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National-Scale Air Toxics Assessment for 1996

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National-Scale Air Toxics Assessment for 1996

EPA Office of Air Quality
Planning and Standards

DRAFT for EPA Science Advisory Board Review: January 18, 2001

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1 Introduction

This document describes the EPA's National-Scale Air Toxics Assessment, based on emissions data for 1996 (called the "national-scale assessment"). The national-scale assessment is a nationwide study of potential inhalation exposures and health risks associated with 32 hazardous air pollutants (hereinafter called air toxics) and diesel particulate matter (diesel PM), based on 1996 data, because 1996 emission inventories were the most complete and up-to-date available. The initial national-scale assessment is one component of the National Air Toxics Assessment (NATA), the technical support component of EPA's National Air Toxics Program Specifically, NATA includes activities such as expanding air toxics monitoring, improving and periodically updating emissions inventories, periodically conducting national- and local-scale air quality, multi-media and exposure modeling, characterizing risks associated with air toxics exposures, and continuing research on health and environmental effects and exposures to both ambient and indoor sources.

The initial national-scale assessment is the first in an anticipated series of national-scale assessments. These national-scale assessments may be repeated every 3 years, to track trends in the reduction of emissions of air toxics, as well as progress in reducing risks from air toxics exposure. The purpose of the national-scale assessment is to gain a better understanding of the air toxics problem. It was not designed, and is not appropriate specifically, for identifying local- or regional-scale air toxics "hot spots," nor is it appropriate for identifying localized risks or individual risks from air toxics. Further analyses on a national scale, and additional assessments on other scales (e.g., urban air toxics assessments and residual risk assessments) are being performed in order to fully characterize risks, especially disproportionate and cumulative risks.

Given the uncertainties and limitations associated with performing this national-scale assessment, the EPA is seeking EPA Science Advisory Board (SAB) review on the appropriateness of the methodologies and tools, as applied in this assessment, and guidance on ways to improve future national-scale assessments. This document was prepared for that SAB review.

The goals of this initial national-scale assessment are to assist in:

- Identifying air toxics of greatest potential concern, in terms of contribution to population risk;

- Characterizing the relative contributions to air toxics concentrations and population exposures from different types of air toxics emission sources;

- Setting priorities for the collection of additional air toxics data (e.g., emission data, ambient monitoring data, data from personal exposure monitoring) for use in local-scale and multipathway modeling and assessments, and for future research to improve estimates of air toxics concentrations and their potential public health impacts;

- Establishing a baseline for tracking trends over time in modeled ambient concentrations of air toxics; and,

- Establishing a baseline for measuring progress toward meeting goals for inhalation risk reduction from ambient air toxics.

The results of the initial national-scale assessment will provide information to guide EPA in developing and implementing various aspects of the national air toxics program. However, the results will not be used to make specific regulatory decisions for air toxics. While regulatory priority-setting will be informed by this and any future national-scale assessments, risk-based regulations will be based on more refined and source-specific data and assessments.

There are several other important limitations in the scope of the national-scale assessment, and they are as follows:

- It is based on 1996 data and does not reflect significant reductions in air toxics emissions that have occurred since that time

- It focuses only on 33 selected air toxics (32 air toxics of greatest concern in urban areas and diesel particulate matter), and does not address the other 156 air toxics listed in section 112(b) of the Clean Air Act

- It does not include risks due to non-inhalation exposure pathways (e.g., ingestion), which have been shown to be significant for some air toxics (e.g., mercury, dioxins)

- It does not include exposure from indoor sources of air toxics

- It focuses on average population risks, rather than individual extremes (i.e., does not identify “hot spots”)

- It does not reliably capture localized impacts and risks

- It currently includes estimates of background levels that are only approximate; more research is needed to treat background contributions more explicitly

- Current model evaluation efforts indicate a tendency to under-predict ambient concentrations, which may contribute toward an underestimation of risk

For these reasons, the initial national-scale assessment provides only a partial indication of the total risk due to all air toxics. Its results should always be interpreted keeping this perspective in mind.

Section 1 of this document provides background information by describing EPA's national air toxics program and by explaining the goals of NATA and the national-scale assessments. Section 2 describes the purpose and goals of the initial national-scale assessment, while Section 3 explains the methodologies used for the initial national-scale assessment. Section 4 provides a summary of the results of that initial assessment. Section 5 of the document presents the risk characterization, which includes an uncertainty and variability analysis, and Section 6 provides a summary of results and recommendations for future actions.

1.1 EPA's National Air Toxics Program

1.1.1 Background on the Air Toxics Program

The air toxics program was authorized under the 1970 Clean Air Act (CAA), and redesigned and reauthorized under the 1990 CAA Amendments. The program is designed to characterize, prioritize, and address, in an equitable manner (i.e., across racial, cultural, and economic groups), the serious impacts of air toxics (also known as hazardous air pollutants, or air toxics) on public health and the environment through a strategic combination of regulatory approaches, voluntary partnerships, ongoing research and assessments, and education and outreach.

Since 1990, EPA has made considerable progress in reducing emissions of air toxics through regulatory, voluntary and other programs. To date, the overall air toxics program has focused on reducing emissions of air toxics from major stationary sources through the implementation of technology-based emissions standards, as required in section 112(d) of the CAA. These actions have resulted in, or are projected to result in, substantial reductions in air toxics emissions. Additionally, actions to address mobile sources under other CAA programs have achieved significant reductions in air toxics emissions (e.g., the phase-out of lead from gasoline). Many motor vehicle and fuel emission control programs of the past have reduced air toxics. Several current EPA programs further reduce air toxics emissions from a wide variety of mobile sources. These include the reformulated gasoline (RFG) program, the national low emission vehicle (NLEV) program, Tier 2 motor vehicle emission standards and gasoline sulfur control requirements, and recently finalized heavy-duty engine and vehicle standards and onroad diesel fuel sulfur control requirements. In addition, certain other mobile source control programs have been specifically aimed at reducing toxics emissions. These actions are projected to reduce emissions substantially.

EPA expects, however, that the emission reductions that will result from these actions may only be part of what is necessary to protect public health and the environment from air toxics. In addition, section 112(f) of the CAA specifically directs EPA to assess the risk remaining after implementation of technology-based standards (i.e., the residual risk) in order to evaluate the need for additional stationary source standards to protect public health and the environment. Under section 112(k), EPA will be performing comprehensive local-scale assessments for several urban areas. In identifying appropriate additional steps, EPA will use a risk-based focus to develop, implement and facilitate additional federal, state and local regulatory and voluntary measures, if necessary. In considering additional steps toward protecting human health and the

environment, EPA will identify and focus on issues of highest priority.

1.1.2 Air Toxics Program Goals

EPA has ten long-range strategic goals [*1*] which establish the focus for the Agency's work in the years ahead. One of these goals, EPA's Clean Air Goal, states that the air in every American community will be safe and healthy to breathe. In particular, children, the elderly, and people with respiratory ailments will be protected from health risks of breathing polluted air. Reducing air pollution will also protect the environment, resulting in many benefits, such as restoring life in damaged ecosystems and reducing health risks to those whose subsistence depends directly on those ecosystems. The specific air toxics objective under this goal is, by 2020, to eliminate unacceptable risks of cancer and other significant health problems from air toxic emissions for at least 95% of the population, with particular attention to children and other sensitive sub-populations, and substantially reduce or eliminate adverse effects on our natural environment. Further, by 2010, the tribes and EPA will have the information and tools to characterize and assess trends in air toxics in Indian country.

EPA will progress toward meeting its air toxics program goals through a combination of statutory authorities, regulatory activities and voluntary initiatives. EPA's overall approach to reducing air toxics consists of the following four key components, which are discussed in greater detail in the July 19, 1999 Federal Register notice for the National Air Toxics Program: [*2*]:

1. *Source-specific standards and sector-based standards.* Section 112 of the CAA specifies emission standards (technology-based standards) and residual risk standards (risk-based standards), as well as area source standards identified by the Integrated Urban Air Toxics Strategy. Additionally, section 129 of the CAA requires standards for solid waste incineration and section 202(l) requires EPA to promulgate reasonable requirements to control air toxics from motor vehicles and their fuels.

2. *National, regional, and community-based initiatives to focus on multi-media and cumulative risks.* Section 112(k)(4) of the CAA requires EPA to "encourage and support area wide strategies developed by the state or local air pollution control agencies." EPA's risk initiatives will include state, local and tribal program activities consistent with the Integrated Urban Air Toxics Strategy on the local level, as well as federal and regional activities associated with the multimedia aspects of air toxics (e.g., the Great Waters program [Section 112(m)] and Agency initiatives concerning mercury and other persistent and bioaccumulative toxics [PBTs]). These Agency initiatives include collaboration between the air and water programs on the impact of air deposition on water quality (e.g., by accounting for the contribution of air deposition to the total maximum daily load (TMDL) of pollutants to a water body), and collaboration between offices within EPA's air program to assess the risks from exposures to air toxics from indoor sources.

3. *National air toxics assessment (NATA) activities.* NATA activities will help EPA

identify areas of concern, characterize risks, and track progress toward meeting the overall air toxics program goals, as well as the risk-based goals of the various activities and initiatives within the program. The NATA activities include expansion of air toxics monitoring, improving and periodically updating emissions inventories, national- and local-scale air quality, multi-media and exposure modeling (including modeling which considers stationary and mobile sources), continued research on health effects and exposures to both ambient and indoor air, and use and improvement of exposure and risk assessment tools. These activities will provide EPA with improved characterizations of air toxics risk and risk reductions resulting from emissions control standards and initiatives for both stationary and mobile source programs.

4. *Education and outreach.* In light of the scientific complexity inherent in air toxics issues, EPA recognizes that the success of the overall air toxics program depends, in part, on the Agency's ability to communicate effectively with the public about air toxics risks and activities necessary to reduce those risks. This includes education and outreach efforts on air toxics in the ambient as well as indoor environments.

Under the CAA, EPA provides leadership and technical and financial assistance for the development of cooperative federal, state, local, and tribal programs to prevent and control air pollution. A strong partnership with and among the different governments that each play a key role in air quality protection is critical to achieving clean air, because it is the sum of these collective efforts that constitutes the national air quality program.

The Agency is committed to working with cities, communities, state, local and tribal agencies, and other groups and organizations that can help implement activities to reduce air toxics emissions. For example, EPA has been working with its regulatory partners and interested stakeholders on activities related to NATA. In addition, the Agency will continue to work with its regulatory partners and stakeholders on regulation development. EPA also expects to involve local communities and industries in the development of local risk initiatives such as the urban community-based pilot projects (i.e., local-scale assessments).

1.2 Overview of National Air Toxics Assessment (NATA) Activities

As described in section 1.1, the overall air toxics program consists of four central elements: (1) source-specific and sector-based standards, (2) national, regional and community-based initiatives, (3) NATA activities, and (4) education and outreach. This section focuses on the third of these elements, the NATA activities.

1.2.1 Scope of NATA Activities

EPA includes, within the scope of the NATA activities, all data gathering, analyses, assessments, characterizations, and related research needed to support the air toxics program, as shown in Figure 1-1. The figure depicts two levels of assessments. National-scale assessments are shown centrally in boldface, and refined local-scale assessments are depicted underneath. Other NATA activities that support each type of

assessment (e.g., emission characterization, meteorological data collection) are shown in boxes at the top of the figure, with arrows depicting the points in the assessment where the information is used. The information from the NATA activities flows to the right side of the figure, where the most important uses for the outputs are shown.

1.2.2 Purpose of NATA Activities

EPA envisions the NATA activities as an ongoing, permanent part of the air toxics program. EPA intends to use NATA products to inform the entire air toxics program with a body of coordinated information that expands and evolves to fit the needs of each part of that program.

EPA's ongoing data collection and research will allow the Agency to continue to improve its understanding of air toxics emissions, ambient concentrations (with a multi-media focus, where appropriate), outdoor and indoor exposures (by inhalation and other pathways, where appropriate), and health and environmental effects. These efforts will also help to improve the tools and methods for assessing and characterizing public health, environmental hazards, and cumulative risks associated with exposures to air toxics. EPA is currently working to ensure stakeholder involvement in the planning and conduct of these activities, and to ensure appropriate peer review of the underlying science and assessment methods.

As shown in Figure 1-1, these NATA activities will serve several purposes. The information and assessments developed by NATA activities will help EPA:

1. Determine priorities for regulatory programs as well as for national, regional, and community-based initiatives;

2. Assess progress toward CAA goals and EPA's long-range strategic goals;

3. Inform state, local, and tribal programs and support public right-to-know initiatives regarding risks associated with exposure to air toxics; and,

4. Support prospective assessments of the benefits estimated to result from implementation of statutory air toxics mandates (as required by section 812 of the CAA).

1.2.3 Links between NATA Activities and Other Program Elements

Figure 1-2 displays important examples of regulatory programs, risk-based initiatives, and special studies that will utilize information from current and future NATA activities. National-scale information produced by these NATA activities will specifically support setting priorities and estimating progress by each of these elements of the air toxics program. In turn, it is anticipated that the regulatory programs, initiatives, and studies shown on Figure 1-2 will result not only in continued reductions in air toxics emissions and risk, but will also lead to enhanced knowledge and tools to improve EPA's ability to characterize air toxics risk. The risk reduction progress that is made, and the improved risk characterization information and tools that become available, will then be reflected in future NATA activities.

1.3 The National-Scale Assessments

The purpose of the national-scale assessments is to gain a better understanding of the air toxics problem on a national scale by compiling and analyzing existing air toxics data. The goal of the national-scale assessments is to assist in: 1) identifying air toxics of greatest potential concern in terms of contribution to population risk; 2) characterizing the relative contributions of various types of emission sources to air toxics concentrations and population exposures; 3) setting priorities for collection of additional air toxics data and research to improve estimates of air toxics concentrations and their potential public health impacts; 4) tracking trends in modeled ambient air toxics concentrations over time; and, 5) measuring progress toward meeting goals for inhalation risk reduction from ambient air toxics. To accomplish these goals, the national-scale assessment will be repeated every three years, with the next national-scale assessment being performed in 2003, with 1999 air toxics data.

EPA particularly notes that, while the initial national-scale assessment (i.e., an assessment which is national in scale, has low resolution, and represents only one part of the overall NATA activities) can help set general programmatic priorities and provide direction for the design of local-scale initiatives and more refined special studies, it is not intended to serve as the basis for setting standards or addressing specific local concerns or community environmental justice issues. EPA will use more refined and local-scale information and assessment tools developed within NATA activities as the basis for risk-based standard setting (e.g., residual risk standards) and for local initiatives.

2 The Initial National-Scale Assessment

2.1 Uses and Limitations

The results of the initial national-scale assessment presented in this document provide important information to help EPA continue to develop and implement various aspects of the national air toxics program. However, it is important to note that these results will not be used directly to regulate sources of air toxics emissions. Although the national-scale assessments (both the initial assessment and future assessments) will inform the regulatory priority-setting process, risk-based regulations will be supported by more refined and source-specific data and assessment tools.

More specifically, the national-scale assessment results will assist in:

- Identifying air toxics of greatest potential concern, in terms of contribution to population risk;

- Characterizing the relative contributions by different types of air toxics emission sources to air toxics concentrations and population exposures (i.e., major, area and other, on-road and non-road mobile, and background sources);

- Setting priorities for the collection of additional air toxics data (e.g., emission data, ambient monitoring data, data from personal exposure monitoring) and research to improve estimates of air toxics concentrations and their potential public health impacts;

- Establishing a baseline for tracking trends over time in modeled ambient concentrations of air toxics; and,

- Establishing a baseline for measuring progress toward meeting goals for inhalation risk reduction from ambient air toxics.

The results of the initial national-scale assessment will help identify areas of the country and pollutants where additional investigation is needed, and will help target locations where more refined, regional- and local-scale analyses should be done.

There are several other important limitations in the scope of the national-scale assessment, and they are as follows:

- It is based on 1996 data and does not reflect significant reductions in air toxics emissions that have occurred since that time

- It focuses only on 33 selected air toxics (32 air toxics of greatest concern in urban areas and diesel particulate matter), and does not address the other 156 air toxics listed in section 112(b) of the Clean Air Act

- It does not include risks due to non-inhalation exposure pathways (e.g., ingestion), which have been shown to be significant for some air toxics (e.g., mercury, dioxins)

- It does not include exposure from indoor sources of air toxics

- It focuses on average population risks, rather than individual extremes (i.e., does not identify "hot spots")

- It does not reliably capture localized impacts and risks

- It currently includes estimates of background levels that are only approximate; more research is needed to treat background contributions more explicitly

- Current model evaluation efforts indicate a tendency to under-predict ambient concentrations, which may contribute toward an underestimation of risk

For these reasons, the initial 1996 national-scale assessment should be considered to provide only a partial indication of the total risk due to all air toxics. Its results should always be interpreted with this perspective in mind.

The initial national-scale assessment is the first step in an iterative and evolving process to assess and characterize risks from exposures to air toxics, measure progress in meeting goals, and inform future directions for EPA's national air toxics program. While there continue to be significant uncertainties and gaps in methods, models, and data that limit EPA's ability to assess risks to public health and the environment associated with exposures to air toxics, continued research will enable future assessment activities, both at the national level and at more refined levels, to yield improved assessments of cumulative air toxics risks. An important component of future NATA activities will be to repeat the national-scale assessment every three years, with the next national-scale assessment being performed in 2003 with 1999 air toxics data.

2.2 The EPA Risk Assessment Paradigm

Because cancer and noncancer health impacts generally cannot be directly isolated and measured with respect to environmental exposures, EPA and others have spent more than two decades developing an extensive set of risk assessment methods, tools, and data that serve the purpose of estimating health risks for many Agency programs. Although significant uncertainties remain, EPA's risk assessment science has been extensively peer-reviewed, is widely used and understood by the scientific community, and continues to expand and evolve as scientific knowledge advances. All NATA risk assessments will be based on the most current and appropriate risk estimation methods.

EPA's framework for assessing and managing risks reflects the risk assessment and risk management paradigm set forth by the National Academy of Sciences (NAS) in 1983 [*3*], shown in Figure 2-1. This figure identifies research, risk assessment, and risk management as three separate but connected elements. The NAS concluded that risk

assessment and risk management are “two distinct elements” between which agencies should maintain a clear conceptual distinction. The 1983 NAS report identified four steps integral to any risk assessment: 1) hazard identification, 2) dose-response assessment, 3) exposure assessment, and 4) risk characterization. As described in the next section, the NAS paradigm for risk assessment serves as the basis for the NATA national-scale assessment.

2.3 Application of the EPA Risk Assessment Paradigm

As described in more detail in section 3, the initial national-scale assessment includes four major components:

(1) compiling a 1996 national emissions inventory of air toxics emissions from outdoor sources;

(2) estimating 1996 air toxics ambient air concentrations for 33 air toxics (32 urban air toxics and diesel particulate matter (diesel PM)) nationwide;

(3) estimating 1996 population inhalation exposures to these air toxics; and,

(4) characterizing potential public health risks associated with these exposures, including both cancer and noncancer effects.

The following sections describe the elements of the initial national-scale assessment.

2.4 Conceptual Model

2.4.1 Scope and Resolution

The initial national-scale assessment was national in scope, covering the contiguous United States, Puerto Rico, and the Virgin Islands. The assessment excluded Alaska, Hawaii, and U.S. territories other than Puerto Rico and the Virgin Islands because the data needed to support the models (e.g., census tract and meteorological information) were not readily available for these areas.

The pollutants identified and peer-reviewed for inclusion in this initial national-scale assessment were the air toxics identified as priority pollutants in EPA's Integrated Urban Air Toxics Strategy (IUATS)[2]. These 33 air toxics are a subset of EPA's list of 188 air toxics, under CAA section 112(b), and have been identified as those pollutants that present the greatest threat to public health in the largest number of urban areas. This assessment also included diesel particulate matter, an indicator of diesel exhaust. EPA has recently listed this likely human carcinogen as a mobile source air toxic and is addressing this pollutant in several regulatory actions.[1] Because the current dioxin exposure and human health reassessment is undergoing review, the initial national-scale assessment did not include the class of compounds known as dioxins. Since the most significant exposure route for dioxin is ingestion rather than inhalation, dioxin’s relative

[1] In the Mobile Source Air Toxics rule (December 2000), under section 202(l)(2) of the CAA, EPA listed diesel particulate matter plus diesel exhaust organic gases, collectively, as a mobile source air toxic.

contribution to this study's inhalation risk estimates would likely not have been great. It is expected that dioxins will be included in future national-scale assessments where ingestion and inhalation exposures are assessed. In summary, 32 of the priority pollutants in EPA's Integrated Urban Air Toxics Strategy, as well as diesel PM, make up the 33 air toxics that were included in the initial national-scale assessment. These 33 air toxics will hereinafter be referred to as "the pollutant set."

The national-scale assessment included a risk characterization based on estimates of inhalation exposure concentrations determined at the census-tract level. Because of uncertainties associated with the accuracy of the results at the census-tract level, the results of the risk characterization have been aggregated and presented at the county level or higher. EPA strongly cautions that census-tract level estimates are not reliable, and results for individual census tracts are not presented. EPA chose the county level of resolution for the assessment for three reasons. First, the inventory data for some pollutants and source sectors was only available at a county level resolution. Second, uncertainties inherent in other model input data (e.g., terrain, meteorology) and the simplifying assumptions made in the models (e.g., transformation chemistry) themselves rendered estimates at the census-tract level highly uncertain. Third, census-tract level exposure estimates included additional variability (e.g., microenvironment data, human activity pattern data) that is introduced only at local levels. For these reasons, EPA's confidence in the accuracy of estimates for any given census tract is low.

2.4.2 Time-Frame for Exposure

The initial national-scale assessment focused on average yearly exposures for effects other than cancer, and assumed lifetime exposures based on annual averages for carcinogenic effects. Subchronic and acute exposures were not included in the initial national-scale assessment. The national-scale assessment excluded acute and subchronic exposures because of the nature of the emissions data, which contained only yearly total emissions. If the emission inventories are later expanded to cover short-term (e.g., hourly, daily) emission rates, EPA may incorporate shorter exposure times into future national-scale assessments.

2.4.3 Rationale for Aggregating Air Toxics and Sources

Exposure to air toxics from all sources is determined by a multiplicity of interactions among complex factors, including the location and nature of the emissions, the existence of multiple sources, local climate, location of receptor populations, and the specific behaviors and physiology of those populations. Risks associated with these exposures are influenced by the particular combination of air toxics that people actually inhale, and the chemical and biological interactions among those air toxics.

Because of this high level of complexity, the magnitude of risks associated with inhalation of air toxics can be usefully depicted by aggregating risk across both substances and sources. Given the goals of this assessment, and the purposes for which EPA intends to use it, currently available risk assessment data, tools, and guidance were judged sufficient.

2.4.4 Details of the Conceptual Model

The following subsections include summary descriptions of the risk dimensions and elements of the national-scale assessment, as recommended by EPA's Cumulative Risk Assessment Guidance [4]. The conceptual model for the national-scale assessment appears in Figure 4.

2.4.4.1 Sources

The dispersion modeling, from which the exposure assessment and risk characterization were developed, included stationary and mobile source emissions for the contiguous US, Puerto Rico, and the Virgin Islands.

By limiting the exposure assessment and risk characterization of the initial national-scale assessment to sources having data within the available emission inventories, EPA excluded all non-inventoried sources. This limitation effectively excluded releases (1) from many natural processes, (2) from indoor sources (e.g., paints, carpets, etc.), and (3) from surface water, groundwater, or soil. While EPA takes these releases and their potential to cause adverse health effects seriously, EPA lacked adequate model inputs (i.e., data on substance identities and release rates) needed to quantify them in the assessment.

2.4.4.2 Stressors

The initial national-scale assessment encompassed the pollutant set described in section 2.5.1. Later national-scale assessments may expand to cover additional Clean Air Act air toxics, to the extent that available emission and toxicity data allow doing so. EPA chose to limit the initial national-scale assessment to this pollutant set for two reasons. First, these air toxics, in aggregate, appeared highly likely to encompass most of the total air toxics-related risk to human populations [2]. Second, the initial national-scale assessment, in combination with similar future assessments, will serve as an important vehicle to fulfill EPA's assessment commitments under the Integrated Urban Air Toxics Strategy, which is focused specifically on these air toxics.

2.4.4.3 Pathways/Media

The dispersion modeling step of the assessment included evaluation of the transport of particles and gases through the air to receptors within 50km of sources. Atmospheric transformation and losses from the air by deposition were included in the modeling, where data permit. For 13 pollutants with available ambient monitoring data, background concentration data due to sources located more than 50 km away were included. These background estimates for each pollutant were assumed constant across the US. The assessment excluded accretion in water, soil, or food associated with deposition from air, and bioaccumulation of air toxics in tissues. Although EPA takes potential transport of air toxics into other media very seriously, refined tools to model multipathway concentrations on the national scale are not yet readily available for use for many pollutants. Future local- or urban-scale assessments will include multipathway calculations, which may be added to national-scale assessments when adequate models and input data become available.

2.4.4.4 Exposure Routes

The national-scale assessment focused on exposures due to inhalation of ambient air. Human receptors were modeled as they moved within 37 separate microenvironments such as residences, offices, schools, outdoor work sites, and automobiles. The exposure assessment estimated air concentrations of each substance within each microenvironment, using the outdoor concentration, proximity of the microenvironment to sources, time of day, air exchange rate, and other. Human activities (e.g., exercising, sleeping) were reflected in the assessment by the amount of time individuals spend in each microenvironment.

The national-scale assessment excluded human exposures via ingestion or dermal contact. This was a consequence of the lack of multipathway models suitable for calculations at the national scale. As modeling tools become available to estimate transfers of substances from air to other media on a national scale, future national-scale assessments may include dermal and ingestion exposures.

2.4.4.5 Subpopulations

The national-scale assessment characterized risks to 40 distinct human subpopulations, divided into five life stage cohorts, two genders, and four racial/ethnic cohorts. Figure 2-3 shows the 40 possible cohorts chosen for this assessment. These cohorts were selected to mirror tract-level demographic census data, to support selection of a representative model population for each census tract. Life stages that were separately assessed included children aged 5 or less, children aged 6-11, children aged 12-17, adults aged 18-65, and adults aged 65 or greater. Racial/ethnic groups included African American, Caucasian, Hispanic, and Asian and other. Exposures and risks are estimated separately for each of the 40 cohorts in each census tract. Within each census tract, the proportion of the total population from each cohort was tailored to demographic census data for that particular tract and presented as the “average cohort” for that tract. Graphs of estimated exposure and risk provide various percentiles only at the county level or higher, for the general population.

2.4.4.6 Non-Human Receptors

The initial national-scale assessment excluded non-human receptors (e.g., wildlife and native plants). This limitation resulted from the extreme complexity of considering potential adverse ecological impacts to the multiplicity of ecosystems that exist within such a large area. Future local- and urban-scale assessments may be expanded to include non-human receptors, contingent on the availability of necessary resources, data, and methodologies. However, EPA does not envision including non-human receptors in future national-scale assessments unless greatly improved models and tools become available.

2.5 Stakeholder Involvement

EPA has worked with the following groups as stakeholders for NATA activities in general, and for the initial national-scale assessment in particular: state, local, and tribal governments; industry; small businesses; and public interest groups.

As part of the outreach efforts on NATA and the national-scale assessment, EPA has held informal discussions with several stakeholder groups, including representatives from the

state and Territorial Air Pollution Program Administrators and the Association of Local Air Pollution Control Officials (STAPPA/ALAPCO), the National Environmental Justice Advisory Council (NEJAC), the Clean Air Act Advisory Committee (CAAAC), the U.S. Conference of Mayors, Congressional representatives, tribal air contacts, and industry groups.

In addition, on October 18 and 19, 1999, EPA held two public meetings in Washington D.C. which were attended by representatives of regulatory, public interest, and business and industry groups and associations. EPA sought input from interested stakeholders as to the most appropriate and effective ways to present the results of the initial air toxics assessment. Participants discussed the components of EPA's current air toxics program provided input on approaches to presenting initial results from the national-scale air toxics assessment activities. The Agency emphasized its desire to clearly communicate to the public and its regulatory partners both what can be learned from such national-scale assessments, as well as the limitations and uncertainties of such information.

Through comments received at the October meetings, and in follow-up comment letters received after the meeting, stakeholders provided ideas, cautions, criticisms, and other substantive input. EPA has factored this input into plans for the presentation of national-scale assessment results. During April and May 2000, EPA conducted a six-week preview of the results of the ambient concentration modeling step of the national-scale assessment with its regulatory partners at the state, local and tribal level. The purpose of this preview was to enable EPA's regulatory partners to provide feedback on the appropriate interpretation and communication of the results of the initial national-scale assessment and to provide a quality assurance assessment of the results.

EPA is currently seeking input from these same stakeholder groups on interpretation and communication of the exposure assessment and risk characterization results, and on quality assurance of these results.

2.6 Peer Review Activities for the Initial National-Scale Assessment

Many of the national-scale assessment components described above have undergone peer-review and public review, either on their own or as critical elements of other analyses. EPA also instituted a peer-review for: (1) the planning and scoping document for the initial national-scale assessment, and (2) for this draft NATA national-scale assessment report, using procedures recommended by the EPA Science Policy Council [5].

2.6.1 Past Reviews of National-Scale Assessment Components

2.6.1.1 List of Urban Air Toxics

In 1997, EPA developed an initial list of potential candidate urban air toxics using a risk-based ranking methodology, and conducted a public review of the national emissions

inventory for those candidate air toxics. During January 1998, a panel of technical experts from outside the U.S. EPA reviewed the air toxics ranking methodology and analysis. The final methodology used to select the 33 urban air toxics identified in the Integrated Urban Air Toxics Strategy incorporated revisions based on comments raised in the 1998 peer review. EPA also received public comments on the draft list of urban air toxics [*6*], which led to further modifications of the identification methodology and the underlying data inputs.

2.6.1.2 1996 National Toxics Inventory

EPA made the draft 1996 National Toxics Inventory (NTI) and documentation available for review and comment between April and August 1999, and received extensive comments and revisions from industry, state and local agencies, and others. Before incorporation into the final NTI, revisions were subjected to a rigorous review process to ensure internal consistency. Further details of the review process are described in section 3.2.1. In addition to review and comment on the 1996 NTI, methods and assumptions used to develop toxic emission estimates for benzene, 1,3-butadiene, formaldehyde, and acetaldehyde from on-highway mobile sources were described in an EPA technical report [*7*] and peer reviewed, in addition to undergoing review by industry groups, state agencies, and local governments (peer review comments are available at: http://www.epa.gov/otaq/toxics.htm).

2.6.1.3 ASPEN National Dispersion Model

In 1996, the EPA Science Advisory Board (SAB) reviewed the Cumulative Exposure Project methodology [*8*], including the underlying scientific basis for the project and specific details of the modeling methodology. This review included the use of the ASPEN dispersion model in developing estimates of ambient concentrations of air toxics. The SAB review found that the overall conceptual framework and underlying scientific foundation was sound but stressed the importance of comparing the ASPEN predictions with measured ambient concentrations.

2.6.1.4 Hazardous Air Pollutant Exposure Model (HAPEM)

The EPA Office of Transportation and Air Quality (formerly the Office of Mobile Sources) has used earlier versions of HAPEM, the exposure model used for this assessment, in analyses of exposure to carbon monoxide and vehicle-related air toxics. Two studies of air toxics exposure associated with vehicle emissions in 1993 and 1999 [9], were peer-reviewed by a panel of independent experts. Details on this version of HAPEM and the peer-review comments can be found at: http://www.epa.gov/oms/toxics.htm.

2.6.1.5 EPA Risk Assessment Guidelines

Since cancer and non-cancer health impacts cannot be directly isolated and measured, EPA and others have spent more than two decades developing an extensive set of risk assessment methods, tools and data that serve the purpose of estimating health risks for many Agency programs. EPA develops and publishes its risk assessment methods in the

form of risk assessment guidelines that have been extensively peer-reviewed, are widely used and understood by the scientific community, and continue to expand and evolve as scientific knowledge advances.

2.6.1.6 Microenvironment Factors

The microenvironment (ME) factors (described in section 3.2.3) represent the relationship between the ambient concentration and the microenvironment of interest. The ME factor approach for determining microenvironment concentrations is the only computationally feasible way (in lieu of indoor/outdoor mass balance models) for predicting exposures on a national scale. To help assure the credibility of the factors, and their appropriateness for use in the national-scale assessment, the ME factors underwent a peer review consistent with EPA's peer-review guidance [5].

2.6.1.7 ASPEN Results

Ambient concentration estimates for the pollutant set, calculated by the initial Assessment System for Population Exposure Nationwide (ASPEN) model runs, were reviewed by state, local, and tribal authorities to find and correct potential input errors, and to obtain further input in developing useful presentation formats. The ASPEN model was subsequently re-run to incorporate the corrections.

2.6.2 Review of Planning and Scoping Document for the Initial National-Scale Assessment

In July 2000, six technical experts from outside the U.S. EPA completed a peer review of the draft planning and scoping document. The reviewers were asked to consider the appropriateness of approaches used to (1) process the state-derived National Toxics Inventory for dispersion modeling, (2) estimate ambient concentrations using the Assessment System for Population Exposure Nationwide (ASPEN) model, (3) estimate human inhalation exposures using the Hazardous Air Pollutant Exposure Model version 4 (HAPEM4), and (4) estimate, aggregate, and interpret associated cancer and non-cancer risks. The detailed charge to the reviewers, a summary of the reviewers' comments, and the EPA's response to comments are provided in Appendix A.

3 Methods

3.1 Introduction

EPA conducted the initial national-scale assessment to demonstrate an approach for characterizing air toxics risks nationwide. This assessment expanded on the approach first used by the EPA in the 1998 Cumulative Exposure Project (CEP) [8], in which annual ambient concentrations of 155 air toxics were estimated for approximately 61,000 census tracts in the contiguous US. In the CEP, estimated concentrations, which are based on 1990 emission estimates, are assumed to equal human exposure concentrations for all persons living in each census tract, and are evaluated, by comparing to currently available "health benchmarks," to assess potential health risks. In this initial national-scale assessment, EPA augmented the CEP approach by: (1) using a 1996 emission inventory compiled by a more rigorous method; (2) incorporating human demographics and behavior into the development of exposure estimates; (3) developing a comprehensive risk characterization, including estimating cumulative risk associated with multiple air toxics; and, (4) expanding geographic coverage to include Puerto Rico and the Virgin Islands.

The four major components of the national-scale assessment were as follows:

1. *Compiling a national emissions inventory for 1996 of the pollutant set emissions from outdoor sources.* The types of emissions sources in the inventory include major stationary sources (e.g., large waste incinerators and factories), area sources (e.g., dry cleaners, small manufacturers, consumer products), and both onroad and nonroad mobile sources (e.g., cars, trucks, and boats).

2. *Estimating 1996 air toxics ambient concentrations.* EPA used the ASPEN air dispersion model and the 1996 inventory to estimate annual average ambient concentrations for the pollutant set (see pollutant names and corresponding CAS numbers listed in Table 3-1) across the contiguous United States, Puerto Rico, and the Virgin Islands. As part of this modeling exercise, estimated concentrations were compared to available ambient monitoring data as a partial evaluation of model performance. Details of this comparison are provided in Appendices I & J.

3. *Estimating 1996 population exposures.* Rather than assume that all people within each census tract were exposed to a concentration that was equivalent to the ASPEN prediction for that census tract, EPA used an inhalation exposure model and the ambient concentrations from item 2 above to estimate human exposure to the pollutant set across the contiguous United States (and Puerto Rico and the Virgin Islands). Exposure modeling was an important step in this assessment because it provided more realistic estimates of population exposures to air toxics from outdoor emission sources by accounting for the time people spend indoors and in other microenvironments (e.g., in vehicles), patterns of movement (e.g., commuting between home and work locations), and activity levels. Detailed information about the structure, function, and use of the HAPEM4 exposure model is provided in the user's guide (Appendix B).

4. *Characterizing potential public health risks.* EPA characterized potential population health risks associated with inhalation of air toxics, including both cancer and noncancer effects, using available information on the health effects of each pollutant, current Agency risk assessment and risk characterization guidelines, and estimated population exposures. This characterization quantified, to the extent possible, potential cumulative risks to public health due to inhalation of air toxics from outdoor emission sources, discussed the uncertainties and limitations of the assessment, and identified other potential risks to public health from exposures that are beyond the scope of this quantitative assessment.

The approach outlined for the national-scale assessment was fundamentally based on national-scale modeling techniques to estimate ambient pollutant concentrations and

Table 3-1. Pollutant set for the initial national-scale assessment.[2]

Pollutant	CAS #
Acetaldehyde	75070
Acrolein	107028
Acrylonitrile	107131
Arsenic compounds	
Benzene	71432
Beryllium compounds	
1,3-Butadiene	106990
Cadmium compounds	
Carbon tetrachloride	56235
Chloroform	67663
Chromium compounds	
Coke Oven Emissions	8007452
1,3-Dichloropropene	542756
Diesel particulate matter	
Ethylene dibromide (1,2-dibromoethane)	106934
Ethylene dichloride (1,2-dichloroethane)	107062
Ethylene oxide	75218
Formaldehyde	50000
Hexachlorobenzene	118741
Hydrazine, hydrazine sulfate	302012
Lead compounds	

[2] This list is the list of 33 urban air toxics, except that dioxins have been removed from the list and diesel particulate matter has been added.

Pollutant	CAS #
Manganese compounds	
Mercury compounds	
Methylene chloride	75092
Nickel compounds	
Polychlorinated biphenyls (PCBs)	1336363
Polycyclic Organic Matter (POM) (including PAHs)	
Propylene dichloride (1,2-dichloropropane)	78875
Quinoline	91225
1,1,2,2-Tetrachloroethane	79345
Tetrachloroethylene (perchloroethylene)	127184
Trichloroethylene (TCE)	79016
Vinyl chloride	75014

population exposures nationwide. While such computer models necessarily required simplifying assumptions and introduced significant uncertainties, they were needed to conduct such a large-scale assessment since direct measurements of ambient air toxics concentrations are limited, and direct personal exposure measurements are relatively rare. Such measurements are available for only a subset of air toxics in relatively few locations and for small study populations. Those ambient air toxics data used in the model to monitor comparison were taken from the air toxics archive. The air toxics archive is a collection of ambient air toxics data that is compiled directly from State and local agencies and which is supplemented with any other ambient air toxics data on AIRS. The specific data used in the model to monitor comparison can be found at: www.epa.gov/ttn/uatw/nata/mtom_pre.html#data. Although EPA is working to expand the number and locations of ambient air toxics monitors and the study of personal exposures, direct measurement of air toxics concentrations or exposures is not currently practical for all air toxics of interest across all areas of the country. As such measurement data become available over time, they can and will be used to evaluate the models so as to better understand some of the uncertainties in such assessments and to improve modeling tools.

In describing what this assessment included, it is also important for the reader to recognize potentially important sources and pathways of risks to public health that were considered beyond the scope of this assessment, and were therefore not included. First, while EPA recognizes that indoor sources of air toxics emissions likely contribute substantially to the total exposures that people experience for a number of these air toxics, additional work is needed to better develop our tools for assessing these indoor sources of exposure on a national scale, that was not a part of this assessment. Further, for a subset of air toxics that persist or bioaccumulate in the environment, dietary intake (e.g., from eating contaminated fish) likely contributes much more to total exposure than does the inhalation pathway that was be addressed in this assessment. Table 3-2 lists

those urban air toxics believed most likely to present important multipathway risks. These and other important aspects of total population exposures to air toxics will be addressed more fully over time as part of future NATA assessment activities, as more comprehensive data and assessment tools become available. As a result of excluding these potentially-important contributors to total risk from this assessment, the results of this assessment should always be viewed with its limitations in mind, and the risks interpreted as only a portion of the total risks which may be associated with these air toxics.

Table 3-2. Urban air toxics believed to present risks from multipathway exposure.

Pollutants
Hexachlorobenzene
Lead compounds
Mercury compounds
Polychlorinated biphenyls (PCBs)
Polycyclic Organic Matter (including 7-PAH)
2,3,7,8-TCDD (dioxin)

3.2 Exposure Assessment

EPA's 1992 guidelines for exposure assessment [10] establish a broad framework for exposure assessments by describing the general concepts of exposure assessment, including definitions and associated measurement units, and by providing general guidance on the planning and conduct of an exposure assessment. The guidelines also provide information on presenting the results of the exposure assessment and characterizing uncertainty.

The guidelines define human exposure as contact with a chemical or agent at the visible external boundary of a person, including skin and openings into the body such as mouth and nostrils (but not necessarily contact with exchange boundaries where absorption may take place, such as skin, lung, and gastrointestinal tract). Therefore, an exposure assessment is the quantitative or qualitative evaluation of contact, and includes such characteristics as intensity, frequency, and duration of contact. Often, an assessment also will evaluate the rate and route at which a chemical crosses the external boundary (dose) and the amount absorbed (internal dose). The numerical output of an exposure assessment may be either exposure or dose, depending on the purpose of the evaluation and availability of appropriate data.

An exposure assessment has three major components: source characterization, environmental fate and transport characterization, and characterization of exposure. These components are discussed individually below.

3.2.1 Source Characterization: Emission Inventories

In the first step of an exposure assessment for air toxics, the specific air toxics emitted

and the sources of their airborne emissions are determined. Data are collected on the emission rates of the pollutants and the release parameters of the source. Knowledge of the emission rate and release characteristics enables the pollutant fate and transport to be estimated.

Ideally, the emission estimates are from direct measurements of representative source emissions. Although such measurements are likely to provide the most accurate data for an emission source, these data are typically not available because such sampling is often too time- and resource-intensive. When specific emission measurements are not feasible or available, other emission estimation methods, including material balances and emission factors, are sometimes used as an alternate method. Emission factors indicate the quantity of a pollutant typically released to the atmosphere for a particular source operation, and are usually considered to be representative of an industry or emission type as a whole. Each approach to estimating emissions, including use of direct measurement data, has an inherent level of uncertainty, which adds to the overall uncertainty of a risk analysis.

Depending on the analysis, source and emissions data can be derived from broad-scale emission inventories, specific data collection efforts with particular industries, or information from regional, state, or local agencies. Other information, such as geographic location of release points, temporal pattern of emissions (e.g., periodic "puffs" vs. constant emission rates), and release height may be necessary, depending on the level of detail needed or types of exposure examined in the assessment.

3.2.1.1 Approach

The majority of emissions information used in the national-scale assessment was extracted from EPA's National Toxics Inventory (NTI), which contains estimates of the emissions of the 188 air toxics listed in section 112(b) of the CAA. In addition to the NTI, EPA's National Emission Trends (NET) inventory was also used as a source of emissions data for air toxics precursors in order to estimate air toxics generated through secondary formation in the atmosphere and for diesel particulate estimates for Puerto Rico and the Virgin Islands. Diesel particulate emission estimates for the contiguous U.S. were extracted from an inventory developed separately by EPA (see further details on this in section 3.2.1.2).

Before the emissions data from the NTI, NET, and diesel particulate inventories could be used as input to dispersion modeling, the emissions data required significant preparation. Some of this preparation work occurred during the compilation of the inventories and some occurred in the Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP) which is a series of computer programs that process emission inventory data for subsequent air quality modeling. Appendix C provides the EMS-HAP User's Guide and the methodologies used to process the emission data for this assessment. The necessary inventory preparation steps are described below:

- Compiling detailed air toxics, air toxics precursors, and diesel PM emissions inputs for all known stationary and mobile sources. The sources of this data are described in section 3.2.1.3.

