Regulatory Impact Analysis

Control of Hazardous Air Pollutants from Mobile Sources

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

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3.1 Air Quality and Exposure Measurements

3.1.1 Ambient Monitoring

Ambient air toxics data are 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 have 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. In 2004, EPA published a draft National Air Toxics Monitoring Strategy to advance this goal.[3] The National Air Toxics Trends Station (NATTS) monitoring network is currently in place, consisting of 23 sites in 22 urban areas nationally.[4]

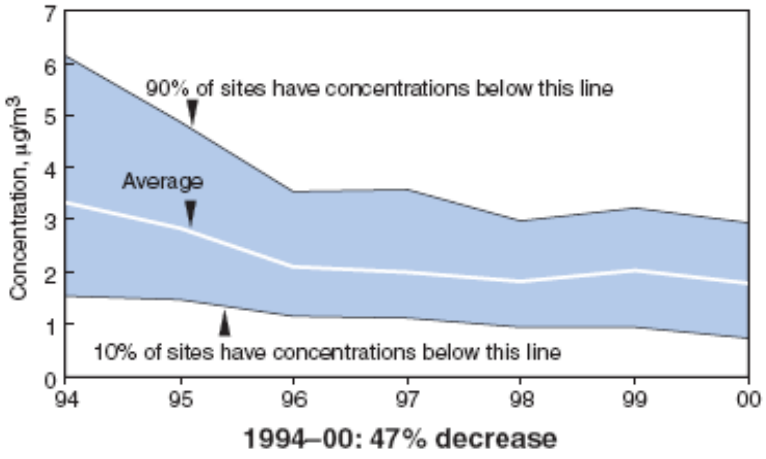
The available monitoring data help air pollution control agencies track trends in toxic air pollutants in various locations around the country. EPA conducted a pilot city monitoring project in 2001 that included sampling in four urban areas and six small city/rural areas (see Figure 3.1-1). This program 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 the NATTS, and numerous community-scale monitoring studies.[5] 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.

EPA recently completed a study of the spatial and temporal trends in ambient air toxics data within the NATTS and other networks from 1990-2003.[6] Most data came from urban monitors. Nationally, citywide average annual concentrations of benzene, formaldehyde, and

acetaldehyde, varied by about a factor of five, and 1,3-butadiene by more than 10 times. The coefficient of variation[a] of annual average concentrations between different monitors within the same city averaged 0.37 for benzene, 0.45 for 1,3-butadiene. Between cities, the coefficient of variation could vary substantially. Different pollutants showed different seasonal trends, with average concentrations of benzene and 1,3-butadiene being highest in colder seasons, while average concentrations of formaldehyde and acetaldehyde were higher during warm seasons, reflecting the high photochemical production of aldehydes. The concentrations of benzene, butadiene, and acetaldehyde fell substantially over different time periods. From 1990-2003, benzene concentrations fell by 57%. Insufficient data existed in earlier years to analyze 1,3-butadiene and acetaldehyde. Formaldehyde increased by 134% over this period, although changes in sampling methodology at some sites around 1995 make this quantification suspect. From 1995-2003, the average annual changes in benzene, 1,3-butadiene, formaldehyde, and acetaldehyde were -47%, -54%, +11%, and -12%. From 1998-2003, the changes were -21%, -46%, +17%, and -4%, respectively. For benzene, these trends were statistically significant, but formaldehyde and acetaldehyde trends after 1995 were not.

One recent publication evaluated the trends in ambient concentrations of benzene and 1,3-butadiene in the Houston, TX metropolitan area.[7] Using data from two air monitoring networks, a state-based network and the Photochemical Assessment Monitoring Systems, the study constructed a statistical model, controlling for meteorology and seasonality, to evaluate trends in ambient toxics over the 1997-2004 time period. Averaged over state monitoring sites with data across the time period, the model estimated 1.7% and 3.7% average annual decrease in ambient benzene and 1,3,-butadiene, respectively. Mobile source and point source emission reductions contributed roughly equally to this change. Examining long-term average concentration differences across monitoring sites, benzene varied by roughly two-fold across monitors while 1,3-butadiene varied roughly six-fold across monitors. This may be attributable to the substantial contribution of industrial sources to the local 1,3-butadiene inventory, while the benzene inventory is dominated by mobile sources. The study also evaluated differences in weekday and weekend concentration, with the model predicting significant meteorologically-adjusted concentration weekday increases relative to weekend only during the 6-9 A.M. morning rush hour period.

A recent study from San Francisco, CA evaluated trends in ambient benzene emissions and air quality throughout the 1990's.[8] The study noted substantial decreases in benzene emissions and ambient concentrations. Unique to the study was the attribution of components of these reduction to specific regulatory programs related to vehicles and fuels. In particular, the study attributed a 1-year drop of 54% in benzene emission rates to a combination of the introduction of California phase 2 RFG (attributed a 50% decrease) and fleet turnover (attributed a 4% decrease). During the same year (1995-1996), a 42% reduction in the ambient concentration of benzene was also observed. Fleet turnover effects were shown to be cumulative over time. The study indicates that in San Francisco both fuel and vehicle effects are important

[a] A "coefficient of variation" is a measure of variability, and for a set of data is defined as the standard deviation over the mean.

contributors to changes in emissions and ambient concentrations of benzene.

New York State has a systematic program in place that has been measuring air toxics since the 1990s.[9] 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 and moving average (KZ filter) on the concentration data. The analysis indicated that site-specific ambient concentration levels of benzene decreased by 50% or more 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.

A similar downward trend was observed in California. In California, the Air Resources Board (ARB) maintains an Almanac of Emissions and Air Quality.[10] 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.

Another recent evaluation of hazardous air pollutant (HAP) trends was conducted for selected metropolitan areas.[11] 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 in reducing ambient concentrations of air toxics over time. Additional reductions are expected with the implementation of additional regulatory measures such as this one. It should be noted that due to the limited spatial and temporal coverage of air toxics monitoring networks, using ambient monitors to represent exposure adds substantial uncertainty in exposure assessment.

Table 3.1-1. Site Descriptions of the Monitoring Stations Along with Mean Benzene Concentration from 1990-2003 and 2001-2003, for Monitoring Stations in New York State.

	Lackawanna	Eastern District High School	Troy	Niagara Falls	Whiteface Mountain Base Lodge
Site Character	Industrial	Urban	Small Urban	Urban Industrial	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
Period 1990-2003 Mean Concentration ($\mu g/m^3$)	5.09	2.85	2.31	1.80	0.86
Period 2001-2003 Mean Concentration ($\mu g/m^3$)	2.26	2.05	1.68	1.08	0.54

3.1.2 Population-Based (Representative) Exposure Measurements

In addition to measurements of outdoor concentrations, an important component of understanding human exposure to air toxics is the body of studies that employ survey techniques to assess microenvironmental and 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.[12] 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 $\mu g/m^3$, with indoor air concentrations averaging 7.21 µg/m3. Outdoor air concentrations averaged 3.61 $\mu g/m^3$. Personal air concentrations were significantly associated with indoor air concentrations, as well as blood concentrations. The preliminary results of the NHEXAS pilot study in Arizona, another study area, indicate that among the 179 statistically-sampled homes, median indoor concentrations were 1.3 $\mu g/m^3$ during the mid-1990's, while outdoor concentrations were 1.0 $\mu g/m^3$.[13] 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.[14] 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, were also obtained for the study area. Overall, median outdoor modeled concentrations of benzene and other fuel-related VOCs corresponded well with measured data in the area (correlation coefficient of median VOC concentrations = 0.97). Average personal exposure to benzene was 4.06 $\mu g/m^3$, while 95th percentile values were 7.30 $\mu g/m^3$. For indoors, the respective values were 3.70 and 8.34 $\mu g/m^3$, while for outdoors the values were 1.84 and 3.14 $\mu g/m^3$. 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 Relationships of 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.[15] 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 $\mu g/m^3$, with a 95th percentile of 10.7 $\mu g/m^3$. Respective statistics

for indoor air were 3.50 μg/m^3 and 10.0 μg/m^3, while outdoor statistics were 2.15 and 5.16 μg/m^3.

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,[16] 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 μg/m^3, with the data skewed toward higher concentrations. The 95th percentile concentration was 12.7 μg/m^3. Homes with attached garages had significantly higher concentrations of benzene indoors ($p < 0.0001$). 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 μg/m^3, with a 95th percentile of 9.1 μg/m^3. Indoor concentrations in the intensive period averaged 3.9 μg/m^3 and outdoor averaged 3.3 μg/m^3. Regression analysis indicated that personal exposures generally were higher than the time-weighted average of indoor and outdoor air. Furthermore, personal exposures to benzene and toluene were elevated for children living in a home with an attached garage, but only the relationship for toluene was significant at the 95% confidence level.

Table 3.1-2. Benzene Emission (Tons Per Year) and Concentration (μg/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

In another study, students recruited from an inner-city school in Minneapolis, MN participated in an exposure study called SHIELD.[17] 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 $\mu g/m^3$ in spring and 2.1 $\mu g/m^3$ in winter.[18]

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.[19] Average winter benzene personal concentrations were 4.70 $\mu g/m^3$, while indoor and outdoor concentrations averaged 5.97 and 2.55 $\mu g/m^3$. Average indoor concentrations exceeding average personal concentrations is unique to the TEACH winter results. Summer values were 3.09, 1.75, and 1.31 $\mu g/m^3$, respectively. The authors noted that VOC concentrations within the city tracked traffic patterns. There was no substantial evidence for indoor sources of benzene.[20] In a subsequent publication, personal exposure concentrations for both cities were reported, averaged across both seasons. New York City average exposure concentrations were 3.82 $\mu g/m^3$, while Los Angeles average exposure concentrations were 4.64 $\mu g/m^3$.[21]

Overall, these studies show that personal and indoor concentrations of benzene and other VOCs are substantially higher than those found outdoors (see Table 3.1-3). In general, these differences are statistically significant. Some of the factors leading to these elevated concentrations are likely 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.

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.

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

Location	Year(s)	Includes Smokers	Personal Average (µg/m^3)	"Upper Bound" (µg/m^3)	Indoor Average (µg/m^3)	Outdoor Average (µg/m^3)	Reference
EPA Region 5	1995-1996	Yes	7.52	13.71[b]	7.21	3.61	Clayton et al. (1999)
Baltimore, MD	2000-2001	No	4.06	7.30[c]	3.70	1.84	Payne-Sturges et al. (2004)
Elizabeth, NJ, Houston, TX, Los Angeles CA	1999-2001	No	3.64	10.7[c], 27.4[g]	3.50	2.15	*Weisel et al. (2005)*
Elizabeth, NJ, Houston, TX, Los Angeles CA	*1999-2001*	*No*	*4.16*	*12.0[c], 43.6[g]*	*N/R[h]*	*N/R[h]*	*Weisel et al. (2005)*
Minneapolis - St. Paul, MN	*1997*	*Yes[e]*	*4.8*	*9.1*	*3.9*	*3.3*	*Adgate et al. (2004a)*
Minneapolis, MN	*2000*	*Yes[e]*	*2.1 Winter 1.5 Spring*	*6.5 Winter[b] 4.2 Spring[b]*	*2.2 Winter 2.1 Spring*	*1.3 Winter 1.1 Spring*	*Adgate et al. (2004b)*
New York, NY	*1999-2000*	*No*	*4.7 Winter 3.1 Summer 3.8 Total*	*11.4 Winter[d] 7.0 Summer[d] 12.3 Total[f]*	*6.0 Winter 1.8 Summer 3.6 Total*	*2.5 Winter 1.3 Summer 1.8 Total*	*Kinney et al. (2002); Sax et al. (2006)*
Los Angeles, CA	*1999-2000*	*No*	*4.64*	*11.27*	*3.87*	*3.32*	*Sax et al. (2006)*

[a] Children's studies in italics
[b] 90th percentile
[c] 95th percentile
[d] Mean +2 standard deviations
[e] Smoking in homes
[f] Maximum measured value
[g] 99th percentile
[h] Not reported

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.[22]

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 $d_p \leq 0.05$ μm 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. $PM_{2.5}$ is particulate matter with an aerodynamic diameter less than or equal to 2.5 μm. $PM_{2.5}$ settles quite slowly in the atmosphere relative to coarse and supercoarse particles. Normal weather patterns can keep $PM_{2.5}$ airborne for several hours to several days and enable these particles to transport hundreds of miles. $PM_{2.5}$ 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.[23,24] 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.[25,26] 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.[27,28] 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.[29] 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.[30,31]

An increase in coarse particles near roads could originate from engine deterioration, brake and tire wear, and secondary aerosol formation.[32,33,34,35] Engine deterioration is generally a function of vehicle age and maintenance condition. Brake wear emissions are highly dependent on brake pad materials.[36] 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.[37] Studies suggest that ambient temperature variation can also affect particle number gradients near roads substantially.[38] Wind direction also affects traffic-related air pollution mass concentrations inside and outside of schools near motorways.[39,40] 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 methods 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.

Air quality monitoring is one way of evaluating pollutant concentrations at locations near sources such as roadways. 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.[41] 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. In a subsequent study, proximity to major roadways, meteorology, and photochemistry were all found to be significant determinants of ambient concentration of a range of aldehyde species, including formaldehyde, acetaldehyde, acrolein, and others. For most aldehydes, spring and summer concentrations were significantly higher than those from colder seasons.[42] However, formaldehyde concentrations were significantly lower in summertime, suggesting greater photochemical destruction than production. On colder days, when photochemical activity was lower, concentrations of formaldehyde, acetaldehyde, acrolein, and other aldehydes were significantly higher with increasing proximity to high-traffic roads.

Several other 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.[43,44,45,46,47,48] For example, measurements near a tollbooth in Baltimore observed mean benzene concentrations to vary by time of day from 3 to 22.3 $\mu g/m^3$ depending on traffic volume, vehicle type, and meteorology.[49] 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 $\mu g/m^3$.[50] Another study measured the average benzene concentration in a relatively high traffic density (~ 16000 automobiles/day) sampling area at 9.6 $\mu g/m^3$ and in rural areas with hardly any traffic (< 50 automobiles/day) at 1.3 $\mu g/m^3$.[51] 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.

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.[52,53,54,55,56] Researchers have demonstrated exponential reductions in concentrations of CO, as well as PM number, and black carbon (as measured by an aethalometer), with increasing downwind distance from a freeway in Los Angeles.[57,58] 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).[59] Troopers operated their vehicles in typical patterns, including highway and city driving and refueling. In-vehicle benzene concentrations averaged 12.8 $\mu g/m^3$, while concentrations measured at an "ambient" site located outside a nearby state environmental office averaged 0.32 $\mu g/m^3$. 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.[60] 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 $\mu g/m^3$), in parking garages above-ground (155 $\mu g/m^3$) and below-ground (61.7 $\mu g/m^3$), in urban street canyons (7.54 $\mu g/m^3$), and during refueling (46.0 $\mu g/m^3$). It should be noted that sample sizes in this screening study were small, usually with only one to two samples per microenvironment. The final report of this study is expected to be released in 2007.

In 1998, the California Air Resources Board published an extensive study of concentrations of in-vehicle air toxics in Los Angeles and Sacramento, CA.[61] The data set is large and included a variety of sampling conditions. On urban freeways, in-vehicle benzene concentrations ranged from 3 to 15 $\mu g/m^3$ in Sacramento and 10 to 22 $\mu g/m^3$ in Los Angeles. In comparison, ambient benzene concentrations ranged from 1 to 3 $\mu g/m^3$ in Sacramento and 3 to 7 $\mu g/m^3$ in Los Angeles.

Studies have also been conducted in diesel buses, such as the one recently conducted of LA school buses.[62,63] 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 $\mu g/m^3$, compared with 1.6 $\mu g/m^3$ 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.[64] Real-time concentrations of black carbon (BC), particle-bound PAH, nitrogen dioxide (NO_2), particle counts (0.3-0.5 μm size range), and $PM_{2.5}$ 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, NO_2, 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 ($\mu g/m^3$)	**2 ± 0.1**	**2 ± 0.3**	**4 ± 0.4**	**3-19 (8)**
Particle Bound -PAH ($\mu g/m^3$)	**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)**
$PM_{2.5}$ ($\mu g/m^3$)	**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.[65] The average in-bus concentration of benzene was 4.5 $\mu g/m^3$, while the average concentrations at three fixed sites taken during the study period ranged from 0.9-2.0 $\mu g/m^3$. 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.[66,67,68,69,70,71]

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.[72,73,74] 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 (μg/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 concentrations of outdoor pollutants can therefore influence indoor concentrations. A review of the literature determined that approximately 100% of gaseous compounds, such as benzene, and 80% of diesel PM can penetrate indoors.[75,76]

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.[77] 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.

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.[78] Concentrations of the traffic pollutants PM_{10}, $PM_{2.5}$, black carbon, total NO_x, and NO_2 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, NOx, NO, and (to a lesser extent) NO_2 associated with roads with heavy traffic within a relatively small geographic area.

A study to assess children's exposure to traffic-related air pollution while attending schools near roadways was performed in the Netherlands.[79] Investigators measured $PM_{2.5}$, NO_2 and benzene inside and outside of 24 schools located within 400 m of roadways. The indoor average benzene concentration was 3.2 $\mu g/m^3$, with a range of 0.6-8.1 $\mu g/m^3$. The outdoor average benzene concentration was 2.2 $\mu g/m^3$, with a range of 0.3-5.0 $\mu g/m^3$. 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, PM_{10}, and NO_2 in twelve schools between the periods of May and August 1995.[80] The schools were located at varying distances from the motorway (35-645 m). Results indicate that black smoke and NO_2 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. PM_{10} 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.

In another Dutch study, researchers monitored children's personal exposure concentrations, and home indoor and home outdoor levels of "soot" (particle blackness), NO, and NO_2.[81] Four-month average concentrations were calculated for each pollutant. Personal exposure to "soot" was 35-38% higher in students living within 75 meters of roads with 10,000 average annual daily traffic, a statistically significant result. Nonsignificant elevations in personal exposure to NO, NO_2, and NOx were also found.

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.[82] 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, spatial and temporal patterns of MTBE concentrations, used as a tracer for motor vehicle pollution, were consistent with traffic patterns.

Average benzene concentrations were determined in a recent evaluation of the exposure of urban inhabitants to atmospheric benzene in Athens, Greece.[83] Home and personal levels of 50 non-smokers in six monitoring campaigns varied between 6.0-13.4 and 13.1-24.6 $\mu g/m^3$, respectively. Urban levels varied between 15.4 and 27.9 $\mu g/m^3$ with an annual mean of 20.4 $\mu g/m^3$. 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 PM_{10} 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.[84] Similarly, researchers found that traffic-related pollutant exposure concentrations of car drivers were higher than for cyclists.[85] 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 NO_2 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.[86] The mean concentration of benzene in the six pedestrian sidewalk samples was 6.8 $\mu g/m^3$. This concentration was lower than the in-vehicle measurement (11.6 $\mu g/m^3$), but higher than the fixed-site measurement (1.9 $\mu g/m^3$) 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.[87] For pedestrians, mean time-weighted concentrations of benzene, toluene, and xylenes of 10.6, 19.8, and 16.7 $\mu g/m^3$, respectively, were reported. For cyclists, the time-weighted concentrations were similar to those of pedestrians, at 9.2, 16.3, and 13.0 $\mu g/m^3$, 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.[88] 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 PM_{10}, $PM_{2.5}$, and PM_1 were made during walking and in-car journeys on two suburban routes.[89] In-car measurements were highest (43.16, 15.54, and 7.03 $\mu g/m^3$ for PM_{10}, $PM_{2.5}$, and PM1, respectively) followed by walking (38.18, 15.06, and 7.14 $\mu g/m^3$, respectively). Background levels were only available for PM_{10} (26.55 $\mu g/m^3$), but were significantly lower than the walking exposure levels. Researchers found similar results for CO exposure levels of schoolchildren commuters.[90] 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.

3.1.3.3 Concentrations and Exposure 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. A recent study from Fairbanks, Alaska used perfluorocarbon tracer (PFT) gases to estimate that 12.2% of air entering a mechanically ventilated energy efficient home and 47.4% of the air entering the living spaces of an older passively ventilated home originated in the homes' attached garages.[92] In an Ann Arbor, Michigan home, researchers used PFT gases to estimate that 16% of the air entering the home entered through the garage.[93] A recent study of a representative sample of homes in Anchorage, Alaska employing PFT estimated that in homes with a forced air furnace in an attached garage, 36.7% of indoor air originated in the garage.[94] In homes that had forced air furnaces indoors or hytronic heat, 17.0% and 18.4% of indoor air originated in the garage, respectively. A study from Minnesota examined homes constructed in 1994, 1998, and 2000.[95] 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.[96] 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.[97] 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.[98]

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) Study was completed in 1985.[99] 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. In the study, a large fraction of an average nonsmoker's benzene exposure originated from sources in attached garages.[100] Work done as part of the TEAM study also identified stored gasoline as an important source of elevated benzene levels indoors.[101] This stored gasoline can be found primarily in gas cans as well as the fuel tanks of lawn and garden equipment, such as lawn mowers and string trimmers. Lawn and garden equipment fuel tank emissions, however, are significantly lower than evaporative emissions from gas cans, because the fuel tanks are much smaller than gas cans, typically 0.3 to 0.4 gallons. Emissions are also higher from gas cans because vents and spouts are left open.

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 outside the occupational setting. The garage also supplies contaminated air to the home to which it is attached. One recent study from Michigan found average garage benzene concentrations of 36.6 $\mu g/m^3$, with a standard deviation of 38.5 $\mu g/m^3$, compared to mean and standard deviation concentrations of 0.4 $\mu g/m^3$ and 0.12 $\mu g/m^3$ in ambient air.[102] In Alaska, where fuel benzene levels tend to be very high and homes may be built very airtight, garage concentrations have been measured at even higher levels. One study from Anchorage measured average garage benzene concentrations of 103 $\mu g/m^3$, with a standard deviation of 135 $\mu g/m^3$.[103] More recently, a two-home study in Fairbanks found garage benzene average concentrations of 119 $\mu g/m^3$ during summer and 189 $\mu g/m^3$ during winter in one well-ventilated home with an air-to-air heat exchanger.[104] In an older home with passive ventilation summer and winter garage benzene concentrations were 421 and 103 $\mu g/m^3$, respectively.

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.[105] 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 $\mu g/m^3$, while during the hot soak, concentrations averaged 13.4 μg/m3. In the garage, concentrations averaged 121 $\mu g/m^3$ during the cold start. Prior to a cold start, indoor benzene concentrations averaged 6.98 $\mu g/m^3$, while for the two hours following cold start, concentrations averaged 25.9 $\mu g/m^3$. In the garage, concentrations averaged 422 μg/m3 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.[106] Homes with attached garages had significantly higher concentrations of indoor benzene compared to homes without attached garages (70.8 μg/m3 vs. 8.6 μg/m3). 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.[107] 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.[108] 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.[109] Homes with attached garages had

median toluene levels of 24 μg/m^3, while homes without garages had median concentrations of 5 μg/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.[110]

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.[111] 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.

Appendix 3A presents an EPA analysis of the effect of attached garages on indoor air under various scenarios. 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. Because it is unclear how well the homes studied to date represent the housing nationally, it is not currently feasible to provide a highly precise estimate of the effect of attached garages on benzene exposure nationally. Depending on how the available data are summarized, overall modeled exposure concentrations would be expected to increase between 1.2 and 6.6 μg/m^3 above average inhalation exposure concentrations to benzene from ambient sources (1.4 μg/m^3, as discussed in Section 3.2). It should be noted that there is considerable uncertainty associated with this estimated range, as discussed in Appendix 3A.

Proposed reductions in fuel benzene content, new standards for cold temperature exhaust emissions during vehicle starts, and reduced emissions from gas cans are all expected to significantly reduce this major source of exposure.

3.1.3.4 Concentrations and Exposure 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 substantially higher than found in outdoor air. Because of the limited amount of data, we include results from some non-U.S. studies, although differences in fuels and control technology limited their applicability to the U.S.

In November 1990, a study of microenvironments, partially funded by the US EPA, evaluated the potential range in concentrations of selected air toxics.[112] 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 $\mu g/m^3$ in the parking garage and 12.8 $\mu g/m^3$ 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.[113] An interim report is available. 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. The final report of the 211(b) is expected in 2007.

A comparative study of indoor air quality in Hong Kong showed that the levels of CO, NOx, and nonmethane hydrocarbons (NMHC) detected in a local park garage were the highest among 13 other indoor sampling locations.[114] 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.[115] 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, CO_2, PM_{10}, 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 PM_{10} 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 $\mu g/m^3$ and 43.1 to 175 $\mu g/m^3$, respectively. The mean concentration of NMHC measured in the enclosed garage (1910 $\mu g/m^3$) was about 17 times higher than in the semi-enclosed garage (94.6 $\mu g/m^3$), 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. The authors 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.[116] 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.[117] Parameters of the parking areas were not specified, but 24-hour geometric means of benzene measured 117.4 and 74.2 $\mu g/m^3$ during the summer, 94.5 and 75.4 $\mu g/m^3$ during the monsoon, and 148.0 and 703.0 $\mu g/m^3$ 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 $\mu g/m^3$, 1.9 to 33.5 $\mu g/m^3$, and 4.7 to 18.8 $\mu g/m^3$, 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 NOx, CO, CO2, BTEX, and PM.[118] 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 NO_2 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 $\mu g/m^3$ and 10 to 350 $\mu g/m^3$, respectively.

In Madrid, Spain, atmospheric pollution produced by vehicles in parking garages was studied.[119] Two parking garages of different design were chosen for measurements of PM_{10}, 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 $\mu g/m^3$ on the weekdays versus 39 $\mu g/m^3$ on the weekend, which was similar to outdoor city average measurement (50 $\mu g/m^3$). 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 Concentrations and Exposure 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 TEAM study from the 1980's, described above, pumping gas and exposure to auto exhaust were significantly associated with elevated 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 exceed 1000 $\mu g/m^3$ (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. Since this study, implementation of fuel controls, onboard vapor recovery, 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.[120] 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 $\mu g/m^3$ with the maximum reported value of 323 $\mu g/m^3$. The median benzene concentration in ambient air was significantly lower at 12.8 $\mu g/m^3$.

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.[121] The mean (and maximum) benzene exposure measurements were 96 (927), 160 (1662), and 192 (607) $\mu g/m^3$, 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.[122] 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.[123] Air samples were collected at service stations in New Jersey, and the mean benzene in-cabin concentration was 54.3 $\mu g/m^3$ (n=46). The background concentration at the pump island measured 9.6 $\mu g/m^3$ (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.

In a study conducted between summer 1998 and spring 1999, self-service gas station customers took part in a study to measure personal and breath concentrations of benzene at gas stations in New Jersey.[124] Benzene exposure concentrations during refueling (with a median duration of three minutes) averaged 2.9 mg/m^3 (SD = 5.8 mg/m^3). Breath concentrations averaged 160 $\mu g/m^3$ (SD = 260 $\mu g/m^3$). Breath benzene concentrations were significantly correlated with refueling exposure concentrations, which was itself significantly associated with refueling duration, time of year, and fuel octane grade.

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.[60] 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 $\mu g/m^3$.

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.[125] The studies reviewed indicate that benzene exposure can range from <1 to more than 10 mg/m^3, 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 fuel benzene reductions to reduce their exposures as well. This statement is echoed by researchers in the occupational literature.[126] Occupational exposures in this range have been associated with increased risk of certain leukemias in occupational epidemiology studies (Section 1.3.1).

Handheld and non-handheld equipment operators may also be exposed to elevated concentrations of air toxics. As discussed below, 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.[127] 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. The study also reported operator breathing-zone concentrations of formaldehyde and acetaldehyde that were higher than background levels in all tests. 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.[128] A study of snowmobile rider exposures in Sweden found benzene concentrations ranging from under 10 $\mu g/m^3$ to 2.5 mg/m^3, 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/m^3. 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, PM_{10}, and CO.[129] Work shift benzene concentrations at a snowmobile entry gate 176.7 $\mu g/m^3$, while snowmobile-bound mobile patrol officers' exposure concentrations averaged 137.20 $\mu g/m^3$. The highest observed work shift concentration in the study was 514.1 $\mu g/m^3$. At major sites of tourist interest where snowmobiles parked, such as the Old Faithful geyser, concentrations averaged 41.3 to 48.8 $\mu g/m^3$. 15-minute "peak" samples of workers' personal air ranged from 46.8 $\mu g/m^3$ to 842.8 $\mu g/m^3$. 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.[130] 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 $\mu g/m^3$ to over 660 $\mu g/m^3$. Additionally, measured concentrations of acetaldehyde, benzene, and formaldehyde were found to be significantly elevated, although concentrations were not presented.

In one recently-published study of diesel exhaust exposures in a representative sample of trucking terminals nationally, investigators applied structural equation modeling to data on personal exposure to diesel exhaust (as elemental carbon).[131] The study found that worker exposure to elemental carbon depended on work area concentrations and worker tobacco use. Work area concentrations depended on the size and type of the trucking terminal, whether the work site was a mechanical shop, work site ventilation, and terminal yard concentrations. Terminal yard concentrations in turn were related to local meteorology, the proximity of interstate highways, surrounding industrial land uses, and region of the country. This study is valuable in showing how personal occupational exposures are a complicated function of many factors. Sophisticated statistical methods are needed to properly estimate models with highly complex covariance structures.

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.

3.1.4 Uncertainties in Air Toxics Measurements

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 and exposure 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 or in personal breathing zones. Air quality monitoring at these central sites often do not represent actual exposures, especially for populations living near roads or with substantial occupational exposure.

Monitoring 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.[132] Thus, this compound can easily break down before samples are analyzed. Also, a vapor pressure of 3.3 atm at 25^{o}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.

Personal exposure monitoring provides greater realism in describing a person's actual exposure to air toxics. However, given the limitations on size of equipment, detection limits in personal exposure monitoring studies are sometimes greater than those found in studies using other techniques.

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 (NATA). It assesses lifetime risks assuming continuous exposure to levels of air toxics estimated for a particular point in time. The most recent NATA was done for the year 1999.[133] It had four steps:

1) Compiled 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) Estimated ambient concentrations based on emissions as input to an air dispersion model (the Assessment System for Population Exposure Nationwide, or ASPEN model).[134]
3) Estimated 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.[135]
4) Characterized 1999 potential public health risks due to inhalation of air toxics. This included cancer and noncancer effects, using available information on air toxics health effects, current EPA risk assessment and risk characterization guidelines, and estimated population exposures.[136]

For this final rule, we have conducted air quality, exposure and risk modeling for the years 1999, 2015, 2020, and 2030, using the same general approach as the 1999 NATA. We modeled all the pollutants in Table 2.2-1 for both the reference case, which includes all control programs currently planned by EPA in regulations, and the control case, which includes the cumulative impacts of the standards proposed in this rule. These pollutants

- Are on EPA's list of hazardous air pollutants in Section 112 of the Clean Air Act
- Are emitted by mobile sources
- Are included in the National Emissions Inventory
- Are included in the 1999 NATA

Note that the modeling did not include diesel PM and diesel exhaust organic gases. EPA has previously done future-year projections of the mobile source contribution to air toxics concentrations, exposure, and risk for selected air toxics,[137, 138, 139, 140] but prior to the proposal for this rule, had never done a comprehensive assessment that includes projections for all mobile source air toxics, as well as the stationary source contribution for those pollutants. It should be noted that the reference case assessment results developed for the proposal have been published in a peer reviewed journal article.[141]

As discussed in Chapter 2, a number of major revisions to inventory methodology have been made relative to what was done for both the 1999 NATA, and air quality exposure and risk modeling for the proposal. These include revisions to cold start emissions, use of NMIM2005 for nonroad equipment, addition of portable fuel container emissions, and changes to gasoline distribution inventories. Also, this final rule modeling for 1999 does not include data submitted by States for the 1999 NEI. In addition, the modeling for the final rule relied on an updated version of the HAPEM model, HAPEM6.[142] HAPEM6 improves on HAPEM5 by accounting for the spatial variability of outdoor concentrations of air toxics within a census tract due to higher outdoor concentrations at locations near major roadways. Other improvements to HAPEM are discussed in section 3.2.1.2.1. This modeling work is discussed in more detail in an EPA technical report, "National Scale Modeling of Air Toxics for the Final Mobile Source Air Toxics Rule; Technical Support Document," Report Number EPA-454/R-07-002. It should be noted that the control case modeling accounted only for the 0.62 percent standard, but not the 1.3 vol% maximum average. Thus, the emission reductions from highway vehicles and other sources attributable to the fuel benzene standard are underestimated in many areas of the country, particularly in areas where fuel benzene levels were highest without control, such as the Northwest.

The NATA 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 ambient "hot spots," as mobile sources are not represented explicitly as roads or other locations of mobile source activity. 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 potentially significant 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 and some reactive compounds. The cancer unit risk estimates for most 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.

Although we do not use it in this modeling, another tool that EPA uses to assess distributions of concentrations of air toxics at the national scale is the Community Multiscale Air Quality (CMAQ) modeling system.[143] CMAQ can account for photochemical destruction and production, deposition and regional transport of toxic air pollutants, and thus can be used to predict the concentrations of HAPs with significant atmospheric production. 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.[144] CMAQ underpredicts monitored benzene levels more than ASPEN, because ASPEN values contain a large, added-on concentration based on monitored values of benzene. 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 concentration gradients, such as benzene.[145] Finally, CMAQ is 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 other potentially significant sources of inhalation exposure, such as benzene emissions from sources in attached garages (such as vehicles, snowblowers, lawnmowers and gas cans).

3.2.1.1 Air Quality Modeling

3.2.1.1.1 Methods

Prior to performing air quality modeling of 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.[146] 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 are processed, they are 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 kilometers away. The model then interpolates concentrations to census tract centroids. For 1999, meteorological conditions in 1999 and 2000 census tract data were used.

In using ASPEN to estimate projected concentrations in 2015, 2020, and 2030 for this final rule, 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 Final 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.[147] 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, for the proposal, a sensitivity analysis was done to evaluate the potential impact of not changing the background concentration (see Section 3.2.1.4).

It should be noted that in the control case scenarios, we have modeled the cumulative impacts on air quality, exposure, and risk for all of the programs finalized today, not the impacts of individual programs. Were we to model each program individually, we anticipate that changes in air quality, exposure, and risk would track the patterns of emission changes closely.

Also, for the final rule, we estimated the contribution of secondary formation to ambient concentrations of MSATs by applying ratios of secondary to primary concentrations from 1999 NATA to the modeled primary concentrations for this rule. This is different from the approach

used in the proposal where we projected precursor emissions and then modeled secondary formation. When we applied the ratio approach to the proposal's primary concentrations, the results were very similar to the full modeling approach (see Section 3.2.1.3). The comparisons are discussed in the technical document cited above.

We estimated the contributions to ambient concentrations for the following source sectors: major, area and other, onroad, nonroad, and background.[b]

3.2.1.1.2 Air Quality Trends for Air Toxics: Reference Case

Table 3.2-1 summarizes nationwide mean census tract ambient concentrations, without the controls being finalized in this rule, 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 over 40% by 2015, with a decrease in ambient benzene concentration from all sources of about 25%. Subsequently, increases in vehicle miles traveled (VMT) are projected to produce increasing concentrations. Summary tables providing data by State, and for reformulated and non-reformulated (i.e., conventional) 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 non-reformulated gasoline areas – about 1.9 $\mu g/m^3$ versus 1.2 $\mu g/m^3$. However the percent reduction in average 2015 ambient concentration is similar regardless of fuel type – 22% for non-reformulated gasoline counties versus 29% for reformulated gasoline counties.

[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.

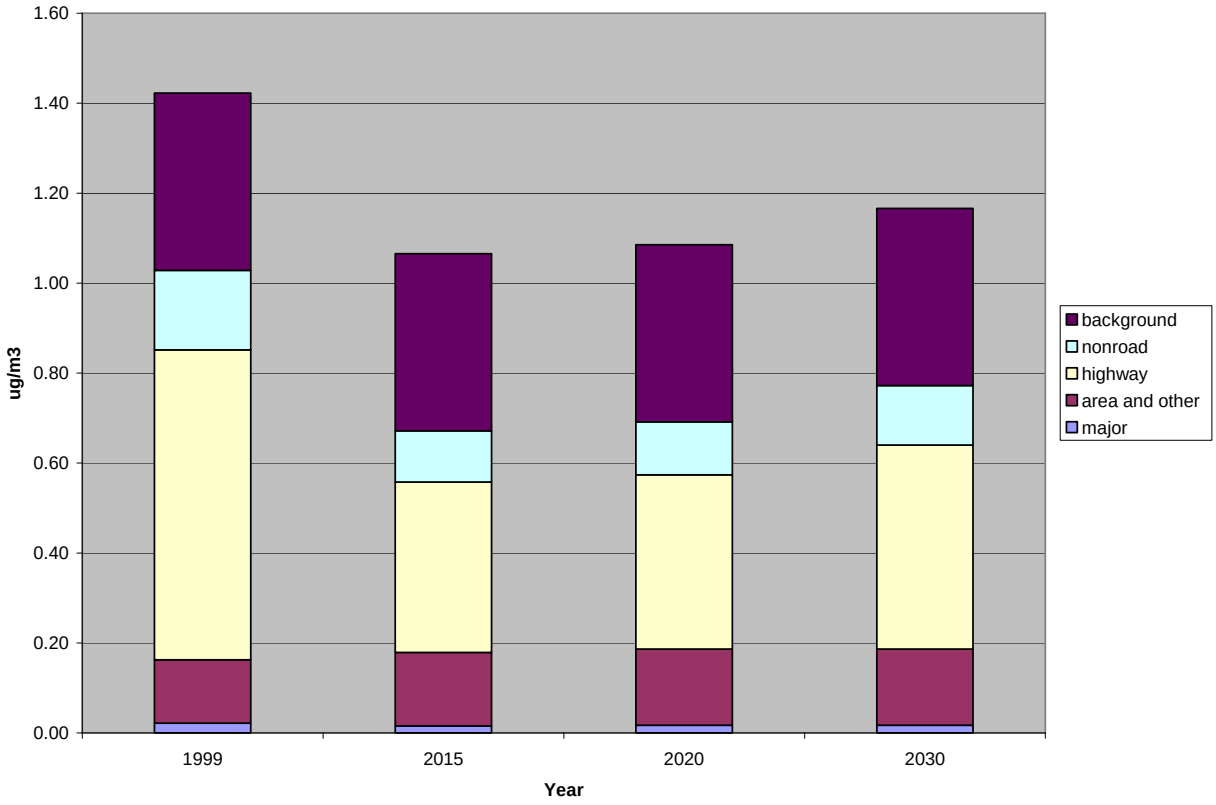
Table 3.2-1. Mean Ambient Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030, Without Controls in this Rule.

