 100 ppb (50 to 99 ppb) can produce these effects in more sensitive plant species, and have the potential over a longer duration of creating chronic stress on vegetation that can lead to effects of concern associated with reduced carbohydrate production and decreased plant vigor. The next few paragraphs present additional information on ozone damage to trees, ecosystems, agronomic crops and urban ornamentals.

Ozone also has been shown conclusively to cause discernible injury to forest trees.^{271,272} In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts.²⁷³ Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function.^{274,275}

Because plants are at the center of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone damages at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors.²⁷⁶ In most instances, responses to chronic or recurrent exposure are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems.^{277,278,279} It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States.

Laboratory and field experiments have also shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN results show that “several economically important crop species are sensitive to ozone levels typical of those found in the United States.”²⁸⁰ In addition, economic studies have shown a relationship between observed ozone levels and crop yields.^{281,282,283}

Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to impact large economic sectors. In the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative analysis has been conducted. It is estimated that more than \$20 billion (1990 dollars) are spent annually on landscaping using ornamentals, both by private property owners/tenants and by governmental units responsible for public areas.²⁸⁴ This is therefore a potentially costly environmental effect. However, methods are not available to allow for plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

3.4 Particulate Matter

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

3.4.1 Science of PM Formation

Particulate matter (PM) represents a broad class of chemically and physically diverse substances. It can be principally characterized as discrete particles that exist in the condensed (liquid or solid) phase spanning several orders of magnitude in size. PM is further described by breaking it down into size fractions. PM_{10} refers to particles with an aerodynamic diameter less than or equal to a nominal 10 micrometers. $PM_{2.5}$ refers to fine particles, those particles with an aerodynamic diameter less than or equal to a nominal 2.5 micrometers. Coarse fraction particles refer to those particles with an aerodynamic diameter less than or equal to a nominal 10 micrometers. Inhalable coarse fraction particles refer to those particles with an aerodynamic diameter greater than 2.5 micrometers, but less than or equal to a nominal 10 micrometers. Ultrafine PM refers to particles with diameters of less than 100 nanometers (0.1 micrometers). Larger particles (greater than 10 micrometers) tend to be removed by the respiratory clearance mechanisms whereas smaller particles (PM_{10}) are deposited deeper in the lungs. Ambient fine particles are a complex mixture including sulfates, nitrates, chlorides, ammonium compounds, organic carbon, elemental carbon, geological material, and metals. Fine particles can remain in the atmosphere for days to weeks and travel through the atmosphere hundreds to thousands of kilometers, while coarse particles generally tend to deposit to the earth within minutes to hours and within tens of kilometers from the emission source.

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

EPA has National Ambient Air Quality Standards (NAAQS) for both $PM_{2.5}$ and PM_{10} . The PM NAAQS consist of a short-term (24-hour) and a long-term (annual) standard. The short-term $PM_{2.5}$ NAAQS is set at a level of $65 \mu\text{g}/\text{m}^3$ based on the 98th percentile concentration averaged over three years. The long-term $PM_{2.5}$ NAAQS specifies an expected annual arithmetic mean not to exceed $15 \mu\text{g}/\text{m}^3$ averaged over three years. The short-term (24-hour) PM_{10} NAAQS is set at a level of $150 \mu\text{g}/\text{m}^3$ not to be exceeded more than once per year. The long-term PM_{10} NAAQS specifies an expected annual arithmetic mean not to exceed $50 \mu\text{g}/\text{m}^3$.

EPA has recently proposed to amend the PM NAAQS.¹ The proposal includes lowering the level of the primary 24-hour fine particle standard from the current level of 65 micrograms per cubic meter ($\mu\text{g}/\text{m}^3$) to 35 $\mu\text{g}/\text{m}^3$, retaining the level of the annual fine standard at 15 $\mu\text{g}/\text{m}^3$, and setting a new primary 24-hour standard for certain inhalable coarse particles (the indicator is qualified so as to include any ambient mix of $\text{PM}_{10-2.5}$ that is dominated by sources typically found in urban environments, such as resuspended road dust from high-density traffic on paved roads and industrial sources, and to exclude any ambient mix of $\text{PM}_{10-2.5}$ dominated by rural windblown dust and soils and agricultural and mining sources) at 70 $\mu\text{g}/\text{m}^3$. The Agency is also requesting comment on various other standards for fine and inhalable coarse PM (71 FR 2620, Jan. 17, 2006).

3.4.2 Health Effects of Particulate Matter

As stated in the EPA Air Quality Criteria Document for PM (PM Criteria Document), available scientific findings “demonstrate well that human health outcomes are associated with ambient PM.”²⁸⁵ We are relying primarily on the data and conclusions in the PM Criteria Document and PM staff paper, which reflects EPA’s analysis of policy-relevant science from the PM Criteria Document, regarding the health effects associated with particulate matter.²⁸⁶ We also present additional recent studies published after the cut-off date for the PM criteria document. Taken together this information supports the conclusion that PM-related emissions from the gasoline vehicles and gas cans being controlled in this action are associated with adverse health effects. Information on PM-related mortality is presented first, followed by information on PM-related morbidity and near-roadway PM exposure.

3.4.2.1 Short-term Exposure Mortality and Morbidity Studies

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

Among the studies of effects from short-term exposure to $\text{PM}_{2.5}$, several studies specifically address the contribution of mobile sources to short-term $\text{PM}_{2.5}$ effects on daily mortality. These studies indicate that there are statistically significant associations between mortality and PM related to mobile source emissions (CD, p.8-85). The analyses incorporate source apportionment tools into daily mortality studies and are briefly mentioned here. Analyses incorporating source apportionment by factor analysis with daily time-series studies of daily death indicated a relationship between mobile source $\text{PM}_{2.5}$ and mortality.^{287,288} Another recent study in 14 U.S. cities examined the effect of PM_{10} exposures on daily hospital admissions for

¹ US EPA, National Ambient Air Quality Standards for Particulate Matter (71 FR 2620, Jan. 17, 2006). This document is also available on the web at: <http://www.epa.gov/air/particlepollution/actions.html>.

cardiovascular disease. They found that the effect of PM_{10} was significantly greater in areas with a larger proportion of PM_{10} coming from motor vehicles, indicating that PM_{10} from these sources may have a greater effect on the toxicity of ambient PM_{10} when compared with other sources.²⁸⁹ These studies provide evidence that PM-related emissions, specifically from mobile sources, are associated with adverse health effects.

3.4.2.2 Long-term Exposure Mortality and Morbidity Studies

Short-term exposure studies provide one way of examining the effect of short-term variations in air quality on morbidity and mortality. However, they do not allow for an evaluation of the effect of long-term exposure to air pollution on human mortality and morbidity.²⁹⁰ Longitudinal cohort studies allow for analysis of such effects.

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

The PM Criteria Document emphasizes the results of two long-term studies, the Six Cities and American Cancer Society (ACS) prospective cohort studies, based on several factors – the large study population in the ACS study, the large air quality data set in the Six Cities study, the generally representative study populations used in the Six Cities and ACS studies, and the fact that these studies have undergone extensive reanalysis (CD, p. 8-306).^{291,292,293} One reanalysis of the ACS cohort data, which was published after the PM criteria document was finalized, found a larger association than had previously been reported between long-term $PM_{2.5}$ exposure and mortality in the Los Angeles area using a new exposure estimation method that accounted for variations in concentration within the city.²⁹⁴ These studies provide strong evidence of a link between $PM_{2.5}$ and mortality, including all-cause, cardiorespiratory, and lung cancer mortality (CD, p. 8-307).

As discussed in the PM Criteria Document, the newer morbidity studies that combine the features of cross-sectional and cohort studies provide the best evidence for chronic exposure effects. Long-term studies evaluating the effect of ambient PM on children's development have shown some evidence indicating effects of $PM_{2.5}$ and/or PM_{10} on reduced lung function growth (CD, Section 8.3.3.2.3). In another recent publication, investigators in southern California reported the results of a cross-sectional study of outdoor $PM_{2.5}$ and measures of atherosclerosis in the Los Angeles basin.²⁹⁵ The study found significant associations between ambient residential $PM_{2.5}$ and carotid intima-media thickness (CIMT), an indicator of subclinical atherosclerosis, an underlying factor in cardiovascular disease.

3.4.2.3 Roadway-Related PM Exposure

A recent body of studies has suggested a link between residential proximity to high-traffic roadways or time spent in traffic and adverse health effects. While many of these studies

did not measure PM specifically, they include potential exhaust exposures which include mobile source PM because they employ indices such as roadway proximity or traffic volumes. One study with specific relevance to PM_{2.5} health effects is a study that was done in North Carolina looking at concentrations of PM_{2.5} inside police cars and corresponding physiological changes in the police personnel driving the cars. The authors report significant elevations in markers of cardiac risk associated with concentrations of PM_{2.5} inside police cars on North Carolina state highways.²⁹⁶ Additional information on near-roadway health effects is included in Section 3.5 of this RIA.

3.4.3 Current and Projected PM Levels

The emission reductions from this proposed rule would assist PM nonattainment areas in reaching the standard by each area's respective attainment date and assist PM maintenance areas in maintaining the PM standards in the future. In this section we present information on current and future attainment of the PM standards.

3.4.3.1 Current PM_{2.5} Levels

A nonattainment area is defined in the Clean Air Act (CAA) as an area that is violating an ambient standard or is contributing to a nearby area that is violating the standard. EPA has recently designated nonattainment areas for the 1997 PM_{2.5} NAAQS by calculating air quality design values (using 2001-2003 or 2002-2004 measurements) and considering other factors.^J The Air Quality Designations and Classifications for the Fine Particles (PM_{2.5}) NAAQS rule lays out the factors that EPA considered in making the nonattainment designations (70 FR 943, Jan. 5, 2005). According to EPA's recent designations, approximately 88 million people live in the 39 PM_{2.5} areas designated as nonattainment for either failing to meet the 1997 PM_{2.5} NAAQS or for contributing to poor air quality in a nearby area. There are 208 full or partial counties that make up the PM_{2.5} nonattainment areas, as shown in Figure 3.3-1. The PM_{2.5} nonattainment counties, areas and populations, as of September 2005, are listed in Appendix 3B to this RIA.

States with PM_{2.5} nonattainment areas will be required to take action to bring those areas into compliance in the future. Most PM_{2.5} nonattainment areas will be required to attain the 1997 PM_{2.5} NAAQS in the 2009 to 2014 time frame and then be required to maintain the PM_{2.5} NAAQS thereafter.^K The emission standards being proposed in this action would become effective between 2009 and 2015. The expected PM_{2.5} and PM_{2.5} precursor inventory reductions

^J The full details involved in calculating a PM_{2.5} design value are given in Appendix N of 40 CFR Part 50.

^K While the final implementation process for bringing the nation's air into attainment with the PM_{2.5} NAAQS is still being completed in a separate rulemaking action, the basic framework is well defined by the statute. The EPA finalized PM_{2.5} attainment and nonattainment areas in April 2005. Following designation, Section 172(b) of the Clean Air Act allows states up to 3 years to submit a revision to their state implementation plan (SIP) that provides for the attainment of the PM_{2.5} standard. Based on this provision, states could submit these SIPs until April 2008. Section 172(a)(2) of the Clean Air Act requires that these SIP revisions demonstrate that the nonattainment areas will attain the PM_{2.5} standard as expeditiously as practicable but no later than 5 years from the date that the area was designated nonattainment. However, based on the severity of the air quality problem and the availability and feasibility of control measures, the Administrator may extend the attainment date "for a period of no greater than 10 years from the date of designation as nonattainment." Based on section 172(a) provisions in the Act, we expect that areas will need to attain the PM_{2.5} NAAQS in the 2010 (based on 2007 - 2009 air quality data) to 2015 (based on 2012 to 2014 air quality data) time frame, and then be required to maintain the NAAQS thereafter.

from the standards proposed in this action would be useful to states to attain and/or maintain the 1997 PM_{2.5} NAAQS.

3.4.3.2 Current PM₁₀ Levels

EPA designated PM₁₀ nonattainment areas in 1990.^L As of September 2005, approximately 29 million people live in the 55 areas that are designated as PM₁₀ nonattainment, for either failing to meet the PM₁₀ NAAQS or for contributing to poor air quality in a nearby area. There are 54 full or partial counties that make up the PM₁₀ nonattainment areas. The PM₁₀ nonattainment areas and populations are listed in Appendix 3B to this RIA.

The attainment date for the initial moderate PM₁₀ nonattainment areas, designated by law on November 15, 1990, was December 31, 1994. Several additional moderate PM₁₀ nonattainment areas were designated in January of 1994, and the attainment date for these areas was December 31, 2000. The initial serious PM₁₀ nonattainment areas had an attainment date set by the Act of December 31, 2001. The Act provides that EPA may grant extensions of the serious area attainment dates of up to 5 years, provided that the area requesting the extension meets the requirements of section 188(e) of the Act. Four serious PM₁₀ nonattainment areas (Phoenix, Arizona; Clark County (Las Vegas), NV; Coachella Valley, and South Coast (Los Angeles), CA) have received extensions of the December 31, 2001 attainment date and thus have new attainment dates of December 31, 2006. We expect that most PM₁₀ nonattainment areas will attain the PM₁₀ standard in the 2006 time frame, depending on an area's classification and other factors, and then be required to maintain the PM₁₀ NAAQS thereafter. The projected reductions in emissions from the proposed controls would be useful to states to maintain the PM₁₀ NAAQS.^M

3.4.3.3 Projected PM_{2.5} Levels

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

3.4.3.3.1 PM Modeling Methodology

The most recent PM air quality analyses were performed for an assessment of the combination of the Clean Air Interstate Rule (CAIR), the Clean Air Mercury Rule (CAMR) and the Clean Air Visibility Rule (CAVR), which were rules promulgated by EPA in 2005. The Community Multiscale Air Quality (CMAQ) model was used as the tool for simulating base and

^L A PM₁₀ design value is the concentration that determines whether a monitoring site meets the NAAQS for PM₁₀. The full details involved in calculating a PM₁₀ design value are given in Appendices H and I of 40 CFR Part 50.

^M As mentioned above, the EPA has recently proposed to amend the PM NAAQS, by establishing a new indicator for certain inhalable coarse particles, and a new primary 24-hour standard for coarse particles described by that indicator. EPA also proposed to revoke the current 24-hour PM₁₀ standard in all areas of the country except in those areas with a population of at least 100,000 people and which contain at least one monitor violating the 24-hour PM₁₀ standard, based on the most recent 3 years of air quality data. In addition, EPA proposed to revoke upon promulgation of this rule the current annual PM₁₀ standard if EPA finalizes the proposed primary standard for PM_{10.2.5} (71 FR 2620, Jan. 17, 2006).

future year concentrations of PM, visibility and deposition in support of the CAIR-CAMR-CAVR air quality assessments. Final rules included in the CAIR-CAMR-CAVR analysis include CAIR and all other federal rules that were finalized prior to CAIR. Details on the air quality modeling are provided in the Air Quality Modeling Technical Support Document for the Final Clean Air Interstate Rule, the Air Quality Modeling Technical Support Document for EPA's Multi-Pollutant Analysis and the Air Quality Modeling results for the CAIR-CAMR-CAVR analysis, included in the docket for this proposed rule.^{297,298,299}

3.4.3.3.2 Areas at Risk of Future PM_{2.5} Violations

Air quality modeling performed for CAIR-CAMR-CAVR indicates that in the absence of additional local, regional or national controls, counties with annual average PM_{2.5} levels above 15 µg/m³ are likely to persist in the future. The CAIR-CAMR-CAVR analysis provided estimates of future PM_{2.5} levels across the country. For example, in 2020 based on emission controls currently adopted or expected to be in place^N, we project that 43 million people will live in 34 counties with average PM_{2.5} levels at and above 15 µg/m³, see Table 3.4-1. The proposed rule would assist these counties in attaining the PM_{2.5} NAAQS. Table 3.4-1 also lists the 47 counties, where 23 million people are projected to live, with 2020 projected design values that do not violate the annual PM_{2.5} NAAQS but are within ten percent of it. These are counties that are not projected to violate the standard, but to be close to it, so the proposed rule would help ensure that these counties continue to maintain their attainment status.

Table 3.4-1. Counties with 2020 Projected Annual PM_{2.5} Design Values Above and within 10% of the Annual PM_{2.5} Standard

State	County	2020 Projected PM _{2.5} Concentration (µg/m ³) ^{a,b}	2000 pop ^c	2020 pop ^d
Alabama	Jefferson Co	17.70	662,046	673,910
Alabama	Montgomery Co	13.75	223,509	257,062
Alabama	Morgan Co	13.85	111,064	133,114
Alabama	Russell Co	14.53	49,756	56,029
California	Butte Co	14.08	203,170	253,664
California	Fresno Co	20.22	799,406	1,012,929
California	Imperial Co	15.32	142,360	183,835
California	Kern Co	24.89	661,644	851,948
California	Kings Co	16.42	129,460	172,415
California	Los Angeles Co	27.57	9,519,334	10,067,663
California	Merced Co	16.65	210,553	263,184
California	Orange Co	23.06	2,846,288	3,690,329
California	Riverside Co	31.13	1,545,386	2,173,672
California	San Bernardino Co	27.48	1,709,433	2,302,697
California	San Diego Co	15.78	2,813,831	3,715,268

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

State	County	2020 Projected PM _{2.5} Concentration ($\mu\text{g}/\text{m}^3$) ^{a,b}	2000 pop ^c	2020 pop ^d
California	San Joaquin Co	15.11	563,597	711,938
California	Stanislaus Co	16.49	446,996	579,349
California	Tulare Co	21.09	368,020	464,651
California	Ventura Co	15.47	753,196	979,467
Delaware	New Castle Co	14.53	500,264	569,213
Georgia	Bibb Co	14.97	153,887	162,876
Georgia	Chatham Co	14.09	232,047	251,910
Georgia	Clarke Co	13.59	101,488	111,243
Georgia	Clayton Co	15.89	236,516	295,993
Georgia	Cobb Co	14.44	607,750	881,392
Georgia	DeKalb Co	15.32	665,864	734,093
Georgia	Floyd Co	16.26	90,565	100,281
Georgia	Fulton Co	16.44	816,005	898,342
Georgia	Muscogee Co	13.90	186,290	201,873
Georgia	Richmond Co	14.24	199,774	216,360
Georgia	Washington Co	13.68	21,176	23,246
Georgia	Wilkinson Co	15.33	10,220	11,620
Illinois	Cook Co	17.65	5,376,739	5,369,914
Illinois	DuPage Co	14.18	904,160	1,134,208
Illinois	Madison Co	16.23	258,940	276,838
Illinois	St. Clair Co	15.80	256,081	250,436
Illinois	Will Co	14.35	502,265	679,024
Indiana	Clark Co	14.64	96,472	117,909
Indiana	Dubois Co	14.23	39,674	43,249
Indiana	Elkhart Co	13.87	182,790	209,907
Indiana	Lake Co	16.46	484,563	492,577
Indiana	Marion Co	15.44	860,453	901,295
Indiana	Porter Co	14.69	146,798	186,219
Indiana	Vanderburgh Co	14.38	171,922	179,060
Kentucky	Jefferson Co	15.02	693,603	717,730
Louisiana	East Baton Rouge Parish	15.38	412,851	516,960
Louisiana	West Baton Rouge Parish	14.88	21,601	23,836
Maryland	Baltimore city	14.86	651,153	575,702
Michigan	Monroe Co	13.83	145,945	161,437
Michigan	Oakland Co	13.59	1,194,155	1,411,792
Michigan	Wayne Co	18.09	2,061,161	1,879,876
Mississippi	Jones Co	14.15	64,958	73,126
Missouri	St. Louis city	14.64	348,188	298,670
Montana	Lincoln Co	16.00	18,837	20,078
New York	New York Co	14.01	1,537,194	1,560,060
Ohio	Butler Co	14.90	332,806	438,844
Ohio	Cuyahoga Co	16.76	1,393,977	1,305,880
Ohio	Franklin Co	14.30	1,068,977	1,220,749
Ohio	Hamilton Co	15.96	845,302	841,465
Ohio	Jefferson Co	15.53	73,894	67,141

State	County	2020 Projected PM _{2.5} Concentration ($\mu\text{g}/\text{m}^3$) ^{a,b}	2000 pop ^c	2020 pop ^d
Ohio	Lawrence Co	13.76	62,319	64,104
Ohio	Lucas Co	13.56	455,053	439,311
Ohio	Scioto Co	15.45	79,195	81,725
Ohio	Stark Co	14.38	378,097	386,952
Ohio	Summit Co	14.00	542,898	564,374
Pennsylvania	Allegheny Co	17.64	1,281,665	1,234,865
Pennsylvania	Delaware Co	13.64	550,863	537,547
Pennsylvania	Philadelphia Co	14.63	1,517,549	1,322,900
Tennessee	Davidson Co	13.97	569,890	610,103
Tennessee	Hamilton Co	14.68	307,895	346,603
Tennessee	Knox Co	15.14	382,031	471,904
Tennessee	Mauzy Co	14.42	69,498	87,456
Tennessee	Shelby Co	14.45	897,471	1,019,065
Tennessee	Sullivan Co	13.63	153,048	167,368
Texas	Harris Co	14.49	3,400,577	4,142,897
Utah	Salt Lake Co	13.69	898,386	1,210,980
West Virginia	Brooke Co	14.29	25,447	24,221
West Virginia	Cabell Co	14.79	96,784	91,003
West Virginia	Hancock Co	14.80	32,667	30,461
West Virginia	Kanawha Co	14.36	200,072	196,337
West Virginia	Wood Co	13.81	87,986	87,965
Number of Violating Counties		34		
Population of Violating Counties			38,776,409	43,594,538
Number of Counties within 10%		47		
Population of Counties within 10%			20,393,336	23,479,151

a) Bolded concentrations indicate levels above the annual PM_{2.5} standard.

b) Concentrations are calculated for counties with Federal Reference Method PM_{2.5} monitoring data.

c) Populations are based on 2000 census data.

d) Populations are based on 2000 census projections.

3.4.4 Environmental Effects of PM Pollution

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

3.4.4.1 Visibility Degradation

Visibility is important because it directly affects people's enjoyment of daily activities in all parts of the country. Individuals value good visibility for the well-being it provides them directly, both in where they live and work, and in places where they enjoy recreational opportunities. Visibility is also highly valued in significant natural areas such as national parks and wilderness areas, because of the special emphasis given to protecting these lands now and for future generations.

Fine particles are the major cause of reduced visibility in parts of the United States. To

address the welfare effects of PM on visibility, EPA set secondary PM_{2.5} standards in 1997 which would work in conjunction with the establishment of a regional haze program. The secondary (welfare-based) PM_{2.5} NAAQS was established as equal to the primary (health-based) NAAQS of 15 µg/m³ (based on a 3-year average of the annual mean) (62 FR 38669, July 18, 1997). Furthermore, in setting the 1997 PM_{2.5} NAAQS, EPA acknowledged that levels of fine particles below the NAAQS may also contribute to unacceptable visibility impairment and regional haze problems in some areas. Section 169 of the Act provides additional authorities to remedy existing visibility impairment and prevent future visibility impairment in the 156 national parks, forests and wilderness areas labeled as Mandatory Class I Federal Areas (62 FR 38680-81, July 18, 1997).^O In July 1999 the regional haze rule (64 FR 35714) was put in place to protect the visibility in Mandatory Class I Federal Areas. A list of the Mandatory Class I Federal Areas is included in Appendix 3C.^P

Data showing PM_{2.5} nonattainment areas and visibility levels above background at the Mandatory Class I Federal Areas demonstrate that unacceptable visibility impairment is experienced throughout the U.S., in multi-state regions, urban areas, and remote mandatory Federal class I areas. The PM and PM precursor emissions from the vehicles and gas cans subject to this proposed rule contribute to these visibility effects.

3.4.4.1.1 Current Visibility Impairment

The need for reductions in the levels of PM_{2.5} is widespread. Currently, high ambient PM_{2.5} levels are measured throughout the country. Fine particles may remain suspended for days or weeks and travel hundreds to thousands of kilometers, and thus fine particles emitted or created in one county may contribute to ambient concentrations in a neighboring region.³⁰⁰

Recently designated PM_{2.5} nonattainment areas indicate that almost 90 million people live in 208 counties that are in nonattainment for the 1997 PM_{2.5} NAAQS, see Appendix 3.4-A. Thus, at least these populations (plus others who travel to these areas) would likely be experiencing visibility impairment that is unacceptable.