- Performing quality assurance of the point source inventory location and stack parameter data and incorporating defaults for missing or erroneous data where possible. In some cases, missing or erroneous information can be found using the Toxics Release Inventory database. EMS-HAP defaults missing point source locations information to the centroid of the zip code (if zip code information is available) or to a census tract within the county. Where possible, stack parameters are defaulted based on the source category classification code or standard industrial classification code. Where not possible (lack of code in the inventory or lack of data for a particular SCC or SIC code, stack parameters) are set to conservative defaults (10 meter stack height, 1 meter stack diameter, 1 meter/second velocity, and a temperature of 295 Kelvin).

- Grouping individual pollutant species into compound groups. The NTI contains approximately 400 different species representing the 188 air toxics listed in section 112(b) of the CAA. Many of the species belong to compound classes. Grouping of these species is necessary for many reasons. One reason is that the individual chemical species belonging to groups are not geographically representative. For example, "lead oxide" may have been reported in just a few counties, whereas other counties aggregated their lead oxide emissions into "lead compounds." Grouping allows for pollutants with similar characteristics to be modeled together for purposes of efficiency. For example, specific lead species and compounds reported as the broad group "lead compound" are grouped to be subsequently modeled as "lead compounds-fine" and "lead compounds-coarse." These groups allow ASPEN to distinguish between the different deposition rates for fine and coarse lead particulates.

- Assigning each pollutant to a reactivity class (high, medium, low, etc.) or particulate size class (fine or coarse particulate) to allow for the ASPEN model to perform decay and deposition calculations. These reactivity classes are based on the rate of reaction of the pollutant with OH and NO3 radicals and had previously been established for the gaseous air toxics in the Cumulative Exposure Project. [*11*]

- Temporally allocating emission values to eight annual 3-hours emission rates. Emissions are temporally allocated based on the type of source using a database of temporal profiles by source classification code. The majority of the profiles are from a database originally developed for regional emission modeling studies under the National Acid Precipitation program. The factors for the National-Scale Air Toxics Assessment consolidate the seasonal and day-of-week classes in order to reflect hourly activity for an annually averaged day.

- Grouping all source categories into the following four source sectors: 1) major, 2) area and other, 3) on-road, 4) non-road. These sectors and the methodology for grouping are discussed below.

  All of the raw inventory inputs for this assessment exist as estimates for point

sources, non-point stationary sources, and mobile sources. "Point" sources provide emissions data at the facility and sub-facility level and include location coordinates (e.g., latitude and longitude). "Non-point" stationary source and "mobile" source data exist as emissions estimates for an entire source category aggregated to the county level. Inventory data files for these different types of sources are generally maintained separately and include different data elements. For the purpose of aggregating air toxics emission sources in the national-scale assessment, all emissions inventory inputs were grouped into four sectors: "major," "area and other," "onroad," and "nonroad." Dispersion modeling was performed for each sector separately, so that concentration estimates of each air toxic could be attributed to each sector. Each sector is further defined as follows:

Major sources are large stationary sources that emit more than 10 tons per year of any listed air toxic (CAA, section 112(b)) or a combination of listed air toxics of 25 tons per year or more. Typical examples of major sources include electric utility plants, chemical plants, steel mills, oil refineries, and large hazardous waste incinerators. These sources may release air toxics from equipment leaks, when materials are transferred from one location to another, or during discharge through emissions stacks or vents.

Area and Other sources are smaller stationary sources that emit less than 10 tons per year of a single air pollutant or less than 25 tons per year of a combination of air toxics. The emission inventory includes facility data for some area sources and aggregated emission estimates at the county level for the remaining area sources. Typical examples of area sources include neighborhood dry cleaners and gas stations. Though emissions from individual area sources are often relatively small, collectively their emissions can be of concern particularly where large numbers of sources are located in heavily populated areas. "Other" stationary sources are sources that may be more appropriately addressed by other programs rather than through regulations developed under certain air toxics provisions (sections 112 or 129) in the Clean Air Act. Examples of other stationary sources include wildfires and prescribed burning, which have emissions that are being addressed through the burning policy agreed to by the EPA and the USDA. For this assessment, the "area" and "other" sectors have been combined in the calculations and presentation of the current national-scale assessment

Onroad mobile sources comprise vehicles used on roads and highways (e.g., cars, trucks, buses).

Nonroad mobile sources are all remaining mobile sources (e.g., trains, lawnmowers, construction vehicles, farm machinery).

Major and area source facilities are drawn from the "point" source inventory files, meaning those with known geographic locations (i.e., latitude and longitude). Area and other source categories that are aggregated as county-level emissions are drawn from the "non-point" source inventory files, meaning those stationary sources that do not have location coordinates but instead exist as county-wide

total emissions by source category. Onroad and nonroad sources exist as distinct sectors in the "mobile" source inventories and are also aggregated to the county level.

- Spatially allocating county-level emissions to the census-tract level using surrogate data, such as population, industrial land or roadway miles, which are available at the census-tract level. A description of the available surrogates for use with EMS-HAP is shown in Table 3-3. Tract-level emissions for a source category are computed based on the percentage of the matching surrogate in the tract for that county. For example, the consumer products usage source category is matched to population. If 10 percent of the population of the county is in tract A, then tract A gets 10 percent of the county's consumer products usage emissions.

Table 3-3. Spatial Allocation Factors (SAF) Developed for EMS-HAP

Code for set of SAFs	Surrogate	Definition	Origin of Data	How EPA developed the set of SAFs
SAF1	Residential land	USGS land use categories: Residential, plus one-third of mixed urban and built-up land plus one-third of other urban and built-up land	mid-70's to 80's	from CEP[a,b]
SAF2	Commercial land	USGS land use categories: Commercial and services, plus one-half of industrial and commercial complexes, plus one-third of mixed urban and built-up land plus one-third of other urban and built-up land	mid-70's to 80's	from CEP[a,b]
SAF3	Industrial land	USGS land use categories: industrial, plus one-half of industrial and commercial complexes, plus one-third of mixed urban and built-up land, plus one-third of other urban and built-up land	mid-70's to 80's	from CEP[a,b]
SAF4	Utility land	USGS land use category: "transportation, communications, and utilities"	mid-70's to 80's	from CEP[a,b]
SAF6	Sum of commercial land and industrial land	Sum of commercial land and industrial land, as defined above	mid-70's to 80's	land use data from developers of CEP[a,b], SAF recomputed
SAF7	Farm land	USGS land use category: "cropland and pasture"	mid-70's to 80's	from CEP[a,b]
SAF8	Orchard land	USGS land use category: "orchards, groves, vineyards, nurseries, and ornamental horticultural areas"	mid-70's to 80's	from CEP[a,b]
SAF9	Confined feeding	USGS land use category "confined feeding"	mid-70's to 80's	from CEP[a,b]
SAF10	Farm land & confined feeding	USGS land use categories "cropland and pasture" plus "confined feeding"	mid-70's to 80's	from CEP[a,b]
SAF12	Rangeland	USGS land use categories: "herbaceous rangeland" plus "scrub and brush" plus "mixed rangeland"	mid-70's to 80's	from CEP[a,b]
SAF13	Forest land	USGS land use categories: "deciduous forest" plus "evergreen forest" plus "mixed forest land"	mid-70's to 80's	from CEP[a,b]
SAF14	Rangeland & forest land	Sum of rangeland and forest land, as defined above	mid-70's to 80's	from CEP[a,b]

Code for set of SAFs	Surrogate	Definition	Origin of Data	How EPA developed the set of SAFs
SAF15	Water	US Census category: water area	1990	from CEP[a,b]
SAF17	Mining & quarry land	USGS land use category: "strip mines, quarries, and gravel pits"	mid-70's to 80's	from CEP[a,b]
SAF18	1/population density	Inverse of: census tract population (defined above) divided by census tract area. Tracts with zero population assigned spatial factors of zero.	1990	from CEP[a,b]
SAF19	1/population density	Inverse of: census tract population (as defined above) divided by census tract land area. Tracts with zero population assigned tract population of one.	1990	population and land area data from CEP[b], SAF recomputed.
SAF20	Population	U.S. Census category: 1990 residential population	1990	from CEP[a,b]
SAF21	Railway miles	Total railway miles, as reported in TIGER/Line	1993	from CEP[a,b]
SAF22	Roadway miles	Total miles of all roadway types in each census tract, as reported in TIGER/Line	1993	from CEP[a,b]
SAF24	50% Population & 50% roadway miles	Surrogate based equally on population fraction and on roadway mile fractions for each of four roadway types	1990-93	0.5*SAF20 + 0.5*SAF22
SAF25	25% Population & 75% roadway miles	Surrogate based on population fraction and roadway mile fractions, respectively weighted by 25% and 75%, for each of four roadway types	1990-93	0.25*SAF20 + 0.75*SAF22
SAF26	Tract area	The area of census tracts (including land and water)	1990	tract areas computed from CEP tract radii[b] data SAF recomputed.
SAF27	Urban: Inverse population density Rural:farmland	Inverse population density (18) for urban[c] counties; farmland (7) for rural[c] counties	1990, mid-70's to 80's	SAF 18 from CEP, SAF 7 from CEP, urban/rural county designations from 1990 and 1996 census data
SAF28	Urban: population Rural: tract area	Population (20) for urban[c] counties; tract area for (26) rural[c] counties	1990	SAF 20 from CEP, SAF 26 from CEP, urban/rural county designations from 1990 and 1996 census data
SAF29	Sum of farmland and orchard land	Sum of farmland and orchard land, as defined above	mid-70's to 80's	land use data from developers of CEP[a,b], SAF recomputed.

[a] except that changes were made to SAFs in Halifax and South Boston in Virginia
[b] except for census tracts in the Virgin Islands and Puerto Rico which were not modeled in the CEP
[c] the designation of urban and rural counties is based on data from the U.S. Census Bureau as follows: a county is considered "urban" if either: 1) it includes a metropolitan statistical area with a population greater than 250,000; or, 2) the U.S. Census Bureau designates more than fifty percent of the population as "urban."

3.2.1.2 Scope

The NTI was the source of the majority of emissions inputs to the national-scale assessment. It contains estimates of air toxics emitted from many anthropogenic source categories for 188 hazardous air pollutants, for the 50 U.S. states, District of Columbia, Puerto Rico and Virgin Islands. With a few exceptions (e.g., wildfires), the NTI does not include emissions of air toxics from natural sources, indoor sources, or accidental

releases. Sources which are not included in the NTI were also excluded from the initial national-scale assessment.

For the purposes of the national-scale assessment, the pollutants were limited to 32 of the 33 air toxics included in EPA's Integrated Urban Air Toxics Strategy [2] (dioxins were not included) and diesel particulate matter. The geographic domain was limited to the contiguous 48 states, District of Columbia, Puerto Rico and Virgin Islands. Background documentation on the development of the 1996 NTI are included in Appendix D or can be accessed on the EPA web site at http://www.epa.gov/ttn/chief/nti. The detailed 1996 NTI data files are available at ftp://ftp.epa.gov/EmisInventory/nti_96.

The NET provides EPA's latest estimates of national emissions for criteria air pollutants: carbon monoxide (CO), nitrogen oxides (NOx), volatile organic compounds (VOCs [excludes certain non-reactive organic compounds]), sulfur dioxide (SO2), particulate matter less than 10 microns (PM10), particulate matter less than 2.5 microns (PM2.5), and lead (Pb). [*12*]

To account for secondary formation of volatile air toxics species, point, non-point stationary and mobile source emission data were input to the ASPEN model for non-air toxics VOC species resulting from a speciation of the 1996 NET (Version 3) inventory. These data were included to account for secondary formation of volatile air toxics species. In addition, NET data were used to estimate diesel particulate emissions for Puerto Rico and the Virgin Islands. The raw NET data are available at ftp://ftp.epa.gov/EmisInventory/net_96.

The diesel PM emissions data used for the 1996 national-scale assessment were extracted from an inventory developed as part of the rulemaking on Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements [*13*]. This inventory is based on Federal Highway Administration estimates of truck operation, estimates of the distribution of fuel type and weight classes of truck from the EPA Office of Transportation Air Quality (OTAQ), and emission factors provided by OTAQ. The nonroad emissions in this inventory were derived from OTAQ's draft June 2000 NONROAD model run for 1996. Both of these PM inventories reflect changes in methods and data sources since the release of versions used for the 1996 NET and 1996 NTI. Time did not allow for estimates of other pollutants from diesel vehicles and equipment to be revised accordingly, but an exploratory analysis indicated that the effect on estimates of other air toxics would not have been large. The diesel particulate emissions are all found in the onroad and nonroad source sectors. More details on the diesel PM inventories can be found at http://www.epa.gov/otaq/hdmodels.htm.

The NET, diesel particulate, and early versions of the 1996 NTI inventories all contain emissions estimates for the 50 states. The geographic domain of the national-scale assessment included the contiguous United States, Puerto Rico, and the Virgin Islands. In all cases, emissions for the territories included in the assessment were derived in part or in total via extrapolation of emissions estimates from surrogate U.S. locations.

3.2.1.3 Sources of Data

EPA prepared the 1996 NTI using various sources of data. The five primary sources of 1996 NTI data are: (1) state and local air toxics inventories developed by state and local air pollution control agencies, (2) existing databases related to EPA's Maximum Achievable Control Technology (MACT) program which requires emission standards under Section 112(d) of the CAA. (www.epa.gov/ttn/uatw/eparules.html) (3) Toxics Release Inventory (TRI) data (www.epa.gov/tri/), (4) emissions estimated by using mobile source methodologies developed by experts in EPA's Office of Transportation and Air Quality, and (5) emission estimates for 30 of 500 non-point source categories generated using emission factors and activity data. Much of the state/local, TRI, and MACT emissions data may have been generated by the sources themselves. The 1996 NTI is the first national modeling emission inventory constructed using state and local air toxic inventory data and containing stationary and mobile source data.

To improve the quality of the draft 1996 NTI, EPA requested comments on the inventory from state and local agencies, industry and others. The draft 1996 NTI and documentation were available to all parties for review and comment from April 30, 1999 - August 13, 1999. A state-only preview of the ASPEN concentration results from March 31, 2000 to May 12, 2000 resulted in additional state/local revisions to the NTI. The EPA received extensive comments and revisions from industry, state and local agencies, and others located in 42 states, Puerto Rico, and the Virgin Islands. Figure 3-1 shows the states and local agencies that ultimately provided inventory data, comments, and revisions to the EPA. Forty-one states provided point source revisions, 27 states provided non-point stationary source revisions and 18 states provided mobile source revisions. All requested revisions were evaluated and incorporated as appropriate into the NTI prior to final ASPEN modeling. Documentation for all emissions estimates in the 1996 NTI is available in Appendix D.

3.2.1.3.1 Point Source Emissions

In compiling point source emissions information for the NTI, preference was given to state- and locally-generated information where available except for utility mercury emissions and municipal waste combustion emissions. While agencies are neither required to compile air toxics emissions data nor to submit such data to EPA, most states did respond to EPA's invitation to do so. EPA did not apply many quality checks on state/local data before incorporating it into the NTI, but in the course of three rounds of state/local review of the draft NTI and national-scale assessment results, many anomalies were noted by EPA or the state/local agencies and resolved by corrections or deletions. Where state or local agency data were not provided, existing emissions data from EPA's regulatory development (MACT) databases were utilized. If these databases differed in pollutant coverage, MACT data were used to add any missing emissions in order to be as comprehensive in pollutant coverage as possible. If neither of state/local or MACT sources contained information for a known stationary source, the NTI used estimates based on information from EPA's TRI.

Most state agencies participated in the development of the 1996 NTI. Forty-seven of the 50 states, Puerto Rico, the Virgin Islands, and the District of Columbia participated in the development of the 1996 NTI either by providing emissions data or by reviewing the

draft inventory. Thirty-six states initially provided draft 1996 air toxics emissions inventories. The data collected from the state or local agencies varied significantly in terms of completeness, coverage, format, and quality. The majority of the 36 states provided data for point sources (primarily large industrial sources which would be defined as major sources). The number of air toxics included in the state or local agency inventories varied. Some state and local agencies compile emission inventories for fewer than the 188 air toxics (e.g., the RAPIDS inventory of Great Lakes states), while other states such as California and Louisiana compile emission inventories for more than the 188 air toxics.

In addition to the 14 states that did not provide draft air toxics inventory data, several data gaps were identified in the state databases provided. Data gaps in the state inventories included: (1) emissions from entire counties missing from the state databases; (2) missing emission sources; (3) lack of stack parameters; and (4) lack of facility location data (latitude/longitude or Universal Transverse Mercator (UTM) coordinates).

In order to find missing facilities for entire states, counties, or individual facilities, EPA compared existing state air toxics inventories first to MACT data, then to TRI data, and then to NET data. For most facilities that appeared in both the state database and in either the MACT or TRI database, EPA assumed that the state databases were more accurate and, thus, no further revisions were made. This assumption of the data quality hierarchy was necessary due to resource limitations that prohibited EPA from comparing various emission estimates for a given facility, obtaining documentation for the disparate estimates, and choosing the most appropriate information across data bases. In at least one case, this hierarchy resulted in the omission of significant emissions in the national-scale assessment. A major source of lead, a lead smelter in Missouri, does not show any lead emissions in the version of the 1996 NTI modeled for this assessment. This resulted because the state agency's emissions estimates for this facility were used but erroneously did not include the lead emissions. Since the facility was already present in the NTI from the state's inventory, TRI estimates were not added. Later, after ASPEN modeling, it was discovered that TRI contained nearly 100 tons of lead emissions for this facility. It is possible that other facilities and emissions could have been omitted from this assessment. Similarly, it is possible that a facility could have been double counted if it appeared in two or more inventories, but with different facility names. These and other potential sources of error are discussed in subsequent sections of this report.

Because the ASPEN model requires a model-ready inventory, the association of stack parameters and location data to each facility's emission estimate was required. If this information was found to be missing, incorrect, or out of range, it was corrected with defaults. Defaulting schemes were performed in both the NTI development and in the subsequent processing in EMS-HAP. Default stack parameters associated with Standard Industrial Classification (SIC) codes were used for emissions reported at the SIC code level. Also default stack parameters associated with source classification codes (SCCs) were employed for emissions reported at the SCC level. Where default parameters were available by either SCC or SIC, the SCC took precedence.

In order to develop appropriate defaults for missing location data, first comparisons were

made to other inventories (e.g., TRI, NET, or the Ozone Transport Assessment Group (OTAG) inventory). Where latitude/longitude coordinates were found they were added to the NTI. Where the facilities could not be matched to other databases, defaulting schemes were employed to place facilities within known zip codes or counties. These schemes are explained in the NTI documentation for major sources (Appendix D) and in the EMS-HAP User's Guide (Appendix C). EPA assigned locations to all facilities except for 87 facilities that were defined as "portable" in Colorado and Idaho. These were not included in the assessment.

3.2.1.3.2 Non-Point Emissions

Where possible, EPA compiled the non-point stationary source emissions (which are included in the "area and other" source sector) from the 1996 state emission data sets. The majority of the 36 states that initially supplied draft 1996 air toxics emission inventories did not provide non-point source data. EPA evaluated and supplemented the state data sets with non-point source data gathered during the development of MACT standards and with TRI data. EPA then generated non-point emission estimates for 30 remaining source categories (listed in Table 3-4) by using activity data and emission factors and then allocating the estimates from the national, state, or regional level to the individual counties. EPA was careful to avoid duplicating emissions (e.g., emissions from large dry cleaners included in point source files would be subtracted from non-point source calculations). For example, if the non-point source estimates were based on raw material usage, the point source fraction already accounted for in this fashion was subtracted prior to calculating the non-point source emissions.

Table 3-4. Non-Point Source Stationary Categories with EPA-Derived Emissions.

Categories

Animal Cremation
Asphalt Paving: Cutback Asphalt
Auto body Refinishing Paint Application
Aviation Gasoline Distribution: Stage I & II
Consumer Products Usage
Dental Preparation and Use
Drum and Barrel Reclamation
Fluorescent Lamp Recycling
Food and Agricultural Products: Cotton Ginning
Gasoline Distribution Stage II
General Laboratory Activities
Geothermal Power
Hospital Sterilizers
Human Cremation
Lamp Breakage
Miscellaneous Organic Chemical Processes
Open Burning: Forest and Wildfires
Open Burning: Prescribed Burnings
Open Burning: Scrap Tires
Pesticide Application
Residential Fuel Use: Anthracite Coal
Residential Fuel Use: Bituminous and Lignite Coal
Residential Fuel Use: Distillate Oil
Residential Fuel Use: Natural Gas
Residential Fuel Use: Wood/Wood Residue
Softwood Drying Kilns
Structure Fires
Surface Coating Operations: Architectural
Surface Coating Operations: Traffic Markings
Surface Coating Operations: Industrial Maintenance

3.2.1.3.3 Mobile Sources

The EPA's Office of Transportation and Air Quality (OTAQ) provided direction and advice on which emission factors and speciation profiles should be used in the development of air toxics

emission estimates for mobile sources. The mobile source estimates were determined using a combination of methods. For highway mobile sources, emission factors for benzene, 1,3-butadiene, formaldehyde and acetaldehyde were modeled for 10 urban areas and 16 geographic regions, using OTAQ's MOBTOX5b model [*14*]. The urban areas and geographic regions modeled were selected to encompass a broad range of inspection and maintenance (I/M) programs, fuel parameters, and temperature regimes. The intent of the selection process was to best characterize the different combinations needed to perform accurate nationwide toxic emissions estimates. Every county in the U.S. was then "mapped" to one of these modeled areas or regions (i.e., the emission factor for the modeled area was also used for the area "mapped" to it). Mapping was done based on a combination of geographic proximity, I/M program, and fuel control programs. The resulting county level emission factors were then multiplied by county-level VMT estimates. To estimate emissions of other air toxics from highway mobile sources, data from speciation profiles were applied to VOC emissions for gaseous air toxics and POM, and to PM emissions estimates for metal emissions and dioxins (although dioxin emissions were subsequently removed from this study). Where emissions of gaseous compounds were impacted by the use of reformulated gasoline or winter oxygenated gasoline, these impacts were accounted for at the county level.

The 1996 NTI contains nonroad mobile emission estimates for 2- and 4-stroke gasoline-powered engines, diesel engines, aircraft, locomotives and commercial marine vessels. For gasoline-powered engines and diesel engines, data from speciation profiles were applied to county level VOC and PM estimates generated by the April, 1999 draft version of the EPA NONROAD model. Again, impacts of gasoline fuel control programs on emissions were accounted for at the county level. Emissions of aircraft, locomotives, and commercial marine vessels were estimated by applying data from speciation profiles to national VOC and PM estimates, then using activity data to allocate nationwide emissions to the county level.

3.2.2 Environmental Fate and Transport Characterization

After the pollutants of interest and their sources and emission rates are defined, the exposure assessment process continues with estimation of pollutant fate and transport in the atmosphere. This step describes how the pollutant is transported, dispersed, and transformed over the area of interest. Initially, the fate of the emitted pollutants is largely determined by the source release characteristics. After pollutants are released to the atmosphere, their transport, dispersion, and transformation are governed by meteorological principles, terrain characteristics, wet and dry deposition rates, and certain chemical properties of the air toxics (e.g., aqueous solubility, vapor pressure, molecular diffusivity, melting point, and adsorption characteristics). For a limited subset of air toxics, it may be important to consider deposition from air to soil, vegetation, or water bodies. For others, such deposition is not important.

Various mathematical models (e.g., Gaussian puff models, Gaussian plume models) [*15*], each with specific data needs, have been developed or are under development to describe the transport and fate of pollutants released to the atmosphere. The model chosen must be appropriate for the intended application, which may range from estimates of short-term peak concentrations immediately adjacent to a facility, to long-term concentrations

over a citywide area or deposition over thousands of miles. The reactivity and persistence of each air pollutant will also influence its fate, and these factors can be important in estimating exposure for certain pollutants. Additionally, secondary transformation products of some air toxics may need to be identified for consideration in risk assessment. Any available air toxics monitoring data can be used either to check the validity of modeled concentration estimates or as a primary or supplemental source of information for the exposure assessment itself.

For a limited subset of air toxics, greater exposures occur through non-inhalation exposures than through inhalation exposures. These air toxics typically are persistent in the environment and have a strong tendency to bioaccumulate. Exposure assessments can consider exposures that occur through routes other than inhalation by using multipathway models. The simplest multipathway exposure assessments require chemical-specific data (e.g., octanol-water partition coefficient) to model the partitioning of the chemical in the environment, and uptake rates (e.g., consumption rate of drinking water) to predict intakes. Combining this information yields general predictions of non-inhalation exposure. EPA's current national-scale exposure models do not have the capability to quantify non-inhalation exposures, so they were not included in this initial assessment, although this may become possible in the future.

3.2.2.1 Overview of the ASPEN Dispersion Model

To develop national-scale estimates of annual average ambient concentrations of air toxics, EPA used the Assessment System for Population Exposure Nationwide (ASPEN) model that was developed and used in EPA's Cumulative Exposure Project (CEP)[*16*].

In general, ASPEN uses a Gaussian model formulation and climatological data to estimate annual average pollutant concentrations at the centroid of each census tract within the modeling domain. Specifically, for each source, the model calculates sets of eight 3-hour ground-level concentrations as a function of radial distance and direction from the source at a set of receptors laid out in a radial grid pattern. These concentrations represent the steady-state concentrations that would occur with constant emissions and meteorological parameters. For each grid receptor, concentrations are calculated for each of a standard set of stability class/wind speed/wind direction combinations. These concentrations are averaged together using the annual frequency of occurrence of each combination (i.e., the climatology) as weightings.

These meteorological frequency distributions are typically prepared for the entire simulation period, usually one or more years. For ASPEN, however, meteorological data are stratified by time of day into eight 3-hour time blocks, to preserve any characteristic diurnal patterns that might be important in subsequent estimation of population exposure. In addition to the climatology, other inputs to ASPEN that are specified by time block include emission rate, mixing height, and reactive decay rates. The resulting output of ASPEN is a grid of annual average concentration estimates for each source/pollutant combination by time block.

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), transformation of one pollutant into another (i.e., secondary formation).

For all pollutants outdoor concentrations include a "background" component. Background is an essential part of the total air quality concentrations. Background includes concentrations due to natural sources, sources not in the emissions inventory, and long-range transport. In this study, except for diesel PM, background concentrations are represented by concentration values measured at "clean air locations" where available. Non-zero background values for 13 pollutants, identified from the technical literature and reported in the CEP study [16] were used to sum with the ASPEN-estimated concentrations in each census tract. Except for diesel PM, the background value is assumed to be constant for all census tracts due to insufficient data for assessment of geographic variations. Where background concentration values were not identified in the technical literature and reported in the CEP study for other air toxics, their background concentrations are assumed to be zero. This result may be an underestimate of outdoor concentrations in some cases.

Annual average concentration estimates for grid receptors surrounding each emission source are spatially interpolated to the census tract centroids within the 50 kilometers impact zone, and contributions from all modeled sources are summed to give estimates of cumulative ambient concentration increments in each census tract [*17*]. By accounting for all identified source categories (including background concentrations), the sum of the concentration increments should yield an estimate of the overall concentration of each air toxic within each census tract. These estimates are designed to represent population-weighted concentration averages (each census tract generally represents between 2,500 and 8,000 people). More detailed information on ASPEN is provided in the ASPEN User's Guide (Appendix E).

3.2.2.2 Application of ASPEN for the Initial National-Scale Assessment

For this assessment, the ASPEN model was run in the same manner (e.g., reactivity class assignments, wet and dry deposition for particulates, secondary formation) as was done in the CEP, with some exceptions:

1. The input inventories were different in data year, approach and some emissions processing techniques. For example, location and stack parameter defaulting techniques were different. Changes were also made to spatial allocation and temporal allocation factors for some emission sources. See section 3.2.1 for a description of inventories and how they were prepared for the ASPEN model.

2. In 1996, there was 75 percent completeness of meteorological data at 357 National Weather Service surface stations versus 214 in 1990. Data from the 357 stations were used for this assessment. Due to the use of an increased number of stations, the average distance between the emission source and the meteorological station improved (decreased) in the 1996 ASPEN run.

3. The modeling domain was extended to Puerto Rico and the Virgin Islands.

4. For diesel PM only, instead of using monitored air quality data to establish background concentrations, a modeling-based approach was developed to provide a rough approximation of concentrations due to transport from sources located between 50 km and 300 km from the receptor. See Appendix F for a more complete discussion of this approach.

To evaluate the quality of ASPEN outputs as a check on input and model execution accuracy, predicted concentrations of toxic pollutants were examined graphically to establish a framework from which "unusual" data values might be identified. The most effective inspection method proved to be the preparation of matrix scatter plots in which predicted concentrations of each pollutant were plotted against the concentrations of all other pollutants at that location. Generally, these plots showed high correlation among pollutants that had common sources (e.g. benzene, toluene, ethyl benzene) and low correlation between pollutants that did not generally share common sources (e.g. benzene and vinyl chloride). Plots were developed separately for each state using the total predicted concentration from ASPEN, but also for each major source component including mobile onroad, mobile nonroad, point and non-point source contributors. Using these plots along with sorted rankings of pollutant concentrations, predicted values were identified that either appeared very large and/or inconsistent with other pollutant levels. These suspect values were then subjected to verification and/or correction through discussion with emission inventory and modeling experts. From this process, a significant number of errors were corrected before the final ASPEN modeling was performed.

3.2.3 Estimating Population Exposure

In the third step of the initial national-scale assessment, ambient air toxics concentrations derived from ASPEN modeling were used to estimate human exposures to air toxics. Inhalation was the only exposure route considered for this initial assessment. For characterization of personal exposure, a model was needed that would allow for consideration of inhalation exposures to various population groups, who may have different levels of exposure as a result of differences in proximity to sources of exposure (due to location of residence, occupational setting, etc.).

An inhalation exposure model, the Hazardous Air Pollutant Exposure Model, Version 4 (HAPEM4) was selected for use to estimate personal exposure and account for differences in exposures among the population.

3.2.3.1 Overview of HAPEM4

The HAPEM4 is an exposure model that is capable of assessing average long-term inhalation exposures of the general population, or a specific sub-population, over spatial scales ranging from urban to national. HAPEM4 utilizes a relatively transparent set of exposure assumptions and approximations (Appendix B). HAPEM4 uses the general exposure modeling approach of tracking representatives of specified demographic groups as they move among indoor and outdoor microenvironments and between geographic

locations performing various activities. Figure 3-2 shows example demographic groups, microenvironments, and activities. The estimated pollutant concentrations in each microenvironment visited are combined into a time-weighted average exposure concentration, which is assigned to members of the demographic group.

HAPEM4 uses four primary sources of information: ambient air concentration data, population data, population activity data, and microenvironmental data. HAPEM4 also contains several special features to improve its exposure prediction capabilities. These data sources and features are described in more detail below.

Ambient Air Concentration Data

The HAPEM4 requires annual-averaged, diurnally-distributed air quality data. In addition, HAPEM4 can also evaluate the contributions of sub-sets of the air quality data (e.g., air concentration values for specific source sectors such as point source, area source, mobile source). While the air concentration data for HAPEM4 must be in a specific format (e.g., annual average and diurnally distributed), the source of the data could be either from an air dispersion model or an ambient monitor. The most common form of ambient air concentration data for HAPEM4 is output data in the ISCLT format.

Population Data

The U.S. Census Bureau is the primary source of most population demographic data. The U.S. Census Bureau collects information on where people live, their demographic makeup (e.g., age, gender, ethnic group), and employment. The HAPEM4 model uses 1990 U.S. Census data reported at the spatial resolution of census tracts, which are small, relatively permanent statistical subdivisions of a county. Census tracts usually contain between 2,500 and 8,000 residents.

Population Activity Data

HAPEM4 uses two types of population activity data: activity pattern data and commuting pattern data. An activity pattern is a series of discrete events of varying time intervals describing an individual's lifestyle and routine. An activity pattern typically includes the amount of time spent in each of a set of microenvironments (e.g., at home, at work, in an automobile, etc.), and a description of what the individual was doing in each microenvironment (e.g., sleeping, eating, exercising, etc.). EPA's Consolidated Human Activity Database (CHAD) [*18*], containing more than 22,000 person-days of activity pattern records from 12 studies, is incorporated into HAPEM4.

Because activity data are not available at a high enough resolution to estimate the exposure of each individual in the population, HAPEM4 groups activity pattern data together for people with similar demographic characteristics that are expected to influence exposure to air pollutants (e.g., age, gender, race), and HAPEM4 estimates exposures for these demographic groups.

Annual average activity pattern sequences are built by randomly selecting (with replacement) 365 daily diary entries. Day of the week, as well as season type, are considered in this selection process. It is important to note that construction of an annual

average activity pattern in this manner results in the loss of day-to-day correlations in activity patterns.

The commuting data contained in HAPEM4 have been derived from a special 1990 US Census database that specifies for each US Census tract the number of residents that work in each US Census tract, i.e., the population associated with each home tract/work tract pair.

Microenvironment Data

In order to calculate the exposure concentration for each demographic group, an estimate is required of the concentration in each microenvironment (ME) specified by the activity pattern. In HAPEM4, these ME concentration estimates are derived from the ambient concentration estimate for the census tract (obtained from ASPEN) and a set of 3 ME factors: PEN, PROX, and ADD. The penetration factor, PEN, is an estimate of the ratio of ME concentration to the concurrent outdoor concentration in the immediate vicinity of the ME. These pollutant-specific estimates are derived from reported measurement studies. The proximity factor, PROX, is an estimate of the ratio of the outdoor concentration in the immediate vicinity of the ME to the outdoor concentration represented by the air concentration data. ADD is an additive factor that accounts for emission sources within or near a particular microenvironment, i.e., indoor emission sources.

In HAPEM4, these ME concentration estimates are derived from the ambient concentration estimate for the census tract (obtained from ASPEN) and a set of 3 ME factors as follows:

$$C_{(i,k,t)} = [ASPEN]_i \times PEN \times PROX + [ADD]$$

Where:

$C_{(i,k,t)}$ =	concentration predicted within exposure district i and microenvironment k in time step t
$[ASPEN]_i$ =	ambient concentration estimated from ASPEN in district i
PEN =	penetration factor
PROX =	proximity factor
ADD =	additive factor accounting for sources within the microenvironment

Features of HAPEM4

It is important to note that the HAPEM4 model has been designed to predict inhalation exposures for population groups, not individuals within these groups. However, the HAPEM4 model contains a stochastic feature to allow the exposure modeler to try and capture some of the variability in activity patterns within these groups. The stochastic feature predicts the annual exposure concentrations for a randomly selected set of 30 estimates for each demographic group.

The HAPEM4 contains a commuting feature that allows the movement of people between home and work locations. In general, the user can specify whether a specific demographic group is a "commuting" group or a "non-commuting" group. For work

related activities, the "commuting" group is then placed in a different census tract location. A default file, developed from 1990 US Census data, that depicts tract-to-tract commuting patterns is included with the HAPEM4 model. The model can be run either with or without the commuting feature.

Details on both of these features can be found in the HAPEM4 User's Guide (Appendix B).

3.2.3.2 Application of HAPEM4 for the Initial National Scale Assessment

For this assessment, the model was applied on the national scale. HAPEM4 estimated personal inhalation exposures from ambient air toxics concentrations by defining the exposed population in terms of geographic and demographic distributions and then accounting for the various microenvironments to which people may be exposed. These microenvironments were addressed by considering the time people may spend in each of these microenvironments and the air toxics concentration in those microenvironments relative to the ambient air toxics concentrations predicted by ASPEN.

Ambient Air Concentration Data

For the national-scale assessment, annual average ambient concentrations for each US Census tract were estimated with the ASPEN model. In order to preserve any characteristic diurnal patterns in ambient concentrations that might be important in the estimation of population exposure, ASPEN annual average concentration estimates are stratified by time of day, with an annual average for each of the (8) 3-hour time blocks (e.g., midnight to 3am, 3am to 6am). ASPEN air quality files were also provided by each of the 4 major source sectors (i.e., major, area, mobile onroad, mobile nonroad). Thus, the results of HAPEM4 can be summarized for each of the 4 groups or a combination of them.

Population Data

For this application, HAPEM4 divided the population into 40 demographic groups, based on combinations of age (5 categories), race (4 categories), and gender. Figure 2-3 depicts the 40 demographic groups identified and used for the initial assessment.

Population Activity Data

For each demographic group, 365 activity patterns were selected randomly (with replacement) and then combined to develop the average fraction of time in each of the 37 microenvironments for each of the (8) 3-hour time blocks. One hundred such annual activity patterns were constructed for each demographic group. Then, for each US Census tract, 30 of the 100 annual patterns were randomly selected (with replacement) to represent typical annual time allocations for group members in that tract. The result was a set of 30 annual exposure concentrations estimates for each demographic group in each census tract. Figure 3-3 shows an example of a daily inhalation exposure scenario and calculations performed to derive a daily exposure value. Because each component of the national-scale assessment has been designed to predict more "typical" or population-based estimates rather than extremes or individuals, the HAPEM4 inhalation concentrations presented for this study, as well as that employed for the subsequent risk

characterization, were derived and aggregated using the median exposure concentration from this group of 30 concentrations.

Microenvironment Data

For most MEs, HAPEM4 uses a PROX value of 1.0. However, when assessing exposure to motor vehicle emissions, for MEs near roadways (e.g., in-vehicle), the pollutant concentration contribution in the immediate vicinity of the ME is generally higher than the average pollutant concentration contribution over the census tract. Thus, PROX values of greater than 1.0 are used. For this application, which addresses only exposure resulting from outdoor emission sources, ADD was uniformly set equal to zero. A complete listing of the ME factors employed in the national scale assessment for each pollutant are presented in the report "Development of Microenvironmental Factors for the HAPEM4 in Support of the National Air Toxics Assessment (NATA) - External Review Draft Report"[*19*]. A summary of the peer review comments on this report and how they were addressed in this assessment is included in Appendix B.

3.3 Dose-Response Assessment

3.3.1 Introduction

Within EPA's paradigm for risk assessment (Figure 2-1, outer circle), the dose-response assessment phase of a risk assessment is based on two sequential analyses. The first analysis is the *hazard identification*, which identifies contaminants that may pose health hazards at environmentally relevant concentrations and qualitatively describes the effects that may occur in humans. The second analysis is the human health *dose-response assessment*, which characterizes the relationship between the concentration, exposure, or dose of a pollutant and the resultant health effects.

The types of effects relevant to each chemical (e.g., cancer, or effects other than cancer) are determined as part of the hazard identification. Factors such as the experimental route of exposure, the type and quality of the effects, the biological plausibility of findings, and the consistency of findings across studies, all contribute to the nature of the hazard identification statement.

The nature of quantitative dose-response assessment typically varies among pollutants. Sufficient data often exist for criteria air pollutants, such as ozone or carbon monoxide, so that relatively complete dose-response relationships can be characterized. In such cases, there is no need for extrapolation to lower doses because adequate human health effects data are available at environmentally relevant levels. However, this has not been the case for most air toxics. Epidemiologic and toxicologic data on air toxics have typically resulted from exposure levels that were high relative to environmental levels.

Generally, dose-response assessment methods for air toxics consist of two parts. First is the evaluation of data in the observable range, and second is the extrapolation from the observable range to low doses/risks. Recent terminology refers to the result of analysis in the observable range as the "point of departure" from which extrapolation begins. The approaches used for evaluation in the observable range are similar for all types of effects,

but EPA's current extrapolation methods differ considerably for cancer and noncancer effects.

3.3.2 Cancer

3.3.2.1 Hazard Identification

The EPA's 1986 *Guidelines for Carcinogen Risk Assessment* [*20*] provide guidance on hazard identification for carcinogens in this assessment. This approach recognizes three broad categories of data: (1) human data (primarily epidemiological); (2) results of long-term experimental animal bioassays; and (3) supporting data, including a variety of short-term tests for genotoxicity and other relevant properties, pharmacokinetic and metabolic studies, physio-chemical properties, and structure-activity relationships. In hazard identification of carcinogens under the 1986 guidelines, human data, animal data, and supporting evidence are combined to characterize the weight-of-evidence (WOE) regarding the agent's potential as a human carcinogen into one of several hierarchic categories:

- Group A ! Carcinogenic to Humans: Agents with adequate human data to demonstrate the causal association of the agent with human cancer (typically epidemiologic data).

- Group B ! Probably Carcinogenic to Humans: Agents with sufficient evidence (i.e., indicative of a causal relationship) from animal bioassay data, but either limited (i.e., indicative of a possible causal relationship, but not exclusive of alternative explanations) human evidence (Group B1), or with little or no human data (Group B2).

- Group C ! Possibly Carcinogenic to Humans: Agents with limited animal evidence and little or no human data.

- Group D ! Not Classifiable as to Human Carcinogenicity: Agents without adequate data either to suggest or refute the suggestion of human carcinogenicity.

- Group E ! Evidence of Non-carcinogenicity for Humans: Agents that show no evidence for carcinogenicity in at least two adequate animal tests in different species or in both adequate epidemiologic and animal studies [*20*].

It is important to note that the WOE categories under the 1986 cancer guidelines express only a relative level of certainty that these agents may cause cancer in humans. The categories specifically do not connote relative level of hazard, or degree of conservatism applied in developing a dose-response assessment. For example, a substance in group C (possible human carcinogen) may very well impart a greater cancer risk to more people than another substance in group A (known human carcinogen). EPA has classified substances as "possible" carcinogens only because the amount and quality of evidence was insufficient to place them in a higher group, not because EPA believes they necessarily present less risk.

EPA's 1986 carcinogen risk assessment guidelines were the product of nearly two decades of experience and scientific consensus building. EPA has since gained considerable experience in applying cancer risk assessment approaches. Likewise, the science of risk assessment and toxicological testing has continued to evolve while EPA has had to address situations not explicitly discussed in the 1986 guidelines, e.g., children's risk assessment. The revision of EPA's carcinogen risk assessment guidelines that is currently underway will consolidate the Agency's experience, provide more comprehensive and transparent guidance on topics not fully developed in the original guidelines, and provide flexibility to accommodate anticipated advances in the science.

In a 1996 Federal Register notice (61 FR No. 123, 32799- 32801) EPA announced that, pending publication of the final revised guidelines, the principles and procedures of the draft revised guidelines would be applied in part or in whole on a case-by-case basis for new assessments. Further, the 1996 proposed guidelines represent the evolution of risk assessment methods rather than a "sea change" in those methods. Application of these approaches is felt to be reflective of EPA's accumulated experience and in keeping with advancing knowledge on cancer assessment and, therefore, provides the Agency with more experience to draw upon in finalizing the guidelines. Accordingly, substances in the current assessment that have EPA hazard identification and dose-response information developed since 1996 reflect the proposed cancer guidelines, using the most recent 1999 guidelines draft [*21*].

3.3.2.2 Dose-Response Assessment for Carcinogens

EPA's 1986 cancer risk assessment guidelines [*20*] adopted a default assumption that chemical carcinogens would exhibit risks at any dose. Extrapolation of cancer risk using the linearized multistage model, which results in a linear extrapolation of risk in the low dose region, was proposed as a reasonable upper-bound on risk (i.e., the true value of the risk is unknown, and may be as low as zero), and this approach has been used for most chemicals with adequate data since then. Extrapolation of cancer risk using other linear extrapolation models, such as have been used with human data available for some known human carcinogens (e.g., benzene, hexavalent chromium) results in estimates which, although conceivably surrounded by less uncertainty, are still characterized by the Agency as plausible upper bound estimates (i.e., the risk is likely to be lower but may be greater). Although the 1986 guidelines also supported the use of non-linear low-dose extrapolations (given adequate mechanistic data), until recently the low-dose linearity assumption has been used without exception in estimating carcinogenic potency.