		1999 average concentrations (μg m^{-3})					2015 annual average concentrations (μg m^{-3})				
Pollutant	**background (μg 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	5.20E-02	1.81E-02	1.44E-01	2.17E-03	2.05E-02	2.28E-02	1.08E-02	1.07E-01
2,2,4-Trimethylpentane	0.00E+00	2.16E-02	2.32E-02	7.29E-01	1.96E-01	9.70E-01	1.09E-02	2.69E-02	3.66E-01	1.15E-01	5.19E-01
Acetaldehyde	5.17E-01	2.94E-02	5.49E-02	6.78E-01	1.47E-01	1.43E+00	2.97E-02	5.71E-02	3.86E-01	1.10E-01	1.10E+00
Acrolein	0.00E+00	3.21E-03	2.93E-02	5.63E-02	2.27E-02	1.11E-01	3.53E-03	2.62E-02	2.42E-02	1.81E-02	7.20E-02
Benzene	3.94E-01	2.20E-02	1.40E-01	6.89E-01	1.77E-01	1.42E+00	1.55E-02	1.63E-01	3.79E-01	1.14E-01	1.07E+00
Chromium III	0.00E+00	8.22E-04	4.53E-04	3.22E-05	5.53E-05	1.36E-03	1.04E-03	6.16E-04	4.40E-05	5.85E-05	1.76E-03
Chromium VI	0.00E+00	1.07E-04	1.98E-04	2.15E-05	1.25E-05	3.39E-04	1.36E-04	2.72E-04	2.94E-05	1.32E-05	4.50E-04
Ethyl Benzene	0.00E+00	1.84E-02	9.00E-02	2.73E-01	9.73E-02	4.79E-01	1.24E-02	1.19E-01	1.35E-01	5.66E-02	3.24E-01
Formaldehyde	7.62E-01	3.99E-02	8.77E-02	4.65E-01	2.21E-01	1.58E+00	4.98E-02	9.82E-02	1.92E-01	1.63E-01	1.27E+00
Hexane	0.00E+00	6.68E-02	4.30E-01	2.34E-01	8.56E-02	8.17E-01	5.94E-02	5.21E-01	1.16E-01	5.93E-02	7.56E-01
MTBE	0.00E+00	1.30E-02	6.04E-02	4.00E-01	4.04E-01	8.77E-01	1.38E-02	6.52E-02	1.05E-01	1.08E-01	2.93E-01
Manganese	0.00E+00	2.71E-03	2.22E-03	1.73E-05	5.46E-06	4.95E-03	3.23E-03	2.92E-03	2.36E-05	6.46E-06	6.17E-03
Naphthalene	0.00E+00	4.56E-03	4.11E-02	1.46E-02	4.36E-03	6.46E-02	3.97E-03	5.01E-02	7.90E-03	4.49E-03	6.65E-02
Nickel	0.00E+00	7.76E-04	1.42E-03	3.96E-05	9.98E-05	2.33E-03	8.87E-04	1.62E-03	5.43E-05	1.15E-04	2.67E-03
POM	0.00E+00	4.93E-03	1.61E-02	1.73E-03	8.60E-04	2.37E-02	3.79E-03	1.86E-02	9.13E-04	7.66E-04	2.40E-02
Propionaldehyde	0.00E+00	1.01E-02	2.33E-02	1.68E-01	4.27E-02	2.45E-01	9.31E-03	2.39E-02	8.24E-02	2.83E-02	1.44E-01
Styrene	0.00E+00	2.52E-02	1.40E-02	2.98E-02	3.65E-03	7.27E-02	3.00E-02	1.89E-02	1.50E-02	2.18E-03	6.61E-02
Toluene	0.00E+00	2.03E-01	8.05E-01	1.81E+00	4.18E-01	3.24E+00	1.43E-01	1.06E+00	9.00E-01	2.50E-01	2.35E+00
Xylenes	1.70E-01	9.98E-02	5.59E-01	1.01E+00	3.99E-01	2.23E+00	8.22E-02	7.60E-01	4.98E-01	2.18E-01	1.73E+00

Table 3.2-1 (cont'd). Mean Ambient Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030, Without Controls in this Rule.

		2020 annual average concentrations (μg m^{-3})					2030 annual average concentrations (μg m^{-3})				
Pollutant	background (μg 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	2.37E-02	1.14E-02	1.09E-01	2.34E-03	2.05E-02	2.78E-02	1.30E-02	1.15E-01
2,2,4-Trimethylpentane	0.00E+00	1.17E-02	2.84E-02	3.66E-01	1.14E-01	5.20E-01	1.17E-02	2.84E-02	4.24E-01	1.24E-01	5.88E-01
Acetaldehyde	5.17E-01	3.10E-02	5.83E-02	3.98E-01	1.09E-01	1.11E+00	3.10E-02	5.83E-02	4.69E-01	1.18E-01	1.19E+00
Acrolein	0.00E+00	3.96E-03	2.54E-02	2.50E-02	1.91E-02	7.34E-02	3.96E-03	2.54E-02	2.94E-02	2.18E-02	8.05E-02
Benzene	3.94E-01	1.70E-02	1.69E-01	3.88E-01	1.18E-01	1.09E+00	1.70E-02	1.69E-01	4.54E-01	1.32E-01	1.17E+00
Chromium III	0.00E+00	1.17E-03	6.96E-04	4.84E-05	5.90E-05	1.97E-03	1.17E-03	6.96E-04	5.94E-05	6.04E-05	1.98E-03
Chromium VI	0.00E+00	1.54E-04	3.07E-04	3.23E-05	1.34E-05	5.07E-04	1.54E-04	3.07E-04	3.96E-05	1.37E-05	5.15E-04
Ethyl Benzene	0.00E+00	1.39E-02	1.31E-01	1.35E-01	5.78E-02	3.38E-01	1.39E-02	1.31E-01	1.57E-01	6.45E-02	3.66E-01
Formaldehyde	7.62E-01	5.65E-02	1.03E-01	1.97E-01	1.64E-01	1.28E+00	5.65E-02	1.03E-01	2.31E-01	1.80E-01	1.33E+00
Hexane	0.00E+00	6.53E-02	5.62E-01	1.07E-01	6.13E-02	7.96E-01	6.53E-02	5.62E-01	1.18E-01	6.87E-02	8.14E-01
MTBE	0.00E+00	1.55E-02	6.67E-02	8.48E-02	1.12E-01	2.79E-01	1.55E-02	6.67E-02	8.42E-02	1.25E-01	2.92E-01
Manganese	0.00E+00	3.59E-03	3.21E-03	2.60E-05	6.83E-06	6.83E-03	3.59E-03	3.21E-03	3.19E-05	7.59E-06	6.84E-03
Naphthalene	0.00E+00	4.46E-03	5.32E-02	7.86E-03	4.80E-03	7.03E-02	4.46E-03	5.32E-02	9.11E-03	5.51E-03	7.23E-02
Nickel	0.00E+00	9.61E-04	1.78E-03	5.97E-05	1.20E-04	2.92E-03	9.61E-04	1.78E-03	7.34E-05	1.31E-04	2.95E-03
POM	0.00E+00	4.21E-03	1.90E-02	9.47E-04	7.71E-04	2.49E-02	4.21E-03	1.90E-02	1.12E-03	8.57E-04	2.52E-02
Propionaldehyde	0.00E+00	9.35E-03	2.45E-02	8.45E-02	2.78E-02	1.46E-01	9.35E-03	2.45E-02	9.84E-02	2.99E-02	1.62E-01
Styrene	0.00E+00	3.44E-02	2.09E-02	1.57E-02	2.21E-03	7.32E-02	3.44E-02	2.09E-02	1.85E-02	2.47E-03	7.63E-02
Toluene	0.00E+00	1.60E-01	1.16E+00	9.11E-01	2.50E-01	2.48E+00	1.60E-01	1.16E+00	1.06E+00	2.75E-01	2.65E+00
Xylenes	1.70E-01	9.29E-02	8.38E-01	5.04E-01	2.18E-01	1.82E+00	9.29E-02	8.38E-01	5.86E-01	2.40E-01	1.93E+00

Figure 3.2-1. Nationwide Average Benzene Concentration, 1999-2030, Without Controls in this Rule.



3.2.1.1.3 Distributions of Air Toxic Concentrations across the U. S.: Reference Case

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, absent the controls being finalized in this rule. 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 percentiles 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

Table 3.2-2. National Distribution of Census Tract Concentrations for Mobile Source Air Toxics in 2020, Without Controls in this Rule.

	2020 concentration (μg m^{-3}) distribution						
Pollutant	5th percentile	10th percentile	25th percentile	Median	75th percentile	90th percentile	95th percentile
1,3-Butadiene	3.03E-03	5.60E-03	3.12E-02	8.36E-02	1.30E-01	1.98E-01	3.28E-01
2,2,4-Trimethylpentane	3.83E-02	7.00E-02	1.74E-01	3.79E-01	6.80E-01	1.12E+00	1.50E+00
Acetaldehyde	5.45E-01	5.82E-01	6.99E-01	9.41E-01	1.29E+00	1.84E+00	2.49E+00
Acrolein	6.04E-03	9.78E-03	2.09E-02	4.41E-02	8.64E-02	1.71E-01	2.71E-01
Benzene	3.42E-01	4.15E-01	6.33E-01	9.37E-01	1.32E+00	1.90E+00	2.36E+00
Chromium III	5.73E-06	1.52E-05	6.40E-05	2.41E-04	7.31E-04	2.34E-03	4.89E-03
Chromium VI	3.52E-06	8.79E-06	3.56E-05	1.22E-04	3.32E-04	9.08E-04	1.55E-03
Ethyl Benzene	2.04E-02	3.79E-02	1.01E-01	2.30E-01	4.06E-01	6.70E-01	9.60E-01
Formaldehyde	4.08E-01	5.29E-01	8.08E-01	1.16E+00	1.52E+00	2.12E+00	2.67E+00
Hexane	3.27E-02	6.16E-02	1.90E-01	4.76E-01	8.93E-01	1.70E+00	2.81E+00
MTBE	3.34E-03	7.88E-03	2.39E-02	7.22E-02	2.44E-01	8.80E-01	1.30E+00
Manganese	1.33E-05	4.35E-05	2.04E-04	8.68E-04	3.53E-03	1.42E-02	2.10E-02
Naphthalene	2.88E-03	5.91E-03	1.86E-02	4.48E-02	8.82E-02	1.67E-01	2.37E-01
Nickel	1.38E-05	3.80E-05	1.67E-04	6.65E-04	2.01E-03	4.78E-03	8.17E-03
POM	1.72E-03	2.94E-03	5.73E-03	1.19E-02	2.08E-02	3.62E-02	5.78E-02
Propionaldehyde	1.24E-02	2.13E-02	4.81E-02	1.07E-01	1.93E-01	3.26E-01	4.33E-01
Styrene	2.52E-03	4.88E-03	1.23E-02	2.70E-02	5.39E-02	1.06E-01	1.75E-01
Toluene	1.54E-01	2.83E-01	7.34E-01	1.64E+00	2.96E+00	5.31E+00	7.43E+00
Xylenes	2.66E-01	3.43E-01	6.35E-01	1.22E+00	2.06E+00	3.61E+00	5.38E+00

indicate higher than average fuel benzene levels in these areas. These areas also have higher benzene emissions in winter due to cold starts. 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.

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 Controls on Ambient Concentrations

The standards being finalized in this rule will substantially reduce ambient concentrations of air toxics across the United States. As noted above, these results reflect the cumulative effects of all of the programs finalized in today's rule, not the individual programs. Table 3.2-3 shows the reduction in nationwide average census tract concentrations of MSATs from all sources in 2015, 2020 and 2030. Table 3.2-4 shows the reduction in the highway vehicle contribution to nationwide average census tract concentrations of MSATs. Table 3.2-5 shows that in 2030, the highway vehicle portion of ambient benzene concentrations will be reduced almost 45% across the U.S., the nonroad equipment contribution will be reduced about 10%, and

Figure 3.2-2. Geographic Distribution of County Median Concentrations (µg/m^3) of Benzene in 2020 Without Controls in this Rule.

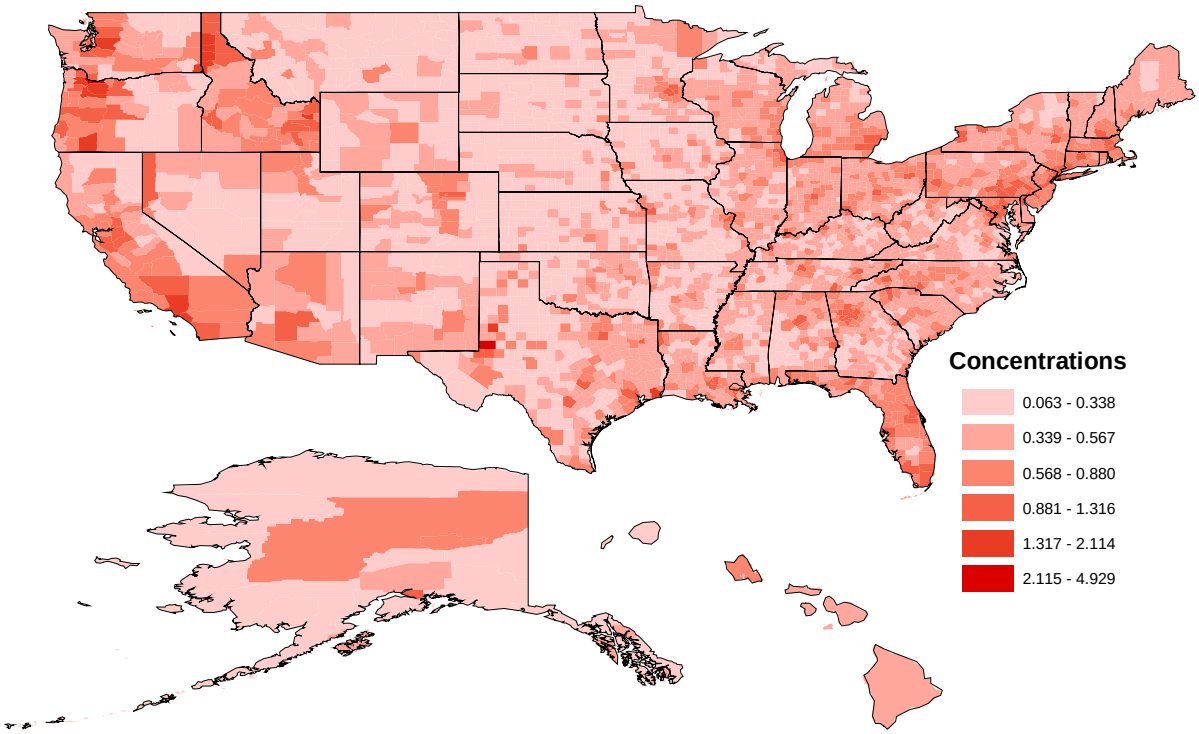


Table 3.2-3. Nationwide Average Census Tract Concentrations of MSATs, With and Without Controls in this Rule, 2015, 2020, and 2030.

	2015			2020			2030		
	Reference	Control	% Reduction	Reference	Control	% Reduction	Reference	Control	% Reduction
1,3-Butadiene	1.07E-01	1.03E-01	3.6	1.09E-01	1.03E-01	5.7	1.15E-01	1.04E-01	9.0
2,2,4-Trimethylpentane	5.19E-01	4.53E-01	12.7	5.20E-01	4.19E-01	19.5	5.88E-01	4.26E-01	27.6
Acetaldehyde	1.10E+00	1.04E+00	5.8	1.11E+00	1.01E+00	9.1	1.19E+00	1.03E+00	13.7
Acrolein	7.20E-02	6.79E-02	5.7	7.34E-02	6.69E-02	8.9	8.05E-02	6.97E-02	13.4
Benzene	1.07E+00	9.56E-01	10.3	1.09E+00	9.38E-01	13.6	1.17E+00	9.50E-01	18.5
Chromium III	1.76E-03	1.76E-03	0.0	1.97E-03	1.97E-03	0.0	1.98E-03	1.98E-03	0.0
Chromium VI	4.50E-04	4.50E-04	0.0	5.07E-04	5.07E-04	0.0	5.15E-04	5.15E-04	0.0
Ethyl Benzene	3.24E-01	2.99E-01	7.5	3.38E-01	3.01E-01	11.1	3.66E-01	3.07E-01	16.3
Formaldehyde	1.27E+00	1.24E+00	2.3	1.28E+00	1.24E+00	3.6	1.33E+00	1.26E+00	5.6
Hexane	7.56E-01	7.37E-01	2.5	7.96E-01	7.70E-01	3.2	8.14E-01	7.76E-01	4.7
MTBE	2.93E-01	2.82E-01	3.5	2.79E-01	2.66E-01	4.6	2.92E-01	2.74E-01	6.0
Manganese	6.17E-03	6.17E-03	0.0	6.83E-03	6.83E-03	0.0	6.84E-03	6.84E-03	0.0
Naphthalene	6.65E-02	6.65E-02	0.0	7.03E-02	7.03E-02	0.0	7.23E-02	7.23E-02	0.0
Nickel	2.67E-03	2.67E-03	0.0	2.92E-03	2.92E-03	0.0	2.95E-03	2.95E-03	0.0
POM	2.40E-02	2.40E-02	0.0	2.49E-02	2.49E-02	0.0	2.52E-02	2.52E-02	0.0
Propionaldehyde	1.44E-01	1.33E-01	7.8	1.46E-01	1.28E-01	12.2	1.62E-01	1.33E-01	18.0
Styrene	6.61E-02	6.33E-02	4.3	7.32E-02	6.87E-02	6.2	7.63E-02	6.89E-02	9.7
Toluene	2.35E+00	2.18E+00	7.1	2.48E+00	2.22E+00	10.4	2.65E+00	2.24E+00	15.7
Xylenes	1.73E+00	1.64E+00	5.3	1.82E+00	1.68E+00	7.8	1.93E+00	1.70E+00	11.8

Table 3.2-4. Nationwide Highway Vehicle Contribution to Average Census Tract Concentrations of MSATs, With and Without Controls in this Rule, 2015, 2020, and 2030.

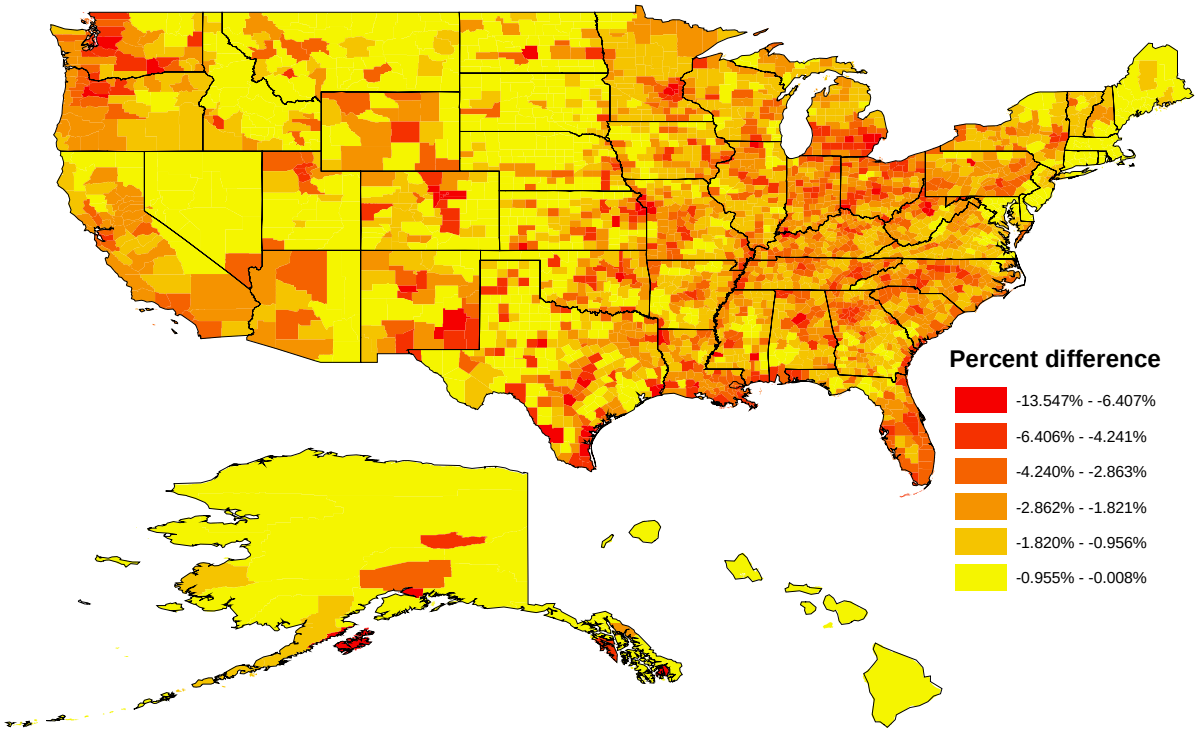
	2015			2020			2030		
	Reference	Control	% Reduction	Reference	Control	% Reduction	Reference	Control	% Reduction
1,3-Butadiene	2.28E-02	1.89E-02	17.0	2.37E-02	1.74E-02	26.3	2.78E-02	1.75E-02	37.0
2,2,4-Trimethylpentane	3.66E-01	3.06E-01	16.3	3.66E-01	2.71E-01	25.9	4.24E-01	2.70E-01	36.4
Acetaldehyde	3.86E-01	3.22E-01	16.5	3.98E-01	2.97E-01	25.4	4.69E-01	3.06E-01	34.8
Acrolein	2.42E-02	2.01E-02	17.0	2.50E-02	1.85E-02	26.2	2.94E-02	1.87E-02	36.6
Benzene	3.79E-01	2.83E-01	25.3	3.88E-01	2.55E-01	34.2	4.54E-01	2.54E-01	44.0
Chromium III	4.40E-05	4.40E-05	0.0	4.84E-05	4.84E-05	0.0	5.94E-05	5.94E-05	0.0
Chromium VI	2.94E-05	2.94E-05	0.0	3.23E-05	3.23E-05	0.0	3.96E-05	3.96E-05	0.0
Ethyl Benzene	1.35E-01	1.14E-01	16.0	1.35E-01	1.01E-01	25.6	1.57E-01	1.00E-01	36.1
Formaldehyde	1.92E-01	1.62E-01	15.3	1.97E-01	1.50E-01	23.6	2.31E-01	1.56E-01	32.4
Hexane	1.16E-01	1.05E-01	9.8	1.07E-01	8.89E-02	16.9	1.18E-01	8.84E-02	25.0
MTBE	1.05E-01	1.01E-01	4.3	8.48E-02	7.77E-02	8.3	8.42E-02	7.30E-02	13.4
Manganese	2.36E-05	2.36E-05	0.0	2.60E-05	2.60E-05	0.0	3.19E-05	3.19E-05	0.0
Naphthalene	7.90E-03	7.90E-03	0.0	7.86E-03	7.86E-03	0.0	9.11E-03	9.11E-03	0.0
Nickel	5.43E-05	5.43E-05	0.0	5.97E-05	5.97E-05	0.0	7.34E-05	7.34E-05	0.0
POM	9.13E-04	9.13E-04	0.0	9.47E-04	9.47E-04	0.0	1.12E-03	1.12E-03	0.0
Propionaldehyde	8.24E-02	7.12E-02	13.6	8.45E-02	6.66E-02	21.1	9.84E-02	6.92E-02	29.6
Styrene	1.50E-02	1.22E-02	18.8	1.57E-02	1.12E-02	28.8	1.85E-02	1.11E-02	39.8
Toluene	9.00E-01	7.47E-01	17.1	9.11E-01	6.66E-01	26.9	1.06E+00	6.62E-01	37.7
Xylenes	4.98E-01	4.14E-01	16.9	5.04E-01	3.69E-01	26.7	5.86E-01	3.67E-01	37.5

Table 3.2-5. Contributions of Source Sectors to Nationwide Average Census Tract Concentrations of Benzene, With and Without Controls in this Rule, 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	1.55E-02	1.63E-01	3.79E-01	1.14E-01	1.07E+00	1.70E-02	1.69E-01	3.88E-01	1.18E-01	1.09E+00	1.70E-02	1.69E-01	4.54E-01	1.32E-01	1.17E+00
Control	1.54E-02	1.61E-01	2.83E-01	1.02E-01	9.56E-01	1.69E-02	1.67E-01	2.55E-01	1.05E-01	9.38E-01	1.69E-02	1.67E-01	2.54E-01	1.18E-01	9.50E-01
% Difference	0	-1	-25	-10	-10	0	-1	-34	-10	-14	0	-1	-44	-10	-19
Average Nationwide Difference in Ambient Benzene Concentration -- Non RFG Areas															
Reference	1.08E-02	1.43E-01	2.96E-01	8.15E-02	8.93E-01	1.20E-02	1.48E-01	3.06E-01	8.34E-02	9.11E-01	1.20E-02	1.48E-01	3.57E-01	9.29E-02	9.71E-01
Control	1.08E-02	1.41E-01	2.17E-01	6.82E-02	7.99E-01	1.20E-02	1.46E-01	2.00E-01	6.95E-02	7.89E-01	1.20E-02	1.46E-01	1.97E-01	7.72E-02	7.94E-01
% Difference	0	-2	-27	-16	-11	0	-2	-35	-17	-13	0	-2	-45	-17	-18
Average Nationwide Difference in Ambient Benzene Concentration -- RFG Areas															
Reference	2.39E-02	1.99E-01	5.29E-01	1.72E-01	1.38E+00	2.58E-02	2.08E-01	5.34E-01	1.79E-01	1.40E+00	2.58E-02	2.08E-01	6.29E-01	2.03E-01	1.52E+00
Control	2.38E-02	1.97E-01	4.02E-01	1.63E-01	1.24E+00	2.58E-02	2.05E-01	3.54E-01	1.70E-01	1.21E+00	2.58E-02	2.05E-01	3.57E-01	1.92E-01	1.23E+00
% Difference	0	-1	-24	-5	-10	0	-1	-34	-5	-14	0	-1	-43	-5	-19

the area source contribution will be reduced about 1 to 2%. The reduction for area sources is due to the impacts of fuel benzene control on gasoline distribution emissions, and reductions in portable fuel container (PFC) emissions from PFC and fuel benzene controls. Reductions in non-reformulated gasoline areas are even larger. It should be noted that the estimated total reductions in ambient concentrations from all sources 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. Figure 3.2-3 presents the distribution of percent reductions in median ambient benzene concentrations for U.S. counties with the controls being finalized in 2030. Again, since the 1.3% maximum average fuel benzene standard is not included in the modeling, reductions in some parts of the country, including the Pacific Northwest, are underestimated. Summary tables providing data by State, as well as maps of MSAT concentrations with controls and percent reductions with controls, can be found in the docket for the rule.

Figure 3.2-3. Distribution of Percent Reductions in Median Ambient Benzene Concentrations, 2030, for U. S. Counties with the Controls in this Rule.



3.2.1.2 Exposure and Risk Modeling

3.2.1.2.1 Methods

The HAPEM6 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.[148, 149, 150] HAPEM6 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. HAPEM6 uses the general approach of tracking representatives of 6 specified age groups as they move among indoor and outdoor microenvironments and among geographic locations (a total of 14, HAPEM5 had 37). 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. HAPEM calculates 30 replicates with different exposures for each demographic group. These data can be used to develop a distribution of exposures for the entire U. S. population.

HAPEM6 uses five primary sources of information: year 2000 population data from the U.S. Census, population activity data, air quality data, roadway locations, and microenvironmental data. The population data used are obtained from the U.S. 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).[151] The commuting data contained in the HAPEM6 default file were derived from the year 2000 U.S. Census, and includes the number of residents of each tract that work in that tract and every other U.S. Census tract, as well as data on commuting times and distances. The air quality data come from ASPEN (after background has been added). The road locations are determined from geographic information system files from the U.S. Census. 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.[152]

New to HAPEM6 are algorithms which account for the gradient in concentrations of primary (directly emitted) mobile source air toxics within 200 meters of major roadways. [153] HAPEM6 adjusts ambient concentrations generated by ASPEN for each census tract using concentration gradients developed with the CALPUFF dispersion model.[154] For locations within 75 meters and from 75 to 200 meters from major roads, ambient concentrations are adjusted upward, while locations further from major roadways are adjusted downward. These adjustments are consistent with results from prior modeling studies that explicitly accounted for concentration gradients around major roads within census tracts.[155] These adjusted concentrations are then employed in microenvironmental concentration calculations.

HAPEM6 has a number of other technical improvements over the previous version of HAPEM. These improvements, along with other details of the model, are described in the HAPEM6 User's Guide.[156] In short, HAPEM6 reduces the number of demographic groups to 6 age-based groups from 10 age-gender groups in HAPEM5, and reduces the number of microenvironments modeled, from 37 to 14. This reduces modeling run time significantly with little impact on results. HAPEM6 also accounts for commuting time better, basing commute times and travel modes for each census tract on distributions reported in the 2000 Census. The HAPEM runs used year 2000 census 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, nonroad, and background.

Once HAPEM runs were completed, cancer risk and noncancer risk were calculated for each of the mobile source air toxic pollutants, based on population exposure distributions. In the HAPEM6 output, for each source category, there are 30 replicate exposure concentrations for each of the six demographic groups (180 concentrations per census tract for each source category). For each source category and each of the 30 replicates, a lifetime exposure concentration was calculated. A risk estimate was then calculated for each of the 30 replicates. The resulting data were used to develop distributions of population risks at various summary levels (census tract, county, state, national). More detail is provided in the technical support document. Table 3.2-6 lists the pollutants with their respective unit risk estimates (UREs) for cancer calculations and reference concentrations (RfCs) for noncancer calculations. These are the same values used in the 1999 NATA, 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 noncancer calculations.

Table 3.2-6. Dose-Response Values Use in Risk Modeling (Concentrations in µg/m^3)

HAP	Carcinogen Class	URE (per µg/m$^{3)}$	Source	Organ Systems	RfC (mg/ m$^{3)}$	Source
1,3-Butadiene	A	$3.0x10^{-5}$	IRIS	Reproductive	$2.0x10^{-3}$	
2,2,4-Trimethylpentane	N/A	N/A		N/A	N/A	
Acetaldehyde	B2	$2.2x10^{-6}$	IRIS	Respiratory	$9.0x10^{-3}$	IRIS
Acrolein		0		Respiratory	$2.0x10^{-5}$	IRIS
Benzene	A	$7.8x10^{-6}$*	IRIS	Immune	$3.0x10^{-2}$	IRIS
Chromium III	N/A	N/A		N/A	N/A	
Chromium VI	A	$1.2x10^{-2}$	IRIS	Respiratory	$1.0x10^{-4}$	IRIS
Ethyl Benzene		0		Developmental	1.0	IRIS
Formaldehyde	B	$5.5x10^{-9}$	CIIT	Respiratory	$9.8x10^{-3}$	ATSDR
Hexane		N/A		Respiratory, Neurological	$2.0x10^{-1}$	IRIS
Manganese		N/A		Neurological	$5.0x10^{-5}$	IRIS
MTBE		N/A		Liver, Kidney, Ocular	3.0	IRIS
Naphthalene	C	$3.4x10^{-5}$	CAL	Respiratory	$3.0x10^{-3}$	IRIS
Nickel	A	$1.6x10^{-4}$	EPA/ OAQPS	Respiratory, Immune	$6.5x10^{-5}$	CAL
POM1	B2	$5.5x10^{-5}$	OAQPS		N/A	
POM2	B2	$5.5x10^{-5}$	OAQPS		N/A	
POM3	B2	$1.0x10^{-1}$	OAQPS		N/A	
POM4	B2	$1.0x10^{-2}$	OAQPS		N/A	
POM5	B2	$1.0x10^{-3}$	OAQPS		N/A	
POM6	B2	$1.0x10^{-4}$	OAQPS		N/A	
POM7	B2	$1.0x10^{-5}$	OAQPS		N/A	
POM8	B2	$2.0x10^{-4}$	OAQPS		N/A	
Styrene		N/A		Neurological	1.0	IRIS
Toluene		N/A		Respiratory, Neurological	$4.0x10^{-1}$	IRIS
Xylenes		N/A		Neurological	$1.0x10^{-1}$	IRIS

*represents upper end of a range of MLE values

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) EPA IRIS assessments.
2) Agency for Toxic Substances and Disease Registry (ATSDR) minimum risk levels (MRLs) for noncancer effects – used as RfC.
3) California Office of Environmental Health Hazard Assessment (OEHHA) values.

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.-6 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 3.2.-6 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 NATA and in Appendix H of the 2001 EPA draft report to the Science Advisory Board on the 1996 National-Scale Assessment.[157]

Individual 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). Except for benzene and chromium, where risks are based on maximum likelihood dose-response values, risks from mobile source air toxics should all be considered upper-bound values. True risks could be greater, but are likely to be lower, and could be zero.

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. However one should evaluate the weight of evidence supporting the RfC value for a particular chemical before determining potential risks. Following the approach used in the 1999 NATA, 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. An 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: Reference Case

Tables 3.2-7 and 3.2-8 summarize nationwide averages of median and 90th percentile census tract exposure concentrations of mobile source air toxics in 1999, 2015, 2020, and 2030, without the controls being finalized in this rule. It should be noted that all the other non-inventoried sources, as well as the contribution from transport, contribute to background levels. Overall, exposure to ambient concentrations tends to be less than ambient concentrations because penetration rates to indoor microenvironments are typically less than one.[c] 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.

[c] In the exposure monitoring studies discussed in section 3.1.2, average measured personal exposure concentrations are greater than those in both indoor and outdoor air. These differences may be attributable to several factors. First, HAPEM6 does not include pollution sources within indoor microenvironments, such as attached garages, environmental tobacco smoke, and solvent storage. Second, measured personal breathing zone concentrations are integrated measurements that account for time-weighted average (TWA) concentrations that incorporate every source, activity, and location with which a monitor comes into contact. Microenvironmental models like HAPEM6 simplify individual time budgets so they fit within the microenvironments modeled or monitored.

Table 3.2-9 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 25 in a million in 1999.

In all projection years, benzene emissions are by far the largest contributor to cancer risk from mobile sources (see Figure 3.2-4). Other significant contributors to cancer risk from mobile source air toxics include 1,3-butadiene, acetaldehyde, naphthalene, and hexavalent chromium. It should be noted, however, that we have no actual measurements of hexavalent chromium emissions from mobile sources, and that the risk estimate for this pollutant is based on an assumption that forty percent of the chromium from highway vehicles and eighteen percent of the chromium from nonroad sources was assumed to be the highly toxic hexavalent form. The estimate for highway vehicles is based on data from utility boilers,[158] and the estimate for nonroad equipment is, based on combustion data from stationary combustion turbines that burn diesel fuel.[159] Thus there is a great deal of uncertainty in estimates for this pollutant.

Despite significant reductions in risk from mobile source air toxics, average inhalation cancer risks for these pollutants in 2030, accounting for both mobile and stationary source contributions, remain well above 20 in 1,000,000 (Figure 3.2-5). In addition, average risk from exposure to benzene remains above 9 in 1,000,000.

Table 3.2-7. National Means of Census Tract Median Population Exposure Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030, Without Controls in this Rule.

		1999 annual average concentrations (μg m^{-3})					2015 annual average concentrations (μg m^{-3})				
Pollutant	**background (μg m^{-3})**	**major**	**area & other**	**onroad**	**nonroad**	**total (including background)**	**major**	**area & other**	**onroad**	**nonroad**	**total (including background)**
1,3-Butadiene	3.96E-02	1.54E-03	1.66E-02	6.39E-02	1.64E-02	1.38E-01	1.72E-03	1.69E-02	2.88E-02	1.01E-02	9.71E-02
2,2,4-Trimethylpentane	0.00E+00	1.70E-02	1.86E-02	8.23E-01	1.57E-01	1.02E+00	8.68E-03	2.18E-02	4.16E-01	9.26E-02	5.39E-01
Acetaldehyde	4.00E-01	2.34E-02	4.33E-02	8.08E-01	1.18E-01	1.39E+00	2.41E-02	4.60E-02	4.70E-01	9.07E-02	1.03E+00
Acrolein	0.00E+00	2.56E-03	2.35E-02	6.62E-02	1.83E-02	1.10E-01	2.91E-03	2.14E-02	2.90E-02	1.49E-02	6.83E-02
Benzene	3.05E-01	1.76E-02	1.16E-01	8.08E-01	1.51E-01	1.40E+00	1.25E-02	1.37E-01	4.53E-01	9.87E-02	1.01E+00
Chromium III	0.00E+00	3.23E-04	1.79E-04	1.93E-05	2.21E-05	5.43E-04	4.11E-04	2.43E-04	2.64E-05	2.34E-05	7.03E-04
Chromium VI	0.00E+00	4.25E-05	7.94E-05	1.30E-05	5.06E-06	1.40E-04	5.40E-05	1.09E-04	1.78E-05	5.38E-06	1.86E-04
Ethyl Benzene	0.00E+00	1.45E-02	7.49E-02	3.22E-01	8.02E-02	4.91E-01	9.92E-03	1.00E-01	1.62E-01	4.69E-02	3.19E-01
Formaldehyde	6.12E-01	3.29E-02	7.20E-02	5.78E-01	1.88E-01	1.48E+00	4.15E-02	8.26E-02	2.46E-01	1.38E-01	1.12E+00
Hexane	0.00E+00	5.50E-02	3.60E-01	2.85E-01	7.13E-02	7.71E-01	4.94E-02	4.41E-01	1.44E-01	4.98E-02	6.85E-01
MTBE	0.00E+00	1.05E-02	4.84E-02	4.61E-01	3.40E-01	8.59E-01	1.26E-03	1.17E-03	1.48E-05	2.84E-06	2.45E-03
Manganese	0.00E+00	1.05E-03	8.93E-04	1.08E-05	2.40E-06	1.96E-03	1.13E-02	5.35E-02	1.24E-01	8.90E-02	2.78E-01
Naphthalene	0.00E+00	3.82E-03	3.37E-02	1.79E-02	3.85E-03	5.92E-02	3.37E-03	4.18E-02	9.89E-03	4.02E-03	5.91E-02
Nickel	0.00E+00	3.02E-04	5.78E-04	2.38E-05	4.17E-05	9.46E-04	3.47E-04	6.50E-04	3.29E-05	4.80E-05	1.08E-03
POM	0.00E+00	2.87E-03	1.00E-02	1.56E-03	5.48E-04	1.50E-02	2.26E-03	1.16E-02	8.33E-04	4.97E-04	1.52E-02
Propionaldehyde	0.00E+00	7.73E-03	1.80E-02	1.93E-01	3.35E-02	2.52E-01	7.24E-03	1.89E-02	9.56E-02	2.28E-02	1.45E-01
Styrene	0.00E+00	2.04E-02	1.14E-02	3.40E-02	3.03E-03	6.88E-02	2.40E-02	1.56E-02	1.73E-02	1.83E-03	5.86E-02
Toluene	0.00E+00	1.61E-01	6.57E-01	2.14E+00	3.42E-01	3.30E+00	1.16E-01	8.80E-01	1.09E+00	2.06E-01	2.29E+00
Xylenes	1.28E-01	8.08E-02	4.66E-01	1.21E+00	3.33E-01	2.22E+00	6.79E-02	6.43E-01	6.11E-01	1.85E-01	1.63E+00

Table 3.2-7 (cont'd). National Means of Census Tract Median Population Exposure Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030, Without Controls in this Rule.