3.4.4.1.2 Current Visibility Impairment at Mandatory Class I Federal Areas

Detailed information about current and historical visibility conditions in Mandatory Class I Federal Areas is summarized in the EPA Report to Congress and the 2002 EPA Trends Report.^{301,302} The conclusions draw upon the Interagency Monitoring of Protected Visual Environments (IMPROVE) network data. One of the objectives of the IMPROVE monitoring network program is to provide regional haze monitoring representing all Mandatory Class I Federal Areas where practical. The National Park Service report also describes the state of national park visibility conditions and discusses the need for improvement.³⁰³

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

^P As mentioned above, the EPA has recently proposed to amend the PM NAAQS (71 FR 2620, Jan. 17, 2006). The proposal would set the secondary NAAQS equal to the primary standards for both PM_{2.5} and PM_{10-2.5}. EPA also is taking comment on whether to set a separate PM_{2.5} standard, designed to address visibility (principally in urban areas), on potential levels for that standard within a range of 20 to 30 µg/m³, and on averaging times for the standard within a range of four to eight daylight hours.

The regional haze rule requires states to establish goals for each affected Mandatory Class I Federal Area to improve visibility on the haziest days (20% most impaired days) and ensure no degradation occurs on the cleanest days (20% least impaired days). Although there have been general trends toward improved visibility, progress is still needed on the haziest days. Specifically, as discussed in the 2002 EPA Trends Report, without the effects of pollution a natural visual range in the United States is approximately 75 to 150 km in the East and 200 to 300 km in the West. In 2001, the mean visual range for the worst days was 29 km in the East and 98 km in the West.³⁰⁴

3.4.4.1.3 Future Visibility Impairment

Recent modeling for the CAIR-CAMR-CAVR was used to project PM_{2.5} levels in the U.S. in 2020. The results suggest that PM_{2.5} levels above the 1997 NAAQS will persist in the future. We predicted that in 2020, there will be 34 counties with a population of 47 million where annual PM_{2.5} levels are above 15 µg/m³, see Table 3.4-1. Thus, in the future, a percentage of the population may continue to experience unacceptable visibility impairment in areas where they live, work and recreate.

The PM and PM precursor emissions from the vehicles and gas cans subject to the proposed controls contribute to visibility impairment. These emissions occur in and around areas with PM_{2.5} levels above the annual 1997 PM_{2.5} NAAQS. Thus, the emissions from these sources contribute to the unacceptable current and anticipated visibility impairment and the proposed emission reductions may help improve future visibility impairment.

3.4.4.1.4 Future Visibility Impairment at Mandatory Class I Federal Areas

Achieving the annual 1997 PM_{2.5} NAAQS will help improve visibility across the country, but it will not be sufficient to meet the statutory goal of no manmade impairment in the Mandatory Class I Federal Areas (64 FR 35722, July 1, 1999 and 62 FR 38680, July 18, 1997). In setting the NAAQS, EPA discussed how the NAAQS in combination with the regional haze program, is deemed to improve visibility consistent with the goals of the Act. In the East, there are and will continue to be areas with PM_{2.5} concentrations above 15 µg/m³ and where light extinction is significantly above natural background. Thus, large areas of the Eastern United States have air pollution that is causing and will continue to cause unacceptable visibility problems. In the West, scenic vistas are especially important to public welfare. Although the annual 1997 PM_{2.5} NAAQS is met in most areas outside of California, virtually the entire West is in close proximity to a scenic Mandatory Class I Federal Area protected by 169A and 169B of the CAA.

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

The overall goal of the regional haze program is to prevent future visibility impairment

and remedy existing visibility impairment in Mandatory Class I Federal Areas. As shown by the future visibility estimates in Appendix 3C there will continue to be Mandatory Class I Federal Areas with visibility levels above background in 2015.³⁰⁵ Additional emission reductions will be needed from the broad set of sources that contribute, including the vehicles and gas cans subject to this proposed rule. The reductions proposed in this action are a part of the overall strategy to achieve the visibility goals of the Act and the regional haze program.

3.4.4.2 Atmospheric Deposition

Wet and dry deposition of ambient particulate matter delivers a complex mixture of metals (e.g., mercury, zinc, lead, nickel, aluminum, cadmium), organic compounds (e.g., POM, dioxins, furans) and inorganic compounds (e.g., nitrate, sulfate) to terrestrial and aquatic ecosystems. The chemical form of the compounds deposited is impacted by a variety of factors including ambient conditions (e.g., temperature, humidity, oxidant levels) and the sources of the material. Chemical and physical transformations of the particulate compounds occur in the atmosphere as well as the media onto which they deposit. These transformations in turn influence the fate, bioavailability and potential toxicity of these compounds. Atmospheric deposition has been identified as a key component of the environmental and human health hazard posed by several pollutants including mercury, dioxin and PCBs.³⁰⁶

Adverse impacts on water quality can occur when atmospheric contaminants deposit to the water surface or when material deposited on the land enters a waterbody through runoff. Potential impacts of atmospheric deposition to waterbodies include adverse effects to human health and welfare through ingestion of contaminated fish, ingestion of contaminated water, damage to the marine ecology, and limited recreational uses. Several studies have been conducted in U.S. coastal waters and in the Great Lakes Region in which the role of ambient PM deposition and runoff is investigated.^{307,308,309,310,311}

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

In the following subsections, atmospheric deposition of heavy metals and particulate organic material is discussed.

3.4.4.2.1 Heavy Metals

Heavy metals, including cadmium, copper, lead, chromium, mercury, nickel and zinc, have the greatest potential for influencing forest growth (CD, p. 4-87).³¹² Investigation of trace metals near roadways and industrial facilities indicate that a substantial burden of heavy metals can accumulate on vegetative surfaces. Copper, zinc, and nickel have been documented to cause direct toxicity to vegetation under field conditions (CD, p. 4-75). Little research has been conducted on the effects associated with mixtures of contaminants found in ambient PM. While metals typically exhibit low solubility, limiting their bioavailability and direct toxicity, chemical

transformations of metal compounds occur in the environment, particularly in the presence of acidic or other oxidizing species. These chemical changes influence the mobility and toxicity of metals in the environment. Once taken up into plant tissue, a metal compound can undergo chemical changes, accumulate and be passed along to herbivores or can re-enter the soil and further cycle in the environment.

Although there has been no direct evidence of a physiological association between tree injury and heavy metal exposures, heavy metals have been implicated because of similarities between metal deposition patterns and forest decline (CD, p. 4-76).³¹³ Contamination of plant leaves by heavy metals can lead to elevated soil levels. Trace metals absorbed into the plant frequently bind to the leaf tissue, and then are lost when the leaf drops (CD, p. 4-75). As the fallen leaves decompose, the heavy metals are transferred into the soil.^{314,315}

The environmental sources and cycling of mercury are currently of particular concern due to the bioaccumulation and biomagnification of this metal in aquatic ecosystems and the potent toxic nature of mercury in the forms in which it is ingested by people and other animals. Mercury is unusual compared with other metals in that it largely partitions into the gas phase (in elemental form), and therefore has a longer residence time in the atmosphere than a metal found predominantly in the particle phase. This property enables mercury to travel far from the primary source before being deposited and accumulating in the aquatic ecosystem. The major source of mercury in the Great Lakes is from atmospheric deposition, accounting for approximately eighty percent of the mercury in Lake Michigan.^{316,317} Over fifty percent of the mercury in the Chesapeake Bay has been attributed to atmospheric deposition.³¹⁸ Overall, the National Science and Technology Council (NSTC, 1999) identifies atmospheric deposition as the primary source of mercury to aquatic systems. Thirty-seven states have issued health advisories for the consumption of fish contaminated by mercury; however, most of these advisories are issued in areas without a mercury point source.

Elevated levels of zinc and lead have been identified in streambed sediments, and these elevated levels have been correlated with population density and motor vehicle use.^{319,320} Zinc and nickel have also been identified in urban water and soils. In addition, platinum, palladium, and rhodium, metals found in the catalysts of modern motor vehicles, have been measured at elevated levels along roadsides.³²¹ Plant uptake of platinum has been observed at these locations.

3.4.4.2.2 Polycyclic Organic Matter

Polycyclic organic matter (POM) is a byproduct of incomplete combustion and consists of organic compounds with more than one benzene ring and a boiling point greater than or equal to 100 degrees centigrade.³²² Polycyclic aromatic hydrocarbons (PAHs) are a class of POM that contain compounds which are known or suspected carcinogens.

Major sources of PAHs include mobile sources. PAHs in the environment may be present as a gas or adsorbed onto airborne particulate matter. Since the majority of PAHs are adsorbed onto particles less than 1.0 μm in diameter, long range transport is possible. However, studies have shown that PAH compounds adsorbed onto diesel exhaust particulate and exposed to ozone have half lives of 0.5 to 1.0 hours.³²³

Since PAHs are insoluble, the compounds generally are particle reactive and accumulate in sediments. Atmospheric deposition of particles is believed to be the major source of PAHs to the sediments of Lake Michigan.^{324,325} Analyses of PAH deposition to Chesapeake and Galveston Bay indicate that dry deposition and gas exchange from the atmosphere to the surface water predominate.^{326,327} Sediment concentrations of PAHs are high enough in some segments of Tampa Bay to pose an environmental health threat. EPA funded a study to better characterize the sources and loading rates for PAHs into Tampa Bay.³²⁸ PAHs that enter a waterbody through gas exchange likely partition into organic rich particles and be biologically recycled, while dry deposition of aerosols containing PAHs tends to be more resistant to biological recycling.³²⁹ Thus, dry deposition is likely the main pathway for PAH concentrations in sediments while gas/water exchange at the surface may lead to PAH distribution into the food web, leading to increased health risk concerns.

Trends in PAH deposition levels are difficult to discern because of highly variable ambient air concentrations, lack of consistency in monitoring methods, and the significant influence of local sources on deposition levels.³³⁰ Van Metre et al. (2000) noted PAH concentrations in urban reservoir sediments have increased by 200-300% over the last forty years and correlates with increases in automobile use.³³¹

Cousins et al. (1999) estimates that greater than ninety percent of semi-volatile organic compound (SVOC) emissions in the United Kingdom deposit on soil.³³² An analysis of polycyclic aromatic hydrocarbon (PAH) concentrations near a Czechoslovakian roadway indicated that concentrations were thirty times greater than background.³³³

3.4.4.3 Materials Damage and Soiling

The deposition of airborne particles can also reduce the aesthetic appeal of buildings and culturally important articles through soiling, and can contribute directly (or in conjunction with other pollutants) to structural damage by means of corrosion or erosion.³³⁴ Particles affect materials principally by promoting and accelerating the corrosion of metals, by degrading paints, and by deteriorating building materials such as concrete and limestone. Particles contribute to these effects because of their electrolytic, hygroscopic, and acidic properties, and their ability to sorb corrosive gases (principally sulfur dioxide). The rate of metal corrosion depends on a number of factors, including the deposition rate and nature of the pollutant; the influence of the metal protective corrosion film; the amount of moisture present; variability in the electrochemical reactions; the presence and concentration of other surface electrolytes; and the orientation of the metal surface.

3.5 Health and Welfare Impacts of Near-Roadway Exposure

In recent years, a large number of epidemiological studies have examined associations between living near major roads and different adverse health endpoints. These studies generally examine people living near heavily-trafficked roadways, typically within several hundred meters, where fresh emissions from motor vehicles are not yet fully diluted with background air.

As discussed in Chapter 3.1.3, many studies have measured elevated concentrations of pollutants emitted directly by motor vehicles near large roadways, as compared to overall urban background levels. These elevated concentrations generally occur within approximately 200 meters of the road, although the distance may vary depending on traffic and environmental conditions. Pollutants measured with elevated concentrations include benzene, polycyclic aromatic hydrocarbons, carbon monoxide, nitrogen dioxide, black carbon, and coarse, fine, and ultrafine particles. In addition, resuspended road dust, and wear particles from tire and brake use also show concentration increases in proximity of major roadways.

The near-roadway health studies provide stronger evidence for some health endpoints than others. Epidemiologic evidence of adverse responses to traffic-related pollution is strongest for non-allergic respiratory symptoms, and several well-conducted epidemiologic studies have shown associations with cardiovascular effects, premature adult mortality, and adverse birth outcomes, including low birth weight and size. Traffic-related pollutants have been repeatedly associated with increased prevalence of asthma-related respiratory symptoms in children, although epidemiologic evidence remains inconclusive for a hypothesized link between traffic and the development of allergies and new onset asthma. For childhood cancer, in particular childhood leukemia, epidemiologic studies have shown less ability to detect the risks predicted from toxicological studies. Several small studies report positive associations, though such effects have not been observed in two larger studies. As described above in Chapter 1.3, benzene and 1,3-butadiene are both known human leukemogens in adults from occupational exposures. As previously mentioned, epidemiologic studies have shown an increased risk of leukemia among children whose parents have been occupationally exposed to benzene. While epidemiologic studies of near-roadway exposures have not always shown a statistically significant association with childhood leukemias, the results are consistent with the risks predicted from the studies at higher exposure levels. As a whole the toxicology and epidemiology are consistent with a potentially serious children's health concern and additional research is needed.

3.5.1 Mortality

Living near major roads has been investigated in both long-term and short-term mortality studies. Long-term studies track subjects over time and investigate the mortality rates among groups with different levels of exposure to ambient pollutants. Short term studies employ daily variation in ambient concentrations to estimate the daily deaths attributable to air pollution.

Among the long-term studies examining adult mortality conducted in Europe and Canada, two have employed the same exposure assessment approach and found similar magnitude of premature risk of mortality associated living near major roads. Canadian vehicles and emission standards largely mirror the U.S. vehicle fleet. Both studies defined living near a major road as having a residence within 100 meters of a highway or within 50 meters of a major urban roadway. In the first study, involving approximately 5,000 people over 55 years old living throughout the Netherlands, residence near major roadways was associated with a 41% increase in the mortality rate from all causes and a 95% increase in the cardiopulmonary mortality rate.³³⁵ The second study involved over 5,200 subjects aged 40 years or more, all living in the Hamilton, Ontario area.³³⁶ This study examined total mortality only, finding a statistically significant 18% increase associated with living near a major roadway. No difference in response was found

among those with pre-existing respiratory illness. The study also calculated “rate advancement periods,” which describe the effect of an exposure in terms of the time period by which exposed persons reach prematurely the same disease risk as unexposed persons reach later on. The rate advancement period for total mortality was 2.5 years. The rate advancement periods were also calculated for other risk factors for mortality, including chronic pulmonary disease excluding asthma (3.4 years), chronic ischemic heart disease (3.1 years), and diabetes mellitus (4.4 years). Despite differences in the vehicle fleets of Europe and Canada, whose emission standards largely mirror those of the U.S., the results of these studies are similar.

In a study involving nearly 190,000 stroke deaths in 1990-1992, Maheswaran and Elliott (2002) examined stroke mortality rates in census districts throughout England and Wales.³³⁷ Census districts closest to major roads showed significant increases in stroke mortality rates for men and women. Compared to those living in census districts whose center was greater than 1000 m from a main road, men and women living in census regions with centers less than 200 m away had stroke mortality rates 7% and 4% higher, respectively.

One study from the Netherlands used time-series analysis to evaluate the change in the magnitude of the association between daily concentrations of black smoke, an air metric related to black carbon, and daily deaths, for populations living along roads with at least 10,000 vehicles per day.³³⁸ Compared with the population living elsewhere, the traffic-exposed population had significantly higher associations between black smoke and daily mortality.

Although the studies of mortality have employed different study designs and metrics of exposure, they provide evidence for increased mortality rates in proximity of heavy traffic. In evaluating the generalizability of these study results, questions remain regarding differences in housing stock, residential ventilation, vehicle type and fuel differences, personal activity patterns, and the appropriate exposure metric.

3.5.2 Non-Allergic Respiratory Symptoms

Studies in Europe, Asia and North America have found increased risk of respiratory symptoms such as wheeze, cough, chronic phlegm production, and dyspnea (shortness of breath) in children and adults with increased proximity to roadways and/or associated with local traffic density. Most of these studies were cross-sectional and relied solely on questionnaire assessments of health outcomes, in combination with simple exposure indicators. There are a large number of studies available, but for the sake of brevity, only studies conducted in the United States are discussed here. European studies reach similar conclusions, as summarized in a recent review of the European literature.³³⁹

Most recently, a study from Cincinnati, OH examined the prevalence of wheezing in a group of infants less than one year of age.³⁴⁰ Infants with at least one atopic parent qualified for enrollment. The study compared infants living near stop-and-go truck traffic with others living near smoothly-flowing truck traffic, and others further from traffic. Infants with wheeze were significantly more likely to live near stop-and-go traffic than either those living near smoothly-flowing traffic or those living away from traffic. Truck volume was not associated with wheeze.

A respiratory health study in the east San Francisco Bay area looked at a series of community schools upwind and downwind of major roads along a major transportation corridor, where ambient air quality was monitored.³⁴¹ Over 1,100 children in grades three through five attending the schools were assessed for respiratory symptoms and physician's diagnosis of asthma. Overall, concentrations of traffic-related air pollutants measured at each school were associated with increased prevalence of bronchitis symptoms and physician confirmed asthma, both within the last 12 months.

A case-control study in Erie County, NY compared home proximity to traffic among children admitted into local hospitals for asthma with those admitted for non-respiratory conditions.³⁴² Overall, children hospitalized for asthma were more likely to live within 200 meters of roads above the 90th percentile of daily vehicle miles traveled, and to have trucks and trailers passing within 200 meters of their residences. However, hospitalization for asthma was not associated with residential distance from major state routes.

A study in San Diego County, CA compared the residential location of asthmatic children with children having a non-respiratory diagnosis within the state Medicaid system.³⁴³ Traffic volumes on streets nearby the home were not associated with the prevalence of asthma. However, among asthmatic children, high street volumes on the nearest street were associated with an increased annual frequency of medical visits for asthma.

In the only U.S. study examining adult respiratory symptoms, Massachusetts veterans were evaluated for traffic-health relationships.³⁴⁴ In the study, living within 50 m of a major roadway was associated with increased reporting of persistent wheeze. This trend held only for roads with at least 10,000 vehicles per day. Patients experiencing chronic phlegm were also more likely to live within 50 meters of roads with at least 10,000 vehicles per day. However, chronic cough was not associated with living near traffic.

Earlier studies have also shown associations between traffic-related exposures and respiratory symptoms. In a study conducted in Kanawha County, WV, outdoor concentrations of petroleum-related volatile organic compounds at 74 elementary schools in the area were associated with chronic lower respiratory symptoms in school children.³⁴⁵

In addition, numerous other studies from around the world also show consistent evidence for increased prevalence of respiratory symptoms among people living near major roads. For a detailed listing, refer to the docket of this proposal. Taken together with the studies presented here, these studies provide substantial evidence that respiratory symptoms are associated with living near major roadways, particularly in children, upon whom the preponderance of studies have focused.

3.5.3 Development of Allergic Disease and Asthma

A significant number of studies have examined evidence of a role of traffic-generated pollution in the development (e.g. new onset) of atopic illnesses (i.e., hypersensitivity to allergens), such as asthma, allergic rhinitis, and dermatitis. A critical review of evidence, primarily generated in European studies, was recently published.³⁴⁶ Overall, the review

concluded that there is some limited evidence of an association between traffic-generated pollutants and asthma incidence. Toxicological evidence provides some evidence that particles from diesel engine exhaust may serve as adjuvants to IgE-mediated immune responses. EPA's Health Assessment Document for Diesel Engine Exhaust addresses many of the toxicological studies on diesel exhaust. However, in community epidemiology studies, the evidence remains tentative, and should not be viewed as conclusive.

As noted above in Section 3.5.2, a recent study in the eastern San Francisco Bay area found pollutant concentrations measured at community schools to be associated with increased asthma prevalence at the school.³⁴⁷

The most recent epidemiologic evidence on the development of asthma associated with traffic derives from two studies. One is a cohort study involving children in 12 southern California communities³⁴⁸ and the other is a cross-sectional study of atopic illness and potentially related metrics in Germany.³⁴⁹ In the southern California study, individual residential ambient concentrations were estimated using different exposure methods: residential outdoor measurements, modeled NO₂ using a roadway-specific dispersion model, and distance from a freeway. NO₂ was used as a general indicator of traffic-related pollution. Overall, lifetime history of physician-diagnosed asthma was significantly associated with outdoor home NO₂, modeled NO₂, and with proximity to freeways. Significant associations with all exposure metrics were also found for recent wheezing, wheezing during exercise, and current use of asthma medication. The German study was based on a population-based sample of over 7,000 subjects. Home traffic intensity and respiratory health history was assessed by questionnaire. Traffic was categorized into three categories. Overall, adjusted for individual-level covariates, living in a high-traffic area was significantly associated with a lifetime history of chronic bronchitis. Subjects with nocturnal coughing, hay fever, and wheezing in the past 12 months had a greater likelihood of living near heavy traffic, but the association was not statistically significant.

One additional study provides more evidence for the role of combustion products in the development of asthma, and points to the complexities involved in epidemiologic studies of asthma onset. The study based in New York City tracked a birth cohort from the third trimester of pregnancy through 24 months of age.³⁵⁰ During their third trimesters, mothers carried samplers analyzed for vapor phase and gas phase PAHs. In the population, total PAH concentration was associated with time spent outdoors.³⁵¹ An interaction between maternal prenatal PAH exposure concentrations and exposure to environmental tobacco smoke (ETS) was reported. Children exposed to ETS postnatally showed a significant, positive association between prenatal PAH exposure and respiratory symptoms. A significant positive exposure-response relationship was also observed for probable asthma at 24 months. Study authors attributed these results to diesel exhaust in the study area. However, infants without ETS exposures at home did not have a significant association between PAH exposures and reported respiratory symptoms or probable asthma. Given that maternal PAH concentrations were not associated with reported ETS or blood cotinine, this study provides evidence that the development of asthma is likely attributable to multiple environmental factors, in addition to whatever genetic predispositions may be present.

Overall, although some of these studies suggest that elevated exposure to traffic-generated pollutants may be associated with development of atopic illnesses such as asthma, the evidence is not universal enough to allow a conclusive statement regarding such an association.

3.5.4 Cardiovascular Effects

Several studies have provided suggestive evidence that exposure to fresh emissions from traffic predispose people to adverse cardiovascular events. Studies have focused on both short-term variations in exposure, as well as long-term residential history. As discussed in the summary section below, there are stressors in the roadway environment in addition to ambient air pollutants (e.g., noise, anxiety) that also have an impact on cardiovascular activity. The potential role of these co-stressors has not been adequately investigated.

A study from Augsburg, Germany interviewed survivors of myocardial infarction (MI) shortly after they had recovered to examine ambient pollution and activities that might predispose someone to having a heart attack.³⁵² Survivors of MI were nearly three times as likely to be in a car, in transit, or on a bicycle in the hour prior to the event as they were to be in traffic at other times. Ambient air pollutants measured in the hour prior to MI at a central site in the city were not associated with the risk of MI.

A study of healthy young North Carolina state patrolmen conducted by EPA's Office of Research and Development monitored in-vehicle concentrations of PM_{2.5}, VOCs, and metals.³⁵³ In-vehicle PM_{2.5} concentrations were associated with altered heart rate variability, an indicator of cardiac stress. In-vehicle concentrations were also associated with increased concentrations of factors in the blood associated with long-term cardiac risk, such as C-reactive protein, an indicator of inflammation. This study provides information on possible mechanisms by which cardiac stress could be induced by exposures to traffic-generated air pollution.

Heart rate variability has also been measured in a study of elderly residents of the Boston area.³⁵⁴ In the study, ambient PM_{2.5} was associated with changes consistent with reduced autonomic control of the heart. However, black carbon, often a more reliable index of traffic-related pollution, was more strongly associated with these changes. In a related study, ST-segment depression, a cardiographic indicator of cardiac ischemia or inflammation, was associated with black carbon levels as well.³⁵⁵

In addition to these short-term studies, several of the previously-mentioned mortality studies focus on specific causes of death. The Dutch mortality study, mentioned in Section 3.5.1, found higher relative risks for cardiopulmonary causes of death. Furthermore, a reanalysis of the Canadian study mentioned previously has found that mortality rates for diseases of the circulatory system were significantly associated with living near a major road.³⁵⁶

3.5.5 Birth Outcomes

Studies examining birth outcomes in populations living near major traffic sources have generally found evidence of low birth weight, preterm birth, reduced head circumference and heart defects among children of mothers living in close proximity to heavy traffic. However, the

studies to date have focused primarily on one area, Southern California. Other studies with less traffic-specific exposure indices have been conducted elsewhere.

One measure of exposure to traffic-generated pollution is “distance-weighted traffic density,” where traffic volume is treated as a measure that “disperses” along a Gaussian bell-shaped curve evenly on both sides of a roadway. This approach captures some of the patterns of dispersion from line sources, but does not account for micrometeorology. One study from Los Angeles County, California employed this metric in a study of birth outcomes for births from 1994 to 1996. The study showed associations between distance-weighted traffic volume near women’s residences during pregnancy and premature birth and low birth weight in their babies.³⁵⁷ The elevated risks occurred primarily for mothers whose third trimesters fell during fall or winter months.