Since the publication of EPA's original cancer guidelines, considerable new knowledge has been developed regarding the processes of chemical carcinogenesis and the evaluation of human cancer risk. The revision of the cancer guidelines currently in progress [21] departs substantially from the original guidelines by distinguishing between linear and nonlinear modes of action. The cancer data in the observable range are to be analyzed using a dose-response model similar to the models used for noncancer effects. The method of extrapolation to lower doses from the point of departure may vary depending on whether the assessment of the available data on the mode of action of the chemical indicates a linear or nonlinear mode of action.

Under the proposed guidelines, a linear extrapolation will remain appropriate when the evidence supports a mode of action of gene mutation due to direct deoxyribonucleic acid (DNA) reactivity or another mode of action that is thought to be linear in the low dose region. A linear mode of action also will serve as a default when available evidence is not sufficient to support a nonlinear extrapolation procedure, even if there is no evidence for DNA reactivity. The linear extrapolation method has also been revised and simplified from that employed under the 1986 guidelines. Nonlinear methods are to be used if there is sufficient evidence to support a nonlinear mode of action.

EPA's process of estimating cancer risk is based on the unit risk estimate (URE). A URE represents an estimate of the increased cancer risk from a lifetime (generally assumed to be 70 years) exposure to a concentration of one unit of exposure. The URE for inhalation exposures is typically expressed as risk per microgram of pollutant per cubic meter of air. The URE is a plausible upper-bound estimate of the risk (i.e., the risk is likely to be lower, but may be greater). EPA defines an upper bound as a plausible upper limit to the true value of a quantity. Because UREs reflect unquantifiable assumptions about effects at low doses, their upper bounds are usually not true statistical confidence limits. Available data were insufficient to support assumptions of threshold or sublinear dose-response for the substances in this assessment, so estimates of cancer risk were developed by linear extrapolation of the URE (i.e., by multiplying the estimated lifetime average daily exposure in micrograms per cubic meter by the URE). UREs used in this assessment were developed by EPA and by the California EPA, and selected for use by a priority system described in Appendix G.

3.3.3 Effects other than cancer

3.3.3.1 Hazard Identification

Due to the wide variety of endpoints, hazard identification procedures for effects other than cancer have not been described as completely in EPA guidance as procedures for the identification of carcinogens. However, the EPA has published guidelines for assessing several specific types of noncancer effects, including mutagenicity [*22*], developmental toxicity [*23*], neurotoxicity [*24*], and reproductive toxicity [*25*].

Under these guidelines for identification of long-term (chronic) hazards other than cancer, scientists from EPA (and from other agencies that assess dose-response relationships) review the health effects literature and characterize its strengths and weaknesses, using a narrative approach rather than a formal classification scheme. Available data on different endpoints are arrayed and discussed, and the effects (and their attendant dose/exposure levels) described. Particular attention is given to the *critical effect* (defined as the first adverse effect, or its known precursor, that occurs to the most sensitive species as the dose rate of an agent increases) in well-designed studies. Information is presented in a narrative description that discusses factors such as the methodological strengths and weaknesses of individual studies (as well as the overall database), the length of time over which the studies were conducted, routes of exposure, and possible biological modes of action. Assessors consider the severity of effects, which may range from severe frank effects that can cause incapacitation or death to

subtle effects that may occur at the cellular level but are early indicators of toxic effects. Not all effects observed in laboratory studies are judged to be adverse. The distinction between adverse and non-adverse effects is not always clear-cut, and considerable professional judgment is applied to identify adverse effects. All of these observations are integrated into a presentation that gives a concise profile of the toxicological properties of the pollutant.

3.3.3.2 Dose-Response Assessment for Non-Carcinogens

The inhalation reference concentration (RfC) is the primary Agency consensus quantitative toxicity metric for use in noncancer risk assessments for chronic inhalation exposure. The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC is derived after a thorough review of the health effects data base for an individual chemical, and identification of the most sensitive and relevant endpoint (i.e., the critical effect) and the principal study(ies) demonstrating that endpoint. Inhalation RfCs are derived according to the Agency's *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* [*26*]. The evaluation of and choice of data on which to base the RfC derivation are critical aspects of the assessment and require scientific judgment.

Derivation of the RfC typically begins with identification of the critical effect from the available valid human and animal study data, followed by identification of a lowest-observed-adverse-effect level (LOAEL) or, preferably, a no-observed-adverse-effect level (NOAEL). Some assessments model the dose-response relationship to interpolate a benchmark dose (BMD), usually the dose at which 5 percent of the organisms are predicted to respond. BMDs are used interchangeably with NOAELs. The LOAELs or NOAELs from animal studies are converted to human equivalent concentrations (HECs) using dosimetric methods [*26*]. The NOAEL[HEC] or LOAEL[HEC] from one or a few well-conducted studies are the key values gleaned from evaluation of the dose-response data. The RfC is then derived by consistent application of uncertainty factors (UFs) to account for recognized uncertainties in the extrapolation from the experimental data and exposure conditions to an estimate (the RfC) appropriate to the assumed human lifetime exposure scenario [*26*]. RfCs (and similar dose-response values) used in this assessment were developed by EPA, the US Agency for Toxic Substances and Disease Registry, and the California EPA. They were selected for use by a priority system described in Appendix G.

3.4 Risk Characterization

3.4.1 Introduction

Under EPA's risk assessment paradigm, the final product in the risk assessment process is the risk characterization, in which the information from the previous steps is integrated and overall conclusions about risk are synthesized and presented in a way that is appropriate and informative for decision-makers. In general, the nature of a risk characterization will depend on the information available, the intended use of the risk information, and the resources (including time) available. In all cases, however, major

issues associated with determining the nature and extent of the risk should be identified and discussed. Further, the EPA Administrator's March 1995 *Policy for Risk Characterization* [*27*] specifies that a risk characterization "be prepared in a manner that is clear, transparent, reasonable, and consistent with other risk characterizations of similar scope prepared across programs in the Agency." The 1995 *Guidance for Risk Characterization* [*28*] lists several guiding principles for defining risk characterization in the context of risk assessment. The three principles with respect to the information content and uncertainty aspects of risk characterization are as follows:

1. The risk characterization integrates the information from the exposure and dose-response assessments, using a combination of qualitative information, quantitative information, and information regarding uncertainties. A good characterization should include different kinds of information from all portions of the foregoing assessment, carefully selected for reliability and relevance.

2. The risk characterization includes a discussion of uncertainty and variability. The risk assessor must distinguish between variability (arising from true heterogeneity) and uncertainty (resulting from a lack of knowledge).

3. Well-balanced risk characterizations present risk conclusions and information regarding the strengths and limitations of the assessment for other risk assessors, EPA decision-makers, and the public. "Truth in advertising" is an integral part of the characterization, discussing all noteworthy limitations while taking care not to become mired in analyzing factors that are not significant.

Risk assessments are intended to address or provide descriptions of risk to: (1) individuals exposed at average levels and those in the high-end portions of the risk distribution; (2) the exposed population as a whole; and (3) important subgroups of the population such as highly susceptible demographic groups or life stages, if known. Because cancer and noncancer dose-response assessment methods are currently quite different, risk characterizations also differ and are discussed separately.

Given the goals of the initial national-scale assessment, the risk characterization's purposes are best served by an analysis of broad geographic scale, recognizing several significant limitations. First, the resolution is coarse. Quantitative estimates of cancer risk and non-cancer hazard are calculated at the census-tract level, but presented only using statistics which summarize their distributions at the county level or higher. Second, the risk characterization limits itself to potential human exposures and health effects, including limited information on variations in exposure and risk among specific subpopulations. Third, the risk characterization includes only inhalation exposure, and excludes estimates of air toxics uptake by ingestion and dermal contact. Fourth, the risk characterization includes only inventoried major, area, and mobile sources of the study pollutants. For these reasons, the results of this assessment represent only a portion of the true risks associated with these air toxics. Interpretation and use of these risk results should thus bear in mind these limitations and focus on their relative aspects rather than their absolute magnitudes.

3.4.2 Cancer

In this assessment, cancer risk is defined as the probability of contracting cancer following exposure to a pollutant over a 70-year period (assumed human lifespan) at the estimated exposure concentration. This estimate of risk focuses on the additional lifetime risk of cancer predicted from the exposure being analyzed, beyond that due to any other factors, and utilizes cancer potency factors which the Agency considers to be plausible upper-bounds (i.e., the true risk potencies are likely to be lower, but may be greater). It is noted that in this assessment, the estimated exposure concentrations are not considered to be upper-bound. Rather, they represent central tendency estimates of exposure concentrations for each demographic group at the geographic unit of analysis (e.g., census tract, county, etc.). Estimates of risk are expressed as a probability, usually represented in scientific notation as a negative exponent of 10. For example, an additional lifetime risk of contracting cancer of 1 chance in 10,000 (or one additional person in 10,000) is written as $1x10^{-4}$ or 1e-4.

The distribution of individual exposures and risks within a given population can also be presented, providing an estimate of the number of people exposed to various predicted levels of risk. The Agency's risk characterization guidelines recommend that risk assessments describe individual risk, population risk, and risk to important subgroups of the population such as highly exposed or highly susceptible groups [*28*]. Quantitative individual cancer risks are calculated by multiplying the corresponding exposure estimate by the URE.

People are typically exposed to multiple chemicals rather than a single chemical. In rare cases where WOE classifications and UREs are available for the chemical mixture of concern or for a similar mixture, risk characterization can be conducted on the mixture using the same procedures used for a single compound. However, cancer dose-response assessments and UREs are usually available only for individual compounds within a mixture. Consequently, in assessments of carcinogens for which there is an assumption of a linear dose-response, the cancer risks predicted for individual chemicals are typically added to estimate cumulative risk associated with groups of chemicals, as recommended by EPA's guidelines for assessment of mixtures [*29*].

For the NATA national-scale assessment the risk estimates for cancer have been expressed in terms of the probability of contracting cancer from a lifetime of exposure. For substances for which UREs have been developed by linear extrapolation to low doses (including all the carcinogenic air toxics in this assessment), probabilities were calculated by multiplying the URE by the estimated lifetime average daily exposure.

Lifetime cancer risks were calculated and aggregated as follows, in order to focus the assessment on those air toxics that drive the assessment:

1. Air toxics-specific cancer risks for each substance having a URE in Table 3-5 were calculated for the median exposure estimates within each census tract. Plots were prepared showing the frequency distribution of risk for each air toxic across all census tracts, and population sizes living in tracts where the median cancer risk estimates exceeded fixed levels.

2. Air toxics for which estimated cancer risk exceeded 1e-6 (1 in 1 million) in the 99th percentile census tract were grouped by WOE (as per the 1986 cancer guidelines), using information shown in Table 3-5. The development of the UREs shown in Table 3-5 for total particulate organic matter (POM) and 7-PAH (polycyclic aromatic hydrocarbon (PAH)) is described in Appendix H. In the spirit of the 1996 proposed classification of "known" and "likely" carcinogens, risks of different air toxics were combined only within two groups: the category A pollutants, and across all category B and C pollutants.

3. Air toxics for which estimated cancer risk did not exceed 1e-6 (1 in 1 million) in the 99th percentile census tract were not included in aggregate risk estimates of multiple air toxics because their relatively small contribution to the risk sum would be within rounding error.

4. In combining risks across multiple carcinogens, this assessment did not consider information supporting non-additive aggregation (as recommended in EPA's draft mixtures assessment guidelines), because such information was not available. Accordingly, the assessment used the mixtures guidelines default assumption of additivity of risks, and combined risks in the manner described above by summing them, using the independence formula in the mixtures guidelines.

3.4.3 Effects Other Than Cancer

Unlike linear dose-response assessments for cancer, in most cases, noncancer risks generally are not expressed as a probability of an individual suffering an adverse effect. Instead, "risk" for noncancer effects typically is quantified by comparing the exposure to the reference level as a ratio. The "hazard quotient" (HQ) is the exposure divided by the reference level (e.g., the RfC or other similar value). For a given air toxic, exposures below the reference level (HQ<1) are not likely to be associated with adverse health effects. With exposures increasingly greater than the reference level (i.e., HQs increasingly greater than 1), the potential for adverse effects increases. The HQ, however, should not be interpreted as a probability of adverse effects.

While some risk assessments may involve significant exposure to only a single compound, exposure to a mixture of compounds that may produce similar or dissimilar health effects more accurately reflects "real" conditions. In a few cases, reference levels may be available for a chemical mixture of concern or for a similar mixture. In such cases, risk characterization can be conducted on the mixture using the same procedures used for a single compound. However, noncancer health effects data are usually available only for individual compounds within a mixture. In screening assessments for such cases, a conservative "hazard index" (HI) approach, in which all the HQs for individual contaminants are summed, is sometimes used. This approach assumes that even when individual pollutant levels are lower than the corresponding reference levels,

Table 3-5. Hazard identification and dose-response information for carcinogenic effects.

Air Toxics	Weight of Evidence		Unit Risk Estimate (per ug/m^3)	Source
	EPA	IARC		
Acetaldehyde	B2	2B	2.2E-06	IRIS[3]
Acrylonitrile	B1	2A	6.8E-05	IRIS[3]
Arsenic compounds	A	1	4.3E-03	IRIS[4]
Benzene	A	1	7.8E-06	IRIS[4,5,6]
Beryllium compounds	B1	1	2.4E-03	IRIS[3,6]
1,3-Butadiene	B2	2A	1E-05	EPA NCEA[4,6,7]
Cadmium compounds	B1	1	1.8E-03	IRIS[3]
Carbon tetrachloride	B2	2B	1.5E-05	IRIS[3]
Chloroform	B2	2B	2.3E-05	IRIS[3]
Chromium compounds	A	1	4.1E-03	IRIS[4,6,8]
Coke Oven Emissions	A	-	6.2E-04	IRIS[3]
1,3-Dichloropropene	B2	2B	4.0E-06	IRIS[3,6]
Ethylene dibromide (1,2-dibromoethane)	B2	2A	2.2E-04	IRIS[3]
Ethylene dichloride (1,2-dichloroethane)	B2	2B	2.6E-05	IRIS[3]
Ethylene oxide	B1	1	8.8E-05	CAL EPA
Formaldehyde	B1	2A	1.3E-05	IRIS[3]
Hexachlorobenzene	B2	2B	4.6E-04	IRIS[3]
Hydrazine, hydrazine sulfate	B2	2B	4.9E-03	IRIS[3]
Lead compounds	B2	2B	1.2E-05	CAL EPA
Methylene chloride	B2	2B	4.7E-07	IRIS[3]
Nickel compounds	A	2B	1.2E-04	[3,8]
Polychlorinated biphenyls (PCBs)	B2	2A	1.1E-04	IRIS[3]
Polycyclic Organic Matter (total)	[9]	[9]	5.5E-05	[10]
Polycyclic Organic Matter (7-PAH)	B2	[9]	2.0E-04	[10]
Propylene dichloride (1,2-dichloropropane)	B2	-	1.9E-05	CONV ORAL[3]
Quinoline	C	-	3.4E-03	CONV ORAL[3]
1,1,2,2-Tetrachloroethane	C	3	5.8E-05	IRIS[3]
Tetrachloroethylene (perchloroethylene)	B2-C	2A	5.9E-06	CAL EPA
Trichloroethylene (TCE)	B2-C	2A	2.0E-06	CAL EPA
Vinyl chloride	A	1	8.8E-06	EPA NCEA[3,6,7,11]

[3] Upper confidence limit URE; (assessments that did not specify method were assumed to use the UCL).
[4] Maximum likelihood URE.
[5] Higher of 2 recommended UREs was selected.
[6] Assessment consistent with 1996 proposed cancer guidelines.
[7] Advanced draft of IRIS assessment, expected to be finalized shortly.
[8] Value includes assumptions on speciation of emissions; details will be provided in report text.
[9] WOE varies among individual compounds.
[10] Note that the California EPA estimates for various polycyclic organic compounds are based on a toxic equivalency approach, where the potency of individual compounds is estimated based on relative activity rather than individual assessments of bioassay data.

some pollutants may work together such that their potential for harm is additive and the combined exposure to the group of chemicals poses greater likelihood of harm. This assumption of dose additivity is most appropriate to compounds that induce the same effect by similar modes of action [*29*]. As with the HQ, the HI should not be interpreted as a probability of adverse effects, or as strict delineation of "safe" and "unsafe" levels [29]. Rather, the HI is a rough measure of potential for risk and needs to be interpreted carefully.

Although the HI approach encompassing all chemicals in a mixture may be appropriate for a screening-level study, it is important to note that application of the HI equation to compounds that may produce different effects, or that act by different mechanisms, could overestimate the potential for effects. Consequently, EPA generally prefers a more refined approach of calculating a separate HI for each noncancer endpoint of concern for which modes of action are known to be similar [29].

For the NATA national-scale assessment, the risk characterization for effects other than cancer has been expressed in terms of the hazard quotient (HQ) for inhalation. As discussed in section 3.3.3.2 above, many RfCs incorporate protective assumptions in the face of uncertain data, so that an HQ greater than one does not necessarily indicate a likelihood of adverse effects. The HQ cannot be translated to a probability that adverse effects will occur, and it is unlikely to be proportional to risk.

Different pollutants may cause completely different adverse health effects or act via completely different modes of action, so it is often inappropriate to aggregate HQs associated with different substances. EPA has drafted revisions to Agency guidelines on assessing the impact of mixtures [*30*], which recognize combining effects of different substances in specific and limited ways. The national-scale assessment has aggregated non-cancer HQs of air toxics that act by similar toxic modes of action, or (where this information is not incorporated in the dose-response assessment) that affect the same target organ. Aggregation in this way produced a "target-organ-specific hazard index" (TOSHI), defined as the sum of hazard quotients for individual air toxics that affect the same organ or organ system.

Non-cancer HQs were calculated and aggregated as follows, in order to focus the assessment on those non-carcinogenic air toxics that drive the assessment:

1. The HQ for each air toxic having an RfC or similar value in Table 3-6 was calculated for the median exposure within each census tract. Plots were prepared showing the frequency distribution of HQ for each air toxic across all census tracts, and population sizes living in tracts where the median HQ exceeded fixed levels.

2. Air toxics for which estimated HQ exceeded 0.01 in the 99th percentile census tract were grouped by target organ, as shown in Table 3-7. Information on target

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The development of UREs for total particulate organic matter (POM) and 7-PAH (polycyclic aromatic hydrocarbon (PAH)) are described in Appendix H.

[11] URE based on whole life exposure was selected over a URE based on adult exposure only.

organs for each pollutant was obtained from dose-response assessments and from the scientific literature. To avoid aggregating HQs with widely divergent levels of uncertainty, HQs for different air toxics were combined only within two groups: those with "high certainty" RfCs (i.e., whose total uncertainty factor was 100 or less) and those with "low certainty" RfCs (whose total uncertainty factor was greater than 100).

3. Air toxics for which HQ did not exceed 0.01 in the 99th percentile census tract were not included in aggregate TOSHIs for multiple air toxics because their relatively small contribution to the TOSHI sum would be within rounding error.

4. For each of the target organs shown in Table 3-7, the HQ for each air toxic was summed to create the TOSHI (data permitting).

Table 3-6. Non-Cancer Dose-Response Information.

Urban Air Toxics	CAS #	RfC (or Equivalent)[12] (mg/m3)	UFxMF[13]	Target Organ for Chronic Critical Effect[14]	Target Organs for Other Chronic Effects	Source
Acetaldehyde	75070	9.0E-03	1000	Nasal epithelium	Growth rate, blood, and kidney	IRIS
Acrolein	107028	2.0E-05	1000	Nasal epithelium	Mucous membranes (irritation)	IRIS
Acrylonitrile	107131	2.0E-03	100/10	Nasal epithelium, brain	Central nervous system (depression)	IRIS
Arsenic compounds		3.0E-05	1000	Skeleton (fetal malformation)	Skin and mucous membranes (irritation)	CAL EPA
Benzene	71432	6.0E-02	10	Blood, bone marrow	Central nervous system (depression)	CAL EPA
Beryllium compounds		2.0E-05	10	Lung	Immune system	IRIS
1,3-Butadiene	106990	8.0E-03	300	Reproductive system	Cardiovascular system, blood	CAL EPA
Cadmium compounds		2.0E-05	30	Kidney	Lung	CAL EPA
Carbon tetrachloride	56235	4.0E-02	300	Liver	Kidney	CAL EPA
Chloroform	67663	9.8E-02	100	Liver, kidney	Central nervous system (depression)	ATSDR
Chromium compounds		1.0E-04	90	Respiratory tract (necrosis)	Liver, kidney, GI tract, immune system	IRIS
1,3-Dichloropropene	542756	2.0E-02	30	Nasal epithelium	Urinary bladder	IRIS

[12] Includes EPA reference concentrations (RfCs), California EPA reference exposure levels (RELs), ATSDR minimum risk levels (MRLs), and HEAST inhalation reference doses (RfDs) converted to concentrations in air.
[13] Modifying factors of 1 are not shown.
[14] Critical effect listed is the adverse effect upon which the RfC or equivalent health-based value is based.

Urban Air Toxics	CAS #	RfC (or Equivalent)[12] (mg/m3)	UFxMF[13]	Target Organ for Chronic Critical Effect[14]	Target Organs for Other Chronic Effects	Source
Ethylene dibromide (1,2-dibromoethane)	106934	8.0E-04	100	Reproductive system	Liver, kidney, testes	CAL EPA
Ethylene dichloride (1,2-dichloroethane)	107062	2.4E+00	90	Kidney	Liver	ATSDR
Ethylene oxide	75218	3.0E-02	100	Blood	Eyes, mucous membranes, central nervous system	CAL EPA
Formaldehyde	50000	9.8E-03	30	Respiratory epithelium	Immune system (sensitization)	ATSDR
Hexachlorobenzene	118741	3.0E-03	100	Liver (developmental)	Immune system, kidney, blood	CAL EPA
Hydrazine, hydrazine sulfate	302012	2.0E-04	300	Liver, thyroid	Respiratory system, spleen	CAL EPA
Lead compounds[15]	PB_CMPDS	1.5E-03	1	Central nervous system (neurobehavioral effects)	Blood, cardiovascular system, kidney	NAAQS
Manganese compounds	MN_CMPDS	5.0E-05	1000	Central nervous system (neurobehavioral effects)	Respiratory system	IRIS
Mercury compounds[16]	HG_CMPDS	3.0E-04	30	Central nervous system	-	IRIS
Methylene chloride	75092	1.0E+00	30	Liver	Kidney, cardiovascular system	ATSDR
Nickel compounds	NI_CMPDS	2.0E-04	30	Respiratory system, immune system	-	ATSDR
Propylene dichloride (1,2-dichloropropane)	78875	4.0E-03	300	Nasal epithelium	Blood	IRIS
Tetrachloroethylene (perchloroethylene)	127184	2.7E-01	100	Central nervous system (depression)	Heart, liver, kidney	ATSDR
Trichloroethylene (TCE)	79016	6.0E-01	100	Central nervous system (depression)	Liver, kidney	IRIS
Vinyl chloride	75014	1.0E-01	300	Liver	Kidney, central nervous system (depression)	CAL EPA

[15] EPA has not developed an RfC for lead. The National-Scale Assessment uses the National Ambient Air Quality Standard for lead, which was developed using the EPA Integrated Exposure, Uptake, Biokinetic Model, and did not use the UF/MF method. Because sensitive human subpopulations were modeled, the effective UF is 1.

[16] Hazard calculations for mercury compounds were based on the RfC for elemental mercury.

Table 3-7. Grouping of compounds by target organ and uncertainty factor for aggregation of effects other than cancer.

Target Organs/Systems for Chronic Effects	UF x MF	Contaminant Groupings
Respiratory system (including nasal epithelium, mucous membranes, and lung)	1-100	Beryllium, cadmium, chromium VI, 1,3-dichloropropene, formaldehyde, nickel
	101 – 1000	Acetaldehyde, acrolein, acrylonitrile, arsenic, propylene dichloride, ethylene oxide, hydrazine, manganese
Blood (including bone marrow and spleen)	1-100	Benzene, ethylene oxide, hexachlorobenzene, lead
	101 – 1000	Arsine, 1,3-butadiene, propylene dichloride, hydrazine
Central nervous system (including neurobehavioral effects and CNS depression)	1-100	Benzene, chloroform, ethylene oxide, lead, manganese, mercury, tetrachloroethylene, trichloroethylene
	101 – 1000	Acrylonitrile, vinyl chloride
Liver and kidney[17]	1-100	Cadmium, chloroform, chromium VI, ethylene dibromide, ethylene dichloride, hexachlorobenzene, lead, methylene chloride, tetrachloroethylene, trichloroethylene,
	101 – 1000	Acetaldehyde, carbon tetrachloride, hydrazine, vinyl chloride
Cardiovascular system	1-100	Lead, methylene chloride, tetrachloroethylene
	101 – 1000	1,3-Butadiene
Immune system (including sensitization)	1-100	Beryllium, chromium VI, formaldehyde, hexachlorobenzene, nickel
	101 – 1000	--

3.4.4 Discussion of Uncertainties in the Dose-Response Assessment

3.4.4.1 Uncertainties in the Unit Risk Estimate

The process of Unit Risk Estimate (URE) development includes the following important sources of uncertainty:

1. Many of the air toxics included in this assessment were classified as probable carcinogens, which means that data were not sufficient to prove these substances definitely cause cancer in humans. It is possible that some are not human carcinogens at environmentally relevant doses, and that true risk associated with these air toxics is zero.

2. All UREs used in this assessment were based on linear extrapolation from high to low doses. To the extent that true dose-response relationships for some air toxics are less than linear, this assumption may result in significant overestimates of risk.

3. UREs for most of these substances were developed from animal data using conservative methods to extrapolate between species. Actual human responses may differ from the predicted ones.

[17] Liver and kidney effects were combined because most HAPs that affect either, affect both.

4. Most UREs used in this assessment (typically those from assessments based on animal data) were based on the statistical upper confidence limit (UCL) of the fitted dose-response curve, but a few (typically those from assessments based on human data) were based on the statistical best fit ("maximum likelihood estimate," or MLE). The reader should be aware that MLE estimates for some known carcinogens are somewhat less conservative than UCL estimates.

Nevertheless, because of the combination of assumptions used in the face of all four sources of uncertainty described above, EPA considers all its UREs to be upper-bound estimates. True risk would probably be less, but could be greater.

For the NATA national-scale assessment, hazard identification and dose-response assessments for carcinogenic effects (Table 3-5) were obtained from peer-reviewed sources and prioritized according to (1) applicability, (2) conceptual consistency with EPA risk assessment guidance, and (3) level of review received. A discussion of sources and details of the prioritization process are presented in Appendix G.

3.4.4.2 Uncertainties in Reference Concentrations

In the development of reference concentrations (RfCs), uncertainty factors (UFs) are applied as appropriate for the following extrapolations or areas of uncertainty:

- Laboratory animal data to humans;
- Average healthy humans to sensitive humans;
- Subchronic to chronic exposure duration;
- LOAEL to NOAEL; and
- Incomplete database.

In addition to UFs (which may be 10, 3 or 1), modifying factors (MFs) may also be applied. A modifying factor is a factor used in the derivation of a reference dose or reference concentration. The magnitude of the MF reflects the scientific uncertainties of the study and database not explicitly treated with standard uncertainty factors (e.g., the completeness of the overall database). A MF is greater than zero and less than or equal to 10, and the default value for the MF is 1.

The composite UF depends on the number of extrapolations required. RfCs have been derived for various substances using composite UFs that range from 10 to 3,000, with most RfCs using factors of 100 to 1,000. The use of order-of-magnitude uncertainty factors for RfCs and the definition of the RfC as having "uncertainty spanning perhaps an order of magnitude" are indications of the general lack of precision in the estimates.

It is important to note that the composite UF expresses only our relative certainty about the dose at which these agents may not cause adverse non-cancer effects in humans and not the absolute hazard when the RfC is exceeded. This is because the magnitude of the UF provides no information on the relative level of hazard or degree of conservatism applied in developing a dose-response assessment. For example, one substance's RfC that is based on a composite UF of 3000 may be no further beneath the true threshold for effects than the RfC for a different substance that is based on a UF of 10. The higher UF

was assigned to the first substance only because the amount and quality of evidence was insufficient to support a lower UF. Thus, RfCs developed using high UFs should not be assumed to be more conservative than those using low UFs.

It should also be noted that exposures above an RfC do not necessarily imply unacceptable risk or that adverse health effects are expected. Because of the inherent conservatism of the RfC methodology, the significance of exceedances must be evaluated on a case-by-case basis, considering such factors as the confidence level of the assessment, the size of UFs used, the slope of the dose-response curve, the magnitude of the exceedance, and the number or types of people exposed at various levels above the RfC.

For the NATA national-scale assessment, hazard identification and dose-response information for non-cancer health effects (3- 5) were obtained from peer-reviewed sources and prioritized according to (1) applicability, (2) conceptual consistency with EPA risk assessment guidance, and (3) level of review received. A discussion of sources and details of the prioritization process are presented in Appendix G.

4 Results and Discussion of Exposure Assessment

4.1 Introduction

It is important to note that the ambient air quality predictions, and subsequent exposure and risk assessment results for this initial national scale assessment are being derived at a broad geographical scale. Because of limitations associated with the emission inventory, dispersion modeling and exposure modeling approaches, local scale "hot spots" (i.e., fence line impacts, complex terrain issues, persistent flow situations, fumigation events, etc.) are not predicted with this assessment. Further, the assessment includes only a single exposure media (i.e., air inhalation). Thus, extreme care should be taken in interpreting the results of this assessment. The results are best used in combination with other media assessments, as well as local scale assessments, to better evaluate the full potential health implications of a particular pollutant. Finally, it is equally important to note that this assessment is still undergoing scientific peer review and the results should be considered draft until that process is complete.

The discussion of results follows the sequence of the four major components of the assessment: emission inventory development, fate and transport characterization, population exposure estimation, and risk characterization. The first three components, the exposure assessment components, are discussed in this section, and the last component, risk characterization, is discussed in section 5. The discussion of each component includes a presentation of important results and a discussion of the major sources of uncertainty associated with that component. The fate and transport section includes a comparison of model performance which utilizes a comparison between modeled and monitored ambient concentrations. In each section, the results presented and discussed represent only a portion of the full results for that component; remaining results for emission inventory and dispersion modeling portions of the assessment are available on the NATA website (http://www.epa.gov/ttn/uatw/nata), and results for the exposure modeling portion of the assessment are provided in Appendix K.

This assessment is the first national-scale assessment, and the results should be considered to be preliminary. The national-scale assessments may be repeated every 3 years, and these future national-scale assessments will expand in scope and will make use of improved data and assessment tools.

4.2 Source Characterization: Emission Inventories

This section provides an overview of the emission estimates in the NTI, which provide the majority of the emissions data inputs to the national-scale assessment. The NATA website contains a complete set of tables, maps and graphics that portray the emission inputs to the national-scale assessment (http://www.epa.gov/ttn/uatw/nata/natsa1.html).

EPA prepared emission inventory ASPEN-input files for 1) direct emissions of air toxics, 2) direct emissions of diesel particulate matter (PM), and 3) pollutants that can transform in the atmosphere to produce air toxics. These inventories are described in section 3.2.1.2. The majority of the emissions data used in the national-scale assessment were

from EPA's 1996 NTI. In addition to the NTI, emissions were used from a diesel PM inventory from mobile sources, and VOC air toxics precursor emissions were derived from the NET criteria database. Table 4-1 summarizes the inventories used as input to EMS-HAP to prepare ASPEN inputs.

Table 4-1. Summary of Inventories Used as Input to EMS-HAP

	Stationary Source Inventory	**Mobile Source Inventory**
Directly emitted air toxics from the 1996 NTI	X	X
Air toxics precursors from the 1996 NTI and the 1996 NET speciated for particular VOC's	X	X
Diesel PM from a mobile source diesel PM inventory (48 states) as discussed in 3.2.1.2 and the 1996 NET (Puerto Rico and the Virgin Islands) for PM-10		X

The NET, diesel PM, and early versions of the 1996 NTI inventories all contained emissions estimates for the 50 states. The geographic domain of the national-scale assessment included the 48 contiguous states, District of Columbia, Puerto Rico, and Virgin Islands. In all of the inventories, emissions for the territories were derived in part or in total via extrapolation of emissions estimates from surrogate U.S. locations. More information regarding how emissions from all inventories were prepared for ASPEN dispersion model input can be found in Appendix C.

4.2.1 Summary and Discussion of 1996 Emission Inventory Results

There are a number of ways that the 1996 NTI data can be summarized. The NATA website (http://www.epa.gov/ttn/uatw/nata/natsa1.html) provides various summaries of the emissions data used in the national scale assessment. Available on the site are county emissions totals by source sector and pollutant, emissions density maps, and pie and bar charts. Figure 4-1 shows that the national total of the 33 urban air toxics (this includes dioxins and excludes diesel PM) emitted from the four source sectors is 1.1 million tons (for 1996). This pie chart illustrates that approximately half of the air toxics in this assessment come from mobile sources and the other half from stationary sources. The majority of these stationary source emissions are from the area and other source sector. This would be expected since these pollutants were selected, in part, based on their presence in urban areas and from area sources.

Another way the data can be examined is by their distribution between urban and rural counties. Counties are defined as urban or rural based on population data provided by the Bureau of the Census. For purposes of developing EPA's Integrated Urban Air Toxics Strategy, a county is considered "urban" if either: 1) it includes a metropolitan statistical area with a population greater than 250,000; or, 2) the U.S. Census Bureau designates more than fifty percent of the population as "urban." The Integrated Urban Air Toxics Strategy (online at www.epa.gov/ttn/uatw/urban/urbanpg.html) is an important part of EPA's national air toxics program. Please note that the definition of "urban" does not necessarily apply for regulatory or implementation purposes.

Figure 4-2 shows a summary of NTI emissions of 33 urban air toxics by urban and rural

designations. As in the previous pie chart (Figure 4-1), the "area and other" category dominates the total emissions, as would be expected for this pollutant set.

Figures 4-3 to 4-6 are maps that show emissions densities by groups of air toxics (metals, semi-volatile organic compounds (SVOCs), etc.) across the U.S., based on EMS-HAP processed emissions. In some cases, sharp contrasts are obvious along state or county lines. This is likely primarily an artifact of variations in state and local agency-reported data. The maps illustrate that the greatest emission densities tend to be in metropolitan areas and in the eastern U.S. This is to be expected since these pollutants were identified based on their presence in urbanized locations. This result is similar across the pollutant groups shown. A complete set of emission density maps for the all the pollutants is available at http://www.epa.gov/ttn/uatw/nata/natsa1.html#emission.

For the purposes of the national-scale assessment, several assumptions were made to accommodate using the NTI data in computer models. For this reason, the emissions input to the ASPEN model, which represents the emissions output from EMS-HAP, is not identical to the NTI data from which it originated. The county-level emission summaries on the NATA website reflect the EMS-HAP processed inventory and therefore, will not match exactly to similar county-level summaries directly from the NTI. National level summaries of the EMS-HAP emissions and NTI emissions are shown in Table 4-2, and the principle ways in which the two differ are described below:

Differences due to pollutant groupings. Proper grouping is essential for assuring that the most accurate deposition and decay characteristics are assigned to air toxics provided in the emission inventory. The grouping decisions made for the national-scale assessment reflect "downstream" data needs, such as making the resultant concentration estimates reflect compounds for which health benchmark information exists. These decisions made for dispersion and exposure modeling are not always the same as those used to summarize pollutant groupings in the NTI.

For the NTI emission summaries, emissions of particular metal compound species (e.g. lead oxide) belonging to groups were summed with no adjustment. For ASPEN modeling, particular metal compound species were adjusted to account only for the moles of the metal in the compound (e.g., emissions of the oxide fraction of lead oxide were subtracted from the emissions of lead oxide). This resulted in apparently lower metal compound emissions in the EMS-HAP summaries than the NTI summaries, but actually this effect is merely the result of two different definitions of "metal compounds."

Differences due to different processing of emissions records that were reported in pounds per hour. For the NTI emission summaries, these records were not included in the summary because the operating schedule was unknown. In EMS-HAP, these records were converted to tons/year assuming a maximum operating schedule (8760 hours per year). This resulted in higher emissions in the EMS-HAP summaries than the NTI summaries.

Differences due to geographic domain. The NTI national summaries include Alaska and Hawaii. EMS-HAP national summaries do not.

Table 4-2. Comparison of EMS-HAP and NTI emission totals.

Pollutant	Major			Area and Other			Onroad Mobile			Nonroad Mobile			Total		
	EMS-HAP Emiss. (tons/yr)	NTI Emiss. (tons/yr)	Diff.	EMS-HAP Emiss. (tons/yr)	NTI Emiss. (tons/yr)	Diff.	EMS-HAP Emiss. (tons/yr)	NTI Emiss. (tons/yr)	Diff.	EMS-HAP Emiss. (tons/yr)	NTI Emiss. (tons/yr)	Diff.	EMS-HAP Emiss. (tons/yr)	NTI Emiss. (tons/yr)	Diff.
1,1,2,2-Tetra-chloroethane	9	9	0%	116	116	0%	0	0	0%	0	0	0%	124	125	0%
1,3-Butadiene	2748	2743	0%	16304	19148	-17%	23596	23488	0%	9460	9864	-4%	52108	55243	-6%
1,3-Dichloro-propene	4	4	0%	21350	21178	1%	0	0	0%	0	0	0%	21354	21181	1%
7-PAH	105	105	0%	707	828	-17%	42	42	1%	17	19	-8%	872	994	-14%
Acetaldehyde - Primary	8815	8799	0%	18826	21691	-15%	28790	28698	0%	40764	40828	0%	97195	100016	-3%
Acrolein - Primary	233	234	0%	16457	19447	-18%	4996	4960	1%	7342	7376	0%	29029	32017	-10%
Acrylonitrile	1001	1001	0%	258	258	0%	0	0	0%	0	0	0%	1259	1259	0%
Arsenic Compounds	316	317	0%	76	77	-2%	0	0	1%	2	2	-5%	395	396	0%
Benzene	13388	13487	-1%	60905	68940	-13%	169060	168212	1%	93623	98703	-5%	336976	349342	-4%
Beryllium Compounds	33	33	0%	7	7	0%	0	0	0%	0	0	-6%	40	40	0%
Cadmium Compounds	68	68	0%	86	84	2%	0	0	0%	0	0	-6%	154	153	1%
Carbon Tetrachloride	380	372	2%	105	104	1%	0	0	0%	0	0	0%	485	476	2%
Chloroform	2752	2693	2%	626	615	2%	0	0	0%	0	0	0%	3378	3308	2%
Chromium Compounds	676	680	-1%	423	429	-2%	14	13	1%	34	35	-1%	1147	1157	-1%
Coke Oven Emissions	1433	1433	0%	4	4	0%	0	0	0%	0	0	0%	1437	1437	0%
Diesel Particulate Matter	N/A	N/A		N/A	N/A		133556	N/A		341241	N/A		474797	N/A	
Ethylene Dibromide	8	8	0%	4	4	-4%	0	0	0%	0	0	0%	13	13	-1%
Ethylene Dichloride	683	684	0%	94	87	8%	0	0	0%	0	0	0%	777	771	1%
Ethylene Oxide	307	279	9%	1114	1112	0%	0	0	0%	0	0	0%	1421	1391	2%
Formaldehyde - Primary	15959	15961	0%	142589	160622	-13%	83354	83006	0%	86374	86440	0%	328276	346028	-5%
Hexachloro-A26benzene	1	1	0%	0	0	-1%	0	0	0%	0	0	0%	1	1	0%
Hydrazine	25	25	0%	2	2	0%	0	0	0%	0	0	0%	28	28	0%
Manganese Compounds	1567	1567	0%	1116	1123	-1%	6	6	1%	35	36	0%	2724	2732	0%
Mercury Compounds	106	106	0%	51	50	1%	0	0	1%	7	7	1%	163	163	0%
Methylene Chloride	32117	30460	5%	52889	52477	1%	0	0	0%	0	0	0%	85006	82937	2%
Nickel Compounds	600	601	0%	500	517	-3%	11	11	1%	89	93	-5%	1199	1221	-2%
Perchloro-ethylene	6403	6411	0%	37510	37092	1%	0	0	0%	0	0	0%	43914	43503	1%
Polychlorinated Biphenyls	0	0	0%	0	0	0%	0	0	0%	0	0	0%	0	0	0%
Propylene Dichloride	81	81	0%	15	15	0%	0	0	0%	0	0	0%	96	96	0%
Quinoline	10	10	0%	4	4	0%	0	0	0%	0	0	0%	15	15	0%
Trichloro-ethylene	10354	10354	0%	15361	15077	2%	0	0	0%	0	0	0%	25715	25431	1%
Vinyl Chloride	851	851	0%	384	384	0%	0	0	0%	0	0	0%	1235	1235	0%

*Only HAPs for which concentration results have been posted are shown; NTI totals include Alaska and Hawaii, EMS-HAP totals do not

Differences due to facilities with missing or incorrect location data. As mentioned previously, locations for some facilities were corrected in EMS-HAP. In addition, 87 facilities in the NTI could not be associated with county coordinates. Forty-three of these sources emit the pollutants targeted in the national-scale assessment, but could not be included because of this missing location information. These differences would account for variations among county totals where location default schemes placed facilities in EMS-HAP in different counties than in the NTI, and would make emissions lower in the EMS-HAP inventory where these facilities are missing (Colorado and Idaho).

4.2.2 Discussion of Inventory Uncertainties

Because the 1996 NTI is a composite of emissions estimates generated by state and local regulatory agencies, EPA, and industry; and because emission estimation techniques vary with the agency providing data, the pollutants and the source categories, it is understandable that the uncertainties should vary widely among emissions estimates. In some cases, an estimate may be derived from few or only one emissions measurement at a similar source. The NTI estimates originated from a variety of sources and estimation methods, as well as for differing purposes, and they will in turn vary in quality, number of pollutants included, level of detail, and geographic coverage. EPA has not attempted to verify estimation methodology from the primary sources submitting data or from other EPA databases (e.g., state inventories, TRI). EPA has not undertaken a full QA/QC evaluation of the NTI because (1) most estimates submitted by state and local agencies did not include supporting documentation, (2) EPA was aware of the uneven quality and planned to view the national-scale assessment results in light of that awareness, (3) this national-scale assessment was planned as a trial effort, not a definitive result, and (4) EPA did not have sufficient time or resources available. Nevertheless, EPA recognizes that the lack of such an evaluation represents an important source of uncertainty in the assessment. Table 4-3 summarizes the origins of the stationary source emission estimates in the NTI.

Table 4-3. Summary of Data Sources to the NTI.

	Emissions of 188 Air Toxics	Percent Emissions	Number of Facilities or Categories	Percent of Facilities or Categories
All Stationary Sources	2,301,700			
All Point Sources	1,174,700			
From state or local agencies	401,300	34% of all point source emissions	55,411 facilities	90 %
From MACT	619,000	53% of all point source emissions	4,310 facilities	7 %
From TRI	153,500	13% of all point source emissions	1,847 facilities	3 %
All Non-point stationary sources	1,128,610			
EPA-generated (via emission factors/activity data)	787,100	70% of all non-point emissions	30 categories	6 %
From state or local agencies and TRI	182,900	16% of all non-point emissions	405 categories	81 %
From MACT	158,600	14% of all non-point emissions	65 categories	13 %

It should also be noted that toxic emissions data for nonroad equipment are much more limited than data for onroad mobile sources. While EPA has basic emission factors for VOC and PM for most of the nonroad categories, there is very little VOC speciation data for the given categories that would allow EPA to develop good estimates of toxic emission rates. Given the large variety of nonroad engine sizes, types and uses, as well as the likelihood that this variety will result in some differences in VOC composition, it is important that EPA obtain or develop speciated VOC data specific to each nonroad category in order to more accurately project nonroad mobile source air toxics (MSAT) inventories. In its section 202(l)(2) rulemaking for mobile source air toxics, the Office of Transportation and Air Quality outlined a strategy to obtain and evaluate these data.