		2020 annual average concentrations (µg m^{-3})					2030 annual average concentrations (µg m^{-3})				
Pollutant	**background (µg m^{-3})**	**major**	**area & other**	**onroad**	**nonroad**	**total (including background)**	**major**	**area & other**	**onroad**	**nonroad**	**total (including background)**
1,3-Butadiene	3.96E-02	1.86E-03	1.69E-02	2.98E-02	1.07E-02	9.88E-02	1.86E-03	1.69E-02	3.49E-02	1.21E-02	1.05E-01
2,2,4-Trimethylpentane	0.00E+00	9.37E-03	2.31E-02	4.16E-01	9.21E-02	5.41E-01	9.37E-03	2.31E-02	4.81E-01	1.00E-01	6.14E-01
Acetaldehyde	4.00E-01	2.52E-02	4.70E-02	4.85E-01	9.01E-02	1.05E+00	2.52E-02	4.70E-02	5.68E-01	9.78E-02	1.14E+00
Acrolein	0.00E+00	3.27E-03	2.07E-02	2.99E-02	1.58E-02	6.97E-02	3.27E-03	2.07E-02	3.51E-02	1.79E-02	7.70E-02
Benzene	3.05E-01	1.37E-02	1.42E-01	4.64E-01	1.02E-01	1.03E+00	1.37E-02	1.42E-01	5.40E-01	1.15E-01	1.12E+00
Chromium III	0.00E+00	4.59E-04	2.74E-04	2.90E-05	2.37E-05	7.86E-04	4.59E-04	2.74E-04	3.56E-05	2.43E-05	7.93E-04
Chromium VI	0.00E+00	6.14E-05	1.23E-04	1.96E-05	5.45E-06	2.09E-04	6.14E-05	1.23E-04	2.40E-05	5.62E-06	2.14E-04
Ethyl Benzene	0.00E+00	1.11E-02	1.10E-01	1.62E-01	4.83E-02	3.32E-01	1.11E-02	1.10E-01	1.87E-01	5.41E-02	3.62E-01
Formaldehyde	6.12E-01	4.71E-02	8.68E-02	2.52E-01	1.38E-01	1.14E+00	4.71E-02	8.68E-02	2.94E-01	1.51E-01	1.19E+00
Hexane	0.00E+00	5.44E-02	4.77E-01	1.33E-01	5.19E-02	7.17E-01	5.44E-02	4.77E-01	1.46E-01	5.83E-02	7.36E-01
MTBE	0.00E+00	1.27E-02	5.48E-02	1.01E-01	9.25E-02	2.61E-01	1.27E-02	5.48E-02	1.00E-01	1.04E-01	2.72E-01
Manganese	0.00E+00	1.40E-03	1.29E-03	1.62E-05	3.00E-06	2.71E-03	1.40E-03	1.29E-03	1.99E-05	3.35E-06	2.71E-03
Naphthalene	0.00E+00	3.78E-03	4.44E-02	9.84E-03	4.31E-03	6.23E-02	3.78E-03	4.44E-02	1.14E-02	4.94E-03	6.45E-02
Nickel	0.00E+00	3.77E-04	7.15E-04	3.62E-05	5.02E-05	1.18E-03	3.77E-04	7.15E-04	4.45E-05	5.47E-05	1.19E-03
POM	0.00E+00	2.51E-03	1.18E-02	8.63E-04	5.01E-04	1.57E-02	2.51E-03	1.18E-02	1.02E-03	5.58E-04	1.59E-02
Propionaldehyde	0.00E+00	7.27E-03	1.94E-02	9.81E-02	2.25E-02	1.47E-01	7.27E-03	1.94E-02	1.14E-01	2.42E-02	1.65E-01
Styrene	0.00E+00	2.74E-02	1.72E-02	1.80E-02	1.87E-03	6.45E-02	2.74E-02	1.72E-02	2.13E-02	2.10E-03	6.80E-02
Toluene	0.00E+00	1.30E-01	9.68E-01	1.10E+00	2.09E-01	2.41E+00	1.30E-01	9.68E-01	1.28E+00	2.30E-01	2.61E+00
Xylenes	1.28E-01	7.68E-02	7.10E-01	6.18E-01	1.87E-01	1.72E+00	7.68E-02	7.10E-01	7.17E-01	2.06E-01	1.84E+00

Table 3.2-8. National Means of Census Tract 90th Percentile Population Exposure Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030, Without Controls in this Rule.

		1999 annual average concentrations (μg m^{-3})					2015 annual average concentrations (μg m^{-3})				
Pollutant	**background (μg m^{-3})**	**major**	**area & other**	**onroad**	**nonroad**	**total (including background)**	**major**	**area & other**	**onroad**	**nonroad**	**total (including background)**
1,3-Butadiene	5.88E-02	2.03E-03	2.23E-02	1.00E-01	2.49E-02	2.08E-01	2.15E-03	2.16E-02	4.11E-02	1.39E-02	1.38E-01
2,2,4-Trimethylpentane	0.00E+00	2.65E-02	3.12E-02	1.42E+00	2.65E-01	1.75E+00	1.30E-02	3.56E-02	7.08E-01	1.54E-01	9.10E-01
Acetaldehyde	5.82E-01	3.48E-02	6.34E-02	1.27E+00	1.80E-01	2.13E+00	3.32E-02	6.27E-02	6.89E-01	1.28E-01	1.49E+00
Acrolein	0.00E+00	3.68E-03	3.36E-02	1.07E-01	2.82E-02	1.72E-01	3.72E-03	2.79E-02	4.36E-02	2.11E-02	9.64E-02
Benzene	4.50E-01	2.57E-02	1.71E-01	1.24E+00	2.28E-01	2.12E+00	1.70E-02	1.91E-01	6.52E-01	1.39E-01	1.45E+00
Chromium III	0.00E+00	4.55E-04	2.59E-04	2.88E-05	3.15E-05	7.74E-04	5.81E-04	3.51E-04	3.97E-05	3.35E-05	1.01E-03
Chromium VI	0.00E+00	6.16E-05	1.15E-04	1.92E-05	7.20E-06	2.03E-04	7.88E-05	1.57E-04	2.63E-05	7.67E-06	2.70E-04
Ethyl Benzene	0.00E+00	2.33E-02	1.19E-01	5.49E-01	1.35E-01	8.27E-01	1.51E-02	1.51E-01	2.63E-01	7.53E-02	5.04E-01
Formaldehyde	8.03E-01	4.21E-02	9.22E-02	7.89E-01	2.52E-01	1.98E+00	4.93E-02	9.74E-02	3.03E-01	1.67E-01	1.42E+00
Hexane	0.00E+00	7.54E-02	5.11E-01	4.32E-01	1.07E-01	1.13E+00	6.51E-02	5.95E-01	2.04E-01	7.06E-02	9.34E-01
MTBE	0.00E+00	1.46E-02	7.13E-02	7.22E-01	5.16E-01	1.32E+00	1.45E-02	7.24E-02	1.92E-01	1.34E-01	4.13E-01
Manganese	0.00E+00	1.44E-03	1.18E-03	1.47E-05	3.25E-06	2.64E-03	1.72E-03	1.55E-03	2.01E-05	3.85E-06	3.30E-03
Naphthalene	0.00E+00	4.81E-03	4.39E-02	2.44E-02	5.09E-03	7.83E-02	4.07E-03	5.13E-02	1.25E-02	4.99E-03	7.29E-02
Nickel	0.00E+00	4.25E-04	8.25E-04	3.52E-05	6.04E-05	1.35E-03	4.94E-04	9.09E-04	4.77E-05	6.89E-05	1.52E-03
POM	0.00E+00	3.68E-03	1.21E-02	2.04E-03	7.05E-04	1.85E-02	2.89E-03	1.38E-02	1.04E-03	6.14E-04	1.84E-02
Propionaldehyde	0.00E+00	1.30E-02	2.79E-02	3.36E-01	5.58E-02	4.33E-01	1.15E-02	2.72E-02	1.60E-01	3.57E-02	2.34E-01
Styrene	0.00E+00	2.87E-02	1.78E-02	5.90E-02	5.23E-03	1.11E-01	3.31E-02	2.31E-02	2.87E-02	3.01E-03	8.79E-02
Toluene	0.00E+00	2.52E-01	1.05E+00	3.61E+00	5.66E-01	5.48E+00	1.70E-01	1.32E+00	1.71E+00	3.21E-01	3.52E+00
Xylenes	2.04E-01	1.23E-01	7.05E-01	1.95E+00	5.25E-01	3.50E+00	9.59E-02	9.14E-01	9.13E-01	2.72E-01	2.40E+00

Table 3.2-8 (cont'd). National Means of Census Tract 90th Percentile Population Exposure Concentrations of Mobile Source Air Toxics in 1999, 2015, 2020, and 2030, Without Controls in this Rule.

		2020 annual average concentrations (μg m^{-3})					2030 annual average concentrations (μg m^{-3})				
Pollutant	background (μg m^{-3})	major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	5.88E-02	2.32E-03	2.16E-02	4.28E-02	1.48E-02	1.40E-01	2.32E-03	2.16E-02	5.11E-02	1.72E-02	1.51E-01
2,2,4-Trimethylpentane	0.00E+00	1.40E-02	3.75E-02	7.09E-01	1.54E-01	9.14E-01	1.40E-02	3.75E-02	8.25E-01	1.69E-01	1.05E+00
Acetaldehyde	5.82E-01	3.47E-02	6.42E-02	7.14E-01	1.28E-01	1.52E+00	3.47E-02	6.42E-02	8.55E-01	1.41E-01	1.68E+00
Acrolein	0.00E+00	4.15E-03	2.70E-02	4.53E-02	2.23E-02	9.87E-02	4.15E-03	2.70E-02	5.37E-02	2.56E-02	1.10E-01
Benzene	4.50E-01	1.86E-02	1.99E-01	6.68E-01	1.44E-01	1.48E+00	1.86E-02	1.99E-01	7.93E-01	1.65E-01	1.63E+00
Chromium III	0.00E+00	6.51E-04	3.97E-04	4.37E-05	3.39E-05	1.13E-03	6.51E-04	3.97E-04	5.40E-05	3.48E-05	1.14E-03
Chromium VI	0.00E+00	8.98E-05	1.77E-04	2.90E-05	7.78E-06	3.04E-04	8.98E-05	1.77E-04	3.58E-05	8.04E-06	3.11E-04
Ethyl Benzene	0.00E+00	1.68E-02	1.65E-01	2.62E-01	7.71E-02	5.21E-01	1.68E-02	1.65E-01	3.06E-01	8.71E-02	5.75E-01
Formaldehyde	8.03E-01	5.60E-02	1.02E-01	3.11E-01	1.67E-01	1.44E+00	5.60E-02	1.02E-01	3.70E-01	1.86E-01	1.52E+00
Hexane	0.00E+00	7.12E-02	6.39E-01	1.86E-01	7.27E-02	9.69E-01	7.12E-02	6.39E-01	2.06E-01	8.21E-02	9.98E-01
MTBE	0.00E+00	1.61E-02	7.34E-02	1.52E-01	1.37E-01	3.78E-01	1.61E-02	7.34E-02	1.50E-01	1.53E-01	3.93E-01
Manganese	0.00E+00	1.92E-03	1.71E-03	2.21E-05	4.07E-06	3.65E-03	1.92E-03	1.71E-03	2.72E-05	4.54E-06	3.66E-03
Naphthalene	0.00E+00	4.55E-03	5.44E-02	1.24E-02	5.33E-03	7.66E-02	4.55E-03	5.44E-02	1.45E-02	6.16E-03	7.96E-02
Nickel	0.00E+00	5.39E-04	1.00E-03	5.25E-05	7.19E-05	1.66E-03	5.39E-04	1.00E-03	6.45E-05	7.86E-05	1.68E-03
POM	0.00E+00	3.21E-03	1.42E-02	1.08E-03	6.21E-04	1.91E-02	3.21E-03	1.42E-02	1.29E-03	6.97E-04	1.94E-02
Propionaldehyde	0.00E+00	1.16E-02	2.78E-02	1.65E-01	3.52E-02	2.39E-01	1.16E-02	2.78E-02	1.94E-01	3.83E-02	2.72E-01
Styrene	0.00E+00	3.78E-02	2.55E-02	2.99E-02	3.07E-03	9.64E-02	3.78E-02	2.55E-02	3.57E-02	3.47E-03	1.03E-01
Toluene	0.00E+00	1.88E-01	1.44E+00	1.73E+00	3.23E-01	3.68E+00	1.88E-01	1.44E+00	2.04E+00	3.61E-01	4.03E+00
Xylenes	2.04E-01	1.08E-01	1.01E+00	9.22E-01	2.74E-01	2.51E+00	1.08E-01	1.01E+00	1.09E+00	3.07E-01	2.71E+00

Table 3.2-9. National Average Cancer Risk Across Census Tracts for 1999, 2015, 2020, and 2030 by Pollutant, Without Controls in this Rule.

	1999 average individual risk					2015 annual average individual risk				
Pollutant	major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	4.36E-08	4.85E-07	2.06E-06	5.39E-07	4.43E-06	4.62E-08	4.50E-07	8.69E-07	3.20E-07	2.97E-06
Acetaldehyde	5.65E-08	1.10E-07	1.96E-06	2.89E-07	3.39E-06	5.59E-08	1.16E-07	1.08E-06	2.12E-07	2.43E-06
Benzene	1.49E-07	9.82E-07	6.79E-06	1.30E-06	1.18E-05	1.00E-07	1.13E-06	3.66E-06	8.25E-07	8.33E-06
Chromium VI	5.32E-07	9.43E-07	1.69E-07	7.18E-08	1.72E-06	6.67E-07	1.25E-06	2.29E-07	8.11E-08	2.23E-06
Formaldehyde	1.81E-10	4.51E-10	3.36E-09	1.11E-09	8.69E-09	2.10E-10	5.18E-10	1.35E-09	7.69E-10	6.43E-09
Naphthalene	1.21E-07	1.22E-06	6.38E-07	1.37E-07	2.11E-06	1.01E-07	1.46E-06	3.43E-07	1.39E-07	2.04E-06
Nickel	4.81E-08	9.79E-08	4.17E-09	6.20E-09	1.56E-07	5.53E-08	1.07E-07	5.65E-09	6.87E-09	1.75E-07
POM	1.77E-07	1.06E-06	1.05E-07	3.62E-08	1.38E-06	1.46E-07	1.25E-06	5.39E-08	3.25E-08	1.48E-06

	2020 annual average individual risk					2030 annual average individual risk				
Pollutant	major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	4.95E-08	4.38E-07	8.92E-07	3.39E-07	3.00E-06	4.82E-08	4.19E-07	1.03E-06	3.86E-07	3.16E-06
Acetaldehyde	5.80E-08	1.19E-07	1.10E-06	2.08E-07	2.46E-06	5.75E-08	1.19E-07	1.28E-06	2.23E-07	2.65E-06
Benzene	1.09E-07	1.17E-06	3.71E-06	8.54E-07	8.45E-06	1.08E-07	1.16E-06	4.29E-06	9.59E-07	9.13E-06
Chromium VI	7.53E-07	1.40E-06	2.50E-07	8.34E-08	2.49E-06	7.48E-07	1.38E-06	3.05E-07	8.78E-08	2.52E-06
Formaldehyde	2.34E-10	5.47E-10	1.38E-09	7.63E-10	6.49E-09	2.28E-10	5.54E-10	1.59E-09	8.22E-10	6.76E-09
Naphthalene	1.12E-07	1.54E-06	3.39E-07	1.48E-07	2.14E-06	1.09E-07	1.52E-06	3.91E-07	1.69E-07	2.19E-06
Nickel	6.02E-08	1.16E-07	6.19E-09	7.10E-09	1.90E-07	6.01E-08	1.15E-07	7.55E-09	7.60E-09	1.90E-07
POM	1.61E-07	1.30E-06	5.54E-08	3.27E-08	1.55E-06	1.61E-07	1.31E-06	6.52E-08	3.59E-08	1.57E-06

Figure 3.2-4. Contributions to Average Inhalation Cancer Risk from Air Toxics Emitted by Mobile Sources, 2020 (Not Including Diesel PM and Diesel Exhaust Organic Gases), Without Controls in this Rule.

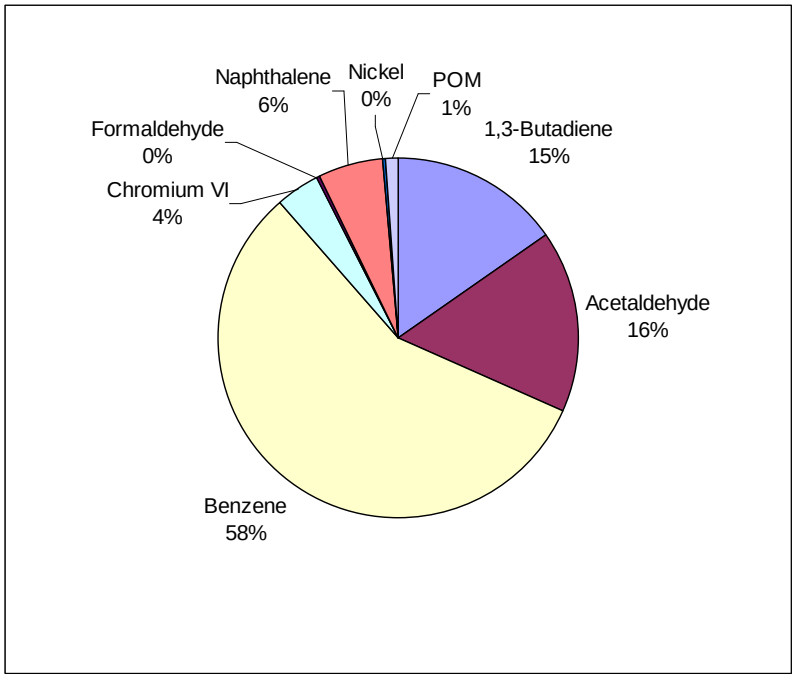
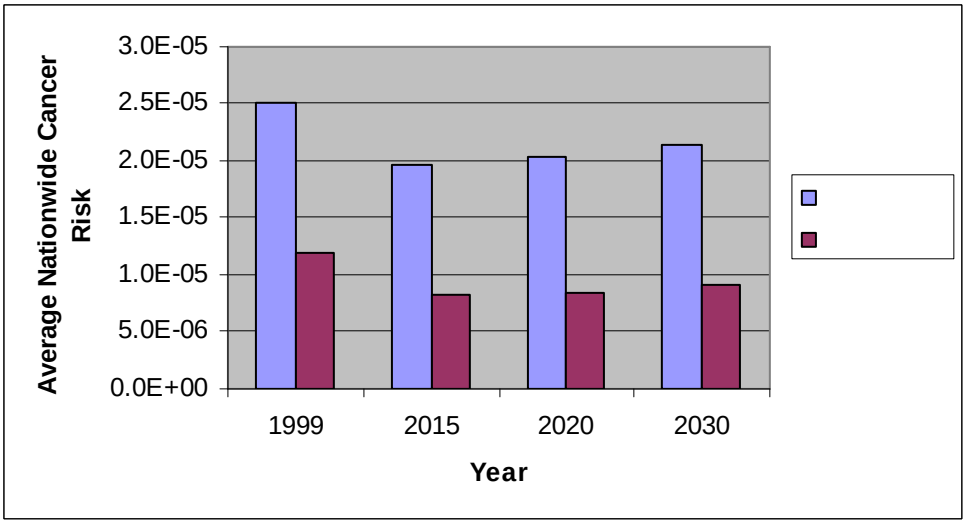
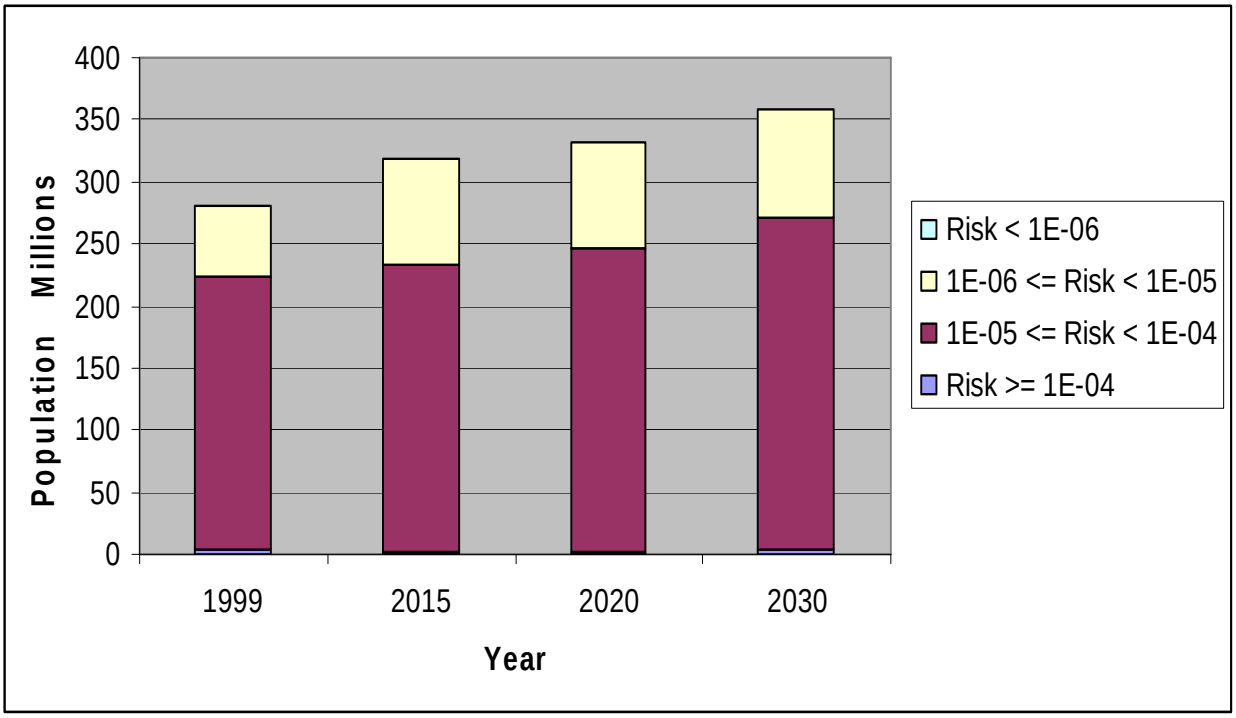


Figure 3.2-5. Average Nationwide Cancer Risk from Emissions of Mobile Source Air Toxics from both Mobile and Stationary Sources across Census Tracts, 1999 to 2030 (Not Including Diesel PM and Diesel Exhaust Organic Gases), Without Controls in this Rule.



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 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),[160] and is also discussed in Chapter 12 of the RIA). These statistics do not include populations in Alaska and Hawaii; thus populations in these States were assumed to remain at year 2000 levels. More details on the methodology used to project the U. S. population above various cancer risk benchmarks are provided in the technical support document "National-Scale Modeling of Mobile Source Air Toxic Emissions, Air Quality, Exposure and Risk for the Final Mobile Source Air Toxics Rule." From Figure 3.2-6 it can be seen that, based on average census tract risks, the vast majority of the population experiences risks between one in a million ($1x10^{-6}$) and one in ten thousand ($1x10^{-4}$). However, the number of people experiencing risks above one in a hundred thousand ($1x10^{-5}$) increases from 223 million in 1999 to 272 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, Without Controls in this Rule.



Tables 3.2-10 and 3.2-11 summarize national average population hazard quotients 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 33% between 1999 and 2030 (Figure 3.2-7), it is still over 4 in 2030, indicating a potential for adverse health effects. The reduction in hazard index occurs despite large increases in activity for highway and nonroad sources. In addition,

about 90% of this non-cancer risk is attributable to acrolein in all projection years. It should be noted that the confidence in the RfC for acrolein is medium. About 25% of primary acrolein emissions are from mobile sources, and about 70% of ambient concentrations of acrolein (and about 75% of exposure) are 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 and increasing vehicle miles traveled will increase the number of Americans with a respiratory hazard index for mobile source air toxics above one, from 258 million in 1999 to 307 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.: Reference Case

Table 3.2-12 gives the distribution of nationwide individual cancer risks for mobile source air toxics in 2020, absent the controls being finalized in this rule. 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 three 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, and separately for 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. Higher risks are also seen in States with colder winters, due to elevated cold start emissions.

Previously discussed changes to the HAPEM exposure model, to account for near road impacts, can impact distributions of risk. In order to evaluate the effect of switching to HAPEM6 from HAPEM5 on individual risks nationally, we conducted model runs using identical input data. Figure 3.2-13 depicts the national distribution of individual cancer risks from benzene, comparing HAPEM6 and HAPEM5. Note that the graph is on a logarithmic scale. As the graph illustrates, when HAPEM6 is used, there are fewer individuals with lower benzene cancer risk levels (e.g. $<1x10^{-6}$) in 1999. The population with higher benzene risk levels (e.g. $>1x10^{-4}$) is higher with HAPEM6 than HAPEM5. In general, the distribution of cancer risks shifts slightly higher when comparing HAPEM6 to HAPEM5, but the largest effects are observed in the populations with the highest and lowest risk levels, which are generally small fractions of the total population.

Table 3.2-13 gives the distribution of nationwide individual 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 over 20 times that at the 5th percentile, and about 4 times the median. Thus, some populations are experiencing much higher hazard indices than others. Figure 3.2-14 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. This error was discovered at too late a date to produce and update emissions inventories for use in the analyses undertaken for this rule. The errors are not expected to affect the analyses of the impacts of controls undertaken for this rule.

Table 3.2-10. National Average Population Hazard Quotient for Chronic Noncancer Effects Across Census Tracts, 1999 – 2030, Without Controls in this Rule.

		1999 average Hazard Quotient					2015 average Hazard Quotient				
Pollutant	Target System	major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	Reproductive	7.27E-04	8.08E-03	3.43E-02	8.98E-03	7.39E-02	7.69E-04	7.49E-03	1.45E-02	5.34E-03	4.96E-02
Acetaldehyde	Respiratory	2.86E-03	5.54E-03	9.92E-02	1.46E-02	1.71E-01	2.82E-03	5.84E-03	5.46E-02	1.07E-02	1.23E-01
Acrolein	Respiratory	1.44E-01	1.28E+00	3.70E+00	1.03E+00	6.16E+00	1.58E-01	1.13E+00	1.54E+00	8.10E-01	3.63E+00
Benzene	Immunological	6.35E-04	4.20E-03	2.90E-02	5.55E-03	5.06E-02	4.29E-04	4.83E-03	1.56E-02	3.53E-03	3.56E-02
Chromium VI	Respiratory	4.43E-04	7.86E-04	1.41E-04	5.98E-05	1.43E-03	5.56E-04	1.04E-03	1.90E-04	6.76E-05	1.86E-03
Ethyl Benzene	Developmental	1.60E-05	8.09E-05	3.60E-04	9.17E-05	5.48E-04	1.05E-05	1.05E-04	1.74E-04	5.30E-05	3.42E-04
Formaldehyde	Respiratory	3.36E-03	8.37E-03	6.23E-02	2.05E-02	1.61E-01	3.90E-03	9.62E-03	2.51E-02	1.43E-02	1.19E-01
Hexane	Neurological, Respiratory	2.76E-04	1.89E-03	1.55E-03	3.95E-04	4.11E-03	2.43E-04	2.21E-03	7.58E-04	2.71E-04	3.48E-03
MTBE	Liver, Kidney, Ocular	3.86E-06	1.76E-05	1.72E-04	1.28E-04	3.21E-04	3.94E-06	1.88E-05	4.43E-05	3.20E-05	9.90E-05
Manganese	Neurological	2.04E-02	1.93E-02	2.27E-04	4.59E-05	3.99E-02	2.65E-02	2.56E-02	3.07E-04	5.32E-05	5.24E-02
Naphthalene	Respiratory	1.19E-03	1.19E-02	6.25E-03	1.35E-03	2.07E-02	9.88E-04	1.43E-02	3.36E-03	1.36E-03	2.00E-02
Nickel	Respiratory, Immunological	4.62E-03	9.42E-03	4.01E-04	5.96E-04	1.50E-02	5.32E-03	1.03E-02	5.43E-04	6.61E-04	1.68E-02
Styrene	Neurological	2.38E-05	1.28E-05	3.77E-05	3.46E-06	7.78E-05	2.85E-05	1.76E-05	1.84E-05	2.05E-06	6.66E-05
Toluene	Respiratory, Neurological	4.55E-04	1.82E-03	5.96E-03	9.69E-04	9.20E-03	3.12E-04	2.39E-03	2.88E-03	5.72E-04	6.16E-03
Xylenes	Neurological	8.47E-04	5.00E-03	1.32E-02	3.72E-03	2.43E-02	6.85E-04	6.69E-03	6.38E-03	2.02E-03	1.72E-02

Table 3.2-10 (cont'd). National Average Population Hazard Quotient for Chronic Noncancer Effects Across Census Tracts, Without Controls in this Rule.

		2020 average Hazard Quotient					2030 average Hazard Quotient				
Pollutant	Target System	major	area & other	onroad	nonroad	total (including background)	major	area & other	onroad	nonroad	total (including background)
1,3-Butadiene	Reproductive	8.25E-04	7.30E-03	1.49E-02	5.64E-03	5.00E-02	8.03E-04	6.98E-03	1.72E-02	6.43E-03	5.26E-02
Acetaldehyde	Respiratory	2.93E-03	5.99E-03	5.58E-02	1.05E-02	1.24E-01	2.90E-03	6.02E-03	6.47E-02	1.13E-02	1.34E-01
Acrolein	Respiratory	1.78E-01	1.09E+00	1.57E+00	8.52E-01	3.69E+00	1.78E-01	1.08E+00	1.82E+00	9.62E-01	4.04E+00
Benzene	Immunological	4.67E-04	4.99E-03	1.58E-02	3.65E-03	3.61E-02	4.63E-04	4.96E-03	1.83E-02	4.10E-03	3.90E-02
Chromium VI	Respiratory	6.28E-04	1.17E-03	2.09E-04	6.95E-05	2.07E-03	6.23E-04	1.15E-03	2.54E-04	7.32E-05	2.10E-03
Ethyl Benzene	Developmental	1.17E-05	1.14E-04	1.72E-04	5.44E-05	3.52E-04	1.15E-05	1.12E-04	1.96E-04	6.09E-05	3.81E-04
Formaldehyde	Respiratory	4.34E-03	1.02E-02	2.55E-02	1.42E-02	1.20E-01	4.23E-03	1.03E-02	2.95E-02	1.53E-02	1.25E-01
Hexane	Neurological, Respiratory	2.66E-04	2.37E-03	6.92E-04	2.82E-04	3.61E-03	2.65E-04	2.32E-03	7.53E-04	3.17E-04	3.66E-03
MTBE	Liver, Kidney, Ocular	4.36E-06	1.91E-05	3.53E-05	3.28E-05	9.16E-05	4.26E-06	1.90E-05	3.44E-05	3.62E-05	9.38E-05
Manganese	Neurological	2.99E-02	2.80E-02	3.37E-04	5.59E-05	5.83E-02	3.08E-02	2.81E-02	4.11E-04	6.15E-05	5.94E-02
Naphthalene	Respiratory	1.09E-03	1.51E-02	3.33E-03	1.45E-03	2.10E-02	1.07E-03	1.49E-02	3.83E-03	1.65E-03	2.14E-02
Nickel	Respiratory, Immunological	5.78E-03	1.12E-02	5.95E-04	6.83E-04	1.83E-02	5.78E-03	1.10E-02	7.26E-04	7.30E-04	1.83E-02
Styrene	Neurological	3.29E-05	1.96E-05	1.90E-05	2.09E-06	7.36E-05	3.32E-05	1.97E-05	2.22E-05	2.32E-06	7.74E-05
Toluene	Respiratory, Neurological	3.47E-04	2.63E-03	2.89E-03	5.78E-04	6.45E-03	3.44E-04	2.63E-03	3.32E-03	6.37E-04	6.93E-03
Xylenes	Neurological	7.69E-04	7.35E-03	6.39E-03	2.04E-03	1.80E-02	7.59E-04	7.26E-03	7.33E-03	2.25E-03	1.90E-02

Table 3.2-11. National Respiratory Hazard Index for Chronic Noncancer Effects across Census Tracts, Without Controls in this Rule.

Respiratory System Average Hazard Index						
Year	background	major	area & other	onroad	nonroad	total (including background)
1999	0.12	0.16	1.32	3.88	1.07	6.54
2015	0.12	0.17	1.17	1.63	0.84	3.92
2020	0.12	0.19	1.14	1.66	0.88	3.99
2030	0.11	0.19	1.13	1.92	0.99	4.35

Figure 3.2-7. Average Respiratory Hazard Index for U.S. Population (Aggregate of Hazard Quotients for Individual Pollutants), Without Controls in this Rule.

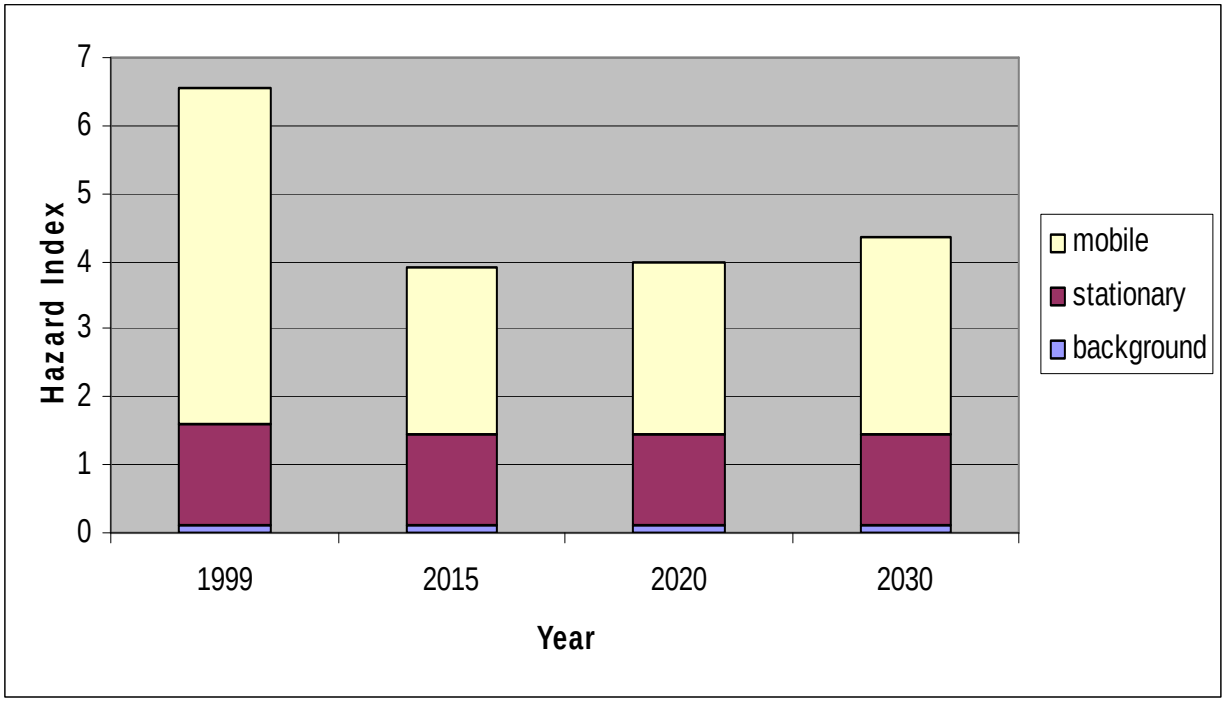


Figure 3.2-8. U. S. Population at Various Non-Cancer Hazard Benchmarks due to Exposure to Mobile Source Air Toxics, 1999 – 2030, Without Controls in this Rule.

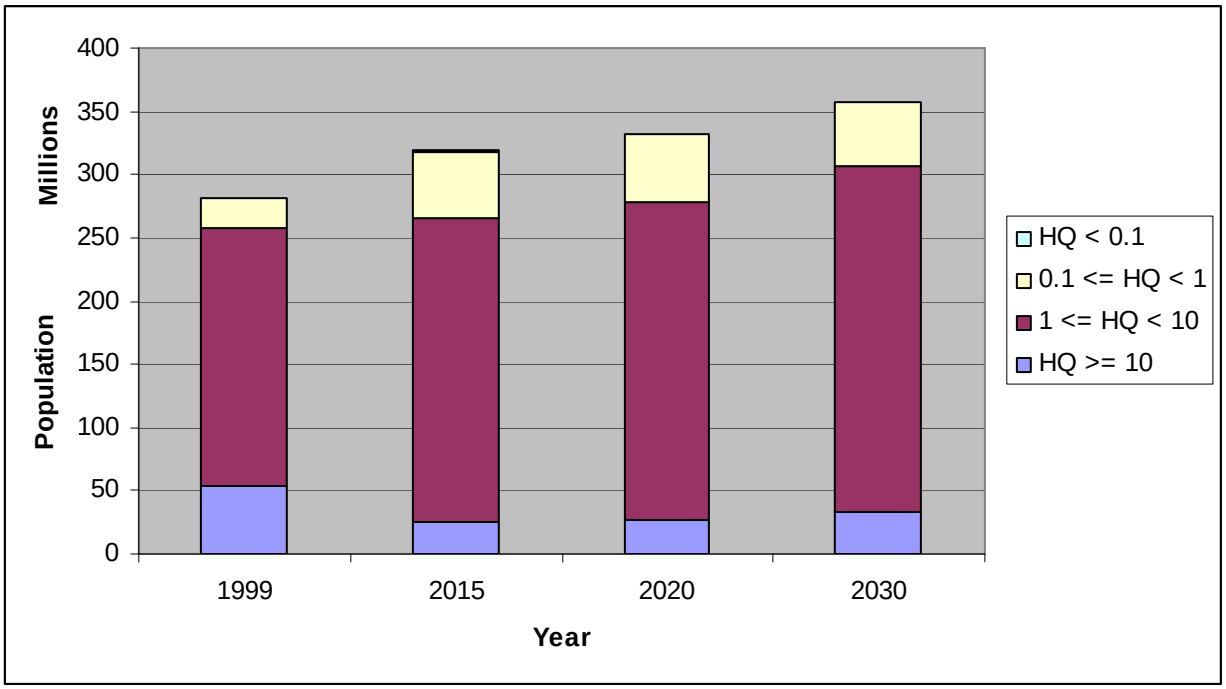


Table 3.2-12. Distribution of Individual Cancer Risks for Mobile Source Air Toxics in 2020, Without Controls in this Rule.

	2020 risk distribution						
Pollutant	**5th percentile**	**10th percentile**	**25th percentile**	**Median**	**75th percentile**		**95th percentile**
Total Risk: All HAPs	4.71E-06	6.08E-06	9.78E-06	1.53E-05	2.37E-05	3.79E-05	4.93E-05
1,3-Butadiene	1.52E-07	2.96E-07	1.06E-06	2.30E-06	3.60E-06	5.47E-06	7.70E-06
Acetaldehyde	1.09E-06	1.19E-06	1.46E-06	1.96E-06	2.81E-06	4.20E-06	5.35E-06
Benzene	2.72E-06	3.36E-06	4.84E-06	6.93E-06	1.00E-05	1.48E-05	1.86E-05
Chromium VI	3.85E-08	7.93E-08	2.38E-07	7.01E-07	1.81E-06	4.54E-06	7.29E-06
Formaldehyde	2.29E-09	2.89E-09	4.12E-09	5.75E-09	7.67E-09	1.05E-08	1.29E-08
Naphthalene	1.59E-07	2.80E-07	6.72E-07	1.39E-06	2.61E-06	4.73E-06	6.68E-06
Nickel	1.84E-09	4.09E-09	1.39E-08	4.60E-08	1.31E-07	3.04E-07	5.06E-07
POM	1.26E-07	1.90E-07	3.48E-07	6.78E-07	1.19E-06	1.99E-06	3.07E-06

Figure 3.2-9. 2020 County Median Cancer Risk for All Mobile Source Air Toxics, Without Controls in this Rule.

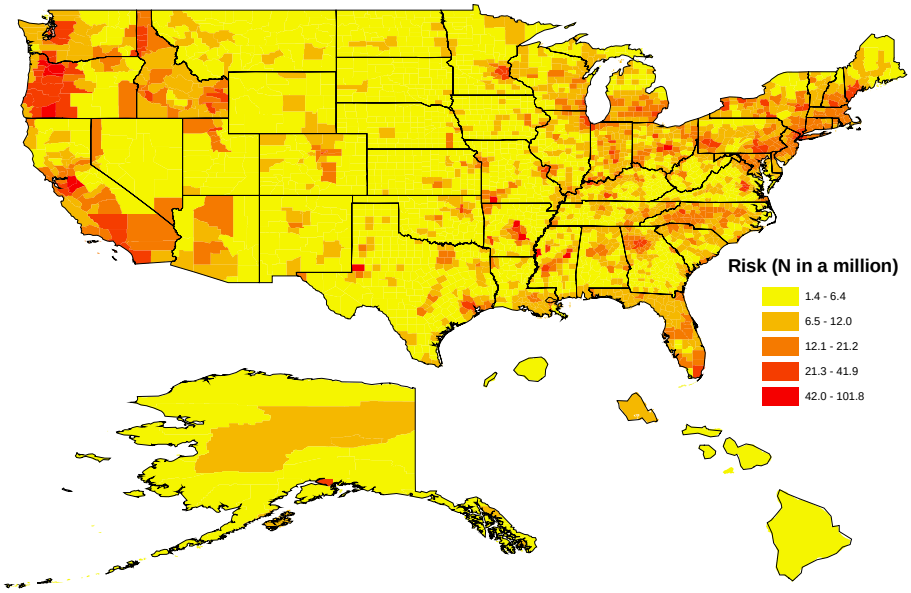


Figure 3.2-10. 2020 County Median Cancer Risk for Benzene, Without Controls in this Rule.