The same researchers had conducted an earlier study of births occurring between 1989 and 1993. In that study, consisting of over 125,000 births, exposures to ambient carbon monoxide (CO), an indicator of traffic pollution, during the third trimester were significantly associated with increased risk of low birth weight.³⁵⁸ In another study, preterm birth was associated with ambient PM₁₀ and CO.³⁵⁹ These authors have also reported in a separate study on the increase in cardiac ventricular septal defects with increasing CO exposure during the second month of pregnancy.³⁶⁰ The role of socioeconomic status and factors associated with it should be investigated in future study design.

Although the exposure metrics employed in these studies are based on surrogate approaches to exposure estimation, other researchers have shown associations between New York mothers’ measured personal exposure to polycyclic aromatic hydrocarbons (PAHs) during pregnancy and an increased risk of low birth weight and size.³⁶¹

Overall, although the number of studies examining perinatal exposures is relatively small, there appears to be consistent evidence that exposure to traffic-related pollutants is associated with adverse birth outcomes, including low birth weight and preterm birth. However, given the variety of exposure metrics employed and the relatively limited geographic extent of studies, the generalization of the conclusions requires a better understanding of relevant sources, pollutants, susceptibility, and local factors.

3.5.6 Childhood Cancer

Several MSATs are associated with cancer in adult populations. However, children have physical and biochemical differences that may affect their susceptibility to and metabolism of MSATs. Particularly in the first year or two after birth, infants’ liver enzyme profiles undergo rapid change. As such, children may respond to MSATs in different ways from adults. Some evidence exists that children may face different cancer risks from adults as a result of exposure to certain MSATs and other components of motor vehicle exhaust. EPA recently recommended default adjustments to cancer risk estimates to account for early life exposures in the Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens.³⁶²

Furthermore, evidence from human and animal studies suggests that increases in childhood leukemia may be associated with in utero exposures to benzene and maternal and paternal exposure prior to conception. Furthermore, there is mounting evidence that key changes related to the development of childhood leukemia occur in the developing fetus.³⁶³

In the last 15 years, several studies have evaluated the association between maternal or childhood residence near busy roads and the risk of cancer in children. Most studies to date have been ecological in nature, with several employing individual-level exposure estimates within cohort designs. The studies employed widely varying exposure metrics, including modeled air quality, proximity to sources, and distance-weighted traffic volumes. Positive studies tend to have used small population sizes, although one recent positive study used a large population. Due to differences in ages studied, study design, exposure metrics, and study location (e.g. Europe vs. U.S.), a systematic comparison between studies is difficult. A description of several key studies from this literature follows.

One early study from Colorado showed significant elevated risk of childhood leukemia in children under age 15 associated with living near roads with higher traffic volumes. The strongest associations were with roads with at least 10,000 vehicles per day.³⁶⁴ The study was reanalyzed using an approach to combine traffic volume with residential distance from major roads to assess “distance-weighted traffic volume.”³⁶⁵ The study found that the significant, monotonically increasing risks associated with increased distance-weighted traffic volume.

Another study used a dispersion model of NO₂ from traffic to conduct a case-control study of childhood cancer in Sweden.³⁶⁶ The study found that in the highest-exposed group, risk of any cancer was significantly elevated. Risks in the most-exposed group were also elevated for leukemia and central nervous system tumors, but were not statistically significant.

These earlier studies were based on relatively small populations of children with cancer. In response, subsequent studies focused on either replicating the earlier studies or studying larger groups of children. A study in Los Angeles, California applied the same distance-weighted traffic volume approach as the earlier Colorado study, but found no elevation in risk in a larger group of children.³⁶⁷ A large study of nearly 2,000 Danish children with cancer found no association between modeled concentrations of benzene and NO₂ at home and the risk of leukemia, central nervous system tumors, or total cancers.³⁶⁸ However, the study did find a dose-dependent relationship between Hodgkin’s disease and modeled air pollution from traffic.

Several large studies were conducted in California using a statewide registry of cancer. These studies employed study sizes of several thousand subjects. In one cross-sectional study, the potency-weighted sum of concentrations of 25 air toxics modeled using EPA’s ASPEN model was not associated with mobile source emissions, but increased rates of childhood leukemia were found when accounting for all sources of air toxics together, and for point sources separately.³⁶⁹ Another study from the same researchers found that roadway density and traffic density within 500 meters of children’s homes was not associated with risk of cancer.³⁷⁰

Most recently, a novel approach to assessing childhood leukemia in relation to early life exposures was employed in the United Kingdom. The study examined all children dying of

cancer between 1955 and 1980, consisting of over 22,000 cases. Birth and death addresses of children with cancer who moved before death were compared with regard to proximity to nearby sources and emissions of specific chemicals.³⁷¹ An excess of births near sources, relative to deaths, was used to indicate sources in early life associated with greatest cancer. Greater risks were associated with birth addresses within 300 meters of high emissions of benzene, 1,3-butadiene, NO_x, PM₁₀, dioxins, and benzo[a]pyrene. In addition, births within 1.0 km of bus stations, hospitals, freight terminals, railways, and oil installations were associated with elevated risk. Overall, locations with the highest emissions of 1,3-butadiene and carbon monoxide showed the greatest risk.

In summary, the lack of consistency in results between large studies and the multiplicity of study designs makes it difficult to draw firm conclusions. Epidemiologic methods for detection of childhood cancer risks may lack sufficient power to detect risks with precision. However, given the well-established carcinogenicity of benzene and 1,3-butadiene in the toxicological and occupational epidemiologic literature, and data suggesting exposure to benzene prior to conception and in utero can lead to increased risk of childhood leukemia, the potential for public health concern is present. The standards proposed in this rule will reduce such exposures.

3.5.7 Summary of Near-Roadway Health Studies

Other significant scientific uncertainties remain in research on health effects near roads, including the exposures of greatest concern, the importance of chronic versus acute exposures, the role of fuel type (e.g. diesel or gasoline) and composition (e.g., % aromatics), and relevant traffic patterns. Research is also needed to understand the role of co-stressors including noise and socioeconomic status (e.g., access to health care, nutritional status), and the role of differential susceptibility within the "exposed" populations near and on-roadways.

Taken together, the available studies of health effects in residents near major roadways highlight more potential health benefits of the emission reductions in this proposal than can currently be quantified.

3.5.8 Size and Characteristics of Populations Living near Major Roads

In assessing the public health implications of near-roadway health concerns, some understanding of the population living near major roads is required. As noted earlier, we conducted a study in three states, Colorado, Georgia, and New York. Geographic information systems were used in the analysis. In Colorado, 22% live within 75 meters of a major road, while an additional 33% live between 75 and 200 meters of major roads. In Georgia, the respective percentages are 17% living within 75 meters and an additional 24% living between 75 and 200 meters. In New York, the percentages are 31% and 36%.

To date, the only source of national data on populations living in close proximity to major transportation sources is the American Housing Survey, conducted by the U.S. Census Bureau.³⁷² This study characterizes the properties and neighborhood characteristics of housing units throughout the U.S. According to the Census Bureau's summaries of the 2003 survey, among

approximately 120,777,000 housing units in the nation, 15,182,000 were within 300 feet of a “4-or-more-lane highway, railroad, or airport.” This constitutes 12.6% of total U.S. housing units. A simple assumption that the U.S. population is uniformly distributed among all types of housing leads to the conclusion that approximately 37.4 million people live in what might be considered a “mobile source hot spot.”

According to the American Housing Survey’s summary tables, occupied housing units in central cities are 35% more likely to be close to major transportation sources than housing units in suburban areas.³⁷³ Furthermore, nationally, housing units that are renter-occupied are 2.3 times more likely to be close to major transportation sources, compared to housing units that are owner-occupied. In the 2003 American Housing Survey, median household income for owner-occupied units was \$52,803, while only \$26,983 for renter-occupied units. These statistics imply that those houses sited near major transportation sources are likely to be lower in income than houses not located near major transportation sources.

A few population-based epidemiology studies have also examined whether discrete groups of people live close to major roadways. In one study of veterans living in southeastern Massachusetts, 23% lived within 50 meters of a “major road,” 33% lived within 100 meters, and 51% within 200 meters.³⁷⁴ In examining traffic volumes, 13% lived within 50 meters of a road with annual average daily traffic of 10,000 vehicles or more, while other distances were not analyzed.

In another study using 150 meters as a definition of “near” a road, 2.3% of California public schools were found to be near a road with more than 50,000 vehicles per day, while 7.2% were near roads with between 25,000 and 49,999 vehicles per day.³⁷⁵ This corresponded to 2.6% and 9.8% of total enrollment, respectively. In that study, traffic exposure increased, the fractions of school populations comprised of black and Hispanic students also increased, as did the fraction of children in government-subsidized meal programs.

Another study in California defined the issue differently, examining the child population living in census block groups and traffic density.³⁷⁶ The study found that approximately 3% of the state child population resided in the highest traffic density census tracts. Furthermore, block groups with lower income were more likely to have high traffic density. Children of color were more likely than white children to live in high traffic density areas.

In summary, a substantial fraction of the U.S. population lives within approximately 200 meters of major roads.

Appendix 3A

Table 3A-1. 8-Hour Ozone Nonattainment Areas and Populations (Data is Current through April 2005 and Population Numbers are from 2000 Census Data)

8-hour Ozone Nonattainment Areas	Population	2001-2003 8-hr Design Value (ppb)	Category / Classification^{a,c,d}	Maximum Attainment Date^b
Albany-Schenectady-Troy, NY	923,778	87	Subpart 1	Jun. 15, 2009
Allegan Co, MI	105,665	97	Subpart 1	Jun. 15, 2009
Allentown-Bethlehem-Easton, PA	637,958	91	Subpart 1	Jun. 15, 2009
Altoona, PA	129,144	85	Subpart 1	Jun. 15, 2009
Amador and Calaveras Cos (Central Mtn), CA	75,654	91	Subpart 1	Jun. 15, 2009
Atlanta, GA	4,228,492	91	Marginal	Jun. 15, 2007
Baltimore, MD	2,512,431	103	Moderate	Jun. 15, 2010
Baton Rouge, LA	636,214	86	Marginal	Jun. 15, 2007
Beaumont-Port Arthur, TX	385,090	91	Marginal	Jun. 15, 2007
Benton Harbor, MI	162,453	91	Subpart 1	Jun. 15, 2009
Benzie Co, MI	15,998	88	Subpart 1	Jun. 15, 2009
Berkeley and Jefferson Counties, WV	118,095	86	Subpart 1 EAC	Dec. 31, 2007
Birmingham, AL	805,340	87	Subpart 1	Jun. 15, 2009
Boston-Lawrence-Worcester (E. MA), MA	5,534,130	95	Moderate	Jun. 15, 2010
Boston-Manchester-Portsmouth(SE),NH	696,713	95	Moderate	Jun. 15, 2010
Buffalo-Niagara Falls, NY	1,170,111	99	Subpart 1	Jun. 15, 2009
Canton-Massillon, OH	378,098	90	Subpart 1	Jun. 15, 2009
Cass Co, MI	51,104	93	Marginal	Jun. 15, 2007
Charleston, WV	251,662	86	Subpart 1	Jun. 15, 2009
Charlotte-Gastonia-Rock Hill, NC-SC	1,476,564	100	Moderate	Jun. 15, 2010
Chattanooga, TN-GA	372,264	88	Subpart 1 EAC	Dec. 31, 2007
Chicago-Gary-Lake County, IL-IN	8,757,808	101	Moderate	Jun. 15, 2010
Chico, CA	203,171	89	Subpart 1	Jun. 15, 2009
Cincinnati-Hamilton, OH-KY-IN	1,891,518	96	Subpart 1	Jun. 15, 2009
Clarksville-Hopkinsville, TN-KY	207,033	85	Subpart 1	Jun. 15, 2009
Clearfield and Indiana Cos, PA	172,987	90	Subpart 1	Jun. 15, 2009
Cleveland-Akron-Lorain, OH	2,945,831	103	Moderate	Jun. 15, 2010
Columbia, SC	494,518	89	Subpart 1 EAC	Dec. 31, 2007
Columbus, OH	1,541,930	95	Subpart 1	Jun. 15, 2009

8-hour Ozone Nonattainment Areas	Population	2001-2003 8-hr Design Value (ppb)	Category / Classification^{a,c,d}	Maximum Attainment Date^b
Dallas-Fort Worth, TX	5,030,828	100	Moderate	Jun. 15, 2010
Dayton-Springfield, OH	950,558	90	Subpart 1	Jun. 15, 2009
Denver-Boulder- Greeley-Ft Collins-Love., CO	2,811,580	87	Subpart 1 EAC	Dec. 31, 2007
Detroit-Ann Arbor, MI	4,932,383	97	Marginal	Jun. 15, 2007
Door Co, WI	27,961	94	Subpart 1	Jun. 15, 2009
Erie, PA	280,843	92	Subpart 1	Jun. 15, 2009
Essex Co (Whiteface Mtn), NY	1,000	91	Subpart 1	Jun. 15, 2009
Evansville, IN	224,305	85	Subpart 1	Jun. 15, 2009
Fayetteville, NC	302,963	87	Subpart 1 EAC	Dec. 31, 2007
Flint, MI	524,045	90	Subpart 1	Jun. 15, 2009
Fort Wayne, IN	331,849	88	Subpart 1	Jun. 15, 2009
Franklin Co, PA	129,313	93	Subpart 1	Jun. 15, 2009
Frederick Co, VA	82,794	85	Subpart 1 EAC	Dec. 31, 2007
Fredericksburg, VA	202,120	99	Moderate	Jun. 15, 2010
Grand Rapids, MI	812,649	89	Subpart 1	Jun. 15, 2009
Greater Connecticut, CT	1,543,919	95	Moderate	Jun. 15, 2010
Greene Co, IN	33,157	88	Subpart 1	Jun. 15, 2009
Greene Co, PA	40,672	89	Subpart 1	Jun. 15, 2009
Greensboro-Winston Salem-High Point, NC	1,285,879	93	Marginal EAC	Dec. 31, 2007
Greenville-Spartanburg- Anderson, SC	799,147	87	Subpart 1 EAC	Dec. 31, 2007
Hancock, Knox, Lincoln & Waldo Cos, ME	92,476	94	Subpart 1	Jun. 15, 2009
Harrisburg-Lebanon- Carlisle, PA	629,401	88	Subpart 1	Jun. 15, 2009
Haywood and Swain Cos (Great Smoky NP), NC	288	85	Subpart 1	Jun. 15, 2009
Hickory-Morganton- Lenoir, NC	309,512	88	Subpart 1 EAC	Dec. 31, 2007
Houston-Galveston- Brazoria, TX	4,669,571	102	Moderate	Jun. 15, 2010
Huntington-Ashland, WV-KY	189,439	91	Subpart 1	Jun. 15, 2009
Huron Co, MI	36,079	87	Subpart 1	Jun. 15, 2009
Imperial Co, CA	142,361	87	Marginal	Jun. 15, 2007
Indianapolis, IN	1,607,486	96	Subpart 1	Jun. 15, 2009
Jackson Co, IN	41,335	85	Subpart 1	Jun. 15, 2009
Jamestown, NY	139,750	94	Subpart 1	Jun. 15, 2009
Jefferson Co, NY	111,738	97	Moderate	Jun. 15, 2010
Johnson City-Kingsport- Bristol, TN	206,611	86	Subpart 1 EAC	Dec. 31, 2007
Johnstown, PA	152,598	87	Subpart 1	Jun. 15, 2009
Kalamazoo-Battle Creek, MI	452,851	86	Subpart 1	Jun. 15, 2009

8-hour Ozone Nonattainment Areas	Population	2001-2003 8-hr Design Value (ppb)	Category / Classification^{a,c,d}	Maximum Attainment Date^b
Kent and Queen Anne's Cos, MD	59,760	95	Marginal	Jun. 15, 2007
Kern Co (Eastern Kern), CA	99,251	98	Subpart 1	Jun. 15, 2009
Kewaunee Co, WI	20,187	93	Subpart 1	Jun. 15, 2009
Knoxville, TN	713,755	92	Subpart 1	Jun. 15, 2009
La Porte, IN	110,106	93	Marginal	Jun. 15, 2007
Lancaster, PA	470,658	92	Marginal	Jun. 15, 2007
Lansing-East Lansing, MI	447,728	86	Subpart 1	Jun. 15, 2009
Las Vegas, NV	1,348,864	86	Subpart 1	Jun. 15, 2009
Lima, OH	108,473	89	Subpart 1	Jun. 15, 2009
Los Angeles South Coast Air Basin, CA	14,593,587	131	Severe 17	Jun. 15, 2021
Los Angeles-San Bernardino Cos(W Mojave),CA	656,408	106	Moderate	Jun. 15, 2010
Louisville, KY-IN	968,313	92	Subpart 1	Jun. 15, 2009
Macon, GA	153,937	86	Subpart 1	Jun. 15, 2009
Madison and Page Cos (Shenandoah NP), VA	2	87	Subpart 1	Jun. 15, 2009
Manitowoc Co, WI	82,887	90	Subpart 1	Jun. 15, 2009
Mariposa and Tuolumne Cos (Southern Mtn),CA	71,631	91	Subpart 1	Jun. 15, 2009
Mason Co, MI	28,274	89	Subpart 1	Jun. 15, 2009
Memphis, TN-AR	948,338	92	Marginal	Jun. 15, 2007
Milwaukee-Racine, WI	1,839,149	101	Moderate	Jun. 15, 2010
Muncie, IN	118,769	88	Subpart 1	Jun. 15, 2009
Murray Co (Chattahoochee Nat Forest), GA	1,000	85	Subpart 1	Jun. 15, 2009
Muskegon, MI	170,200	95	Marginal	Jun. 15, 2007
Nashville, TN	1,097,810	86	Subpart 1 EAC	Dec. 31, 2007
Nevada Co. (Western Part), CA	77,735	98	Subpart 1	Jun. 15, 2009
New York-N. New Jersey-Long Island,NY- NJ-CT	19,634,122	102	Moderate	Jun. 15, 2010
Norfolk-Virginia Beach- Newport News (HR),VA	1,542,144	90	Marginal	Jun. 15, 2007
Parkersburg-Marietta, WV-OH	151,237	87	Subpart 1	Jun. 15, 2009
Philadelphia-Wilmin- Atlantic Ci,PA-NJ-MD- DE	7,333,475	106	Moderate	Jun. 15, 2010
Phoenix-Mesa, AZ	3,086,045	87	Subpart 1	Jun. 15, 2009
Pittsburgh-Beaver Valley, PA	2,431,087	94	Subpart 1	Jun. 15, 2009
Portland, ME	456,508	91	Marginal	Jun. 15, 2007

8-hour Ozone Nonattainment Areas	Population	2001-2003 8-hr Design Value (ppb)	Category / Classification^{a,c,d}	Maximum Attainment Date^b
Poughkeepsie, NY	717,262	94	Moderate	Jun. 15, 2010
Providence (All RI), RI	1,048,319	95	Moderate	Jun. 15, 2010
Raleigh-Durham-Chapel Hill, NC	1,244,053	94	Subpart 1	Jun. 15, 2009
Reading, PA	373,638	91	Subpart 1	Jun. 15, 2009
Richmond-Petersburg, VA	919,277	94	Marginal	Jun. 15, 2007
Riverside Co, (Coachella Valley), CA	324,750	108	Serious	Jun. 15, 2013
Roanoke, VA	235,932	85	Subpart 1 EAC	Dec. 31, 2007
Rochester, NY	1,098,201	88	Subpart 1	Jun. 15, 2009
Rocky Mount, NC	143,026	89	Subpart 1	Jun. 15, 2009
Sacramento Metro, CA	1,978,348	107	Serious	Jun. 15, 2013
San Antonio, TX	1,559,975	89	Subpart 1 EAC	Dec. 31, 2007
San Diego, CA	2,813,431	93	Subpart 1	Jun. 15, 2009
San Francisco Bay Area, CA	6,541,828	86	Marginal	Jun. 15, 2007
San Joaquin Valley, CA	3,191,367	115	Serious	Jun. 15, 2013
Scranton-Wilkes-Barre, PA	699,312	86	Subpart 1	Jun. 15, 2009
Sheboygan, WI	112,646	100	Moderate	Jun. 15, 2010
South Bend-Elkhart, IN	448,350	93	Subpart 1	Jun. 15, 2009
Springfield (Western MA), MA	814,967	94	Moderate	Jun. 15, 2010
St Louis, MO-IL	2,504,603	92	Moderate	Jun. 15, 2010
State College, PA	135,758	88	Subpart 1	Jun. 15, 2009
Steubenville-Weirton, OH-WV	132,008	86	Subpart 1	Jun. 15, 2009
Sutter Co (Sutter Buttes), CA	1	88	Subpart 1	Jun. 15, 2009
Terre Haute, IN	105,848	87	Subpart 1	Jun. 15, 2009
Tioga Co, PA	41,373	86	Subpart 1	Jun. 15, 2009
Toledo, OH	576,119	93	Subpart 1	Jun. 15, 2009
Ventura Co, CA	753,197	95	Moderate	Jun. 15, 2010
Washington Co (Hagerstown), MD	131,923	86	Subpart 1 EAC	Dec. 31, 2007
Washington, DC-MD-VA	4,452,498	99	Moderate	Jun. 15, 2010
Wheeling, WV-OH	153,172	87	Subpart 1	Jun. 15, 2009
York, PA	473,043	89	Subpart 1	Jun. 15, 2009
Youngstown-Warren-Sharon, OH-PA	715,039	95	Subpart 1	Jun. 15, 2009
Total	159,271,919			

a) Under the CAA these nonattainment areas are further classified as subpart 1 or subpart 2 (subpart 2 is further classified as marginal, moderate, serious, severe or extreme) based on their design values. An Early Action Compact (EAC) area is one that has entered into a compact with the EPA and have agreed to reduce ground level ozone pollution earlier than the CAA would require in exchange the EPA will defer the effective date of the nonattainment designation. The severe designation is denoted as severe-15 or severe-17 based on the maximum attainment date associated with the classification.

b) The nonattainment areas covered under subpart 1 will be required to attain the standard no later than 5 years after designation and, in limited circumstances; they may apply for an additional extension of up to 5 years (e.g., 2009 to 2014). The areas classified under subpart 2 have attainment dates ranging from up to 3 years for marginal areas (2007) to up to 20 years for extreme areas (2024).

c) Boston-Manchester-Portsmouth (SE), NH has the same classification as Boston-Lawrence- Worcester (E. MA), MA.

d) Fredericksburg, VA has the same classification as Washington, DC-MD-VA.