Some comparisons to other inventory databases and discussion of the primary sources of uncertainty in the NTI are discussed in Section 4.2.1.2.1.

4.2.2.1 Uncertainties in Completeness of Point Source Universe

Although there are not any other national-level air toxics inventories that include point and non-point stationary and mobile sources available for comparison to the NTI, there are other emissions data sets that have been used in other dispersion modeling exercises. The only two nationwide air toxics data sets are the one used in the 1990 Cumulative Exposure Project (CEP) study and the Toxics Release Inventory. In addition to these air toxics inventories, the NET is a nationwide criteria pollutant inventory that includes emissions from all source sectors mentioned above, but with different source category definitions (e.g., major sources in the NET have 100 tons of criteria pollutant actual emissions or other limits selected by the states submitting the data).

Cumulative Exposure Project (CEP)

The 1990 CEP included 1990 TRI data, but also relied heavily on VOC and PM emission estimates from the interim 1990 NET inventory. These criteria pollutant emissions were converted to individual pollutant emissions via speciation profiles. In general, speciation profiles are industry-specific conversion factors that are used to estimate individual air toxics emission rates from criteria pollutant emissions. This method creates an air toxics emission inventory with minimal resources but produces an uncertain inventory, particularly for stationary sources where industry-specific speciation profile information is very limited. In addition much of the NET inventory is “grown” from prior years’ estimates and is thought to be less accurate than the year-specific emissions data compiled in the NTI. Therefore, the NTI would be deemed to be of superior quality to the CEP emissions estimates. A comparison of 1990 CEP inventory to 1996 NTI emission totals for selected air toxics is presented in Table 4-4.

Table 4-4. 1990 CEP and 1996 NTI National Emission Totals.

	1990 CEP	1996 NTI
POLLUTANT NAME	**Total**	**Total**
Benzene	587285	349342
Formaldehyde	412450	346028
Tetrachloroethylene	116435	43503
Methylene Chloride	115340	82937
Acetaldehyde	112639	100016
1,3-Butadiene	87600	55243
Trichloroethylene	82344	25431
POM as 7-PAH	76504	994
Acrolein	72628	32017
Coke Oven Emissions	29127	1437
1,3-Dichloropropene	20732	21181
Chloroform	17520	3308
Ethylene Dichloride	11570	771
Vinyl Chloride	6351	1235
Acrylonitrile	5110	1259
Manganese Compounds	4030	2732
Nickel Compounds	4015	1221
Carbon Tetrachloride	3978	476
Ethylene Oxide	1836	1391
Arsenic Compounds	1095	396
Chromium Compounds	996	1157
1,2-Dichloropropane	735	96.1
Cadmium Compounds	299	153
Mercury Compounds	267	163
Ethylene Dibromide	223	12.8
1,1,2,2-Tetrachloroethane	35.1	125
Beryllium Compounds	33.8	40.2
PCBs	29.2	0.21
Hydrazine	15.4	27.5
Quinoline	13.8	14.8
Hexachlorobenzene	9.96	1.00

Toxics Release Inventory (TRI)

The TRI differs from the NTI in several fundamental ways. Most significantly is the fact that the TRI universe of sources is a subset of the NTI universe, although the TRI contains many pollutants other than the 188 air toxics. The TRI contains data from only industrial sources that are large enough (in terms of chemicals usage, emissions, number of employees, etc.) to meet certain reporting requirements. In addition to these TRI sources, the NTI also contains emission estimates for smaller sources, sources not associated with industry (e.g., wildfires, consumer product usage) and mobile sources. The two inventories also differ in the level of detail. For instance, TRI sources report "stack" and "fugitive" emissions, while the NTI contains point source process parameter

details necessary for computer modeling (e.g., stack height, flow rate, temperatures, etc.) that may include many emission points (stacks) at a given facility. EPA uses information from the TRI and other EPA programs, as well as information reported voluntarily by state and local agencies, to monitor emissions and emission trends across a broad spectrum of source categories.

National Emission Trends (NET)

The NET contains criteria pollutant emissions for point, non-point stationary, and mobile sources, compiled by EPA in conjunction with state and local criteria pollutant inventory submittals. The NET and NTI inventories are designed for different purposes and contain emissions estimates for different pollutants (criteria pollutants in the NET, air toxics in the NTI). However, there may be significant overlap of included point sources, particularly among large point facilities. Therefore, the NET provides a good resource for checking the NTI's completeness and for gleaning missing location or facility details. Table 4-5 compares the number of major source NTI facilities with major source facilities in the TRI and unique facilities in the NET. Note that for states that did not provide air toxics inventories to EPA, the 1996 NET includes approximately four times the number of facilities as the NTI. This fact suggests that large point source facilities may be missing from the NTI in these states.

Table 4-5. Facility Count Summary.

State	Unique Facilities in NTI	Major Source Facilities in TRI	Unique Facilities in NET	State	Unique Facilities in NTI	Major Source Facilities in TRI	Unique Facilities in NET
AK*	16	3	28	MT*	28	10	217
AL	240	151	811	NC	2,369	230	925
AR	301	99	114	ND	56	11	65
AZ	244	24	309	NE	290	40	704
CA	7,416	176	18,870	NH	53	13	184
CO	3,424	20	4,645	NJ	148	62	864
CT	68	49	660	NM	34	6	299
DC*	1	0	14	NV*	12	4	109
DE	48	18	87	NY	3,528	103	1,584
FL	359	102	509	OH*	421	275	1,900
GA*	240	158	417	OK*	94	71	373
HI*	16	3	155	OR	187	61	395
IA*	138	101	63	PA	586	206	977
ID	56	6	21	RI	505	15	110
IL	8,813	220	9,713	SC	406	116	489
IN	1,587	287	1,321	SD	24	16	22
KS	150	75	1,963	TN	502	193	588
KY	250	98	370	TX	2,426	288	1,203
LA	301	89	664	UT	137	24	333
MA	311	55	495	VA	1,460	137	2,299
MD	730	35	439	VT	86	3	126
ME	144	20	223	WA	269	66	276
MI*	341	168	1,966	WI	744	147	1,585
MN	210	103	696	WV	172	39	229
MO	791	116	758	WY	115	8	270
MS	150	104	131	TOTAL	40,997	4,424	61,568

* States that did not provide point source data files to draft NTI

4.2.2.2 Uncertainties Due to the Dynamic Nature of Emission Inventories

Like other air pollution inventories, the NTI data set for 1996 is dynamic, and it will likely change, as resources permit, with periodic updates as new, more reliable data become available. For this reason, future assessments based on the 1996 NTI may produce somewhat different results. As the results of the ASPEN and subsequent HAPEM models are evaluated, modifications to the emission inventory input will continue to surface such as the lead emissions that are known to be missing from the Missouri lead smelter discussed in section 3.2.1.3.1. Also, it is likely that some facilities were double counted under different names, and that some plants that had closed have not been omitted from the 1996 data set.

The county-level mobile, area and other sources may be based on VMT, activity data, and/or emission factors, and these components are updated routinely. For example, the nonroad mobile source air toxics emissions are based on VOC and PM estimates from the June 2000 version of the NONROAD model. When this model run was complete, the VOC and PM nonroad estimates from individual equipment categories were consolidated into gasoline 2-stroke and 4-stroke and diesel PM source sectors and used to prepare the NTI air toxics estimates. However, subsequently a decision was made to revise inventory estimates for recreational equipment. When the revised 1996 NET was developed, these estimates were replaced with significantly lower recreational equipment estimates originally developed for the 1991 EPA Nonroad Engine and Vehicle Emission Study. The change affects both 2 and 4-stroke emissions, but it is most noticeable with 2-strokes, since that is what makes up most of the recreation equipment VOC inventory. Additional revisions to inventory estimates for recreational equipment and other equipment categories are anticipated before a final version of NONROAD is released. (More background on the draft NONROAD model, its history and recent developments are available at http://www.epa.gov/otaq/nonrdmdl.htm)

4.2.2.3 Uncertainties in Emission Locations

The location of emissions is important for all types of sources, whether expressed at a given latitude/longitude or at the county level. Many locations are unknown or incomplete, yet for dispersion modeling each facility point source must have an exact location. If the location coordinates were missing from point sources, they were placed via the default mechanisms in EMS-HAP. How well this default location reflects reality will affect the model results and the uncertainty of the overall analysis. To illustrate this point, Table 4-6 shows, by state, the percent of point sources, and the corresponding percent of emissions of lead, chromium, and cadmium emissions, that were located via default algorithms. The table shows the percentages based on sources with default locations determined by the facilities' zip codes or counties, as well as the percentage of facilities were not defaulted because their coordinates were within the correct county or within reasonable bounds outside the county. Although this comparison does not provide a quantitative assessment of uncertainty, the magnitude of sources and emissions defaulted in a given state suggests the uncertainty in the results due to potential location errors.

Table 4-6. Summary of Defaulted Sources and Emissions for Lead, Chromium, and Cadmium.

		Percent of Sources				Percent of Emissions			
State	Metal	County Defaulted	Zip Code Defaulted	Not Defaulted, Outside County	Not Defaulted, Inside County	County Defaulted	Zip Code Defaulted	Not Defaulted, Outside County	Not Defaulted, Inside County
All	All	10.7	0.6	4.4	84.4	10.5	0.2	3.7	85.6
All	Lead	10.8	0.5	4.1	84.6	12.8	0.3	3.8	83.1
All	Cadmium	10.3	0.4	5.2	84.2	25.0	0.2	6.4	68.4
All	Chromium	10.9	0.5	4.7	83.8	3.5	0.1	3.0	93.4
Alabama	All	23.3	0.0	3.8	72.9	58.6	0.0	4.1	37.3
Arizona	All	1.3	0.0	9.0	89.7	2.0	0.0	29.3	68.7
Arkansas	All	9.7	0.0	2.9	87.4	10.5	0.0	5.3	84.2
California	All	19.1	0.1	0.7	80.1	9.6	0.0	0.3	90.1
Colorado	All	6.4	0.0	0.0	93.6	1.1	0.0	0.0	98.9
Connecticut	All	37.5	0.0	4.2	58.3	26.4	0.0	0.3	73.3
Delaware	All	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100.0
District of Columbia	All	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100.0
Florida	All	11.3	0.0	4.0	84.8	6.7	0.0	0.6	92.8
Georgia	All	17.6	0.0	1.6	80.8	46.0	0.0	1.4	52.7
Idaho	All	13.0	0.0	4.3	82.6	0.9	0.0	0.0	99.1
Illinois	All	1.7	0.0	0.4	97.8	0.4	0.0	0.0	99.6
Indiana	All	14.3	0.3	40.3	45.0	21.2	0.2	32.4	46.2
Iowa	All	3.4	1.7	0.0	94.9	0.1	1.3	0.0	98.6
Kansas	All	2.0	0.0	0.0	98.0	0.0	0.0	0.0	100.0
Kentucky	All	6.6	0.0	0.0	93.4	7.0	0.0	0.0	93.0
Louisiana	All	22.5	0.0	2.2	75.3	25.8	0.0	0.1	74.0
Maine	All	5.8	1.9	5.8	86.5	0.5	0.2	0.2	99.1
Maryland	All	19.5	0.8	2.3	77.4	6.1	0.0	4.1	89.8
Massachusetts	All	3.2	0.0	0.0	96.8	0.3	0.0	0.0	99.7
Michigan	All	12.7	1.6	0.4	85.3	16.7	0.0	0.1	83.2
Minnesota	All	22.7	0.0	0.0	77.3	35.7	0.0	0.0	64.3
Mississippi	All	16.3	0.0	2.3	81.4	40.8	0.0	1.0	58.2
Missouri	All	14.2	0.0	3.1	82.7	4.5	0.0	0.4	95.1
Montana	All	13.6	0.0	0.0	86.4	8.9	0.0	0.0	91.1
Nebraska	All	34.9	0.0	0.0	65.1	3.7	0.0	0.0	96.3
Nevada	All	12.5	0.0	0.0	87.5	0.3	0.0	0.0	99.7
New Hampshire	All	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100.0
New Jersey	All	1.5	0.0	3.1	95.4	0.0	0.0	0.1	99.9

Table 4-6. Summary of Defaulted Sources and Emissions for Lead, Chromium, and Cadmium.

		Percent of Sources				Percent of Emissions			
State	Metal	County Defaulted	Zip Code Defaulted	Not Defaulted, Outside County	Not Defaulted, Inside County	County Defaulted	Zip Code Defaulted	Not Defaulted, Outside County	Not Defaulted, Inside County
New Mexico	All	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100.0
New York	All	1.2	0.1	0.7	97.9	13.7	0.0	0.0	86.3
North Carolina	All	2.6	0.1	1.9	95.3	1.5	0.0	0.1	98.4
North Dakota	All	16.7	0.0	0.0	83.3	2.0	0.0	0.0	98.0
Ohio	All	3.1	0.4	1.3	95.1	21.3	0.1	0.8	77.7
Oklahoma	All	8.3	0.0	5.6	86.1	15.1	0.0	2.9	82.0
Oregon	All	48.8	3.1	1.6	46.5	28.3	0.7	0.1	70.9
Pennsylvania	All	10.2	0.4	1.3	88.1	1.9	0.0	5.1	93.0
Puerto Rico	All	20.0	0.0	80.0	0.0	2.8	0.0	97.2	0.0
Rhode Island	All	36.4	59.1	0.0	4.5	10.7	88.0	0.0	1.3
South Carolina	All	4.8	0.0	5.7	89.6	34.8	0.0	0.1	65.1
South Dakota	All	12.5	0.0	0.0	87.5	5.5	0.0	0.0	94.5
Tennessee	All	61.1	16.6	0.6	21.7	16.4	6.2	0.0	77.4
Texas	All	10.2	0.1	1.6	88.1	24.9	0.1	0.8	74.3
Utah	All	12.5	0.0	0.0	87.5	0.8	0.0	0.0	99.2
Vermont	All	10.3	0.0	6.9	82.8	1.2	0.0	10.6	88.2
Virginia	All	17.7	0.0	1.0	81.3	40.8	0.0	0.6	58.7
Washington	All	24.1	0.0	1.7	74.1	39.2	0.0	0.5	60.3
West Virginia	All	1.9	0.0	0.0	98.1	0.8	0.0	0.0	99.2
Wisconsin	All	5.4	1.5	1.5	91.7	0.5	0.0	0.4	99.1
Wyoming	All	0.0	0.0	0.0	100.0	0.0	0.0	0.0	100.0

Nationwide, for primary emissions of pollutants that were modeled (i.e., no reactive precursors to secondary formation), the following statistics apply to default locations:

Out of the 48,657 sites (there can be multiple sites at a single facility) from the point source inventory simulated via the ASPEN model (i.e., those that contained the 33 urban air toxics and were not dropped due to lack of information on locations or zero emissions), 4212 were defaulted by county-level defaults (census tract centroids) and 460 were defaulted by zip-code (zip code centroids). There were also 43 sites that were dropped from the EMS-HAP input prior to modeling due to the fact that their locations were identified as “portable” and therefore could not be defaulted.

In addition to point sources that required exact location coordinates, all county-level emissions needed to be spatially allocated to the census-tract level prior to ASPEN modeling. Each source category’s emissions were placed based on an appropriate surrogate such as population or land use. In some cases, the initial emission estimate may have been made at the national or state level and allocated to counties by the same

surrogate. This type of spatial placement may lead to emissions being too dispersed or too concentrated at a given location. For example, landfill emissions were first placed in the appropriate counties based on information collected via MACT standard development. Subsequently, within EMS-HAP, these emissions were further allocated to census tracts based on reciprocal population density. This assumption is based on the concept that landfills are placed away from populated areas. In reality, landfills are located at specific geographic coordinates rather than spread throughout census tracts within a county. Since these exact location data were not available, the surrogate approach was used as a best approximation.

The spatial allocation of county emissions, which takes place within EMS-HAP is described above in 3.2.1.1. This necessary approximation of emission location adds uncertainty to the overall analysis although it represents the best method available. As shown in Table 3-3, some of the allocation surrogate information used in this process is very old (e.g., 1970's).

Uncertainty due to spatial allocation schemes is also particularly pronounced for mobile source categories, especially within the nonroad sector where, in order to simplify computation, the county-level emissions from almost 100 source categories (e.g., recreational marine vessels, lawn mowers, construction equipment, etc.) were consolidated into three source category groups (2-stroke gas, 4-stroke gas, and diesel engines) prior to spatial allocation.

The spatial allocation methods used in this study were revised for some source categories as ASPEN concentration results were reviewed in order to improve the overall analysis.

4.2.2.4 Uncertainty Due to Stack Parameter Defaults

A facility in the NTI can contain many emissions release points. Each release point requires several process parameters in order to characterize it sufficiently for dispersion modeling. When these parameters were missing from the original inventory data or were out of reasonable range, they were replaced with defaults either within the NTI or EMS-HAP. Table 4-7 describes the number of vertical emission release points that had defaulted process parameters. Of the 97,365 unique vertical stacks, 63,292 contained at least one defaulted stack parameter.

Table 4-7. Stack Parameter Default Statistics.

Parameter	Number of Defaults		Percent of Total Vertical Stacks
	in NTI	in EMS-HAP	
NTI Stack Height	28,550	16,048	45.8
NTI Stack Diameter	32,176	20,722	54.3
NTI Exit Velocity	40,361	20,816	62.8
NTI Exit Temperature	28,890	12,982	43.0

4.2.2.5 Variations in Reported Emissions

Since no standardized requirements for collecting or submitting air toxics inventories to EPA currently exist to make these databases consistent, and because resources among the agencies are variable, the quality, coverage and number of air toxics in these inventories vary significantly. For instance, the emissions of polychlorinated biphenyls (PCB) appear in only 121 counties in the NTI out of a national total of 3,145 counties. It is unclear whether the reported emissions are correct and additional emissions are missing from the other counties, or if PCB, a banned substance, is no longer emitted from anthropogenic sources.

4.2.2.6 Uncertainties Due to Pollutant Groupings

As discussed previously, many of the air toxics from the NTI were reported either as groups of compounds (e.g., lead compounds) or individual chemical species (e.g., lead oxide). For subsequent modeling, these pollutants had to be grouped together in order to create consistency across the model's geographic domain. This creates uncertainties because the groupings made for the national-scale assessment do not necessarily account for the difference in toxicological characteristics among individual species in the group. In addition, numerous assumptions were made to establish the pollutant groups and the resulting pollutant group characteristics (e.g., deposition).

Another related uncertainty in grouping metal species stems from the adjustments made to the mass of metal species emissions to allow for modeling only the metal portion of the compound. First there is an assumption that when emissions for a particular species are reported, they are for the entire compound and not just the metal portion. Second, for metals reported as broad compound groups or subgroups (e.g., lead & compounds, alkylated lead), no adjustments were made since the particular species was not known.

4.2.2.7 Particle Size and Reactivity Assignments

All of the emissions input for ASPEN modeling must be identified by particle size fractions (coarse versus fine) and reactivity. This tells the dispersion model how to account for deposition and subsequent chemical reactions. None of the emission inventories used in this assessment contained this information, and so it had to be assigned by EMS-HAP. Since ASPEN uses different deposition rates for fine and coarse particulates, each metal particulate must be classified by its particle size. Except for mercury, the percentage of a species assigned to coarse/fine particulate (the coarse/fine split) was estimated as the percentage of coarse/fine particulate obtained from the inventory used for the Cumulative Exposure Project. These percentages vary among the onroad, nonroad and stationary source categories, but do not vary within those broad groups. For example, the coarse/fine split for chromium compounds emitted from a chromium electroplating operation is the same as for chromium emitted from an incinerator. For mercury, it was assumed that all mercury species reported are gaseous except for mercury emissions from mobile sources and mercuric chloride. Mercuric chloride was assumed to be 100 percent fine particulate. These assignments add to the overall uncertainty.

4.3 Environmental Fate and Transport Characterization

4.3.1 Summary of ASPEN Modeling Results

The ASPEN model was used to estimate ambient concentrations at each census tract in the contiguous United states, plus Puerto Rico and the Virgin Islands. Based on these estimates, annual average modeled concentrations (in μg/m3) were calculated for each state and territory, and the statistical distributions described by the 25th, median, and 75th percentiles across all census tracts were calculated and displayed graphically for each of the 33 air toxics; national averages were similarly calculated. These "statewide estimate" charts can be viewed at www.epa.gov/ttn/uatw/nata/chartconc.html. A value for background concentration, except for diesel PM, was also displayed. In addition, the percent contribution to the statewide annual average ambient concentration estimates were calculated and displayed. These "percent contribution" charts can be viewed at www.epa.gov/ttn/uatw/nata/chartconc.html. Figures 4-7 and 4-8 provide an example of each type of display for benzene. As can be seen in Figure 4-7, benzene is fairly ubiquitous, is prevalent in all states, and as expected, the median value is generally higher in the more populated or industrial states. As can be seen in Figure 4-8, the percent contribution for benzene is higher for mobile sources (both onroad and nonroad) than stationary sources. The contribution from background is also appreciable.

When examining the source contribution plots for all of the modeled air toxics (at http://www.epa.gov/ttn/uatw/nata/chartconc.html), it can be seen that no single source sector is the main contributor to the estimated concentrations. Table 4-8 summarizes the dominant contributor for each of the modeled air toxics. The results show that, on a national level, about half of the pollutants have "area and other sources" as the dominant contributing source sector. As seen in the table, the dominant source sector may be different when the source contributions are examined at the state level. For example, except for coke oven emissions, all of the pollutants in which major is the dominant source sector at the National level show the area and other category to be dominant for a number of states. For those pollutants where background is dominant, the next highest contributing source sector is the "area and other" sector.

To further explore the geographic variability of the source sector on the ambient concentrations, we examined the dominant source sector for the counties with the 10 highest median concentrations. Table 4-9 shows the number (out of 10) of these concentrations dominated by the various source types. The asterisked pollutants in the table are those in which the dominant source type differed from the dominant types at the national level and state levels as shown in Table 4-8. Tables 4-8 and 4-9 show that most pollutants vary geographically in their dominant source sector.

Table 4-8. Pollutants grouped by the dominant source sector affecting their national average concentrations.

Area and Other	Major	Mobile (onroad and nonroad combined)	Background
Arsenic[M]	Acrylonitrile[A]	Acetaldehyde	Carbon tetrachloride[a]
Beryllium[M]	Coke oven emissions	Acrolein[A]	Chloroform[a]
Cadmium[M]	Hydrazine[A]	Benzene	Ethylene dibromide[a]
Chromium[M]	Propylene dichloride[A]	1,3-Butadiene	Ethylene dichloride[a]
1,3-dichloropropene	QuinolineA	Formaldehyde	Hexachlorobenzene[a]
Ethylene oxide[M]			Mercury[a]
Manganese[M]			Polychlorinated biphenyls (PCB) [a]
Methylene chloride			Trichloroethylene[A, a]
Nickel			
Perchloroethylene			
POM[M]			
1,1,2,2-Tetrachloroethane			
Vinyl chloride[M]			

[M] = for several states, the dominant sector is major;
A= for several states, the dominant sector is area and other
A= next highest sector after background is area and other

Table 4-9. Number of Counties of Each Dominant Source Sector for the 10 Highest County Median Concentrations.

Pollutant	Major	Area and Other	Mobile	Background
Acetaldehyde			10	
Acrolein	2	3	5	
Acrylonitrile		10		
Arsenic Compounds	3	7		
Benzene			10	
Beryllium Compounds	7	3		
1,3-Butadiene		7	3	
Cadmium Compounds	3	7		
Carbon Tetrachloride				10
Chloroform	8			2
Chromium Compounds	7	3		
Coke Oven Emissions	10			
1,3-Dichloropropene		10		
Ethylene Dibromide				10
Ethylene Dichloride				10
Ethylene Oxide		10		

Pollutant	Major	Area and Other	Mobile	Background
Formaldehyde		6	4	
Hexachlorobenzene		3		7
Hydrazine	7	3		
Manganese Compounds	7	3		
Mercury Compounds		6		4
Methylene Chloride	8	2		
Nickel Compounds	6	4		
Perchloroethylene	1	9		
Polychlorinated Biphenyls		1		9
Polycyclic Organic Matter		10		
Propylene Dichloride		10		
Quinoline	7	3		
1,1,2,2-Tetrachloroethane	1	9		
Trichloroethylene	2	8		
Vinyl Chloride	9	1		

A comparison of estimated annual average concentration distribution for 10 selected pollutants is shown in Figure 4-9. These pollutants were selected to include air toxics that are relatively inert (e.g., benzene), air toxics that are subject to chemical transformation in the atmosphere (e.g., formaldehyde), and metal particles that are subject to deposition. As can be seen in this figure, the distribution of the annual average concentrations among pollutants spans several orders of magnitude. Benzene and formaldehyde have much higher annual average concentrations than metals. One to two orders of magnitude variability exists for each air toxic.

Figure 4-10 provides a comparison of estimated annual average concentration for the same 10 selected pollutants with each census tract categorized as urban or rural. Each census tract is designated as either urban or rural as part of the dispersion modeling process, since dispersion parameters differ for these two types of locations. In general, census tracts with population density greater than 750 people/km^2 are designated as urban, while other census tracts are designated as rural [15]. This results in approximately an even split of census tracts into the urban and rural designations. As can be seen in this figure, the annual average concentrations are generally higher in the urban census tracts than rural tracts; this result is likely due both to higher emission densities for many pollutants in urban areas and to a closer proximity of many emission sources to the modeled receptors (i.e., census tract centroids).

Figure 4-11 shows, for each State, the relative contributions of pollutants, broken out into three categories: mobile source dominated pollutants, metals, and stationary source non-metals. In each bar of this figure, 49 boxes representing 48 states and the District of Columbia are displayed. Concentrations are scaled by a maximum value and sorted from minimum to maximum. Thus, each box represents the magnitude of relative average concentration for the state. As can be seen from this figure, some pollutants (e.g.,

propylene dichloride) have an impact in only one or two states while other pollutants (e.g., benzene) appear more uniformly distributed across the country.

4.3.2 Discussion of Results

From an examination of the ASPEN dispersion modeling results, the following general observations can be made:

- Concentration estimates are a complex function of a number of factors, including emissions density (number of sources in a particular area), meteorology and source characteristics, rather than just total emissions.

- Of the four main source sectors (area and other, major, onroad, nonroad), no one type is a dominant contributor to the estimated concentrations. On a national level, about half of the pollutants have "area and other sources" as the dominant contributing source sector.

- There is considerable variability among the national, state and the county levels in terms of contributions of certain pollutants by source type.

- Both emissions and estimated concentrations are generally higher in urban than in rural areas.

- Because different types of sources contribute to emissions in different areas of the country, the highest ambient average concentrations of the individual pollutants occur in different states (i.e., no one state has the highest concentrations of all the pollutants).

- Some pollutants (e.g., benzene) are more evenly distributed around the country while other pollutants (e.g., vinyl chloride) are more related to isolated areas of industrial activity.

- Most of the stationary non-metal pollutants have higher concentrations in only a few states whereas the concentrations from metals and mobile source-dominated pollutants are more evenly distributed across the country. Among the 20 modeled non-metal pollutants, twelve have impacts on one to three states, and only four are widely distributed across states. For metals, two pollutants out of eight have a major impact on one or two states, while the other six pollutants have an impact in more than ten states. Mobile source-dominated pollutants are generally more uniformly distributed across the country.

- Background concentrations are an essential part of the total air quality concentrations and include pollutant concentrations due to natural sources, sources not in the emissions inventory, and long-range transport. Based on the CEP study, non-zero estimates of background concentrations for only 13 pollutants are available. For seven of these (PCBs, ethylene dibromide, carbon

tetrachloride, hexachlorobenzene, ethylene dichloride, chloroform, and mercury), the background dominates the total estimated average concentration.

4.3.3 Comparison of ASPEN Modeling System to Monitoring Data

The ASPEN system as designed and applied in the initial national scale assessment provides broad-scale air quality impacts (census tract resolution). The assessment system is not designed to capture more localized areas of high concentration or "hot-spot" impacts. The system evaluation presented below (model-to-monitoring comparison study) was conducted to evaluate the ASPEN system's ability to predict an air quality concentration at a given point in space (i.e., an air quality monitor). No detailed evaluation of the ASPEN system's inability to predict "hot-spot" impacts was conducted. It is anticipated that, following completion of more detailed local scale assessments that are to be conducted as part of future NATA activities, such an evaluation can be made. However, EPA conducted a few crude comparisons to assess the potential for the ASPEN system to underestimate "hot spots", as predicted by local-scale modeling assessments. One such comparison, for a major source in an urban environment found local fence line impacts approximately 30 times higher then those predicted at the census tract centroid by ASPEN in the initial national scale assessment. A similar comparison in a more rural setting found maximum predicted fence line impacts greater than 2 orders of magnitude above those predicted by ASPEN at the census tract centroid. While these two comparisons are not a complete evaluation of expected differences between the ASPEN initial national scale assessment predictions and those from local scale "hot spot" assessments, they are simple examples of why other scale assessments must be considered to fully understand the air toxic issues in a particular location.

The initial National-Scale Assessment does not capture local-scale or "hot spot" impacts. Such impacts could be orders of magnitude greater than those predicted at the census tract resolution provided by the ASPEN modeling system.

The remainder of this section compares the modeled air quality estimates with currently available, but geographically limited, ambient air monitoring data. A representative subset of seven air toxics (benzene, perchloroethylene, formaldehyde, acetaldehyde, cadmium, chromium, and lead) was selected for this evaluation. These pollutants were selected because they represent impacts by different combinations of mobile, county-level and point sources; include reactive and non-reactive compounds; and include those with both primary emissions and secondary formation in the atmosphere. They also include those air toxics with the largest number of monitoring sites.

For each monitor-pollutant combination, a comparison was made between the monitored concentration and the concentration estimated by the ASPEN model at the monitor location to get point-to-point comparisons. Steps in this comparison, for each pollutant at each monitor, were as follows:

The ASPEN model was used to estimate concentrations at the exact locations of the monitors. Using the monitor latitude and longitude coordinates, the monitor location is assumed to represent a census tract with 100 m radius. Concentrations are interpolated to

this “census tract” using the log-linear interpolation scheme used in ASPEN to get the annual average estimate.

For monitored concentrations, an annual average estimate was calculated by averaging all of the measured values obtained by a monitor across the course of a year. In calculating the average monitored concentration, any individual samples where the concentration was found to be below the detection limit were assigned a value of one-half the detection limit. Further details are available in the full report (Appendix J, Section II.b.ii.)

Model-monitor ratios are calculated by dividing the modeled concentration by the monitored concentration. A ratio of 1.0 means that the modeled concentration equals the monitored concentration, while a ratio less than 1.0 means that the model underestimates the monitored concentration, and a ratio greater than 1.0 means that the model overestimates the monitored concentration. A detailed explanation of the results are presented in the full report (Appendix J), Section V.

In general, the modeled estimates for most of the pollutants examined are typically lower than the measured ambient annual average concentrations when evaluated at the exact location of the monitors. However, when the maximum modeled estimate for distances up to 10-20 km from the monitoring location are compared to the measured concentrations, the modeled estimates are closer to monitored concentrations. This result can be attributed, in part, to spatial uncertainty of the underlying emission and meteorological data, and the tendency of current air toxics monitoring networks to measure the higher, if not highest, local air pollution impact areas. It also shows that the model estimates are more uncertain at the census-tract level but are more reliable for larger geographic scales, like county or state. Nevertheless, there are many locations for several of the studied air toxics (including the aldehydes and metals) for which the model estimates are still significantly lower than the measured concentrations even at distances up to 50 km. For these instances, the difference between modeled and monitored concentrations might be attributed to underestimated or missing emissions data in the model as well as uncertainty in chemical transformation for the aldehydes. The limitations of modeled concentrations resulting from isolated point sources using a geographically sparse ASPEN receptor network in rural areas may be another contributing factor. Yet another reason for the discrepancies may be attributed to monitors being sited to find peak concentrations. Often, the ambient concentration decays quickly around the peak area. Even under the scenario of a “perfect” model and “perfect” monitors, if the monitor is sited exactly at the peak and the emissions (or meteorological inputs) inputs are even slightly inaccurate, the model will tend to underestimate results. This would be especially likely for pollutants dominated by point sources with elevated releases, because any errors in release height, exit velocity, and/or emissions location will likely cause the model to find a peak concentration different from the true peak level. A more detailed discussion of the uncertainties of ASPEN and the monitoring data can be found in Appendix J of this document. A description/basis of the methodology used for the model-to-monitor comparison is given in Appendix I, and a detailed discussion of the model to monitor comparison results is provided in Appendix J.

A summary of the results from the model-to-model comparison are presented below. Table 4-10 summarizes the comparisons on a point-to-point basis. The best agreement is observed for benzene. The results are within a factor of two for 89 percent of the cases. The median ratio of model to monitor comparisons is 0.92. The lack of agreement for the other air toxics on a point to point basis can also be seen in Table 4-8, which shows the median ratios varying between 0.65 for formaldehyde to 0.17 for lead. The number of points with agreement within a factor of 2 (or within 30 percent) are also correspondingly lower. These results can also be observed using the ratio box plot graphs, shown in Figure 4.12.

Table 4-10. Comparison of the Measurement Data to Modeled Concentration.

Pollutant	Number of Sites	Median of Ratios*	Percentage within factor of two**	Percentage within factor of 30%***	Percentage Under-estimated
Benzene	87	0.92	89	59	59
Perchloroethylene	44	0.52	55	32	86
Formaldehyde	32	0.65	53	28	88
Acetaldehyde	32	0.60	59	22	91
Lead	242	0.17	18	10	91
Cadmium	20	0.18	15	5	85
Chromium	36	0.15	28	19	83

* Ratio represents (Ambient Measurement Concentration/Modeled Concentration).
** This represents the percentage of sites for which the model estimate is somewhere between half and double the monitor average.
*** This represents the percentage of sites for which the model/monitor ratio is between 0.7 and 1.3.

While all the air toxics except benzene show relatively poor agreement on a point-to-point basis (with the modeled estimates being systematically lower than the monitor averages), they compare more favorably when the maximum model-estimated concentration is examined within 30 km of the monitoring site as shown below in Table 4-11.

This technique is referred to as “MAXTOMON” and compares the maximum model estimate within x kilometers of the monitor TO the monitor average. All model estimates are considered (both estimates at monitor sites as well as the estimates at census tract centroids) in computing the maximum values. This is an example of a point-to-range tool. This tool is used here to test whether the frequent underestimation by the model at monitoring sites was due to location uncertainties or due to systematic underestimation. The reader is referred to Appendix J for further details on this technique.

Table 4-11. Maximum Modeled Concentration Compared to Monitored Value.

		Percent Missing Low at a Radius of:			
Pollutant	Number of Sites	0 km (Exact Monitor Site)	10 km	20 km	30 km
Benzene	87	59	25	20	11
Perchloroethylene	44	86	43	23	9
Formaldehyde	32	88	56	31	31
Acetaldehyde	32	91	56	38	34
Lead	242	91	65	51	40
Cadmium	20	85	60	35	25
Chromium	36	83	39	28	25

The improved comparisons, using the MAXTOMON technique, shown in Table 4-12 can be attributed to three reasons:

- Many emission sources are not precisely located. The EMS-HAP model defaults locations when they are not provided or when total emissions exist for the county. Note, however, that this could contribute to either under or over predicting and is a “non-bias” source of uncertainty.

- Many of the monitors were likely sited to find peak concentrations. For the point source situations with elevated emission releases, the monitors frequently represent hot spot locations where the ambient concentration falls off quickly around the peak concentration area.

- Since the emissions inventory is likely missing sources, the modeling assessment is under predicting air concentrations.

In the following sections, the model-to-monitor comparisons are discussed for benzene, other gases, and for the metals.

Benzene

The relationship between model estimates and monitored values for benzene can be described by the scatter plot shown in Figure 4-12. Figure 4-12 shows the point to point comparison of modeled and monitored annual average concentrations. As also shown in Figure 4-13 and Table 4-10, most of the points in the scatter plot fall between the 2:1 and 1:2 lines, showing good agreement between model predictions and monitor measurements.

This result is not surprising given the availability of good monitoring and emissions data for this ubiquitous pollutant.

Perchloroethylene, Formaldehyde, and Acetaldehyde

The model-to-monitor relationship on a point-to-point basis is similar for the other three gases investigated (perchloroethylene, formaldehyde, and acetaldehyde). In the ratio box plots in Figure 4-13, however, one can see that the model's estimates tend to be lower than the monitor averages. The typical values, however, agree well with the median ratios all within a factor of 2. Nevertheless, a large percentage of the modeled estimates are less than the monitored concentration for these gases on a point-to-point basis (see Table 4-10 and Figure 4-13). This can be attributed, in part, to spatial uncertainty in the underlying emissions for these pollutants; missing source emissions data; and/or underestimated emissions.

To examine spatial uncertainty in the modeling system for the gases, the monitored concentration is compared to the maximum modeled estimate in its vicinity (the "MAXTOMON" technique described above). The results for these gases are presented in Table 4-11. Table 4-11 shows nearby modeled concentrations which are greater than the measured average concentration for many of the monitors.

This is especially true for perchloroethylene. In close vicinity (10 km or less) of most monitors, higher modeled concentrations are observed. This result for perchloroethylene suggests that uncertainties in the magnitude and location of the nearby area sources may at least be partly responsible for the underestimation on a point-to-point basis. In other words, the model may be systematically underestimating ambient concentrations or it may just be finding the peak concentration in the wrong place.

For the two aldehydes, many monitors also have nearby modeled concentrations which are greater than their measured values. However, a large fraction of the aldehyde monitors cannot be associated with larger modeled values, even within 50 km. This suggests systematic underestimation by the modeling system for the aldehydes, at least for some of the areas. This may be attributed, in part, to the nature and treatment of these air toxics. It may also be due to underestimated emissions. The two aldehydes are mobile-source dominated, but a large fraction of their ambient concentrations are secondarily formed. The chemical reactions resulting in their formation are simulated in ASPEN. This adds an additional source of uncertainty to the modeling system and distinguishes the aldehydes from the other air toxics in this comparison.

Metals

For the metals, the monitored concentrations are typically much higher than the modeled concentrations when compared at the same location. The difference is most dramatic for source-oriented monitors. Based on the median ratio, the source-oriented lead monitors are typically underestimated by a factor of 7.5, and the others are underestimated by a factor of 4.9. Only 17 percent of the source-oriented monitors and 18 percent of the other monitors are estimated within a factor of two at the exact location of the model estimate.

A combination of several factors may be responsible for these discrepancies:

- Missing emissions from the inventory (e.g., missing point sources, lack of treatment for possible re-entrainment effects)

- Spatial uncertainty in emission locations due to defaulted locations for point sources.

- Spatial uncertainty of nearby impacts from elevated point sources (i.e., narrow plume impact) together with a small number of receptors.

- High coarse particle deposition velocities.

The effects of missing source location data and defaulted emission locations were explored. This analysis focused on 30 of the 42 monitors which were underestimated by a factor of 10 or greater. This analysis demonstrated that, for the included monitoring locations, several nearby lead sources are missing location information (have uncertain locations). The effects for spatial uncertainty of lead emissions were also examined using the MAXTOMON technique. Because this analysis did not reveal higher predicted concentrations within a relatively large region surrounding the monitor, it also suggests that emission sources are likely missing or underestimated. However, this analysis may be less definitive for point source pollutants in rural areas. Such monitors tend to represent small (less than 0.5 km) areas. The surrounding regions also have few census tracts and the small number of ASPEN receptors limit the opportunity to find other peak concentrations. Regarding deposition velocities, it was estimated that ASPEN has a bias to under predict average lead concentrations by 20-30 percent, because of high coarse particle deposition velocities.

It appears that the current modeling system is underestimating lead for a large percentage of the monitors used in this evaluation. It should be noted that the monitors do not represent a random sampling of all census tracts. To attain better modeled results in the vicinity of isolated point sources, emissions and source locations should be more accurately characterized. In addition, a denser receptor network may be required.

The results for cadmium and chromium also show many locations with low modeled concentrations. However, the amount of disagreement between the modeled estimates and monitored concentrations appear to be dependent on the different source regions represented. This suggests possible differences in the state inventories; however, generalizations are difficult because of the limited number of monitoring locations included.

4.3.4 Discussion of ASPEN Dispersion Modeling Uncertainties

In this discussion we will summarize the studies and conclusions reached regarding uncertainties in the ASPEN modeling results. The main sources for uncertainty in the ASPEN dispersion modeling results are 1) emission characterization uncertainties (e.g., specification of source location, emission rates and release characteristics); 2)

meteorological characterization uncertainties (e.g., representativeness); 3) model formulation and methodology uncertainties (e.g., characterization of dispersion, plume rise, deposition, and uncertainties associated with using a “net” of receptors for characterizing concentration impacts at particular locations); and 4) uncertainty of background concentrations.

4.3.4.1 Emission Characterization Uncertainties

When performing comparisons between modeled and monitored concentration values, it is of key importance to properly locate sources relative to monitoring locations and properly characterize whether the emissions are low-level (i.e., those closer to the ground such as fugitive emissions) or elevated stack emissions. Inspection of the Gaussian plume formulations reveals that the simulated impact at the surface generally decreases as $1/(\text{distance})^{á}$, where á is on the order of 1 to 2. The low-level emissions affect a receptor by a factor of 5 to 8 times more than if the emissions were from an elevated stack (as the elevated emissions will be diluted during transport and dispersion to the surface) [*31*, *32*].

The National Toxics Inventory (NTI) provides the “raw” emissions data that are processed and made "model-ready" for ASPEN by the Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP). Annual emission rates are provided for point sources. Emissions not allocated to point sources are in the form of “non-point” county-level summaries. The ASPEN model requires higher resolution both temporally and spatially. Temporally, EMS-HAP allocates the annual total emissions equally into days and then allocates daily emissions into eight three-hour periods using temporal profiles based on the type of emission source. The county-wide area and mobile emissions are allocated by EMS-HAP to a specific census tract centroid within the county, based on surrogates such as population density, land use, roadway miles, etc., depending on the source category. For point source emissions, EMS-HAP processing is required whenever latitude/longitude coordinates or source emission characteristics (e.g., stack parameters) are missing. When the location coordinates are missing or suspect, EMS-HAP assigns a location using the zip code’s centroid (or if this is missing to a census tract centroid chosen at random within the source’s county).

To assess the extent of point source emissions that were assigned coordinates by EMS-HAP, the point source inventory for three metals (lead, cadmium, chromium) was investigated. Table 4-12 shows the percentage of sources and the corresponding percentage of mass for which point source coordinates were deemed suspect. It would appear that, although the percentage of sources with suspect locations was approximately the same (about 15 percent) for all three metals across the United States, the amount of mass differed greatly, ranging from 7 to 32 percent.

In part to check the accuracy of the stack release heights in the inventory, members of the EPA project team visited a lead smelter in Herculaneum, Missouri. According to data files available to the modeling team (Richard Daye, EPA Region 7, personnel communication), 89.91 tons per year of lead are emitted from the 550-foot stack in the center of the facility, and 7.7 tons per year are released as "fugitive" emissions, that is, escaping from the facility through open doors, windows, etc. However, the emissions for

an ongoing 2-month study at the facility suggest that the "fugitive" emissions are of order 50 tons rather than 7.7 tons. EPA cannot generalize to all other sources from one site visit, but this does reveal the possibility of the types of emission characterization uncertainties that can occur. All other factors being equal, an increase from 7.7 to 50 tons in low-level emissions would likely increase the modeled annual average concentration at the monitoring site by a factor of 3.