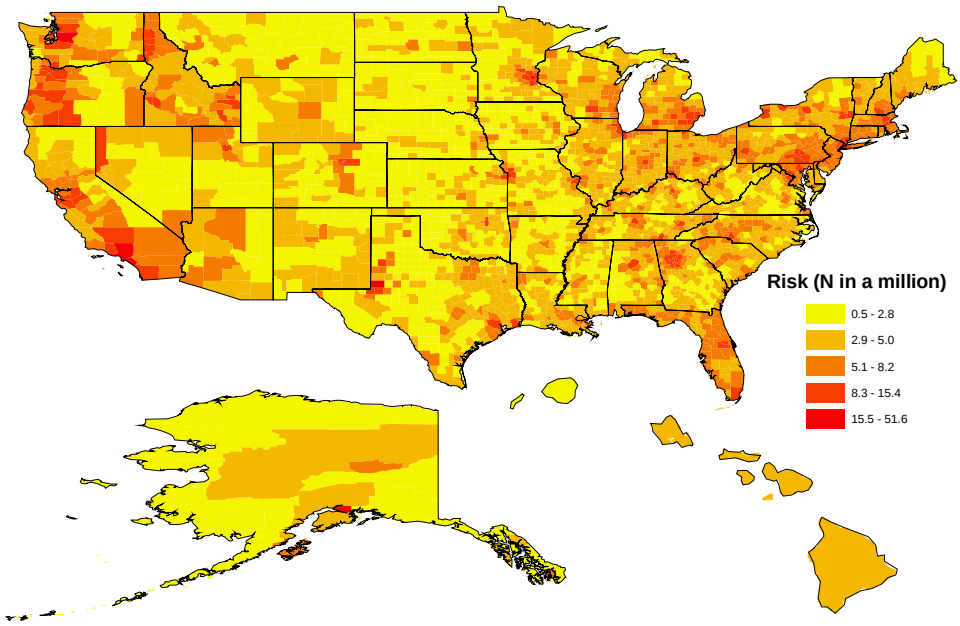


Figure 3.2-11. 2020 County Median Cancer Risk for Acetaldehyde, Without Controls in this Rule.

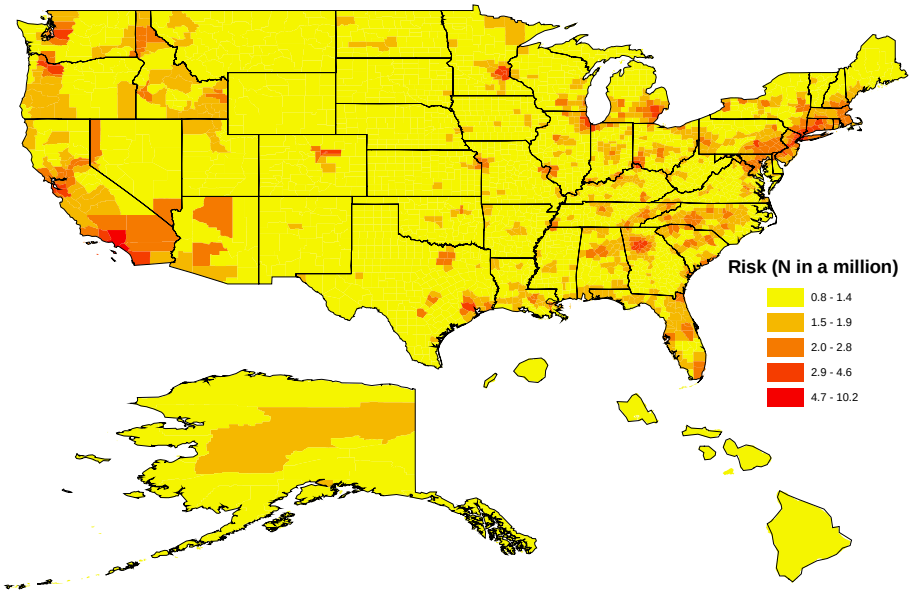


Figure 3.2-12. 2020 County Median Cancer Risk for 1,3-Butadiene, Without Controls in this Rule.

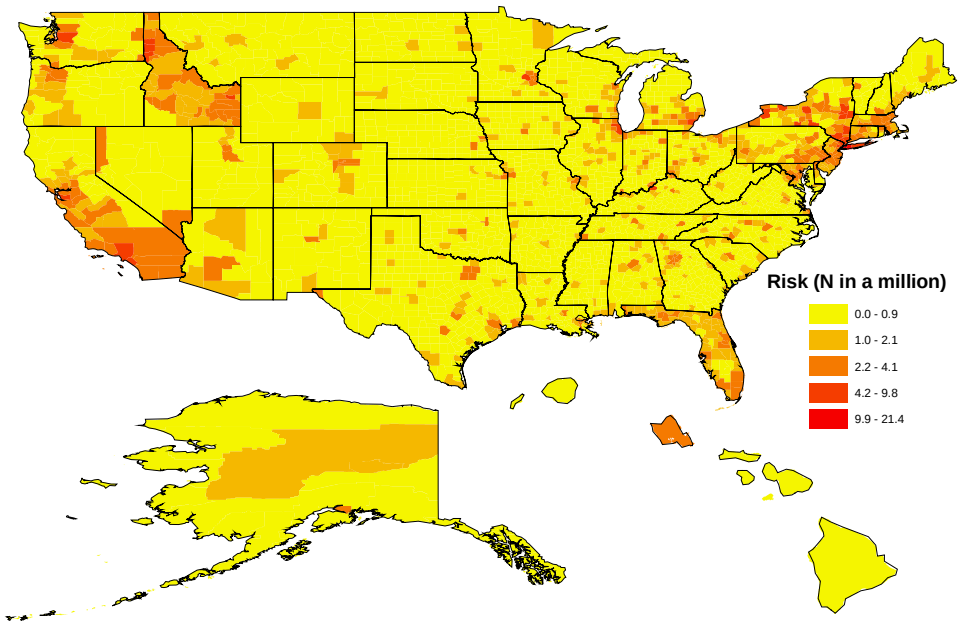


Figure 3.2-13. 1999 Comparison Between HAPEM6 and HAPEM5 Nationwide Individual Benzene Cancer Risk, Without Controls in this Rule.

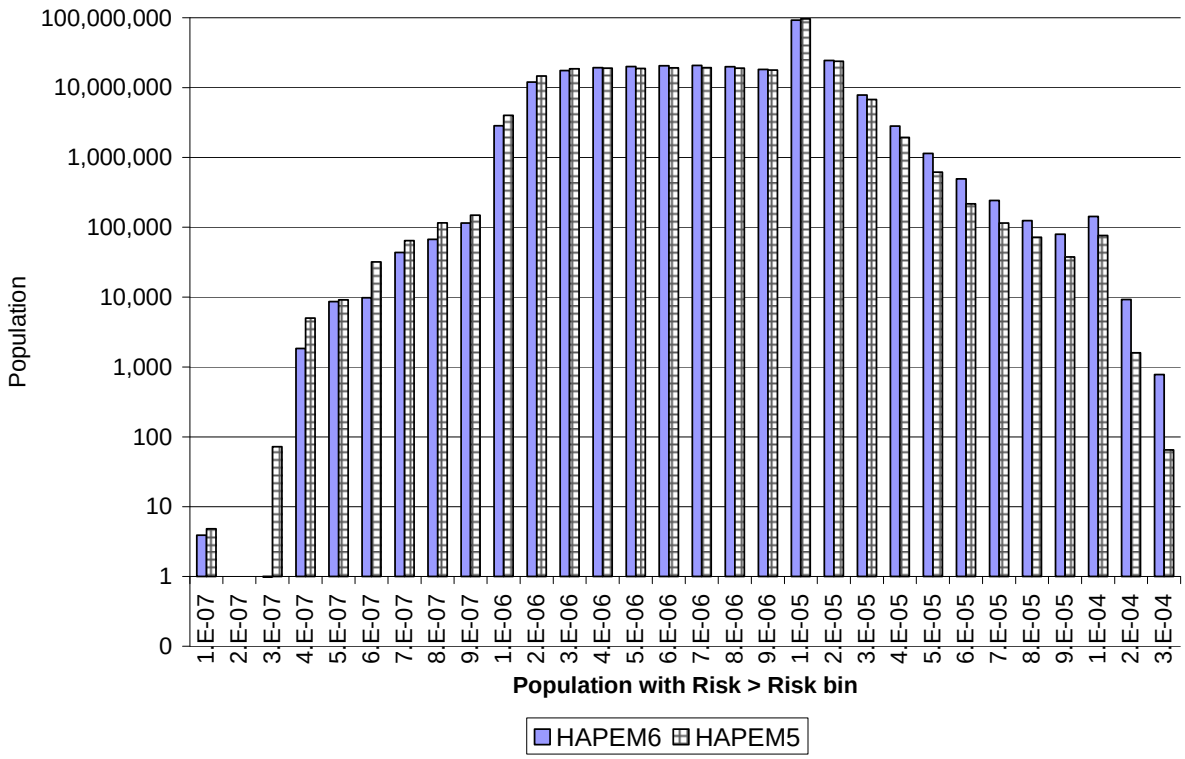
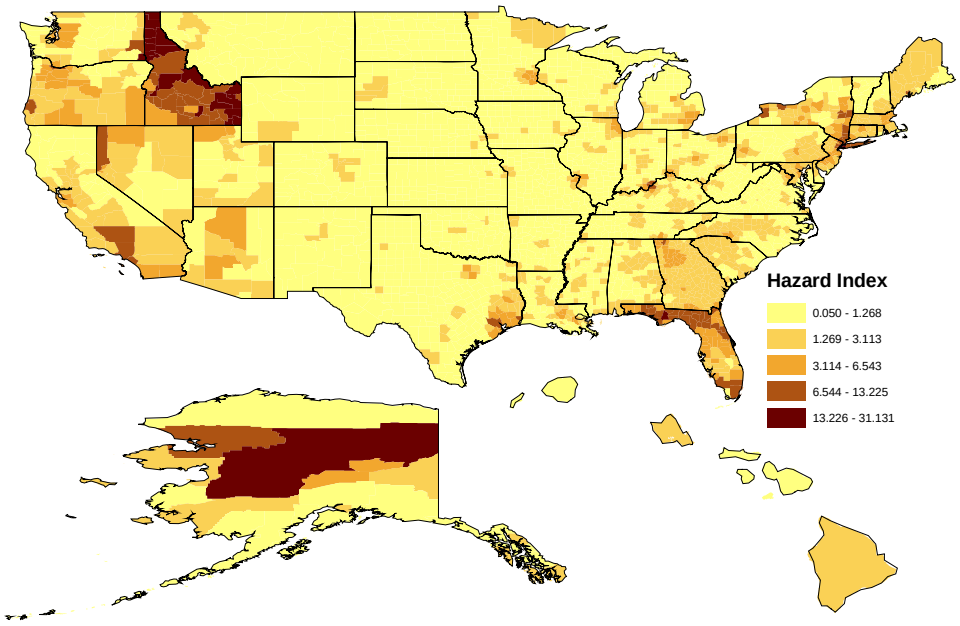


Table 3.2-13. Distribution of Individual Hazard Quotients/Hazard Indices for Mobile Source Air Toxics (from both Mobile and Stationary Sources) in 2020, Without Controls in this Rule.

	2020 average Hazard Quotient or Hazard Index						
Pollutant	5th percentile	10th percentile	25th percentile	Median	75th percentile	90th percentile	95th percentile
Acrolein	0.41	0.61	1.18	2.31	4.47	8.05	11.3
Respiratory System	0.53	0.75	1.36	2.57	4.83	8.54	11.9

Figure 3.2-14. 2020 County Median Non-Cancer Hazard Index Respiratory Mobile Source Air Toxics, Without Controls in this Rule.



3.2.1.2.4 Impacts of Controls on Average Inhalation Cancer Risks and Noncancer Hazards

The standards being finalized in this rule will substantially reduce inhalation cancer and noncancer risk from exposure to air toxics emitted by mobile sources across the United States. Table 3.2-14 shows that in 2030, the highway vehicle contribution to MSAT cancer risk will be reduced on average 36% across the U.S., and the nonroad equipment contribution will be reduced about 6%. In 2030, the highway vehicle contribution to benzene cancer risk will be reduced on average by 43% across the U.S., and the nonroad contribution will be reduced by 11%. Table 3.2-15 summarizes the change in median and 95th percentile inhalation cancer risks from benzene and all MSATs attributable to all outdoor sources in 2015, 2020, and 2030, with the controls being finalized in this rule. Reductions are significantly larger for individuals in the 95th percentile than in the 50th percentile. Thus, this rule is providing bigger benefits to individuals experiencing the highest levels of risk. In states with high fuel benzene levels and high cold start emissions, the cancer risk reduction from total MSATs is about 40% or higher (Table 3.2-16).[d] Figure 3.2-15 depicts the impact on the mobile source contribution to nationwide average population cancer risk from all MSATs and benzene in 2030. Nationwide, the cancer risk attributable to total MSATs would be reduced by 30%, and the risk from mobile source benzene would be reduced by 37%. Figures 3.2-16 and 3.2.-17 present the distribution of percent reductions in average MSAT and benzene cancer risk, respectively, from all sources in 2030 with the controls being finalized in 2030. Table 3.2-17 shows reductions in hazard quotients and hazard indices for acrolein and respiratory effects, respectively. Nationwide, the mobile source contribution to the acrolein hazard quotient and respiratory hazard index would both be reduced about 23%, and the highway vehicle contribution will be reduced about 35%. Summary tables providing exposure and risk data by State, as well as maps of cancer risks and noncancer hazards with controls and percent reductions with controls, can be found in the docket for the rule.

It should be noted that the estimated total relative 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. Again, as noted previously, since this modeling did not include the 1.3 vol% maximum average fuel benzene level, reductions in risk for some parts of the country, such as the Pacific Northwest, are underestimated.

[d] Reductions are likely to be higher than estimated by this modeling, due to the 1.3% maximum average fuel benzene level.

Table 3.2-14. Contributions of Source Sectors to Nationwide Average Cumulative MSAT Cancer Risk, With and Without Controls, 2015, 2020, and 2030

	2015 Average Risks					2020 Average Risks					2030 Average Risks				
	major	area & other	total onroad	total nonroad	total (including background)	major	area & other	total onroad	total nonroad	total (including background)	major	area & other	total onroad	total nonroad	total (including background)
Total MSATs															
Reference	1.17E-06	5.76E-06	6.24E-06	1.62E-06	1.97E-05	1.30E-06	6.08E-06	6.35E-06	1.67E-06	2.03E-05	1.29E-06	6.02E-06	7.37E-06	1.87E-06	2.14E-05
Control	1.17E-06	5.74E-06	4.98E-06	1.53E-06	1.83E-05	1.30E-06	6.06E-06	4.58E-06	1.58E-06	1.84E-05	1.29E-06	6.01E-06	4.69E-06	1.77E-06	1.86E-05
% Difference	0.0	0.3	20.2	5.3	6.9	0.0	0.3	27.9	5.5	9.3	0.0	0.3	36.3	5.6	13.1
Benzene															
Reference	1.00E-07	1.13E-06	3.66E-06	8.25E-07	8.33E-06	1.09E-07	1.17E-06	3.71E-06	8.54E-07	8.45E-06	1.08E-07	1.16E-06	4.29E-06	9.59E-07	9.13E-06
Control	1.00E-07	1.12E-06	2.73E-06	7.38E-07	7.30E-06	1.09E-07	1.15E-06	2.45E-06	7.62E-07	7.09E-06	1.08E-07	1.15E-06	2.43E-06	8.54E-07	7.15E-06
% Difference	0.3	1.3	25.4	10.5	12.3	0.3	1.3	34.0	10.8	16.2	0.3	1.3	43.4	10.9	21.7

Table 3.2-15. Change in Median and 95th Percentile Inhalation Cancer Risk from Benzene and all MSATs Attributable to Outdoor Sources in 2015, 2020, and 2030 with the Controls Being Finalized in this Rule.

	2015		2020		2030	
	median	95th	median	95th	median	95th
All MSATs						
Without Controls	$1.50x10^{-5}$	$4.75x10^{-5}$	$1.53x10^{-5}$	$4.93x10^{-5}$	$1.61x10^{-5}$	$5.28x10^{-5}$
With Controls	$1.41x10^{-5}$	$4.37x10^{-5}$	$1.40x10^{-5}$	$4.40x10^{-5}$	$1.42x10^{-5}$	$4.49x10^{-5}$
Percent Change	6	8	8	11	12	15
Benzene						
Without Controls	$6.86x10^{-6}$	$1.82x10^{-5}$	$6.93x10^{-6}$	$1.86x10^{-5}$	$7.37x10^{-6}$	$2.06x10^{-5}$
With Controls	$6.17x10^{-6}$	$1.53x10^{-5}$	$6.02x10^{-6}$	$1.47x10^{-5}$	$6.06x10^{-6}$	$1.49x10^{-5}$
Percent Change	10	16	13	21	18	28

Table 3.2-16. States with Highest Reductions in Average Benzene Cancer Risk Resulting from Mobile Source Emissions, 2030.

State	Average Risk – Reference Case	Average Risk – Control Case	Percent Difference
Alaska	$1.01x10^{-5}$	$4.23x10^{-6}$	-58%
North Dakota	$2.92x10^{-6}$	$1.68x10^{-6}$	-42
Washington	$1.39x10^{-5}$	$8.10x10^{-6}$	-42
Minnesota	$1.21x10^{-5}$	$7.08x10^{-6}$	-42
Wyoming	$2.38x10^{-6}$	$1.39x10^{-6}$	-41
Montana	$3.12x10^{-6}$	$1.87x10^{-6}$	-40
Idaho	$5.03x10^{-6}$	$3.02x10^{-6}$	-40
Michigan	$1.09x10^{-5}$	$6.55x10^{-6}$	-40
South Dakota	$2.73x10^{-6}$	$1.66x10^{-6}$	-39
Oregon	$1.01x10^{-5}$	$6.17x10^{-6}$	-39

Figure 3.2-15. Contribution to Nationwide Average Population Cancer Risk from Mobile Source MSATs and Benzene Emitted by Mobile Sources in 2030, Without and With Controls in this Rule.

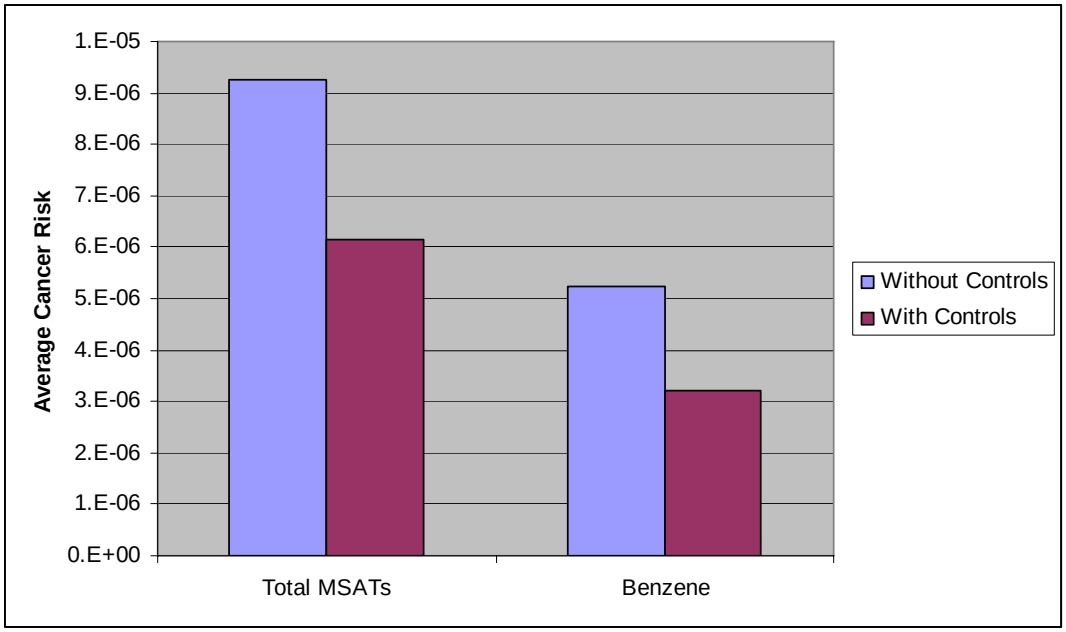


Figure 3.2-16. Distribution of Percent Reductions in Median MSAT Cancer Risk, 2030, for U.S. Counties with Controls in this Rule.

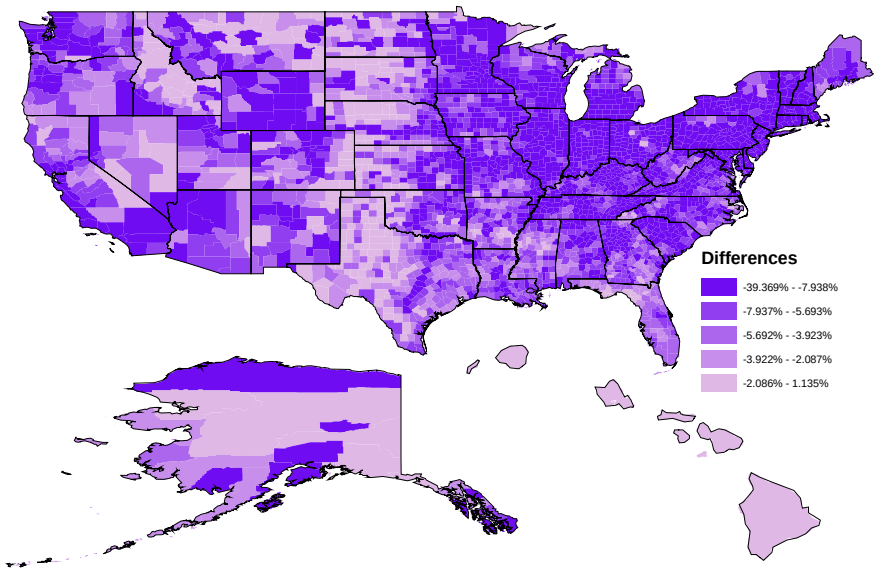
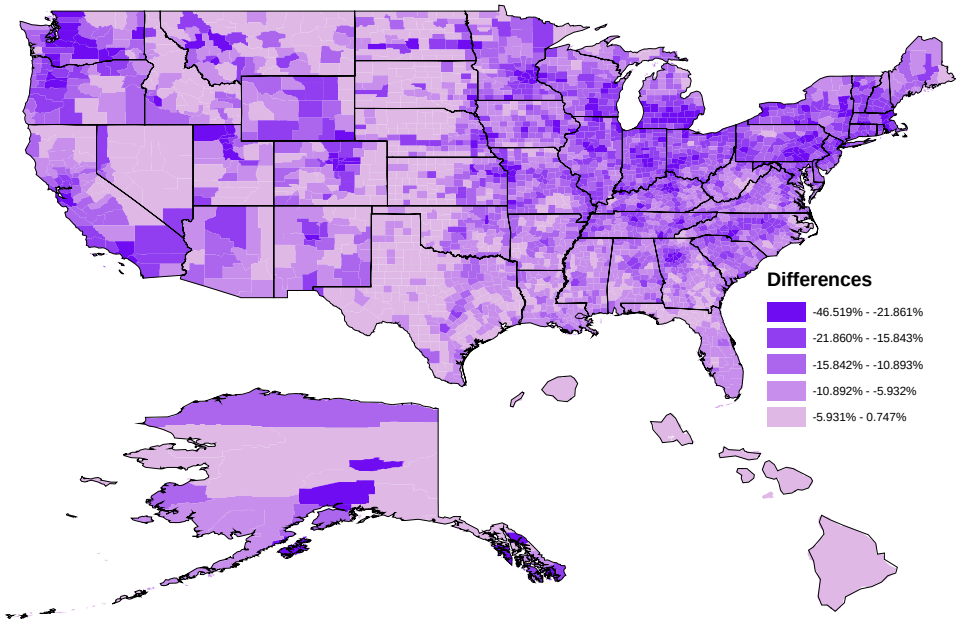


Figure 3.2-17. Distribution of Percent Reductions in Median Benzene Cancer Risk, 2030, for U.S. Counties With Controls in this Rule.



As a result of the controls being finalized in this rule, 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 11 million in 2020 and by about 17 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 about 30 million in 2020 and 46 million in 2030 (Table 3.2-18).

Table 3.2.-17. Reductions in Hazard Quotients and Hazard Indices for Acrolein and Respiratory Effects Due to MSAT Controls.

2015 Average Hazard Index/Quotient				2020 Average Hazard Index/Quotient					2030 Average Hazard Index/Quotient				
area & other	total onroad	total nonroad	total (including background)	major	area & other	total onroad	total nonroad	total (including background)	major	area & other	total onroad	total nonroad	total (including background)
1.17	1.63	0.84	3.92	0.19	1.14	1.66	0.88	3.99	0.19	1.13	1.92	0.99	4.35
1.17	1.35	0.84	3.65	0.19	1.14	1.24	0.88	3.56	0.19	1.13	1.24	0.99	3.67
0.0	16.7	0.0	6.9	0.0	0.0	25.6	0.0	10.7	0.0	0.0	35.4	0.0	15.6
1.13	1.54	0.81	3.63	0.18	1.09	1.57	0.85	3.69	0.18	1.08	1.82	0.96	4.04

Table 3.2-18. 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 Controls in this Rule.

Year	Benzene	All Mobile Source Air Toxics
2015	21,697,000	8,149,000
2020	30,031,000	11,257,000
2030	46,360,000	16,737,000

The standards being finalized will also impact on the number of people above various respiratory hazard index levels (Table 3.2-19).

Table 3.2-19. Decrease in Number of People with Inhalation Exposure above a Respiratory Hazard Index of One due to Inhalation Exposure from Ambient Sources, With Controls in this Rule.

Year	Decrease in Population with Respiratory HI > 1
2015	5,639,000
2020	10,227,000
2030	16,919,000

3.2.1.3 Strengths and Limitations

Air quality, exposure, and risk were assessed using the best available suite of tools for national-scale analysis of air toxics. The same general suite of tools was used in 1996 and 1999 NATA. The 1996 NATA 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.[161]
- 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.

In addition to the improvements for the 1999 NATA, improvements were made in analyses for this rule, including inventory improvements and updates to HAPEM discussed earlier.

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 in this rule 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 are 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. Uncertainties in dose-response parameters are discussed in Chapter 1 of the RIA. The modeling also has certain key limitations: results are most accurate for large geographic areas, 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.[162] If warranted, 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 3.2-20).

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

Table 3.2-20. 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. Assumptions about future-year meteorology introduce additional uncertainty in ambient concentrations and resulting exposures. Another limitation is the use of 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. For the proposed rule 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 proposed rule 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 3.2-21 compares national average total concentrations from the

proposed rule using 1999 versus scaled backgrounds. More details are provided in the technical support document for the proposed rule.[163]

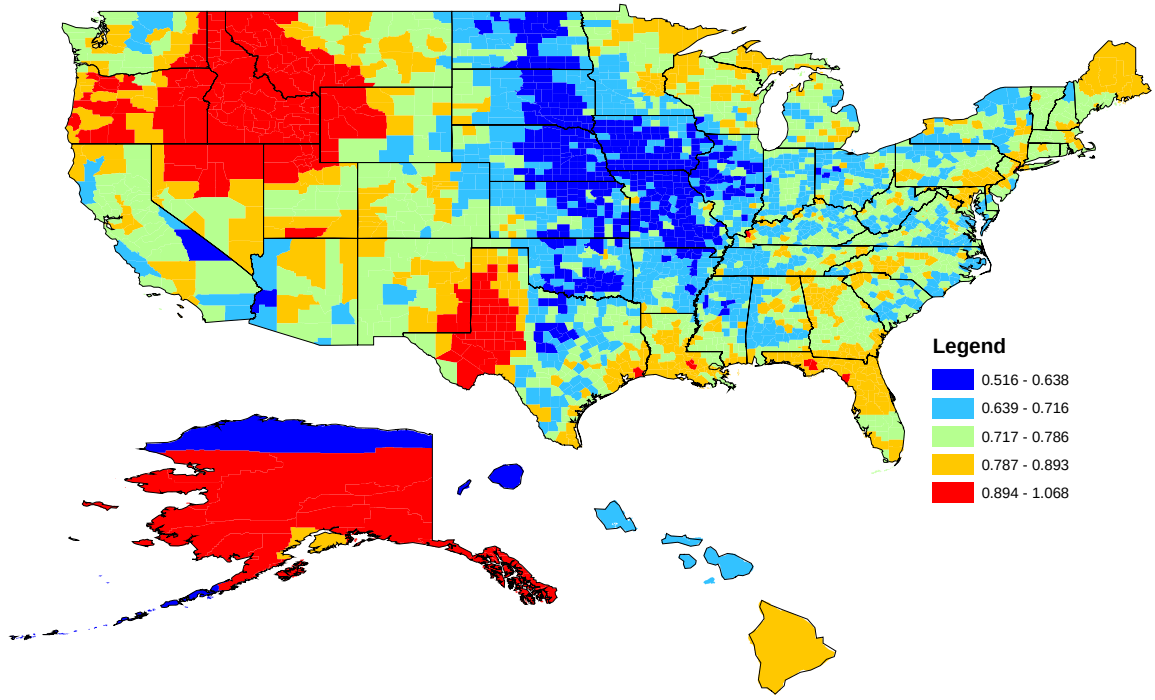
Table 3.2-21. National Average Total Concentrations (All Sources and Background) for 2015, 2020, and 2030 using both the 1999 Background and the Scaled Backgrounds (Data from Proposed Rule).

	Total Concentrations (μg m^{-3}) using 1999 Background			Total Concentrations (μg m^{-3}) using Scaled Concentrations		
HAP	**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 3.2-19, 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 equipment and fuels in attached garages and increased risks from early lifetime exposures, estimated risks and risk reductions from fuel benzene control would be larger.

Figure 3.2-19. Ratios of Benzene Concentrations with and without an Adjusted Background, 2020 (from modeling done to support proposed rule).



3.2.1.4. Perspective on Cancer Cases

We have not quantified the cancer-related health benefits of expected MSAT reductions in terms of avoided cancer cases or dollars. The EPA Science Advisory Board (SAB) specifically commented in their review of the 1996 National Air Toxics Assessment (NATA) that these tools were not yet ready for use in a national-scale benefits analysis, because they did not consider the full distribution of exposure and risk, or address sub-chronic health effects.[164] While EPA has since improved many of these tools, there remain critical limitations for estimating cancer incidence. For the MSATs of greatest concern, for example, we are currently unable to estimate cessation lag, which is the time between reduction in exposure and decline in risk to "steady state level."[165] We have also not resolved the analytical challenges associated with quantifying partial lifetime probabilities of cancer for different age groups or estimating changes in survival rates over time. Indeed, some of these issues are likely to remain highly uncertain for the foreseeable future.

We can, however, present some perspective on how average individual risks could translate into cumulative excess cancer cases across the U.S. population over a lifetime, assuming continuous exposure at a given level for 70 years. Cancer cases were estimated by summing the distribution of individual cancer risks from the national-scale modeling done to support this rule.

To estimate annual incidence, this would be divided by 70. However, without knowing when within a lifetime cancer is more likely to occur, and without accounting for time-varying

exposure, any estimate of incidence for a given calendar year is highly uncertain. We also note that a proper calculation would entail the use of a life table of incidence rates within discrete age ranges and a dose-response formulation expressing rate ratios as a function of benzene inhalation exposure concentration.

In 2030, the cumulative excess average individual cancer risk from outdoor emissions of mobile source air toxics is estimated at 2.1×10^{-5}. If the entire U. S. population (projected to be about 364 million)[166] were exposed to this level of risk over a 70-year lifetime, it would result in about 7700 cancer cases, which translates into 110 annual cancer cases.

In its review of the 1996 NATA, SAB recommended that if cancer cases were calculated for benefits assessment, a "best estimate" of risk (rather than an upper bound), should be used. We believe that the maximum likelihood unit risk range for benzene represents a best estimate. In our analyses, we have used the upper end of this range, as did the 1999 NATA. If we used the lower end of this range, incidence estimates would be lower by a factor of about 3.5. Following is a discussion related to benzene specifically, including a discussion of the potential implications of the limitations of our national-scale modeling, which were noted in Section 3.2.1.4.

In 2030, the national average inhalation individual cancer risk from outdoor mobile and stationary sources of benzene, in the absence of the standards being finalized in this rule, is estimated at approximately 9.1×10^{-6}, based on the modeling done for this rule. If the entire U. S. population were exposed to that level of risk over a 70-year lifetime, it would result in approximately 47 excess cancer cases per year (Equation 1).

(1) *Excess Cancer Cases at* 2030 *Exposure Level* =

$$(\textit{Average Individual Cancer Risk}) \times (2030\ \textit{Population})$$

$$= 9.1 \times 10^{-6} \times 3.64 \times 10^{8} \approx 3300$$

$$\textit{Annual Cancer Cases} = 3300 / 70 = 47$$

As discussed in Section 3.1.3.3, EPA's estimate of risk due to exposure to benzene could increase significantly if the influence of attached garages were included. When the exposures for people with attached garages are averaged across the population, time-weighted average individual exposures to benzene could increase by roughly 1.2 to 6.6 $\mu g/m^3$ (Appendix 3A). There is a great deal of uncertainty associated with these estimates. This could result in about another 3400 to 18700 excess cancer cases (equation 3). The numerical ranges expressed here may not fully address all sources of uncertainty involved in making these projections.

(3) *Attached Garage Excess Cancer Cases* =

$$(\textit{Average Exposure})\ x\ (\textit{Benzene URE}) \times (\textit{Population})$$

$$= (1.2 - 6.6\ \mu g / m^3)\ x\ (7.8 x 10^{-6} / \mu g / m^3)\ x\ (3.64\ x\ 10^{8}) = 3400 - 18700$$

$$\textit{Annual Cancer Cases} = 49 - 268$$

Thus, including attached garages would increase the number of benzene-related excess cancer cases to somewhere between 96 and 315 annually. This estimate would still not include higher exposure levels from occupational exposures, vapor emissions from leaking underground storage tanks, or other accidental releases into the environment. Any population risk characterization that does not account for these factors underestimates the excess cancer related to benzene.

With the controls being finalized in this rule, average individual risk, not including attached garage exposures, is reduced to 7.3×10^{-6}, which results in approximately 37 cancer cases per year. Thus, excess leukemia cases would be reduced by 10 annually. A roughly 40% reduction in overall benzene emissions could reduce attached garage exposures by approximately 0.5-2.6 $\mu g/m^3$ as well, thus reducing excess annual cancer cases from this source of exposure by another estimated 20 to 100 excess cancer cases. Thus, this rule would prevent roughly 30 to 110 benzene-related excess cancer cases annually, assuming continuous lifetime exposure to 2030 levels, given the assumptions of population size and lifetime above, and not including excess leukemia from occupational exposure or from leaking underground storage tanks. Emission reductions in 2030 would reduce cancer cases not just in 2030, but also well beyond this period. There would also be further unquantified reductions in incidence due to the other air toxics reductions.

Such estimates should be interpreted with extreme caution since they could imply an artificial sense of precision. Serious limitations include:

- As discussed in Chapter 1, the current unit risk estimate for benzene may underestimate risk from leukemia, because some recent epidemiology data, including key studies published after the most recent IRIS assessment, suggest a supralinear rather than linear dose-response at low doses. However, the studies published after the most recent IRIS assessment have not yet been formally evaluated by EPA as part of the IRIS review process, and it is not clear whether these data provide sufficient evidence to reject a linear dose-response curve. A better understanding of the biological mechanism of benzene-induced leukemia is needed.
- Geographically heterogeneous percentage emissions reductions do not translate directly into changes in ambient levels, exposure, and risk.
- The U.S. population would have experienced higher average exposures in previous years, but this is not accounted for.
- The extent to which available studies of indoor air homes in with attached garage are representative of the national housing stock is unknown.
- Cessation lag between reduction in exposure and reduction in risk is not accounted for.
- Differences in risk among various age groups are not known, and the age structure of the U.S. population is expected to change over time.

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.[167] A case study of diesel exhaust particulate matter in Wilmington, CA was recently conducting employing some of these advances.[168] 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.

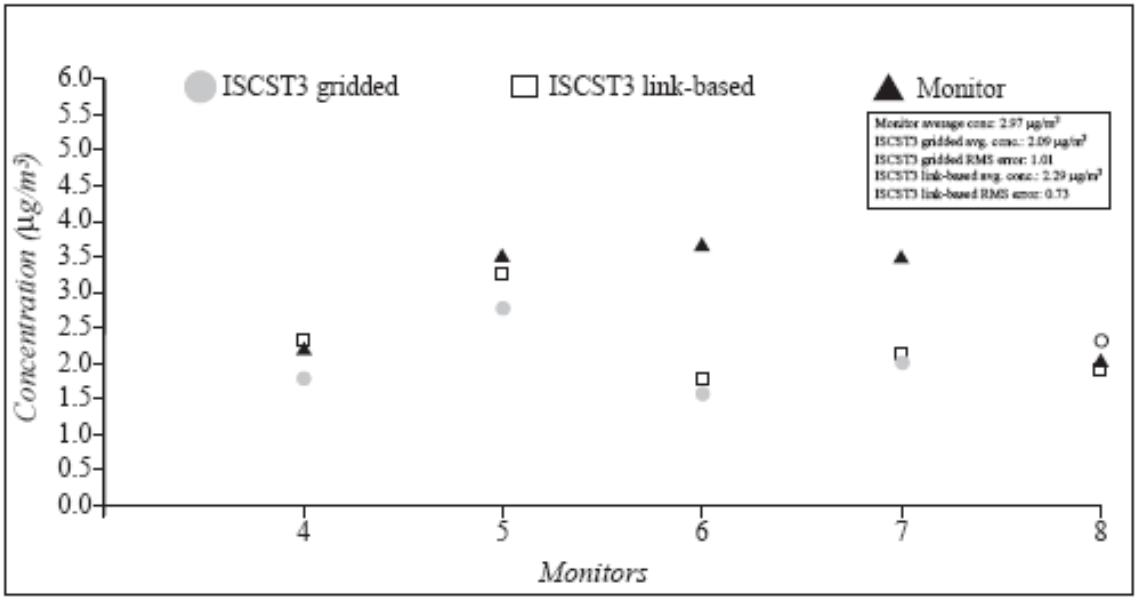
The Wilmington study discussed above also allocated motor vehicle emissions to individual road "links," rather than using spatial surrogates to allocate county level vehicle emissions to grid cells. In using spatial surrogates to allocate emissions, high local concentrations may not be captured for environments 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.[169] 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.

As discussed in Section 3.1.3.2, local-scale modeling in Houston assigned emissions to

individual road links. [170] 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. 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 3.2-20. 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.

Recent air quality modeling in Portland, OR using the CALPUFF dispersion model assigned emissions to specific roadway links.[171] 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 $\mu g/m^3$, 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 $\mu g/m^3$, with concentrations increasing to 1.29 $\mu g/m^3$ 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.

Figure 3.2-20. Model to Monitor Comparisons of Houston Benzene Concentrations



A recent review of local-scale modeling studies concluded that:[172]

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.[173] 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 the Philadelphia area differed significantly between the top-down and the bottom-up methodologies as shown in Table 3.2-22.

Table 3.2-22. 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 County, using local registration distribution data resulted in significantly lower air toxics emission factors and resultant emissions, while Montgomery County showed higher emissions. In the 1999 National Air Toxics Assessment, higher county-level emissions were generally associated with higher county-level average concentrations, so it is anticipated that county-level concentrations will follow similar trends. However, in microscale settings near specific road links, these results may not apply.

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 final 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 pollution 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.

The science of ozone formation, transport, and accumulation is complex.[174] Ground-level ozone is produced and destroyed in a cyclical set of chemical reactions, many of which are sensitive to temperature and sunlight. When ambient temperatures and sunlight levels remain high for several days and the air is relatively stagnant, ozone and its precursors can build up and result in more ozone than typically would occur on a single high-temperature day. Ozone also can be transported into an area from pollution sources found hundreds of miles upwind, resulting in elevated ozone levels even in areas with low VOC or NO_x emissions.