Table 3A-2. 8-Hour Ozone Nonattainment Counties and Populations (Data is Current through April 13, 2005 and Population Numbers are from 2000 Census Data)

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
AL	Jefferson Co	W	662,047
AL	Shelby Co	W	143,293
AZ	Maricopa Co	P	3,054,504
AZ	Pinal Co	P	31,541
AR	Crittenden Co	W	50,866
CA	Alameda Co	W	1,443,741
CA	Amador Co	W	35,100
CA	Butte Co	W	203,171
CA	Calaveras Co	W	40,554
CA	Contra Costa Co	W	948,816
CA	El Dorado Co	P	124,164
CA	Fresno Co	W	799,407
CA	Imperial Co	W	142,361
CA	Kern Co	P	649,471
CA	Kings Co	W	129,461
CA	Los Angeles Co	P	9,519,338
CA	Madera Co	W	123,109
CA	Marin Co	W	247,289
CA	Mariposa Co	W	17,130
CA	Merced Co	W	210,554
CA	Napa Co	W	124,279
CA	Nevada Co	P	77,735
CA	Orange Co	W	2,846,289
CA	Placer Co	P	239,978
CA	Riverside Co	P	1,519,609
CA	Sacramento Co	W	1,223,499
CA	San Bernardino Co	P	1,689,509
CA	San Diego Co	P	2,813,431
CA	San Francisco Co	W	776,733
CA	San Joaquin Co	W	563,598
CA	San Mateo Co	W	707,161
CA	Santa Clara Co	W	1,682,585
CA	Solano Co	P	394,542
CA	Sonoma Co	P	413,716
CA	Stanislaus Co	W	446,997
CA	Sutter Co	P	25,014
CA	Tulare Co	W	368,021
CA	Tuolumne Co	W	54,501
CA	Ventura Co	P	753,197
CA	Yolo Co	W	168,660
CO	Adams Co	W	348,618
CO	Arapahoe Co	W	487,967

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
CO	Boulder Co	W	269,814
CO	Broomfield Co	W	38,272
CO	Denver Co	W	554,636
CO	Douglas Co	W	175,766
CO	Jefferson Co	W	525,507
CO	Larimer Co	P	239,000
CO	Weld Co	P	172,000
CT	Fairfield Co	W	882,567
CT	Hartford Co	W	857,183
CT	Litchfield Co	W	182,193
CT	Middlesex Co	W	155,071
CT	New Haven Co	W	824,008
CT	New London Co	W	259,088
CT	Tolland Co	W	136,364
CT	Windham Co	W	109,091
DE	Kent Co	W	126,697
DE	New Castle Co	W	500,265
DE	Sussex Co	W	156,638
DC	Entire District	W	572,059
GA	Barrow Co	W	46,144
GA	Bartow Co	W	76,019
GA	Bibb Co	W	153,887
GA	Carroll Co	W	87,268
GA	Catoosa Co	W	53,282
GA	Cherokee Co	W	141,903
GA	Clayton Co	W	236,517
GA	Cobb Co	W	607,751
GA	Coweta Co	W	89,215
GA	De Kalb Co	W	665,865
GA	Douglas Co	W	92,174
GA	Fayette Co	W	91,263
GA	Forsyth Co	W	98,407
GA	Fulton Co	W	816,006
GA	Gwinnett Co	W	588,448
GA	Hall Co	W	139,277
GA	Henry Co	W	119,341
GA	Monroe Co	P	50
GA	Murray Co	P	1,000
GA	Newton Co	W	62,001
GA	Paulding Co	W	81,678
GA	Rockdale Co	W	70,111
GA	Spalding Co	W	58,417
GA	Walton Co	W	60,687
IL	Cook Co	W	5,376,741
IL	Du Page Co	W	904,161
IL	Grundy Co	P	6,309

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
IL	Jersey Co	W	21,668
IL	Kane Co	W	404,119
IL	Kendall Co	P	28,417
IL	Lake Co	W	644,356
IL	Mc Henry Co	W	260,077
IL	Madison Co	W	258,941
IL	Monroe Co	W	27,619
IL	St Clair Co	W	256,082
IL	Will Co	W	502,266
IN	Allen Co	W	331,849
IN	Boone Co	W	46,107
IN	Clark Co	W	96,472
IN	Dearborn Co	P	10,434
IN	Delaware Co	W	118,769
IN	Elkhart Co	W	182,791
IN	Floyd Co	W	70,823
IN	Greene Co	W	33,157
IN	Hamilton Co	W	182,740
IN	Hancock Co	W	55,391
IN	Hendricks Co	W	104,093
IN	Jackson Co	W	41,335
IN	Johnson Co	W	115,209
IN	Lake Co	W	484,564
IN	La Porte Co	W	110,106
IN	Madison Co	W	133,358
IN	Marion Co	W	860,454
IN	Morgan Co	W	66,689
IN	Porter Co	W	146,798
IN	St Joseph Co	W	265,559
IN	Shelby Co	W	43,445
IN	Vanderburgh Co	W	171,922
IN	Vigo Co	W	105,848
IN	Warrick Co	W	52,383
KY	Boone Co	W	85,991
KY	Boyd Co	W	49,752
KY	Bullitt Co	W	61,236
KY	Campbell Co	W	88,616
KY	Christian Co	W	72,265
KY	Jefferson Co	W	693,604
KY	Kenton Co	W	151,464
KY	Oldham Co	W	46,178
LA	Ascension Par	W	76,627
LA	East Baton Rouge Par	W	412,852
LA	Iberville Par	W	33,320
LA	Livingston Par	W	91,814
LA	West Baton Rouge Par	W	21,601

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
ME	Androscoggin Co	P	3,390
ME	Cumberland Co	P	252,907
ME	Hancock Co	P	29,805
ME	Knox Co	P	33,563
ME	Lincoln Co	P	28,504
ME	Sagadahoc Co	W	35,214
ME	Waldo Co	P	604
ME	York Co	P	164,997
MD	Anne Arundel Co	W	489,656
MD	Baltimore Co	W	754,292
MD	Calvert Co	W	74,563
MD	Carroll Co	W	150,897
MD	Cecil Co	W	85,951
MD	Charles Co	W	120,546
MD	Frederick Co	W	195,277
MD	Harford Co	W	218,590
MD	Howard Co	W	247,842
MD	Kent Co	W	19,197
MD	Montgomery Co	W	873,341
MD	Prince George's Co	W	801,515
MD	Queen Anne's Co	W	40,563
MD	Washington Co	W	131,923
MD	Baltimore (City)	W	651,154
MA	Barnstable Co	W	222,230
MA	Berkshire Co	W	134,953
MA	Bristol Co	W	534,678
MA	Dukes Co	W	14,987
MA	Essex Co	W	723,419
MA	Franklin Co	W	71,535
MA	Hampden Co	W	456,228
MA	Hampshire Co	W	152,251
MA	Middlesex Co	W	1,465,396
MA	Nantucket Co	W	9,520
MA	Norfolk Co	W	650,308
MA	Plymouth Co	W	472,822
MA	Suffolk Co	W	689,807
MA	Worcester Co	W	750,963
MI	Allegan Co	W	105,665
MI	Benzie Co	W	15,998
MI	Berrien Co	W	162,453
MI	Calhoun Co	W	137,985
MI	Cass Co	W	51,104
MI	Clinton Co	W	64,753
MI	Eaton Co	W	103,655
MI	Genesee Co	W	436,141
MI	Huron Co	W	36,079

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
MI	Ingham Co	W	279,320
MI	Kalamazoo Co	W	238,603
MI	Kent Co	W	574,335
MI	Lapeer Co	W	87,904
MI	Lenawee Co	W	98,890
MI	Livingston Co	W	156,951
MI	Macomb Co	W	788,149
MI	Mason Co	W	28,274
MI	Monroe Co	W	145,945
MI	Muskegon Co	W	170,200
MI	Oakland Co	W	1,194,156
MI	Ottawa Co	W	238,314
MI	St Clair Co	W	164,235
MI	Van Buren Co	W	76,263
MI	Washtenaw Co	W	322,895
MI	Wayne Co	W	2,061,162
MO	Franklin Co	W	93,807
MO	Jefferson Co	W	198,099
MO	St Charles Co	W	283,883
MO	St Louis Co	W	1,016,315
MO	St Louis	W	348,189
NV	Clark Co	P	1,348,864
NH	Hillsborough Co	P	336,518
NH	Merrimack Co	P	11,721
NH	Rockingham Co	P	266,340
NH	Strafford Co	P	82,134
NJ	Atlantic Co	W	252,552
NJ	Bergen Co	W	884,118
NJ	Burlington Co	W	423,394
NJ	Camden Co	W	508,932
NJ	Cape May Co	W	102,326
NJ	Cumberland Co	W	146,438
NJ	Essex Co	W	793,633
NJ	Gloucester Co	W	254,673
NJ	Hudson Co	W	608,975
NJ	Hunterdon Co	W	121,989
NJ	Mercer Co	W	350,761
NJ	Middlesex Co	W	750,162
NJ	Monmouth Co	W	615,301
NJ	Morris Co	W	470,212
NJ	Ocean Co	W	510,916
NJ	Passaic Co	W	489,049
NJ	Salem Co	W	64,285
NJ	Somerset Co	W	297,490
NJ	Sussex Co	W	144,166
NJ	Union Co	W	522,541

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
NJ	Warren Co	W	102,437
NY	Albany Co	W	294,565
NY	Bronx Co	W	1,332,650
NY	Chautauqua Co	W	139,750
NY	Dutchess Co	W	280,150
NY	Erie Co	W	950,265
NY	Essex Co	P	1,000
NY	Genesee Co	W	60,370
NY	Greene Co	W	48,195
NY	Jefferson Co	W	111,738
NY	Kings Co	W	2,465,326
NY	Livingston Co	W	64,328
NY	Monroe Co	W	735,343
NY	Montgomery Co	W	49,708
NY	Nassau Co	W	1,334,544
NY	New York Co	W	1,537,195
NY	Niagara Co	W	219,846
NY	Ontario Co	W	100,224
NY	Orange Co	W	341,367
NY	Orleans Co	W	44,171
NY	Putnam Co	W	95,745
NY	Queens Co	W	2,229,379
NY	Rensselaer Co	W	152,538
NY	Richmond Co	W	443,728
NY	Rockland Co	W	286,753
NY	Saratoga Co	W	200,635
NY	Schenectady Co	W	146,555
NY	Schoharie Co	W	31,582
NY	Suffolk Co	W	1,419,369
NY	Wayne Co	W	93,765
NY	Westchester Co	W	923,459
NC	Alamance Co	W	130,800
NC	Alexander Co	W	33,603
NC	Burke Co	P	69,970
NC	Cabarrus Co	W	131,063
NC	Caldwell Co	P	64,254
NC	Caswell Co	W	23,501
NC	Catawba Co	W	141,685
NC	Chatham Co	P	21,320
NC	Cumberland Co	W	302,963
NC	Davidson Co	W	147,246
NC	Davie Co	W	34,835
NC	Durham Co	W	223,314
NC	Edgecombe Co	W	55,606
NC	Forsyth Co	W	306,067
NC	Franklin Co	W	47,260

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
NC	Gaston Co	W	190,365
NC	Granville Co	W	48,498
NC	Guilford Co	W	421,048
NC	Haywood Co	P	28
NC	Iredell Co	P	39,885
NC	Johnston Co	W	121,965
NC	Lincoln Co	W	63,780
NC	Mecklenburg Co	W	695,454
NC	Nash Co	W	87,420
NC	Orange Co	W	118,227
NC	Person Co	W	35,623
NC	Randolph Co	W	130,454
NC	Rockingham Co	W	91,928
NC	Rowan Co	W	130,340
NC	Swain Co	P	260
NC	Union Co	W	123,677
NC	Wake Co	W	627,846
OH	Allen Co	W	108,473
OH	Ashtabula Co	W	102,728
OH	Belmont Co	W	70,226
OH	Butler Co	W	332,807
OH	Clark Co	W	144,742
OH	Clermont Co	W	177,977
OH	Clinton Co	W	40,543
OH	Columbiana Co	W	112,075
OH	Cuyahoga Co	W	1,393,978
OH	Delaware Co	W	109,989
OH	Fairfield Co	W	122,759
OH	Franklin Co	W	1,068,978
OH	Geauga Co	W	90,895
OH	Greene Co	W	147,886
OH	Hamilton Co	W	845,303
OH	Jefferson Co	W	73,894
OH	Knox Co	W	54,500
OH	Lake Co	W	227,511
OH	Licking Co	W	145,491
OH	Lorain Co	W	284,664
OH	Lucas Co	W	455,054
OH	Madison Co	W	40,213
OH	Mahoning Co	W	257,555
OH	Medina Co	W	151,095
OH	Miami Co	W	98,868
OH	Montgomery Co	W	559,062
OH	Portage Co	W	152,061
OH	Stark Co	W	378,098
OH	Summit Co	W	542,899

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
OH	Trumbull Co	W	225,116
OH	Warren Co	W	158,383
OH	Washington Co	W	63,251
OH	Wood Co	W	121,065
PA	Adams Co	W	91,292
PA	Allegheny Co	W	1,281,666
PA	Armstrong Co	W	72,392
PA	Beaver Co	W	181,412
PA	Berks Co	W	373,638
PA	Blair Co	W	129,144
PA	Bucks Co	W	597,635
PA	Butler Co	W	174,083
PA	Cambria Co	W	152,598
PA	Carbon Co	W	58,802
PA	Centre Co	W	135,758
PA	Chester Co	W	433,501
PA	Clearfield Co	W	83,382
PA	Cumberland Co	W	213,674
PA	Dauphin Co	W	251,798
PA	Delaware Co	W	550,864
PA	Erie Co	W	280,843
PA	Fayette Co	W	148,644
PA	Franklin Co	W	129,313
PA	Greene Co	W	40,672
PA	Indiana Co	W	89,605
PA	Lackawanna Co	W	213,295
PA	Lancaster Co	W	470,658
PA	Lebanon Co	W	120,327
PA	Lehigh Co	W	312,090
PA	Luzerne Co	W	319,250
PA	Mercer Co	W	120,293
PA	Monroe Co	W	138,687
PA	Montgomery Co	W	750,097
PA	Northampton Co	W	267,066
PA	Perry Co	W	43,602
PA	Philadelphia Co	W	1,517,550
PA	Tioga Co	W	41,373
PA	Washington Co	W	202,897
PA	Westmoreland Co	W	369,993
PA	Wyoming Co	W	28,080
PA	York Co	W	381,751
RI	Bristol Co	W	50,648
RI	Kent Co	W	167,090
RI	Newport Co	W	85,433
RI	Providence Co	W	621,602
RI	Washington Co	W	123,546

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
SC	Anderson Co	W	165,740
SC	Greenville Co	W	379,616
SC	Lexington Co	P	181,265
SC	Richland Co	P	313,253
SC	Spartanburg Co	W	253,791
SC	York Co	P	102,000
TN	Anderson Co	W	71,330
TN	Blount Co	W	105,823
TN	Cocke Co	P	20
TN	Davidson Co	W	569,891
TN	Hamilton Co	W	307,896
TN	Hawkins Co	W	53,563
TN	Jefferson Co	W	44,294
TN	Knox Co	W	382,032
TN	Loudon Co	W	39,086
TN	Meigs Co	W	11,086
TN	Montgomery Co	W	134,768
TN	Rutherford Co	W	182,023
TN	Sevier Co	W	71,170
TN	Shelby Co	W	897,472
TN	Sullivan Co	W	153,048
TN	Sumner Co	W	130,449
TN	Williamson Co	W	126,638
TN	Wilson Co	W	88,809
TX	Bexar Co	W	1,392,931
TX	Brazoria Co	W	241,767
TX	Chambers Co	W	26,031
TX	Collin Co	W	491,675
TX	Comal Co	W	78,021
TX	Dallas Co	W	2,218,899
TX	Denton Co	W	432,976
TX	Ellis Co	W	111,360
TX	Fort Bend Co	W	354,452
TX	Galveston Co	W	250,158
TX	Guadalupe Co	W	89,023
TX	Hardin Co	W	48,073
TX	Harris Co	W	3,400,578
TX	Jefferson Co	W	252,051
TX	Johnson Co	W	126,811
TX	Kaufman Co	W	71,313
TX	Liberty Co	W	70,154
TX	Montgomery Co	W	293,768
TX	Orange Co	W	84,966
TX	Parker Co	W	88,495
TX	Rockwall Co	W	43,080
TX	Tarrant Co	W	1,446,219

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
TX	Waller Co	W	32,663
VA	Arlington Co	W	189,453
VA	Botetourt Co	W	30,496
VA	Charles City Co	W	6,926
VA	Chesterfield Co	W	259,903
VA	Fairfax Co	W	969,749
VA	Frederick Co	W	59,209
VA	Gloucester Co	W	34,780
VA	Hanover Co	W	86,320
VA	Henrico Co	W	262,300
VA	Isle Of Wight Co	W	29,728
VA	James City Co	W	48,102
VA	Loudoun Co	W	169,599
VA	Madison Co	P	1
VA	Page Co	P	1
VA	Prince George Co	W	33,047
VA	Prince William Co	W	280,813
VA	Roanoke Co	W	85,778
VA	Spotsylvania Co	W	90,395
VA	Stafford Co	W	92,446
VA	York Co	W	56,297
VA	Alexandria	W	128,283
VA	Chesapeake	W	199,184
VA	Colonial Heights	W	16,897
VA	Fairfax	W	21,498
VA	Falls Church	W	10,377
VA	Fredericksburg	W	19,279
VA	Hampton	W	146,437
VA	Hopewell	W	22,354
VA	Manassas	W	35,135
VA	Manassas Park	W	10,290
VA	Newport News	W	180,150
VA	Norfolk	W	234,403
VA	Petersburg	W	33,740
VA	Poquoson	W	11,566
VA	Portsmouth	W	100,565
VA	Richmond	W	197,790
VA	Roanoke	W	94,911
VA	Salem	W	24,747
VA	Suffolk	W	63,677
VA	Virginia Beach	W	425,257
VA	Williamsburg	W	11,998
VA	Winchester	W	23,585
WV	Berkeley Co	W	75,905
WV	Brooke Co	W	25,447
WV	Cabell Co	W	96,784

State	8-hour Ozone Nonattainment County	Whole (W) or Partial (P) County?	Population
WV	Hancock Co	W	32,667
WV	Jefferson Co	W	42,190
WV	Kanawha Co	W	200,073
WV	Marshall Co	W	35,519
WV	Ohio Co	W	47,427
WV	Putnam Co	W	51,589
WV	Wayne Co	W	42,903
WV	Wood Co	W	87,986
WI	Door Co	W	27,961
WI	Kenosha Co	W	149,577
WI	Kewaunee Co	W	20,187
WI	Manitowoc Co	W	82,887
WI	Milwaukee Co	W	940,164
WI	Ozaukee Co	W	82,317
WI	Racine Co	W	188,831
WI	Sheboygan Co	W	112,646
WI	Washington Co	W	117,493
WI	Waukesha Co	W	360,767
		Total	159,271,919

Appendix 3B

Table 3B-1. PM_{2.5} Nonattainment Areas and Populations (data is current through September 2005 and the population numbers are from 2000 census data)

PM _{2.5} Nonattainment Area	Population
Atlanta, GA	4,231,750
Baltimore, MD	2,512,431
Birmingham, AL	807,612
Canton-Massillon, OH	378,098
Charleston, WV	251,662
Chattanooga, AL-TN-GA	423,809
Chicago-Gary-Lake County, IL-IN	8,757,808
Cincinnati-Hamilton, OH-KY-IN	1,850,975
Cleveland-Akron-Lorain, OH	2,775,447
Columbus, OH	1,448,503
Dayton-Springfield, OH	851,690
Detroit-Ann Arbor, MI	4,833,493
Evansville, IN	277,402
Greensboro-Winston Salem-High Point, NC	568,294
Harrisburg-Lebanon-Carlisle, PA	585,799
Hickory, NC	141,685
Huntington-Ashland, WV-KY-OH	340,776
Indianapolis, IN	1,329,185
Johnstown, PA	164,431
Knoxville, TN	599,008
Lancaster, PA	470,658
Libby, MT	2,626
Liberty-Clairton, PA	21,600
Los Angeles-South Coast Air Basin, CA	14,593,587
Louisville, KY-IN	938,905
Macon, GA	154,837
Martinsburg, WV-Hagerstown, MD	207,828
New York-N. New Jersey-Long Island,NY-NJ-CT	19,802,587
Parkersburg-Marietta, WV-OH	152,912
Philadelphia-Wilmington, PA-NJ-DE	5,536,911
Pittsburgh-Beaver Valley, PA	2,195,054
Reading, PA	373,638
Rome, GA	90,565
San Joaquin Valley, CA	3,191,367
St. Louis, MO-IL	2,486,562
Steubenville-Weirton, OH-WV	132,008
Washington, DC-MD-VA	4,377,935
Wheeling, WV-OH	153,172
York, PA	381,751
Total	88,394,361

Table 3B-2. PM₁₀ Nonattainment Areas and Populations (data is current through September 29, 2005 and the population numbers are from 2000 census data)

PM10 Nonattainment Areas Listed Alphabetically	Classification	Number of Counties NAA	2000 Population (thousands)	EPA Region	State
Ajo (Pima County), AZ	Moderate	1	8	9	AZ
Anthony, NM	Moderate	1	3	6	NM
Bonner Co (Sandpoint), ID	Moderate	1	37	10	ID
Butte, MT	Moderate	1	35	8	MT
Clark Co, NV	Serious	1	1,376	9	NV
Coachella Valley, CA	Serious	1	182	9	CA
Columbia Falls, MT	Moderate	1	4	8	MT
Coso Junction, CA	Moderate	1	7	9	CA
Douglas (Cochise County), AZ	Moderate	1	16	9	AZ
Eagle River, AK	Moderate	1	195	10	AK
El Paso Co, TX	Moderate	1	564	6	TX
Eugene-Springfield, OR	Moderate	1	179	10	OR
Flathead County; Whitefish and vicinity, MT	Moderate	1	5	8	MT
Fort Hall Reservation, ID	Moderate	2	1	10	ID
Hayden/Miami, AZ	Moderate	2	4	9	AZ
Imperial Valley, CA	Serious	1	120	9	CA
Juneau, AK	Moderate	1	14	10	AK
Kalispell, MT	Moderate	1	15	8	MT
LaGrande, OR	Moderate	1	12	10	OR
Lake Co, OR	Moderate	1	3	10	OR
Lamar, CO	Moderate	1	9	8	CO
Lame Deer, MT	Moderate	1	1	8	MT
Lane Co, OR	Moderate	1	3	10	OR
Libby, MT	Moderate	1	3	8	MT
Los Angeles South Coast Air Basin, CA	Serious	4	14,594	9	CA
Lyons Twsp., IL	Moderate	1	109	5	IL
Medford-Ashland, OR	Moderate	1	78	10	OR
Missoula, MT	Moderate	1	52	8	MT
Mono Basin, CA	Moderate	1	0	9	CA
Mun. of Guaynabo, PR	Moderate	1	92	2	PR
New Haven Co, CT	Moderate	1	124	1	CT
New York Co, NY	Moderate	1	1,537	2	NY
Nogales, AZ	Moderate	1	25	9	AZ
Ogden, UT	Moderate	1	77	8	UT
Owens Valley, CA	Serious	1	7	9	CA
Paul Spur, AZ	Moderate	1	1	9	AZ
Phoenix, AZ	Serious	2	3,112	9	AZ
Pinehurst, ID	Moderate	1	2	10	ID
Polson, MT	Moderate	1	4	8	MT
Portneuf Valley, ID	Moderate	2	66	10	ID
Rillito, AZ	Moderate	1	1	9	AZ
Ronan, MT	Moderate	1	3	8	MT
Sacramento Co, CA	Moderate	1	1,223	9	CA

Salt Lake Co, UT	Moderate	1	898	8	UT
San Bernardino Co, CA	Moderate	1	199	9	CA
San Joaquin Valley, CA	Serious	7	3,080	9	CA
Sanders County (part);Thompson Falls and vicinity,MT	Moderate	1	1	8	MT
Sheridan, WY	Moderate	1	16	8	WY
Shoshone Co, ID	Moderate	1	10	10	ID
Southeast Chicago, IL	Moderate	1	3	5	IL
Trona, CA	Moderate	1	4	9	CA
Utah Co, UT	Moderate	1	369	8	UT
Washoe Co, NV	Serious	1	339	9	NV
Weirton, WV	Moderate	2	15	3	WV
Yuma, AZ	Moderate	1	82	9	AZ
55 Total Areas		54	28,918		

Appendix 3C

Table 3C-1. List of 156 Mandatory Class I Federal Areas Where Visibility is an Important Value (As Listed in 40 CFR 81)*

State	Area Name	Acreage	Federal Land Manager
Alabama	Sipsey Wilderness Area	12,646	USDA-FS
Alaska	Bering Sea Wilderness Area	41,113	USDI-FWS
	Denali NP (formerly Mt. McKinley NP)	1,949,493	USDI-NPS
	Simeonof Wilderness Area	25,141	USDI-FWS
	Tuxedni Wilderness Area	6,402	USDI-FWS
Arizona	Chiricahua National Monument Wilderness Area	9,440	USDI-NPS
	Chiricahua Wilderness Area	18,000	USDA-FS
	Galiuro Wilderness Area	52,717	USDA-FS
	Grand Canyon NP	1,176,913	USDI-NPS
	Mazatzal Wilderness Area	205,137	USDA-FS
	Mount Baldy Wilderness Area	6,975	USDA-FS
	Petrified Forest NP	93,493	USDI-NPS
	Pine Mountain Wilderness Area	20,061	USDA-FS
	Saguaro Wilderness Area	71,400	USDI-FS
	Sierra Ancha Wilderness Area	20,850	USDA-FS
	Superstition Wilderness Area	124,117	USDA-FS
	Sycamore Canyon Wilderness Area	47,757	USDA-FS
Arkansas	Caney Creek Wilderness Area	4,344	USDA-FS
	Upper Buffalo Wilderness Area	9,912	USDA-FS
California	Agua Tibia Wilderness Area	15,934	USDA-FS
	Caribou Wilderness Area	19,080	USDA-FS
	Cucamonga Wilderness Area	9,022	USDA-FS
	Desolation Wilderness Area	63,469	USDA-FS
	Dome Land Wilderness Area	62,206	USDA-FS
	Emigrant Wilderness Area	104,311	USDA-FS
	Hoover Wilderness Area	47,916	USDA-FS
	John Muir Wilderness Area	484,673	USDA-FS
	Joshua Tree Wilderness Area	429,690	USDI-NPS
		36,300	USDI-BLM
	Kaiser Wilderness Area	22,500	USDA-FS
	Kings Canyon NP	459,994	USDI-NPS
	Lassen Volcanic NP	105,800	USDI-NPS
	Lava Beds Wilderness Area	28,640	USDI-NPS
	Marble Mountain Wilderness Area	213,743	USDA-FS
	Minarets Wilderness Area	109,484	USDA-FS
	Mokelumme Wilderness Area	50,400	USDA-FS
	Pinnacles Wilderness Area	12,952	USDI-NPS
	Point Reyes Wilderness Area	25,370	USDI-NPS
	Redwood NP	27,792	USDI-NPS