Table 4-12. Source location uncertainties for point source inventory for three metals.

	Lead	Cadmium	Chromium
Percent of sources with suspect coordinates	15.4	15.8	16.2
Percent of mass for which sources were assigned coordinates	16.9	31.6	6.6

EPA concluded that since surrogates (such as population) are used to allocate county-wide emissions to specific locations (rather than placing emissions at their true locations), and since almost 15 percent of the point source inventory have uncertain coordinates, EPA can anticipate that the ASPEN modeling system will likely not provide good correlation with observed concentration values at specific locations. If the modeling results are in accord with observations, it will be for those instances where point source emissions (location and release height) are well characterized, and where use of a county-level inventoried source category for the characterization of the other emissions is appropriate (i.e., the emissions are ubiquitous with respect to the monitoring location).

4.3.4.2 Meteorological Characterization Uncertainties

Meteorological data are a critical input for ISC-based Gaussian air quality models like ASPEN. EPA analyzed the effects of using 1990 Hourly United States Weather Observations (HUSWO) and 1996 International Surface Weather Observations (INSWO) databases on the annual average concentrations predictions. The 1990 and 1996 meteorological inputs differed in three respects: the methods in which the data were obtained, the number of stations employed, and differences in meteorological conditions between 1990 and 1996. Because the same emissions input and fixed receptor locations were used for both model simulations, differences in annual average concentration estimates between the model simulations are entirely attributable to the differences between the meteorological inputs for these two years.

The number of surface stations in most states increased in going from the 1990 HUSWO data set to the 1996 INSWO data set. In the 1990 model simulation, the mean source-to-meteorological station separation distance was approximately 70 km; this mean separation distance was reduced to under 50 km in the 1996 simulation by the inclusion of more meteorological observation locations. The increased density of surface stations in 1996 is anticipated to result in a better representation of the overall climate at most emission sources. Due to differences in reported variables, number and location of valid

stations, and meteorology itself, statewide annual-averaged concentration estimates were found to vary from 16.9 percent lower to 84.4 percent higher in 1996, as compared to 1990. Greater variability in modeled concentrations was found when comparisons were made at the receptor level demonstrating the importance of meteorology in dispersion-based models.

4.3.4.3 Model Formulation and Methodology Uncertainties

The ASPEN air quality model employs a Gaussian plume model for the characterization of the transport and dispersion. The particular algorithms employed within the model were extracted from version 2 of the Industrial Source Complex Long-Term model (ISCLT2). ISCLT2 has a history of development that dates back to the 1960's, so there is a history of usage of this model that allows EPA to anticipate the uncertainty in its estimates (given well-characterized emissions). In general, field study comparisons have shown that approximately 90 percent of the estimated annual averages are within a factor of two of those observed [*33*, *34*, *35*, *36*, *37*, *38*]. These comparisons were for sulfur dioxide concentrations at receptors that were generally within 20 km of the sources and so transformation and deposition effects would be considered negligible.

To verify that the model formulation algorithms were performing as anticipated, several investigations were conducted. In the first, EPA investigated whether the deposition algorithms were working properly. In the second, EPA jointly tested whether the dispersion characterizations were operating as expected, and whether the use of a “net” of receptors might lead to a bias in the modeling results.

The ASPEN model simulates the effect of dry deposition of particulate by adding an additional decay term to the emission rate in calculation of ambient concentrations. The decay term is a function of the deposition velocity, downwind distance from the source, and plume dimensions with respect to the mixing height. Deposition velocity is also a function of the particle size, wind speed, and the land-use type. The ASPEN model allows different deposition options for fine and coarse particulate and urban/rural environments. In order to analyze the effect of these options on the modeled ambient concentrations, EPA performed test case simulations using lead emissions from mobile nonroad sources in Colorado. Different compositions of fine/coarse fractions were used while the total emission rate was held constant. Five different scenarios were used for this test case: 10 percent fine and 90 percent coarse, 25 percent fine and 75 percent coarse, 50 percent fine and 50 percent coarse, 75 percent fine and 25 percent coarse, and 90 percent fine and 10 percent coarse. Emissions from 17 pseudo-point sources of 10m height, 1m/s exit velocity, and T = 295 K were considered. For fine particles, the ASPEN deposition velocities are generally similar to those estimated by ISC and scattered around the 1:1 ratio line. The deposition velocities for coarse particles are much higher for ASPEN than for ISCST3. The effects of these differences were extrapolated to the national scale by estimating the fraction of lead emissions that were assigned as coarse particles and as fine particles. For the entire U.S., the total lead emissions were 66.5 g/s and the percent contribution from different source categories was as follows: 49 percent of all lead emissions were accounted for by major sources, 28 percent by area sources, less than 0.01 percent by mobile onroad, and 23 percent by

mobile nonroad sources. For the ASPEN simulations this means that about 50 percent of all lead emission sources were treated as point sources and about 50 percent - as pseudo point sources. By this means, it was estimated that ASPEN would predict average lead concentrations in the air 20-30 percent lower than would typically be predicted by ISCST3 because of differences in coarse particle deposition velocities between the two models.

Gaussian dispersion models are designed to work with inert (non-reactive) pollutants. Although they can be made to handle linear production or removal effects, nonlinear chemistry effects are typically not treated. To address this limitation, a simple mechanism was built to estimate the concentration from those species that could be formed or destroyed in the atmosphere through secondary chemical reactions (secondary formation), and within the ASPEN modeling system, this secondary formed concentration is added to the concentration attributable to primary emissions. Analysis of ASPEN modeled nationwide mean values for formaldehyde and acrolein suggest that 23 percent and 44 percent (respectively) of the total modeled concentration is attributable to secondary formation. Results from a recent study using the research version of the EPA's Ozone Isopleth Plotting Program (OZIPR, a photochemical grid model), suggest that on a national scale, secondary formation for formaldehyde and acrolein would account for 90 percent and 85 percent (respectively) of the total modeled concentration [*39*]. However, there has not been any direct comparison of ASPEN and OZIPR modeled concentrations with equivalent emissions and meteorology, accounting for the fact that ASPEN provides values at specific locations while OZIPR provides grid-averaged values. Therefore, generalizing current results is problematic. However, these results do suggest that the ASPEN modeling system for reactive species may be underestimating (or overestimating) the total concentration, because ASPEN may be underestimating the component of the concentration that is produced (or destroyed) through nonlinear chemical reactions. At this point, EPA cautions that concentration estimates for reactive species should be considered more uncertain than for non-reactive species, all other factors being equal.

EPA specifically investigated whether the interpolation scheme used within ASPEN might be underestimating the modeled impacts. This concern arose because a "net" of receptors is employed by ASPEN, and then concentrations at specific points are estimated by interpolating within the "net". EPA wondered whether ASPEN might underestimate peak ambient concentrations because it might "average out" the peak values by combining them with lower concentrations nearby. To do this, EPA simulated three types of emissions sources, and compared the ASPEN estimates downwind from each source to the estimates derived from a more recent dispersion model, the Industrial Source Complex Long-Term Model Version 3 (ISCLT3). The simulations were run under a variety of wind speed conditions. The ASPEN estimates are lower than ISCLT3's by about 10 percent in the near distances, with the underestimation increasing to about 25 percent at 30 km downwind.

4.3.4.4 Uncertainty Due to Background

For all air toxics except for diesel PM, a uniform "background" concentration was

applied across all geographic areas to account for long-range transport. (The ASPEN model does not simulate the transport of any pollutant beyond 50 km from its original emission point or for emissions from natural sources.) The use of a uniform background concentration is a simplifying assumption because such extrinsic concentrations vary geographically for many of these pollutants.

4.3.4.5 Summary of Model Uncertainty Investigations

Past studies of the performance of long-term air quality models (for air toxics at more localized scales) suggest that 90 percent of the estimated concentrations should be within a factor of 2 of those observed, if the emissions are well characterized and the meteorological data are representative. If differences between observed and estimated concentration values differ greatly from what has historically been seen, one questions whether the model formulations are in error, whether the meteorological data is unrepresentative, or whether the emission characterization is in error. The investigations of the model formulation and methodology characterization uncertainties suggest that the differences in the dispersion algorithms between standard long-term algorithms and those within ASPEN are minor, contributing to differences of less than 10 percent in the near field. Significant differences were seen in the ASPEN deposition velocities for coarse particles, which were estimated to result in a bias to underestimate lead concentrations by roughly 30 percent. This bias is of importance only for those toxics simulated as having a significant fraction of coarse particles, as there was no bias seen in ASPEN deposition velocities for fine particles. Preliminary investigation of uncertainties associated with chemical reaction effects suggests that we should consider concentration estimates for reactive species as more uncertain than for non-reactive species. To improve the representativeness of the meteorological data, the INSWO surface weather observations data archive was employed. This reduced the average separation distance between source locations and observation meteorological sites, and is anticipated to aid in providing more representative meteorological data. The spot checks on location uncertainties for the three metals suggest that 6 to 30 percent of these emissions were assigned to default locations, rather than specified at their true locations. A site visit to one lead smelter suggests that close inspection of facilities in the vicinity of monitors might reveal other emissions not reported. These emission characterization uncertainties could have a greater impact on the model-to-monitor comparison results (resulting in differences of a factor of 3 or more) than uncertainties seen elsewhere in the modeling algorithms or the meteorological characterizations (which appear to result in differences of 30 to 80 percent).

4.4 Estimating Population Exposure

In this phase of the national-scale assessment, characterization of population exposure, ambient air toxics concentrations derived from ASPEN modeling were used to estimate human exposures to the 33 pollutants included in the ambient concentration analysis. The exposure assessment was conducted with the HAPEM4 model, which predicts annual average inhalation exposure estimates. Under the national-scale assessment, the exposure results serve two purposes. First, the results of the exposure assessment are directly presented on a pollutant-by-pollutant or geographic area-specific basis, for comparative purposes. Second, the inhalation exposure estimates serve as input to the

risk characterization (see section 4.3).

As applied in the national-scale assessment, the HAPEM4 model predicted a series of inhalation exposure concentrations for each pollutant, for 40 demographic groups, at over 61,000 census tract locations in the contiguous US, as well as Puerto Rico and the Virgin Islands. In all, the HAPEM4 model predicts over 74 million exposure estimates as part of the national-scale assessment. Therefore, to be useful, these results must be aggregated into the geographic areas and exposure demographic groups of concern. The aggregation of the 74 million exposure estimates occurs at two levels: at the census-tract level and across census tracts. At the census-tract level, aggregation is across the predicted exposures within and across different demographic groups. Across census tracts, aggregation of results to a larger geographic area (i.e., counties, states) is made to match the reliability of exposure estimates with that of the emissions inventory and air quality phases of the assessment.

For presentation and comparison of the exposure assessment results over a broad geographic area (i.e., counties, states), the study focuses on exposures derived for the "general population." For the risk characterization, an "age-related" breakdown of the exposure results is important in defining a lifetime (i.e., 70 years) exposure and because health responses to pollutants vary with age. Details on the aggregation of the HAPEM4 model results for these two purposes, in the national-scale assessment, are presented below.

4.4.1 HAPEM4 Census Tract Level Exposure Estimates

The HAPEM4 model is designed to predict inhalation exposure concentrations at the census-tract level. As applied in the national-scale assessment, the HAPEM4 model predicted a set of 30 annual exposure concentration estimates (i.e., 30 different activity pattern scenarios) for each of 40 demographic groups at each census tract, or a total of 1200 exposure concentration estimates per census tract.

It is important to note that, although these results from the HAPEM4 assessment are derived at the census-tract level, the results are this level are not reliable, due to recognized limitations in the emissions inventory and dispersion model results. However, to minimize data aggregation errors, the census tract results serve as the basis for determining pollutant exposure and risk distributions over larger geographic areas.

In the exposure assessment, certain demographic groups include commuting to other census tracts. As applied in the national-scale assessment, the exposure concentrations from these groups (for time spent in the home or work tracts) are recorded in their "home" census tract. Thus, if a person spends 8-hours in his "work tract" location, the exposure concentration predicted during this 8-hour period would be included in the person's "home tract" exposure concentration. In urban settings, the commuting feature can tend to "spread" the predicted exposure concentrations from central business districts (which generally have higher ambient concentrations) to the suburban areas (which generally have lower ambient concentrations).

Finally, for census tracts with "zero population," the HAPEM4 model will predict an exposure estimate of "zero," even though the tract may have a significant ambient concentration.

Variability Within a Census Tract

It is important to note that the predicted range of exposure estimates for each census tract (i.e.,1200 exposure concentrations) results from variability in demographic makeup, variations in activity patterns, and variations in commuting patterns and are derived from an ambient air quality estimate from a single location (at the census tract centroid as predicted by the ASPEN model). The modeling exercise did not attempt to quantify the variability in exposures across individuals within demographic groups. Thus, the HAPEM4 model, as applied in the national-scale assessment, does not predict a "complete" distribution of inhalation exposure concentrations across a census tract, only the distribution that results from "exposure-related" variations. This portion of the distribution is expected to be small compared to the actual variations in air quality across the tract. An air quality model applied at a more localized scale would be required to predict the "more complete" distribution of exposure concentrations across a census tract. Local scale modeling efforts have shown that ambient concentration gradients can easily span several orders of magnitude across a small geographic area, such as a census tract. The distribution resulting from "exposure-related" variations alone is expected to be far less than an order of magnitude at most locations across the study domain.

Figure 4-14 shows an example of this "exposure- related" variation across an example urban census tract. In this example, predicted POM exposure concentrations vary by less than 5 percent between different demographic groups and less than 1 percent within a specific demographic group. In general the youngest and oldest demographic groups considered (i.e., 0-5 year olds and 65+ years old) are predicted to have the lowest exposure levels. These groups are expected to spend more time indoors, where POM concentration levels are about 30 percent lower than outdoor levels (as defined by the HAPEM4 model). Thus, EPA can expect the annual average exposure levels for these groups to be lower than demographic groups which spend more time outdoors. This relatively small variation in exposure concentrations resulting from "exposure-related" variations can be found for most of the pollutants and locations included in the assessment.

Aggregation of Exposure Estimates Within a Census Tract

As noted above, the HAPEM4 model predicts 1200 exposure estimates at each census tract. These exposure estimates have been aggregated in two different ways. For presentation and comparison of the exposure assessment results over a broad geographic area, an assessment of exposure estimates to "the general population" is the most feasible approach. For the risk characterization, an "age-related" breakdown of the exposure results is important. These approaches are discussed further below:

Aggregation of Exposures for County, State, and National Level Estimates

Since the goal of the national-scale assessment is to assess exposure and risk levels across

a wide population distribution, and the variability of exposure concentrations resulting from "exposure-related" factors is small, for presentation and area-to-area comparison of exposure results (i.e., in the tables and charts discussed below), the 1200 exposure concentrations were characterized by a single "median exposure concentration" at the census-tract level. This was done by population-weighting the median exposure for each of the 40 demographic groups. This population-weighted "median exposure concentration" represents a "best estimate" of the population exposure for a given census tract. For the national-scale assessment, census-tract level "median exposure concentrations" were estimated for the contiguous United States, plus Puerto Rico and the Virgin Islands. This population exposure approach should be carefully considered when examining and interpreting the results of this national scale assessment. The national-scale assessment cannot be used to predict local scale "hot spot" exposures or to capture the range of exposures experienced by individuals, including the maximum exposed individuals.

Aggregation of Exposures for the Risk Characterization

Although the variation across different demographic groups was small (about 5 percent or less), for defining carcinogenic risks (see section 4.3), the variation across different age groups was retained to build a 70-year lifetime exposure period for a "typical" person at each census tract. This was done by aggregating the predicted median exposures for each of 5 age groups (0 - 5, 6 - 11, 12 - 17, 18 - 64, and 65+ years). For accessing non-cancer risks (see section 4.3), population weighted exposures were determined for 2 age groups, children (<12 years) and adults (12+ years).

4.4.2 National-Scale Assessment Exposure Estimates

As previously noted, the exposure results under the national scale assessment serve two purposes. First, the results of the exposure assessment are directly presented on a pollutant-by-pollutant or geographic area specific basis for comparative purposes. Under this assessment, the median census tract exposure concentrations (as described above) were used as the basis to derive the statistical distributions at the county, state, and national levels. Results of these distributions have been prepared in both a tabular and graphical format and are discussed further below. Second, the inhalation exposure estimates serve as input to the risk characterization. Results of these exposure estimates are presented as part of the risk characterization in section 4-3.

Presentation of County, State, and National Exposure Estimates

Aggregated exposure information, at the county, state, and national level is available in both tabular and graphical formats. The tabular information is summarized on a pollutant-by-pollutant basis for the entire study domain. Figure 4-15 shows an example of part of the tabular distribution tables (actual tables contain over 3300 lines). Graphically, three types of chart presentations are available, "Statewide Exposure Concentration Estimates," "Percent Contribution," and "Individual State Exposure Concentration Estimates." The "Statewide Exposure Concentration Estimates" and "Percent Contribution" charts have been prepared on a pollutant-by-pollutant basis. The "Individual State Exposure Concentration Estimates" are presented on a state-by-state pollutant basis. In addition, state and national maps of the exposure estimates on a

pollutant-by-pollutant basis have also been prepared. Examples of each chart, and a map, are presented in Figures 4-16 through 4-19, respectively. Also attached to each exposure assessment chart/map will be an additional text page (called “page 2”) that contains a set of caveats that the user is encouraged to review. A copy of “page 2,” along with a complete set of charts, is provided in Appendix K. The contents of each presentation type are described in further detail below.

Exposure Distribution Tables:
The tables contain exposure concentration distributions (i.e., as percentiles), in addition to a breakdown of exposure concentrations into major, area and other, onroad mobile, and nonroad mobile sources, as well as background concentrations for each pollutant at the county, state, and national levels. Results are also segregated by urban and rural county designation.

Statewide Exposure Concentration Estimates Charts:
These charts display a horizontal bar for each state, to represent the central part of the distribution of the exposure concentrations in that state (the 25th to 75th percentile), as well as the median value for the selected pollutant. If the air pollutant is a carcinogen, the bar chart also displays a line at the exposure concentrations representing the "1 in a Million Cancer Risk", the "10 in a Million Cancer Risk" and the "100 in a Million Cancer Risk"[18]. For pollutants with noncancer risks, the bar chart displays a line at the exposure concentrations representing a " Hazard Quotient = 1.0" (for reference purposes, some charts may contain a line representing a Hazard Quotient = 0.1). An example is provided in Figure 4-16.

Percent Contribution Charts:
These charts contain horizontal bars representing the percent contribution of four source types (major, area and other, onroad mobile, and nonroad mobile) as well as the contribution from the estimated background to the statewide average exposure concentration estimate for the selected pollutant (see Figure 4-17).

Individual State Exposure Concentration Estimates Charts:
These charts depict the median exposure concentrations for each pollutant in the NATA analysis on a single chart. A chart is provided for each state. Pollutant exposure concentration symbols are plotted to give the relationship of the state's median exposure concentrations to the cancer or noncancer risk level, where applicable. An example is provided in Figure 4-18.

[18] Concentrations at various levels of cancer risk and non-cancer hazard were determined using procedures described in section 3.3 and assume a continuous lifetime exposure to the concentrations predicted by this assessment. It is important to note that none of these risk-based concentrations represent regulatory standards.

National and State Maps of Modeled Exposure Concentrations:
These maps illustrate the modeled human exposure concentration to air toxics by county in 1996. Map colors indicate categories of exposure risk / hazard, and the corresponding ranges of inhalation exposure concentration. The exposure concentration value displayed in maps is the county median. Pollutant exposure concentration is expressed in micrograms per cubic meter of air (ì g/m3). Figure 4-19 provides an example of a state map with estimated exposure concentrations.

Interpretation of County, State, and National Exposure Estimates
It is important to note that the exposure distributions presented in this part of the national scale assessment represent the range of census tract median exposure concentrations across a geographic area (i.e., county, state, and national levels). As previously noted, the available information about the range of exposures across individuals or, spatially, within census tracts, is not reliable. This information is best suited to estimate "trends" in exposure or general population level exposures. The results of the exposure assessment presented at this level are best used to define broad geographic areas and pollutants of initial concern. A relatively low predicted exposure value does not necessarily mean that a particular area or pollutant is exempt from health-related air toxic issues. In certain situations, a more refined or local scale assessment may be needed to assess whether there is an air toxics problem. In areas with a relatively high predicted exposure value, the reliability of the estimate, and the data used to derive that estimate must be further examined before concluding that concern is warranted.

The results of the exposure assessment are only meaningful when examined at the individual county level or above. Comparison across geographic areas can be made by examining the graphical and tabular exposure results. However, it is important to note that the confidence in the predictions may be better in some geographic areas than others or may be better for certain pollutants. The variability in the quality of the predictions can vary from not only state-to-state but county-to-county within the same state. Thus, while the presentation materials contain multiple geographic areas (i.e., states compared on a single chart), the data are presented in this fashion for convenience and to minimize the number of charts/graphs that is required to summarize the assessment.

For reference, cancer and noncancer health criteria lines have been included on the graphical presentations. These have been placed only to compare the presented exposure estimates in a general context to the health criteria. It is strongly advised that the risk characterization section, as well as the assessment's limitations, be reviewed before assessing a particular health concern in a specific geographic area. A discussion of exposure results on a pollutant-by-pollutant basis is made in the risk characterization results section.

Care should be taken when trying to interpret the geographic distribution of exposure concentrations across a county. Figure 4-20 presents the range of exposure concentrations across tracts within a county. This shows that, for benzene exposure estimates, the range of exposures within a county (the tract-to-tract variations) appears to

be directly related to the population of that county. Thus, at least for benzene, the most populated counties are predicted to have the largest range in exposure concentrations, and thus the greatest uncertainty in the geographic distribution of these estimates. It is not clear whether this is a true relationship or an artifact of the assessment, in that a denser modeling (both ambient and exposure) grid is utilized in more populated areas (more census tracts).

4.4.3 Comparison of HAPEM4 Exposure Concentrations to ASPEN Ambient Concentrations

To illustrate the contribution of the HAPEM4 model to the assessment, census tract median exposure concentrations were compared to corresponding census tract median ambient concentrations as predicted by ASPEN. Table 4-13 shows the average ratio of HAPEM4 to ASPEN for each of the study pollutants. This ratio is presented for total levels as well as for each of the five source sectors considered in the assessment (i.e., major, area and other, onroad mobile, nonroad mobile, and background). In general the HAPEM4 exposure predictions are 5-40 percent lower than the corresponding predicted air quality values. This reduction likely results from the inability of many pollutants to penetrate efficiently into an indoor environment (see section 3.2.3). Exposures resulting from emissions originating from major, area, nonroad mobile, and background source sectors are seen to be about 20-30 percent lower than the corresponding predicted air quality values for most gaseous pollutants, with some metals approaching a 40 percent reduction from air quality levels. Exposures resulting from emissions from onroad mobile sources range from 40 percent lower than the corresponding predicted air quality values for chromium to 24 percent greater than the predicted air quality values for benzene. Benzene exposure levels for this source sector are most likely higher than their corresponding ambient levels because of the relatively high “proximity term” (see section 3.2.3) assigned to the “in-vehicle” microenvironment. This “proximity term” is required to adjust the ASPEN predicted ambient level, which is assumed representative of the census tract centroid, to that which EPA would expect immediately outside of the microenvironment. For most microenvironments this term was set to unity. However, for the transportation related microenvironments it is presumed that the ambient concentrations immediately outside the vehicle (i.e., very close to the pollutant source) are considerably higher than the census tract centroid value predicted by ASPEN. Thus for pollutants where emissions are significantly dominated by onroad mobile sources an appropriate proximity term was developed and applied specific for this assessment. For example, for benzene, a proximity term of 6.6 was applied to the ASPEN census tract centroid levels to predict the expected ambient concentration immediately outside a vehicle.

Table 4-13. HAPEM4 to ASPEN Average Ratio[1].

Pollutant	Source Sector					Total
	Major	Area and Other	Onroad Mobile	Nonroad Mobile	Estimated Background	
Acetaldehyde	0.79	0.79	0.93	0.80		0.87
Acrolein	0.82	0.80	1.06	0.82		0.92
Acrylonitrile	0.75	0.75				0.75
Arsenic	0.79	0.79	0.80	0.79		0.79
Benzene	0.85	0.85	1.24	0.85	0.85	0.98
Beryllium	0.79	0.79		0.79		0.79
1,3-Butadiene	0.83	0.83	0.93	0.84		0.90
Cadmium	0.79	0.79		0.79		0.79
Carbon Tetrachloride	0.71	0.71			0.72	0.72
Chloroform	0.82	0.82			0.82	0.82
Chromium	0.64	0.63	0.66	0.66		0.64
Coke Oven Emissions	0.75	0.75				0.75
1,3-Dichloropropene	0.82	0.83				0.83
Diesel Particulate Matter			0.72	0.70		0.71
Ethylene Dibromide	0.79	0.79			0.79	0.79
Ethylene Dichloride	0.87	0.87			0.87	0.87
Ethylene Oxide	0.79	0.79				0.79
Formaldehyde	0.69	0.69	0.89	0.72	0.69	0.75
Hexachlorobenzene	0.81	0.81			0.81	0.81
Hydrazine	0.83	0.83				0.83
Lead	0.84	0.84	0.84	0.84		0.84
Manganese	0.74	0.73	0.75	0.75		0.73
Mercury	0.79	0.79	0.80	0.80	0.79	0.79
Methylene Chloride	0.80	0.81			0.80	0.80
Nickel	0.79	0.79	0.80	0.79		0.79
Polychlorinated biphenyls (PCBs)	0.79	0.79			0.79	0.79
Perchloroethylene	0.75	0.76			0.75	0.75
Polycyclic Organic Matter (7-PAH)	0.75	0.75	0.76	0.78		0.75
Polycyclic Organic Matter (Total)	0.76	0.76	0.76	0.77		0.76
Propylene Dichloride	0.79	0.79				0.79
Quinoline	0.75	0.75				0.75
1,1,2,2-Tetrachloroethane	0.79	0.79				0.79
Trichloroethylene	0.84	0.84			0.84	0.84
Vinyl Chloride	0.75	0.75				0.75

[1] Average ratio developed by comparing (HAPEM4 Concentration)/(ASPEN Concentration) at each census tract

4.4.4 Discussion of HAPEM4 Limitations and Uncertainties

Like any model, especially when applied on a very broad geographical scale, HAPEM4 relies on a number of assumptions and approximations in estimating inhalation exposures. In general, the accuracy of the results is primarily limited by:

1. The emissions inventory and dispersion modeling results;

2. Reliability of ME factors for various pollutants;

3. Activity pattern information (e.g., patterns of time spent in various microenvironments for the populations in the geographic areas modeled); and

4. The ability of the chosen population cohorts to adequately represent the true demographics of every census tract.

More specifically, limitations and uncertainties include:

1. Uncertainty with the emissions inventory and the ASPEN dispersion modeling, which was discussed in the previous sections.

2. Indoor emission sources - The exposure estimates do not include exposures related to emissions of air toxics from indoor sources (e.g., off-gassing from building or consumer products, smoking, internal combustion sources, etc.). Indoor sources of some air toxics are likely to be important in assessing total inhalation exposures, and will be addressed in the future as additional data are obtained and new analyses are conducted.

3. Spatial resolution - Because of the spatial uncertainties associated with air toxics emission inventory data and the associated ASPEN model ambient concentration estimates, results of the HAPEM4 inhalation exposure model are compiled and presented at county level or larger spatial scales.

4. Exposure routes - The HAPEM4 model only estimates inhalation exposures, since it is based on ambient air concentration data as input. Thus, it does not address exposures through other pathways (e.g., ingestion). This is especially important for toxic pollutants that are persistent and bioaccumulate, such as mercury, dioxins, and PCBs. Emissions of these pollutants disperse through the atmosphere and eventually deposit to land or water bodies. Once deposited, they can bioaccumulate up the food chain. For example, mercury bioaccumulates most efficiently in the aquatic food chain (e.g., fish). Fish consumption is the primary route of exposure to mercury [*40*]. For dioxins, (which bioaccumulate mainly in animal fat tissue) population exposures are primarily due to ingestion of dairy products, fish, beef, pork, and poultry [*41*]. Multimedia exposure models are needed to address such multipathway exposures. These will be included in future national-scale assessments as appropriate tools and data become available.

5. Population exposure estimates - The exposure estimates represent midrange estimates of population exposures. Due to a number of factors, some individuals may have substantially higher or lower exposures. It is important to note that the exposure model, as applied on the national scale, is not designed to quantify these extreme values of individual exposures.

6. Representativeness of the ME factors - Exposure models must predict the relationship between the ambient air quality (outside) and that in a microenvironment (inside or outside). When applied on a local scale, exposure models can employ detailed mass balance equations to predict this relationship. However, on a national scale, the development of such a detailed relationship is not feasible. Thus, the HAPEM4 model, applied on a national scale, relies on generalized ME factors, which do not account for variability (e.g., penetration affected by air exchange rate, which is a function of ambient temperature, heating/cooling system, and open windows). These factors can be represented as simple first-order relationships between the outdoor and the indoor air quality. For some pollutants and microenvironments this relationship is well-documented and well-understood. However, for many pollutants and microenvironments this relationship is either not clear or has not been measured. As part of this assessment, EPA conducted a detailed study of exposure literature to develop ME factors for each air toxic in the study [19]. This ME study has undergone a separate technical peer review. The technical reviewers agreed that there is a great degree of uncertainty associated with these numbers. They have suggested that this uncertainty be assessed either qualitatively or quantitatively as part of this study. In future versions of this assessment EPA plans to define these ME factors as distributions rather than fixed "best estimate" values as was employed for this initial assessment.

7. Representativeness of the population cohorts - The assessment assumed that the 40 cohort groups selected can represent the activity patterns of the general population in all areas of the country. The groups were selected to represent variability in population activity patterns while at the same time maintaining the ability to present the exposure assessment results in a manner that will allow for an adequate lifetime exposure aggregation. It is also possible that members of the same cohort may have significantly different activity patterns in different census tracts. The usage of the 40 cohort groups reflects the finest resolution that EPA believes is possible with currently-available models.

8. Representativeness of the activity pattern sequence - When selecting multiple 24-hour activity patterns to construct an annual average pattern, patterns are combined that pertain to different individuals, so that day-to-day correlations in activities are not preserved. For example, for day 1 the pattern may specify a house with an attached garage, and for day 2 a house without an attached garage. In this situation, the HAPEM4 model would underestimate the annual average exposure for a person residing in a house with an attached garage, and

overestimate the exposure of the person in the house without an attached garage, and overestimate the exposure of the person in the house without an attached garage. As a result the aggregated activity pattern is more representative of a population average pattern for the demographic group, than any individual pattern. Thus, the distribution of exposure concentrations for the group estimated by HAPEM4 represents the uncertainty in the population average exposure concentration, rather than the variability of the individual exposure concentrations among members of the group. Uncertainty and variability of input data other than activity data are not considered, so that the resulting uncertainty information provided by the prediction distributions is an underestimate of the overall uncertainty.

5 Risk Characterization

5.1 Introduction

As described in Section 2, this assessment was based on EPA's paradigm for risk assessment, a framework to assess and manage risks developed by the National Academy of Sciences in 1983. The paradigm divides the risk assessment and risk management process into different general phases (as shown in Figure 2-1). The phases that comprise risk assessment are (1) exposure assessment (which describes how humans come into contact with pollutants), (2) dose-response assessment (which describes the adverse health effects the pollutants may produce and at what doses these effects may occur), and (3) risk characterization (which combines the exposure and dose-response information to draw qualitative or quantitative inferences about risk). The risk characterization section presents an interpretation and discussion of the results of the NATA national-scale assessment, including the uncertainties associated with each element, and makes specific recommendations for future directions of air toxics assessments on the basis of this information. Complete results for the risk characterization portion of the assessment are provided in Appendix L.

This risk characterization examines inhalation risks for 32 hazardous air pollutants associated with 1996 emissions in urban and rural areas nationwide. This assessment was conducted on a coarse spatial resolution (i.e., census tracts) and used many simplifying assumptions. Therefore, it should be noted that individuals within a census tract may have substantially higher or lower exposures (and concomitant risks) than estimated here. The characterization is intended to help identify pollutants of greatest potential concern (among these 32), prioritize efforts to reduce emissions, provide a baseline for measuring future trends, and to help set research priorities. EPA plans to update this assessment every three years. The next assessment will focus on 1999 emissions, concentrations and risks.

As described in Section 3, several risk presentation formats have been used. Cancer risks to individuals are presented as lifetime (e.g., 70-year) individual risks. These risks are expressed in terms of the estimated "upper-bound" (i.e., likely actually to be lower, but may be higher) probability that a person with the median exposure in a census tract will contract cancer. Non-cancer hazard to individuals is expressed in terms of the hazard quotient, defined as the ratio between the estimated median exposure in an individual's census tract and the reference concentration (or similar value). The reference concentration is an exposure that is believed to be without significant risk of adverse non-cancer health effects in a chronically-exposed population, including sensitive individuals.

Cancer risks to individuals exposed to multiple pollutants were combined by summing, with known human carcinogens kept separate from probable and possible carcinogens. Non-cancer hazards for multiple pollutants were combined by summing hazard quotients for pollutants affecting the same target organs to create a target organ-specific hazard index (TOSHI) for non-carcinogenic effects. Non-carcinogenic pollutants having "high certainty" RfCs were summed separately from those with "low certainty" RfCs. Due to

the broad scale of the assessment, the risk characterization focused on results at the national level, which is the level at which EPA believes the results are most meaningful.

Estimated census tract median cancer risks and non-cancer hazards to individuals have been shown as box plots. The plots include a range extending from the 5th to the 99th percentile census tracts. Cancer risks and non-cancer hazards to populations have been expressed as total numbers of people (i.e., not truncated at the 5th and 99th percentiles) who reside in census tracts where the estimated median risk (or hazard quotient) exceeds fixed levels within the contiguous US, Puerto Rico, and the Virgin Islands.

For a complete understanding of the risk characterization, it is important to remember that the scope and methods of this assessment have imposed the following limitations and uncertainties:

1. Risk and hazard quotient levels are not regulatory levels. The determination of what is an acceptable or unacceptable risk depends on additional factors and more refined information.

2. The risk estimates presented are based on the assumption that pollutant exposures would remain at 1996 levels over a lifetime. They did not take into account significant reductions that have taken effect since 1996, or future reductions expected from: 1) mobile sources; 2) major industrial sources; 3) State or industry initiatives; and 4) facility closures. For example, EPA expects exposures to some gaseous air toxics from onroad mobile sources to be reduced about 50% by 2007 and 60% by 2020. While such a "snapshot" type of assessment may not be able to provide direct estimates of absolute risk levels, it nonetheless provides a useful yardstick against which one may evaluate the risk-reduction potential of hypothetical risk reduction scenarios, and which may be used to assess the relative contributions of various pollutants and source groupings to national-scale risks.

3. The risk estimates for chromium, nickel, and polycyclic organic matter were based on conservative assumptions of speciation that were applied uniformly to all areas. Actual risks associated with these pollutants may be lower than estimated in some areas.

4. The risk estimates represent risks associated with midrange estimates of population exposures within each census tract. Some individuals may have had substantially higher or lower exposures and risks. It is important to understand that the dispersion and exposure models used by the assessment were not designed to quantify inter-individual variability in exposures.

5. All cancer risk estimates should be considered as conservative, but not worst-case. Because they represent a composite of upper bound UREs and median population exposures (which may be underestimated), the true risks would

probably be less, but could be greater. For more highly exposed segments of the population, these cancer risk estimates would be correspondingly higher.

6. EPA is currently reassessing the carcinogenic effects of 19 of the air pollutants included in this study. Cancer unit risk estimates could change substantially as a result of these reassessments.

7. For pollutants that have more than one unit risk estimate (e.g., benzene and vinyl chloride), this characterization uses the highest available unit risk. For more details about these unit risks, see Appendix G.

8. This characterization presents non-cancer hazard in terms of the hazard quotient, which is the ratio of a given exposure level to the reference concentration (RfC) or similar value for a pollutant. The RfC is an estimate of the continuous lifetime inhalation exposure that the EPA believes is likely to have no appreciable risk of deleterious non-cancer effects. Although hazard quotients below 1.0 (i.e., exposures below the RfC) are believed safe, hazard quotients above 1.0 are not necessarily harmful. Nevertheless, as the hazard quotient increases above 1, potential for adverse effects also increases.

9. EPA combined non-cancer hazard estimates for multiple pollutants using the target-organ-specific hazard index (TOSHI), defined as the sum of hazard quotients for individual toxic air pollutants that affect the same organ or organ system. This method is a simplified approximation of the potential aggregate effect because different substances may affect the same organ in different and non-additive ways. As with the hazard quotient, aggregate exposures below a hazard index of 1.0 will likely not result in adverse non- cancer health effects over a lifetime of exposure. However, a hazard index greater than 1.0 does not necessarily suggest a likelihood of adverse effects.

10. Estimates of the number of people at various cancer risk or non-cancer hazard levels were based on census tract population estimates from 1990, the most recent available. The total population of the contiguous US, Puerto Rico, and the Virgin Islands in 1990 was 251 million, including 207 million adults and 44 million children.

11. Exposure pathways other than inhalation, as well as indoor sources of air toxics, may contribute substantially to total risks for some pollutants. This assessment does not address oral or dermal exposures, or inhalation exposure resulting from indoor sources.

12. The simplifying assumptions necessary for national-scale modeling have introduced significant uncertainties into each component of the assessment. Because of these uncertainties, EPA will not use the results of this assessment to determine source-specific contributions or to set regulatory requirements.

These uncertainties are discussed in greater detail in Section 5.4. This section includes qualitative descriptions of all major sources of uncertainty and variability in the national-scale assessment, provides a simple illustration of one method by which the magnitude of variability and uncertainty can be estimated, and provides recommendations for future efforts for a more complete quantification of variability and uncertainty in the future.

5.2 Cancer Risks

5.2.1 Pollutant-Specific Cancer Risks to Individuals

Figure 5-1 shows the distribution of estimated cancer risks for each of the pollutants nationwide, from the 5th to the 99th percentile. Risks were based on the median exposure within each of approximately 61,000 census tracts nationwide. Because census tracts are intended to include more or less similar population sizes, the distribution of tract median risks should be generally representative of the distribution of risks for "typical" individuals in the US. It should be noted that this distribution does not represent risks to all individuals in the US. Some individuals within a census tract may have higher or lower exposures (and concomitant risks) that those shown. (For example, the 5th percentile risk level on the figure indicates that approximately 5% of the population lives in census tracts where the median risks are at that level or lower. The 99th percentile risk level indicates that 99% of the population lives in census tracts where the median risks are at that level or lower.)

The extremes at either end of the distributions (i.e., less than the 5th and greater than the 99th percentiles) were not shown because EPA believes that, given the broad scope of this assessment, these risk estimates were less reliable than the information shown on the graphs. That does not mean these risks are unimportant, however. For example, approximately 2.5 million people resided in census tracts where the median estimated risks were higher than the 99th percentile risk shown. Because this assessment was designed to evaluate average exposure and risk at the national scale, more refined local-scale assessments will be needed to adequately characterize exposures and risks at the upper end of the national range.

In general, narrow distributions on Figure 5-1 (e.g., for PCBs or carbon tetrachloride) suggest that background sources were dominant. Risks associated with these background-dominated pollutants were similar in all tracts. Broad risk distributions (e.g., for coke oven emissions) suggest dominance by major sources that can strongly affect limited areas. Risks associated with these pollutants varied with location by many orders of magnitude. Distributions intermediate between narrow and broad (e.g., for benzene or perchloroethylene) suggest dominance by mobile or area (and other) sources whose impacts were more widespread than major sources but less widespread than background. Detailed risk distributions for major, area, mobile, and background sources are shown in Appendix L (Figures 1-5).

Based on an examination of Figure 5-1, pollutants can be grouped into scale-related categories as follows:

National risk drivers: Figure 5-1 shows that, for at least 50% of the US population, estimated inhalation cancer risks associated with three pollutants – benzene, carbon tetrachloride, and formaldehyde – approached or exceeded 10 in 1 million. Benzene and formaldehyde risks were associated primarily with mobile and background sources, whereas carbon tetrachloride risks were virtually all from background.

Regional risk drivers: Four more pollutants – chromium, ethylene oxide, polycyclic organic matter, and coke oven emissions – showed estimated cancer risks exceeding 10 in 1 million for more than 1% of the US population. The first three of these pollutants originated primarily from area (and other) sources; the fourth was associated entirely with major sources.

Important national contributors: Estimated risks to 50% or more of the US population for five more pollutants– acetaldehyde, 1,3-butadiene, ethylene dibromide, ethylene dichloride, and perchloroethylene – were near or above 1 in 1 million. Mobile sources were most important for emissions of acetaldehyde and 1,3-butadiene, background sources for ethylene dibromide and ethylene dichloride, and area sources for perchloroethylene.

Important regional contributors: Nine more pollutants – arsenic, nickel, acrylonitrile, cadmium, chloroform, 1,3-dichloropropene, hydrazine, quinoline, and trichloroethylene – showed estimated cancer risks exceeding 1 in 1 million for some of the US population. Major sources contributed important amounts of hydrazine and quinoline, although area (and other) sources were also important for these pollutants. Risks for the remaining seven pollutants were dominated by area (and other) sources. (Note that one pollutant that met this condition, 7-PAH, was omitted from this discussion because it is redundant with total POM. Because total POM is a more inclusive group, discussion focuses on it.)

Cancer risk to individuals:

- National risk drivers: benzene, carbon tetrachloride, formaldehyde
- Regional risk drivers: chromium, ethylene oxide, polycyclic organic matter, coke oven emissions
- Important national contributors: acetaldehyde, 1,3-butadiene, ethylene dibromide, ethylene dichloride, perchloroethylene
- Important regional contributors: arsenic, nickel, acrylonitrile, cadmium, chloroform, 1,3-dichloropropene, hydrazine, quinoline, trichloroethylene

For the remaining carcinogenic pollutants in the national-scale assessment – vinyl chloride, beryllium, hexachlorobenzene, lead, methylene chloride, PCBs, propylene dichloride, and 1,1,2,2-tetrachloroethane – 99% of the US population lived in census tracts where the median cancer risk did not exceed 1 in 1 million. It is important to note, however, that this result does not rule out potential concerns for these pollutants. First, this national-scale assessment lacks the resolution to evaluate all local inhalation risk hot spots. Therefore, there may be locations where these substances pose significant risks due to concentrations in air, but this assessment lacked the resolution to detect them.

Additionally, there is the potential for the omission of important sources in the inventory, especially since this is the first time such a comprehensive inventory has been attempted. Furthermore, beryllium, hexachlorobenzene, lead, and PCBs all tend to accumulate in soil and are persistent in the environment. Because this assessment was limited to inhalation exposure, these substances may pose important risks via the ingestion pathway that were beyond the scope of this assessment.

In general, the most important national-scale "risk drivers" originated predominantly from mobile (i.e., benzene, formaldehyde) or background (i.e., carbon tetrachloride, formaldehyde) sources. Most of the "second-tier" pollutants originated mostly from area or background sources. Pollutants associated with major sources were generally not seen to be as important nationally, but were very important in some regions. Given the resolution of this national-scale assessment, this result is not very surprising; the assessment tools are simply inadequate to quantify localized impacts around isolated stationary sources. More rigorous localized assessments will be needed to quantify risks associated with such individual sources; this is one of the goals of the assessments being carried out as a part of EPA's residual risk assessment efforts. Finally, it is important to realize that "background" in this assessment was really a miscellaneous category, attempting to capture sources that were not inventoried or sources beyond 50 km that were inventoried. The category included natural background emissions, historic emissions of persistent substances, international transport, and interstate transport. The first three of these subcategories are not controllable under the Clean Air Act, but elements of the fourth – interstate transport – are. Thus, some part of the background sources component of risk for each substance may be addressable by the air toxics program. This addressable portion likely varies among different substances, and the current state of EPA's dispersion models and databases is insufficient for quantitative estimation.