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 (NO_2); as the air moves downwind and the cycle continues, the NO_2 forms additional ozone. The importance of this reaction depends, in part, on the relative concentrations of 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.[e] These health effects are well documented and are critically assessed in the EPA ozone Air Quality Criteria Document (ozone AQCD) and EPA staff paper.[175,176] We are relying on the data and conclusions in the ozone AQCD and staff paper, regarding the health effects associated with ozone exposure.

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

Based on a large number of scientific studies, EPA has identified several key health effects associated with exposure to levels of ozone found today in many areas of the country. Short-term (1 to 3 hours) and prolonged exposures (6 to 8 hours) to higher ambient ozone concentrations have been linked to lung function decrements, respiratory symptoms, increased hospital admissions and emergency room visits for respiratory problems.[177, 178, 179, 180, 181, 182] Repeated exposure to ozone can increase susceptibility to respiratory infection and lung inflammation and can aggravate preexisting respiratory diseases, such as asthma.[183, 184, 185, 186, 187] Repeated exposure to sufficient concentrations of ozone can also cause inflammation of the lung, impairment of lung defense mechanisms, and possibly irreversible changes in lung structure, which over time could lead to premature aging of the lungs and/or chronic respiratory illnesses, such as emphysema and chronic bronchitis.[188, 189, 190, 191]

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.[192] Children and outdoor workers tend to have higher ozone exposures because they typically are active outside, working, playing and exercising, during times of day and seasons (e.g., the summer) when ozone levels are highest.[193] For example, summer camp studies in the Eastern United States and Southeastern Canada have reported significant reductions in lung

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

function in children who are active outdoors.[194, 195, 196, 197, 198, 199, 200, 201] Further, children are more at risk of experiencing health effects from ozone exposure than adults because their respiratory systems are still developing. These individuals (as well as people with respiratory illnesses such as asthma, especially asthmatic children) can experience reduced lung function and increased respiratory symptoms, such as chest pain and cough, when exposed to relatively low ozone levels during prolonged periods of moderate exertion.[202, 203, 204, 205]

3.3.3 Current 8-Hour Ozone Levels

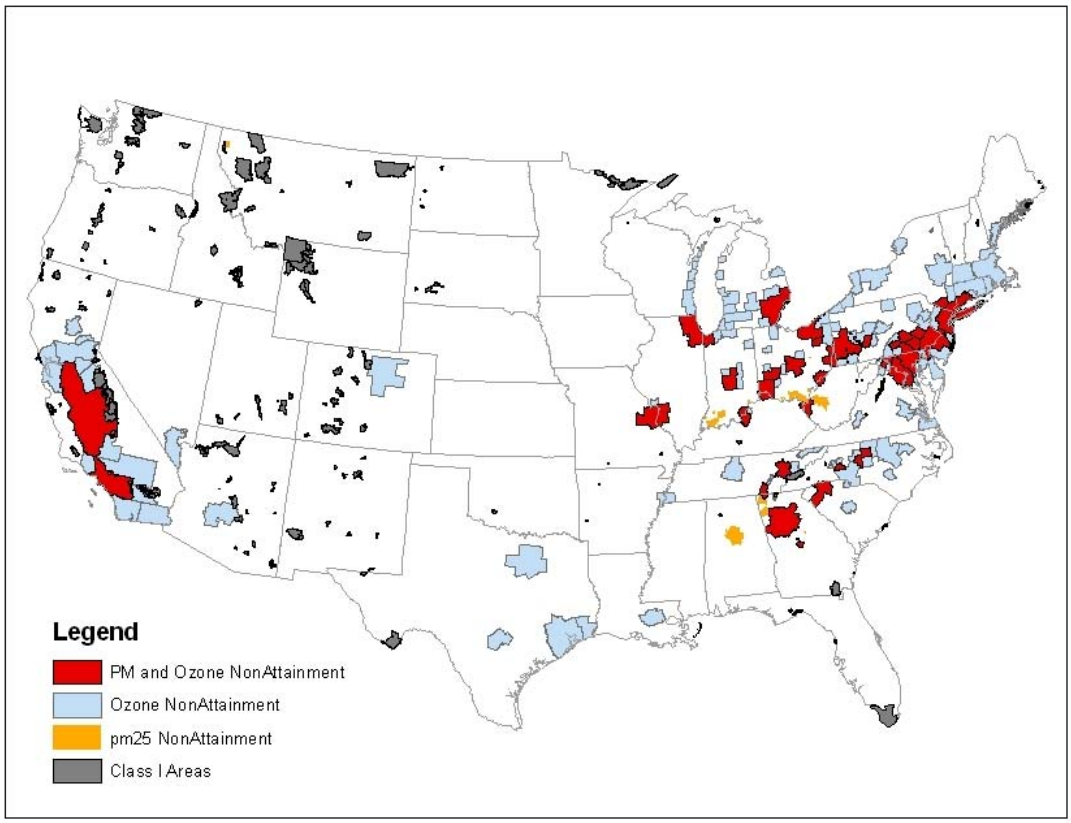
The gas can emission reductions will assist 8-hour ozone nonattainment areas in reaching the standard by each area's respective attainment date and assist 8-hour ozone maintenance areas in maintaining the 8-hour ozone standard in the future. In this section and the next section we present information 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.[f]

As of October 26, 2006, approximately 157 million people live in the 116 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 461 full or partial counties that make up the 116 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 3B to this RIA.

[f] An ozone design value is the concentration that determines whether the ozone levels recorded at a monitoring site meet the NAAQS for ozone. The level of a design value is 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. Greater detail on how these values are determined (including how to account for missing values and other complexities) is given in Appendices H and I of 40 CFR Part 50. Due to the precision with which the standards are expressed (0.08 ppm for the 8-hour NAAQS value), a violation of the 8-hour standard is defined as any design value greater than or equal to 0.085 ppm, or 85 ppb. For any particular county, the design value is the highest design value from amongst all the monitors having valid design values within that county. If there are no ozone monitors located in a particular county, that county is not assigned a design value. However, readers should note that ozone design values represent air quality over a broad area and the absence of a design value for a specific county does not imply that that county is in compliance with the NAAQS for ozone**.** Therefore, our analysis may underestimate the number of counties with ozone levels, i.e., design values, which are above the level of the ozone NAAQS.

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 categorized, on the basis of their one-hour ozone design value, as Subpart 1 or Subpart 2 (69 FR 23951, April 30, 2004). Areas categorized as Subpart 2 were then further classified, on the basis of their 8-hour ozone design value, as marginal, moderate, serious, severe or extreme. The maximum attainment date assigned to an ozone nonattainment area is based on the area's classification.

Table 3B-1 presents the 8-hour ozone nonattainment areas, their 8-hour design values, and their category or classification. States with 8-hour ozone nonattainment areas are required to take action to bring those areas into compliance prior to the ozone season in the attainment year. Based on the final rule designating and classifying 8-hour ozone nonattainment areas, most 8-hour ozone nonattainment areas will be required to attain the 8-hour ozone NAAQS in the 2007 to 2013 time frame and then be required to maintain the 8-hour ozone NAAQS thereafter.[g] The gas can emission standards being finalized in this action will become effective in 2009. Thus,

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

the expected ozone precursor emission inventory reductions from the standards finalized in this action will be useful to States in attaining and/or maintaining the 8-hour ozone NAAQS.

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

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 some areas in the future. In the following sections we describe recent ozone air quality modeling from the CAIR 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 for gas cans.

3.3.4.1 CAIR Ozone Air Quality Modeling

Recently ozone air quality analyses were performed for the Clean Air Interstate Rule (CAIR), which was promulgated by EPA in 2005. The Comprehensive Air Quality Model with Extension (CAMx) was used as the tool for simulating base and future year concentrations of ozone in support of the CAIR ozone air quality assessment. The CAIR analysis included all final federal rules up to and including CAIR controls. Details on the air quality modeling are provided in the Air Quality Modeling Technical Support Document for the Final Clean Air Interstate Rule, included in the docket for this final rule.[206]

Air quality modeling performed for CAIR 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 analysis provided estimates of future ozone levels across the country. For example, in 2010, in the absence of controls beyond those relied on for the CAIR modeling, we project that 24 million people would live in 37 Eastern counties with 8-hour ozone concentrations at and above 85 ppb, see Table 3.3-1.[h] Table 3.3-1 also lists the 148 Eastern counties, where 61 million people are projected to live, with 2010 projected design values that do not violate the 8-hour ozone NAAQS but are within ten percent of it, in the absence of emission reductions beyond those considered in the CAIR modeling. These are counties that are not projected to violate the standard, but to be close to it. The rule may help ensure that these counties continue to maintain their attainment status and the emission reductions from this final rule will be included by the states in their baseline inventory modeling for their ozone maintenance plans.

[h] 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 ozone standard by their statutory date.

Table 3.3-1. Eastern Counties with 2010 projected 8-hour Ozone Concentrations Above and within 10% of the 8-hour Ozone Standard

State	County	2010 Projected 8-hour Ozone Concentration (ppb)[a]	2000 pop[b]	2010 pop[c]
Arkansas	Crittenden Co	80.8	50,866	52,889
Connecticut	Fairfield Co	**92.2**	882,567	891,694
Connecticut	Hartford Co	80.1	857,183	859,080
Connecticut	Middlesex Co	**90.6**	155,071	164,202
Connecticut	New Haven Co	**91.3**	824,008	829,181
Connecticut	New London Co	83.4	259,088	267,199
Connecticut	Tolland Co	82.7	136,364	142,988
D.C.	Washington Co	**85.0**	572,058	554,474
Delaware	Kent Co	78.7	126,697	139,376
Delaware	New Castle Co	84.7	500,264	534,631
Delaware	Sussex Co	80.3	156,638	181,962
Georgia	Bibb Co	80.0	153,887	158,291
Georgia	Cobb Co	79.4	607,750	744,488
Georgia	Coweta Co	76.6	89,215	111,522
Georgia	De Kalb Co	81.9	665,864	698,335
Georgia	Douglas Co	78.7	92,174	114,380
Georgia	Fayette Co	76.7	91,263	117,580
Georgia	Fulton Co	**85.1**	816,005	855,826
Georgia	Henry Co	80.3	119,341	153,957
Georgia	Rockdale Co	80.4	70,111	87,977
Illinois	Cook Co	81.8	5,376,739	5,363,464
Illinois	Jersey Co	77.0	21,668	22,905
Illinois	Lake Co	76.8	644,356	731,690
Illinois	McHenry Co	76.6	260,077	307,400
Indiana	Boone Co	78.1	46,107	54,035
Indiana	Clark Co	78.4	96,472	107,096
Indiana	Hamilton Co	81.7	182,740	230,565
Indiana	Hancock Co	80.4	55,391	65,282
Indiana	La Porte Co	81.8	110,106	111,566
Indiana	Lake Co	82.8	484,563	489,220
Indiana	Madison Co	78.6	133,358	137,710
Indiana	Marion Co	79.6	860,453	879,932
Indiana	Porter Co	81.1	146,798	165,350
Indiana	Shelby Co	81.6	43,445	46,565
Indiana	St Joseph Co	77.8	265,559	275,031
Kentucky	Campbell Co	81.5	88,616	92,109
Louisiana	Bossier Parish	77.0	98,310	110,838
Louisiana	East Baton Rouge Parish	80.6	412,852	465,411
Louisiana	Iberville Parish	79.4	33,320	33,089
Louisiana	Jefferson Parish	78.6	455,466	493,359
Louisiana	Livingston Parish	77.8	91,814	124,895
Louisiana	West Baton Rouge Parish	78.8	21,601	22,672
Maine	Hancock Co	80.5	51,791	53,886

State	County	2010 Projected 8-hour Ozone Concentration (ppb)[a]	2000 pop[b]	2010 pop[c]
Maine	York Co	80.2	186,742	201,082
Maryland	Anne Arundel Co	**88.6**	489,656	543,785
Maryland	Baltimore Co	83.7	754,292	792,284
Maryland	Carroll Co	80.0	150,897	179,918
Maryland	Cecil Co	**89.5**	85,951	96,574
Maryland	Charles Co	78.7	120,546	145,763
Maryland	Frederick Co	78.1	195,277	234,304
Maryland	Harford Co	**92.8**	218,590	268,207
Maryland	Kent Co	**85.8**	19,197	20,233
Maryland	Montgomery Co	79.3	873,341	940,126
Maryland	Prince Georges Co	84.2	801,515	842,221
Massachusetts	Barnstable Co	83.6	222,230	249,495
Massachusetts	Bristol Co	83.0	534,678	558,460
Massachusetts	Essex Co	81.7	723,419	747,556
Massachusetts	Hampden Co	80.2	456,228	452,718
Massachusetts	Hampshire Co	78.0	152,251	158,130
Massachusetts	Middlesex Co	79.1	1,465,396	1,486,428
Massachusetts	Suffolk Co	78.1	689,807	674,179
Michigan	Allegan Co	82.1	105,665	121,415
Michigan	Benzie Co	77.9	15,998	17,849
Michigan	Berrien Co	78.1	162,453	164,727
Michigan	Cass Co	78.2	51,104	53,544
Michigan	Genesee Co	76.7	436,141	441,196
Michigan	Macomb Co	**85.4**	788,149	838,353
Michigan	Mason Co	78.9	28,274	30,667
Michigan	Muskegon Co	82.0	170,200	175,901
Michigan	Oakland Co	80.7	1,194,155	1,299,592
Michigan	Ottawa Co	76.6	238,314	277,400
Michigan	St Clair Co	80.6	164,235	178,391
Michigan	Washtenaw Co	81.0	322,895	344,398
Michigan	Wayne Co	84.7	2,061,161	1,964,209
Missouri	Clay Co	76.5	184,006	213,643
Missouri	Jefferson Co	76.7	198,099	230,539
Missouri	St Charles Co	80.5	283,883	341,686
Missouri	St Louis City	79.4	348,188	324,156
Missouri	St Louis Co	80.5	1,016,315	1,024,964
New Hampshire	Hillsborough Co	76.6	380,841	412,071
New Jersey	Atlantic Co	80.4	252,552	269,754
New Jersey	Bergen Co	**86.0**	884,118	898,450
New Jersey	Camden Co	**91.6**	508,932	509,912
New Jersey	Cumberland Co	84.4	146,438	149,595
New Jersey	Gloucester Co	**91.3**	254,673	278,612
New Jersey	Hudson Co	84.3	608,975	607,256
New Jersey	Hunterdon Co	**88.6**	121,989	139,641
New Jersey	Mercer Co	**95.2**	350,761	359,912
New Jersey	Middlesex Co	**92.1**	750,162	805,537

State	County	2010 Projected 8-hour Ozone Concentration (ppb)[a]	2000 pop[b]	2010 pop[c]
New Jersey	Monmouth Co	**86.4**	615,301	670,971
New Jersey	Morris Co	**85.5**	470,212	500,033
New Jersey	Ocean Co	**100.3**	510,916	572,364
New Jersey	Passaic Co	79.7	489,049	495,610
New York	Bronx Co	79.7	1,332,649	1,298,206
New York	Chautauqua Co	81.8	139,750	139,909
New York	Dutchess Co	81.0	280,150	291,098
New York	Erie Co	**86.9**	950,265	953,085
New York	Essex Co	77.6	38,851	39,545
New York	Jefferson Co	80.5	111,738	113,075
New York	Monroe Co	76.9	735,343	745,350
New York	Niagara Co	82.3	219,846	220,407
New York	Orange Co	77.1	341,367	371,434
New York	Putnam Co	82.3	95,745	107,967
New York	Queens Co	78.3	2,229,379	2,239,026
New York	Richmond Co	**87.1**	443,728	488,728
New York	Suffolk Co	**90.8**	1,419,369	1,472,127
New York	Westchester Co	84.7	923,459	944,535
North Carolina	Mecklenburg Co	81.4	695,453	814,088
North Carolina	Rowan Co	80.1	130,340	143,729
North Carolina	Wake Co	77.2	627,846	787,707
Ohio	Allen Co	76.8	108,473	106,900
Ohio	Ashtabula Co	83.5	102,728	104,850
Ohio	Butler Co	78.0	332,806	384,410
Ohio	Clermont Co	78.0	177,977	205,365
Ohio	Clinton Co	81.4	40,543	47,137
Ohio	Cuyahoga Co	77.3	1,393,977	1,348,313
Ohio	Delaware Co	77.3	109,989	136,125
Ohio	Franklin Co	81.9	1,068,977	1,142,894
Ohio	Geauga Co	**86.6**	90,895	102,083
Ohio	Hamilton Co	78.6	845,302	843,226
Ohio	Knox Co	76.5	54,500	59,435
Ohio	Lake Co	82.2	227,511	237,161
Ohio	Lorain Co	78.5	284,664	292,040
Ohio	Lucas Co	80.0	455,053	447,302
Ohio	Medina Co	76.5	151,095	173,985
Ohio	Portage Co	79.8	152,061	162,685
Ohio	Summit Co	82.4	542,898	552,567
Ohio	Trumbull Co	79.7	225,116	226,157
Ohio	Warren Co	80.0	158,383	186,219
Ohio	Wood Co	77.4	121,065	129,124
Oklahoma	Tulsa Co	79.2	563,299	610,536
Pennsylvania	Allegheny Co	81.9	1,281,665	1,259,040
Pennsylvania	Armstrong Co	79.7	72,392	72,829
Pennsylvania	Beaver Co	79.6	181,412	183,693
Pennsylvania	Berks Co	81.7	373,637	388,194

State	County	2010 Projected 8-hour Ozone Concentration (ppb)[a]	2000 pop[b]	2010 pop[c]
Pennsylvania	Bucks Co	**94.3**	597,635	648,796
Pennsylvania	Cambria Co	76.9	152,598	146,811
Pennsylvania	Chester Co	**85.4**	433,501	478,460
Pennsylvania	Dauphin Co	80.8	251,798	265,019
Pennsylvania	Delaware Co	84.0	550,863	543,169
Pennsylvania	Erie Co	79.1	280,843	284,835
Pennsylvania	Franklin Co	80.2	129,313	135,088
Pennsylvania	Lancaster Co	83.6	470,657	513,684
Pennsylvania	Lehigh Co	82.1	312,090	323,215
Pennsylvania	Mercer Co	78.1	120,293	122,546
Pennsylvania	Montgomery Co	**87.6**	750,097	772,849
Pennsylvania	Northampton Co	81.8	267,066	279,797
Pennsylvania	Philadelphia Co	**89.9**	1,517,549	1,420,803
Pennsylvania	Washington Co	77.3	202,897	205,153
Pennsylvania	Westmoreland Co	76.7	369,993	372,941
Pennsylvania	York Co	79.4	381,750	404,807
Rhode Island	Kent Co	**86.2**	167,090	174,126
Rhode Island	Providence Co	81.2	621,602	621,355
Rhode Island	Washington Co	84.2	123,546	137,756
South Carolina	Richland Co	76.9	320,677	349,826
Tennessee	Sevier Co	76.5	71,170	96,097
Tennessee	Shelby Co	76.7	897,471	958,501
Texas	Brazoria Co	84.1	241,767	281,960
Texas	Collin Co	82.5	491,675	677,868
Texas	Dallas Co	82.2	2,218,899	2,382,657
Texas	Denton Co	**86.8**	432,976	554,033
Texas	Galveston Co	84.6	250,158	283,963
Texas	Gregg Co	79.1	111,379	121,241
Texas	Harris Co	**97.4**	3,400,577	3,770,129
Texas	Jefferson Co	**85.0**	252,051	260,847
Texas	Johnson Co	78.2	126,811	157,545
Texas	Montgomery Co	81.2	293,768	413,048
Texas	Tarrant Co	**87.2**	1,446,219	1,710,920
Virginia	Alexandria City	80.9	128,283	130,422
Virginia	Arlington Co	**86.0**	189,453	193,370
Virginia	Charles City Co	77.7	6,926	7,382
Virginia	Fairfax Co	**85.4**	969,749	1,085,483
Virginia	Hampton City	78.7	146,437	153,246
Virginia	Hanover Co	80.9	86,320	98,586
Virginia	Henrico Co	78.2	262,300	294,174
Virginia	Loudoun Co	78.6	169,599	214,469
Virginia	Suffolk City	77.5	63,677	69,003
Wisconsin	Door Co	82.1	27,961	30,508
Wisconsin	Kenosha Co	**91.0**	149,577	166,359

State	County	2010 Projected 8-hour Ozone Concentration (ppb)[a]	2000 pop[b]	2010 pop[c]
Wisconsin	Kewaunee Co	79.9	20,187	20,538
Wisconsin	Manitowoc Co	80.0	82,887	83,516
Wisconsin	Milwaukee Co	82.1	940,164	922,943
Wisconsin	Ozaukee Co	**85.8**	82,317	95,549
Wisconsin	Racine Co	83.9	188,831	199,178
Wisconsin	Sheboygan Co	**87.7**	112,646	118,866
Number of Violating Counties		37		
Population of Violating Counties			22,724,010	24,264,574
Number of Counties within 10%		148		
Population of Counties within 10%			58,453,962	61,409,062

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

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 rule.[207]

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.[208] 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.

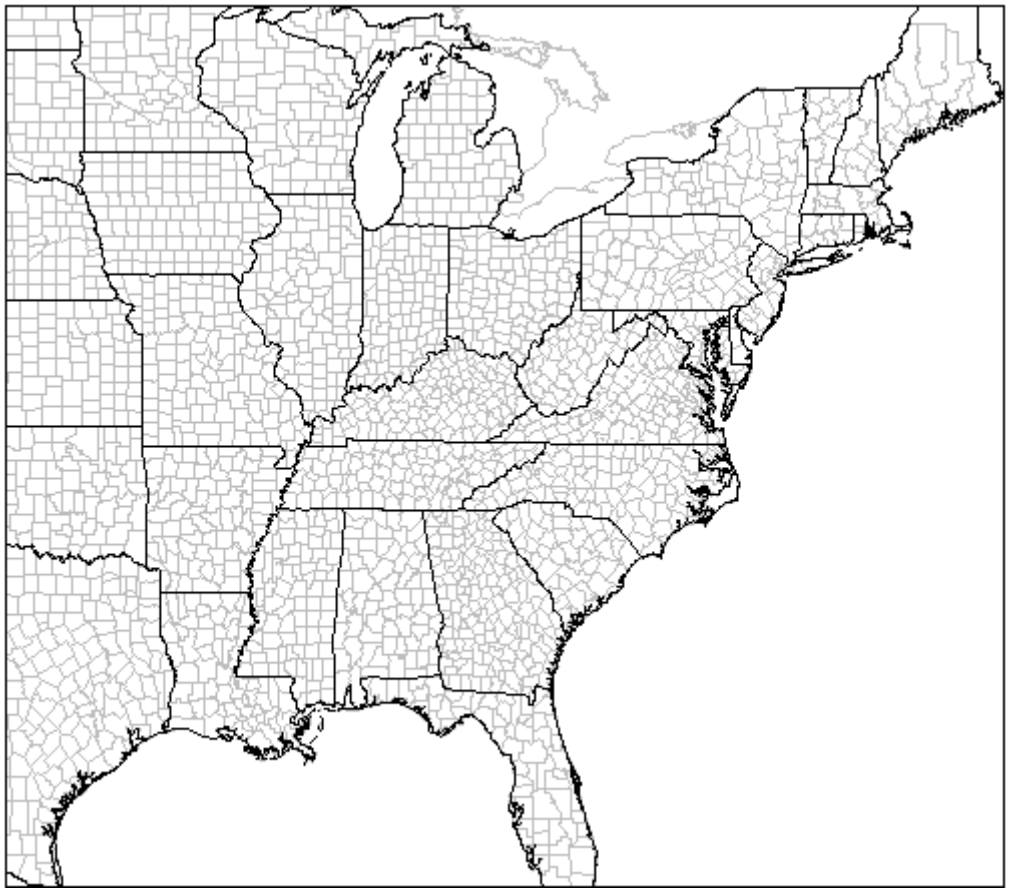
The ozone RSM uses the 2015 controlled CAIR emissions inventory as its baseline.[209] This inventory does not include the gas can emissions that are being controlled in this rule. The uncontrolled and controlled gas can emissions have been incorporated into the base and control runs of the ozone RSM (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. Finally, the inventory includes an error in mobile source NOx for 13 Northeastern states. The impact of this error is minimized as the model is used in a relative way. 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 final rule.[210]

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.[211] 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 gas can controls. Section 2.1.1.2 also details the states that have their own gas can control programs and how the controls finalized 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 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 (NOx 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 154 runs (a base case plus 139 control runs plus 10 evaluation runs plus 4 boundary condition 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 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 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 reductions in gas can emissions. Based upon our previous CAIR 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 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.[i] Nonetheless, there are some areas where the ozone improvement is more significant. These areas include Chicago, Milwaukee, Detroit and New York City. 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 have the highest ozone design values. Those counties that are projected to experience an extremely small increase in ozone design values generally have design values that are lower, below 70 ppb. The results from the metamodeling projections indicate a net overall improvement in future 8-hour

[i] Appendix I of 40 CFR Part 50.

ozone design values due to the gas can controls, when weighted by population. The AQMTSD, contained in the docket for this final rule, 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.[212] 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 ozone AQCD notes that "ozone affects vegetation throughout the United States, impairing crops, native vegetation, and ecosystems more than any other air pollutant."[213] Like carbon dioxide (CO_2) and other gaseous substances, ozone enters plant tissues primarily through apertures (stomata) in leaves in a process called "uptake." To a lesser extent, ozone can also diffuse directly through surface layers to the plant's interior.[214] Once sufficient levels of ozone, a highly reactive substance, (or its reaction products) reaches the interior of plant cells, it can inhibit or damage essential cellular components and functions, including enzyme activities, lipids, and cellular membranes, disrupting the plant's osmotic (i.e., water) balance and energy utilization patterns.[215,216] 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.[217] With fewer resources available, the plant reallocates existing resources away from root growth and storage, above ground growth or yield, and reproductive processes, toward leaf repair and maintenance. Studies have shown that plants stressed in these ways may exhibit a general loss of vigor, which can lead to secondary impacts that modify plants' responses to other environmental factors. Specifically, plants may become more sensitive to other air pollutants, more susceptible to disease, insect attack, harsh weather (e.g., drought, frost) and other environmental stresses. Furthermore, there is some evidence that ozone can interfere with the formation of mycorrhiza, essential symbiotic fungi associated with the roots of most terrestrial plants, by reducing the amount of carbon available for transfer from the host to the symbiont.[218]

Ozone can produce both acute and chronic injury in sensitive species depending on the concentration level and the duration of the exposure. Ozone effects also tend to accumulate over the growing season of the plant, so that even lower concentrations experienced for a longer duration have the potential to create chronic stress on sensitive vegetation. Not all plants, however, are equally sensitive to ozone. Much of the variation in sensitivity between individual plants or whole species is related to the plant's ability to regulate the extent of gas exchange via leaf stomata (e.g., avoidance of O_3 uptake through closure of stomata).[219,220,221] 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.[222] Because of the differing sensitivities among plants to ozone, ozone pollution can also exert a selective pressure that leads to changes in plant community composition. Given the range of plant sensitivities and the fact that

numerous other environmental factors modify plant uptake and response to ozone, it is not possible to identify threshold values above which ozone is consistently toxic for all plants. The next few paragraphs present additional information on ozone damage to trees, ecosystems, agronomic crops and urban ornamentals.

Ozone also has been shown conclusively to cause discernible injury to forest trees.[223,224] In terms of forest productivity and ecosystem diversity, ozone may be the pollutant with the greatest potential for regional-scale forest impacts.[225] Studies have demonstrated repeatedly that ozone concentrations commonly observed in polluted areas can have substantial impacts on plant function.[226,227]

Because plants are at the center of the food web in many ecosystems, changes to the plant community can affect associated organisms and ecosystems (including the suitability of habitats that support threatened or endangered species and below ground organisms living in the root zone). Ozone impacts at the community and ecosystem level vary widely depending upon numerous factors, including concentration and temporal variation of tropospheric ozone, species composition, soil properties and climatic factors.[228] In most instances, responses to chronic or recurrent exposure in forested ecosystems are subtle and not observable for many years. These injuries can cause stand-level forest decline in sensitive ecosystems.[229,230,231] It is not yet possible to predict ecosystem responses to ozone with much certainty; however, considerable knowledge of potential ecosystem responses has been acquired through long-term observations in highly damaged forests in the United States.

Laboratory and field experiments have also shown reductions in yields for agronomic crops exposed to ozone, including vegetables (e.g., lettuce) and field crops (e.g., cotton and wheat). The most extensive field experiments, conducted under the National Crop Loss Assessment Network (NCLAN) examined 15 species and numerous cultivars. The NCLAN results show that "several economically important crop species are sensitive to ozone levels typical of those found in the Unites States."[232] In addition, economic studies have shown reduced economic benefits as a result of predicted reductions in crop yields associated with observed ozone levels.[233,234,235]

Urban ornamentals represent an additional vegetation category likely to experience some degree of negative effects associated with exposure to ambient ozone levels and likely to impact large economic sectors. It is estimated that more than $20 billion (1990 dollars) are spent annually on landscaping using ornamentals, both by private property owners/tenants and by governmental units responsible for public areas.[236] This is therefore a potentially costly environmental effect. However, in the absence of adequate exposure-response functions and economic damage functions for the potential range of effects relevant to these types of vegetation, no direct quantitative analysis has been conducted. Methods are not available to allow for plausible estimates of the percentage of these expenditures that may be related to impacts associated with ozone exposure.

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 levels of ambient PM above the NAAQS. Emissions of PM and VOC from the vehicles subject to this 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 generally less than or equal to 10 micrometers (μm) in diameter. $PM_{2.5}$ refers to fine particles, those particles generally less than or equal to 2.5 μm in diameter. Inhalable (or "thoracic") coarse particles refer to those particles generally greater than 2.5 μm but less than or equal to 10 μm in diameter. Ultrafine PM refers to particles with diameters generally less than 100 nanometers (0.1 μm). Larger particles (>10 μm) tend to be removed by the respiratory clearance mechanisms, whereas smaller particles are deposited deeper in the lungs.

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

The vehicles that will be covered by the 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 will be covered by the 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 recently amended the PM NAAQS (71 FR 61144, October 17, 2006). The final rule, signed on September 21, 2006 and published on October 17, 2006, addressed revisions to the primary and secondary NAAQS for PM to provide increased protection of public health and welfare, respectively. The primary $PM_{2.5}$ NAAQS include a short-term (24-hour) and a long-term (annual) standard. The level of the 24-hour $PM_{2.5}$ NAAQS has been revised from 65μg/m^3 to 35 μg/m^3 to provide increased protection against health effects associated with short-term

exposures to fine particles. The current form of the 24-hour $PM_{2.5}$ standard was retained (e.g., based on the 98th percentile concentration averaged over three years). The level of the annual $PM_{2.5}$ NAAQS was retained at 15$\mu g/m^3$, continuing protection against health effects associated with long-term exposures. The current form of the annual $PM_{2.5}$ standard was retained as an annual arithmetic mean averaged over three years, however, the following two aspects of the spatial averaging criteria were narrowed: (1) the annual mean concentration at each site shall be within 10 percent of the spatially averaged annual mean, and (2) the daily values for each monitoring site pair shall yield a correlation coefficient of at least 0.9 for each calendar quarter. With regard to the primary PM_{10} standards, the 24-hour PM_{10} NAAQS was retained at a level of 150 $\mu g/m^3$ not to be exceeded more than once per year on average over a three-year period. Given that the available evidence does not suggest an association between long-term exposure to coarse particles at current ambient levels and health effects, EPA has revoked the annual PM_{10} standard.

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

3.4.2 Health Effects of Particulate Matter

As stated in the EPA Particulate Matter Air Quality Criteria Document (PMAQCD), available scientific findings "demonstrate well that human health outcomes are associated with ambient PM."[j] We are relying primarily on the data and conclusions in the PM AQCD and PM staff paper, which reflects EPA's analysis of policy-relevant science from the PM AQCD, regarding the health effects associated with particulate matter.[237,238] We also present additional recent studies[k] published after the cut-off date for the PM AQCD.[239] Taken together this information supports the conclusion that PM-related emissions such as those controlled in this action are associated with adverse health effects.

3.4.2.1 Short-Term Exposure Mortality and Morbidity Studies

As discussed in the PM AQCD, short-term exposure to $PM_{2.5}$ is associated with premature mortality from cardiopulmonary diseases (PM AQCD, p. 8-305), hospitalization and

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

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

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

Among the studies of effects from short-term exposure to $PM_{2.5}$, several studies specifically address the contribution of mobile sources to short-term $PM_{2.5}$ effects on daily mortality. These studies indicate that there are statistically significant associations between mortality and PM related to mobile source emissions (PM AQCD, p.8-85). The analyses incorporate source apportionment tools into daily mortality studies and are briefly mentioned here. Analyses incorporating source apportionment by factor analysis with daily time-series studies of daily death indicated a relationship between mobile source $PM_{2.5}$ and mortality.[240,241] Another recent study in 14 U.S. cities examined the effect of PM_{10} exposures on daily hospital admissions for cardiovascular disease. They found that the effect of PM_{10} was significantly greater in areas with a larger proportion of PM_{10} coming from motor vehicles, indicating that PM_{10} from these sources may have a greater effect on the toxicity of ambient PM_{10} when compared with other sources.[242] 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

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

The PM AQCD and PM Staff Paper emphasize the results of two long-term studies, the Six Cities and American Cancer Society (ACS) prospective cohort studies, based on several factors – the inclusion of measured PM data, the fact that the study populations were similar to the general population, and the fact that these studies have undergone extensive reanalysis (PM AQCD, p. 8-306, Staff Paper, p.3-18).[243,244,245] These studies indicate that there are significant associations for all-cause, cardiopulmonary, and lung cancer mortality with long-term exposure to $PM_{2.5}$. A variety of studies have been published since the completion of the AQCD. One such study, which was summarized in EPA's provisional assessment, was an analysis of a subset of the ACS cohort data, which was published after the PM AQCD was finalized but in time for the 2006 Provisional Assessment, found a larger association than had previously been reported between long-term $PM_{2.5}$ exposure and mortality in the Los Angeles area using a new exposure estimation method that accounted for variations in concentration within the city.[246] EPA is assessing the significance of this study within the context of the broader literature.

As discussed in the PM AQCD, the morbidity studies that combine the features of cross-

sectional and cohort studies provide the best evidence for chronic exposure effects. Long-term studies evaluating the effect of ambient PM on children's development have shown some evidence indicating effects of $PM_{2.5}$ and/or PM_{10} on reduced lung function growth (PM AQCD, Section 8.3.3.2.3). A variety of studies have been published since the completion of the AQCD. One such study, which was summarized in EPA's provisional assessment, reported the results of a cross-sectional study of outdoor $PM_{2.5}$ and measures of atherosclerosis in the Los Angeles basin.[247] The study found significant associations between ambient residential $PM_{2.5}$ and carotid intima-media thickness (CIMT), an indicator of subclinical atherosclerosis, an underlying factor in cardiovascular disease. EPA is assessing the significance of this study within the context of the broader literature.

3.4.2.3 Roadway-Related Pollution Exposure

A recent body of studies reinforces the findings of these PM morbidity and mortality effects by looking at traffic-related exposures, PM measured along roadways, or time spent in traffic and adverse health effects. While many of these studies did not measure PM specifically, they include potential exhaust exposures which include mobile source PM because they employ indices such as roadway proximity or traffic volumes. One study with specific relevance to $PM_{2.5}$ health effects is a study that was done in North Carolina looking at concentrations of $PM_{2.5}$ inside police cars and corresponding physiological changes in the police personnel driving the cars. The authors report significant elevations in markers of cardiac risk associated with concentrations of $PM_{2.5}$ inside police cars on North Carolina state highways.[248] A number of studies of traffic-related pollution have shown associations between fine particles and adverse respiratory outcomes in children who live near major roadways.[249,250,251] 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 rule will 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. In 2005, EPA designated 39 nonattainment areas for the 1997 $PM_{2.5}$ NAAQS based on air quality design values (using 2001-2003 or 2002-2004 measurements) and a number of other factors.[1] (70 FR 943, January 5, 2005; 70 FR 19844, April 14, 2005). These areas are comprised of 208 full or partial counties with a total population exceeding 88 million. The 1997 $PM_{2.5}$ nonattainment areas and populations, as of October 2006, are listed in Appendix 3C to this RIA. As mentioned in Section 3.4.1, the 1997 $PM_{2.5}$ NAAQS was recently revised and the 2006 $PM_{2.5}$ NAAQS became effective on December 18, 2006. Nonattainment areas will be designated with respect to the 2006 $PM_{2.5}$ NAAQS in early 2010. Table 3.4-1 presents the number of counties in areas

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

currently designated as nonattainment for the 1997 $PM_{2.5}$ NAAQS as well as the number of additional counties which have monitored data that is violating the 2006 $PM_{2.5}$ NAAQS.

Table 3.4-1. $PM_{2.5}$ Standards: Current Nonattainment Areas and Other Violating Counties

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

1) Population numbers are from 2000 census data.
2) This table provides an estimate of the counties violating the 2006 $PM_{2.5}$ NAAQS based on 2003-05 air quality data. The areas designated as nonattainment for the 2006 $PM_{2.5}$ NAAQS will be based on 3 years of air quality data from later years. Also, the county numbers in the summary table includes only the counties with monitors violating the 2006 $PM_{2.5}$ NAAQS. The monitored county violations may be an underestimate of the number of counties and populations that will eventually be included in areas with multiple counties designated nonattainment.

States with $PM_{2.5}$ nonattainment areas will be required to take action to bring those areas into compliance in the future. Most $PM_{2.5}$ nonattainment areas will be required to attain the 1997 $PM_{2.5}$ NAAQS in the 2010 to 2015 time frame and then be required to maintain the 1997 $PM_{2.5}$ NAAQS thereafter.[m] The attainment dates associated with the potential nonattainment areas based on the 2006 $PM_{2.5}$ NAAQS would likely be in the 2015 to 2020 timeframe. The emission standards being finalized in this action will become effective between 2009 and 2015. The expected $PM_{2.5}$ and $PM_{2.5}$ precursor inventory reductions from the standards finalized in this action will be useful to states in attaining or maintaining the $PM_{2.5}$ NAAQS.

3.4.3.2 Current PM_{10} Levels

EPA designated PM_{10} nonattainment areas in 1990.[n] As of October 2006, approximately 28 million people live in the 46 areas that are designated as PM_{10} nonattainment, for either failing to meet the PM_{10} NAAQS or for contributing to poor air quality in a nearby area. There are 46 full or partial counties that make up the PM_{10} nonattainment areas. The PM_{10} nonattainment areas and populations are listed in Appendix 3C to this RIA.

As mentioned in Section 3.4.1, the 1997 PM NAAQS was recently revised and the 2006 PM NAAQS became effective on December 18, 2006. The annual PM_{10} NAAQS was revoked and the 24 hour PM_{10} NAAQS was not changed. The projected reductions in emissions from the controls finalized in this action will be useful to states to maintain the PM_{10} NAAQS.

[m] The EPA finalized $PM_{2.5}$ attainment and nonattainment areas in April 2005. The EPA proposed the PM Implementation rule in November 2005 (70 FR 65984).
[n] A PM_{10} design value is the concentration that determines whether a monitoring site meets the NAAQS for PM_{10}. The full details involved in calculating a PM_{10} design value are given in Appendices H and I of 40 CFR Part 50.