State	Area Name	Acreage	Federal Land Manager
	San Gabriel Wilderness Area	36,137	USDA-FS
	San Gorgonio Wilderness Area	56,722	USDA-FS
		37,980	USDI-BLM
	San Jacinto Wilderness Area	20,564	USDA-FS
	San Rafael Wilderness Area	142,722	USDA-FS
	Sequoia NP	386,642	USDI-NS
	South Warner Wilderness Area	68,507	USDA-FS
	Thousand Lakes Wilderness Area	15,695	USDA-FS
	Ventana Wilderness Area	95,152	USDA-FS
	Yolla Bolly-Middle Eel Wilderness Area	111,841	USDA-FS
		42,000	USDI-BLM
	Yosemite NP	759,172	USDI-NPS
Colorado	Black Canyon of the Gunnison Wilderness Area	11,180	USDI-NPS
	Eagles Nest Wilderness Area	133,910	USDA-FS
	Flat Tops Wilderness Area	235,230	USDA-FS
	Great Sand Dunes Wilderness Area	33,450	USDI-NPS
	La Garita Wilderness Area	48,486	USDA-FS
	Maroon Bells-Snowmass Wilderness Area	71,060	USDA-FS
	Mesa Verde NP	51,488	USDI-NPS
	Mount Zirkel Wilderness Area	72,472	USDA-FS
	Rawah Wilderness Area	26,674	USDA-FS
	Rocky Mountain NP	263,138	USDI-NPS
	Weminuche Wilderness Area	400,907	USDA-FS
	West Elk Wilderness Area	61,412	USDA-FS
Florida	Chassahowitzka Wilderness Area	23,360	USDI-FWS
	Everglades NP	1,397,429	USDI-NPS
	St. Marks Wilderness Area	17,745	USDI-FWS
Georgia	Cohotta Wilderness Area	33,776	USDA-FS
	Okefenokee Wilderness Area	343,850	USDI-FWS
	Wolf Island Wilderness Area	5,126	USDI-FWS
Hawaii	Haleakala NP	27,208	USDI-NPS
	Hawaii Volcanoes NP	217,029	USDI-NPS
Idaho	Craters of the Moon Wilderness Area ^a	43,243	USDI-NPS
	Hells Canyon Wilderness Area	83,800	USDA-FS
	Sawtooth Wilderness Area	216,383	USDA-FS
	Selway-Bitterroot Wilderness Area ^b	988,770	USDA-FS
	Yellowstone NP ^c	31,488	USDI-NPS
Kentucky	Mammoth Cave NP	51,303	USDI-NPS
Louisiana	Breton Wilderness Area	5,000+	USDI-FWS
Maine	Acadia National Park	37,503	USDI-NPS
	Moosehorn Wilderness Area	7,501	USDI-FWS
	Edmunds Unit	2,706	USDI-FWS
	Baring Unit	4,680	USDI-FWS
Michigan	Isle Royale NP	542,428	USDI-NPS
	Seney Wilderness Area	25,150	USDI-FWS

State	Area Name	Acreage	Federal Land Manager
Minnesota	Boundary Waters Canoe Area Wilderness Area	747,840	USDA-FS
	Voyageurs NP	114,964	USDI-NPS
Missouri	Hercules-Glades Wilderness Area	12,315	USDA-FS
	Mingo Wilderness Area	8,000	USDI-FWS
Montana	Anaconda-Pintlar Wilderness Area	157,803	USDA-FS
	Bob Marshall Wilderness Area	950,000	USDA-FS
	Cabinet Mountains Wilderness Area	94,272	USDA-FS
	Gates of the Mtn Wilderness Area	28,562	USDA-FS
	Glacier NP	1,012,599	USDI-NPS
	Medicine Lake Wilderness Area	11,366	USDI-FWS
	Mission Mountain Wilderness Area	73,877	USDA-FS
	Red Rock Lakes Wilderness Area	32,350	USDI-FWS
	Scapegoat Wilderness Area	239,295	USDA-FS
	Selway-Bitterroot Wilderness Area ^d	251,930	USDA-FS
	U. L. Bend Wilderness Area	20,890	USDI-FWS
	Yellowstone NP ^e	167,624	USDI-NPS
Nevada	Jarbidge Wilderness Area	64,667	USDA-FS
New Hampshire	Great Gulf Wilderness Area	5,552	USDA-FS
	Presidential Range-Dry River Wilderness Area	20,000	USDA-FS
New Jersey	Brigantine Wilderness Area	6,603	USDI-FWS
New Mexico	Bandelier Wilderness Area	23,267	USDI-NPS
	Bosque del Apache Wilderness Area	80,850	USDI-FWS
	Carlsbad Caverns NP	46,435	USDI-NPS
	Gila Wilderness Area	433,690	USDA-FS
	Pecos Wilderness Area	167,416	USDA-FS
	Salt Creek Wilderness Area	8,500	USDI-FWS
	San Pedro Parks Wilderness Area	41,132	USDA-FS
	Wheeler Peak Wilderness Area	6,027	USDA-FS
	White Mountain Wilderness Area	31,171	USDA-FS
North Carolina	Great Smoky Mountains NP ^f	273,551	USDI-NPS
	Joyce Kilmer-Slickrock Wilderness Area ^g	10,201	USDA-FS
	Linville Gorge Wilderness Area	7,575	USDA-FS
	Shining Rock Wilderness Area	13,350	USDA-FS
	Swanquarter Wilderness Area	9,000	USDI-FWS
North Dakota	Lostwood Wilderness	5,557	USDI-FWS
	Theodore Roosevelt NP	69,675	USDI-NPS
Oklahoma	Wichita Mountains Wilderness	8,900	USDI-FWS
Oregon	Crater Lake NP	160,290	USDA-NPS
	Diamond Peak Wilderness	36,637	USDA-FS
	Eagle Cap Wilderness	293,476	USDA-FS
	Gearhart Mountain Wilderness	18,709	USDA-FS
	Hells Canyon Wilderness ^a	108,900	USDA-FS
		22,700	USDI-BLM
	Kalmiopsis Wilderness	76,900	USDA-FS

State	Area Name	Acreage	Federal Land Manager
	Mountain Lakes Wilderness	23,071	USDA-FS
	Mount Hood Wilderness	14,160	USDA-FS
	Mount Jefferson Wilderness	100,208	USDA-FS
	Mount Washington Wilderness	46,116	USDA-FS
	Strawberry Mountain Wilderness	33,003	USDA-FS
	Three Sisters Wilderness	199,902	USDA-FS
South Carolina	Cape Romain Wilderness	28,000	USDI-FWS
South Dakota	Badlands Wilderness	64,250	USDI-NPS
	Wind Cave NP	28,060	USDI-NPS
Tennessee	Great Smoky Mountains NP ^f	241,207	USDI-NPS
	Joyce Kilmer-Slickrock Wilderness ^g	3,832	USDA-FS
Texas	Big Bend NP	708,118	USDI-NPS
	Guadalupe Mountains NP	76,292	USDI-NPS
Utah	Arches NP	65,098	USDI-NPS
	Bryce Canyon NP	35,832	USDI-NPS
	Canyonlands NP	337,570	USDI-NPS
	Capitol Reef NP	221,896	USDI-NPS
	Zion NP	142,462	USDI-NPS
Vermont	Lye Brook Wilderness	12,430	USDA-FS
Virgin Islands	Virgin Islands NP	12,295	USDI-NPS
Virginia	James River Face Wilderness	8,703	USDA-FS
	Shenandoah NP	190,535	USDI-NPS
Washington	Alpine Lakes Wilderness	303,508	USDA-FS
	Glacier Peak Wilderness	464,258	USDA-FS
	Goat Rocks Wilderness	82,680	USDA-FS
	Mount Adams Wilderness	32,356	USDA-FS
	Mount Rainier NP	235,239	USDI-NPS
	North Cascades NP	503,277	USDI-NPS
	Olympic NP	892,578	USDI-NPS
	Pasayten Wilderness	505,524	USDA-FS
West Virginia	Dolly Sods Wilderness	10,215	USDA-FS
	Otter Creek Wilderness	20,000	USDA-FS
Wyoming	Bridger Wilderness	392,160	USDA-FS
	Fitzpatrick Wilderness	191,103	USDA-FS
	Grand Teton NP	305,504	USDI-NPS
	North Absaroka Wilderness	351,104	USDA-FS
	Teton Wilderness	557,311	USDA-FS
	Washakie Wilderness	686,584	USDA-FS
	Yellowstone NP ^h	2,020,625	USDI-NPS
New Brunswick, Canada	Roosevelt Campobello International Park	2,721	i

* U.S. EPA (2001) Visibility in Mandatory Federal Class I Areas (1994-1998): A Report to Congress. EPA-452/R-01-008. This document is available in Docket EPA-HQ-OAR-2005-0036.

- a) Hells Canyon Wilderness Area, 192,700 acres overall, of which 108,900 acres are in Oregon and 83,800 acres are in Idaho.
- b) Selway Bitterroot Wilderness Area, 1,240,700 acres overall, of which 988,700 acres are in Idaho and 251,930 acres are in Montana.
- c) Yellowstone National Park, 2,219,737 acres overall, of which 2,020,625 acres are in Wyoming, 167,624 acres are in Montana, and 31,488 acres are in Idaho
- d) Selway-Bitterroot Wilderness Area, 1,240,700 acres overall, of which 988,770 acres are in Idaho and 251,930 acres are in Montana.
- e) Yellowstone National Park, 2,219,737 acres overall, of which 2,020,625 acres are in Wyoming, 167,624 acres are in Montana, and 31,488 acres are in Idaho.
- f) Great Smoky Mountains National Park, 514,758 acres overall, of which 273,551 acres are in North Carolina, and 241,207 acres are in Tennessee.
- g) Joyce Kilmer-Slickrock Wilderness Area, 14,033 acres overall, of which 10,201 acres are in North Carolina, and 3,832 acres are in Tennessee.
- h) Yellowstone National Park, 2,219,737 acres overall, of which 2,020,625 acres are in Wyoming, 167,624 acres are in Montana, and 31,488 acres are in Idaho.
- i) Chairman, RCIP Commission.

Abbreviations Used in Table:

USDA-FS: U.S. Department of Agriculture, U.S. Forest Service
USDI-BLM: U.S. Department of Interior, Bureau of Land Management
USDI-FWS: U.S. Department of Interior, Fish and Wildlife Service
USDI-NPS: U.S. Department of Interior, National Park Service

Table 3C-2. Current (1998-2002) Visibility, Projected (2015) Visibility, and Natural Background Levels for the 20% Worst Days at 116 IMPROVE Sites

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

Class I Area Name^a	State	1998-2002 Baseline Visibility (deciviews)^b	2015 CAIR Control Case Visibility^c (deciviews)	Natural Background (deciviews)
James River Face	VA	28.5	25.1	11.2
Jarbridge	NV	12.6	12.8	7.1
Joshua Tree	CA	19.5	20.3	7.1
Joyce Kilmer - Slickrock	NC	29.5	26.1	11.5
Kalmiopsis	OR	14.8	14.4	7.7
Kings Canyon	CA	23.5	24.1	7.1
La Garita	CO	11.6	11.5	7.1
Lassen Volcanic	CA	14.8	14.6	7.3
Lava Beds	CA	16.6	16.5	7.5
Linville Gorge	NC	27.9	24.6	11.4
Lostwood	ND	19.6	18.7	7.3
Lye Brook	VT	23.9	21.1	11.3
Mammoth Cave	KY	30.2	27.0	11.5
Marble Mountain	CA	17.1	16.8	7.7
Maroon Bells - Snowmass	CO	11.3	11.3	7.1
Mazatzal	AZ	13.1	13.5	6.9
Medicine Lake	MT	17.7	17.1	7.3
Mesa Verde	CO	12.8	12.8	7.1
Mingo	MO	27.5	25.9	11.3
Mission Mountains	MT	14.2	14.0	7.4
Mokelumne	CA	12.9	12.8	7.1
Moosehorn	ME	21.4	20.3	11.4
Mount Hood	OR	14.0	13.7	7.8
Mount Jefferson	OR	15.7	15.2	7.8
Mount Rainier	WA	18.9	19.4	7.9
Mount Washington	OR	15.7	15.2	7.9
Mount Zirkel	CO	11.7	11.8	7.1
North Cascades	WA	14.0	14.0	7.8
Okefenokee	GA	26.4	24.7	11.5
Otter Creek	WV	27.6	24.0	11.3
Pasayten	WA	14.7	14.5	7.8
Petrified Forest	AZ	13.5	13.8	7.0
Pine Mountain	AZ	13.1	13.4	6.9
Presidential Range - Dry	NH	23.2	20.9	11.3
Rawah	CO	11.7	11.7	7.1
Red Rock Lakes	WY	12.1	12.1	7.1
Redwood	CA	16.5	16.5	7.8
Rocky Mountain	CO	14.1	14.1	7.1
Roosevelt Campobello	ME	21.4	20.1	11.4
Salt Creek	NM	17.7	17.3	7.0
San Geronio	CA	21.5	22.1	7.1
San Jacinto	CA	21.5	21.4	7.1
San Pedro Parks	NM	11.4	11.4	7.0
Sawtooth	ID	13.6	13.5	7.2
Scapegoat	MT	14.2	14.1	7.3
Selway - Bitterroot	MT	12.3	12.1	7.3
Seney	MI	23.8	22.6	11.4
Sequoia	CA	23.5	24.1	7.1
Shenandoah	VA	27.6	23.4	11.3

Class I Area Name ^a	State	1998-2002 Baseline Visibility (deciviews) ^b	2015 CAIR Control Case Visibility ^c (deciviews)	Natural Background (deciviews)
Sierra Ancha	AZ	13.4	13.7	6.9
Sipsey	AL	28.7	26.1	11.4
South Warner	CA	16.6	16.5	7.3
Strawberry Mountain	OR	19.6	19.2	7.5
Superstition	AZ	14.7	15.0	6.9
Swanquarter	NC	24.6	21.9	11.2
Sycamore Canyon	AZ	16.1	16.6	7.0
Teton	WY	12.1	12.1	7.1
Theodore Roosevelt	ND	17.6	16.8	7.3
Thousand Lakes	CA	14.8	14.6	7.3
Three Sisters	OR	15.7	15.2	7.9
UL Bend	MT	14.7	14.1	7.2
Upper Buffalo	AR	25.5	24.3	11.3
Voyageurs	MN	18.4	17.6	11.1
Weminuche	CO	11.6	11.4	7.1
West Elk	CO	11.3	11.3	7.1
Wind Cave	SD	16.0	15.4	7.2
Wolf Island	GA	26.4	24.9	11.4
Yellowstone	WY	12.1	12.1	7.1
Yolla Bolly - Middle Eel	CA	17.1	16.9	7.4
Yosemite	CA	17.6	17.4	7.1
Zion	UT	13.5	13.3	7.0

a) 116 IMPROVE sites represent 155 of the 156 Mandatory Class I Federal Areas. One isolated Mandatory Class I Federal Area (Bering Sea, an uninhabited and infrequently visited island 200 miles from the coast of Alaska), was considered to be so remote from electrical power and people that it would be impractical to collect routine aerosol samples. U.S. EPA (2003) guidance for Tracking Progress Under the Regional Haze Rule. EPA-454/B-03-004. This document is available in Docket EPA-HQ-OAR-2005-0036.

b) The deciview metric describes perceived visual changes in a linear fashion over its entire range, analogous to the decibel scale for sound. A deciview of 0 represents pristine conditions. The higher the deciview value, the worse the visibility, and an improvement in visibility is a decrease in deciview value.

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

References for Chapter 3

- ¹ U. S. EPA (2003) National Air Quality and Trends Report, 2003 Special Studies Edition. Office of Air Quality Planning and Standards, Research Triangle Park, NC. Publication No. EPA 454/R-03-005. <http://www.epa.gov/air/airtrends/aqtrnd03/> This document is available in Docket EPA-HQ-OAR-2005-0036.
- ² U. S. EPA (2004) Air Toxics Website. <http://www.epa.gov/ttn/atw/stprogs.html>
- ³ U. S. EPA (2004) National Air Toxics Monitoring Strategy, Draft. Office of Air Quality Planning and Standards, Research Triangle Park, NC. January 2004. <http://www.epa.gov/ttn/amtic/files/ambient/airtox/atstrat104.pdf>
- ⁴ Kenski, D; Koerber, M.; Hafner, H. et al. (2005) Lessons learned from air toxics data. A national perspective. *Environ Manage.* June 2005: 19-22.
- ⁵ Aleksic, N.; Boynton, G.; Sistla, G.; Perry, J. (2005) Concentrations and trends of benzene in ambient air over New York State during 1990-2003. *Atmos Environ* 39: 7894-7905.
- ⁶ California Air Resources Board (2005) The California Almanac of Emissions and Air Quality - 2005 Edition. <http://www.arb.ca.gov/aqd/almanac/almanac05/almanac2005all.pdf>
- ⁷ Oommen, R.; Hauser, J.; Dayton, D; Brooks, G. (2005) Evaluating HAP trends: A look at emissions, concentrations, and regulation analyses for selected metropolitan statistical areas. Presentation at the 14th International Emissions Inventory Conference: Transforming Emission Inventories Meeting Future Challenges Today. April 12-14, 2005.
- ⁸ Clayton, C.A.; Pellizzari, E.D.; Whitmore, R.W.; et al. (1999) National Human Exposure Assessment Survey (NHEXAS): distributions and associations of lead, arsenic, and volatile organic compounds in EPA Region 5. *J Exposure Analysis Environ Epidemiol* 9: 381-392.
- ⁹ Gordon, S.M.; Callahan, P.J.; Nishioka, M.G.; et al. (1999) Residential environmental measurements in the National Human Exposure Assessment Survey (NHEXAS) pilot study in Arizona: preliminary results for pesticides and VOCs. *J Exposure Anal Environ Epidemiol* 9: 456-470.
- ¹⁰ Payne-Sturges, D.C.; Burke, T.A.; Beyse, P.; et al. (2004) Personal exposure meets risk assessment: a comparison of measured and modeled exposures and risk in an urban community. *Environ Health Perspect* 112: 589-598.
- ¹¹ Weisel, C.P.; Zhang, J.; Turpin, B.J.; et al. (2005) Relationships of Indoor, Outdoor, and Personal Air (RIOPA). Part I. Collection methods and descriptive analyses. *Res Rep Health Effects Inst* 130.
- ¹² Kwon, J. (2005) Development of a RIOPA database and evaluation of the effect of proximity on the potential residential exposure to VOCs from ambient sources. Rutgers, the State University of New Jersey and University of Medicine and Dentistry of New Jersey. PhD dissertation.
- ¹³ Weisel, C.P. (2004) Assessment of the contribution to personal exposures of air toxics from mobile sources. Final report to U.S. Environmental Protection Agency, Office of Transportation and Air Quality. This document is available in Docket EPA-HQ-OAR-2005-0036.

-
- ¹⁴ Adgate, J.L.; Eberly, L.E.; Stroebel, C.; et al. (2004) Personal, indoor, and outdoor VOC exposures in a probability sample of children. *J Exposure Analysis Environ Epidemiol* 14: S4-S13.
- ¹⁵ Adgate, J.L.; Church, T.R.; Ryan, A.D.; et al. (2004) Outdoor, indoor, and personal exposures to VOCs in children. *Environ Health Perspect* 112: 1386-1392.
- ¹⁶ Sexton, K.; Adgate, J.L.; Church, T.R.; et al. (2005) Children's exposure to volatile organic compounds as determined by longitudinal measurements in blood. *Environ Health Perspect* 113: 342-349.
- ¹⁷ Kinney, P.L.; Chillrud, S.N.; Ramstrom, S.; et al. (2002) Exposures to multiple air toxics in New York City. *Environ Health Perspect* 110 (suppl 4): 539-546.
- ¹⁸ Sax, S.N.; Bennett, D.H.; Chillrud, S.N.; et al. (2004) Differences in source emission rates of volatile organic compounds in inter-city residences of New York City and Los Angeles. *J Exposure Analysis Environ Epidemiol* 14: S95-S109.
- ¹⁹ <http://www.epa.gov/eogapti1/module3/category/category.htm> This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁰ Kittelson, D.; Watts, W.; Johnson, J. (2002) Diesel aerosol sampling methodology. Coordinating Research Council report E-43. <http://www.crcao.com> This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²¹ Zhang, K.M.; Wexler, A.S.; Zhu, Y.F.; et al. (2004) Evolution of particle number distribution near roadways. Part II: the 'Road-to-Ambient' process. *Atmos Environ* 38: 6655-6665.
- ²² Zhu, Y.; Hinds, W.C.; Kim, S.; et al. (2002) Study of ultrafine particles near a major highway with heavy-duty diesel traffic. *Atmos Environ* 36: 4323-4335.
- ²³ Zhu, Y.; Hinds, W.C.; Kim, S.; Sioutas, C. (2002) Concentration and size distribution of ultrafine particles near a major highway. *J Air & Waste Manage Assoc* 52: 1032-1042.
- ²⁴ Kittelson, D.B.; Watts, W.F.; Johnson, J.P. (2004) Nanoparticle emissions on Minnesota highways. *Atmos Environ* 38: 9-19.
- ²⁵ Reponen, T.; Grinshpun, S.A.; Trakumas, S.; et al. (2003) Concentration gradient patterns of aerosol particles near interstate highways in the Greater Cincinnati airshed. *J Environ Monit* 5: 557-562.
- ²⁶ Bunn, H.J.; Dinsdale, D.; Smith, T.; Grigg, J. (2001) Ultrafine particles in alveolar macrophages from normal children. *Thorax* 56: 932-934.
- ²⁷ Kittelson, D.B.; Watts, W.F.; Johnson, J.P. (2004) Nanoparticle emissions on Minnesota highways. *Atmos Environ* 38: 9-19.
- ²⁸ Kittelson, D.B.; Watts, W.F.; Johnson, J.P. (2004) Nanoparticle emissions on Minnesota highways. *Atmos Environ* 38: 9-19.
- ²⁹ Sanders, P.G.; Xu, N.; Dalka, T.M.; Maricq, M.M. (2003) Airborne brake wear debris: size distributions, composition, and a comparison of dynamometer and vehicle tests. *Environ Sci Technol* 37: 4060-4069.

-
- ³⁰ Kamens, R.M.; Jang, M.; Lee, S.; et al. (2003) Secondary organic aerosol formation: some new and exciting insights. *American Geophysical Union* 5: 02915.
- ³¹ Kupiainen, K.J.; Tervahattu, H; Raisanen, M.; et al. (2005) Size and composition of airborne particles from pavement wear, tires, and traction sanding. *Environ Sci Technol* 39: 699-706.
- ³² Sanders, P.G.; Xu, N; Dalka, T.M.; Maricq, M.M. (2003) Airborne brake wear debris: size distributions, composition, and a comparison of dynamometer and vehicle tests. *Environ Sci Technol* 37: 4060-4069.
- ³³ Hitchins, J.; Morawska, L.; Wolff, R.; Gilbert, D. (2000) Concentrations of submicrometre particles from vehicle emissions near a major road. *Atmos Environ* 34: 51-59.
- ³⁴ Janssen, N.A.H.; van Vliet, P.H.N.; Aarts, F.; et al. (2001) Assessment of exposure to traffic related air pollution of children attending schools near motorways. *Atmos Environ* 35: 3875-3884.
- ³⁵ Roorda-Knape M.C.; Janssen N.A.H.; De Hartog J.J.; et al. (1998) Air pollution from traffic in city districts near major motorways. *Atmos Environ* 32: 1921-1930.
- ³⁶ U.S. EPA (2002) National Air Toxics Assessment. <http://www.epa.gov/ttn/atw/nata/>
- ³⁷ Kinnee, E.J.; Beidler, A.; Touma, J.S.; et al. (2004) Allocation of onroad mobile emissions to road segments for air toxics modeling in an urban area. *Transportation Res Part D* 9: 139-150.
- ³⁸ Cohen, J.; Cook, R.; Bailey, C.R.; Carr, E. (2005) Relationship between motor vehicle emissions of hazardous pollutants, roadway proximity, and ambient concentrations in Portland, Oregon. *Environ Modeling & Software* 20: 7-12.
- ³⁹ Skov, H.; Hansen, A.B.; Lorenzen, G.; et al. (2001) Benzene exposure and the effect of traffic pollution in Copenhagen, Denmark. *Atmos Environ* 35: 2463-2471.
- ⁴⁰ Jo, W.; Kim, K.; Park, K.; et al. (2003) Comparison of outdoor and indoor mobile source-related volatile organic compounds between low- and high-floor apartments. *Environ Res* 92: 166-171.
- ⁴¹ Fischer, P.H.; Joek, G.; van Reeuwijk, H.; et al. (2000) Traffic-related differences in outdoor and indoor concentrations of particle and volatile organic compounds in Amsterdam. *Atmos Environ* 34: 3713-3722.
- ⁴² Ilgen, E.; Karfich, N.; Levsen, K.; et al. (2001) Aromatic hydrocarbons in the atmospheric environment: part I. Indoor versus outdoor sources, the influence of traffic. *Atmos Environ* 35: 1235-1252.
- ⁴³ Rodes, C.; Sheldon, L.; Whitaker, D.; et al. (1998) Measuring concentrations of selected air pollutants inside California vehicles. Final report to California Air Resources Board. Contract No. 95-339.
- ⁴⁴ Sapkota, A.; Buckley, T.J. (2003) The mobile source effect on curbside 1,3-butadiene, benzene, and particle-bound polycyclic aromatic hydrocarbons assessed at a tollbooth. *J Air Waste Manage Assoc* 53: 740-748.