5.2.2 Pollutant-Specific Cancer Risks to Populations

Figure 5-2 shows numbers of people residing in census tracts for which the median exposure corresponds with estimated lifetime cancer risks exceeding three fixed levels (100, 10, and 1 in one million) for emissions from all sources combined. The same information for each source sector (major, area, mobile, and background sources) is provided in Appendix L (Figures 13-17). Unlike the individual risk figures (which exclude the extreme ends of the distribution), these population figures include the entire assessed population in order to increase sensitivity to those potential urban-scale hot spots that affect the median exposure estimate at the census-tract level.

Based on an examination of Figure 5-2, pollutants can be grouped into scale-related categories as follows:

National risk drivers: Figure 5-2 shows that three pollutants – benzene, formaldehyde, and chromium – presented estimated risks of 10 in 1 million or higher to more than 10 million people. The first two pollutants posed such risks for more than 100 million people. Contributions of individual source sectors (shown in Appendix L, Figures 13-17) suggest that benzene originated predominantly from a mixture of onroad and nonroad

mobile sources, but formaldehyde was emitted predominantly by nonroad mobile sources. Both of these pollutants had an important background component (i.e., greater than 1 in 1 million risk to the entire population) as well. In contrast, chromium emissions originated predominantly from a mixture of major and area sources.

Regional risk drivers: Coke oven emissions posed estimated risks of 100 in 1 million or higher to more than 100,000 people, and acrylonitrile, arsenic, and hydrazine presented such risks to more than 10,000 people. Arsenic is emitted by both major and mobile sources; the other three pollutants are emitted predominantly or exclusively by major sources. These substances may be good candidates for more refined, local-scale analyses in areas where emissions sources exist.

Cancer risk to populations:

- National risk drivers: benzene, formaldehyde, chromium
- Regional risk drivers: arsenic, coke oven emissions, acrylonitrile, hydrazine
- Important national contributors: acetaldehyde, carbon tetrachloride, ethylene dibromide, ethylene dichloride, polycyclic organic matter, perchloroethylene
- Important regional contributors: 1,3-butadiene, cadmium, 1,3-dichloropropene, ethylene oxide

Important national contributors: Six more pollutants – acetaldehyde, carbon tetrachloride, ethylene dibromide, ethylene dichloride, polycyclic organic matter, and perchloroethylene – presented risks of 1 in 1 million or higher to more than 100 million people. Mobile sources were most important for emissions of acetaldehyde, background sources for carbon tetrachloride, ethylene dibromide, and ethylene dichloride, and area (and other) sources for polycyclic organic matter and perchloroethylene.

Important regional contributors: Four more pollutants –1,3-butadiene, cadmium, 1,3-dichloropropene, and ethylene oxide – posed estimated cancer risks of 1 in 1 million or higher to more than 10 million people. Cadmium, 1,3-dichloropropene and ethylene oxide originated mostly from area sources, and 1,3-butadiene from mobile sources (mostly onroad).

The remaining carcinogenic pollutants in the national-scale assessment – nickel, vinyl chloride, beryllium, chloroform, hexachlorobenzene, lead, methylene chloride, PCBs, propylene dichloride, quinoline, 1,1,2,2-tetrachloroethane, and trichloroethylene – were estimated to exceed risks of 1 in 1 million for fewer than 10 million people and to exceed risks of 10 in 1 million for fewer than 1 million people. As noted in the discussion of individual risk, this result does not exonerate these pollutants. In fact, Figure 5-2 shows that beryllium, chloroform, and quinoline contribute estimated inhalation risks exceeding 10 in 1 million for more than 100,000 people, making them potentially important urban-scale concerns. Also, as noted previously, potentially important ingestion exposures for beryllium, hexachlorobenzene, lead, and PCBs were not included in the assessment.

5.2.3 Aggregate Cancer Risks of Multiple Pollutants to Individuals

Figure 5-3 shows the distribution of estimated cancer risks for multiple pollutants combined, from the 5th to the 99th percentile in the US population. The risks are based on median exposures within each census tract nationwide. Known human carcinogens were aggregated separately from probable and possible carcinogens, and the figure shows risks by individual source sector, and for all source sectors combined.

The median aggregate risk estimate for all known carcinogens and source sectors combined was 10 in 1 million and the 99th percentile risk was 60 in 1 million. The median aggregate risk estimate for all probable and possible carcinogens and source sectors combined was 20 in 1 million and the 99th percentile risk was 100 in 1 million. While EPA has not combined risk estimates for known and probable/possible carcinogens because of their different magnitudes of relative uncertainty, it appears reasonable to assume that total risk associated will all carcinogens in this assessment could approach 30 in 1 million risk to the median US receptor.

Background sources alone were estimated to provide an aggregate risk of approximately 3 per million for known carcinogens and 4 per million for probable/possible carcinogens. Because background exposures were assumed to be ubiquitous, they effectively established the "floor" for the distributions of total risk. Major sources contributed little to the central part of the total risk distributions, but made dominant contributions to the upper end of the distribution for known carcinogens. Mobile sources were the most important contributors to total risk for known carcinogens; area and mobile sources were of roughly equal importance in determining total risk for probable and possible carcinogens.

5.2.4 Aggregate Cancer Risks of Multiple Pollutants to Populations

Figure 5-4 shows numbers of people residing in census tracts for which the median exposures to all carcinogens combined corresponded with estimated lifetime cancer risks exceeding three fixed levels – 100, 10, and 1 in one million. As before, known carcinogens were aggregated separately from probable and possible carcinogens. The figure shows affected populations by source sector, and for all source sectors combined.

Based on Figure 5-4, approximately 130 million people resided in census tracts where cancer risk exceeded 10 in 1 million, and 800,000 lived in tracts where risk exceeded 100 in 1 million from known carcinogens. For probable and possible carcinogens, approximately 200 million people lived in tracts where risks exceeded 10 in 1 million and 2 million lived in tracts where risks were above 100 in 1 million.

As already noted, the entire assessed population experienced estimated risks exceeding 1 in 1 million from background sources alone. For known carcinogens, major sources were responsible for most risks exceeding 100 in 1 million. For probable/possible carcinogens, risks above 100 in 1 million were associated more or less equally with major, area, and nonroad mobile sources. The greatest contributors to "intermediate risks" (i.e., between 10 and 100 in a million) were onroad and nonroad mobile sources.

5.3 Non-Cancer Hazard

5.3.1 Pollutant-Specific Hazard Quotient for Individuals

Figure 5-5 shows the distribution of non-cancer hazard quotient (HQ) for 27 pollutants nationwide, from the 5th to the 99th percentile. As with the cancer risk figures, HQs were based on the median exposure within each census tract nationwide and are therefore likely to be similar to the distribution of risks for individuals in the US. The extremes at either end of the distributions were truncated at the 5th and 99th percentiles, but readers should keep in mind that approximately 2.5 million people reside in census tracts where the median HQs are higher than the upper end of the distribution on the graphs.

In general, a very narrow distribution on Figure 5-5 (e.g., chloroform, ethylene dibromide) suggests that background sources were dominant. HQs associated with these pollutants were similar in all tracts. A very broad risk distribution (e.g., lead) suggests dominance by major sources or by a few large area sources. Hazards associated with these pollutants varied with location by several orders of magnitude. Distributions in between narrow and broad (e.g., benzene, manganese) suggest that dominant sources were either mobile or area sources that impacted more areas than major sources but were less ubiquitous than background. Detailed risk distributions for major, area, mobile, and background sources are shown in Appendix L (Figures 7-11).

National hazard drivers: Figure 5-5 shows that for at least 50% of the US population, the inhalation HQ associated with a single pollutant – acrolein – was approximately 4. The HQ for the most exposed 1% of the population was approximately 20. No other pollutants approached within an order of magnitude of acrolein's HQ distribution. Most of the acrolein exposure was associated with mobile sources, with area (and other) sources also contributing.

It is important to note that many reference concentrations incorporate protective assumptions in the face of uncertain data. For this reason, an HQ greater than 1.0 does not necessarily suggest a likelihood of adverse effects, whereas an HQ less than 1.0 does suggest that adverse effects are unlikely. Furthermore, the HQ cannot be translated to a probability that adverse effects will occur, and is not likely to be proportional to risk. An HQ greater than one can be best described as indicating that a potential may exist for adverse health effects, warranting further investigation.

Three more pollutants – formaldehyde, acetaldehyde, and manganese – showed HQs exceeding 0.1 for some of the US population. While an HQ of 0.1 does not suggest a present potential for adverse health effects, the fact that these HQs are within an order of magnitude of 1.0 suggests that some potential may exist for adverse health effects if emissions increase in the future. Also, these substances could potentially combine with other pollutants to present an aggregate threat, or pose local threats. The remaining pollutants in the national-scale assessment were found not to contribute HQs exceeding 0.1 for 99% of the US population. As with carcinogens, this result does not exonerate these pollutants because of the coarse resolution of the assessment, potential omissions in the inventory, and the fact that ingestion exposure was not assessed.

5.3.2 Pollutant-Specific Hazard Quotient for Populations

Figures 5-6 and 5-7 show numbers of adults and children, respectively, residing in census tracts where median exposures exceeded three fixed HQ levels (0.1, 1.0, and 10) for emissions from all sources combined. The same information is provided in Appendix L (Figures 19-23 and 25-29) for each source sector (major, area, mobile, and background sources). Unlike the individual HQ figures (which exclude the extreme ends of the distribution), these population figures include the entire assessed population, with the goal of increasing sensitivity to identify potential urban-scale areas of concern. (As already noted, the national-scale assessment was designed to assess exposure and risk levels across large populations. Therefore, this use to identify urban-scale areas may produce false negatives.)

Pollutants can be grouped in categories of decreasing importance in the assessment as follows:

National hazard drivers: Figures 5-6 and 5-7 show that one pollutant, acrolein, presented an HQ exceeding 10 to more than 20 million adults and 4 million children. Virtually all adults and children in the US population lived in census tracts where the median HQ exceeded 1.0. Similar results were obtained by considering mobile sources alone (Appendix L, Figures 21, 22, 27, and 28).

Regional hazard drivers: Three more pollutants – formaldehyde, acetaldehyde, and manganese – presented HQs greater than 1.0 to more than 100,000 adults and 20,000 children. Most of the formaldehyde and acetaldehyde exposures were attributable to nonroad mobile sources, while manganese exposures originated primarily from area sources.

Important regional contributors: Two more pollutants – cadmium and arsenic – presented HQs greater than 1.0 to more than 10,000 people. Arsenic contributions originated mostly from a mixture of major and area sources, and cadmium came mostly from area sources alone.

5.3.3 Aggregate Target Organ Specific Hazard Index of Multiple Pollutants to Individuals

Aggregating hazards of multiple pollutants that have the potential to cause adverse health effects other than cancer is inherently more complex than assessing cancer risk because (1) it is necessary to consider different toxic effects and mechanisms that may not be additive, and (2) it is necessary to consider adults and children separately.

Regarding the first issue, the most effective way to aggregate hazards for multiple pollutants would be to combine hazard quotients for pollutants that cause the same adverse effects by the same toxic mechanism. However, because detailed information on toxic mechanisms was not available for most of the substances in this assessment, EPA used a simpler and more conservative method, the target organ-specific hazard index (TOSHI). The TOSHI is the sum of hazard quotients for pollutants that affect the same organ or organ system. This assessment calculated TOSHIs for the respiratory system,

blood and blood-forming tissues, central nervous system, liver and kidney, cardiovascular system, and immune system.

Exposures to children and adults were estimated separately to respect children's potentially greater susceptibility to some toxic effects. This susceptibility may be due to physiological differences from adults, different activity patterns that may lead to higher exposures, or both. However, dose-response assessments for non-cancer effects developed by EPA and other agencies do not currently include separate reference concentrations (or similar values) for adults and children. Therefore, adult and child hazard quotients all had the same denominators. Also, the HAPEM4 exposure model takes into account the different activity patterns of children but does not consider their higher mass-specific inhalation rates. Because adults tend to commute to areas of higher concentrations more frequently than children do, HAPEM4 systematically produced higher inhalation exposure estimates, and higher TOSHIs, for adults than for children. For this reason, most of the discussion of individual non-cancer hazards in this section will focus on adults.

To avoid mixing hazard quotients for well-understood pollutants with those from pollutants for which data are sparse, separate TOSHIs were calculated for two groups – pollutants for which the total RfC uncertainty factor ranged from 1 to 100, and those for which the uncertainty factor exceeded 100.

Figure 5-8 shows distributions of individual TOSHIs for the respiratory system for adults. Distributions of individual TOSHIs for six organs or organ systems, for adults and for children are included in Appendix L (Figures 33-38 for adults and Figures 45-50 for children). Of the six TOSHIs computed, only the respiratory TOSHI for low-certainty pollutants exceeded 1.0 for more than 1% of the US population. This result was dominated by a single substance, acrolein. The median of this TOSHI distribution was approximately 4, and its 99th percentile was approximately 20. More than three quarters of the population had a respiratory TOSHI greater than 1.0.

Much of the respiratory TOSHI was contributed by onroad mobile sources, with nonroad mobile and area (and other) sources contributing lesser but still important amounts. Major sources did not add much to the respiratory TOSHI nationally, and none of the pollutants in this TOSHI had an estimated background contribution.

5.3.4 Aggregate TOSHI of Multiple Pollutants to Populations

Figures in Appendix L (Figures 39-44 and 51-56) show numbers of adults and children, respectively, residing in census tracts for which the median exposures to multiple pollutants produced TOSHIs exceeding three fixed levels (0.1, 1.0, and 10) for emissions from all sources combined. Unlike the individual TOSHI figures (which exclude the extreme ends of the distribution), these population figures include the entire assessed population in order to increase sensitivity to potential urban-scale hot spots.

Pollutants can be grouped in categories of decreasing importance in the assessment as follows:

National hazard drivers: Results show that nearly the entire US population resided in census tracts where the median TOSHI for respiratory effects exceeded 1.0, and that about 25 million adults and 4 million children resided in tracts where the respiratory TOSHI exceeded 10 (see Appendix L, Figures 39 and 51). These results are for the low-certainty group of pollutants alone, and were dominated by acrolein.

Regional hazard drivers: TOSHIs for four more organs or organ systems – blood and blood-forming tissues, central nervous system, liver or kidney, and immune system – exceeded 1.0 for more than 100,000 adults and 20,000 children. Results for the immune system were for high-certainty pollutants; results for the other three target organs were for low-certainty pollutants. Of these, the central nervous system TOSHI potentially affected the largest populations. Approximately 600,000 adults and 100,000 children resided in tracts where the CNS TOSHI exceeded 1.0, and 10,000 adults lived in tracts where the CNS TOSHI exceeded 10.

The sixth TOSHI, for the cardiovascular system, did not exceed 1.0 for more than 10,000 people nationwide.

5.4 Discussion of the Risk of Diesel Exhaust

Although EPA is providing concentration exposure information on diesel particulate matter as a surrogate for diesel exhaust, the Agency is unable to provide the same quantitative information in this risk characterization as is provided for the other 32 air toxics. At the national level, EPA believes that diesel exhaust is one of the air toxics that poses the greatest risks to the public based on its potential carcinogenic effects and other health effects related to diesel exhaust, especially since diesel engine emissions provide a substantial contribution to fine particle emissions.

EPA=s Clean Air Scientific Advisory Committee (CASAC) recently approved conclusions that EPA has reached regarding the lung cancer hazard and risk of diesel exhaust [*42*]. In EPA=s draft Health Assessment Document for Diesel Exhaust (HAD), the Agency concluded that diesel exhaust is likely to be carcinogenic to humans at environmental exposure levels that the public faces (classifying it as a “probable” human carcinogen in the scheme used in this NATA report)[*43*]. However, as stated in the "Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Rule" [*44*], EPA has concluded that the available data are not sufficient to develop a confident estimate of cancer unit risk**.** The Agency concluded in developing its perspective on risk in the HAD that there is a reasonable potential that environmental lifetime cancer risks (›environmental risk=) from diesel exhaust may exceed one in a hundred thousand and could be as high as one in a thousand. The environmental risk estimates included in the Agency=s risk perspective are meant only to gauge the possible magnitude of risk to provide a means to understand the potential significance of the lung cancer hazard. The estimates are not to be construed as cancer unit risk estimates and are not suitable for use in analyses which would estimate possible lung cancer cases in the exposed populations.

EPA recognizes that, as in all such risk assessments, there are uncertainties in this assessment of the environmental risk range including limitations in exposure data, uncertainty with respect to the most accurate characterization of the risk increases observed in the epidemiological studies, chemical changes in diesel exhaust over time, and extrapolation of the risk from occupational to ambient environmental exposures. As with any such risk assessment for a carcinogen, despite EPA=s thorough examination of the available epidemiologic evidence and exposure information, at this time EPA can not rule out the possibility that the lower end of the risk range includes zero.[19] However, it is the Agency=s best scientific judgment that the assumptions and other elements of this analysis are reasonable and appropriate for identifying the risk potential based on the scientific information currently available.

Even the lower end of the risk range (presented in the risk perspectives section of the Diesel Exhaust HAD) is above the level that has historically warranted regulatory concern at EPA for air toxics. The Agency believes that areas of the U.S. that have relatively higher annual exposure levels for diesel exhaust, certainly those counties and States with annual exposure average levels above 2 micrograms per cubic meter, should consider the scientific judgments that the Agency has made in the risk perspectives section of the HAD while considering the important limitations in their efforts to compare air toxic risks and set priorities for their programs. At the higher exposure levels found in a number of urban areas in NATA, there is an overlap between what the occupational levels were in the epidemiological studies that EPA considered and environmentally equivalent exposures.

There is substantial evidence that diesel exhaust alone and as part of mixture of fine particles is associated with harmful respiratory and cardiovascular health effects including an association with premature mortality. In addition to the direct emissions of diesel engines of fine particulate, the NOx, SO2, and VOC emissions from these sources are transformed into substantial concentrations of fine particles in the atmosphere (e.g. nitrates and sulfates). The Agency provided an assessment of the seriousness of the health effects associated with human exposure to fine particles in a Criteria Document (CD) in 1996 [*45*]. Recent major reanalysis of two of the most critical studies regarding the health effects of long-term exposure to fine particles examined in that CD confirmed the findings of associations between long-term fine particle exposure and mortality [*46*].

[19]EPA's scientific judgment (which CASAC has supported) is that diesel exhaust is likely to be carcinogenic to humans. Notably, similar scientific judgments about the carcinogenicity of diesel exhaust have been recently made by the National Toxicology Program for the Department of Health and Human Services, NIOSH, WHO, and OEHA of the State of California. In the risk perspective discussed above, EPA recognizes the possibility that the lower end of the environmental risk range includes zero. The risks could be zero because (1) some individuals within the population may have a high tolerance level to exposure from diesel exhaust and therefore are not susceptible to the cancer risks from environmental exposure and (2) although EPA has not seen evidence of this, there could be a threshold of exposure below which there is no cancer risk.

5.5 Uncertainty and Variability Analysis for the NATA National-Scale Assessment

5.5.1 Introduction

EPA's guidelines for risk characterization recommend that estimates of health risk be presented in the context of uncertainties and limitations in the data and methodology. The degrees to which different types of uncertainty and variability need to be quantified, and the amount of uncertainty that is considered acceptable, may vary with the scope and purpose of the assessment. Because the national-scale assessment is generally intended for prioritization, tracking national trends and progress, and setting the research agenda for the air toxics program, EPA can accept a higher degree of uncertainty than if the assessment were intended for supporting regulatory actions or as a final assessment of risk at the local scale. Instead, the national-scale assessment is only a part of the analyses that EPA intends to conduct to guide and inform the Air Toxics Program. It is inevitable that local details in a nationwide study will be more uncertain than in an optimized local study, and that the uncertainty of the nationwide study will be described in more general terms.

Here, we refer to "uncertainty" as imperfect knowledge regarding the values of specific parameters included in the assessment, and "variability" as real differences in the values of specific parameters among places or individuals included in the assessment. Generally, uncertainty can be reduced by gathering better data, whereas variability cannot be reduced but can be characterized through more refined information or model resolution.

EPA had hoped to undertake a quantitative analysis of uncertainty and variability within each component of the national-scale assessment, using a "bottom-up" approach. The intent was to methodically estimate the range of possible values (and use frequency distributions where supported by more complete data) for each parameter used in the risk calculations. These ranges and distributions would then be used as input for separate calculations of the propagation of uncertainty and variability for all variables combined. However, the EPA technical experts who contributed the various components of this assessment could not with confidence place quantitative estimates, or even semi-quantitative order-of-magnitude estimates, on uncertainty and variability for many of the input parameters. It appears unlikely that a complete "bottom-up" approach to characterizing uncertainty will be feasible without significant additional work. Nevertheless, in order to obtain a minimal estimate of how much higher or lower the risks calculated by this assessment are likely to be, we have provided an illustration of a "top-down" approach to estimating some of the uncertainty and variability associated with (1) modeling of ambient concentrations, (2) estimation of personal exposure, and (3) dose-response assessment. This illustration also shows how this uncertainty and variability might propagate into the final estimates of risks.

The illustration has several important limitations that readers should keep in mind. First, it was not possible to fully separate variability from uncertainty with this "top-down"

approach. Therefore, the propagated characterization of uncertainty incorporates both. Second, the quantitative uncertainty estimates did not include all sources of uncertainty, and the combined estimates of uncertainty seem likely to be underestimates. True uncertainty and variability may be greater, but are not likely to be less.

The uncertainty and variability section ends with recommendations to develop a plan for a more rigorous and comprehensive assessment of uncertainty and variability in this and future national-scale assessments the future, based on more refined information for individual components of the assessment.

5.5.2 Source Characterization

5.5.2.1 Data Sources

As described in Section 4.2.1.3, the 1996 NTI is a composite of emissions estimates generated by State and local regulatory agencies and EPA using emission estimation techniques determined by agency, pollutant, and source category. These emissions estimates differed in quality, number of pollutants included, level of detail, and geographic coverage. Furthermore, EPA did not attempt to verify the methods by which emissions were estimated or undertake a full quality assurance and quality control evaluation of the NTI.

As discussed in Section 4.2.1.2.1, EPA compared the information in the NTI to the emissions in the CEP, and the point sources included in the TRI, and NET data sets, which have been used in other dispersion modeling exercises. However, each of these comparison data sets was developed for different purposes and had different facility and pollutant coverage. Also, their data were compiled by methods likely to be surrounded by at least as much uncertainty as the NTI. In general, the NTI compares favorably to these data sets considering its inclusion of all anthropogenic source sectors and the level of emissions detail required for this study (e.g., location coordinates, stack parameters, etc.).

The quality and associated uncertainty in the NTI emissions data varies according to source sector and individual source categories and pollutants. For point sources, including those categorized as “major sources,” emissions estimates and the associated facility/stack details are more certain where more effort has been concentrated on improving estimates (e.g., under certain MACT standards development efforts that included gathering individual facility emissions data or within states that have gathered emissions data for certain facilities for permitting or planning purposes). By their nature, nonpoint emissions (area, other, or mobile), that are compiled in the NTI as county-wide emissions estimates, rather than at known location coordinates, are less certain than point sources. Many of these nonpoint emissions are estimated by using emission factors, that may be out dated, and surrogate information (e.g., industry sales, population. Uncertainty due to such surrogate allocation schemes are particularly pronounced for mobile source categories, especially within the nonroad sector where, in order to simplify computation, the county-level emissions from hundreds of nonroad sources (e.g., recreational marine vessels, lawn mowers, construction equipment, etc.) were consolidated into three source

category groups (2-stroke gas, 4-stroke gas, and diesel engines) prior to spatial allocation.

It is important to note that the 1996 NTI is not static. EPA intends to periodically update it, as resources allow and as more reliable data (e.g., addition of missing information, removal of double-counting, improved emission factors) become available for that year. Thus future assessments based on the 1996 NTI may produce different results. These factors represent important sources of uncertainty in the assessment.

5.5.2.2 Emission Locations

5.5.2.2.1 Point Sources

Locations for many point sources in the NTI were unknown, and had to be placed via default mechanisms (e.g., using zip codes or counties). EPA prepared a State-by-State analysis of proportions of emissions of three metals that were located by default. This analysis suggested that "area and other" sources dominated the total emissions, as expected for these pollutants. Although this comparison did not provide a quantitative assessment of uncertainty, the magnitude of sources and emissions defaulted in a given State suggests that there is uncertainty in the results due to potential location errors. Nationwide, fewer than 10% of point source sites of primary modeled emissions of the 32 pollutants in this assessment were based on default locations.

5.5.2.2.2 County-Level Emissions Sources

All county-level emissions (i.e., mobile sources and many area and other sources) were spatially allocated to the census-tract level prior to ASPEN modeling, using appropriate surrogates such as population or land use using methods described in Appendix 3. In addition to uncertainties introduced by the use of surrogates for allocation, the surrogate information itself was subject to substantial uncertainty due to age or variable quality.

5.5.2.3 Stack Parameter Defaults

A facility in the NTI may have contained many emissions release points, and each release point required several process parameters for dispersion modeling. When these parameters were missing or were out of reasonable range they were replaced by defaults. EPA compiled a table of the percentage of facilities for which defaults were used for eight individual parameters, and found that on average, about one-quarter of the facility data had to be augmented by one or more defaults.

5.5.2.4 Particle Size and Reactivity Assignments

All emissions input for ASPEN modeling had to be categorized according to particle size fraction (coarse versus fine) and reactivity to support the model's accounting for deposition and subsequent chemical reactions. None of the emissions inventories used in this assessment contained this information; all had to be assigned by EMS-HAP. These assignments added to the overall uncertainty.

5.5.2.5 Chemical Speciation Data

The NTI did not include uniform speciation information for HAP groups (e.g., polycyclic organic matter, chromium and compounds) uniformly. In the process of preparing the

NTI data for ASPEN modeling, EPA made important assumptions about chemical speciation within these groups, creating additional uncertainty in the modeling results.

5.5.3 Ambient Concentration Estimation

5.5.3.1 Temporal Resolution of Emissions

The NTI provided the raw emissions data, which were processed and made "model-ready" for ASPEN by EMS-HAP. In addition to the spatial allocation described in section 1.2.1 above, the ASPEN model also required higher temporal resolution. To support this, EMS-HAP allocated annual emissions into days and daily emissions into three-hour periods.

5.5.3.2 Simplifying Assumptions

The ASPEN model does not simulate local terrain effects and the local meteorological conditions associated with terrain effects, which may have caused local-scale inaccuracies in the predicted concentrations. Also, ASPEN does not model transport of any pollutant beyond 50 km from its original emission point. For some air toxics in this assessment (e.g., reactive volatile organic compounds), this assumption was reasonable, while for others (e.g., persistent metal compounds), this assumption represented a significant source of uncertainty. Finally, the ambient concentration estimates included uniform “background” concentrations for 13 pollutants, applied across all geographic areas. This was an important simplifying assumption, because such extrinsic concentrations may vary geographically for many of these pollutants. These three simplifying assumptions create currently unquantifiable but important uncertainties in the ASPEN outputs.

5.5.3.3 Meteorological Characterization Uncertainties

Meteorological data are a critical input for the ASPEN model. EPA’s analysis of the influence of two different sources of input data showed that ambient concentration estimates varied within a range of minus 17% to plus 84%. EPA used the 1996 database, which had a lower mean source-to-meteorological station separation distance that was more representative, for this assessment.

5.5.3.4 Model Formulation and Methodology Uncertainties

5.5.3.4.1 Deposition and Dispersion Algorithms

The ASPEN air quality model employs a Gaussian plume model to characterize transport and dispersion, using algorithms extracted from version 2 of the Industrial Source Complex Long-Term model (ISCLT2). This model has a history of usage and development that dates back to the 1960's, suggesting that we can anticipate its level of uncertainty. As discussed in Section 4.2.2.4, comparisons have suggested that approximately 90% of the model’s estimates are within a factor of two of those observed.

As described in Section 4.2.2.4, EPA verified that the model formulation algorithms were performing as anticipated by using lead emissions to test both the deposition and dispersion algorithms were working properly, and whether the use of a “net” of receptors

might lead to a bias in the modeling results. The results suggested that ASPEN may predict average ambient lead concentrations 20 to 30% lower than a more refined model, because ASPEN uses coarse particle deposition velocities that are higher than typically used in local-scale modeling.

5.5.3.4.2 Atmospheric Transformation Algorithms

Although Gaussian dispersion models can be modified to handle zero-order (linear) production or removal, they typically do not treat nonlinear chemistry effects. To address this limitation EPA developed a mechanism to separately estimate exponential formation and added (or subtracted) the secondary formed concentration to that attributable to primary emissions. Using this mechanism, ASPEN results for formaldehyde and acrolein were 23% and 44% attributable secondary formation. However, results from the more refined OZIPR model suggest that secondary formation for formaldehyde and acrolein would account for 90% and 85% (respectively) of the total. These results should be considered only approximate, because the two models were not directly compared (i.e., using equivalent emissions and meteorology), but they do suggest that ASPEN may be underestimating changes in concentration due to reactivity.

5.5.3.4.3 Interpolation Between Census Tract Centroids

EPA's comparison of ASPEN and ISCLT3 outputs (to determine if ASPEN's interpolation scheme might be underestimating actual impacts) found that ASPEN estimates were lower than ISCLT3's by about 10% in the near distances, with the underestimation increasing to about 25% at 30 km downwind.

5.5.3.4.4 Summary

As discussed above, the use of ASPEN to estimate ambient concentrations added important elements of uncertainty to the national-scale assessment. The simplifying assumptions that made the a national-scale dispersion modeling possible at all – lack of terrain effects, a 50-km radius for effects, and the application of uniform national background concentrations for some pollutants – each have uncertainties that would be difficult to quantify but that may be important.

However, it was possible to make some rough estimates of the performance of the entire modeling system, and of some of its parts. Past studies with air toxics at the local scale have suggested that about 90% of estimated concentrations should be within a factor of 2 of those observed, assuming well-characterized emissions and representative meteorological data. EPA's comparisons of the formulation and methodology used by ASPEN and other dispersion models suggested that differences in dispersion algorithms were minor, likely leading to performance differences of less than 10% in the near field. ASPEN's deposition velocities for coarse particles showed larger differences, and were estimated to bias concentrations roughly 30% low, a bias that affects only pollutants simulated as having coarse particles. Investigations of uncertainties associated with chemical reaction effects suggested that concentration estimates for reactive species should be considered more uncertain than for non-reactive species.

Spot checks on location uncertainties for three metals suggested that 6 to 30% of these emissions were assigned to locations rather than modeled at their true locations. A site visit to one lead smelter found fugitive emissions that appeared greater than those reported in the NTI, suggesting that close inspection of facilities might reveal other under-reported non-point emissions. These emission characterization uncertainties could have a greater impact on the model-to-monitor comparison results (resulting in differences of a factor of 3 or more) than uncertainties seen elsewhere in the modeling algorithms or the meteorological characterizations (which appear to result in differences of 30 to 80%).

5.5.3.5 Illustration of Uncertainty and Variability Associated With Ambient Concentration Estimates

EPA's statistical comparison of ASPEN model estimates with monitored ambient concentrations at the same locations (Section 4.2.2.3) provides an opportunity to estimate the uncertainty in the model's performance for seven substances (benzene, perchloroethylene, formaldehyde, acetaldehyde, cadmium, chromium and lead) representing different source types, deposition characteristics, and atmospheric reactivity. The model-to-monitor comparison showed that modeled estimates for most of the pollutants examined were on average lower than measured concentrations. The degree to which the model underestimated the measured concentration was greater for reactive gases than for stable ones, and greater still for particulates. In addition, the variance of the ratios generally appeared to increase with the degree of underestimation.

If there were sufficient monitored values, and these values represented truth, this information would be a reasonable approximation of total uncertainty contributed by the National Toxics Inventory, all ASPEN model inputs, and dispersion model error. Although there is uncertainty regarding the monitored values, the following illustration shows how a model-to-monitor comparison approach can be used to estimate model system uncertainty. The illustration also provides, for a limited set of pollutants, calculated values based on this approach.

As described in Section 4.2.2.3, the tendency of the model to underestimate measured levels decreased when the comparison was made with the highest modeled concentrations within 10 and 20 km of monitor locations. This result suggested that some part of the underestimation could be attributed to spatial uncertainty of the underlying emission and meteorological data and the tendency of air monitoring networks to select sites in high-impact areas. Nevertheless, there were many locations for which maximum model estimates were still lower than measured concentrations, even at distances up to 50 km. These underpredictions were judged most likely due to underestimated (or missing) emissions data or uncertainty in chemical transformation assumptions.

In this illustration, total uncertainty surrounding the modeled estimates of ambient concentrations was estimated quantitatively, as follows: (1) The model-to-monitor ratios from the original analysis were inverted, to provide an estimate of the factor by which the ASPEN model under-predicted each monitored concentration. (2) The populations of resulting monitor-to-model ratios for seven pollutants were fitted to lognormal

distributions[20] using Crystal Ball 4.0 software. The parameters of the fitted distributions are shown in Table 5-1. Variability could not be estimated because only the 1996 NTI was modeled. Therefore, comparisons to monitored data for other years were not possible.

In addition to expressing the uncertainty of the ASPEN estimates, the fact that the mean monitor-to-model ratio for all seven pollutants exceeded 1.0 was consistent with the reported tendency of ASPEN to under-predict ambient concentrations. Furthermore, both the degree of bias and the total range of uncertainty varied between classes of pollutants. Accordingly, to better support extrapolation of these results to pollutants that were not monitored in 1996, EPA developed composite monitor-to-model frequency distributions for three classes of pollutants: stable gases; reactive gases (i.e., subject to atmospheric transformation), and particulates using standard Monte Carlo simulation techniques[21]. Stable gases were represented by the geometric mean of randomly-selected ratios for benzene and perchloroethylene; reactive gases were represented by the geometric mean of ratios for formaldehyde and acetaldehyde. Particulates were represented by the geometric mean of ratios for lead, cadmium, and chromium. Results are shown in Table 5-2 and Figures 5-9 to 5-11.

Table 5-1. *Illustration*: Parameters for lognormal distributions fitted to monitor-to-model ratios for seven pollutants.

Pollutant	Mean	Standard Deviation
Benzene	1.19	0.51
Perchloroethylene	2.26	1.56
Formaldehyde	2.28	1.60
Acetaldehyde	2.69	2.25
Lead	15.37	37.23
Cadmium	12.53	24.40
Chromium	6.05	9.51

Table 5-2. *Illustration*: Calculated percentiles for monitor:model ratio distribution.

Monitor:Model Ratio for:	2.5%	5%	50%	95%	97.5%
Stable gas	0.69	0.78	1.4	2.6	2.9
Reactive gas	0.76	0.88	2.0	4.3	5.0
Particulate	1.2	1.4	4.9	16	20

A monitor-to-model ratio of 1.0 indicates perfect agreement. For stable gases, 95% of the ratios fell between 0.69 and 2.9. For gases subject to transformation, 95% of the ratios fell between 0.76 and 5.0. For particulates, 95% of the ratios fell between 1.2 and 20. Because these ranges clearly show different amounts of both bias and variance, these three groups of pollutants have been considered separately in the analysis of aggregate uncertainty.

[20] The lognormal distribution was the continuous distribution of best fit for five of the pollutants, and provided second-best fit for the other two.
[21] Simulation was performed with Crystal Ball 4.0 software, using 10,000 iterations with Monte Carlo sampling and an initial random number seed of 0.

This characterization of uncertainties in the emissions inventory and dispersion modeling portions inherently assumed that monitored ambient concentrations were measured without error, and that ASPEN's divergence from monitored levels was the result of either model error or errors in inputs to the model. Of course, ambient concentrations cannot be measured without error, and further errors may have occurred when these data were compiled and reported. Nevertheless, EPA believes that this approach has the potential to provide a fair approximation of the total uncertainty associated with the use of ASPEN for this assessment.

5.5.4 Personal Exposure Assessment

5.5.4.1 Microenvironment Factors

To predict personal exposure, HAPEM4 modeled the relationship between ambient air quality and concentrations in microenvironments occupied by human receptor populations. Because its use on a national-scale precluded detailed mass balance equations, HAPEM4's microenvironment modeling relied on microenvironment factors (simple first-order relationships between outdoor and indoor air quality). However, the outdoor-indoor relationship is not well documented or understood for many pollutants and technical reviewers of this approach agreed there is a great degree of uncertainty associated with it, suggesting that this uncertainty be assessed qualitatively or quantitatively.

5.5.4.1.1 Population Cohorts

The assessment assumed that information from EPA's CHAD database for 40 cohort groups adequately represented the activity patterns of the general population in all areas of the country. Groups were selected to represent variability by age, race, and gender, in order to support comparisons of various demographic groups while still allowing aggregation of exposure across ages. However, there is no way to measure whether these data actually captured the full range of human activity in each tract. For example, it is possible and even probable that members of the same cohort behave differently in different census tracts. It is also possible that selecting different types of cohorts (e.g., economic classes, inside vs. outside workers, etc.) may have encompassed more variability.

5.5.4.2 Activity Pattern Sequence

HAPEM4 constructed annual average activity patterns from multiple 24-hour activity patterns by combining patterns from different individuals, so that day-to-day correlations in activity were not preserved. These aggregated activity patterns were therefore more representative of population averages for that demographic group, rather than individual patterns. For this reason, exposure distributions for the each group represented uncertainty in the population average rather than variability in individual exposures. Uncertainty and variability of input data other than activity pattern were not considered, so that the resulting uncertainty information provided by the prediction distributions is an underestimate of the overall uncertainty.

5.5.4.3 Illustration of Uncertainty and Variability Associated with Exposure Estimates

A comparison of HAPEM4 exposure estimates against personal monitoring data (analogous to the ASPEN model-to-monitor comparison) would be valuable to quantify the aggregate uncertainty of the exposure estimation methods. However, EPA is not aware of any body of personal monitoring data that has not already been used in developing HAPEM4, making any such comparison invalid. Therefore, it is not possible to determine if the HAPEM4 exposure estimates were biased relative to measured exposures. Nonetheless, it is possible as an illustration to compare ratios of ambient concentrations to personal exposures from modeling and monitoring studies for other pollutants to determine if HAPEM4 has captured an appropriate range of variability. For this illustration, EPA utilized data collected for two criteria pollutants, ozone and particulate matter, to develop frequency distributions for the exposure modeling portion of the risk calculation.

Several simple statistics confirmed that HAPEM4 did not capture inter-individual variability in exposure, in that it produced exposure estimates that varied too little among people living in the same tract. For example, in no case (considering all tracts and pollutants) did the 90th percentile exposure exceed the 50th percentile by more than 2%. An analysis of the full range of exposures within several tracts showed that exposures for the least and most exposed individuals differed by less than 20%. Additionally, a tract-by-tract correlation analysis of ASPEN and HAPEM4 results produced correlation coefficients equal to or greater than 0.998. These results contrasted sharply with real-world measurements of personal exposure, which tend to have much greater interpersonal variability.

Databases of matched ambient and personal monitoring data sufficient to support estimating uncertainty and variability in personal exposure among people living in the same area were not available for the pollutants in this assessment. As a surrogate for this illustration, EPA used Spearman correlation coefficients for ozone and particulate matter as examples of “typical” gases and particulates. These data may not be directly applicable because (1) they were developed from monitoring data at a coarser resolution than used in the national-scale assessment, and (2) pollutant characteristics may differ. However, they are used here to illustrate data needs for “top down” estimates of uncertainty and variability for air toxics.

The correlation coefficients (seasonal averages of 0.49 for ozone and 0.13 for PM, from correlation analyses of ambient and personal measurements reported in EPA’s draft exposure assessment for particulate matter were input into a Monte Carlo simulation, which developed a set of estimated personal-to-ambient ratios by sampling two correlated uniform distributions[22].

[22] Populations of personal-to-ambient ratios were calculated using 10,000 iterations with Crystal Ball 4.0, by dividing an personal exposure selected from a uniform distribution between 0.1 and 10 by an ambient concentration linked to the exposure level by the appropriate correlation coefficient.

The resulting distributions of ambient-to-personal ratios shown in Table 5-3 and Figures 5-12 and 5-13. For "typical" gases, 95% of simulated ambient-to-personal ratios fell between 0.09 and 13.0. For particulates 95% of the ratios fell between 0.13 and 7.08. The relatively weak observed correlation between ambient and personal concentrations for ozone and PM results from elements of both uncertainty (e.g., errors in measurement of ambient and personal concentrations) and variability (e.g., varying characteristics of different microenvironments). Although data were too sparse to support separating variability and uncertainty, it appears likely that variability among microenvironments is the dominant factor.

Table 5-3. *Illustration*: Percentiles for uncertainty and variability in the personal: ambient ratio distribution.

Personal:ambient ratio for:	2.5%	5%	50%	95%	97.5%
Gas	0.09	0.14	1.0	7.6	13
Particulate	0.13	0.21	1.0	4.5	7.1

While these distributions are obviously limited in their applicability to the use of HAPEM4 to predict individual exposures, EPA currently lacks the information on correlated ambient and personal data that would be needed to do better. Furthermore, this analysis focuses only on the variations in ambient-to-personal ratios, and assumes that HAPEM4 contributed no bias. EPA intends to seek better data in the hope of extending and improving this part of the uncertainty analysis for future national-scale assessments.

5.5.5 Illustration of Uncertainty and Variability for Dose-Response Assessment

Uncertainty in dose-response assessments is amenable at least to partial quantification. Specifically, assessments that use statistical methods to determine benchmark doses and to fit dose-response relationships to toxicological data often provide confidence intervals for the results. This information is available for many RfCs and UREs. In addition, EPA generically considers RfCs for effects other than cancer as being surrounded by an uncertainty "spanning perhaps one order of magnitude." These uncertainty characterizations clearly incorporate both uncertainty and variability in a manner which does not allow them to be separated. Additionally, it is important to realize that the confidence intervals surrounding RfCs and UREs include only the statistical uncertainty in interpreting the data. Uncertainties inherent in the choice of models to extrapolate from animals to humans and from high to low doses are potentially far larger, and cannot be quantified.

5.5.5.1 Unit Risk Estimates (UREs)

The UREs used in the national-scale assessment are subject to four major areas of variability and uncertainty. First, many of the pollutants were classified as probable carcinogens because data were not sufficient to prove causality in humans. It is possible that some of these pollutants do not cause cancer at environmentally relevant doses, and that true risk associated with these air toxics is zero. Second, all UREs in this study were based on linear extrapolation from high to low doses. It is possible that the true dose-response relationships for some pollutants may be less than linear, resulting in an

overestimate of risk. Third, most UREs in this study were developed from animal data using conservative methods to extrapolate between species. Human responses may differ from the predicted ones. The first three elements are comprised entirely of uncertainty. Fourth, most UREs in this study were based on statistical upper confidence limits, though some were based on statistical best fits. (While this does not affect overall uncertainty, UREs based on best fits should be unbiased, while those based on upper confidence limits should be biased high.) This fourth element represents a combination of variability (i.e., based on variation responses of different people or animals) and uncertainty (i.e., potential errors in the measurement of exposure and response). Because of the aggregate treatment all four sources of variability and uncertainty described above, EPA considers all its UREs to be upper-bound estimates.