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

Recently PM air quality analyses were performed for the PM NAAQS final rule, which was promulgated by EPA in 2006. The Community Multiscale Air Quality (CMAQ) model was used as the tool for simulating base and future year concentrations of PM, visibility and deposition in support of the PM NAAQS air quality assessment. The PM NAAQS analysis included all final federal rules up to and including Clean Air Interstate Rule (CAIR) and all final mobile source rule controls as of October 2006. Details on the air quality modeling are provided in the Regulatory Impact Analysis (RIA) for the Final PM NAAQS Rule, included in the docket for this final rule.[252]

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

Air quality modeling performed for the final PM NAAQS indicates that in the absence of additional local, regional or national controls, there will likely continue to be counties that will not attain some combination of the annual 2006 $PM_{2.5}$ standard (15 $\mu g/m^3$) and the daily 2006 $PM_{2.5}$ standard (35 $\mu g/m^3$). The PM NAAQS analysis provides estimates of future $PM_{2.5}$ levels across the country. For example, in 2015 based on emission controls currently adopted or expected to be in place[o], we project that 53 million people will live in 52 counties with projected $PM_{2.5}$ design values at and above the 2006 standard, see Table 3.4-2.[p] The rule will assist these counties in attaining the $PM_{2.5}$ NAAQS. Table 3.4-2 also lists the 54 counties, where 27 million people are projected to live, with 2015 projected design values that do not violate the $PM_{2.5}$ NAAQS but are within ten percent of it. The rule may help ensure that these counties continue to maintain their attainment status.

Table 3.4-2. Counties with 2015 Projected Annual and Daily $PM_{2.5}$ Design Values Above and within 10% of the 2006 $PM_{2.5}$ Standard[a]

State	County	2015 Projected Annual $PM_{2.5}$ Design Value ($\mu g/m^3$)	2015 Projected Daily $PM_{2.5}$ Design Value ($\mu g/m^3$)	2015 Population[b]

[o] 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 rule will help these areas attain the PM standards by their statutory date.

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

Alabama	Jefferson Co	**15.9**	**36.9**	669,850
California	Alameda Co	**13.3**	**59.4**	1,628,698
California	Butte Co	**13.4**	**50.7**	242,166
California	Colusa Co	9.5	33.5	23,066
California	Contra Costa Co	**12.6**	**61.3**	1,155,323
California	Fresno Co	**20.1**	**73.0**	960,934
California	Imperial Co	**14.8**	**45.7**	173,482
California	Inyo Co	**6.1**	**38.1**	19,349
California	Kern Co	**21.3**	**81.4**	804,940
California	Kings Co	**17.2**	**70.6**	161,607
California	Los Angeles Co	**23.7**	**62.2**	9,910,805
California	Merced Co	**15.8**	**54.4**	250,152
California	Orange Co	**20.0**	**41.1**	3,467,120
California	Placer Co	**11.4**	**38.1**	403,624
California	Riverside Co	**27.8**	**73.5**	2,015,955
California	Sacramento Co	**12.2**	**49.8**	1,488,456
California	San Bernardino Co	**24.6**	**65.7**	2,157,926
California	San Diego Co	**15.8**	**40.7**	3,489,368
California	San Francisco Co	**11.3**	**52.5**	765,846
California	San Joaquin Co	**15.4**	**51.1**	675,362
California	San Luis Obispo Co	**9.4**	**35.8**	304,079
California	San Mateo Co	**10.5**	**41.9**	785,949
California	Santa Clara Co	**10.7**	**48.5**	1,899,727
California	Solano Co	**11.7**	**57.7**	529,784
California	Sonoma Co	**10.0**	**38.9**	569,486
California	Stanislaus Co	**16.6**	**61.9**	547,041
California	Sutter Co	**11.2**	**39.3**	99,716
California	Tulare Co	**21.2**	**77.2**	441,185
California	Ventura Co	**14.1**	**38.8**	923,205
California	Yolo Co	10.2	33.0	206,388
Connecticut	Fairfield Co	11.0	31.6	893,629
Georgia	Bibb Co	13.7	27.0	160,468
Georgia	Clayton Co	13.9	28.7	280,476
Georgia	DeKalb Co	13.6	31.5	715,947
Georgia	Floyd Co	14.0	30.9	97,674
Georgia	Fulton Co	**15.5**	**32.2**	877,365
Georgia	Muscogee Co	13.4	34.2	197,634
Georgia	Wilkinson Co	13.6	29.3	11,259
Idaho	Ada Co	8.9	32.2	397,456
Idaho	Bannock Co	**9.1**	**40.2**	88,033
Idaho	Canyon Co	9.2	32.6	154,137
Idaho	Power Co	**10.5**	**36.6**	8,932
Idaho	Shoshone Co	**12.4**	**36.2**	15,646
Illinois	Cook Co	**15.5**	**37.1**	5,362,931
Illinois	Madison Co	**15.2**	**35.5**	271,854
Illinois	St. Clair Co	14.6	30.4	251,612
Illinois	Will Co	13.2	32.0	634,068
Indiana	Clark Co	13.6	31.1	112,523
Indiana	Lake Co	**13.4**	**40.8**	490,795

Indiana	Marion Co	13.5	33.1	889,645
Kentucky	Jefferson Co	13.8	33.4	710,231
Maryland	Anne Arundel Co	11.1	33.2	574,322
Maryland	Baltimore city	13.0	35.5	596,076
Maryland	Baltimore Co	11.3	32.6	810,172
Massachusetts	Hampden Co	11.6	32.9	452,055
Michigan	Kalamazoo Co	12.8	32.7	257,817
Michigan	Kent Co	12.0	31.9	654,449
Michigan	Oakland Co	13.0	33.2	1,355,670
Michigan	St. Clair Co	12.5	32.5	185,970
Michigan	Wayne Co	**17.4**	**39.0**	1,921,253
Montana	Lincoln Co	**15.0**	**42.4**	19,875
Montana	Missoula Co	10.6	32.1	118,303
New Jersey	Camden Co	11.1	32.1	512,135
New Jersey	Hudson Co	12.0	32.8	604,036
New Jersey	Union Co	12.2	32.8	525,096
New York	Bronx Co	12.8	33.2	1,283,316
New York	New York Co	14.0	33.2	1,551,641
Ohio	Cuyahoga Co	**15.4**	**40.0**	1,325,507
Ohio	Franklin Co	13.7	33.5	1,181,578
Ohio	Hamilton Co	14.3	34.2	841,858
Ohio	Jefferson Co	14.2	34.2	68,909
Ohio	Lucas Co	12.5	32.2	443,230
Ohio	Scioto Co	**15.6**	**34.3**	81,013
Ohio	Trumbull Co	12.1	34.2	227,546
Oregon	Jackson Co	**10.9**	**37.6**	250,169
Oregon	Klamath Co	**10.1**	**39.1**	69,423
Oregon	Lane Co	**12.9**	**53.6**	387,237
Oregon	Washington Co	9.0	32.0	639,839
Pennsylvania	Allegheny Co	**16.5**	**53.4**	1,245,917
Pennsylvania	Beaver Co	12.1	33.2	184,648
Pennsylvania	Berks Co	**12.0**	**35.5**	396,410
Pennsylvania	Dauphin Co	11.0	33.3	272,748
Pennsylvania	Lancaster Co	12.2	33.7	535,622
Pennsylvania	Lehigh Co	10.5	34.7	328,523
Pennsylvania	Mercer Co	11.0	31.6	123,577
Pennsylvania	Northampton Co	10.9	35.0	286,838
Pennsylvania	Philadelphia Co	13.3	35.2	1,372,037
Pennsylvania	York Co	**12.3**	**35.9**	417,408
Tennessee	Knox Co	13.6	29.6	448,931
Utah	Box Elder Co	**8.6**	**39.0**	49,878
Utah	Cache Co	**12.5**	**51.9**	114,729
Utah	Salt Lake Co	**12.6**	**49.3**	1,133,410
Utah	Utah Co	**9.3**	**36.7**	508,106
Utah	Weber Co	**9.1**	**36.2**	229,807
Washington	Clark Co	9.2	34.3	479,002
Washington	King Co	10.8	34.0	2,013,808
Washington	Pierce Co	**11.1**	**43.0**	879,363
Washington	Snohomish Co	**11.3**	**40.1**	782,319
Washington	Thurston Co	8.9	34.9	264,364

Washington	Yakima Co	9.6	34.9	261,452
West Virginia	Berkeley Co	12.0	32.7	99,349
West Virginia	Hancock Co	13.4	32.7	30,857
West Virginia	Kanawha Co	13.9	28.9	196,498
Wisconsin	Milwaukee Co	12.1	32.1	908,336
Wisconsin	Waukesha Co	11.8	32.4	441,482
Wyoming	Sheridan Co	10.5	31.8	28,623
Number of Violating Counties			52	
Population of Violating Counties				53,468,515
Number of Counties within 10%			54	
Population of Counties within 10%				26,896,926

a) Bolded concentrations indicate levels above the $PM_{2.5}$ standard.
b) 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 Impairment

Visibility can be defined as the degree to which the atmosphere is transparent to visible light.[253] Visibility impairment manifests in two principal ways: as local visibility impairment and as regional haze.[q] Local visibility impairment may take the form of a localized plume, a band or layer of discoloration appearing well above the terrain as a result from complex local meteorological conditions. Alternatively, local visibility impairment may manifest as an urban haze, sometimes referred to as a "brown cloud." This urban haze is largely caused by emissions from multiple sources in the urban areas and is not typically attributable to only one nearby source or to long-range transport. The second type of visibility impairment, regional haze, usually results from multiple pollution sources spread over a large geographic region. Regional haze can impair visibility over large regions and across states.

Visibility is important because it has direct significance to people's enjoyment of daily activities in all parts of the country. Individuals value good visibility for the well-being it provides them directly, where they live and work, and in places where they enjoy recreational opportunities. Visibility is also highly valued in significant natural areas such as national parks and wilderness areas, and special emphasis is given to protecting visibility in these areas. For more information on visibility see the 2004 PMAQCD as well as the 2005 PM Staff Paper.[254,255]

Fine particles are the major cause of reduced visibility in parts of the United States. To address the welfare effects of PM on visibility, EPA set secondary $PM_{2.5}$ standards which would

q See discussion in U.S. EPA , National Ambient Air Quality Standards for Particulate Matter; Proposed Rule; January 17, 2006, Vol71 p 2676. This information is available electronically at http://epa.gov/fedrgstr/EPA-AIR/2006/January/Day-17/a177.pdf.

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

Data showing $PM_{2.5}$ nonattainment areas and visibility levels above background at the Mandatory Class I Federal Areas demonstrate that 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.[256]

As mentioned above the secondary $PM_{2.5}$ standards were set as equal to the suite of primary $PM_{2.5}$ standards. Recently designated $PM_{2.5}$ nonattainment areas indicate that almost 90 million people live in 208 counties that are in nonattainment for the 1997 $PM_{2.5}$ NAAQS, see Appendix 3C. Thus, at least these populations (plus others who travel to these areas) would likely be experiencing visibility impairment.

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.[257,258] 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.[259]

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

[r] 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.

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. [260]

3.4.4.1.3 Future Visibility Impairment

Recent modeling for the final PM NAAQS rule was used to project $PM_{2.5}$ levels in the U.S. in 2015. The results suggest that $PM_{2.5}$ levels above the 2006 NAAQS will persist in the future. We predicted that in 2015, there will be 52 counties with a population of 53 million where annual $PM_{2.5}$ levels will exceed the 2006 $PM_{2.5}$ NAAQS, see Table 3.4-1. Thus, in the future, a percentage of the population may continue to experience 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 current and anticipated visibility impairment and the emission reductions finalized here may help improve future visibility impairment.

3.4.4.1.4 Future Visibility Impairment at Mandatory Class I Federal Areas

Achieving the $PM_{2.5}$ NAAQS will help improve visibility across the country, but it will not be sufficient to meet the statutory goal of no manmade impairment in the mandatory class I federal areas (64 FR 35714, July 1, 1999 and 62 FR 38652, 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 the $PM_{2.5}$ NAAQS and where light extinction is significantly above natural background. Thus, large areas of the Eastern United States have air pollution that is causing and will continue to cause visibility impairment. In the West, scenic vistas are especially important to public welfare. Although the $PM_{2.5}$ NAAQS is met in most areas outside of California, virtually the entire West is in close proximity to a scenic mandatory class I federal area protected by 169A and 169B of the CAA.

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

The overall goal of the regional haze program is to prevent future visibility impairment and remedy existing visibility impairment in mandatory class I federal areas. As shown by the future visibility estimates in Appendix 3D it is projected that there will continue to be mandatory class I federal areas with visibility levels above background in 2015. [261] Additional emission reductions will be needed from the broad set of sources that contribute, including the vehicles and gas cans subject to this rule. The reductions being finalized 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.[262]

Adverse impacts on water quality can occur when atmospheric contaminants deposit to the water surface or when material deposited on the land enters a waterbody through runoff. Potential impacts of atmospheric deposition to waterbodies include those related to both nutrient and toxic inputs. Adverse effects to human health and welfare can occur from the addition of excess particulate nitrate nutrient enrichment which contributes to toxic algae blooms and zones of depleted oxygen, which can lead to fish kills, frequently in coastal waters. Particles contaminated with heavy metals or other toxins may lead to the ingestion of contaminated fish, ingestion of contaminated water, damage to the marine ecology, and limited recreational uses. Several studies have been conducted in U.S. coastal waters and in the Great Lakes Region in which the role of ambient PM deposition and runoff is investigated.[263,264,265,266,267]

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

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 (PM AQCD, p. 4-87).[268] Investigation of trace metals near roadways and industrial facilities indicate that a substantial burden of heavy metals can accumulate on vegetative surfaces. Copper, zinc, and nickel have been documented to cause direct toxicity to vegetation under field conditions (PM AQCD, p. 4-75). Little research has been conducted on the effects associated with mixtures of contaminants found in ambient PM. While metals typically exhibit low solubility, limiting their bioavailability and direct toxicity, chemical transformations of metal compounds occur in the environment, particularly in the presence of acidic or other oxidizing species. These chemical changes influence the mobility and toxicity of metals in the environment. Once taken up into plant tissue, a metal compound can undergo chemical changes, accumulate and be passed along to herbivores or can re-enter the soil and further cycle in the environment.

Although there has been no direct evidence of a physiological association between tree injury and heavy metal exposures, heavy metals have been implicated because of similarities between metal deposition patterns and forest decline (PM AQCD, p. 4-76).[269] Contamination of plant leaves by heavy metals can lead to elevated soil levels. Some trace metals absorbed into the plant and can bind to the leaf tissue (PM AQCD, p. 4-75). When these leaves fall and decompose, the heavy metals are transferred into the soil.[270,271]

The environmental sources and cycling of mercury are currently of particular concern due to the bioaccumulation and biomagnification of this metal in aquatic ecosystems and the potent toxic nature of mercury in the forms in which is it ingested by people and other animals. Mercury is unusual compared with other metals in that it largely partitions into the gas phase (in elemental form), and therefore has a longer residence time in the atmosphere than a metal found predominantly in the particle phase. This property enables a portion of emitted mercury to travel far from the primary source before being deposited and accumulating in the aquatic ecosystem. Localized or regional impacts are also observed for mercury emitted from combustion sources. The major source of mercury in the Great Lakes is from atmospheric deposition, accounting for approximately eighty percent of the mercury in Lake Michigan.[272,273] Over fifty percent of the mercury in the Chesapeake Bay has been attributed to atmospheric deposition.[274] Overall, the National Science and Technology Council (NSTC, 1999) identifies atmospheric deposition as the primary source of mercury to aquatic systems. Forty-four states have issued health advisories for the consumption of fish contaminated by mercury; however, most of these advisories are issued in areas without a mercury point source.

Elevated levels of zinc and lead have been identified in streambed sediments, and these elevated levels have been correlated with population density and motor vehicle use.[275,276] 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.[277] 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.[278] Polycyclic aromatic hydrocarbons (PAHs) are a class of POM that contains compounds which are known or suspected carcinogens.

Major sources of PAHs include mobile sources. PAHs in the environment may be present as a gas or adsorbed onto airborne particulate matter. Since the majority of PAHs are adsorbed onto particles less than 1.0 μm in diameter, long range transport is possible. However, studies have shown that PAH compounds adsorbed onto diesel exhaust particulate and exposed to ozone have half lives of 0.5 to 1.0 hours.[279]

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.[280,281] Analyses of PAH deposition to Chesapeake and Galveston Bay indicate that dry deposition and gas exchange from the atmosphere to the surface water predominate.[282,283] 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.[284] 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.[285] 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.[286] 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.[287]

Cousins et al. (1999) estimates that greater than ninety percent of semi-volatile organic compound (SVOC) emissions in the United Kingdom deposit on soil.[288] An analysis of polycyclic aromatic hydrocarbon (PAH) concentrations near a Czechoslovakian roadway indicated that concentrations were thirty times greater than background.[289]

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.[290] 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

Over the years there have been a large number of studies that 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.

As noted in section 3.2, HAPEM6 estimates the changes in time-weighted exposures associated with proximity to roadways for individual pollutants. The studies discussed in this section address exposures and health effects that are at least partially captured by our modeling, but there may be additional exposures and health effects associated with pollutants, singly or in combination, that are not explicitly quantified. However, because the studies discussed in this section often employ exposure estimation metrics associated with multiple pollutants, exposure-response information from these studies may not be suitable for risk assessment geared around one or several chemicals.

At this point, there exists no exposure metric specific to "traffic," although as noted above, a wide variety of gaseous, particulate, and semi-volatile species are elevated near roadways. As a result, the exposure metrics employed generally indicate the presence and/or intensity of a mixture of air pollutants for exposure assessment. Many of the health studies discussed below employ non-specific exposure metrics, including traffic on roads nearest home or school, distance to the nearest road, measured outdoor nitrogen dioxide concentrations, air quality dispersion modeling of specific traffic-generated chemicals, and exposure assignment based on land use. These exposure metrics represent the mixture of traffic-generated pollutants, rather than individual pollutants. Accordingly, such results are not directly comparable with community epidemiology studies that employ ambient measurements of particulate matter or ozone over a fixed time period, or to toxicological studies employing a single pollutant to evaluate responses in humans or animals.

A wide range of health effects are reported in the literature related to near roadway and in-vehicle exposures. This is not unexpected, given the chemical and physical complexity of the

mixture to which people are exposed in this environment. These effects overlap with those identified in our discussion of the effects of PM and ozone. The discussion below addresses the studies in detail. However, in general terms, 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.

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., percent aromatics), and relevant traffic patterns. Furthermore, in these studies, it is often difficult 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.

3.5.1 Mortality

The quantifiable effects of this rule on premature mortality associated with exposure to $PM_{2.5}$ are assessed as part of the benefits estimates for this rule. In addition to studies that have documented the relationship between ambient PM and premature mortality, a few recent studies have investigated the relationship between premature mortality and broader indicators of transportation emissions, such as residence near traffic. The extent to which these studies are detecting any additional effects not accounted for in the ambient PM-premature mortality relationship is unclear.

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.

A total of three cohort studies have examined premature mortality in relation to residence near traffic, another examined county-level traffic density, while one other has examined stroke mortality. In addition, one study accounted for the effect of residence along a major road on associations with daily deaths in a time-series study. These studies constitute all of the studies examining mortality with reference to proximity to traffic.

Premature mortality in adults in association with living near high-traffic roadways has been studied in three recent cohort studies for all-cause and cardiopulmonary mortality from the Netherlands, Ontario, Canada, and most recently, Germany.[291,292,293] 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.[294]

The second study involved over 5,200 subjects aged 40 years or more, all living in the Hamilton, Ontario area. This study examined total mortality, 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). A subsequent follow-up study found elevated mortality rates from circulatory causes in the Canadian study population.

Most recently, German investigators followed up a series of cross-sectional studies on women age 50-59 living in the North Rhine-Westphalia region during the late 1980's and 1990's, tracking vital status and migration to the years 2002-2003.[295] In total, the cohort consisted of approximately 4800 women. Exposures were categorized using ambient NO_2 and PM_{10} (estimated from TSP), and an indicator of residence within 50 m of a "major road", defined at ≥10,000 cars/day. Overall, living within 50 meters of a major road was associated with a significant 70% increase in the rate of cardiopulmonary mortality. Nearest-monitor NO_2 and PM_{10} were also associated with a 57% and 34% increase in the rate of cardiopulmonary mortality. Exposure to NO_2 was also associated with a 17% increase in all-cause mortality.

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 another study evaluating a cohort of older, hypertensive male U.S. veterans, county-level traffic index and pollution estimates were employed in estimating exposure to traffic activity and other air pollutants.[296] Area-based traffic density was significantly associated with increased mortality rates, as were constituents of motor vehicle exhaust, such as elemental carbon.

One cohort study conducted in the United Kingdom examined cardiocerebral (stroke) mortality in relation to living near traffic.[297] Those living in census areas near roadways had significantly higher stroke mortality rates. 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. 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.[298] 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. Furthermore, in the cohort studies, although controls for income level were incorporated based on postal code or census area, it is possible that other unmeasured covariates explain the associations with traffic.

3.5.2 Non-Allergic Respiratory Symptoms

Our analysis of the benefits associated with reduced exposure to $PM_{2.5}$ includes chronic bronchitis, hospital admissions for respiratory causes, emergency room visits for asthma, acute bronchitis, upper and lower respiratory symptoms and exacerbation of asthma. In addition, 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.[299] The discussion below covers all studies conducted in the United States. EPA has not formally evaluated the extent to which these studies may be documenting health effects that are already included in the benefits analysis associated with PM.

Most recently, a study from Cincinnati, OH examined the prevalence of wheezing in a group of infants less than one year of age.[300] 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.[301] 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.[302] 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.[303] 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.[304] 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.

The studies described above employ different exposure metrics and health endpoints, making evaluation difficult. However, numerous other studies from around the world also provide evidence for increased prevalence of respiratory symptoms among people living near major roads. For a detailed listing, refer to the docket of this rule. Taken together, these studies provide evidence that respiratory symptoms may be 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.[305] Overall, the review concluded that there is some limited evidence of an association between traffic-generated pollutants and asthma incidence. More recent studies have also found significant associations between prevalent asthma and living near major roads.[306] 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. The potential for these effects is not taken into account in the benefits analysis for PM because EPA's various scientific advisors have argued that the literature is not strong enough to support a causal association.

3.5.4 Cardiovascular Effects

Cardiovascular effects are currently seen as a potentially important set of mechanisms whereby $PM_{2.5}$ may be leading to premature mortality. In Chapter 12, we estimate the quantifiable benefits of PM-related non-fatal acute myocardial infarction and cardiovascular hospital admissions. The studies described in Section 3.5.1 found higher relative risks for cardiopulmonary causes of death.

In addition to cardiopulmonary mortality, some studies have looked at morbidity. A recent study from Germany also found significant increased odds of coronary heart disease (CHD) in a cohort of approximately 3400 participants.[307] Residents living within 150 meters of major roads were compared to those living further ways. Overall, controlling for background air pollution and individual risk factors, the adjusted odds ratio for CHD prevalence was significantly elevated (1.85). Subgroup analyses indicated stronger effects in men, in participants under 60 year of age, and in never-smokers.

Several additional 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.[308] 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.[309] 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.[310] In the study, ambient $PM_{2.5}$ was associated with changes consistent with reduced

autonomic control of the heart. Black carbon, often a more reliable index of traffic-related pollution, was also 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.[311] These studies further document a hypothesized mechanism associated with motor vehicle emissions, but do not necessarily suggest effects independent of those identified in our discussion of PM health effects.

3.5.5 Birth Outcomes

A few studies examining birth outcomes in populations living near major traffic sources have 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. Our discussion of PM health effects also quantitatively accounts for premature mortality effects in infants and qualitatively accounts for low birth weight.

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.[312] 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.[313] In another study, preterm birth was associated with ambient PM_{10} and CO.[314] 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.[315] 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.[316] Subsequent follow-up of the same birth cohort to age three found evidence of neurodevelopmental deficits associated with maternal exposure to PAHs during pregnancy, particularly in cognitive development.[317]

Overall, although the number of studies examining perinatal exposures is small, there is some evidence that exposure to traffic-related pollutants may be 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 for compounds with a mutagenic mode of action to account for early life exposures in the Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens.[318]

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 some evidence that key changes related to the development of childhood leukemia occur in the developing fetus.[319]

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.[320] The study was reanalyzed using an approach to combine traffic volume with residential distance from major roads to assess "distance-weighted traffic volume."[321] The study found that the significant, monotonically increasing risks associated with increased distance-weighted traffic volume.

NO_2 has been used as an indicator of traffic emissions in some studies; however, it is important to note that NO_2 is not implicated as causing cancer. For instance, a study used a dispersion model of NO_2 from traffic to conduct a case-control study of childhood cancer in Sweden.[322] 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.[323] A large study of nearly 2,000 Danish children with cancer found no association between modeled concentrations of benzene and NO_2 at home and the risk of leukemia, central nervous system tumors, or total cancers.[324] 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.[325] 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.[326]

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.[327] 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, PM_{10}, 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

Taken together, the available studies of health effects in residents near major roadways suggest a possible public health concern. These studies' exposure metrics are reflective of a complex mixture from traffic, and the standards will reduce a broad range of pollutants present in higher concentrations near roadways. It is unclear to what extent these health effects are attributable to PM versus other components of the complex mixture. Note that the benefits associated with the direct PM reductions from the cold temperature vehicle standards are presented in Chapter 12 of this RIA.

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. Those living near major roadways are a subpopulation of the total population included in quantitative analysis, and to the extent that there may be additional exposures and health effects not captured in analyses for the total population, we enumerate the size and characteristics of the subpopulation. A study of the populations nationally using geographic information systems indicated that more than half of the population lives within 200 meters of a major road (see file USbytract.txt in the docket for this rule).[s] It should be noted that this analysis relied on the Census Bureau definition of a major road, which is not based on traffic volume. Thus, some of the roads designated as "major" may carry a low volume of traffic. Detailed analyses of data were conducted in three states, Colorado, Georgia, and New York. 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%.[328]

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.[329] 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.[330] 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. [331] 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

[s] Major roads are defined as those roads defined by the U.S. Census as one of the following: "limited access highway," "highway," "major road (primary, secondary and connecting roads)," or "ramp."

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.[332] 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.[333] 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: Influence of Emissions in Attached Garages on Indoor Air Benzene Concentrations and Human Exposure

Introduction

Measurement studies provide strong evidence that VOC sources in attached garages can significantly increase VOC concentrations inside homes.[334] Preliminary analyses of data from a pilot study for the National Human Exposure Assessment Survey (NHEXAS) in Arizona also found indoor concentrations of mobile source-related VOC compounds significantly higher in homes with attached garages than in homes without them.[335] This population-based exposure study included measurements from 187 homes. A study in 50 Alaska residences found that in homes with attached garages, indoor benzene levels averaged 70.8 $\mu g/m^3$, while in homes without attached garages, concentrations averaged 8.6 $\mu g/m^3$.[336] Multiple factors, including house architecture, ventilation design, garage configuration, and climate can all play roles as well.

National-scale air toxics modeling efforts, such as those discussed in RIA Section 3.2.1.2, employ Gaussian dispersion models in combination with human exposure models to calculate the concentrations of air toxics in various microenvironments. Exposure models calculate an average exposure resulting from the movement of a simulated population through a time-activity pattern that brings them into contact with air in the various microenvironments.

At this point, the NATA and the analyses performed for this rulemaking have only included exposures from outdoor sources. Although the HAPEM6 exposure model is capable of addressing indoor sources, more thorough analyses of the prevalence and use of emission sources within attached garages are required to develop quantitative estimates of model parameters to address attached garage contributions across the U.S. population.

This appendix addresses the potential impact of all benzene sources within an attached garage on residential indoor air quality.

Methods

Calculation of Within-garage Source Emission Rate

Emission rates for indoor sources of VOCs can be derived by several methods. Most accurately, the actual emission rates of an indoor VOC source can be measured through the use of a Sealed Housing for Evaporative Determination (SHED). However, test conditions must be representative of real world applications. Short of SHED-based measurement, several surrogate approaches may be employed. For evaporative losses from a sealed container, the change in weight of a container over time may be used to calculate a total mass loss rate, which can be assumed to be in the form of VOC. Alternatively, if the air concentrations and ventilation conditions of a defined indoor space are known, mass balance equations can be employed to derive a "virtual" emission rate for all sources within the space.

This appendix employs the latter approach in calculating source emission factors. The general approach of a mass balance equation is to calculate the change in mass over a given time, accounting for the mass of a pollutant transported into a space, the mass of pollutant transported out of a space, the emission rate of a source within the space, and the decay of any pollutants

within the space, which can be treated as a first-order decay. A simple space like a garage can be treated as a single zone. The differential equation representing this mass balance is as follows:

$$(1) \qquad \frac{dM_{t,i}}{dt} = C_o k \frac{dV}{dt} + \frac{dM_i}{dt} - C_i \frac{dV}{dt}$$

Here, $dM_{t,i}/dt$ represents the rate of change of total indoor mass, C_i is the indoor concentration, C_o is the outdoor concentration, dV/dt is the volumetric air flow through the space, k is the penetration fraction indicating the proportion of mass that passes through the wall of the compartment, and dM_i/dt represents the mass emission rate inside the space. Note that all air entering the garage is assumed to enter from outdoors.

Assuming steady-state conditions, $dM_{t,i}/dt$ assumes the value of zero, meaning that the concentration in the garage does not change over time. Algebraically, this allows the equation above to be represented as:

$$(2) \qquad \left(\frac{dV}{dt}\right)\left(C_i - C_o k\right) = \frac{dM_i}{dt}$$

In other words, the indoor source terms can be calculated if the volumetric flow through the space and concentrations indoor and outdoor are known. Any gradient in concentration between indoor and outdoor concentrations is explained by indoor sources and the fraction of mass that does not penetrate from indoors to outdoors.

The volumetric flow can be calculated by multiplying the volume of the space by the number of times per hour that the air within the space is turned over. As such:

$$(3) \qquad \frac{dV}{dt} = \alpha V$$

Here, α is the "air exchange rate," expressed in air changes per hour (ACH). Combining equations (2) and (3), the mass emission rate is represented as:

$$(4) \qquad \alpha V\left(C_i - C_o k\right) = \frac{dM_i}{dt}$$

A recent study in Ann Arbor, MI measured the air exchange rates and the in-garage and outdoor concentrations of VOCs needed to perform these calculations.[337] The homes in the study were based on a convenience sample, and so may not be generally representative of the local or national housing stock. All garages but one adjoined a house. All attached garages had between one and three walls adjoining a residence. The distributions of garage benzene concentration and ACH are shown in Figure 3A-1. The distributions of each were not significantly different from lognormal, judging by the Kolmogorov-Smirnov Z statistic.

Values of k, the penetration factor, are dependent on the physical pathways through which air passes into a garage, as well as the presence and chemical composition of any insulating material through which air passes. In the case of garages, the infrequency of insulated garages and the low reactivity of benzene justifies the assumption that k=1.[338]

These data from the Ann Arbor, MI study were used to solve equation (2) to derive a distribution of benzene mass emission rates in each garage in the study, based on variability in measurements of outdoor concentrations. Equation 4 was implemented using a Microsoft Excel spreadsheet with the @Risk probabilistic simulation add-in (version 4.5).[339] Monte Carlo sampling was used for all terms in deriving the emission rates.

As described below, this distribution can be used to evaluate the effect of various fuel control measures on indoor benzene concentrations. A single lognormal distribution was used to

represent C_o in equation 4, based on other studies of ambient air, which have found that many pollutants' concentrations are lognormally distributed.

Calculation of Garage Contributions to Indoor Air

In the same way that a mass balance calculation can be used to calculate emission rates for sources within garages, a mass balance equation can be used to estimate the additional concentration in a home that will occur as a result of elevated concentrations in the garage. However, unlike the garage case, it is not valid to assume that all air entering the home comes directly from outdoors.

Recent studies have provided indications that over multiple sequential days, variability in within-home benzene concentration is relatively small. A recent study from Ann Arbor, MI found a coefficient of variation (COV) of 4.6% for benzene.[340] Furthermore, recent data obtained by EPA through the Environmental and Occupational Health Sciences Institute (EOHSI) on homes in the Elizabeth, NJ area indicates no significant differences in within-home concentrations at a 95% confidence level.[t,341] These data are preliminary, and analyses are still in progress.

Given the fraction of air entering the home through the home-garage interface, the appropriate mass balance equation for a single-compartment (e.g. well-mixed) home can be represented as such:

$$\frac{dM_{t,i}}{dt} = kC_o(1 - f_g)\frac{dV}{dt} + kC_g f_g \frac{dV}{dt} - C_i \frac{dV}{dt} \quad (5)$$

Here, C_i is the in-house concentration, C_o is the outdoor concentration, C_g is the concentration in the garage, dV/dt is the volumetric air flow through the house, and f_g is the fraction of air entering the home from the garage. One assumption made here is that the penetration factor for the air moving through the house-garage interface is the same as air moving through the house-outdoors interface. Reactive decay is assumed to be zero. Such mass balance equations are standard approaches in environmental science and engineering, and are frequently found in textbooks on these subjects.[342]

Again assuming steady-state conditions, $dM_{t,i}/dt = 0$, the equation above simplifies to:

$$C_i = kC_o(1 - f_g) + kC_g f_g \quad (6)$$

Or more simply, the indoor concentration under steady state conditions is proportional to the fraction of air entering the house through the garage.

Figure 3A-2 is a contour plot illustrating the range of average indoor air concentrations that could plausibly arise given a range of values of C_g and f_g, with a background concentration of zero. However, Figure 3A-2 does not answer the question of what the likely indoor air values are in a sample of real homes.

The text below describes procedures and results of a small-scale modeling study.

Modeling Approach

[t] In that study, one air sample was obtained in the room adjacent to an attached garage in each home and another was obtained in another location. The New Jersey Department of Environmental Protection and EPA provided joint funding for the study. A two-sided paired t-test was applied to data obtained from 36 homes over approximately 24 hours.

All modeling analyses employed Equation 6 in a Microsoft Excel spreadsheet with the @Risk probabilistic modeling add-in was the software employed in all modeling analyses. Where appropriate, each of the terms in Equation 6 was treated as a random variable represented as either a parametric distribution or as an empirical distribution based on measured data.

Often, in employing data obtained from more than one study, combining data into a single distribution was not justified on *a priori* grounds. In ventilation studies, ambient conditions such as temperature and geography can substantially affect air flow patterns and building constructions. For instance, residential air exchange rates differ significantly between regions with substantially different climates.[343] Furthermore, based on the limited number of studies available, combining data from multiple studies into a single data set had the potential to apply *de facto* weights to data, potentially shifting the fitted model parameters away from truly "representative" distributions.

Another consideration is the potential for independence of the f_g and C_g variables. There is no *a priori* reason why the "leakiness" of the house-garage envelope should be related to the concentration of benzene in the garage.

Because of these considerations, data on f_g or C_g from studies in different areas were not formally combined. Rather, distributions fit separately to data from each study were used to develop several model "scenarios." As described below, four different studies provided data for C_g and three different studies provided data for f_g. As such, a minimum of 12 (3 x 4) scenarios were needed to represent the totality of available data.

For each scenario modeled, @Risk sampled from each distribution 20,000 times using a proprietary Latin Hypercube sampling framework. The large number of samples and Latin Hypercube strategy were employed to ensure that modeled concentration distributions achieved stability.

Lastly, for comparison to the current approaches for exposure modeling, the following equation was used, paralleling the approach taken by HAPEM5 with no garage emissions:

$$C_i = kC_o \qquad (7)$$

Data for Populating Model Parameters

Fraction of Air Entering Home through the Garage (f_g)

Several studies have examined the fraction of air entering the home from the garage. Except for one, all of these studies took place in northern states and Canada, where homes are built with more insulation. A recent study of a set of homes in Ontario, Canada found that approximately 13% of the air entering the home came from the garage.[344] One study from Minnesota found that in newer homes, houses built in the year 1994 had an average of 17.4% of total air leakage coming through their garages, houses build in 1998 had an average leakage fraction of 10.5%, and houses built in 2000 had an average leakage fraction of 9.4%.[345] Two recent studies have employed perfluorocarbon tracer (PFT) gases to estimate air transport between different "zones" of houses with attached garages. A recent study by Isbell et al. (2005) based in Fairbanks, Alaska found that in a modern air-tight Alaskan home ventilated with an air-to-air heat exchanger, 12.2% of the air entering a home entered through the garage, while 47.4% of the air entering an older home ventilated passively by structural defects came through the attached garage.[346] Another study of a home in Ann Arbor, Michigan built in 1962 found that

16% of the air entering the home originated in the garage.[347] In a more recent study from Ann Arbor, investigators deployed PFT tracers in 15 homes and calculated the fraction of air entering each home through an attached garage, with an average of 6.5±5.3% of the air entering through the garage.[348] From these studies, it is apparent that across homes, the fraction of air entering through the garage is highly variable, making it necessary to acknowledge significant uncertainties in characterizing "typical" infiltration patterns.

Benzene Concentrations in Garage Air (C_g)

Four sources of in-garage concentration data are available in the format relevant for steady-state modeling over extended periods of time. First, there is the study by Batterman et al. (2005), in which average garage concentrations of benzene were measured over a period of four days in each of 15 homes using passive sampling badges. The average garage concentration reported was 36.6 μg/m^3, with a standard deviation of 38.5 μg/m^3.

Second, a study in Alaska by George et al. (2002) measured benzene concentrations in 28 Alaska homes and 48 garages with passive diffusion badges.[349] One disadvantage of this study is the relatively high detection limit for benzene, 7 ppb (22 μg/m^3). As a result, many of the data available are based on a reported value of 50% of the detection limit. In the Alaska study, in-garage benzene concentrations averaged 103 μg/m^3, and the standard deviation was 135 μg/m^3. The study included concurrent in-home measurement of benzene in homes with attached garages, allowing evaluation of the modeled indoor concentrations. However, it is not apparent that this study underwent scientific peer review.

A third study in one New Jersey home also evaluated garage and indoor benzene, as part of an investigation into in-garage emissions of vehicles fueled with methanol blends.[350] Only one home was sampled, but it was sampled multiple times inside the garage and at multiple locations inside the residence. A fourth study from Fairbanks, Alaska conducted measurements in 12-hour periods on four separate days in two houses in two seasons, summer and winter.[351] The study obtained two daily measurements of benzene concentration within each garage over a 12-hour sampling period. One home was a modern, well-insulated home with an air-to-air heat exchanger for ventilation. The other was an older home ventilated passively by structural defects in the building envelope. Because of the large differences in concentrations between homes and seasons, data from each home-season combination was treated as a separate distribution within the indoor air model (Equation 6 in Excel/@Risk). Treating these data as separate distributions increased the number of modeled "scenarios" to 21 (3 f_g x 7 C_g).

Penetration Factor (k)

The values of k in this case were obtained from the HAPEM5 user's manual, using the PEN-1 factor, representing the fraction of benzene from outdoor air penetrating indoors. The values in HAPEM5 are presented as a distribution that assigns a 2/3 weight to the value 0.8 and a 1/3 weight to the value 1. These estimates are based on a comprehensive review of indoor and outdoor air quality studies.

Outdoor Ambient Concentration (C_o)

The 1999 National Air Toxics Assessment (NATA) provided ambient concentration estimates for every census tract in the U.S. For this modeling exercise, a lognormal distribution was fit to these data.

Results

Within-Garage Emission Rates

Equation 4 was used with Monte Carlo sampling to calculate a distribution of emission factors for each home, based on the variability in outdoor concentrations reported in Batterman et al. (2005). As shown in Figure 3-A3, the within-garage variation was a very small component of overall variability compared to between-garage variation. This finding implies that the factors in individual garages, such as storage of vehicles, nonroad equipment, and fuels, have a major effect on in-garage concentrations.

In aggregate, the mean emission rate for all garages sampled fell along a lognormal distribution ($p > 0.05$). The mean emission rate was 3049 μg/hr (73 mg/day), with a standard deviation of 4220 μg/hr (101 mg/day).

To evaluate the plausibility of these steady-state emission factors, known emission factors for other emission sources were evaluated. The California Air Resources Board (CARB) conducted a study of emissions from portable fuel containers, finding that volume-specific emissions rates for total VOC due to evaporation and permeation was 0.37 g/gal-day. Assuming an average fuel container volume of two gallons, the average emission factor per can would be 0.74 g VOC/day.