-
- ⁴⁵ Sapkota, A.; Buckley, T.J. (2003) The mobile source effect on curbside 1,3-butadiene, benzene, and particle-bound polycyclic aromatic hydrocarbons assessed at a tollbooth. *J Air Waste Manage Assoc* 53: 740-748.
- ⁴⁶ <http://www.mde.state.md.us/Programs/AirPrograms/airData/dataReport.asp> This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁴⁷ Ilgen, E.; Karfich, N.; Levsen, K.; et al. (2001) Aromatic hydrocarbons in the atmospheric environment: part I. Indoor versus outdoor sources, the influence of traffic. *Atmos Environ* 35: 1235-1252.
- ⁴⁸ Kwon, J. (2005) Development of a RIOPA database and evaluation of the effect of proximity on the potential residential exposure to VOCs from ambient sources. Rutgers, the State University of New Jersey and University of Medicine and Dentistry of New Jersey. PhD dissertation. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁴⁹ Hoek G.; Meliefste K.; Cyrus J.; et al. (2002) Spatial variability of fine particle concentrations in three European areas. *Atmos. Environ.* 36: 4077-4088.
- ⁵⁰ Etyemezian V.; Kuhns H.; Gillies J.; et al. (2003) Vehicle-based road dust emission measurement (III): effect of speed, traffic volume, location, and season on PM10 road dust emissions in the Treasure Valley, ID. *Atmos. Environ.* 37: 4583-4593.
- ⁵¹ Harrison R.M.; Tilling R.; Romero M.S.C.; et al. (2003) A study of trace metals and polycyclic aromatic hydrocarbons in the roadside environment. *Atmos. Environ.* 37: 2391-2402.
- ⁵² Zhang K.M.; Wexler A.S.; Zhu Y.F.; et al. (2004) Evolution of particle number distribution near roadways. Part II: the 'Road-to-Ambient' process. *Atmos. Environ.* 38: 6655-6665.
- ⁵³ Zhu Y.F.; Hinds W.C.; Shen S.; Sioutas C. (2004) Seasonal trends of concentration and size distribution of ultrafine particles near major highways in Los Angeles. *Aerosol Sci. Technol.* 38: 5-13.
- ⁵⁴ Zhu, Y.; Hinds, W.C.; Kim, S.; et al. (2002) Study of ultrafine particles near a major highway with heavy-duty diesel traffic. *Atmos Environ* 36: 4323-4335.
- ⁵⁵ Zhu, Y.; Hinds, W.C.; Kim, S.; Sioutas, C. (2002) Concentration and size distribution of ultrafine particles near a major highway. *J Air & Waste Manage Assoc* 52: 1032-1042.
- ⁵⁶ Riediker, M.; Williams, R.; Devlin, R.; et al. (2003) Exposure to particulate matter, volatile organic compounds, and other air pollutants inside patrol cars. *Environ Sci Technol* 37: 2084-2093.
- ⁵⁷ Zielinska, B.; Fujita, E.M.; Sagebiel, J.C.; et al. (2002) Interim data report for Section 211(B) Tier 2 high end exposure screening study of baseline and oxygenated gasoline. Prepared for American Petroleum Institute. November 19, 2002. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁵⁸ Rodes, C.; Sheldon, L.; Whitaker, D.; et al. (1998) Measuring concentrations of selected air pollutants inside California vehicles. Final report to California Air Resources Board. Contract No. 95-339.

-
- ⁵⁹ Fitz, D. R.; Winer, A. M.; Colome, S.; et al. (2003) Characterizing the Range of Children's Pollutant Exposure During School Bus Commutes. Final Report Prepared for the California Resources Board. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁶⁰ Sabin, L.D.; Behrentz, E.; Winer, A.M.; et al. (2005) Characterizing the range of children's air pollutant exposure during school bus commutes. *J Expos Anal Environ Epidemiol* 15: 377-387.
- ⁶¹ Behrentz, E.; Sabin, L.D.; Winer, A.M.; et al. (2005) Relative importance of school bus-related microenvironments to children's pollutant exposure. *J Air & Waste Manage Assoc* 55: 1418-1430.
- ⁶² Batterman, S.A.; Peng, C.Y.; and Braun, J. (2002) Levels and composition of volatile organic compounds on commuting routes in Detroit, Michigan. *Atmos Environ* 36: 6015-6030.
- ⁶³ Fruin, S.A.; Winer, A.M.; Rodes, C.E. (2004) Black carbon concentrations in California vehicles and estimation of in-vehicle diesel exhaust particulate matter exposures. *Atmos Environ* 38: 4123-4133.
- ⁶⁴ Adams, H.S.; Nieuwenhuijsen, M.J.; Colville, R.N. (2001) Determinants of fine particle (PM_{2.5}) personal exposure levels in transport microenvironments, London, UK. *Atmos Environ* 35: 4557-4566.
- ⁶⁵ Leung, P.-L.; Harrison, R.M. (1999) Roadside and in-vehicle concentrations of monoaromatic hydrocarbons. *Atmos Environ* 33: 191-204.
- ⁶⁶ Weinhold, B. (2001) Pollutants lurk inside vehicles. *Environ Health Perspec* 109 (9): A422-A427.
- ⁶⁷ Riediker, M.; Williams, R.; Devlin, R.; et al. (2003) Exposure to particulate matter, volatile organic compounds, and other air pollutants inside patrol cars. *Environ Sci Technol* 37: 2084-2093.
- ⁶⁸ Van Wijnen J.H.; Verhoeff A.P.; Jans H.W.A.; Van Bruggen M. (1995) The exposure of cyclists, car drivers and pedestrians to traffic-related air pollutants. *Int Arch Occup Environ Health* 67: 187-193.
- ⁶⁹ Chan C.-C.; Ozkaynak H.; Spengler J.D.; Sheldon L. (1991) Driver Exposure to Volatile Organic Compounds, CO, Ozone, and NO₂ under Different Driving Conditions. *Environ. Sci. Technol.* 25: 964-972.
- ⁷⁰ Shikiya, D.C., C.S. Liu, M.I. Kahn, et al. (1989) In-vehicle air toxics characterization study in the south coast air basin. South Coast Air Quality Management District, El Monte, CA. May, 1989. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁷¹ Chan C.-C., Spengler J. D., Ozkaynak H., and Lefkopoulou M. (1991) Commuter Exposures to VOCs in Boston, Massachusetts. *J. Air Waste Manage. Assoc.* 41, 1594-1600.
- ⁷² U.S. EPA (2000) Development of microenvironmental factors for the HAPEM4 in support of the National Air Toxics Assessment (NATA). External Review Draft Report Prepared by ICF Consulting and TRJ Environmental, Inc. for the U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC. This document is available in Docket EPA-HQ-OAR-2005-0036.

-
- ⁷³ U.S. EPA (2000) Determination of microenvironmental factors for diesel PM. An addendum to: Development of microenvironmental factors for the HAPEM4 in support of the National Air Toxics Assessment (NATA). External Review Draft Report Prepared by ICF Consulting and TRJ Environmental, Inc. for the U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park, NC. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁷⁴ Personal communication with FACES Investigators Fred Lurmann, Paul Roberts, and Katharine Hammond. Data is currently being prepared for publication.
- ⁷⁵ Kim J.J.; Smorodinsky S.; Lipsett M.; et al. (2004) Traffic-related air pollution near busy roads. *Am J Respir Crit Care Med* 170: 520-526.
- ⁷⁶ Korenstein, S. and Piazza, B. (2002) An Exposure Assessment of PM10 from a Major Highway Interchange: Are Children in Nearby Schools at Risk? *J Environ Health* 65(2): 9-17.
- ⁷⁷ Janssen, N.A.H.; van Vliet, P.H.N.; Aarts, F.; et al. (2001) Assessment of exposure to traffic related air pollution of children attending schools near motorways. *Atmos Environ* 35: 3875-3884.
- ⁷⁸ Roorda-Knape M.C.; Janssen N.A.H.; De Hartog J.J.; et al. (1998) Air pollution from traffic in city districts near major motorways. *Atmos Environ* 32: 1921-1930.
- ⁷⁹ Kinney, P.L.; Chillrud, S.N.; Ramstrom, S.; et al. (2002) Exposures to multiple air toxics in New York City. *Environ Health Perspect* 110 (Suppl 4): 539-546.
- ⁸⁰ Weisel, C.P. (2002) Assessing exposure to air toxics relative to asthma. *Environ Health Perspect* 110 (Suppl 4): 527-537.
- ⁸¹ Naumova, Y.Y.; Eisenreich, S.J.; Turpin, B.J.; et al. (2002) Polycyclic aromatic hydrocarbons in the indoor and outdoor environment of three cities in the U.S. *Environ Sci Technol* 36: 2552-2559.
- ⁸² Chatzis, C.; Alexopoulos, E.C.; and Linos, A. (2005) Indoor and outdoor personal exposure to benzene in Athens, Greece. *Sci Total Environ* 349: 72-80.
- ⁸³ Gulliver J.; Briggs D.J. (2004) Personal exposure to particulate air pollution in transport microenvironments. *Atmos Environ* 38: 1-8.
- ⁸⁴ Van Wijnen J.H.; Verhoeff A.P.; Jans H.W.A.; Van Bruggen M. (1995) The exposure of cyclists, car drivers and pedestrians to traffic-related air pollutants. *Int Arch Occup Environ Health* 67: 187-193.
- ⁸⁵ Chan C.-C.; Ozkaynak H.; Spengler J.D.; Sheldon L. (1991) Driver Exposure to Volatile Organic Compounds, CO, Ozone, and NO2 under Different Driving Conditions. *Environ. Sci. Technol.* 25: 964-972.
- ⁸⁶ Chan C.-C., Spengler J. D., Ozkaynak H., and Lefkopoulou M. (1991) Commuter Exposures to VOCs in Boston, Massachusetts. *J. Air Waste Manage. Assoc.* 41, 1594-1600.
- ⁸⁷ Duci, A.; Chaloulakou, A.; Spyrellis N. (2003) Exposure to carbon monoxide in the Athens urban area during commuting. *Sci Total Environ* 309: 47-58.
- ⁸⁸ Gulliver J.; Briggs D.J. (2004) Personal exposure to particulate air pollution in transport microenvironments. *Atmos Environ* 38: 1-8.

-
- ⁸⁹ Ashmore, M.R.; Batty, K.; Machin, F.; et al. (2000) Effects of traffic management and transport mode on the exposure of schoolchildren to carbon monoxide. *Environ Monitoring and Assessment* 65: 49-57.
- ⁹⁰ Atkinson, R.; Arey, J.; Hoover, S.; Preston, K. (2005) Atmospheric Chemistry of Gasoline-Related Emissions: Formation of Pollutants of Potential Concern. Draft Report Prepared for California Environmental Protection Agency. September 2005. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁹¹ U.S. EPA (1997) Exposure factors handbook. This document is available in Docket EPA-HQ-OAR-2005-0036. <http://www.epa.gov/ncea>
- ⁹² Sheltersource, Inc. (2002) Evaluating Minnesota homes. Final report. Prepared for Minnesota Department of Commerce. U.S. Department of Energy grant DE-FG45-96R530335. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁹³ Fugler, D.; Grande, C.; Graham, L. (2002) Attached garages are likely path for pollutants. *ASHRAE IAQ Applications* 3(3): 1-4.
- ⁹⁴ Emmerich, S.J.; Gorfain, J.E.; Howard-Reed, C. (2003) Air and pollutant transport from attached garages to residential living spaces – literature review and field tests. In *J Ventilation* 2: 265-276.
- ⁹⁵ Isbell, M.; Gordian, M.E.; Duffy, L. (2002) Winter indoor air pollution in Alaska: identifying a myth. *Environ Pollution* 117: 69-75.
- ⁹⁶ Wallace, L. (1996) Environmental exposure to benzene: an update. *Environ Health Perspect* 104 (Suppl 6): 1129-1136.
- ⁹⁷ Batterman, S.; Hatzivasilis, G. (2005) Concentrations and emissions of gasoline and other vapors from residential vehicle garages. *Atmos Environ* (Accepted for publication).
- ⁹⁸ George, M.; Kaluza, P.; Maxwell, B.; et al. (2002) Indoor air quality & ventilation strategies in new homes in Alaska. Alaska Building Science Network. [Online at <http://www.cchrc.org>] This document is available in Docket EPA-HQ-OAR-2005-0036.
- ⁹⁹ Graham, L.A.; Noseworthy, L.; Fugler, D.; et al. (2004) Contribution of vehicle emissions from an attached garage to residential indoor air pollution levels. *J Air & Waste Manage Assoc* 54: 563-584.
- ¹⁰⁰ Schlapia, A.; Morris, S.S. (1998) Architectural, behavioral, and environmental factors associated with VOCs in Anchorage homes. Proceedings of the Air & Waste Management Association's 94th Annual Conference & Exhibition. Paper 98-A504.
- ¹⁰¹ Isbell, M.; Ricker, J.; Gordian, M.E.; Duffy, L.K. (1999) Use of biomarkers in an indoor air study: lack of correlation between aromatic VOCs with respective urinary biomarkers. *Sci Total Environ* 241: 151-159.
- ¹⁰² Isbell, M.A.; Stolzberg, R.J.; Duffy, L.K. (2005) Indoor climate in interior Alaska: simultaneous measurement of ventilation, benzene and toluene in residential indoor air of two homes. *Sci Total Environ* 345: 31-40.

-
- ¹⁰³ Gordon, S.M.; Callahan, P.J.; Nishioka, M.G.; et al. (1999) Residential environmental measurements in the National Human Exposure Assessment Survey (NHEXAS) pilot study in Arizona: preliminary results for pesticides and VOCs. *J Exposure Analysis Environ Epidemiol* 9: 456-470.
- ¹⁰⁴ Bonanno, L.J.; Freeman, N.C.G.; Greenberg, M.; Liroy, P.J. (2001) Multivariate analysis on levels of selected metals, particulate matter, VOC, and household characteristics and activities from the Midwestern states NHEXAS. *Appl Occup Environ Hygiene* 16: 859-874.
- ¹⁰⁵ Tsai, P.; Weisel, C.P. (2000) Penetration of evaporative emissions into a home from an M85-fueled vehicle parked in an attached garage. *J Air & Waste Manage Assoc* 50: 371-377.
- ¹⁰⁶ Bailey, C.R. (2005) Additional contribution to benzene exposure from attached garages. Memorandum to docket. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹⁰⁷ Wilson, A.L.; Colome, S.D.; and Tian, Y. (1991) Air toxics microenvironment exposure and monitoring study. Final Report. Prepared for South Coast Air Quality Management District and U.S. Environmental Protection Agency. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹⁰⁸ Zielinska, B.; Fujita, E.M.; Sagebiel, J.C.; et al. (2002) Interim data report for Section 211(B) Tier 2 high end exposure screening study of baseline and oxygenated gasoline. Prepared for American Petroleum Institute. November 19, 2002. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹⁰⁹ Lee, S.C.; Chan, L.Y.; and Chiu, M.Y. (1999) Indoor and outdoor air quality investigation at 14 public places in Hong Kong. *Environ Int* 25: 443-450.
- ¹¹⁰ Wong, Y.-c.; Sin, D.W.-m.; and Yeung L.L. (2002) Assessment of the air quality in indoor car parks. *Indoor & Built Environment* 11: 134-145.
- ¹¹¹ Chaloulakou, A.; Duci, A.; Spyrellis, N. (2002) Exposure to carbon monoxide in enclosed multi-level parking garages in the central Athens urban area. *Indoor & Built Environment* 11: 191-201.
- ¹¹² Srivastava, A.; Joseph, A.E.; and Nair, S. (2004) Ambient levels of benzene in Mumbai city. *Int J Environ Health Res* 14 (3): 215-222.
- ¹¹³ Schwar, M.; Booker, J.; Tait, L. (1997) Car Park Air Pollution Exposure of Operatives and the General Public. *Clean Air & Environ Protection* 27 (5): 129-137
- ¹¹⁴ Morillo, P.; Dos Santos, S.G.; Santamaria, J.; et al. (1998) A study of the atmospheric pollution produced by vehicles in car parks in Madrid, Spain. *Indoor Built Environ* 7: 156-164.
- ¹¹⁵ U.S. EPA (1987) The Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis: Volume I. Office of Research and Development, Washington, D.C. June 1987. EPA Report No. EPA/600/6-87/002a.
- ¹¹⁶ Wilson, A.L.; Colome, S.D.; and Tian, Y. (1991) Air toxics microenvironment exposure and monitoring study, Final Report. Prepared for South Coast Air Quality Management District and U.S. Environmental Protection Agency. This document is available in Docket EPA-HQ-OAR-2005-0036.

-
- ¹¹⁷ Hartle, R. (1993) Exposure to methyl tert-butyl ether and benzene among service station attendants and operators. *Environ Health Perspect Supplements*: 101 (Suppl. 6): 23-26.
- ¹¹⁸ Northeast States for Coordinated Air Use Management (1999) RFG/MTBE Findings and Recommendations. August 1999. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹¹⁹ Vayghani, S.A.; Weisel, C. (1999) The MTBE air concentrations in the cabin of automobiles while fueling. *J Expos Analysis Environ Epidemiol* 9: 261-267.
- ¹²⁰ Verma, D.K.; Johnson, D.M.; Shaw, M.L.; et al. (2001) Benzene and total hydrocarbon exposures in the downstream petroleum industries. *Am Indust Hygiene Assoc J* 62: 176-194.
- ¹²¹ Verma, D.K. and des Tombe, K. (2002) Benzene in gasoline and crude oil: occupational and environmental implications. *Am Indust Hygiene Assoc J* 63: 225-230.
- ¹²² Baldauf R.; Fortune C.; Weinstein J.; et al. (2005) Air contaminant exposures during the operation of lawn and garden equipment. *J Expos Anal Environ Epidemiol* *in press*.
- ¹²³ Eriksson, K.; Tjerner, D.; Marqvardsen, I.; et al. (2003) Exposure to benzene, toluene, xylenes, and total hydrocarbons among snowmobile drivers in Sweden. *Chemosphere* 50: 1343-1347.
- ¹²⁴ Kado, N.Y.; Kuzmicky, P.A.; and Okamoto, R.A. (2001) Environmental and occupational exposure to toxic air pollutants from winter snowmobile use in Yellowstone National Park. Final report to the Yellowstone Park Foundation, Pew Charitable Trusts, and National Park Service. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹²⁵ NESCAUM (2003) Evaluating the occupational and environmental impact of nonroad diesel equipment in the Northeast. Interim Report June 9, 2003. This document is available in Docket EPA-HQ-OAR-2005-0036. <http://www.nescaum.org/resources/reports/index.html>
- ¹²⁶ Reference available when the NATA website opens up.
- ¹²⁷ U.S. EPA (2000) User's Guide for the Assessment System for Population Exposure Nationwide (ASPEN, Version 1.1) Model. Office of Air Quality Planning and Standards, Research Triangle Park, NC, Report No. EPA-454/R-00-017. This document is available in Docket EPA-HQ-OAR-2005-0036. <http://www.epa.gov/scram001/userg/other/aspenug.pdf>
- ¹²⁸ Rosenbaum, A. (2005) The HAPEM5 User's Guide: Hazardous Air Pollutant Exposure Model, Version 5. Prepared by ICF, Inc. for U. S. EPA. This document is available in Docket EPA-HQ-OAR-2005-0036. http://www.epa.gov/ttn/fera/hapem5/hapem5_guide.pdf
- ¹²⁹ U. S. EPA (2005) Risk - Air Toxics Risk Assessment. http://www.epa.gov/ttn/fera/risk_atoxic.html.
- ¹³⁰ U. S. EPA (1993) Motor Vehicle-Related Air Toxics Study. Report No. EPA420-R-93-005. http://www.epa.gov/otaq/regs/toxics/tox_archive.htm#2
- ¹³¹ U. S. EPA (2000) Technical Support Document: Control of Hazardous Air Pollutants from Motor Vehicles and Motor Vehicle Fuels. Office of Transportation and Air Quality. Report No. EPA-420-R-00-023. <http://www.epa.gov/otaq/toxics.htm>

-
- ¹³² Cook, R., Jones, B., Cleland, J. (2004) A Cohort Based Approach for Characterizing Lifetime Inhalation Cancer Risk from Time-Varying Exposure to Air Toxics from Ambient Sources. *Environmental Progress* 23(2): 120-125.
- ¹³³ Cook, R., Strum, M., Touma, J., et al. 2002. Trends in Mobile Source-Related Ambient Concentrations of Hazardous Air Pollutants, 1996 – 2007. SAE Paper No. 2002-01-1274. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹³⁴ Byun, D. W., Ching, J. K. S. 1999. Science Algorithms of the EPA Models-3 Community Multiscale Air Quality (CMAQ) Modeling System. U. S. EPA, Office of Research and Development, Washington, DC. Report No. EPA/600/R-99/030. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹³⁵ Luecken, D. J., Hutzell, W. T., Gipson, G. J. 2005. Development and Analysis of air quality modeling simulations for hazardous air pollutants. Submitted to *Atmospheric Environment*.
- ¹³⁶ Seigneur, C., Pun, B., Lohman, K., Wu, S.-Y. 2003. Regional modeling of the atmospheric fate and transport of benzene and diesel particles. *Environ. Sci. Technol.* 37: 5236-5246.
- ¹³⁷ U. S. EPA. 2004. User's Guide for the Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP, Version 3.0), Office of Air Quality Planning and Standards, Research Triangle Park, NC, Report No. EPA-454/B-00-007. This document is available in Docket EPA-HQ-OAR-2005-0036. <http://www.epa.gov/scram001/userg/other/emshapv3ug.pdf>
- ¹³⁸ Battelle. 2003. Estimated background concentrations for the National-Scale Air Toxics Assessment. Prepared for U. S. EPA, Office of Air Quality Planning and Standards. Contract No. 68-D-02-061. Work Assignment 1-03. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹³⁹ U. S. EPA. 1993. Motor Vehicle-Related Air Toxics Study. Office of Mobile Sources, Ann Arbor, MI. Report No. EPA 420-R-93-005. This document is available in Docket EPA-HQ-OAR-2005-0036. http://www.epa.gov/otaq/regs/toxics/tox_archive.htm
- ¹⁴⁰ U. S. EPA. 1999. Analysis of the Impacts of Control Programs on Motor Vehicle Toxics Emissions and Exposure in Urban Areas and Nationwide. Prepared for U. S. EPA, Office of Transportation and Air Quality, by Sierra Research, Inc., and Radian International Corporation/Eastern Research Group. Report No. EPA 420 –R-99-029/030. This document is available in Docket EPA-HQ-OAR-2005-0036. http://www.epa.gov/otaq/regs/toxics/tox_archive.htm
- ¹⁴¹ U. S. EPA. 2002. 1996 National-Scale Air Toxics Assessment. <http://www.epa.gov/ttn/atw/nata/>
- ¹⁴² Glen, G., Lakkadi, Y., Tippet, J. A., del Valle-Torres M. 1997. Development of NERL/CHAD: The National Exposure Research Laboratory Consolidated Human Activity Database. Prepared by ManTech Environmental Technology, Inc. EPA Contract No. 68-D5-0049. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹⁴³ Long, T.,; Johnson, T. ; Laurensen, J.; Rosenbaum, A. 2004. Development of Penetration and Proximity Microenvironment Factor Distributions for the HAPEM5 in Support of the 1999 National-Scale Air Toxics Assessment (NATA). Memorandum from TRJ Consulting and ICF Consulting, Inc. to Ted Palma, U. S. EPA, Office of Air Quality Planning and Standards, RTP,

NC., April 5, 2004. This document is available in Docket EPA-HQ-OAR-2005-0036.
http://www.epa.gov/ttn/fera/human_hapem.html

¹⁴⁴ Rosenbaum, A. 2005. The HAPEM5 User's Guide: Hazardous Air Pollutant Exposure Model, Version 5. Prepared by ICF, Inc. for Ted Palma, U. S. EPA. This document is available in Docket EPA-HQ-OAR-2005-0036. http://www.epa.gov/ttn/fera/hapem5/hapem5_guide.pdf

¹⁴⁵ U. S. EPA. 2001. National-Scale Air Toxics Assessment for 1996: Draft for EPA Science Advisory Board Review. Report No. EPA-453/R-01-003 This document is available in Docket EPA-HQ-OAR-2005-0036. <http://www.epa.gov/ttn/atw/sab/natareport.pdf>

¹⁴⁶ U. S. EPA. 2004. Benefits of the Proposed Inter-State Air Quality Rule. Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. Report No. EPA 452/-03-001. This document is available in Docket EPA-HQ-OAR-2005-0036.
<http://www.epa.gov/CAIR/technical.html> or <http://www.epa.gov/CAIR/tsd0175.pdf>

¹⁴⁷ Memorandum from Arlene Rosenbaum and Kevin Wright, ICF Consulting, to Chad Bailey, U.S. Environmental Protection Agency. Subject: Estimating near roadway populations and areas for HAPEM6. This document is available in Docket EPA-HQ-OAR-2005-0036.