Of these four areas of uncertainty and variability, only the variability element of the fourth is amenable to quantitative analysis for this illustration. Some dose-response assessments that determine points-of-departure or fit dose-response curves to data using statistical methods also include enough information to support fitting a frequency distribution to the URE. To illustrate this approach, EPA developed a frequency distribution for benzene (Table 5-4 and Figure 5-14), using the lognormal distribution and confidence interval reported in the recent IRIS reassessment. To be consistent with the use of ratios to describe uncertainty in the exposure assessment, the distribution was assigned a mean of 1.0.

Table 5-4. *Illustration*: Percentiles for variability in the benzene URE.

Ratio of "true" URE to the estimated URE	2.5%	5%	50%	95%	97.5%
Benzene	0.14	0.19	1.0	5.3	7.2

It is important to remember that this illustration is based on a single, well-understood substance. While aggregated uncertainty illustration at the end of this section assumes this distribution is typical of other carcinogens, it is possible that other substances (many of which lack distributional information) may have greater statistical error terms. More importantly, this frequency distribution represents only the statistical error term in the dose-response assessment and does not address the other three important sources of uncertainty in dose-response assessment. Information sufficient to quantify these other uncertainties does not currently exist, and this estimate of uncertainty and variability should be considered a minimum for the dose-response assessment as a whole. The true aggregate of uncertainty and variability is likely to be much greater.

5.5.5.2 Reference Concentrations (RfCs)

EPA and other agencies express uncertainty in reference concentrations for effects other than cancer using a series of uncertainty and modifying factors (UFs and MFs). UFs are assigned for extrapolation (1) between species, (2) to sensitive individuals within a species, (3) from subchronic to chronic exposure duration, (4) to estimate no-effect levels from lowest-effect levels, and (5) to account for incomplete data. MFs may also be assigned to consider other issues not covered by the standard UF categories. The aggregate UF/MF depends on the number of extrapolations required, and is best viewed

as an expression of the possible range within which the RfC could change when more complete data become available.

Quantitative estimates of the uncertainty associated with each of these factors are not available. In general, if sufficient data were available to support a quantitative assessment of these factors for any substance, the factors would not be needed in the first place. For this reason, it is not possible to develop total uncertainty ranges for RfCs by aggregating uncertainties of each UF or MF. However, EPA's definition for the RfC[23] specifically describes it as having "uncertainty spanning perhaps one order of magnitude." Because the variability among test organisms is likely to be only a small part of this order-of-magnitude range, this component can be considered to be dominated by uncertainty. Although the actual uncertainty in the RfC may vary among substances, this order-of-magnitude generic range is probably the most reasonable characterization for the purposes of the national-scale assessment.

Thus, for illustrating the uncertainty and variability surrounding RfCs and similar values, EPA assigned the RfC a uniform distribution with upper and lower bounds of 3.0 and 0.3, respectively, as shown in Figure 5-15.

5.5.6 Illustration of Propagation of Uncertainty and Variability

As discussed above, EPA has developed an illustration of a "top-down" uncertainty assessment that quantitatively estimates a portion of the variability and uncertainty associated with three major components of this assessment – the estimation of ambient concentrations, the estimation of personal exposures associated with the ambient concentrations, and the assessment of dose-response.

The illustration for ambient concentrations inherently includes all the uncertainties in the NTI, the other inputs to ASPEN, and modeling error within ASPEN itself. Although the relative contributions of each element cannot be distinguished, the method used has the potential to fairly portray both bias and uncertainty for the aggregate NTI-ASPEN component of the assessment. Because the annual average concentration for a specific time and location has no variation, variability should not be an important component in this part of the illustration.

The uncertainty and variability analysis for personal exposure characterizes the "expected" variation between ambient and personal exposures noted for ozone and particulates in other studies. In contrast, HAPEM4 produced essentially perfect correlations between ambient levels and personal exposures in the national-scale assessment. Therefore, applying an interpersonal variation term developed elsewhere may better describe the variability that should be present in the HAPEM4 personal exposure estimates, but was not. However, this illustration does not attempt to estimate potential bias in the HAPEM4 personal exposure estimates, which would require personal monitoring data not already used in the development of HAPEM4. As described, this

[23] An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

element of the uncertainty/variability illustration should be dominated by variability, most likely among microenvironments.

The uncertainty/variability analysis for dose-response should be considered a generic illustration that would need to be repeated on a pollutant-specific basis. For carcinogens, the variability surrounding the URE has been fitted to a distribution that should be considered a lower estimate of the URE's plausible variability and uncertainty. However, readers should keep in mind that most carcinogens are far less studied than benzene and may have correspondingly greater uncertainty. Also, the analysis for benzene includes only the statistical error term; other important sources of uncertainty that may be far larger are outside the scope of the current analysis.

For noncarcinogens the uncertainty/variability analysis is based on the stated order-of-magnitude of uncertainty in EPA's definition for the RfC (which should be dominated by uncertainty rather than variability). All values within a 10-fold range were considered equally likely. While obviously represents an oversimplification of the uncertainty associated with the RfC, it nonetheless provides a quantitative illustration of the stated uncertainties associated with the RfC derivation process. Better characterization of the distribution of possible RfC values would require significant additional research.

The last step in the "top-down" illustration of a quantitative variability/uncertainty assessment approach was the aggregation of uncertainty and variability associated with ASPEN, HAPEM4, and the dose-response assessment process were combined by Monte Carlo simulation[24], as follows:

$$(Mon2Mod)(Pers2Amb)(DR) = RR$$

Where:

Mon2Mod = ratio of monitored to modeled concentration
Pers2Amb = ratio of ambient concentration to personal exposure concentration
DR = of "true" RfC or URE to deterministic estimate
RR = ratio of "true" risk to deterministic estimate

The calculation was repeated 10,000 times, with each of the three input distributions sampled randomly and without correlation to the others. The risk ratio was estimated separately for carcinogens and noncarcinogens, and for stable gases, transformed gases, and particulates, for a total of six aggregate uncertainty estimates (Table 5-5 and Figures 5-16 to 5-21 below). As discussed, uncertainty and variability could not be separated for this demonstration exercise because they were inextricably mixed in some elements.

[24] Simulations were conducted with Crystal Ball 4.0 software, using 10,000 iterations, Monte Carlo sampling and an initial seed value of 0.

Because the propagation of variability and uncertainty did not capture important uncertainties in the dose-response assessment process, and did not include potential bias in the personal exposure or dose-response elements, these percentiles should be considered a low estimate of the total plausible uncertainty and variability surrounding the risk characterization.

Table 5-5. *Illustration*: Combined uncertainty and variability, in terms of the risk ratio (i.e., the ratio of "true" risk to estimated risk).

Risk Ratio for:	2.5%	5%	50%	95%	97.5%
Cancer: stable gas	0.06	0.11	1.4	20	36
Cancer: reactive gas	0.08	0.14	2.0	29	51
Cancer: particulate	0.23	0.41	4.7	61	100
Noncancer: stable gas	0.13	0.22	2.1	19	33
Noncancer: reactive gas	0.16	0.27	2.9	29	48
Noncancer: particulate	0.48	0.76	7.0	57	92

For stable gases, 95% of likely cancer risk and noncancer hazard quotient values lay within a range from approximately one order of magnitude below and 1.5 orders of magnitude above the deterministic risk estimate. For reactive gases, 95% of likely values fell between approximately one order of magnitude below and 1.7 orders of magnitude above the deterministic value. For particulates, 95% of likely values fell between approximately half an order of magnitude below and 2 orders of magnitude above the point values. In general, stable gases had the smallest plausible risk range, followed by reactive gases, and then by particulates.

This illustration of an approach to estimating combined variability and uncertainty from the individual components of the assessment is a relatively crude estimate that is nevertheless somewhat useful for putting the risk results for individual census tracts into perspective. For example, a typical census tract median risk estimate for benzene was about 10 in 1 million. Since benzene is a stable gas, one can use the table to provide a minimal 95% confidence interval for benzene-related risks in that tract extending from 0.6 to 360 in 1 million. Similarly, a typical census tract median cancer risk for chromium was about 2 in a million. The illustration provides a 90% confidence interval on this tract estimate ranging from 0.8 in a million to about 120 in a million. While these confidence interval illustrations are only approximations, EPA believes that they provide an appropriate sense of the imprecision of risk estimates that deterministic risk estimates do not give.

5.5.7 Aggregation of Risk Across Pollutants

Because the above illustration does not characterize the variability and uncertainty associated with all 32 pollutants, it was not logical to finish it with a quantitative propagation of uncertainty associated with aggregating risk across pollutants. However, some insights about this can be gained from procedures for propagation of statistical error. When aggregating risks across multiple pollutants, it is apparent from Table 5-5 that differences among classes of pollutants will lead to differences in aggregate uncertainty/variability. Also, by simple error propagation theory, we can show that if:

$$R_A = \sum_i r_i$$

where R_A is aggregate risk and r_i is the risk associated with pollutant i, the uncertainty associated with the aggregate risk, U_R, can be shown to be:

$$U_R = \left[\sum_i U_{ri}^2 \right]^{1/2}$$

where U_{ri} is the uncertainty of the risk associated with pollutant i, and the uncertainties associated with each pollutant risk are assumed to be independent. If we simplify this equation by dividing each side by R_A, we obtain:

$$\frac{U_R}{R_A} = \left[\sum_i U_{ri}^{\ 2} \Big/ R_A^{\ 2} \right]^{1/2}$$

where the left-hand side of the equation now corresponds to the fractional or relative uncertainty in the aggregate risk. Examination of the right-hand side of the equation shows that (1) pollutants with larger absolute uncertainties in their risks will contribute more to the aggregate uncertainty, and (2) when individual pollutant risk uncertainties are comparable, pollutants that dominate or drive the aggregate risk will also tend to dominate the uncertainty in that result.

The implications of the partial quantification of uncertainties in this case study are multiple. First, the uncertainties were large, limiting the use of the absolute values of the risk results in any decision-making process. Second, the magnitudes of the uncertainties tended to be similar across pollutants, reinforcing the notion that the relative interpretation of risks can be useful in terms of setting priorities for further analysis or data gathering. Third, while the uncertainties associated with the dose-response component of the assessment were large, there appeared to be equally large uncertainties associated with the emissions and dispersion modeling component of the assessment, especially for particulates and reactive gases. Finally, mathematical procedures for propagation of uncertainties associated with the aggregation of risks across pollutants shows that pollutants which dominate or drive aggregate risk levels will also contribute the most to the uncertainties in those aggregate risks.

5.5.8 Recommendations for Further Characterization of Uncertainty

5.5.8.1 Role of Uncertainty Analysis

Although the quantitative estimates of uncertainty described above are the best that can currently be developed, they have important limitations. They are based on available indicators of uncertainty and variability, which resulted in all the sources of uncertainty being aggregated into only three steps and in the use results from one or a few pollutants being applied to many other pollutants. For example, the aggregate uncertainty and

variability in the emissions estimates and the dispersion and transformation modeling are characterized by the monitor-to-model concentration comparisons, which in turn are limited to the relatively few pollutants and locations with monitoring data. Thus, the estimates described above likely do not properly capture all the important sources of uncertainty in the population risk estimates. To the extent that this or future national scale assessments are used in decision making, whether to set priorities among pollutants, to decide on control actions, or to judge the progress of the control program, better understanding of uncertainties will assist in that decision making process. Also, because of the aggregation approach, the uncertainty estimates described above do not indicate how the uncertainty in the risk estimates would be reduced if uncertainty in specific components of the assessment were reduced. That is, they do not tell us much about how to get the best "bang for the buck" in reducing the uncertainty in future assessments. For both reasons, we believe that it should be a goal to improve the characterization of the uncertainties in the national-scale risk assessment process.

EPA plans to repeat the national scale assessment in 2002-2003, for calendar year 1999 emissions and meteorological conditions, and we believe that steps already taken or underway will lead to improved inputs. For example, we expect more states to provide emission inventory data, and both EPA and states will have more experience with such inventories than in this assessment for 1996. Because the 1999 national-scale assessment will give us risk estimates for a second point in time, and incorporate improvements in several important areas, reducing and better understanding the uncertainties the 1999 assessment will be arguably more important than better understanding the uncertainties in this 1996 assessment. Nevertheless, EPA will be pursuing better approaches to characterizing the uncertainties in this 1996 assessment, both to help focus the efforts to improve the 1999 assessment and to test and refine techniques that may ultimately be applied to the 1999 assessment.

5.5.8.2 Technical Issues in Further Characterization of Uncertainty

In a formal bottom-up uncertainty analysis, many individual components or input values are considered to have uncertainty or variability, and that uncertainty or variability is represented by a frequency distribution. Variability is then propagated upwards through the assessment calculations using Monte Carlo methods, similar to the general approach used in the case study. Given the broad geographic, multi-source, and multi-pollutant scope of the national scale assessment, there are very many components or input values that affect the final risk estimates, and hence that contribute to the uncertainty of the risk estimates. Table 5-6 below is a structured list of components. The list provides a framework for considering how a more complete, "bottom-up" uncertainty assessment might be conducted. It should be noted that for most of the components listed there are actually many individual quantitative input values that could be further distinguished and subject to a formal uncertainty analysis. Moreover, many listed components of the national scale assessment have "structural" uncertainties that are not directly addressable by a Monte Carlo approach. Also, the chain of modeling steps involved in producing one "run" from one set of inputs is time consuming. Repeating it hundreds or thousands of time to create a distribution of risk estimates would be a demanding approach, and less demanding alternatives should be considered. It may be sufficient, for example, to

characterize the uncertainty of each main modeling step separately, and then consider how these uncertainties affect the aggregate uncertainty.

Table 5-6. Sources of uncertainty for the national-scale assessment.

Emissions Data
Stationary source emission data sources:
❑ HAP inventories developed by State and local air agencies
❑ Databases related to EPA's MACT program
❑ Toxics Release Inventory data
❑ Emission factors and activity data
Mobile source emission data sources:
❑ On-road sources:
❑ Non-road sources
Preparing Emissions Data for Dispersion Modeling
Use of compound classes to group pollutant species
Assumptions used for other input data
❑ Use of default physical release characteristics (i.e., temporal pattern, spatial pattern, release height, etc.) by SIC of SCC code
❑ Estimation of chemical characteristics (e.g., vapor-particle ratio, secondary formation, reactivity class, particle size class)
❑ Use of surrogate data to allocate county emissions to census tracts
ASPEN Dispersion Modeling
Model error
❑ Limitations of Gaussian models (e.g., 50-km limit)
❑ Use of a single background assumption for selected HAPs
❑ Use of an assumption of flat terrain
❑ Use of meteorological data from nearest airport
HAPEM4 Exposure Modeling
❑ Use of ME factors to extrapolate from census tract to microenvironment concentrations
❑ ME factors assumed independent of geography
❑ Use of CHAD activity data to represent behavior of entire demographic cohorts
❑ Annualized behavior of cohorts assembled from daily diaries
Dose-Response Assessment
Carcinogens
❑ Probable and possible human carcinogens assumed carcinogenic
❑ UREs based on linear extrapolation from high to low doses

❑ Most UREs developed from animal data extrapolated to humans
❑ Most UREs based on the statistical upper confidence limit of dose-response curve; some based on maximum likelihood estimate
❑ Grouping of aggregate cancer risk by weight-of-evidence
Non-Carcinogens
❑ Laboratory animal data to humans;
❑ Average healthy humans to sensitive humans;
❑ Subchronic to chronic exposure duration;
❑ LOAEL to NOAEL
❑ Incomplete database.
Risk Characterization
Cancer
❑ Use of assumption that cancer risks for different pollutants are additive
❑ Degree to which aggregation of risks based on upper-bound UREs may propagate overestimates
Non-Cancer
❑ Assumption that effects to the same organ or organ system are additive
❑ Use of the RfC for the critical effect (i.e., the adverse effect appearing at the lowest dose) to all effects (i.e., that may appear at substantially higher doses)
❑ Grouping of aggregate noncancer hazard by total uncertainty and modifying factor

5.5.8.3 Future Plans

EPA intends to consider over the next year how to formulate an uncertainty analysis that is appropriate to the particular nature of the national scale assessment process, with its very large number of numerical inputs, particular structural approaches, and multiple modeling steps. We will have to consider where to treat critical inputs individually and where and how to use a more aggregated approach. We will also have to estimate the bias, uncertainty, distributional shape, etc, of the inputs that are treated through frequency distributions. This may involve the use of panels of experts in each of the following areas: (1) emission inventories, (2) dispersion modeling, (3) exposure modeling, (4) dose-response assessment, and (5) risk characterization. Additional data collection efforts might need to be initiated to provide needed quantitative information to support the process. Although it may not be possible to develop true frequency distributions to characterize uncertainties surrounding some of the items in Table 5-6 without additional data, these panels might be charged with providing bounding estimates at the least and more, if possible.

6 Summary and Recommendations

6.1 Perspective on the National-Scale Assessment for 1996

The purpose of the national-scale assessment is to gain a better understanding of the air toxics problem. The national-scale assessment was not designed, and is not appropriate specifically, for identifying local- or regional-scale air toxics "hot spots," nor is it appropriate for identifying localized risks or individual risks from air toxics. Further analyses on a national scale, and additional assessments on other scales (e.g., urban air toxics assessments and residual risk assessments) are being performed in order to fully characterize risks, especially disproportionate and cumulative risks. This means that this initial national-scale assessment is not a complete characterization of the exposures and risks associated with air toxics. Therefore, before considering the results, it is important to understand the context of the assessment.

This initial application of the national-scale assessment should be viewed more as a step in building the analytical framework necessary to estimate risks of air toxics on a national scale – not as producing a definitive estimate of these risks. That is, the individual emissions, dispersion/fate, exposure and risk characterization tools have been integrated for the first time and an initial evaluation of the limitations and uncertainty has been completed. In addition, much of the data collection, while representing a significant effort on the part of many contributors, was performed for the first time in this initial national-scale assessment. Also, some of the pre-existing tools were used in their existing form in the interest of time, even though approximations and simplifications could be improved with more effort. Accordingly, EPA is continuing to develop the process and an understanding of the uncertainties and limitations of the national-scale assessment. Nevertheless, this application is complete enough to warrant a scientific peer review that will provide a basis for making improvements to any future national-scale assessment, through improved data collection, revisions to the tools and appropriate research.

Key Limitations of this National-Scale Assessment:

- **It is based on 1996 data**
- **It only includes 33 air toxics**
- **It only addresses inhalation exposures and risks (not ingestion, a significant exposure pathway for some air toxics (e.g., mercury))**
- **It does not capture localized impacts and risks**
- **It focuses on average population risks, rather than individual extremes, for ambient outdoor exposure only**

It is critical to understand that the national-scale assessment does not include other relevant exposure scenarios (or scales). For example, the national-scale assessment does

not estimate exposures and risks at a local-scale. At best, such high exposures are partly reflected in average exposure concentrations calculated in each census tract. The national-scale assessment also does not include risks associated with emissions of air toxics indoors. In addition, the national-scale assessment does not include exposures and risks from ingestion and dermal exposure that may be posed by these pollutants. These non-inhalation risks are an important component of the air toxics program and should not be ignored in an overall characterization of the risks associated with air toxics. This means that this initial national-scale assessment is not a complete characterization of the exposures and risks associated with air toxics. Therefore, it is not appropriate to determine that a particular air toxic is not significant based solely on this analysis.

As a consequence, the results should neither be viewed as final nor considered definitive or in isolation. The assessment does, however represent our best effort to develop a process for risk characterization for air toxics on a national scale. Additionally, the results provide a picture from which to review and evaluate the technical aspects of a national-scale assessment, to gain insight into the areas of greatest uncertainty, to aid in identifying research priorities, and to provide an initial sense of relative priorities among these first 33 air toxics for the air toxics program.

6.2 Summary of Initial Results of National Scale Assessment

In the risk characterization presented in Section 5, EPA grouped pollutants into four categories based on the magnitude of the risk or hazard estimates and the number of people potentially affected. Magnitude of risk was expressed by classifying a substance as a “driver” (i.e., contributing a relatively large share of the total) or an “important contributor” (i.e., contributing a smaller but still important share of the total). The number of people affected was expressed by assigning a substance national scope (i.e., with potential impacts to millions of people) or regional scope (i.e., with potential impacts to tens or hundreds of thousands of people). This categorization scheme produced four groupings: national drivers, regional drivers, important national contributors, and important regional contributors. Twenty-three of the 32 pollutants were placed in one of these groups. One pollutant – polycyclic organic matter – was grouped both with regional drivers and important national contributors.

National drivers included acrolein, benzene, carbon tetrachloride, chromium, and formaldehyde. Regional drivers included acrylonitrile, arsenic, coke oven emissions, ethylene oxide, hydrazine, manganese, and polycyclic organic matter. Important national contributors were acetaldehyde, 1,3-butadiene, ethylene dibromide, ethylene dichloride, perchloroethylene, and polycyclic organic matter. Important regional contributors were cadmium, chloroform, 1,3-dichloropropene, nickel, quinoline, and trichloroethylene.

In addition, as explained in Section 5.4, EPA believes that diesel exhaust is also one of the air toxics that poses the greatest risks to the public based on its potential carcinogenic effects and other health effects related to diesel exhaust, especially since diesel engine emissions provide a substantial contribution to fine particle emissions. For the nine air toxics not found to be important contributors to inhalation risks on a national or regional scale, this result does not necessarily mean these pollutants are not important. It could

indicate that their main impacts may be limited to the local or neighborhood scales at which we expect the national-scale assessment methodology to under-predict individual risks. These pollutants would therefore be better investigated with local-scale data and assessment tools. It may also be that the initial national-scale assessment underestimated ambient concentrations, and therefore exposures and risks, as appears to be the case with many of the metals.

Mobile sources air toxics showed a strong association with national-scale risks, but the remaining mobile source pollutants appeared to have limited potential for national- or regional-scale risks. Major sources, in contrast, showed a strong association with regional risks rather than national risks. Area sources appeared to produce important risks on both the national and regional scales. Background sources were associated exclusively with nationwide risks, as expected. Because background was assumed to be the same in all tracts, exposure to background pollutants varied only with different human activity.

In summary, the results of the national-scale assessment suggest that 23 of the 32 air toxics have the potential to present some noteworthy risk to some group of people in the U.S. It is important to remember the previously stated limitations when interpreting the results of this assessment.

- Non-inhalation exposures such as ingestion and dermal, are not included. A complete picture of risk would include additional pathways for exposure. This is especially important for pollutants that persist in the environment and bioaccumulate, such as mercury, dioxins, and PCBs.

- The highest localized exposures and risks are not captured by the national-scale approach. As mentioned earlier in this report, two comparisons between the results of the 1996 national-scale assessment and results from local-scale refined assessments indicate that this limitation can lead to significant underestimation of risks in the vicinity of individual point sources. These two comparisons showed an under prediction of local-scale risks by a factor of 30 in an urban area and by a factor greater than 100 in a rural setting.

- In a direct comparison with ambient monitoring data, the ASPEN model was found to consistently underpredict annual average concentrations. While the best agreement between the ASPEN model and ambient data was found for benzene (which ASPEN underpredicted by about 10%), concentration estimates of some metal s were found to be underpredicted by more than a factor of 5.

- Information on dioxins is still under review, and therefore, this pollutant has not been included in the risk characterization. Since dioxins are considered to be an important potential health threat, efforts will be made to include them in future assessments.

- Indoor sources of pollution are not included. While these are considered outside the scope of the current study, it is important to recognize that, for certain hazardous air

pollutants, total long-term human exposures can be significantly influenced and sometimes dominated by exposures due to indoor sources.

- Sources, emission estimates, and exposure factors have a high degree of uncertainty.

6.3 ***Recommendations***

As stated in section 2.1, the results of the initial national-scale assessment (in addition to other assessments) are intended to help inform EPA as it continues to develop and implement various aspects of the national air toxics program. The initial national-scale assessment will assist by:

- Identifying air toxics of greatest potential concern, in terms of contribution to population risk;

- Characterizing the relative contributions to air toxics concentrations and population exposures from different types of air toxics emission sources;

- Setting priorities for the collection of additional air toxics data (e.g., emission data, ambient monitoring data, data from personal exposure monitoring) for use in local-scale and multipathway modeling and assessments, and for future research to improve estimates of air toxics concentrations and their potential public health impacts;

- Establishing a baseline for tracking trends over time in modeled ambient concentrations of air toxics; and,

- Establishing a baseline for measuring progress toward meeting goals for inhalation risk reduction from ambient air toxics.

The recommendations have been categorized below according to the assessment goal that they support.

6.3.1 Identifying Air Toxics of Greatest Concern

Given the limitations and uncertainties of this initial assessment, it is not possible, at this time, to identify definitively the air toxics of greatest concern from among these 33. Nevertheless, it is possible to identify air toxics that appear to be important***;*** that is, those air toxics posing estimated risks at or above levels typically addressed by EPA. However, it is not possible to eliminate other air toxics from consideration.

Of the 33 hazardous air pollutants assessed, those that appear to pose the greatest health threats to individuals (from inhalation exposure) in all parts of the U.S. are *chromium, acrolein, benzene, formaldehyde, and carbon tetrachloride*. Pollutants having the greatest potential to pose health threats to individuals in some regions of the US include *acrylonitrile, coke oven emissions, hydrazine, ethylene oxide, manganese, and polycyclic organic matter*. In addition, as explained in Section 5.4, EPA believes that diesel exhaust is also one of the air toxics that poses the greatest risks to the public based on its potential

carcinogenic effects and other health effects related to diesel exhaust, especially since diesel engine emissions provide a substantial contribution to fine particle emissions. Although other pollutants besides these twelve substances may also be important contributors of health risk in some areas, these eleven pollutants account for most of the total estimated national air toxics-related health risk in this assessment.

6.3.2 Prioritizing Efforts to Reduce Emissions

As discussed above, the results of the initial national-scale assessment should be used cautiously when planning efforts to reduce air toxics emissions at the national level. The national-scale assessment results may be used, in conjunction with other information, for planning program activities, assessment activities and emission reduction efforts. For example, the program decisions related to the urban air toxics program and priorities associated with national technology-based standards for area and mobile sources can be informed by results from the initial national-scale assessment. However, these decisions should not be based solely on the results of the initial national-scale assessment. Because other pollutants included in the initial national-scale assessment may have been inadequately characterized or may be important contributors of health risk at a local scale or health risk due to non-inhalation exposure (e.g., ingestion), they should not be excluded from future consideration or assessments based on results from the initial national-scale assessment alone.

Given the limitations and uncertainties, state and local agencies need to be particularly careful in interpreting the initial national-scale assessment results. While these results can be helpful in planning assessments within a state or local area, they should not be used without confirming information to reduce the pollutant scope of an assessment, or to decide on major control steps. The results alone should not be used to dismiss local complaints about air toxics pollutants.

6.3.3 Characterizing Contributions of Sources

Given the limitations and uncertainties of this initial assessment, it is not possible, at this time, to definitively characterize the relative contribution of sources. In particular, the emissions for metals (with their corresponding low ambient and exposure concentrations and risk estimates) may not be adequately characterized by the models in the assessment, particularly at the national scale. Thus, at this time, it is difficult to describe the relative risk contribution from different sources.

Accordingly, characterizing the relative contribution of sources based on the results of the initial national-scale assessment can only be done in general terms. The results appear to show that mobile and area sources of air toxics are responsible for a majority of the health risk concerns that can be identified on a national scale. Nevertheless, certain air toxics from major sources may present significantly greater levels of individual risk to smaller portions of the population than the risks posed to a broader portion of the population from mobile or area sources. Local-scale assessments (such as those being performed as part of EPA's residual risk program and those being anticipated as part of future NATA activities under the urban strategy) will be needed to more accurately characterize the exposures and risks associated with major sources before a complete

characterization of the relative contribution of all sources can be made.

As discussed in Section 5.4, the Agency is unable to provide the same quantitative information for diesel PM in this risk characterization as is provided for the other 32 air toxics. At the national level, EPA believes that diesel exhaust is also one of the air toxics that poses the greatest risks to the public based on its potential carcinogenic effects and other health effects related to diesel exhaust, especially since diesel engine emissions provide a substantial contribution to fine particle emissions. Diesel PM emissions result mainly from mobile sources and in the national-scale assessment EPA focused on estimating the ambient concentrations and exposure from only these sources. EPA spent considerable effort estimating the 1966 emissions inventories from onroad sources (primarily diesel trucks) and nonroad sources (such as construction and farm equipment) for this study. The national-scale assessment results suggest that throughout the country most of the population's exposure to diesel PM emissions results from nonroad sources. There is a relatively wide range of exposures concentrations that occurs in various counties throughout the country with the highest exposures levels occurring as expected in heavily populated urban areas.

6.3.4 Tracking Trends and Progress

EPA plans to update this assessment every three years and the next assessment will occur in 2002-2003. The next assessment will focus on 1999 emissions, concentrations and risks. The assessment of 1999 data will provide results that can be used to assess relative reductions in air toxics emissions, ambient levels, exposures, and risks since 1996 (by comparing with results of this assessment). It will also be important to determine how effective this tool is in accomplishing that objective, to address the limitations and uncertainties in the initial national-scale assessment, as well as the limitations and uncertainties in the second national-scale assessment, to provide an adequate basis for comparison. EPA will attempt to compare on a national-scale the relative impact of reductions of the 33 air toxics since 1996. As previously mentioned, EPA plans to develop a more complete approach for assessing uncertainties and variability of national-scale assessment results over the course of the next year.

However, EPA will also begin to analyze its pilot monitoring results, residual risk assessments, other localized efforts in an attempt to measure progress and the effectiveness of the air toxics program.

EPA can also utilize the national-scale assessment approach in a predictive mode to evaluate potential changes in population-based risks for candidate emission-reduction scenarios. Results from such assessments can be used to inform discussions on future voluntary or regulatory actions to reduce emissions of air toxics and their associated risks.

6.3.5 Setting Data Collection and Research Priorities

The evaluation of the NATA national-scale assessment results is an iterative process. The current evaluation has demonstrated the need for better information that, in turn, will permit an improved evaluation in the future. As a consequence, EPA's Office of

Research and Development has drafted an air toxics research strategy. This draft strategy is expected to undergo peer review by the EPA's Science Advisory Board in the spring of 2001.

The initial national-scale assessment should assist EPA in understanding our risk assessment tools (the National Toxics Inventory, dispersion and exposure modeling, and dose-response information) the proper uses and limitations of each, and how to most effectively improve them. The results of the assessment have shown that the following steps should be taken to improve the quality of the next such assessment:

Improve the quality of emission data. EPA has already requested that State, local, and tribal authorities that submit emission information to the National Toxics Inventory, for the assessment year 1999, make specific improvements in the way they speciate polycyclic organic matter, chromium, and nickel. Other important improvements include (1) entering location coordinates and stack heights for all point source emissions, (2) entering the specific method by which emissions were quantified, and (3) better characterizing the spatial nature of mobile source emissions. In addition, it would be desirable to conduct a study on a small sample of sources, to see if the emissions are accurately located and that their rates are accurately estimated.

Improve support for urban-scale modeling. EPA plans to evaluate the initial national-scale assessment by assessing air toxics on urban and local scales using more refined air quality modeling tools that factor in specific local information such as terrain and local weather patterns. The results of national-, urban-, and local-scale modeling would be compared to provide a more complete context for the characterization of air toxics. State, local, and tribal authorities have been encouraged to revisit and revise inventories in areas of relatively high risk where urban-scale modeling would be valuable. EPA should focus on improving spatial allocation methods for area and mobile sources and reducing the need to rely on these methods by better characterizing the spatial resolution of its emission inventory.

Improve the characterization of background. Background sources of five substances -- benzene, carbon tetrachloride, formaldehyde, ethylene dibromide, and ethylene dichloride – were found to contribute substantially to estimated air toxics-associated health risks. However, the assumption of a ubiquitous background concentration for each of these represents an important simplification that is unlikely to hold true. With the exception of diesel PM (see Appendix F), background was defined as that part of ambient concentrations not caused by modeled sources within 50 kilometers of the receptor population. This definition encompasses natural sources, international transport, intranational transport, and persistent historic emissions, all of which may vary with location. In addition, one component of background – intranational transport – most likely includes sources that EPA has the authority to control and is therefore not properly "background" at all. The uncertainties surrounding the treatment of background should be reduced by (1) establishing a remote monitoring network for these substances, and (2) improving the capabilities of EPA's national dispersion modeling approach to extend beyond the current 50-km range.

Provide support for future model-to-monitor comparisons for ambient air toxics concentrations. Data from existing State and local air monitoring programs have already been compiled to summarize EPA's current knowledge about ambient air toxics, and to serve as a "reality check" on modeling results. As a result of Congressional direction and SAB review of EPA's air toxics monitoring concept paper, an improved, expanded and more representative air toxics monitoring network will be available in the near future to better support model evaluation. To assist with the development of this network and to provide better model evaluation information in the short-term, pilot monitoring studies have been initiated which will have multiple monitors in four urban areas (Seattle, Tampa, Providence, and Detroit) and also provide information in six smaller communities across the country. Information from this assessment will also be used to inform the monitor siting process. Limited information on background concentrations will also be provided. These new data will provide a wider range of information than the current monitoring data set.

Provide support for future model-to-monitor comparisons for exposure. Personal exposure monitoring studies are needed to evaluate the ability of the exposure modeling approach embodied in HAPEM4 to capture the average exposure levels for air toxics in individual census tracts as well as to describe the complete distribution of that can be expected across each census tract. Without such information, exposure models cannot be improved to capture the full range of risks experienced by the exposed population.

Improve dose-response information. Within the scope of this assessment, most of the cancer risk and virtually the entire non-cancer hazard are associated with substances in the "lower-certainty" groups. For example, formaldehyde and carbon tetrachloride are probable (i.e., less certain) carcinogens, and acrolein has a reference concentration that has a total uncertainty factor of more than 100. EPA already has new assessments in progress for these and other substances that are important sources of risk in this assessment, but may want to consider expediting these assessments and subjecting them to review by the EPA's SAB in addition to regular internal and external peer review. Other areas in which dose-response information can be improved include development of (1) organ-specific RfC (to reduce conservatism associated with applying critical-effect RfCs to all target organs when combining non-cancer hazard across pollutants) and (2) UREs based on maximum-likelihood rather than upper-bound estimates (to reduce conservatism associated with combining cancer risks across pollutants).

Extend EPA risk assessment guidelines to be more inclusive of children and other vulnerable subpopulations. EPA's current IRIS assessments for effects other than cancer are severely limited by their use of the average inhaled concentration as the exposure metric. In this way the IRIS assessments fail to support consideration of the different inhalation rates and body weights of children. This prevented the initial national-scale assessment from fully differentiating between adults and children. For the next such assessment, it will be desirable to either adjust estimated inhalation concentrations to children's equivalence, or to develop separate reference concentrations for children.

Improve modeling to include multipathway exposures. One major limitation of this assessment is that it does not fully account for exposure pathways, such as ingestion, which are important for many persistent, bioaccumulative toxics. There are a variety of approaches for accommodating other than inhalation exposures to air pollutants, each with attendant strengths and limitations. In the Mercury Study Report to Congress [40], non-inhalation mercury exposures (from fish ingestion) were estimated using monitoring data (fish tissue), with a recognition that a portion of these exposures result from air emissions. For local scale assessments, EPA has been developing an improved multi-media fate and transport model for air pollutants (TRIM.FaTE) which will be getting some initial applications in 2001-2002. Additionally, development of a more refined ingestion exposure model for air pollutants (ingestion component of TRIM.Expo) is planned for 2002. These tools are expected to contribute to our multipathway exposure modeling capabilities.

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44 Federal Register, 2001. USEPA; 40 CFR Parts 69, 80, and 86; [AMS-FRL-6923-7] RIN 2060-AI69; "Control of Air Pollution from New Motor Vehicles: Heavy-Duty Engine and Vehicle Standards and Highway Diesel Fuel Sulfur Control Requirements"; http://www.epa.gov/fedrgstr/EPA-AIR/2001/January/Day-18/a01a.htm

45 U.S. EPA. 1996. Air Quality Criteria for Particulate Matter. U.S. Environmental Protection Agency, Office of Research and Development, Washington, D.C. 20460. EPA/600/P-95/001a-c.

46 Krewski D, Burnett RT, Goldbert MS, Hoover K. Siemiatycki J, Jerrett M, Abrahamowicz M, and White WH. 2000. Reanalysis of the Harvard Six Cities Study and the American Cancer Society Study of Particulate Air Pollution and Mortality. Special Report to the Health Effects Institute, Cambridge MA, July 2000.

Figures

Figure 1-1. Overview of National Air Toxics Assessment (NATA) Activities.

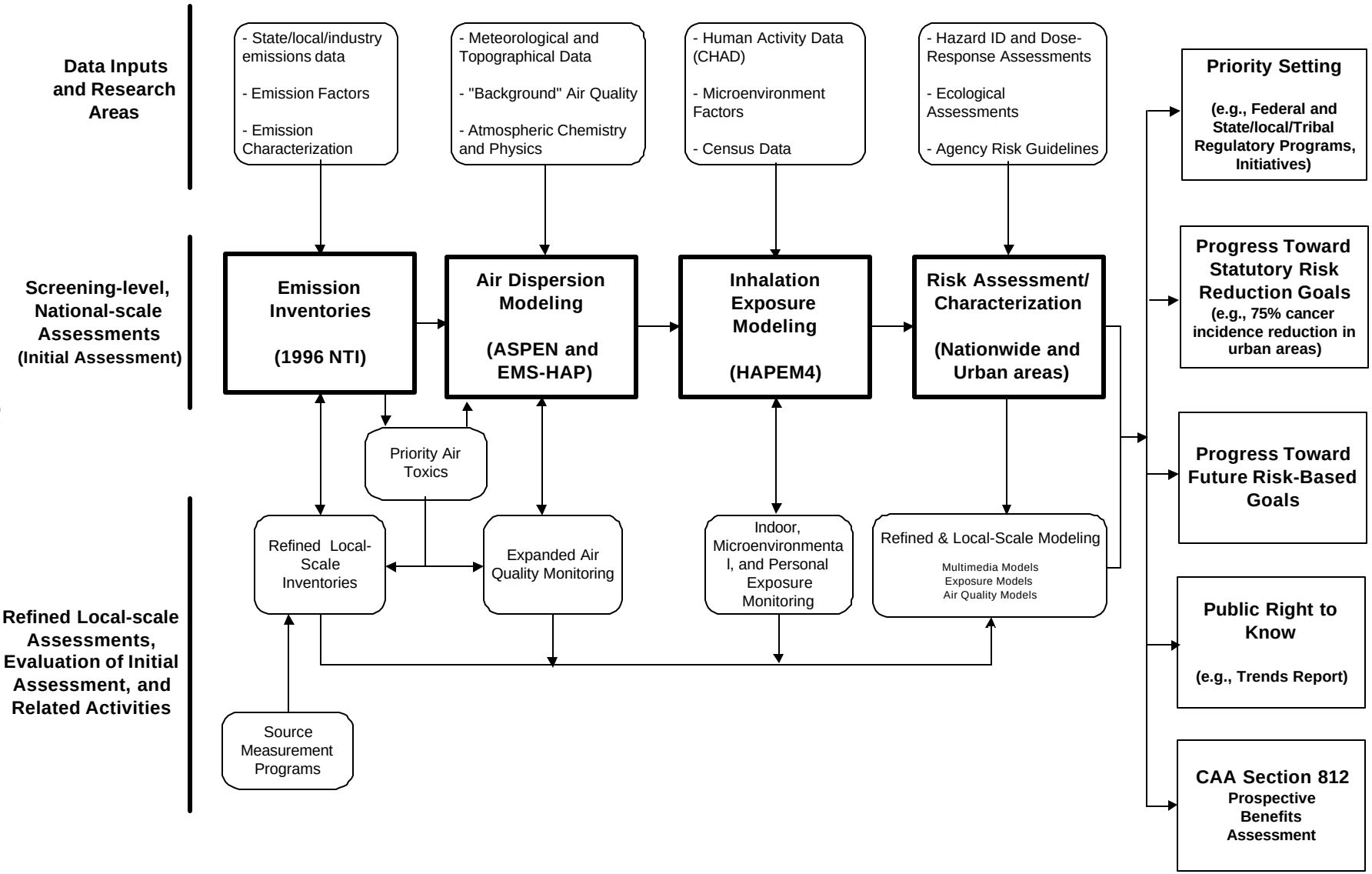


Figure 1-2. Links Between NATA Activities and Other Program Elements.