To evaluate the derived emission rates relative to CARB's measurements, a benzene fuel vapor pressure fraction of 0.5-1% was assumed, based on MOBILE6.2 evaporative emission factors. Given that assumption, the average benzene emission rate from CARB's study is 3.7-7.4 mg/day. This value is in the lower range of emission rates shown in Figure 3A-3. This comparison suggests that emissions due to permeation and evaporation from portable fuel containers may be a relatively small fraction of overall garage benzene.

Subsequently, one additional study used perfluorocarbon tracers (PFT) and VOC measurement in two Fairbanks, Alaska homes to estimate two garages' "source strengths" for benzene.[352] For a new, energy efficient "tight" home with an air-to-air heat exchanger, median garage emission estimates for benzene were 21 mg/h in summer and 14 mg/h in winter. In an older home with passive ventilation due to structural defects, median benzene source strengths were calculated at 40 and 22 mg/h in summer and winter, respectively. These values are substantially higher than those calculated based on Batterman et al. (2005). However, the

difference may be attributable to higher fuel benzene in Fairbanks than in Michigan. Study design may also play a key role. In the Fairbanks study, the measurement periods were 12 hours each in duration. In the Michigan study, measurement periods lasted four days each. The Michigan study's longer duration may have allowed for a broader range of emissions activities than the Fairbanks study.

Garage Contributions to Benzene in Indoor Air

Figures 3A-4 to 3A-8 display the results of @Risk simulations of indoor air. Each figure represents the modeled outputs as cumulative probability distributions. In the legend of each figure, the label of each distribution describes its f_g and C_g sources. For instance, "George et al. (2002) / Fugler FG Ci" indicates a distribution using garage concentration data from George et al. (2002) and f_g data from Fugler et al.

Figure 3A-4 presents the output of Equation 6, a daily average indoor benzene concentration including contributions from outdoor air and from attached garages. As noted in the "Methods" section of this appendix, it was necessary to run a large number of scenarios to account for different combinations of f_g and C_g data sources. The figure depicts results using studies that contain C_g data from multiple homes as bold solid lines, while the model simulations based on studies that employ C_g data from only one home are shown in dashed lines. As indicated in the figure, there is no major difference in the C_i distributions predicted by using C_g data from multiple homes or by using C_g measured from a single home. The average modeled indoor benzene concentrations ranged from 2.9 to 16.4 $\mu g/m^3$.

For comparison, Figure 3A-5 presents cumulative distributions of the observed results from several studies that measured indoor air concentrations in homes with attached garages. Schlapia and Morris (1998) measured integrated 24-hour benzene concentrations inside 91 homes with attached garages in Anchorage, Alaska between 1994 and 1996.[353] George et al. (2002) reported average benzene concentrations in 36 homes in Anchorage, Alaska, but no distributional data. Mentioned above, Isbell et al. (2005) also measured integrated 12-hour benzene in two seasons in one modern air-tight home ("Home V" in Figures 3A-4 to 3A-8) and one older passively-ventilated home ("Home NV" in Figures 3A-4 to 3A-8).[354] Both homes were located in Fairbanks, Alaska. Batterman et al. (2006) measured indoor air benzene concentrations in 15 homes in southeastern Michigan over four-day sampling periods throughout spring and summer of 2005.[355] Lastly, Weisel (2006) conducted a study of indoor air in 21 homes in Union County, NJ between April 2005 and January 2006. One monitor in each home was sited in the room adjacent to the garage, while another was located in another part of the house.[356]

Comparing Figures 3A-4 and 3A-5, it is apparent that the distributions of modeled indoor air concentrations of benzene are very similar to those observed in monitoring studies. Both figures indicate that there is substantial variability in concentrations between homes and between studies.

Figure 3A-6 presents the mean concentrations from modeling scenarios and from monitoring studies. In general, the range of mean concentrations is close to the values monitored in the indoor air studies. Notable exceptions are the indoor air values by George et al. (2002), the winter data from the passively-ventilated "NV" home from Isbell et al. (2005), and by Schlapia and Morris (1998). All of these studies took place in Alaska, which may have uniquely high benzene fuel levels or housing architectures that create higher garage air infiltration indoors.

Of particular note, all of these studies included substantial numbers of homes with "tuck-under" garages where one or more rooms of a house are situated above a garage. Schlapia and Morris (1998) reported a very high average value that was not matched by the "average" conditions of any other run. It is notable that this high value is the average across 91 homes with attached garages.

Another consistent trend shown in Figure 3A-6 is that scenarios employing f_g data from Batterman et al. (2006) produced consistently lower average benzene concentrations than scenarios employing other sources. This trend is attributable to the lower average f_g reported in Batterman et al. (2006), 6.5%, as compared to values found in Sheltersource (11.7%) and Fugler et al. (13.6%).

It is unclear whether the studies measuring C_g, f_g, and C_i constitute a representative sample of homes. In general Alaskan studies report higher concentrations, but not consistently. The relatively greater prevalence of homes with "tuck under" garages in some Alaskan studies may explain this discrepancy.

In comparison to the values reported in Figures 3A-4 and 3A-5, indoor air concentrations calculated with the default $C_i = kC_o$ approach, similar to that employed in the national-scale modeling for this rule, averaged 1.2 $\mu g/m^3$.

Overall, modeled concentrations presented here appear to provide a credible estimate of indoor benzene concentration in homes with attached garages. However, it is unclear whether the homes included in the studies employed herein may be considered "representative."

Implications

Effect on Exposures Nationwide

In calculating the hypothetical effect of attached garage on national estimates of chronic, time-weighted average (TWA) human exposure, precise estimates are not possible. As noted previously, the extent to which available studies of indoor air of homes with attached garages is representative of the entire population of such homes is unclear. Furthermore, the distribution of housing stock by climate and meteorology is not well understood. However, despite these limitations, a bounding exercise is still feasible.

One simple approach for such a bounding exercise is determined by the following equation:

$$(8) \qquad E_g = C_{i,g} * P_g * T_g$$

Here, E_g represents the national average exposure to benzene in air attributable to attached garages. $C_{i,g}$ represents the average indoor concentration attributable to an attached garage, P_g represents the fraction of the population living in a home with an attached garage, and T_g represents the time spent in a home with an attached garage.

$C_{i,g}$ is derived from Equation 6, and can be derived by setting the outdoor concentration term (C_o) to zero. An estimate of the attached garage contribution to indoor air can be made for studies with only indoor measurements as well. This can be accomplished by substituting ASPEN concentration estimates for the county in which each study took place. For Equation 6, C_o estimates from NATA for each census tract in the relevant county were assembled into a lognormal distribution. With this data and the other assumptions of Equation 6, an estimate of $C_{i,g}$ could be derived from the measurement studies.

To estimate P_g, an estimate of the national fraction of homes with attached garages is required. The Residential Energy Consumption Survey (RECS), run by the U.S. Energy Information Administration, provides an estimate of the fraction of homes with attached garages.[357] RECS estimates a total of 107.0 million housing units nationally, 37.1 million (34.7%) of which are homes with attached garages. Assuming that the population is uniformly distributed across housing units allows this figure to serve as an estimate of P_g.

Information on the fraction of time spent in a residence (T_g) is required to determine how the microenvironmental concentration in homes with attached garages affects overall time-weighted exposure concentrations. As cited in EPA's Exposure Factors Handbook, the average person studied by the National Human Activity Pattern Survey (NHAPS) spent 1001.39 minutes (16.68 hours) per day indoors within any room of a residence.[358]

Results of model simulations using Equation 7 are shown in Figure 3A-7. As before, the results of each combination of C_g and f_g data source are shown. For each study, the legend lists the source for both C_g and f_g data. As described above, the estimates $C_{i,g}$ derived from indoor air measurements are also presented in Figure 3A-7. In the legend of Figure 3A-6, these studies are denoted by the term "Direct Ci Measure." As shown, there tends to be a greater degree of agreement between modeling scenarios for lower concentration estimates, but less agreement for higher concentration estimates.

As described above, it is unclear to what the extent to which the homes studied for benzene related to attached garages are representative of homes nationally. As such, in summarizing the scenarios, several different approaches to "averaging" across scenarios are presented here. Figure 3A-8 shows the results of these different averaging scenarios. In the "All Data" distribution shown in the figure, all scenarios are averaged together. In the "Weighted Average" distribution, weights are equal to the number of homes included in each study. In the "Model Only" distribution, only scenarios involving modeling C_i are shown. In the "Measure Only" distribution, only those studies in which C_i was measured directly are shown. In the "AK Only" distribution, only scenarios employing Alaskan C_g or f_g studies are shown. In the "Non-AK Only" distribution, only scenarios excluding Alaskan C_g or f_g data are shown. These scenarios are intended to span a range of estimates for the national estimate.

The average concentrations from these "summary scenarios" are shown in Table 3A-1. As shown in Table 3A-1 and in Figure 3A-8, scenarios employing only measured indoor data resulted in higher predicted benzene TWA exposure concentrations than the studies employing only modeling. Scenarios employing Alaskan data result in higher benzene concentrations than scenarios excluding Alaskan data. Also weighting scenarios by the number of homes resulted in higher benzene concentrations.

Accordingly, the national average TWA exposure concentration attributable to attached garages is estimated to be 1.2 – 6.6 $\mu g/m^3$. This range is intended to span possible values of average TWA exposure from attached garages, given currently available information. The actual average TWA exposure concentration due to attached garages could be outside of this range. Because of limited information on the representativeness of the homes studied, a more precise "central estimate" is not appropriate at this time. The width of the range, with the upper end being 5.5 times the lower end, is an indicator of the magnitude of uncertainty in the estimate. It is not a confidence interval in the traditional sense. As more data become available, more precise estimates will hopefully emerge.

In comparison, the national average exposure concentration of census tract median exposure concentrations in this rule is estimated at 1.4 $\mu g/m^3$ for calendar year 1999.

Accordingly, if the attached garage exposure contribution is considered, the estimate of national average exposure to benzene rises to 2.6 – 8.0 μg/m^3, corresponding to an increase of 85-471%.

Effects of Emission Standards

Several limitations prevent precise estimation of the effect of the standards in this rule on garage-related exposures. First, cold temperature vehicle ignition and evaporative vehicle, engine, and fuel container emissions can occur either in a garage or outdoors. Second, detailed tracking of the time during which people are inside a house during cold vehicle starting or hot vehicle soaking, when a majority of benzene emissions are likely to occur, is limited. However, a bounding exercise can provide some estimates as to the effect of the standards in this rule.

First, assuming full mixing and steady-state conditions, concentrations within a garage is estimable[359] as:

$$C_g = (dM_i/dt) / \alpha V \quad (9)$$

Here, the terms are similar to Equations 1-7.

Given a change in the mass benzene emission rates from vehicle cold temperature ignition, fuel evaporation from vehicles, engines, and fuel containers, an estimate of a change in C_g is feasible. Table 2.2-52 of the RIA displays the emission reductions attributable to each program. By splitting the emission reductions into evaporative and exhaust emissions and applying several simple assumptions about where emissions occur (in garage vs. outdoors), the fraction of emission reductions occurring within attached garages can be estimated. This estimate is calculated by assuming ranges of values for the fraction of evaporative and exhaust emissions from each program that occur within an attached garage.[u] As such, while the total benzene mobile source and PFC emission reductions occurring as a result of the rule in 2030 are 37% less than the projected emissions without controls (Table 2.2-52 of the RIA), emissions inside attached garages are reduced by an estimated 43-44%.

Applying this fraction to Equation 8 and Equation 7, for the "average" scenarios modeled presented in Table 1, this amounts to a national average exposure reduction of approximately 0.5 – 2.6 μg/m^3.

Limitations

As apparent in the wide range of "scenario" averages, there remains considerable uncertainty in ascertaining the true magnitude of attached garage exposure contributions nationally. There are a number of limitations in the approaches undertaken here. First, although comparison with measured indoor data shows reasonable performance for the modeling approach employed here, the selection of simple one-compartment mass balance models for both garage and home modeling may substantially understate the variation in concentrations within these microenvironments. All estimates here assumed steady-state conditions, and this may not be

[u] The assumed fraction of evaporative and exhaust emission reductions from each source occurring within an attached garage are as follows. Ranges are represented as [min, max]. For LDGV, about 90% of emission reductions are exhaust-related, of which P_g*[25%,75%] occur within attached garages; the fraction of evaporative reductions occurring within attached garages are P_g *[25%,50%]. For small nonroad gasoline equipment, about 72% of emissions are from exhaust, of which P_g *[0%,2%] occur in attached garages; 24% are evaporative, of which P_g *[90%,100%] occur in attached garages, 4% are refilling-related, of which P_g *[25%,75%] occur in attached garages. For portable fuel containers, P_g *[25%,75%] of emissions are assumed to occur in attached garages.

appropriate for a source like a garage, where door opening, car entry and ignition, and other major sources of benzene are likely to produce short-term spikes in exposure not accounted for with steady-state assumptions.

Second, the preponderance of these data were collected in locations with cold climates, so the results may not be applicable to warmer locations where houses are not built with the same degree of weather-tightness. Furthermore, studies suggest that indoor concentrations arising from attached garages vary considerably in response to emission-related activities in a garage such as cold vehicle ignition and parking a hot vehicle.[360] Ambient temperatures may affect the magnitude of emissions from these activities.

Lastly, the extent to which the houses studied in the publications cited here are "representative" of the national housing stock is unknown.

Conclusions

Modeled indoor benzene concentrations indicate that indoor air concentrations in homes with attached garages may be substantially higher than in homes without attached garages.

Garage concentrations of benzene appear to be a major source of indoor benzene in homes with attached garages. According to the modeling conducted here, this source could explain the majority of exposures experienced by typical residents of such homes. Given this finding, interventions that result in a reduction in emissions within the garage would be a relatively efficient means of reducing overall personal exposure, particularly in areas geographically similar to the areas of the studies upon which this analysis relies. Given the proximity of this source to homes, one major set of beneficiaries of the rule's emission controls is likely to be people with homes with attached garages, particularly in areas with high fuel benzene levels. Emissions from vehicles and fuel containers also may have greater relative impacts on those with attached garages. An elementary calculation of the intake fraction (iF) of emissions occurring within attached garages with very basic assumptions indicates that for benzene emitted in a garage, approximately 3-18 parts per thousand are inhaled by a person in an attached garage. This estimate is far in excess of estimated iF from ambient sources, and similar to estimated iF estimates for indoor sources.[361]

Table 3A-1. Summary of National Average Exposure Estimates Attributable to Attached Garages. Different "averaging" assumptions shown.

"Averaging" Scenario	Benzene TWA (ug/m3)
All Data	4.3
Weighted Average	6.6
Measure Only	6.1
Model Only	3.4
AK Only	5.5
Non-AK Only	1.2

Figure 3A-1a. Density of Garage Benzene Concentrations from Batterman et al. (2005)

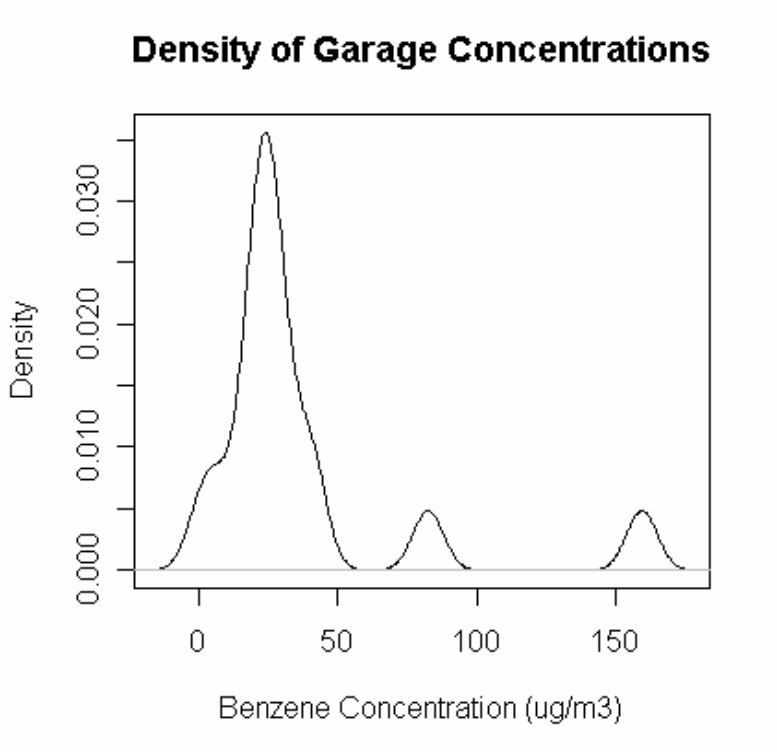


Figure 3A-1b. Density of Air Exchange Rates (ACH)

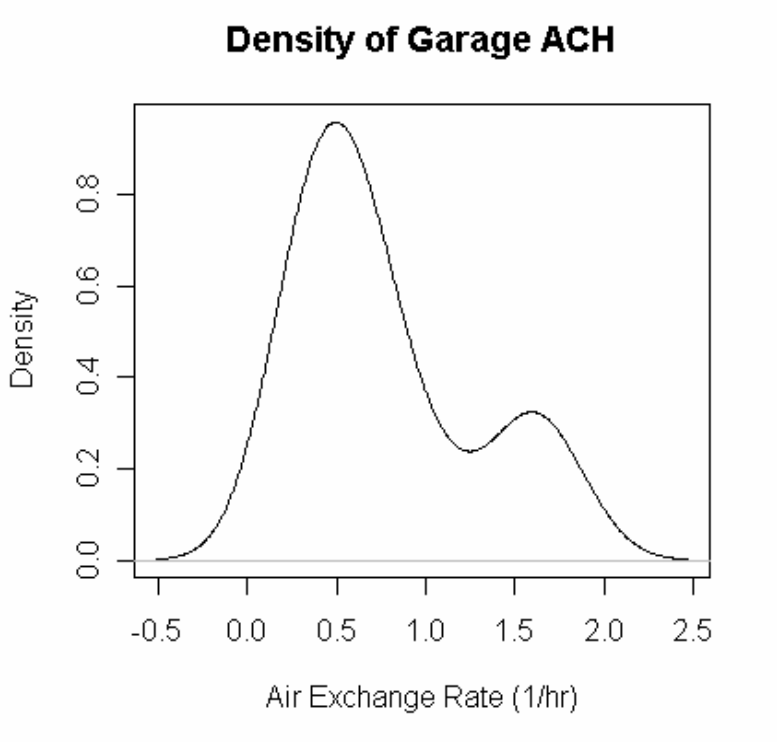


Figure 3A-2. Additional Indoor Air Concentrations from Garage as a Function of C_g and f_g

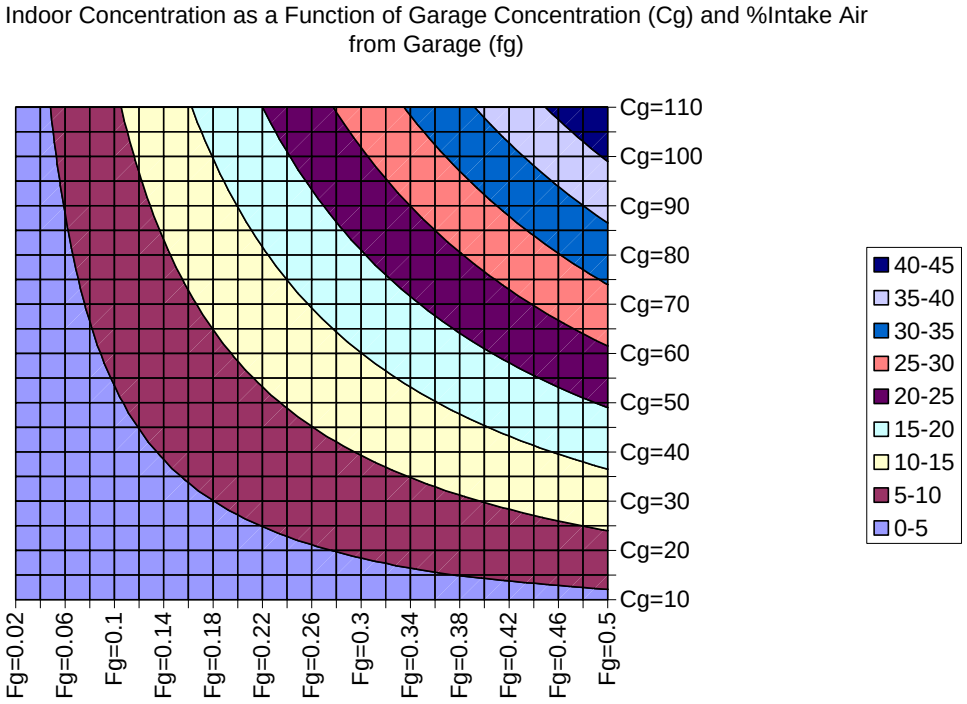


Figure 3A-3. Distributions of Individual Garage Emission Factors

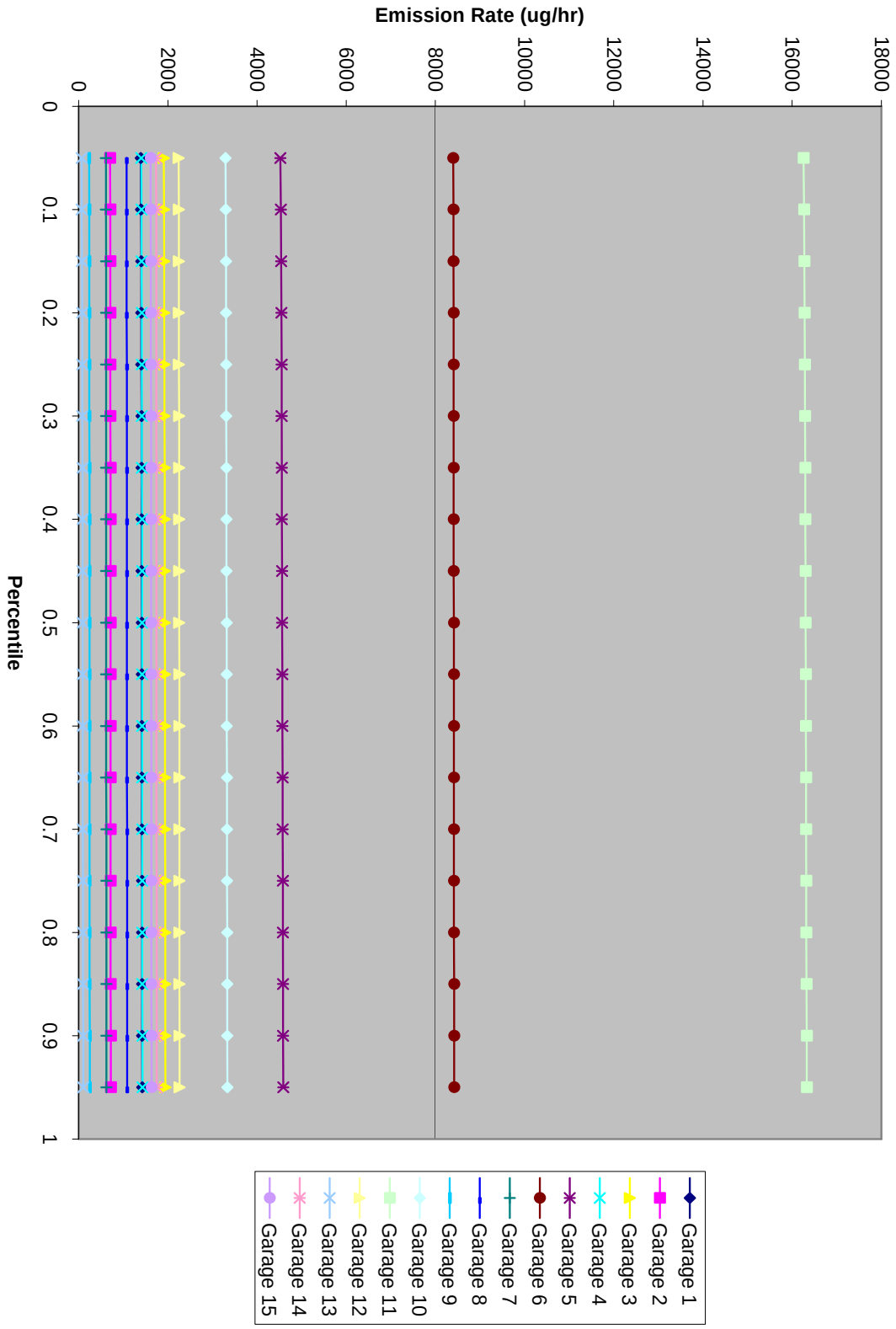


Figure 3A-4. Cumulative Distribution of Modeled Indoor Benzene Concentrations

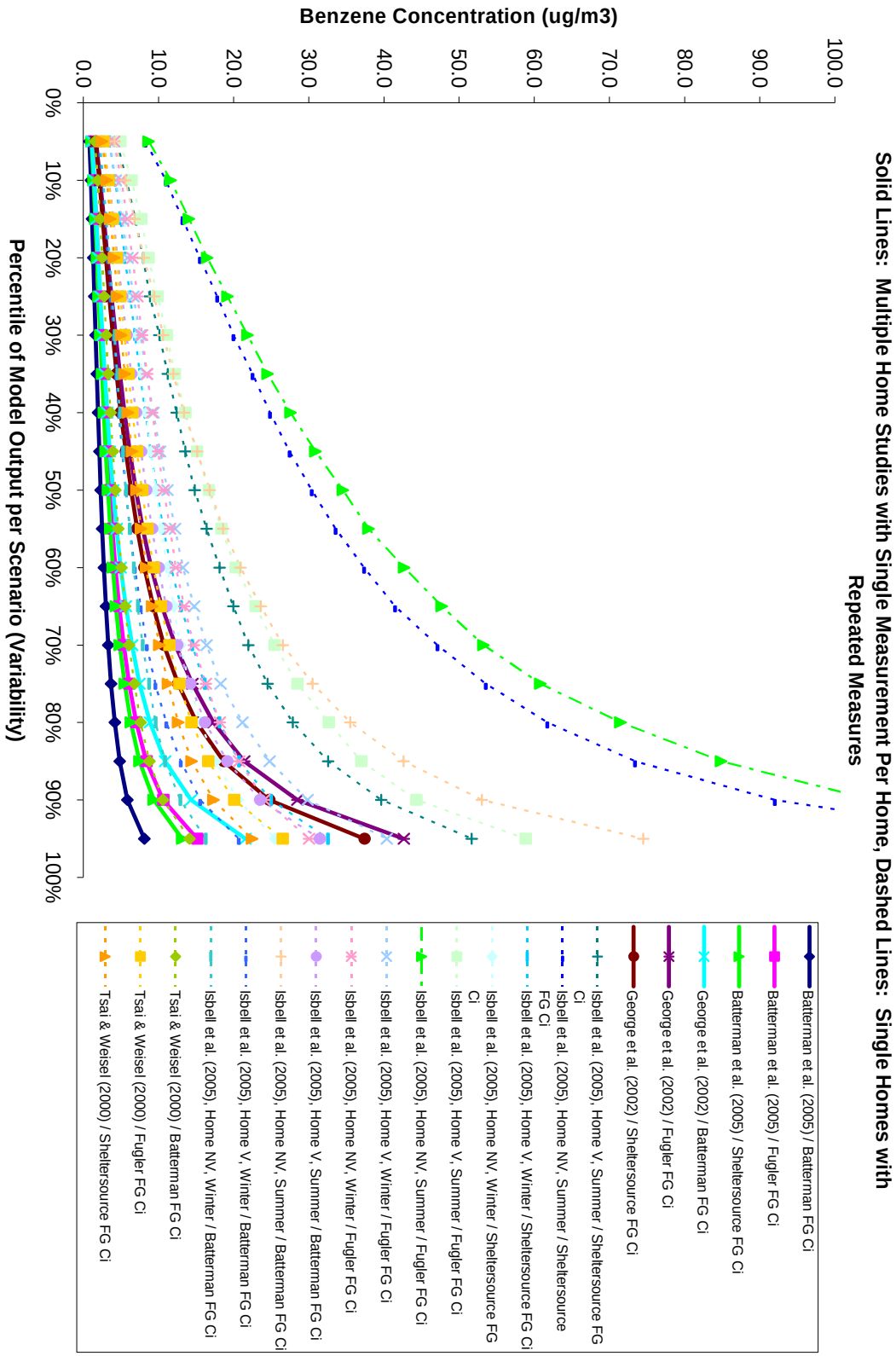


Figure 3A-5. Cumulative Distributions of Observed Benzene Levels in Homes with Attached Garages

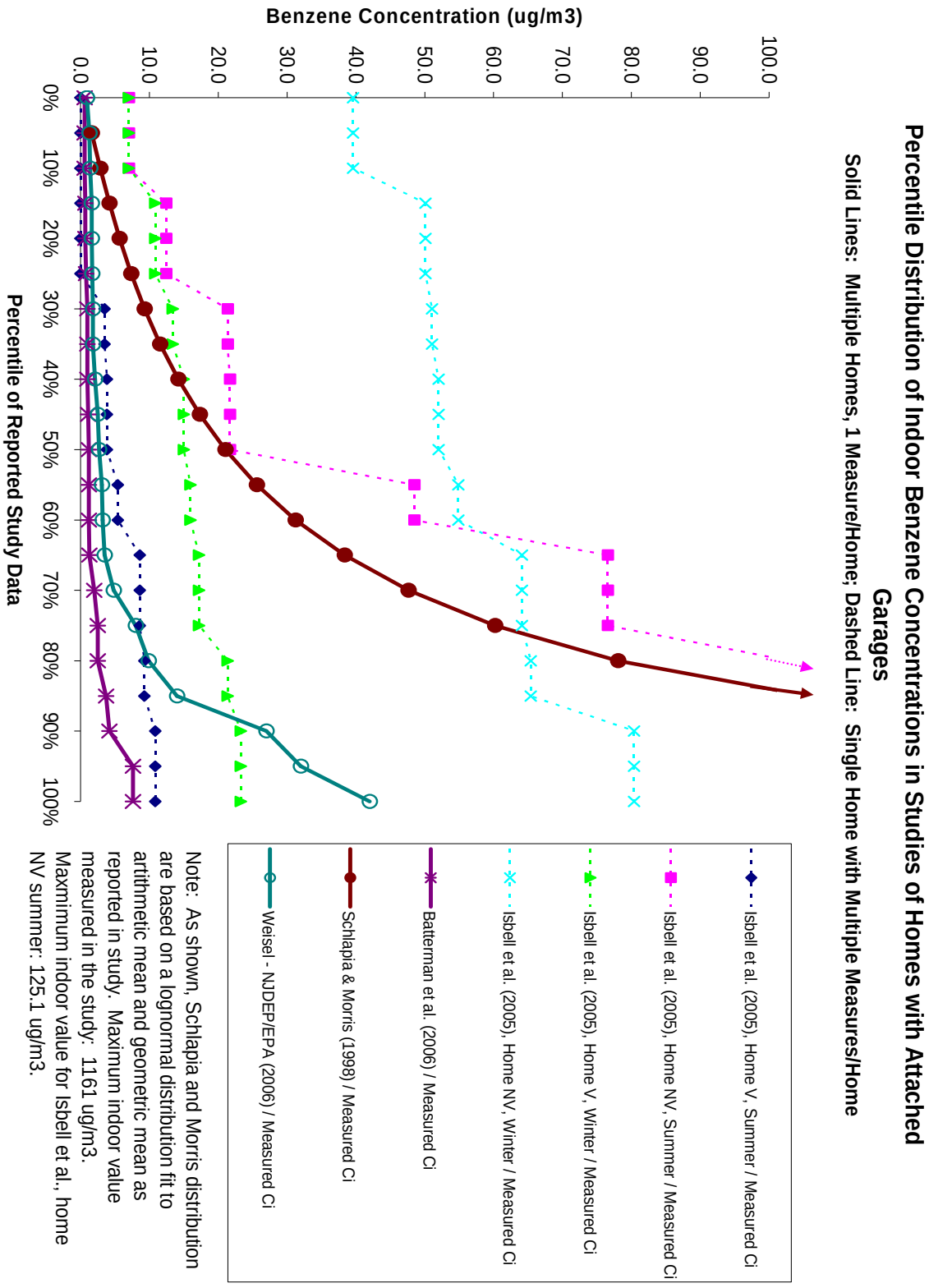


Figure 3A-6. Comparison of Modeled and Observed Indoor Benzene Concentrations

Figure 3A-7. Multiple Scenario Output of Predicted National Average Benzene Exposure Attributable to Attached Garages

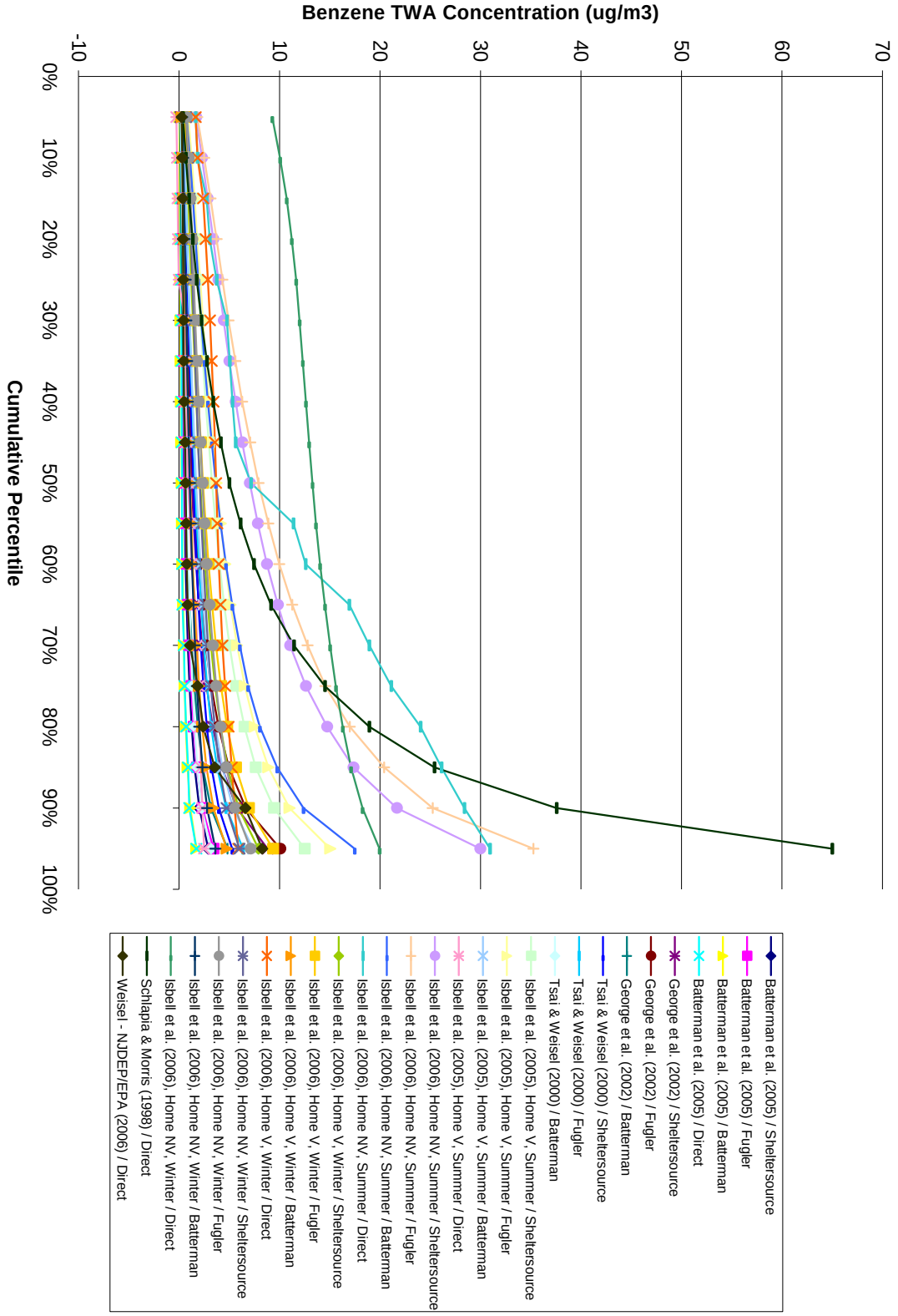
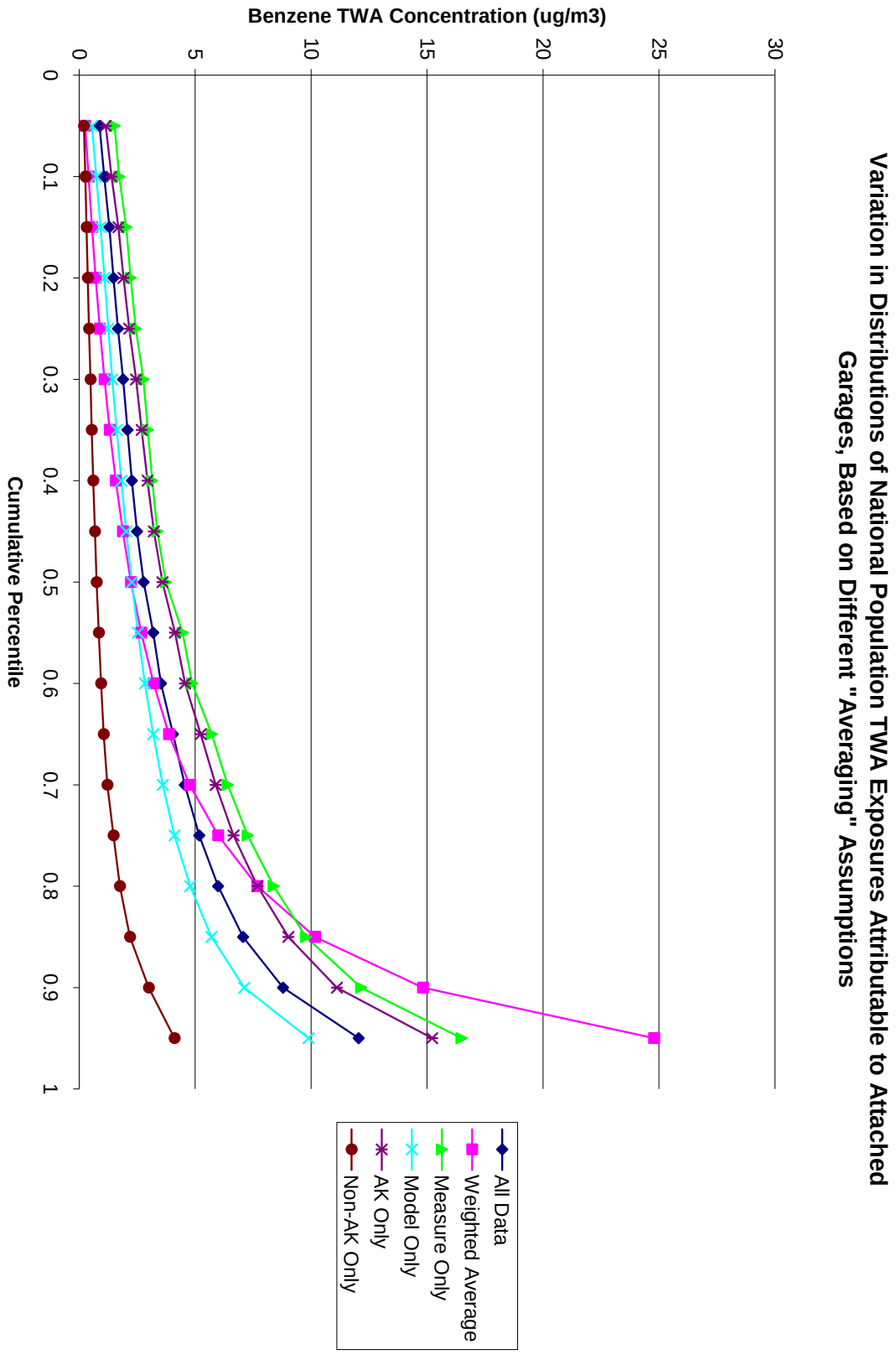