¹⁴⁸ Riediker M, Williams, R., Devlin, R., et al. 2003. Exposure to particulate matter, volatile organic compounds, and other air pollutants inside patrol cars. *Environ. Sci. Technol.* 2003, 37, 2084-2093.

¹⁴⁹ Rodes, C., Sheldon, L., Whitaker, D., et al. 1998. Measuring concentrations of selected air pollutants inside California vehicles. Main Study Report for California ARB. Contract 95-339. <http://www.arb.ca.gov/research/abstracts/95-339.htm>

¹⁵⁰ Zhu, Y, Hinds, W. C., Kim, S., et al. 2002. Study of ultrafine particles near a major highway with heavy-duty diesel traffic. *Atmos Environ* 36 (2002) 4323-4335.

¹⁵¹ Kwon, J. 2005. Development of a RIOPA database and evaluation of the effect of proximity on the potential residential exposure to VOCs from ambient sources. PhD. dissertation. Graduate School, New Brunswick, Rutgers, the State University of New Jersey and the University of Medicine and Dentistry of New Jersey. This document is available in Docket EPA-HQ-OAR-2005-0036.

¹⁵² Meng, Q. Y., Turpin, B. J., Korn, L., et al. 2005. Influence of ambient (outdoor) sources on residential indoor and personal PM_{2.5} concentrations: Analyses of RIOPA data. *Journal of Exposure Analysis and Environ Epidemiology* 15: 17-28.

¹⁵³ Weisel, C. P., Zhang, J. J., Turpin, B. J., et al. 2004. Relationship of Indoor, Outdoor and Personal Air (RIOPA) study; study design, methods and quality assurance / control results. *Journal of Exposure Analysis and Environ Epidemiology* 15: 123-137.

¹⁵⁴ Cohen, J., Cook, R., Bailey, C. R., Carr, E. 2005. Relationship between motor vehicle emissions of hazardous pollutants, roadway proximity, and ambient concentrations on Portland, OR. *Environmental Modelling and Software* 20: 7-12.

¹⁵⁵ U. S. EPA (2003) Estimated Background Concentrations for the National-Scale Air Toxics Assessment. Emissions, Monitoring, and Analysis Division, Office of Air Quality Planning and

Standards, Research Triangle Park, NC. <http://www.epa.gov/ttn/atw/nata1999/background.html>
This document is available in Docket EPA-HQ-OAR-2005-0036. NOT YET RELEASED.

¹⁵⁶ U. S. EPA (2005) Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. Report No. EPA/630/R-03/003F. This document is available in Docket EPA-HQ-OAR-2005-0036.

<http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=116283>

¹⁵⁷ Touma, J. S.; Isakov, V.; Ching, J.; Seigneur, C. (2006). Air Quality Modeling of Hazardous Air Pollutants: Current Status and Future Directions. J. Air and Waste Manage. Assoc., in press.

¹⁵⁸ Isakov, V.; Venkatram, A. (2005) Resolving neighborhood scale in air toxics modeling: a case study in Wilmington, California. J Air & Waste Manage Assoc *submitted*.

¹⁵⁹ Pratt, G. C.; Wu, C. Y.; Bock, D.; et al. (2004) Comparing air dispersion model predictions with measured concentrations of VOCs in urban communities. Environ. Sci. Technol. 38: 1949-1959.

¹⁶⁰ Kinnee, E. J.; Touma, J. S.; Mason, R.; et al. (2004) Allocation of Onroad Mobile Emissions to Road Segments for Air Toxics Modeling in Harris County, Texas. Transport Res Part D: Transport and Environ 9:139-150.

¹⁶¹ Cohen, J.; Cook, R.; Bailey, C.R.; Carr, E. (2005) Relationship between motor vehicle emissions of hazardous pollutants, roadway proximity, and ambient concentrations in Portland, Oregon. Environ Modeling & Software 20: 7-12.

¹⁶² Touma, J. S.; Isakov, V.; Ching, J.; Seigneur, C. (2006). Air Quality Modeling of Hazardous Air Pollutants: Current Status and Future Directions. J. Air and Waste Manage. Assoc., in press.

¹⁶³ Cook, R.; Beidler, A.; Touma, J.S.; Strum M. (2005) Preparing Highway Emissions Inventories for Urban Scale Modeling: A Case Study in Philadelphia. *Submitted to* Transportation Research Part D: Transport and Environment.

¹⁶⁴ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.

¹⁶⁵ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.

¹⁶⁶ 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. This document is available in Docket EPA-HQ-OAR-2005-0036.

¹⁶⁷ U.S. EPA. 2005. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Second External Review Draft). This document is available in Docket EPA-HQ-OAR-2005-0036.

¹⁶⁸ American Academy of Pediatrics, Committee on Environmental Health. 2004. "Ambient air pollution: health hazards to children." *Pediatrics* 114: 1699-1707.

¹⁶⁹ Mortimer, K. M.; Tager, I. B.; Dockery, D. W.; et al. 2000. "The effect of ozone on inner-city children with asthma: identification of susceptible subgroups." *Am. J. Respir. Crit. Care Med.* 162: 1838-1845.

-
- ¹⁷⁰ Gent, J. F.; Triche, E. W.; Holford, T. R.; et al. 2003. "Association of low-level ozone and fine particles with respiratory symptoms in children with asthma." *J. Am. Med. Assoc.* 290: 1859-1867.
- ¹⁷¹ Romieu, I.; Sienra-Monge, J. J.; Ramírez-Aguilar, M.; et al. 2002. "Antioxidant supplementation and lung functions among children with asthma exposed to high levels of air pollutants." *Am. J. Respir. Crit. Care Med.* 166: 703-709.
- ¹⁷² Romieu, I.; Sienra-Monge, J. J.; Ramírez-Aguilar, M.; et al. 2004. "Genetic polymorphism of *GSTM1* and antioxidant supplementation influence lung function in relation to ozone exposure in asthmatic children in Mexico City." *Thorax* 59: 8-10.
- ¹⁷³ Bates, D.V.; Baker-Anderson, M.; Sizto, R. 1990. "Asthma attack periodicity: a study of hospital emergency visits in Vancouver." *Environ. Res.* 51: 51-70.
- ¹⁷⁴ Thurston, G.D.; Ito, K.; Kinney, P.L.; Lippmann, M. 1992. "A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers." *J. Exposure Anal. Environ. Epidemiol.* 2:429-450.
- ¹⁷⁵ Thurston, G.D.; Ito, K.; Hayes, C.G.; et al. 1994. "Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols." *Environ. Res.* 65: 271-290.
- ¹⁷⁶ Lipfert, F.W.; Hammerstrom, T. 1992. "Temporal patterns in air pollution and hospital admissions." *Environ. Res.* 59: 374-399.
- ¹⁷⁷ Burnett, R.T.; Dales, R.E.; Raizenne, M.E.; et al. 1994. "Effects of low ambient levels of ozone and sulfates on the frequency of respiratory admissions to Ontario hospitals." *Environ. Res.* 65: 172-194.
- ¹⁷⁸ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹⁷⁹ Delfino, R. J.; Zeiger, R. S.; Seltzer, J. M.; Street, D. H. 1998. "Symptoms in pediatric asthmatics and air pollution: differences in effects by symptom severity, anti-inflammatory medication use and particulate averaging time." *Environ. Health Perspect.* 106: 751-761.
- ¹⁸⁰ Gold, D. R.; Damokosh, A. I.; Pope, C. A., III; et al. 1999. "Particulate and ozone pollutant effects on the respiratory function of children in southwest Mexico City." *Epidemiology* 10: 8-16.
- ¹⁸¹ Mortimer, K. M.; Tager, I. B.; Dockery, D. W.; et al. 2000. "The effect of ozone on inner-city children with asthma: identification of susceptible subgroups." *Am. J. Respir. Crit. Care Med.* 162: 1838-1845.
- ¹⁸² Newhouse, C. P.; Levetin, B. S.; Levetin, E. 2004. "Correlation of environmental factors with asthma and rhinitis symptoms in Tulsa, OK." *Ann. Allergy Asthma Immunol.* 92: 356-366.
- ¹⁸³ Koken, P. J.; Piver, W. T.; Ye, F.; et al. 2003. "Temperature, air pollution, and hospitalization for cardiovascular diseases among elderly people in Denver." *Environ. Health Perspect.* 111: 1312-1317.

-
- ¹⁸⁴ Gwynn, R. C.; Thurston, G. D. 2001. "The burden of air pollution: impacts among racial minorities." *Environ. Health Perspect. Suppl.* 109(4): 501-506.
- ¹⁸⁵ Wilson, A. M.; Wake, C. P.; Kelly, T.; Salloway, J. C. 2005. "Air pollution, weather, and respiratory emergency room visits in two northern New England cities: an ecological time-series study." *Environ. Res.* 97: 312-321.
- ¹⁸⁶ Weisel, C. P.; Cody, R. P.; Georgopoulos, P. G.; et al. 2002. "Concepts in developing health-based indicators for ozone." *Int. Arch. Occup. Environ. Health* 75: 415-422.
- ¹⁸⁷ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹⁸⁸ Devlin, R. B.; McDonnell, W. F.; Mann, R.; et al. 1991. "Exposure of humans to ambient levels of ozone for 6.6 hours causes cellular and biochemical changes in the lung." *Am. J. Respir. Cell Mol. Biol.* 4: 72-81.
- ¹⁸⁹ Koren, H. S.; Devlin, R. B.; Becker, S.; et al. 1991. "Time-dependent changes of markers associated with inflammation in the lungs of humans exposed to ambient levels of ozone." *Toxicol. Pathol.* 19: 406-411.
- ¹⁹⁰ Koren, H. S.; Devlin, R. B.; Graham, D. E.; et al. 1989. "Ozone-induced inflammation in the lower airways of human subjects." *Am. Rev. Respir. Dis.* 139: 407-415.
- ¹⁹¹ Schelegle, E.S.; Siefkin, A.D.; McDonald, R.J. 1991. "Time course of ozone-induced neutrophilia in normal humans." *Am. Rev. Respir. Dis.* 143:1353-1358.
- ¹⁹² Romieu, I.; Meneses, F.; Ruiz, S.; et al. 1996. "Effects of air pollution on the respiratory health of asthmatic children living in Mexico City." *Am. J. Respir. Crit. Care Med.* 154: 300-307.
- ¹⁹³ Romieu, I.; Meneses, F.; Ruiz, S.; et al. 1997. "Effects of intermittent ozone exposure on peak expiratory flow and respiratory symptoms among asthmatic children in Mexico City." *Arch. Environ. Health* 52: 368-376.
- ¹⁹⁴ Gielen, M. H.; Van Der Zee, S. C.; Van Wijnen, J. H.; et al. 1997. "Acute effects of summer air pollution on respiratory health of asthmatic children." *Am. J. Respir. Crit. Care Med.* 155: 2105-2108.
- ¹⁹⁵ Just, J.; Ségala, C.; Sahraoui, F.; et al. 2002. "Short-term health effects of particulate and photochemical air pollution in asthmatic children." *Eur. Respir. J.* 20: 899-906.
- ¹⁹⁶ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ¹⁹⁷ Hodgkin, J.E.; Abbey, D.E.; Euler, G.L.; Magie, A.R. 1984. "COPD prevalence in nonsmokers in high and low photochemical air pollution areas." *Chest* 86: 830-838.
- ¹⁹⁸ Euler, G.L.; Abbey, D.E.; Hodgkin, J.E.; Magie, A.R. 1988. "Chronic obstructive pulmonary disease symptom effects of long-term cumulative exposure to ambient levels of total oxidants and nitrogen dioxide in California Seventh-day Adventist residents." *Arch. Environ. Health* 43: 279-285.

-
- ¹⁹⁹ Abbey, D.E.; Petersen, F.; Mills, P.K.; Beeson, W.L. 1993. "Long-term ambient concentrations of total suspended particulates, ozone, and sulfur dioxide and respiratory symptoms in a nonsmoking population." *Arch. Environ. Health* 48: 33-46.
- ²⁰⁰ Frischer, T. M.; Kühr, J.; Pullwitt, A.; et al. 1993. "Ambient ozone causes upper airways inflammation in children." *Am. Rev. Respir. Dis.* 148: 961-964.
- ²⁰¹ Kinney, P. L.; Nilsen, D. M.; Lippmann, M.; et al. 1996. "Biomarkers of lung inflammation in recreational joggers exposed to ozone." *Am. J. Respir. Crit. Care Med.* 154: 1430-1435.
- ²⁰² U.S. EPA. 1996. Review of National Ambient Air Quality Standards for Ozone, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA452-R-96-007. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁰³ Brauer, M.; Brook, J. R. 1997. "Ozone personal exposures and health effects for selected groups residing in the Fraser Valley." In: Steyn, D. G.; Bottenheim, J. W., eds. The Lower Fraser Valley Oxidants/Pacific '93 Field Study. *Atmos. Environ.* 31: 2113-2121.
- ²⁰⁴ Chan, C.-C.; Wu, T.-H. 2005. "Effects of ambient ozone exposure on mail carriers' peak expiratory flow rates." *Environ. Health Perspect.* 113: 735-738.
- ²⁰⁵ Brauer, M.; Blair, J.; Vedal, S. 1996. "Effect of ambient ozone exposure on lung function in farm workers." *Am. J. Respir. Crit. Care Med.* 154: 981-987.
- ²⁰⁶ Korrick, S. A.; Neas, L. M.; Dockery, D. W.; et al. 1998. "Effects of ozone and other pollutants on the pulmonary function of adult hikers." *Environ. Health Perspect.* 106: 93-99.
- ²⁰⁷ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁰⁸ U.S. EPA. 1996. Review of National Ambient Air Quality Standards for Ozone, Assessment of Scientific and Technical Information, OAQPS Staff Paper, EPA452-R-96-007. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁰⁹ Brauer, M.; Blair, J.; Vedal, S. 1996. "Effect of ambient ozone exposure on lung function in farm workers." *Am. J. Respir. Crit. Care Med.* 154: 981-987.
- ²¹⁰ Korrick, S. A.; Neas, L. M.; Dockery, D. W.; et al. 1998. "Effects of ozone and other pollutants on the pulmonary function of adult hikers." *Environ. Health Perspect.* 106: 93-99.
- ²¹¹ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²¹² U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²¹³ Avol, E. L.; Trim, S. C.; Little, D. E.; et al. 1990. "Ozone exposure and lung function in children attending a southern California summer camp." Presented at: 83rd annual meeting and exhibition of the Air & Waste Management Association; June; Pittsburgh, PA. Pittsburgh, PA: Air & Waste Management Association; paper no. 90-150.3.
- ²¹⁴ Higgins, I. T. T.; D'Arcy, J. B.; Gibbons, D. I.; et al. 1990. "Effect of exposures to ambient ozone on ventilatory lung function in children." *Am. Rev. Respir. Dis.* 141: 1136-1146.

-
- ²¹⁵ Raizenne, M. E.; Burnett, R. T.; Stern, B.; et al. 1989. "Acute lung function responses to ambient acid aerosol exposures in children." *Environ. Health Perspect.* 79: 179-185.
- ²¹⁶ Raizenne, M.; Stern, B.; Burnett, R.; Spengler, J. 1987. "Acute respiratory function and transported air pollutants: observational studies." Presented at: 80th annual meeting of the Air Pollution Control Association; June; New York, NY. Pittsburgh, PA: Air Pollution Control Association; paper no. 87-32.6.
- ²¹⁷ Spektor, D. M.; Lippmann, M. 1991. "Health effects of ambient ozone on healthy children at a summer camp." In: Berglund, R. L.; Lawson, D. R.; McKee, D. J., eds. Tropospheric ozone and the environment: papers from an international conference; March 1990; Los Angeles, CA. Pittsburgh, PA: Air & Waste Management Association; pp. 83-89. (A&WMA transaction series no. TR-19).
- ²¹⁸ Spektor, D. M.; Thurston, G. D.; Mao, J.; et al. 1991. "Effects of single- and multiday ozone exposures on respiratory function in active normal children." *Environ. Res.* 55: 107-122.
- ²¹⁹ Spektor, D. M.; Lippman, M.; Lioy, P. J.; et al. 1988. "Effects of ambient ozone on respiratory function in active, normal children." *Am. Rev. Respir. Dis.* 137: 313-320.
- ²²⁰ Thurston, G. D.; Lippmann, M.; Scott, M. B.; Fine, J. M. 1997. "Summertime haze air pollution and children with asthma." *Am. J. Respir. Crit. Care Med.* 155: 654-660.
- ²²¹ US EPA. 2002. New Ozone Health and Environmental Effects References, Published Since Completion of the Previous Ozone AQCD. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²²² Thurston, G.D., M.L. Lippman, M.B. Scott, and J.M. Fine. 1997. "Summertime Haze Air Pollution and Children with Asthma." *American Journal of Respiratory Critical Care Medicine*, 155: 654-660.
- ²²³ Ostro, B, M. Lipsett, J. Mann, et al. 2001. "Air pollution and exacerbation of asthma in African-American children in Los Angeles." *Epidemiology* 12(2): 200-208.
- ²²⁴ Jalaludin, B. B.; Chey, T.; O'Toole, B. I.; et al. 2000. "Acute effects of low levels of ambient ozone on peak expiratory flow rate in a cohort of Australian children." *Int. J. Epidemiol.* 29: 549-557.
- ²²⁵ Desqueyroux, H.; Pujet, J.-C.; Prosper, M.; et al. 2002. "Short-term effects of low-level air pollution on respiratory health of adults suffering from moderate to severe asthma." *Environ. Res. A* 89: 29-37.
- ²²⁶ Just, J.; Ségala, C.; Sahraoui, F.; et al. 2002. "Short-term health effects of particulate and photochemical air pollution in asthmatic children." *Eur. Respir. J.* 20: 899-906.
- ²²⁷ McDonnell, W.F., D.E. Abbey, N. Nishino and M.D. Lebowitz. 1999. "Long-term ambient ozone concentration and the incidence of asthma in nonsmoking adults: the ahsmog study." *Environmental Research.* 80(2 Pt 1): 110-121.
- ²²⁸ McConnell, R.; Berhane, K.; Gilliland, F.; et al. 2002. "Asthma in exercising children exposed to ozone: a cohort study." *Lancet* 359: 386-391.

-
- ²²⁹ Burnett, R. T.; Smith_Doiron, M.; Stieb, D.; et al. 2001. "Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age." *Am. J. Epidemiol.* 153: 444-452.
- ²³⁰ Gouveia, N.; Fletcher, T. 2000. "Respiratory diseases in children and outdoor air pollution in Sao Paulo, Brazil: a time series analysis." *Occup. Environ. Med.* 57: 477-483.
- ²³¹ Petroschevsky, A.; Simpson, R. W.; Thalib, L.; Rutherford, S. 2001. "Associations between outdoor air pollution and hospital admissions in Brisbane, Australia." *Arch. Environ. Health* 56: 37-52.
- ²³² Wong, T. W.; Lau, T. S.; Yu, T. S.; et al. 1999. "Air pollution and hospital admissions for respiratory and cardiovascular diseases in Hong Kong." *Occup. Environ. Med.* 56: 679-683.
- ²³³ Burnett, R. T.; Smith-Doiron, M.; Stieb, D.; et al. 2001. "Association between ozone and hospitalization for acute respiratory diseases in children less than 2 years of age." *Am. J. Epidemiol.* 153: 444-452.
- ²³⁴ Chen, L.; Jennison, B. L.; Yang, W.; Omaye, S. T. 2000. "Elementary school absenteeism and air pollution." *Inhalation Toxicol.* 12: 997-1016.
- ²³⁵ Gilliland, F.D.; Berhane, K.; Rappaport, E.B.; et al. 2001. "The effects of ambient air pollution on school absenteeism due to respiratory illnesses." *Epidemiology* 12:43-54.
- ²³⁶ Park, H.; Lee, B.; Ha, E.-H.; et al. 2002. "Association of air pollution with school absenteeism due to illness." *Arch. Pediatr. Adolesc. Med.* 156: 1235-1239.
- ²³⁷ Chen, L.; Jennison, B. L.; Yang, W.; Omaye, S. T. 2000. "Elementary school absenteeism and air pollution." *Inhalation Toxicol.* 12: 997-1016.
- ²³⁸ Gilliland, F.D.; Berhane, K.; Rappaport, E.B.; et al. 2001. "The effects of ambient air pollution on school absenteeism due to respiratory illnesses." *Epidemiology* 12:43-54.
- ²³⁹ Park, H.; Lee, B.; Ha, E.-H.; et al. 2002. "Association of air pollution with school absenteeism due to illness." *Arch. Pediatr. Adolesc. Med.* 156: 1235-1239.
- ²⁴⁰ Samet, J. M.; Zeger, S. L.; Dominici, F.; et al. 2000. The national morbidity, mortality, and air pollution study. Part II: morbidity, mortality, and air pollution in the United States. Cambridge, MA: Health Effects Institute; research report no. 94, part II. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁴¹ Dominici, F.; McDermott, A.; Daniels, M.; et al. 2003. "Mortality among residents of 90 cities." In: Revised analyses of time-series studies of air pollution and health. Special report. Boston, MA: Health Effects Institute; pp. 9-24. Available: <http://www.healtheffects.org/Pubs/TimeSeries.pdf> [12 May, 2004]. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁴² Bell, M. L.; McDermott, A.; Zeger, S. L.; et al. 2004. "Ozone and short-term mortality in 95 US urban communities, 1987-2000." *JAMA J. Am. Med. Assoc.* 292: 2372-2378.
- ²⁴³ Huang, Y.; Dominici, F.; Bell, M. L. 2005. "Bayesian hierarchical distributed lag models for summer ozone exposure and cardio-respiratory mortality." *Environmetrics* 16: 547-562.

-
- ²⁴⁴ Schwartz, J. 2005. "How sensitive is the association between ozone and daily deaths to control for temperature?" *Am. J. Respir. Crit. Care Med.* 171: 627-631.
- ²⁴⁵ Gryparis, A.; Forsberg, B.; Katsouyanni, K.; et al. 2004. Acute effects of ozone on mortality from the "Air Pollution and Health: A European Approach" project. *Am J Respir Crit Care Med* 170: 1080-1087.
- ²⁴⁶ Touloumi, G.; Katsouyanni, K.; Zmirou, D.; et al. 1997. "Short-term effects of ambient oxidant exposure on mortality: a combined analysis within the APHEA project." *Am. J. Epidemiol.* 146: 177-185.
- ²⁴⁷ U.S. EPA. 2005. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Second External Review Draft). This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁴⁸ Bell, M. L.; Dominici, F.; Samet, J. M. 2005. "A meta-analysis of time-series studies of ozone and mortality with comparison to the national morbidity, mortality, and air pollution study." *Epidemiology* 16: 436-445.
- ²⁴⁹ Ito, K.; De Leon, S. F.; Lippmann, M. 2005. "Associations between ozone and daily mortality, analysis and meta-analysis." *Epidemiology* 16: 446-457.
- ²⁵⁰ Levy, J. I.; Chemerynski, S. M.; Sarnat, J. A. 2005. "Ozone exposure and mortality, an empiric Bayes metaregression analysis." *Epidemiology* 16: 458-468.
- ²⁵¹ U.S. EPA. 2005. Air Quality Criteria for Ozone and Related Photochemical Oxidants (Second External Review Draft). This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵² U.S. EPA. 2005. Technical Support Document for the Final Clean Air Interstate Rule - Air Quality Modeling. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵³ U.S. EPA. 2005. Technical Support Document for EPA's Multi-Pollutant Analysis, Methods for Projecting Air Quality Concentrations for EPA's Multi-Pollutant Analysis of 2005. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵⁴ U.S. EPA. 2005. CAIR-CAMR-CAVR Air Quality Results. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵⁵ U.S. EPA. 2005. Technical Support Document for the Proposed Mobile Source Air Toxics Rule – Air Quality Modeling. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵⁶ U.S. EPA. 2005. Technical Support Document for the Final Clean Air Interstate Rule - Air Quality Modeling. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵⁷ U.S. EPA. 2005. Clean Air Interstate Rule Emissions Inventory Technical Support Document. Available from: <http://www.epa.gov/cair/technical.html>. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵⁸ U.S. EPA. 2005. Technical Support Document for the Proposed Mobile Source Air Toxics Rule – Ozone Air Quality Modeling. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁵⁹ Pielke, R.A., W.R. Cotton, R.L. Walko, et al. 1992. "A Comprehensive Meteorological Modeling System – RAMS." *Meteor. Atmos. Phys.*, Vol. 49, pp. 69-91.