Figure 3A-8. Average “Summarized” Benzene Exposure Distributions



Appendix 3B: 8-Hour Ozone Nonattainment

Table 3B-1. 8-Hour Ozone Nonattainment Areas, Counties and Populations (Data is Current through October 2006 and Population Numbers are from 2000 Census Data)

8-hour Ozone Nonattainment Area	State	Classification[a,b]	County Name	Whole /Part	2000 Cty Pop
Albany-Schenectady-Troy Area	NY	Subpart 1	Albany Co	W	294,565
Albany-Schenectady-Troy Area	NY	Subpart 1	Greene Co	W	48,195
Albany-Schenectady-Troy Area	NY	Subpart 1	Montgomery Co	W	49,708
Albany-Schenectady-Troy Area	NY	Subpart 1	Rensselaer Co	W	152,538
Albany-Schenectady-Troy Area	NY	Subpart 1	Saratoga Co	W	200,635
Albany-Schenectady-Troy Area	NY	Subpart 1	Schenectady Co	W	146,555
Albany-Schenectady-Troy Area	NY	Subpart 1	Schoharie Co	W	31,582
Allegan County Area	MI	Subpart 1	Allegan Co	W	105,665
Allentown-Bethlehem-Easton Area	PA	Subpart 1	Carbon Co	W	58,802
Allentown-Bethlehem-Easton Area	PA	Subpart 1	Lehigh Co	W	312,090
Allentown-Bethlehem-Easton Area	PA	Subpart 1	Northampton Co	W	267,066
Altoona Area	PA	Subpart 1	Blair Co	W	129,144
Amador and Calaveras Counties (Central Mountain Counties) Area	CA	Subpart 1	Amador Co	W	35,100
Amador and Calaveras Counties (Central Mountain Counties) Area	CA	Subpart 1	Calaveras Co	W	40,554
Atlanta Area	GA	Subpart 2/Marginal	Barrow Co	W	46,144
Atlanta Area	GA	Subpart 2/Marginal	Bartow Co	W	76,019
Atlanta Area	GA	Subpart 2/Marginal	Carroll Co	W	87,268
Atlanta Area	GA	Subpart 2/Marginal	Cherokee Co	W	141,903
Atlanta Area	GA	Subpart 2/Marginal	Clayton Co	W	236,517
Atlanta Area	GA	Subpart 2/Marginal	Cobb Co	W	607,751
Atlanta Area	GA	Subpart 2/Marginal	Coweta Co	W	89,215
Atlanta Area	GA	Subpart 2/Marginal	De Kalb Co	W	665,865
Atlanta Area	GA	Subpart 2/Marginal	Douglas Co	W	92,174
Atlanta Area	GA	Subpart 2/Marginal	Fayette Co	W	91,263
Atlanta Area	GA	Subpart 2/Marginal	Forsyth Co	W	98,407
Atlanta Area	GA	Subpart 2/Marginal	Fulton Co	W	816,006
Atlanta Area	GA	Subpart 2/Marginal	Gwinnett Co	W	588,448
Atlanta Area	GA	Subpart 2/Marginal	Hall Co	W	139,277

Atlanta Area	GA	Subpart 2/Marginal	Henry Co	W	119,341
Atlanta Area	GA	Subpart 2/Marginal	Newton Co	W	62,001
Atlanta Area	GA	Subpart 2/Marginal	Paulding Co	W	81,678
Atlanta Area	GA	Subpart 2/Marginal	Rockdale Co	W	70,111
Atlanta Area	GA	Subpart 2/Marginal	Spalding Co	W	58,417
Atlanta Area	GA	Subpart 2/Marginal	Walton Co	W	60,687
Baltimore Area	MD	Subpart 2/Moderate	Anne Arundel Co	W	489,656
Baltimore Area	MD	Subpart 2/Moderate	Baltimore (City)	W	651,154
Baltimore Area	MD	Subpart 2/Moderate	Baltimore Co	W	754,292
Baltimore Area	MD	Subpart 2/Moderate	Carroll Co	W	150,897
Baltimore Area	MD	Subpart 2/Moderate	Harford Co	W	218,590
Baltimore Area	MD	Subpart 2/Moderate	Howard Co	W	247,842
Baton Rouge Area	LA	Subpart 2/Marginal	Ascension Par	W	76,627
Baton Rouge Area	LA	Subpart 2/Marginal	East Baton Rouge Par	W	412,852
Baton Rouge Area	LA	Subpart 2/Marginal	Iberville Par	W	33,320
Baton Rouge Area	LA	Subpart 2/Marginal	Livingston Par	W	91,814
Baton Rouge Area	LA	Subpart 2/Marginal	West Baton Rouge Par	W	21,601
Beaumont-Port Arthur Area	TX	Subpart 2/Marginal	Hardin Co	W	48,073
Beaumont-Port Arthur Area	TX	Subpart 2/Marginal	Jefferson Co	W	252,051
Beaumont-Port Arthur Area	TX	Subpart 2/Marginal	Orange Co	W	84,966
Benton Harbor Area	MI	Subpart 1	Berrien Co	W	162,453
Benzie County Area	MI	Subpart 1	Benzie Co	W	15,998
Berkeley and Jefferson Counties Area	WV	Subpart 1 - EAC	Berkeley Co	W	75,905
Berkeley and Jefferson Counties Area	WV	Subpart 1 - EAC	Jefferson Co	W	42,190
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Barnstable Co	W	222,230
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Bristol Co	W	534,678
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Dukes Co	W	14,987
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Essex Co	W	723,419

Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Middlesex Co	W	1,465,396
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Nantucket Co	W	9,520
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Norfolk Co	W	650,308
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Plymouth Co	W	472,822
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Suffolk Co	W	689,807
Boston-Lawrence-Worcester (E. Mass) Area	MA	Subpart 2/Moderate	Worcester Co	W	750,963
Boston-Manchester-Portsmouth (SE) Area	NH	Subpart 2/Moderate	Hillsborough Co	P	336,518
Boston-Manchester-Portsmouth (SE) Area	NH	Subpart 2/Moderate	Merrimack Co	P	11,721
Boston-Manchester-Portsmouth (SE) Area	NH	Subpart 2/Moderate	Rockingham Co	P	266,340
Boston-Manchester-Portsmouth (SE) Area	NH	Subpart 2/Moderate	Strafford Co	P	82,134
Buffalo-Niagara Falls Area	NY	Subpart 1	Erie Co	W	950,265
Buffalo-Niagara Falls Area	NY	Subpart 1	Niagara Co	W	219,846
Canton-Massillon Area	OH	Subpart 1	Stark Co	W	378,098
Cass County Area	MI	Subpart 2/Marginal	Cass Co	W	51,104
Charlotte-Gastonia-Rock Hill Area	NC	Subpart 2/Moderate	Cabarrus Co	W	131,063
Charlotte-Gastonia-Rock Hill Area	NC	Subpart 2/Moderate	Gaston Co	W	190,365
Charlotte-Gastonia-Rock Hill Area	NC	Subpart 2/Moderate	Iredell Co	P	39,885
Charlotte-Gastonia-Rock Hill Area	NC	Subpart 2/Moderate	Lincoln Co	W	63,780
Charlotte-Gastonia-Rock Hill Area	NC	Subpart 2/Moderate	Mecklenburg Co	W	695,454
Charlotte-Gastonia-Rock Hill Area	NC	Subpart 2/Moderate	Rowan Co	W	130,340
Charlotte-Gastonia-Rock Hill Area	NC	Subpart 2/Moderate	Union Co	W	123,677
Charlotte-Gastonia-Rock Hill Area	SC	Subpart 2/Moderate	York Co	P	102,000
Chattanooga Area	GA	Subpart 1 - EAC	Catoosa Co	W	53,282
Chattanooga Area	TN	Subpart 1 - EAC	Hamilton Co	W	307,896
Chattanooga Area	TN	Subpart 1 - EAC	Meigs Co	W	11,086
Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Cook Co	W	5,376,741
Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Du Page Co	W	904,161
Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Grundy Co	P	6,309
Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Kane Co	W	404,119
Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Kendall Co	P	28,417

Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Lake Co	W	644,356
Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Mc Henry Co	W	260,077
Chicago-Gary-Lake County Area	IL	Subpart 2/Moderate	Will Co	W	502,266
Chicago-Gary-Lake County Area	IN	Subpart 2/Moderate	Lake Co	W	484,564
Chicago-Gary-Lake County Area	IN	Subpart 2/Moderate	Porter Co	W	146,798
Chico Area	CA	Subpart 1	Butte Co	W	203,171
Cincinnati-Hamilton Area	IN	Subpart 1	Dearborn Co	P	10,434
Cincinnati-Hamilton Area	KY	Subpart 1	Boone Co	W	85,991
Cincinnati-Hamilton Area	KY	Subpart 1	Campbell Co	W	88,616
Cincinnati-Hamilton Area	KY	Subpart 1	Kenton Co	W	151,464
Cincinnati-Hamilton Area	OH	Subpart 1	Butler Co	W	332,807
Cincinnati-Hamilton Area	OH	Subpart 1	Clermont Co	W	177,977
Cincinnati-Hamilton Area	OH	Subpart 1	Clinton Co	W	40,543
Cincinnati-Hamilton Area	OH	Subpart 1	Hamilton Co	W	845,303
Cincinnati-Hamilton Area	OH	Subpart 1	Warren Co	W	158,383
Clearfield and Indiana Counties Area	PA	Subpart 1	Clearfield Co	W	83,382
Clearfield and Indiana Counties Area	PA	Subpart 1	Indiana Co	W	89,605
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Ashtabula Co	W	102,728
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Cuyahoga Co	W	1,393,978
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Geauga Co	W	90,895
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Lake Co	W	227,511
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Lorain Co	W	284,664
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Medina Co	W	151,095
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Portage Co	W	152,061
Cleveland-Akron-Lorain Area	OH	Subpart 2/Moderate	Summit Co	W	542,899
Columbia Area	SC	Subpart 1 - EAC	Lexington Co	P	181,265
Columbia Area	SC	Subpart 1 - EAC	Richland Co	P	313,253
Columbus Area	OH	Subpart 1	Delaware Co	W	109,989
Columbus Area	OH	Subpart 1	Fairfield Co	W	122,759
Columbus Area	OH	Subpart 1	Franklin Co	W	1,068,978
Columbus Area	OH	Subpart 1	Knox Co	W	54,500
Columbus Area	OH	Subpart 1	Licking Co	W	145,491
Columbus Area	OH	Subpart 1	Madison Co	W	40,213
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Collin Co	W	491,675
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Dallas Co	W	2,218,899

Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Denton Co	W	432,976
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Ellis Co	W	111,360
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Johnson Co	W	126,811
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Kaufman Co	W	71,313
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Parker Co	W	88,495
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Rockwall Co	W	43,080
Dallas-Fort Worth Area	TX	Subpart 2/Moderate	Tarrant Co	W	1,446,219
Dayton-Springfield Area	OH	Subpart 1	Clark Co	W	144,742
Dayton-Springfield Area	OH	Subpart 1	Greene Co	W	147,886
Dayton-Springfield Area	OH	Subpart 1	Miami Co	W	98,868
Dayton-Springfield Area	OH	Subpart 1	Montgomery Co	W	559,062
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Adams Co	W	348,618
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Arapahoe Co	W	487,967
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Boulder Co	W	269,814
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Broomfield Co	W	38,272
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Denver Co	W	554,636
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Douglas Co	W	175,766
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Jefferson Co	W	525,507
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Larimer Co	P	239,000
Denver-Boulder-Greeley-Ft. Collins-Love. Area	CO	Subpart 1 - EAC	Weld Co	P	172,000
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	Lenawee Co	W	98,890
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	Livingston Co	W	156,951
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	Macomb Co	W	788,149
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	Monroe Co	W	145,945
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	Oakland Co	W	1,194,156
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	St Clair Co	W	164,235
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	Washtenaw Co	W	322,895
Detroit-Ann Arbor Area	MI	Subpart 2/Marginal	Wayne Co	W	2,061,162
Door County Area	WI	Subpart 1	Door Co	W	27,961
Erie Area	PA	Subpart 1	Erie Co	W	280,843

Essex County (Whiteface Mtn.) Area	NY	Subpart 1	Essex Co	P	1,000
Fayetteville Area	NC	Subpart 1 - EAC	Cumberland Co	W	302,963
Flint Area	MI	Subpart 1	Genesee Co	W	436,141
Flint Area	MI	Subpart 1	Lapeer Co	W	87,904
Fort Wayne Area	IN	Subpart 1	Allen Co	W	331,849
Franklin County Area	PA	Subpart 1	Franklin Co	W	129,313
Frederick County Area	VA	Subpart 1 - EAC	Frederick Co	W	59,209
Frederick County Area	VA	Subpart 1 - EAC	Winchester	W	23,585
Grand Rapids Area	MI	Subpart 1	Kent Co	W	574,335
Grand Rapids Area	MI	Subpart 1	Ottawa Co	W	238,314
Greater Connecticut Area	CT	Subpart 2/Moderate	Hartford Co	W	857,183
Greater Connecticut Area	CT	Subpart 2/Moderate	Litchfield Co	W	182,193
Greater Connecticut Area	CT	Subpart 2/Moderate	New London Co	W	259,088
Greater Connecticut Area	CT	Subpart 2/Moderate	Tolland Co	W	136,364
Greater Connecticut Area	CT	Subpart 2/Moderate	Windham Co	W	109,091
Greene County Area	PA	Subpart 1	Greene Co	W	40,672
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Alamance Co	W	130,800
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Caswell Co	W	23,501
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Davidson Co	W	147,246
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Davie Co	W	34,835
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Forsyth Co	W	306,067
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Guilford Co	W	421,048
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Randolph Co	W	130,454
Greensboro-Winston-Salem-High Point Area	NC	Subpart 2/Marginal - EAC	Rockingham Co	W	91,928
Greenville-Spartanburg-Anderson Area	SC	Subpart 1 - EAC	Anderson Co	W	165,740
Greenville-Spartanburg-Anderson Area	SC	Subpart 1 - EAC	Greenville Co	W	379,616
Greenville-Spartanburg-Anderson Area	SC	Subpart 1 - EAC	Spartanburg Co	W	253,791
Hancock, Knox, Lincoln and Waldo	ME	Subpart 1	Hancock Co	P	29,805

Counties (Central Maine Coast) Area					
Hancock, Knox, Lincoln and Waldo Counties (Central Maine Coast) Area	ME	Subpart 1	Knox Co	P	33,563
Hancock, Knox, Lincoln and Waldo Counties (Central Maine Coast) Area	ME	Subpart 1	Lincoln Co	P	28,504
Hancock, Knox, Lincoln and Waldo Counties (Central Maine Coast) Area	ME	Subpart 1	Waldo Co	P	604
Harrisburg-Lebanon-Carlisle Area	PA	Subpart 1	Cumberland Co	W	213,674
Harrisburg-Lebanon-Carlisle Area	PA	Subpart 1	Dauphin Co	W	251,798
Harrisburg-Lebanon-Carlisle Area	PA	Subpart 1	Lebanon Co	W	120,327
Harrisburg-Lebanon-Carlisle Area	PA	Subpart 1	Perry Co	W	43,602
Haywood and Swain Counties (Great Smoky NP) Area	NC	Subpart 1	Haywood Co	P	28
Haywood and Swain Counties (Great Smoky NP) Area	NC	Subpart 1	Swain Co	P	260
Hickory-Morganton-Lenoir Area	NC	Subpart 1 - EAC	Alexander Co	W	33,603
Hickory-Morganton-Lenoir Area	NC	Subpart 1 - EAC	Burke Co	P	69,970
Hickory-Morganton-Lenoir Area	NC	Subpart 1 - EAC	Caldwell Co	P	64,254
Hickory-Morganton-Lenoir Area	NC	Subpart 1 - EAC	Catawba Co	W	141,685
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Brazoria Co	W	241,767
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Chambers Co	W	26,031
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Fort Bend Co	W	354,452
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Galveston Co	W	250,158
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Harris Co	W	3,400,578
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Liberty Co	W	70,154
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Montgomery Co	W	293,768
Houston-Galveston-Brazoria Area	TX	Subpart 2/Moderate	Waller Co	W	32,663
Huntington-Ashland Area	KY	Subpart 1	Boyd Co	W	49,752
Huron County Area	MI	Subpart 1	Huron Co	W	36,079
Imperial County Area	CA	Subpart 2/Marginal	Imperial Co	W	142,361
Indianapolis Area	IN	Subpart 1	Boone Co	W	46,107
Indianapolis Area	IN	Subpart 1	Hamilton Co	W	182,740
Indianapolis Area	IN	Subpart 1	Hancock Co	W	55,391
Indianapolis Area	IN	Subpart 1	Hendricks Co	W	104,093
Indianapolis Area	IN	Subpart 1	Johnson Co	W	115,209
Indianapolis Area	IN	Subpart 1	Madison Co	W	133,358
Indianapolis Area	IN	Subpart 1	Marion Co	W	860,454
Indianapolis Area	IN	Subpart 1	Morgan Co	W	66,689
Indianapolis Area	IN	Subpart 1	Shelby Co	W	43,445

Jamestown Area	NY	Subpart 1	Chautauqua Co	W	139,750
Jefferson County Area	NY	Subpart 2/Moderate	Jefferson Co	W	111,738
Johnson City-Kingsport-Bristol Area	TN	Subpart 1 - EAC	Hawkins Co	W	53,563
Johnson City-Kingsport-Bristol Area	TN	Subpart 1 - EAC	Sullivan Co	W	153,048
Johnstown Area	PA	Subpart 1	Cambria Co	W	152,598
Kalamazoo-Battle Creek Area	MI	Subpart 1	Calhoun Co	W	137,985
Kalamazoo-Battle Creek Area	MI	Subpart 1	Kalamazoo Co	W	238,603
Kalamazoo-Battle Creek Area	MI	Subpart 1	Van Buren Co	W	76,263
Kent and Queen Anne's Counties Area	MD	Subpart 2/Marginal	Kent Co	W	19,197
Kent and Queen Anne's Counties Area	MD	Subpart 2/Marginal	Queen Annes Co	W	40,563
Kern County (Eastern Kern) Area	CA	Subpart 1	Kern Co	P	99,251
Kewaunee County Area	WI	Subpart 1	Kewaunee Co	W	20,187
Knoxville Area	TN	Subpart 1	Anderson Co	W	71,330
Knoxville Area	TN	Subpart 1	Blount Co	W	105,823
Knoxville Area	TN	Subpart 1	Cocke Co	P	20
Knoxville Area	TN	Subpart 1	Jefferson Co	W	44,294
Knoxville Area	TN	Subpart 1	Knox Co	W	382,032
Knoxville Area	TN	Subpart 1	Loudon Co	W	39,086
Knoxville Area	TN	Subpart 1	Sevier Co	W	71,170
La Porte County Area	IN	Subpart 2/Marginal	La Porte Co	W	110,106
Lancaster Area	PA	Subpart 2/Marginal	Lancaster Co	W	470,658
Lansing-East Lansing Area	MI	Subpart 1	Clinton Co	W	64,753
Lansing-East Lansing Area	MI	Subpart 1	Eaton Co	W	103,655
Lansing-East Lansing Area	MI	Subpart 1	Ingham Co	W	279,320
Las Vegas Area	NV	Subpart 1	Clark Co	P	1,348,864
Lima Area	OH	Subpart 1	Allen Co	W	108,473
Los Angeles and San Bernardino Counties (W Mojave Desert) Area	CA	Subpart 2/Moderate	Los Angeles Co	P	297,058
Los Angeles and San Bernardino Counties (W Mojave Desert) Area	CA	Subpart 2/Moderate	San Bernardino Co	P	359,350
Los Angeles-South Coast Air Basin Area	CA	Subpart 2/Severe 17	Los Angeles Co	P	9,222,280
Los Angeles-South Coast Air Basin Area	CA	Subpart 2/Severe 17	Orange Co	W	2,846,289
Los Angeles-South Coast Air Basin Area	CA	Subpart 2/Severe 17	Riverside Co	P	1,194,859
Los Angeles-South Coast Air Basin Area	CA	Subpart 2/Severe 17	San Bernardino Co	P	1,330,159
Louisville Area	IN	Subpart 1	Clark Co	W	96,472
Louisville Area	IN	Subpart 1	Floyd Co	W	70,823
Louisville Area	KY	Subpart 1	Bullitt Co	W	61,236
Louisville Area	KY	Subpart 1	Jefferson Co	W	693,604
Louisville Area	KY	Subpart 1	Oldham Co	W	46,178
Macon Area	GA	Subpart 1	Bibb Co	W	153,887
Macon Area	GA	Subpart 1	Monroe Co	P	50
Manitowoc County Area	WI	Subpart 1	Manitowoc Co	W	82,887

Mariposa and Tuolumne Counties (Southern Mountain Counties) Area	CA	Subpart 1	Mariposa Co	W	17,130
Mariposa and Tuolumne Counties (Southern Mountain Counties) Area	CA	Subpart 1	Tuolumne Co	W	54,501
Mason County Area	MI	Subpart 1	Mason Co	W	28,274
Memphis Area	AR	Subpart 2/Marginal	Crittenden Co	W	50,866
Memphis Area	TN	Subpart 2/Marginal	Shelby Co	W	897,472
Milwaukee-Racine Area	WI	Subpart 2/Moderate	Kenosha Co	W	149,577
Milwaukee-Racine Area	WI	Subpart 2/Moderate	Milwaukee Co	W	940,164
Milwaukee-Racine Area	WI	Subpart 2/Moderate	Ozaukee Co	W	82,317
Milwaukee-Racine Area	WI	Subpart 2/Moderate	Racine Co	W	188,831
Milwaukee-Racine Area	WI	Subpart 2/Moderate	Washington Co	W	117,493
Milwaukee-Racine Area	WI	Subpart 2/Moderate	Waukesha Co	W	360,767
Murray County (Chattahoochee Nat Forest) Area	GA	Subpart 1	Murray Co	P	1,000
Muskegon Area	MI	Subpart 2/Marginal	Muskegon Co	W	170,200
Nashville Area	TN	Subpart 1 - EAC	Davidson Co	W	569,891
Nashville Area	TN	Subpart 1 - EAC	Rutherford Co	W	182,023
Nashville Area	TN	Subpart 1 - EAC	Sumner Co	W	130,449
Nashville Area	TN	Subpart 1 - EAC	Williamson Co	W	126,638
Nashville Area	TN	Subpart 1 - EAC	Wilson Co	W	88,809
Nevada County (Western part) Area	CA	Subpart 1	Nevada Co	P	77,735
New York-N. New Jersey-Long Island Area	CT	Subpart 2/Moderate	Fairfield Co	W	882,567
New York-N. New Jersey-Long Island Area	CT	Subpart 2/Moderate	Middlesex Co	W	155,071
New York-N. New Jersey-Long Island Area	CT	Subpart 2/Moderate	New Haven Co	W	824,008
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Bergen Co	W	884,118
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Essex Co	W	793,633
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Hudson Co	W	608,975
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Hunterdon Co	W	121,989
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Middlesex Co	W	750,162
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Monmouth Co	W	615,301
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Morris Co	W	470,212
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Passaic Co	W	489,049

New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Somerset Co	W	297,490
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Sussex Co	W	144,166
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Union Co	W	522,541
New York-N. New Jersey-Long Island Area	NJ	Subpart 2/Moderate	Warren Co	W	102,437
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Bronx Co	W	1,332,650
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Kings Co	W	2,465,326
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Nassau Co	W	1,334,544
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	New York Co	W	1,537,195
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Queens Co	W	2,229,379
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Richmond Co	W	443,728
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Rockland Co	W	286,753
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Suffolk Co	W	1,419,369
New York-N. New Jersey-Long Island Area	NY	Subpart 2/Moderate	Westchester Co	W	923,459
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Chesapeake	W	199,184
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Gloucester Co	W	34,780
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Hampton	W	146,437
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Isle Of Wight Co	W	29,728
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	James City Co	W	48,102
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Newport News	W	180,150
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Norfolk	W	234,403
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Poquoson	W	11,566
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Portsmouth	W	100,565
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Suffolk	W	63,677
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Virginia Beach	W	425,257
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	Williamsburg	W	11,998
Norfolk-Virginia Beach-Newport News (Hampton Roads) Area	VA	Subpart 2/Marginal	York Co	W	56,297
Parkersburg-Marietta Area	OH	Subpart 1	Washington Co	W	63,251
Parkersburg-Marietta Area	WV	Subpart 1	Wood Co	W	87,986

Philadelphia-Wilmington-Atlantic City Area	DE	Subpart 2/Moderate	Kent Co	W	126,697
Philadelphia-Wilmington-Atlantic City Area	DE	Subpart 2/Moderate	New Castle Co	W	500,265
Philadelphia-Wilmington-Atlantic City Area	DE	Subpart 2/Moderate	Sussex Co	W	156,638
Philadelphia-Wilmington-Atlantic City Area	MD	Subpart 2/Moderate	Cecil Co	W	85,951
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Atlantic Co	W	252,552
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Burlington Co	W	423,394
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Camden Co	W	508,932
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Cape May Co	W	102,326
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Cumberland Co	W	146,438
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Gloucester Co	W	254,673
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Mercer Co	W	350,761
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Ocean Co	W	510,916
Philadelphia-Wilmington-Atlantic City Area	NJ	Subpart 2/Moderate	Salem Co	W	64,285
Philadelphia-Wilmington-Atlantic City Area	PA	Subpart 2/Moderate	Bucks Co	W	597,635
Philadelphia-Wilmington-Atlantic City Area	PA	Subpart 2/Moderate	Chester Co	W	433,501
Philadelphia-Wilmington-Atlantic City Area	PA	Subpart 2/Moderate	Delaware Co	W	550,864
Philadelphia-Wilmington-Atlantic City Area	PA	Subpart 2/Moderate	Montgomery Co	W	750,097
Philadelphia-Wilmington-Atlantic City Area	PA	Subpart 2/Moderate	Philadelphia Co	W	1,517,550
Phoenix-Mesa Area	AZ	Subpart 1	Maricopa Co	P	3,054,504
Phoenix-Mesa Area	AZ	Subpart 1	Pinal Co	P	31,541
Pittsburgh-Beaver Valley Area	PA	Subpart 1	Allegheny Co	W	1,281,666
Pittsburgh-Beaver Valley Area	PA	Subpart 1	Armstrong Co	W	72,392
Pittsburgh-Beaver Valley Area	PA	Subpart 1	Beaver Co	W	181,412
Pittsburgh-Beaver Valley Area	PA	Subpart 1	Butler Co	W	174,083
Pittsburgh-Beaver Valley Area	PA	Subpart 1	Fayette Co	W	148,644
Pittsburgh-Beaver Valley Area	PA	Subpart 1	Washington Co	W	202,897
Pittsburgh-Beaver Valley Area	PA	Subpart 1	Westmoreland Co	W	369,993
Portland Area	ME	Subpart 2/Marginal	Androscoggin Co	P	3,390
Portland Area	ME	Subpart 2/Marginal	Cumberland Co	P	252,907
Portland Area	ME	Subpart 2/Marginal	Sagadahoc Co	W	35,214
Portland Area	ME	Subpart	York Co	P	164,997

		2/Marginal			
Poughkeepsie Area	NY	Subpart 2/Moderate	Dutchess Co	W	280,150
Poughkeepsie Area	NY	Subpart 2/Moderate	Orange Co	W	341,367
Poughkeepsie Area	NY	Subpart 2/Moderate	Putnam Co	W	95,745
Providence (all of RI) Area	RI	Subpart 2/Moderate	Bristol Co	W	50,648
Providence (all of RI) Area	RI	Subpart 2/Moderate	Kent Co	W	167,090
Providence (all of RI) Area	RI	Subpart 2/Moderate	Newport Co	W	85,433
Providence (all of RI) Area	RI	Subpart 2/Moderate	Providence Co	W	621,602
Providence (all of RI) Area	RI	Subpart 2/Moderate	Washington Co	W	123,546
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Chatham Co	P	21,320
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Durham Co	W	223,314
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Franklin Co	W	47,260
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Granville Co	W	48,498
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Johnston Co	W	121,965
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Orange Co	W	118,227
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Person Co	W	35,623
Raleigh-Durham-Chapel Hill Area	NC	Subpart 1	Wake Co	W	627,846
Reading Area	PA	Subpart 1	Berks Co	W	373,638
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Charles City Co	W	6,926
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Chesterfield Co	W	259,903
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Colonial Heights	W	16,897
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Hanover Co	W	86,320
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Henrico Co	W	262,300
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Hopewell	W	22,354
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Petersburg	W	33,740
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Prince George Co	W	33,047
Richmond-Petersburg Area	VA	Subpart 2/Marginal	Richmond	W	197,790
Riverside County (Coachella Valley) Area	CA	Subpart 2/Serious	Riverside Co	P	324,750
Roanoke Area	VA	Subpart 1 - EAC	Botetourt Co	W	30,496
Roanoke Area	VA	Subpart 1 - EAC	Roanoke	W	94,911
Roanoke Area	VA	Subpart 1 - EAC	Roanoke Co	W	85,778
Roanoke Area	VA	Subpart 1 - EAC	Salem	W	24,747
Rochester Area	NY	Subpart 1	Genesee Co	W	60,370
Rochester Area	NY	Subpart 1	Livingston Co	W	64,328

Rochester Area	NY	Subpart 1	Monroe Co	W	735,343
Rochester Area	NY	Subpart 1	Ontario Co	W	100,224
Rochester Area	NY	Subpart 1	Orleans Co	W	44,171
Rochester Area	NY	Subpart 1	Wayne Co	W	93,765
Rocky Mount Area	NC	Subpart 1	Edgecombe Co	W	55,606
Rocky Mount Area	NC	Subpart 1	Nash Co	W	87,420
Sacramento Metro Area	CA	Subpart 2/Serious	El Dorado Co	P	124,164
Sacramento Metro Area	CA	Subpart 2/Serious	Placer Co	P	239,978
Sacramento Metro Area	CA	Subpart 2/Serious	Sacramento Co	W	1,223,499
Sacramento Metro Area	CA	Subpart 2/Serious	Solano Co	P	197,034
Sacramento Metro Area	CA	Subpart 2/Serious	Sutter Co	P	25,013
Sacramento Metro Area	CA	Subpart 2/Serious	Yolo Co	W	168,660
San Antonio Area	TX	Subpart 1 - EAC	Bexar Co	W	1,392,931
San Antonio Area	TX	Subpart 1 - EAC	Comal Co	W	78,021
San Antonio Area	TX	Subpart 1 - EAC	Guadalupe Co	W	89,023
San Diego Area	CA	Subpart 1	San Diego Co	P	2,813,431
San Francisco Bay Area	CA	Subpart 2/Marginal	Alameda Co	W	1,443,741
San Francisco Bay Area	CA	Subpart 2/Marginal	Contra Costa Co	W	948,816
San Francisco Bay Area	CA	Subpart 2/Marginal	Marin Co	W	247,289
San Francisco Bay Area	CA	Subpart 2/Marginal	Napa Co	W	124,279
San Francisco Bay Area	CA	Subpart 2/Marginal	San Francisco Co	W	776,733
San Francisco Bay Area	CA	Subpart 2/Marginal	San Mateo Co	W	707,161
San Francisco Bay Area	CA	Subpart 2/Marginal	Santa Clara Co	W	1,682,585
San Francisco Bay Area	CA	Subpart 2/Marginal	Solano Co	P	197,508
San Francisco Bay Area	CA	Subpart 2/Marginal	Sonoma Co	P	413,716
San Joaquin Valley Area	CA	Subpart 2/Serious	Fresno Co	W	799,407
San Joaquin Valley Area	CA	Subpart 2/Serious	Kern Co	P	550,220
San Joaquin Valley Area	CA	Subpart 2/Serious	Kings Co	W	129,461
San Joaquin Valley Area	CA	Subpart 2/Serious	Madera Co	W	123,109
San Joaquin Valley Area	CA	Subpart 2/Serious	Merced Co	W	210,554
San Joaquin Valley Area	CA	Subpart 2/Serious	San Joaquin Co	W	563,598
San Joaquin Valley Area	CA	Subpart	Stanislaus Co	W	446,997

		2/Serious			
San Joaquin Valley Area	CA	Subpart 2/Serious	Tulare Co	W	368,021
Scranton-Wilkes-Barre Area	PA	Subpart 1	Lackawanna Co	W	213,295
Scranton-Wilkes-Barre Area	PA	Subpart 1	Luzerne Co	W	319,250
Scranton-Wilkes-Barre Area	PA	Subpart 1	Monroe Co	W	138,687
Scranton-Wilkes-Barre Area	PA	Subpart 1	Wyoming Co	W	28,080
Sheboygan Area	WI	Subpart 2/Moderate	Sheboygan Co	W	112,646
South Bend-Elkhart Area	IN	Subpart 1	Elkhart Co	W	182,791
South Bend-Elkhart Area	IN	Subpart 1	St Joseph Co	W	265,559
Springfield (W. Mass) Area	MA	Subpart 2/Moderate	Berkshire Co	W	134,953
Springfield (W. Mass) Area	MA	Subpart 2/Moderate	Franklin Co	W	71,535
Springfield (W. Mass) Area	MA	Subpart 2/Moderate	Hampden Co	W	456,228
Springfield (W. Mass) Area	MA	Subpart 2/Moderate	Hampshire Co	W	152,251
St. Louis Area	IL	Subpart 2/Moderate	Jersey Co	W	21,668
St. Louis Area	IL	Subpart 2/Moderate	Madison Co	W	258,941
St. Louis Area	IL	Subpart 2/Moderate	Monroe Co	W	27,619
St. Louis Area	IL	Subpart 2/Moderate	St Clair Co	W	256,082
St. Louis Area	MO	Subpart 2/Moderate	Franklin Co	W	93,807
St. Louis Area	MO	Subpart 2/Moderate	Jefferson Co	W	198,099
St. Louis Area	MO	Subpart 2/Moderate	St Charles Co	W	283,883
St. Louis Area	MO	Subpart 2/Moderate	St Louis	W	348,189
St. Louis Area	MO	Subpart 2/Moderate	St Louis Co	W	1,016,315
State College Area	PA	Subpart 1	Centre Co	W	135,758
Steubenville-Weirton Area	OH	Subpart 1	Jefferson Co	W	73,894
Steubenville-Weirton Area	WV	Subpart 1	Brooke Co	W	25,447
Steubenville-Weirton Area	WV	Subpart 1	Hancock Co	W	32,667
Sutter County (part) (Sutter Buttes) Area	CA	Subpart 1	Sutter Co	P	1
Tioga County Area	PA	Subpart 1	Tioga Co	W	41,373
Toledo Area	OH	Subpart 1	Lucas Co	W	455,054
Toledo Area	OH	Subpart 1	Wood Co	W	121,065
Ventura County (part) Area	CA	Subpart 2/Moderate	Ventura Co	P	753,197
Washington Area	DC	Subpart 2/Moderate	Entire District	W	572,059
Washington Area	MD	Subpart 2/Moderate	Calvert Co	W	74,563
Washington Area	MD	Subpart	Charles Co	W	120,546

		2/Moderate			
Washington Area	MD	Subpart 2/Moderate	Frederick Co	W	195,277
Washington Area	MD	Subpart 2/Moderate	Montgomery Co	W	873,341
Washington Area	MD	Subpart 2/Moderate	Prince George's Co	W	801,515
Washington Area	VA	Subpart 2/Moderate	Alexandria	W	128,283
Washington Area	VA	Subpart 2/Moderate	Arlington Co	W	189,453
Washington Area	VA	Subpart 2/Moderate	Fairfax	W	21,498
Washington Area	VA	Subpart 2/Moderate	Fairfax Co	W	969,749
Washington Area	VA	Subpart 2/Moderate	Falls Church	W	10,377
Washington Area	VA	Subpart 2/Moderate	Loudoun Co	W	169,599
Washington Area	VA	Subpart 2/Moderate	Manassas	W	35,135
Washington Area	VA	Subpart 2/Moderate	Manassas Park	W	10,290
Washington Area	VA	Subpart 2/Moderate	Prince William Co	W	280,813
Washington County (Hagerstown) Area	MD	Subpart 1 - EAC	Washington Co	W	131,923
Wheeling Area	OH	Subpart 1	Belmont Co	W	70,226
Wheeling Area	WV	Subpart 1	Marshall Co	W	35,519
Wheeling Area	WV	Subpart 1	Ohio Co	W	47,427
York Area	PA	Subpart 1	Adams Co	W	91,292
York Area	PA	Subpart 1	York Co	W	381,751
Youngstown-Warren-Sharon Area	OH	Subpart 1	Columbiana Co	W	112,075
Youngstown-Warren-Sharon Area	OH	Subpart 1	Mahoning Co	W	257,555
Youngstown-Warren-Sharon Area	OH	Subpart 1	Trumbull Co	W	225,116
Youngstown-Warren-Sharon Area	PA	Subpart 1	Mercer Co	W	120,293

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

Appendix 3C: PM Nonattainment

Table 3C-1. $PM_{2.5}$ Nonattainment Areas and Populations (data is current through October 2006 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 3C-2. PM_{10} Nonattainment Areas and Populations (data is current through March 2006 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
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
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 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
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
51 Total Areas		51	28,674		

Appendix 3D: Visibility Tables

Table 3D-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 Rainer 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 3D-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
Jarbidge	NV	12.6	12.8	7.1
Joshua Tree	CA	19.5	20.3	7.1
Joyce Kilmer - Slickrock	NC	29.5	26.1	11.5
Kalmiopsis	OR	14.8	14.4	7.7
Kings Canyon	CA	23.5	24.1	7.1
La Garita	CO	11.6	11.5	7.1
Lassen Volcanic	CA	14.8	14.6	7.3
Lava Beds	CA	16.6	16.5	7.5
Linville Gorge	NC	27.9	24.6	11.4
Lostwood	ND	19.6	18.7	7.3
Lye Brook	VT	23.9	21.1	11.3
Mammoth Cave	KY	30.2	27.0	11.5
Marble Mountain	CA	17.1	16.8	7.7
Maroon Bells - Snowmass	CO	11.3	11.3	7.1
Mazatzal	AZ	13.1	13.5	6.9
Medicine Lake	MT	17.7	17.1	7.3
Mesa Verde	CO	12.8	12.8	7.1
Mingo	MO	27.5	25.9	11.3
Mission Mountains	MT	14.2	14.0	7.4
Mokelumne	CA	12.9	12.8	7.1
Moosehorn	ME	21.4	20.3	11.4
Mount Hood	OR	14.0	13.7	7.8
Mount Jefferson	OR	15.7	15.2	7.8
Mount Rainier	WA	18.9	19.4	7.9
Mount Washington	OR	15.7	15.2	7.9
Mount Zirkel	CO	11.7	11.8	7.1
North Cascades	WA	14.0	14.0	7.8
Okefenokee	GA	26.4	24.7	11.5
Otter Creek	WV	27.6	24.0	11.3
Pasayten	WA	14.7	14.5	7.8
Petrified Forest	AZ	13.5	13.8	7.0
Pine Mountain	AZ	13.1	13.4	6.9
Presidential Range - Dry	NH	23.2	20.9	11.3
Rawah	CO	11.7	11.7	7.1
Red Rock Lakes	WY	12.1	12.1	7.1
Redwood	CA	16.5	16.5	7.8
Rocky Mountain	CO	14.1	14.1	7.1
Roosevelt Campobello	ME	21.4	20.1	11.4
Salt Creek	NM	17.7	17.3	7.0
San Gorgonio	CA	21.5	22.1	7.1
San Jacinto	CA	21.5	21.4	7.1
San Pedro Parks	NM	11.4	11.4	7.0
Sawtooth	ID	13.6	13.5	7.2
Scapegoat	MT	14.2	14.1	7.3
Selway - Bitterroot	MT	12.3	12.1	7.3
Seney	MI	23.8	22.6	11.4
Sequoia	CA	23.5	24.1	7.1
Shenandoah	VA	27.6	23.4	11.3

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).

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