-
- ²⁶⁰ U.S. EPA. 1999. The Benefits and Costs of the Clean Air Act, 1990-2010. Prepared for U.S. Congress by U.S. EPA, Office of Air and Radiation, Office of Policy Analysis and Review, Washington, DC, November; EPA report no. EPA410-R-99-001. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁶¹ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁶² Winner, W.E., and C.J. Atkinson. 1986. "Absorption of air pollution by plants, and consequences for growth." *Trends in Ecology and Evolution* 1:15-18.
- ²⁶³ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁶⁴ Tingey, D.T., and Taylor, G.E. 1982. "Variation in plant response to ozone: a conceptual model of physiological events." In: *Effects of Gaseous Air Pollution in Agriculture and Horticulture* (Unsworth, M.H., Omrod, D.P., eds.) London, UK: Butterworth Scientific, pp. 113-138.
- ²⁶⁵ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁶⁶ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁶⁷ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁶⁸ Ollinger, S.V., J.D. Aber and P.B. Reich. 1997. "Simulating ozone effects on forest productivity: interactions between leaf canopy and stand level processes." *Ecological Applications* 7:1237-1251.
- ²⁶⁹ Winner, W.E., 1994. "Mechanistic analysis of plant responses to air pollution." *Ecological Applications*, 4(4):651-661.
- ²⁷⁰ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁷¹ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁷² Fox, S., and R. A. Mickler, eds. 1996. *Impact of Air Pollutants on Southern Pine Forests*. Springer-Verlag, NY, Ecol. Studies, Vol. 118, 513 pp.
- ²⁷³ National Acid Precipitation Assessment Program (NAPAP), 1991. *National Acid Precipitation Assessment Program. 1990 Integrated Assessment Report*. National Acid Precipitation Program. Office of the Director, Washington DC.
- ²⁷⁴ De Steiguer, J., J. Pye, C. Love. 1990. "Air Pollution Damage to U.S. Forests." *Journal of Forestry*, Vol 88(8) pp. 17-22.
- ²⁷⁵ Pye, J.M. 1988. "Impact of ozone on the growth and yield of trees: A review." *Journal of Environmental Quality* 17 pp.347-360.

-
- ²⁷⁶ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁷⁷ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁷⁸ McBride, J.R., P.R. Miller, and R.D. Laven. 1985. "Effects of oxidant air pollutants on forest succession in the mixed conifer forest type of southern California." In: Air Pollutants Effects On Forest Ecosystems, Symposium Proceedings, St. P, 1985, p. 157-167.
- ²⁷⁹ Miller, P.R., O.C. Taylor, R.G. Wilhour. 1982. Oxidant air pollution effects on a western coniferous forest ecosystem. Corvallis, OR: U.S. Environmental Protection Agency, Environmental Research Laboratory (EPA600-D-82-276). This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁸⁰ U.S. EPA. 1996. Air Quality Criteria for Ozone and Related Photochemical Oxidants, EPA600-P-93-004aF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁸¹ Kopp, R. J.; Vaughn, W. J.; Hazilla, M.; Carson, R. 1985. "Implications of environmental policy for U.S. agriculture: the case of ambient ozone standards." *J. Environ. Manage.* 20:321-331.
- ²⁸² Adams, R. M.; Hamilton, S. A.; McCarl, B. A. 1986. "The benefits of pollution control: the case of ozone and U.S. agriculture." *Am. J. Agric. Econ.* 34: 3-19.
- ²⁸³ Adams, R. M.; Glycer, J. D.; Johnson, S. L.; McCarl, B. A. 1989. "A reassessment of the economic effects of ozone on U.S. agriculture." *JAPCA* 39:960-968.
- ²⁸⁴ Abt Associates, Inc. 1995. Urban ornamental plants: sensitivity to ozone and potential economic losses. U.S. EPA, Office of Air Quality Planning and Standards, Research Triangle Park. Under contract to RADIANT Corporation, contract no. 68-D3-0033, WA no. 6. pp. 9-10.
- ²⁸⁵ U.S. EPA. 2004. Air Quality Criteria for Particulate Matter. EPA/600/P-99/002aF and EPA/600/P-99/002bF. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁸⁶ U.S. EPA. 2005. Review of the National Ambient Air Quality Standard for Particulate Matter: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. EPA-452/R-05-005. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁸⁷ Laden F; Neas LM; Dockery DW; et al. 2000. "Association of fine particulate matter from different sources with daily mortality in six U.S. cities." *Environ Health Perspectives* 108(10):941-947.
- ²⁸⁸ Schwartz J; Laden F; Zanobetti A. 2002. "The concentration-response relation between PM(2.5) and daily deaths." *Environ Health Perspect* 110(10): 1025-1029.
- ²⁸⁹ Janssen NA; Schwartz J; Zanobetti A.; et al. 2002. "Air conditioning and source-specific particles as modifiers of the effect of PM10 on hospital admissions for heart and lung disease." *Environ Health Perspect* 110(1):43-49.
- ²⁹⁰ Kunzli, N; Medina, S; Kaiser, R; et al. 2001. "Assessment of deaths attributable to air pollution: should we use risk estimates based on time series or on cohort studies?" *Am J Epidemiol* 153(11): 1050-1055.

-
- ²⁹¹ Dockery, DW; Pope, CA, III; Xu, X; et al. 1993. “An association between air pollution and mortality in six U.S. cities.” *N Engl J Med* 329:1753-1759.
- ²⁹² Pope, CA, III; Burnett, RT; Thun, MJ; Calle, EE; et al. 2002. “Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution.” *J Am Med Assoc* 287: 1132-1141.
- ²⁹³ Krewski, D; Burnett, RT; Goldberg, M S; et al. 2000. “Reanalysis of the Harvard Six Cities study and the American Cancer Society study of particulate air pollution and mortality. A special report of the Institute's Particle Epidemiology Reanalysis Project.” Cambridge, MA: Health Effects Institute. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁹⁴ Jerrett, M; Burnett, RT; Ma, R; et al. 2005. “Spatial Analysis of Air Pollution and Mortality in Los Angeles.” *Epidemiology*. 16(6):727-736.
- ²⁹⁵ Künzli, N.; Jerrett, M.; Mack, W.J.; et al. 2005. “Ambient air pollution and atherosclerosis in Los Angeles.” *Environ Health Perspect*. 113:201-206.
- ²⁹⁶ Riediker, M.; Cascio, W.E.; Griggs, T.R.; et al. 2004. “Particulate matter exposure in cars is associated with cardiovascular effects in healthy young men.” *Am J Respir Crit Care Med* 169: 934–940.
- ²⁹⁷ U.S. EPA. 2005. Technical Support Document for the Final Clean Air Interstate Rule - Air Quality Modeling. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁹⁸ U.S. EPA 2005. Technical Support Document for EPA’s Multi-Pollutant Analysis, Methods for Projecting Air Quality Concentrations for EPA’s Multi-Pollutant Analysis of 2005. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ²⁹⁹ U.S. EPA. 2005. CAIR-CAMR-CAVR Air Quality Results. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³⁰⁰ U.S. EPA. 2005. Review of the National Ambient Air Quality Standard for Particulate Matter: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. EPA-452/R-05-005. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³⁰¹ U.S. EPA. 1993. Effects of the 1990 Clean Air Act Amendments on Visibility in Class I Areas: An EPA Report to Congress. EPA452-R-93-014.
- ³⁰² U.S. EPA (2002) Latest Findings on National Air Quality – 2002 Status and Trends. EPA 454/K-03-001. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³⁰³ National Park Service. Air Quality in the National Parks, Second edition. NPS, Air Resources Division. D 2266. September 2002. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³⁰⁴ U.S. EPA (2002) Latest Findings on National Air Quality – 2002 Status and Trends. EPA 454/K-03-001. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³⁰⁵ U.S. EPA (2005). Technical Support Document for the Final Clean Air Interstate Rule - Air Quality Modeling. This document is available in Docket EPA-HQ-OAR-2005-0036.

-
- ³⁰⁶ U.S. EPA (2000) Deposition of Air Pollutants to the Great Waters: Third Report to Congress. Office of Air Quality Planning and Standards. EPA-453/R-00-0005.
- ³⁰⁷ U.S. EPA (2004) National Coastal Condition Report II. Office of Research and Development/Office of Water. EPA-620/R-03/002.
- ³⁰⁸ Gao, Y., E.D. Nelson, M.P. Field, et al. 2002. "Characterization of atmospheric trace elements on PM_{2.5} particulate matter over the New York-New Jersey harbor estuary." *Atmos. Environ.* 36: 1077-1086.
- ³⁰⁹ Kim, G., N. Hussain, J.R. Scudlark, and T.M. Church. 2000. "Factors influencing the atmospheric depositional fluxes of stable Pb, ²¹⁰Pb, and ⁷Be into Chesapeake Bay." *J. Atmos. Chem.* 36: 65-79.
- ³¹⁰ Lu, R., R.P. Turco, K. Stolzenbach, et al. 2003. "Dry deposition of airborne trace metals on the Los Angeles Basin and adjacent coastal waters." *J. Geophys. Res.* 108(D2, 4074): AAC 11-1 to 11-24.
- ³¹¹ Marvin, C.H., M.N. Charlton, E.J. Reiner, et al. 2002. "Surficial sediment contamination in Lakes Erie and Ontario: A comparative analysis." *J. Great Lakes Res.* 28(3): 437-450.
- ³¹² Smith, W.H. 1991. "Air pollution and Forest Damage." *Chemical Engineering News*, 69(45): 30-43.
- ³¹³ Gawel, J.E.; Ahner, B.A.; Friedland, A.J.; and Morel, F.M.M. 1996. "Role for heavy metals in forest decline indicated by phytochelatin measurements." *Nature*, 381: 64-65.
- ³¹⁴ Cotrufo, M.F.; DeSanto, A.V.; Alfani, A.; et al. 1995. "Effects of urban heavy metal pollution on organic matter decomposition in *Quercus ilix* L. woods." *Environmental Pollution*, 89: 81-87.
- ³¹⁵ Niklinska, M.; Laskowski, R.; Maryanski, M. 1998. "Effect of heavy metals and storage time on two types of forest litter: basal respiration rate and exchangeable metals." *Ecotoxicological Environmental Safety*, 41: 8-18.
- ³¹⁶ Mason, R.P. and Sullivan, K.A. 1997. "Mercury in Lake Michigan." *Environmental Science & Technology*, 31: 942-947. (from Delta Report "Atmospheric deposition of toxics to the Great Lakes")
- ³¹⁷ Landis, M.S. and Keeler, G.J. 2002. "Atmospheric mercury deposition to Lake Michigan during the Lake Michigan Mass Balance Study." *Environmental Science & Technology*, 21: 4518-24.
- ³¹⁸ U.S. EPA. 2000. EPA453/R-00-005, "Deposition of Air Pollutants to the Great Waters: Third Report to Congress," Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³¹⁹ Callender, E. and Rice, K.C. 2000. "The Urban Environmental Gradient: Anthropogenic Influences on the Spatial and Temporal Distributions of Lead and Zinc in Sediments." *Environmental Science & Technology*, 34: 232-238.
- ³²⁰ Rice, K.C. 1999. "Trace Element Concentrations in Streambed Sediment Across the Conterminous United States." *Environmental Science & Technology*, 33: 2499-2504.

-
- ³²¹ Ely, JC; Neal, CR; Kulpa, CF; et al. 2001. "Implications of Platinum-Group Element Accumulation along U.S. Roads from Catalytic-Converter Attrition." *Environ. Sci. Technol.* 35: 3816-3822.
- ³²² U.S. EPA. 1998. EPA454/R-98-014, "Locating and Estimating Air Emissions from Sources of Polycyclic Organic Matter," Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³²³ U.S. EPA. 1998. EPA454/R-98-014, "Locating and Estimating Air Emissions from Sources of Polycyclic Organic Matter," Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³²⁴ Simcik, M.F.; Eisenreich, S.J.; Golden, K.A.; et al. 1996. "Atmospheric Loading of Polycyclic Aromatic Hydrocarbons to Lake Michigan as Recorded in the Sediments." *Environmental Science and Technology*, 30: 3039-3046.
- ³²⁵ Simcik, M.F.; Eisenreich, S.J.; and Lioy, P.J. 1999. "Source apportionment and source/sink relationship of PAHs in the coastal atmosphere of Chicago and Lake Michigan." *Atmospheric Environment*, 33: 5071-5079.
- ³²⁶ Arzayus, K.M.; Dickhut, R.M.; and Canuel, E.A. 2001. "Fate of Atmospherically Deposited Polycyclic Aromatic Hydrocarbons (PAHs) in Chesapeake Bay." *Environmental Science & Technology*, 35, 2178-2183.
- ³²⁷ Park, J.S.; Wade, T.L.; and Sweet, S. 2001. "Atmospheric distribution of polycyclic aromatic hydrocarbons and deposition to Galveston Bay, Texas, USA." *Atmospheric Environment*, 35: 3241-3249.
- ³²⁸ Poor, N.; Tremblay, R.; Kay, H.; et al. 2002. "Atmospheric concentrations and dry deposition rates of polycyclic aromatic hydrocarbons (PAHs) for Tampa Bay, Florida, USA." *Atmospheric Environment* 38: 6005-6015.
- ³²⁹ Arzayus, K.M.; Dickhut, R.M.; and Canuel, E.A. 2001. "Fate of Atmospherically Deposited Polycyclic Aromatic Hydrocarbons (PAHs) in Chesapeake Bay." *Environmental Science & Technology*, 35, 2178-2183.
- ³³⁰ U.S. EPA. 2000. EPA453/R-00-005, "Deposition of Air Pollutants to the Great Waters: Third Report to Congress," Office of Air Quality Planning and Standards, Research Triangle Park, North Carolina. This document is available in Docket EPA-HQ-OAR-2005-0036.
- ³³¹ Van Metre, P.C.; Mahler, B.J.; and Furlong, E.T. 2000. "Urban Sprawl Leaves its PAH Signature." *Environmental Science & Technology*, 34: 4064-4070.
- ³³² Cousins, I.T.; Beck, A.J.; and Jones, K.C. 1999. "A review of the processes involved in the exchange of semi-volatile organic compounds across the air-soil interface." *The Science of the Total Environment*, 228: 5-24.
- ³³³ Tuhackova, J., Cajthaml, T.; Novak, K.; et al. 2001. "Hydrocarbon deposition and soil microflora as affected by highway traffic." *Environmental Pollution*, 113: 255-262.
- ³³⁴ U.S. EPA (2005) Review of the National Ambient Air Quality Standard for Particulate Matter: Policy Assessment of Scientific and Technical Information, OAQPS Staff Paper. EPA-452/R-05-005. This document is available in Docket EPA-HQ-OAR-2005-0036.

-
- ³³⁵ Hoek, G.; Brunekreef, B.; Goldbohm, S.; et al. (2002) Association between mortality and indicators of traffic-related air pollution in the Netherlands: a cohort study. *Lancet* 360: 1203-1209.
- ³³⁶ Finkelstein, M.M.; Jerrett, M.; Sears, M.R. (2004) Traffic air pollution and mortality rate advancement periods. *Am J Epidemiol* 160: 173-177.
- ³³⁷ Maheswaran, M.; Elliott, P. (2003) Stroke mortality associated with living near main roads in England and Wales. A geographical study. *Stroke* 34: 2776-2780.
- ³³⁸ Roemer, W.H.; van Wijnen, J.H. (2001) Daily mortality and air pollution along busy streets in Amsterdam, 1987-1998. *Epidemiol* 12: 649-653.
- ³³⁹ Heinrich, J.; Wichmann, H-E. (2004) Traffic related pollutant in Europe and their effect on allergic disease. *Current Opin Clinical Epidemiol* 4: 341-348.
- ³⁴⁰ Ryan, P.H.; LeMasters, G.; Biagnini, J.; et al. (2005) Is it traffic type, volume, or distance? Wheezing in infants living near truck and bus traffic. *J Allergy Clin Immunol* 116: 279-284.
- ³⁴¹ Kim, J.J.; Smorodinsky, S.; Lipsett, M.; et al. (2004) Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. *Am J Respir Crit Care Med* 170: 520-526.
- ³⁴² Lin, S.; Munsie, J.P.; Hwang, S-A.; et al. (2002) Childhood asthma hospitalization and residential exposure to state route traffic. *Environ Res* 88: 73-81.
- ³⁴³ English, P.; Neutra, R.; Scalf, R.; et al. (1999) Examining associations between childhood asthma and traffic flow using a geographic information system. *Environ Health Perspect* 107: 761-768.
- ³⁴⁴ Garshick, E.; Laden, F.; Hart, J.E.; Caron, A. (2003) Residence near a major road and respiratory symptoms in U.S. veterans. *Epidemiology* 14: 728-736.
- ³⁴⁵ Ware, J.H.; Spengler, J.D.; Neas, L.M.; et al. (1993) Respiratory and irritant health effects of ambient volatile organic compounds. *Am J Epidemiol* 137: 1287-1301.
- ³⁴⁶ Heinrich, J.; Wichmann, H-E. (2004) Traffic related pollutant in Europe and their effect on allergic disease. *Current Opin Clinical Epidemiol* 4: 341-348.
- ³⁴⁷ Kim, J.J.; Smorodinsky, S.; Lipsett, M.; et al. (2004) Traffic-related air pollution near busy roads: the East Bay Children's Respiratory Health Study. *Am J Respir Crit Care Med* 170: 520-526.
- ³⁴⁸ Gauderman, W.J.; Avol, E.; Lurmann, F.; et al. (2005) Childhood asthma and exposure to traffic and nitrogen dioxide. *Epidemiol* 16: 737-43.
- ³⁴⁹ Heinrich, J.; Topp, R.; Gehring, U.; Thefeld, W. (2005) Traffic at residential address, respiratory health, and atopy in adults: the National German Health Survey 1998. *Environ Res* 98: 240-249.
- ³⁵⁰ Miller, R.L.; Garfinkel, R.; Horton, M.; et al. (2004) Polycyclic aromatic hydrocarbons, environmental tobacco smoke, and respiratory symptoms in an inner-city birth cohort. *Chest* 126: 1071-1078.

-
- ³⁵¹ Tonne, C.C.; Whyatt, R.M.; Camann, D.E.; et al. (2004) Predictors of personal polycyclic aromatic hydrocarbon exposures among pregnant minority women in New York City. *Environ Health Perspect* 112: 754-769.
- ³⁵² Peters, A.; von Klot, S.; Heier, M.; et al. (2004) Exposure to traffic and the onset of myocardial infarction. *N Engl J Med* 351: 1721-1730.
- ³⁵³ Riediker, M.; Cascio, W.E.; Griggs, T.R.; et al. (2003) Particulate matter exposures in cars is associated with cardiovascular effects in healthy young men. *Am J Respir Crit Care Med* 169: (page nos.?)
- ³⁵⁴ Schwartz, J.; Litonjua, L.; Suh, H.; et al. (2005) Traffic related pollution and heart rate variability in a panel of elderly subjects. *Thorax* 60: 455-461.
- ³⁵⁵ Gold, D.R.; Litonjua, A.A.; Zanobetti, A.; et al. (2005) Air pollution and ST-segment depression in elderly subjects. *Environ Health Perspect* 113: 883-887.
- ³⁵⁶ Finkelstein, M.M.; Jerrett, M.; Sears, M.R. (2005) Environmental inequality and circulatory disease mortality gradients. *J Epidemiol Community Health* 59: 481-486.
- ³⁵⁷ Wilhelm, M.; Ritz, B. (2002) Residential proximity to traffic and adverse birth outcomes in Los Angeles County, California, 1994-1996. *Environ Health Perspect* 111:207-216.
- ³⁵⁸ Ritz B; Yu F. (1999) The effect of ambient carbon monoxide on low birth weight among children born in southern California between 1989 and 1993. *Environ Health Perspect* 107:17-25.
- ³⁵⁹ Ritz B; Yu F; Capa G; Fruin S. (2000) Effect of air pollution on premature birth among children born in southern California between 1989 and 1993. *Epidemiology* 11:502-511.
- ³⁶⁰ Ritz B; Yu F; Fruin S; et al. (2002) Ambient air pollution and risk of birth defects in Southern California. *Am J Epidemiol* 155:17-25.
- ³⁶¹ Perera, F.P.; Rauh, V.; Tsai, W-Y.; et al. (2002) Effect of transplacental exposure to environmental pollutants on birth outcomes in a multiethnic population. *Environ Health Perspect* 111: 201-205.
- ³⁶² U. S. EPA (2005) Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens. Report No. EPA/630/R-03/003F. This document is available in Docket EPA-HQ-OAR-2005-0036. <http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=116283>
- ³⁶³ U. S. EPA. 2002. Toxicological Review of Benzene (Noncancer effects). Report No. EPA 635/R-02/001-F.
- ³⁶⁴ Savitz, D.A.; Feingold, L. (1989) Association of childhood cancer with residential traffic density. *Scand J Work Environ Health* 15: 360-363.
- ³⁶⁵ Pearson, R.L.; Wachtel, H.; Ebi, K.L. (2000) Distance-weighted traffic density in proximity to a home is a risk factor for leukemia and other childhood cancers. *J Air Waste Manage Assoc* 50: 175-180.
- ³⁶⁶ Feychting, M.; Svensson, D.; Ahlbom, A. (1998) Exposure to motor vehicle exhaust and childhood cancer. *Scand J Work Environ Health* 24(1): 8-11.

-
- ³⁶⁷ Langholz, B.; Ebi, K.L.; Thomas, D.C.; et al. (2002) Traffic density and the risk of childhood leukemia in a Los Angeles case-control study. *Ann Epidemiol* 12: 482-487.
- ³⁶⁸ Raaschou-Nielsen, O.; Hertel, O.; Thomsen, B.L.; Olsen, J.H. (2001) Air pollution from traffic at the residence of children with cancer. *Am J Epidemiol* 153: 433-443.
- ³⁶⁹ Reynolds, P.; Von Behren, J.; Gunier, R.B.; et al. (2003) Childhood cancer incidence rates and hazardous air pollutants in California: an exploratory analysis. *Environ Health Perspect* 111: 663-668.
- ³⁷⁰ Reynolds, P.; Von Behren, J.; Gunier, R.B.; et al. (2004) Residential exposure to traffic in California and childhood cancer. *Epidemiol* 15: 6-12.
- ³⁷¹ Knox, E.G. (2005) Oil combustion and childhood cancers. *J Epidemiol Community Health* 59: 755-760.
- ³⁷² United States Census Bureau. (2004) American Housing Survey web page. [Online at <http://www.census.gov/hhes/www/housing/ahs/ahs.html>]
- ³⁷³ This statistic is based on the odds ratio of being near major transportation sources, compared to not being near transportation sources for housing units located in different geographic regions.
- ³⁷⁴ Garshick, E.; Laden, F.; Hart, J.E.; Caron, A. (2003) Residence near a major road and respiratory symptoms in U.S. veterans. *Epidemiol* 14: 728-736.
- ³⁷⁵ Green, R.S.; Smorodinsky, S.; Kim, J.J.; McLaughlin, R.; Ostro, B. (2004) Proximity of California public schools to busy roads. *Environ Health Perspect* 112: 61-66.
- ³⁷⁶ Gunier, R.B.; Hertz, A.; Von Behren, J.; Reynolds, P. (2003) Traffic density in California: socioeconomic and ethnic differences among potentially exposed children. *J Expos Anal Environ Epidemiol* 13: 240-246.