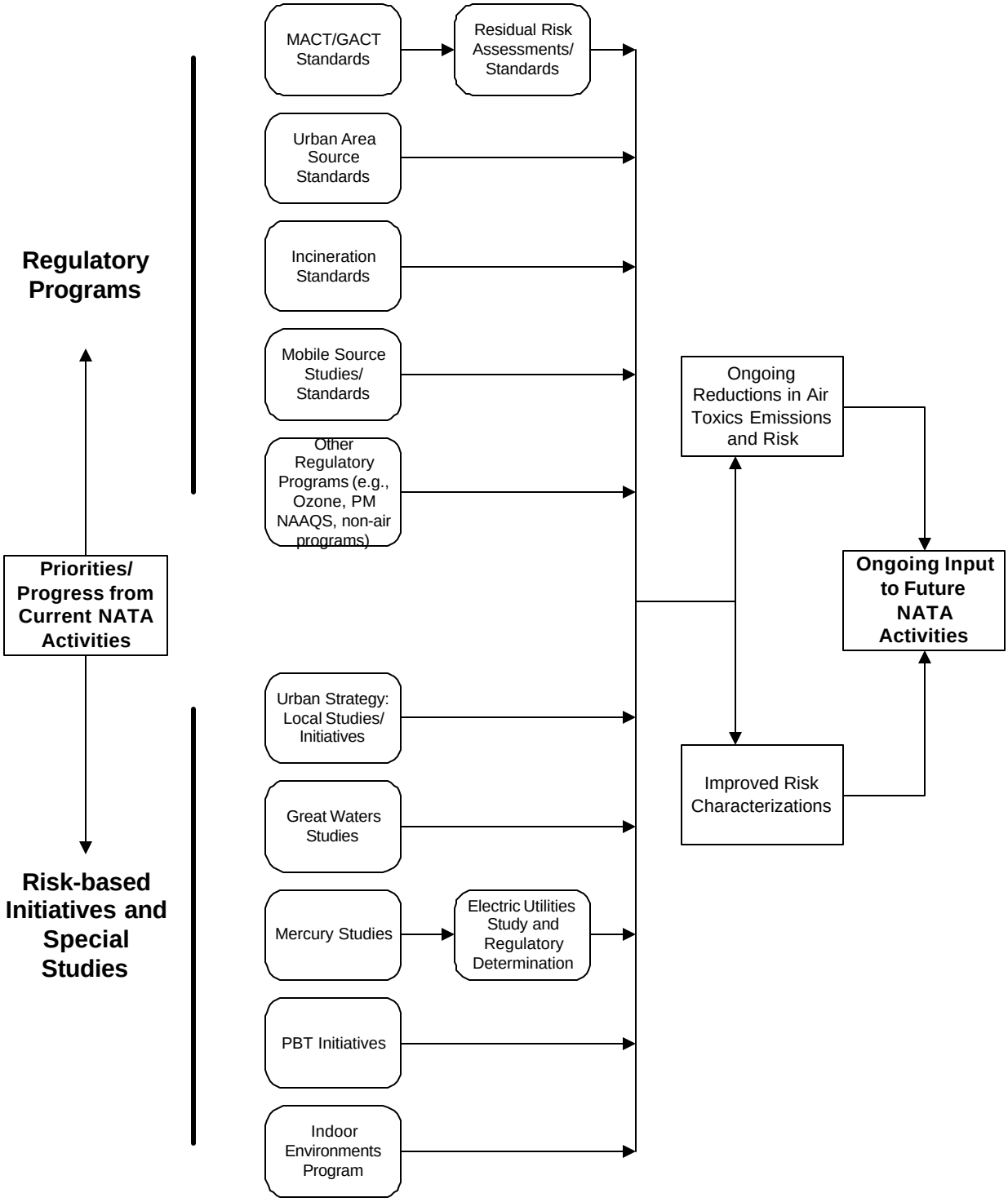
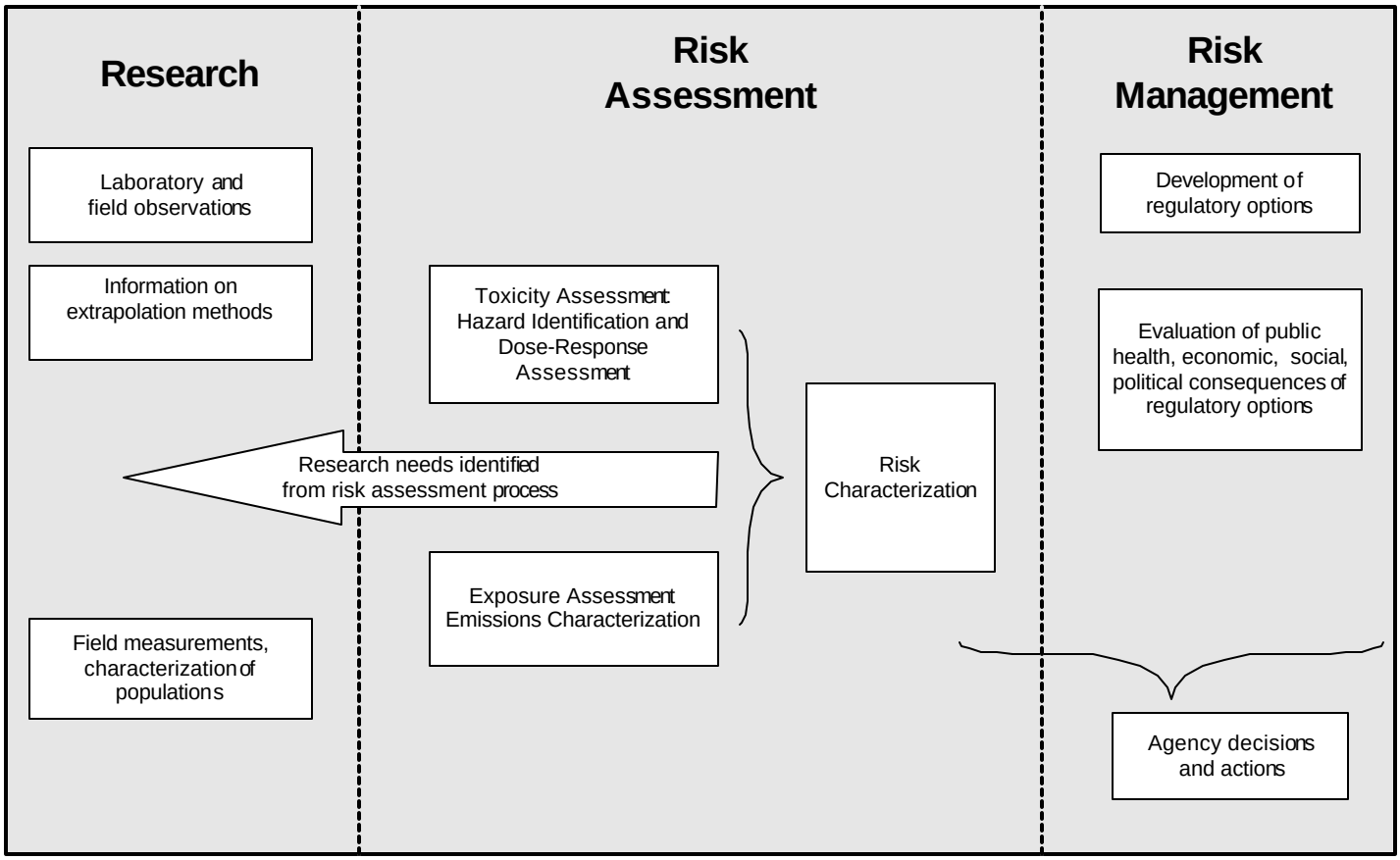


Figure 2-1. NAS Risk Assessment/Risk Management Paradigm



Developed from: NAS, 1983

Figure 2-2. National-Scale Air Toxics Health Assessment: Conceptual Model.

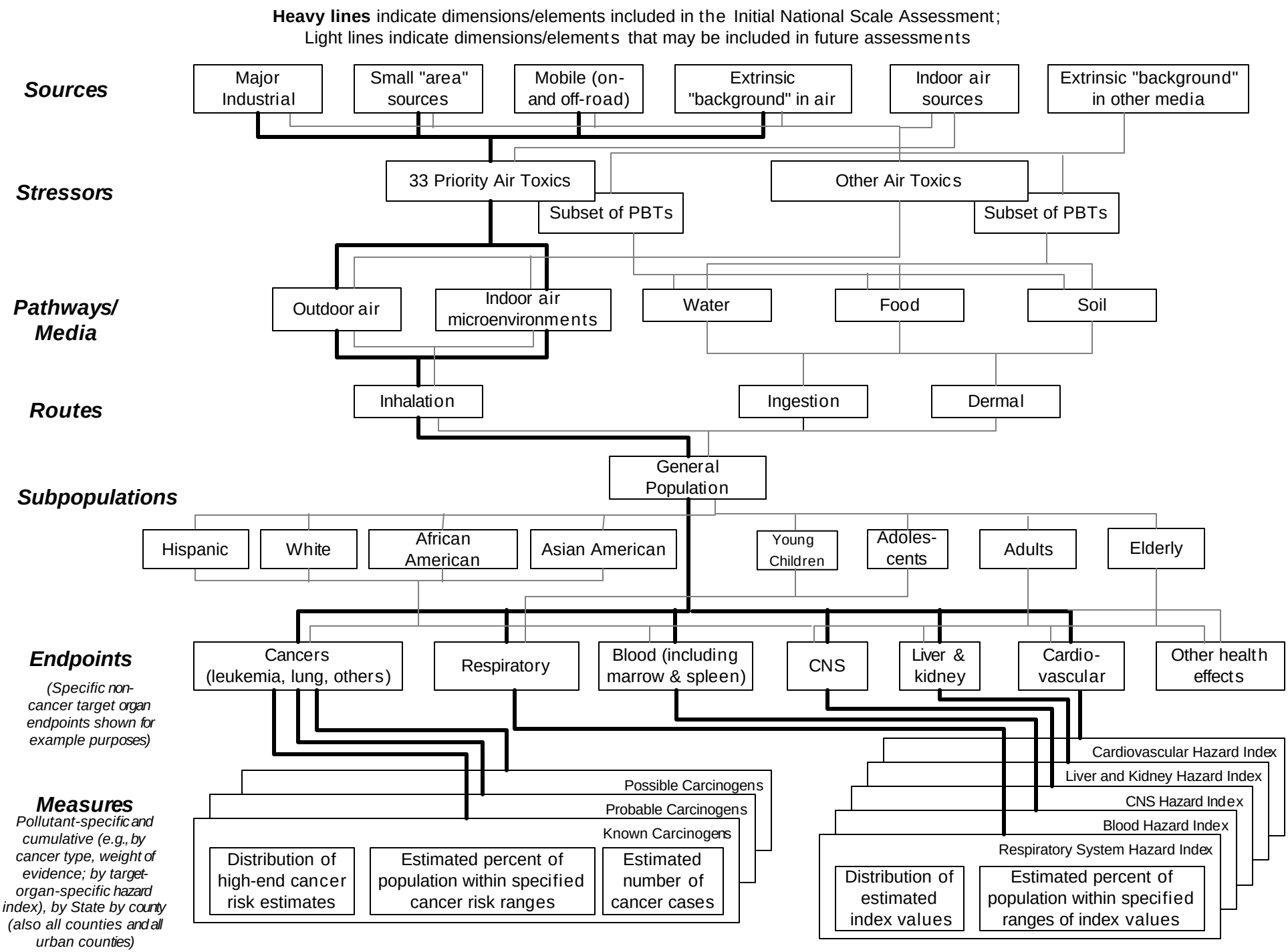


Figure 2-3. Diagram of the HAPEM4 40 Cohort Groups (2x4x5=40).

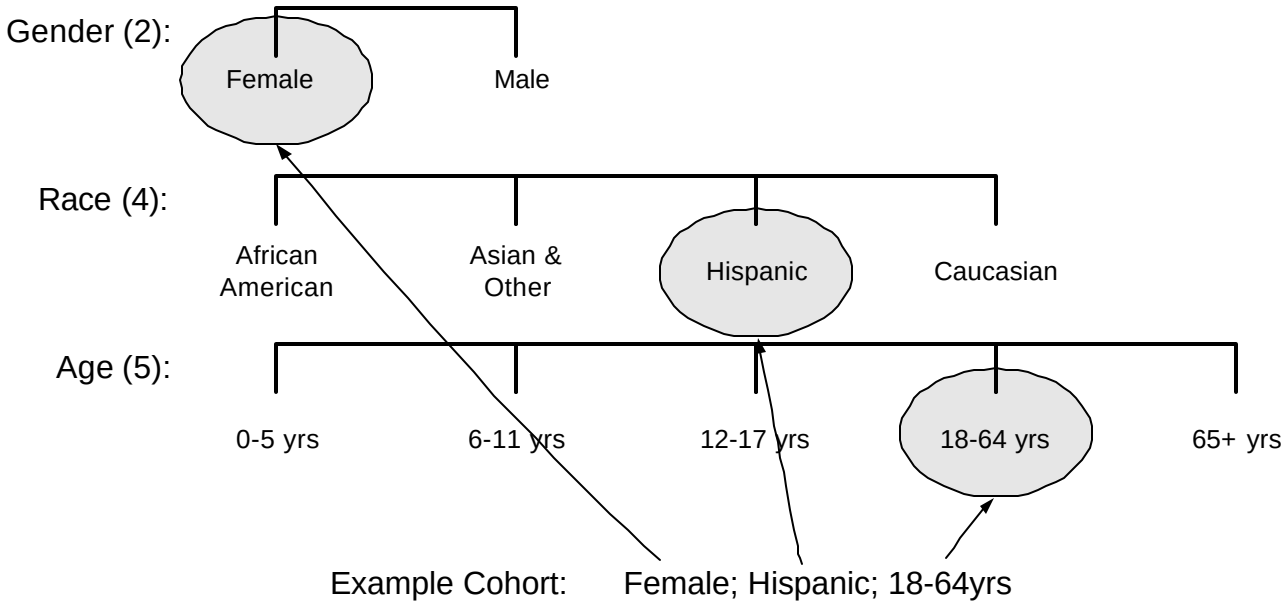
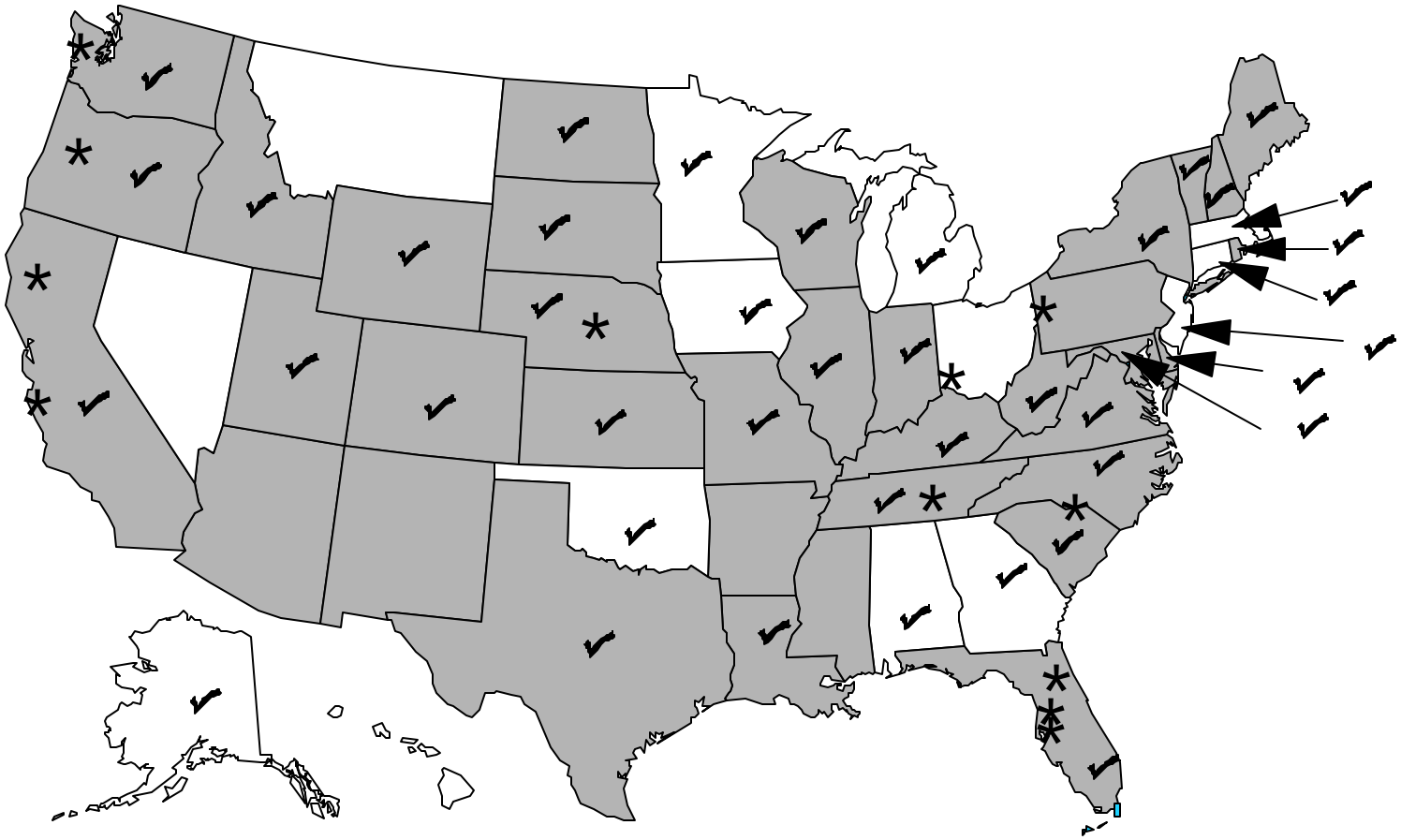


Figure 3-1. 1996 NTI State and Local Agency Data Summary.



Gray - states who submitted air toxics inventory data

✓ - states who submitted revisions

* - local agencies who submitted revisions

Figure 3-2. Example Demographic Groups, Microenvironment, and Activities.

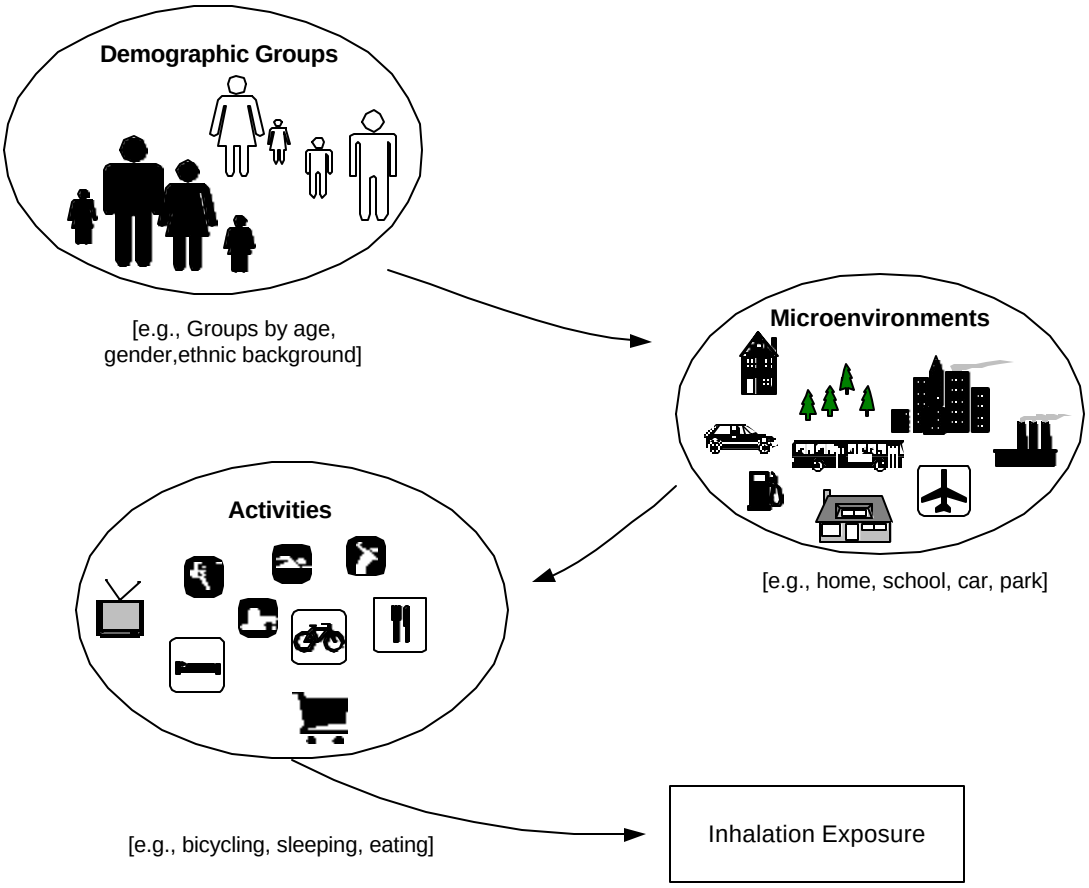


Figure 3-3. Example of a Daily Exposure Scenario for a Cohort.

Cohort: Black Male 18-64
Daytype: Weekday

Daily Exposure Concentration:
$X_{Day}=(X_1+X_2+X_3+X_4+X_5+X_6+X_7)/24hrs)$

	Midnite–3am	3am–6am	6am–9am	9am–Noon	Noon–3pm	3pm–6pm	6pm–9pm	9pm–Midnite
Home Census Tract Concentration X_H	1.5	1.2	1.1	1.2	2.5	2.8	1.7	1.5
Work Census Tract Concentration X_W	1.5	1.1	1.2	1.8	3.8	4.5	2.8	1.8
Activity/Location	Sleeping @ Home	Jog in Park	Drive to Work	Work in Office			Drive tHome	Eat In resturant	Sleeping @ Home

$X_1=(X_{H(0\text{-}3)}* 3hrs+ X_{H(3\text{-}6)}* 1.5hrs) * ME_{Home}$

$X_2=(X_{H(3\text{-}6)}* 1.5hrs+ X_{H(6\text{-}9)}* 0.5hrs) * ME_{Park}$

$X_3=(X_{H(6\text{-}9)}* 1.2hrs) * ME_{Car}$

$X_4=(X_{w(6\text{-}9)}*1.3hrs+ X_{w(9\text{-}12)}* 3hrs)+ X_{w(12\text{-}3)}* 3hrs+ X_{w(3\text{-}6)}* 2hrs)) * ME_{Office})$

$X_5=(X_{w(3\text{-}6)}* 1hr+ X_{w(6\text{-}9)}*1hr) * ME_{Car}$

$X_6=(X_{H(6\text{-}9)}* 2hrs) * ME_{Rest}$

$X_7=(X_{H(9\text{-}12)}* 3hrs) * ME_{Home}$

Figure 4-1. Summary of 1996 NTI Emissions for 33 Air Toxics by Source Sector.

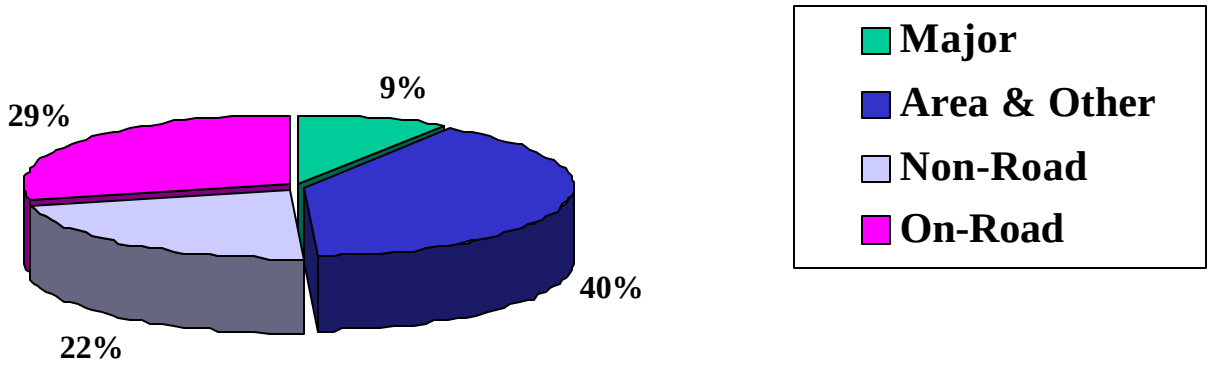


Figure 4-2. Summary of 1996 NTI Emissions of 33 Air Toxics by Urban and Rural Designations.

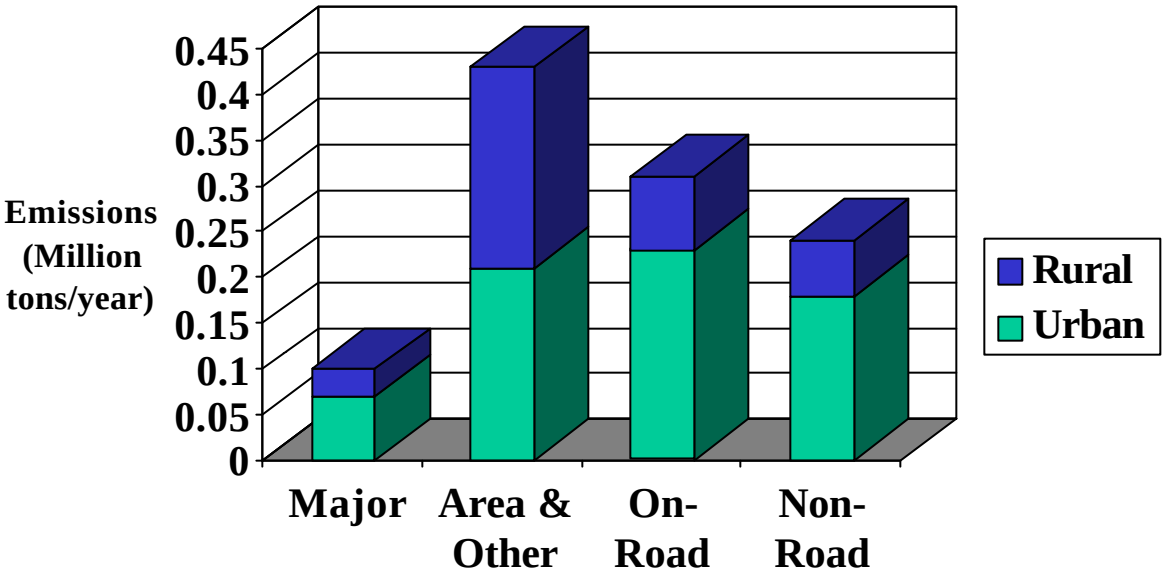


Figure 4-3. 1996 NTI - Aldehydes Emission Densities.

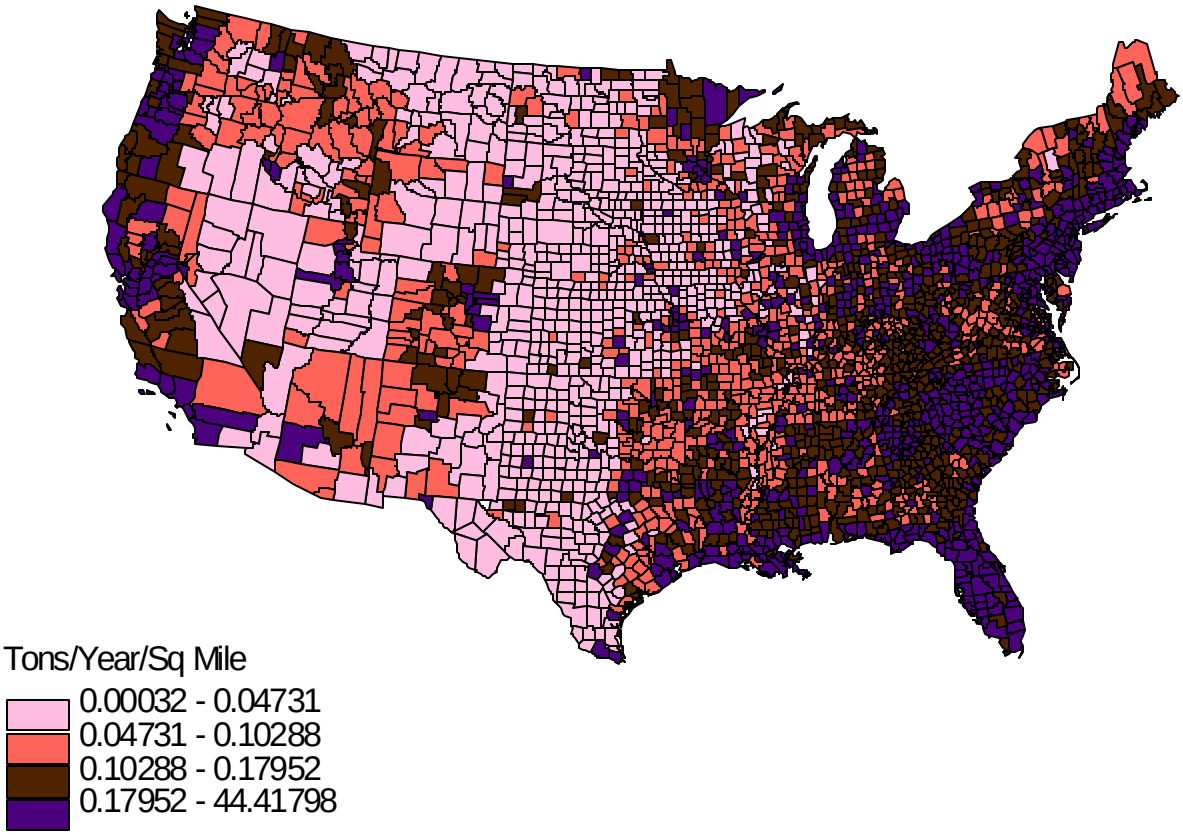


Figure 4-4. 1996 NTI - Metals Emission Densities.

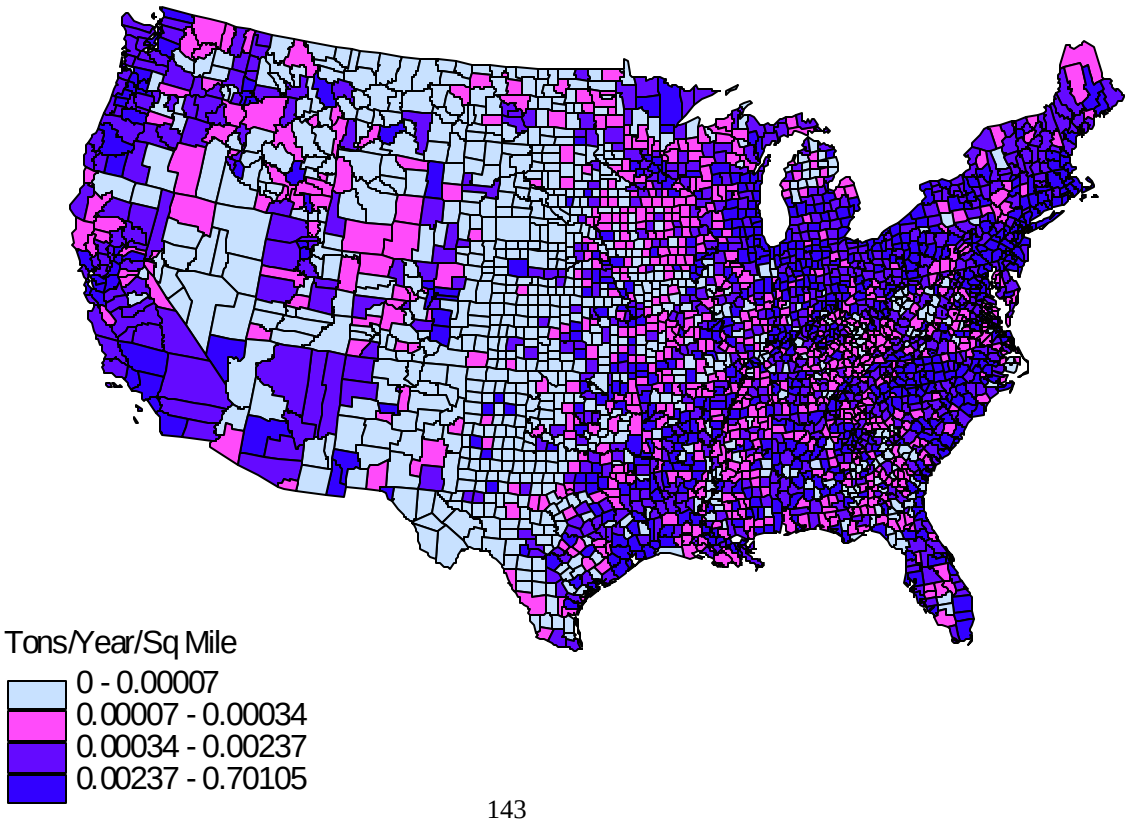


Figure 4-5. 1996 NTI -Halides Emission Densities.

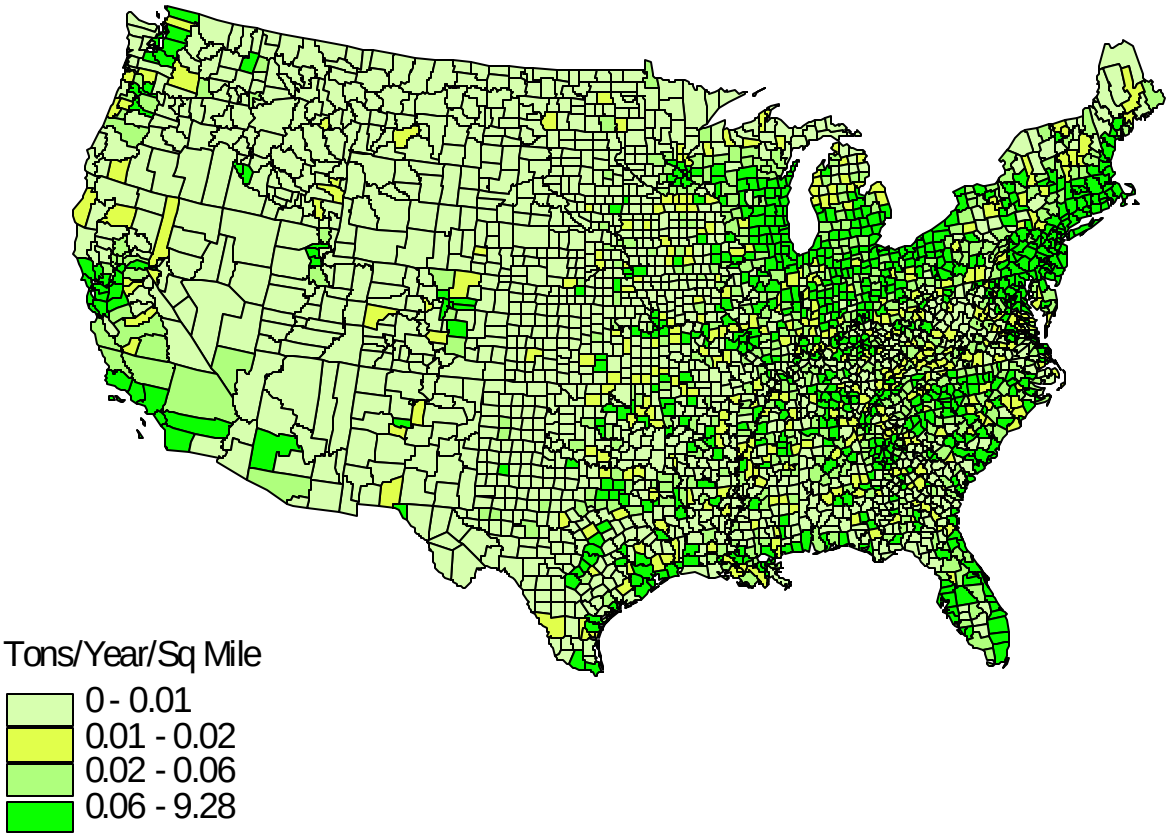


Figure 4-6. 1996 NTI -POM and Hydrocarbons Emission Densities.

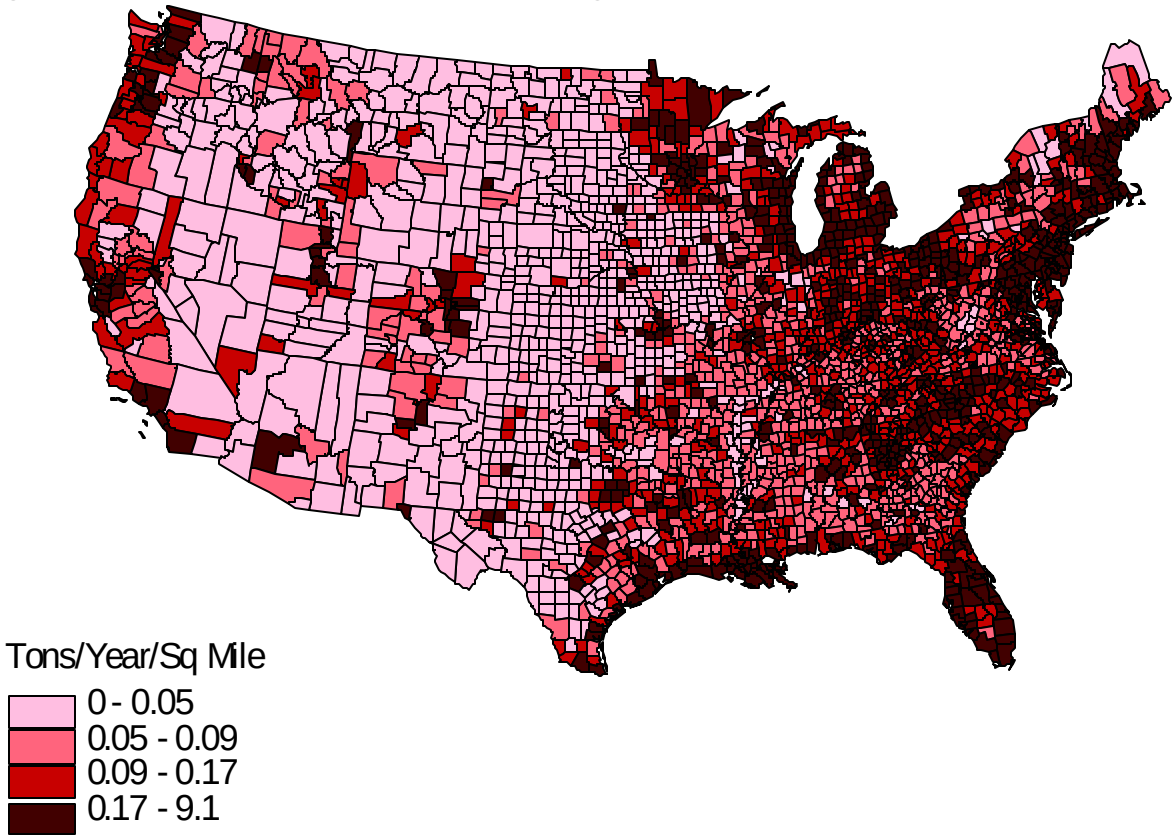


Figure 4-7. Estimated Annual Average Concentrations (ug/m3) for Benzene.

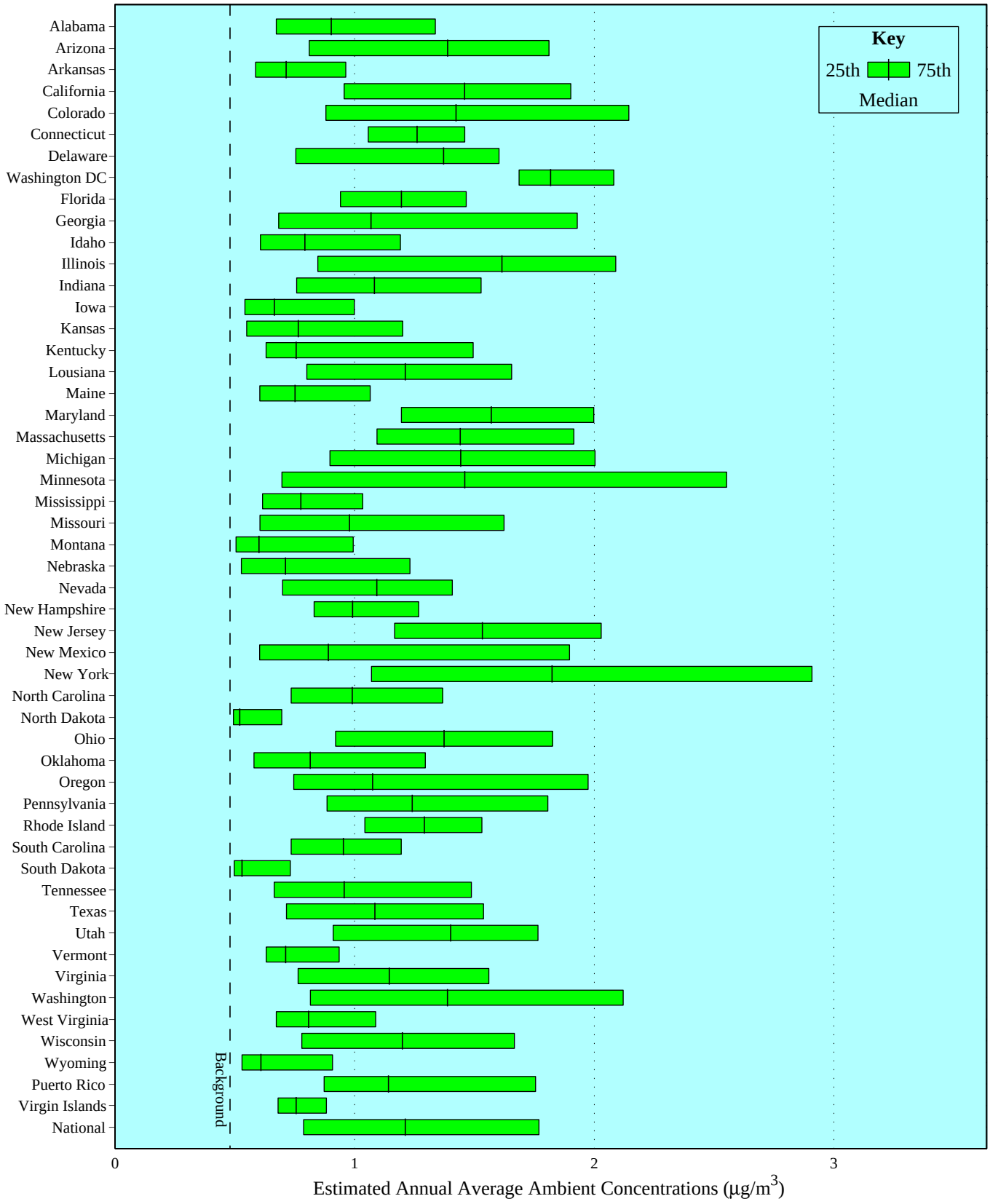


Figure 4-8. Percent Contribution to the Statewide Annual Average Ambient Benzene Concentration Estimates

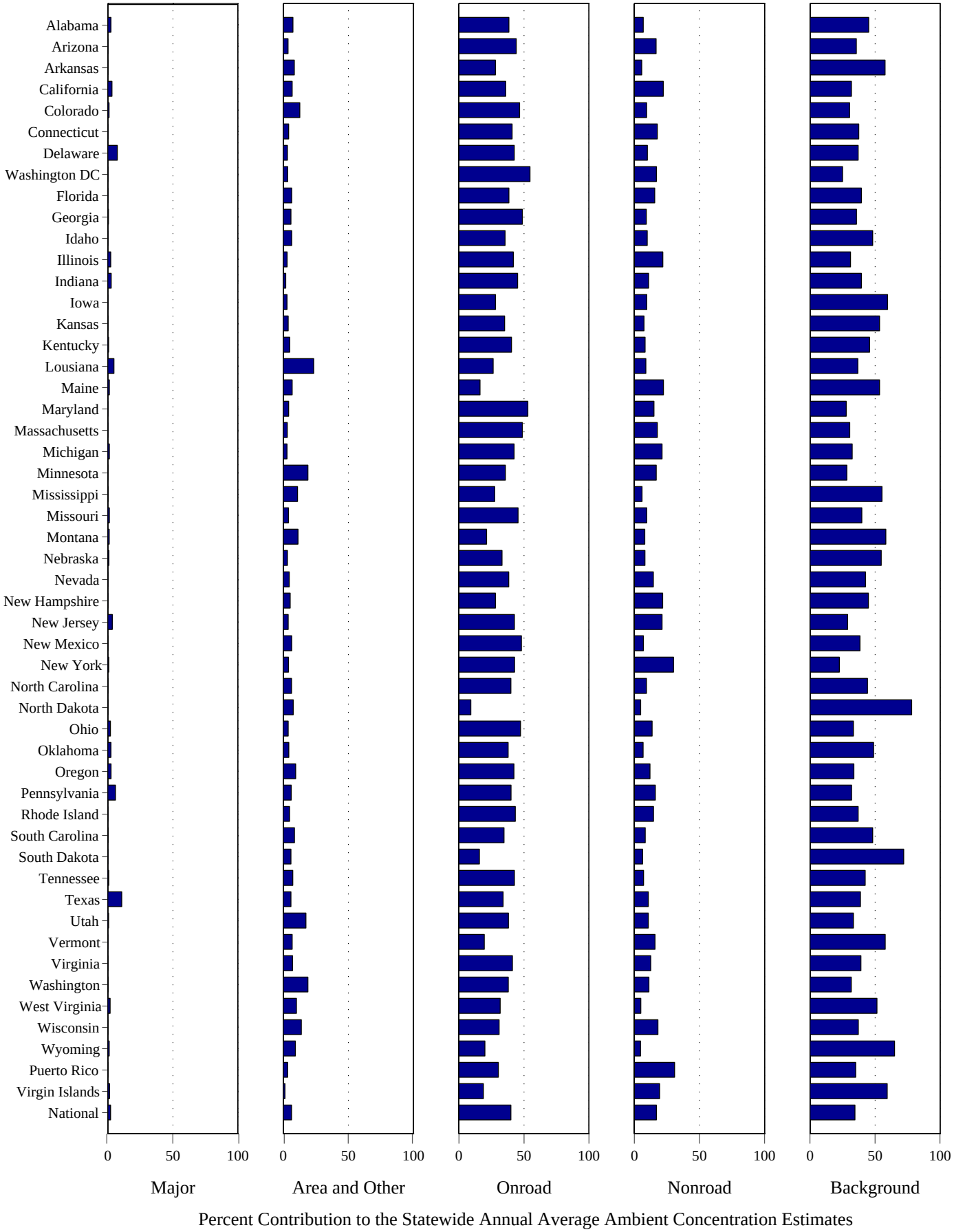


Figure 4-9. Comparison of Annual Average Model Concentrations for 10 Pollutants.

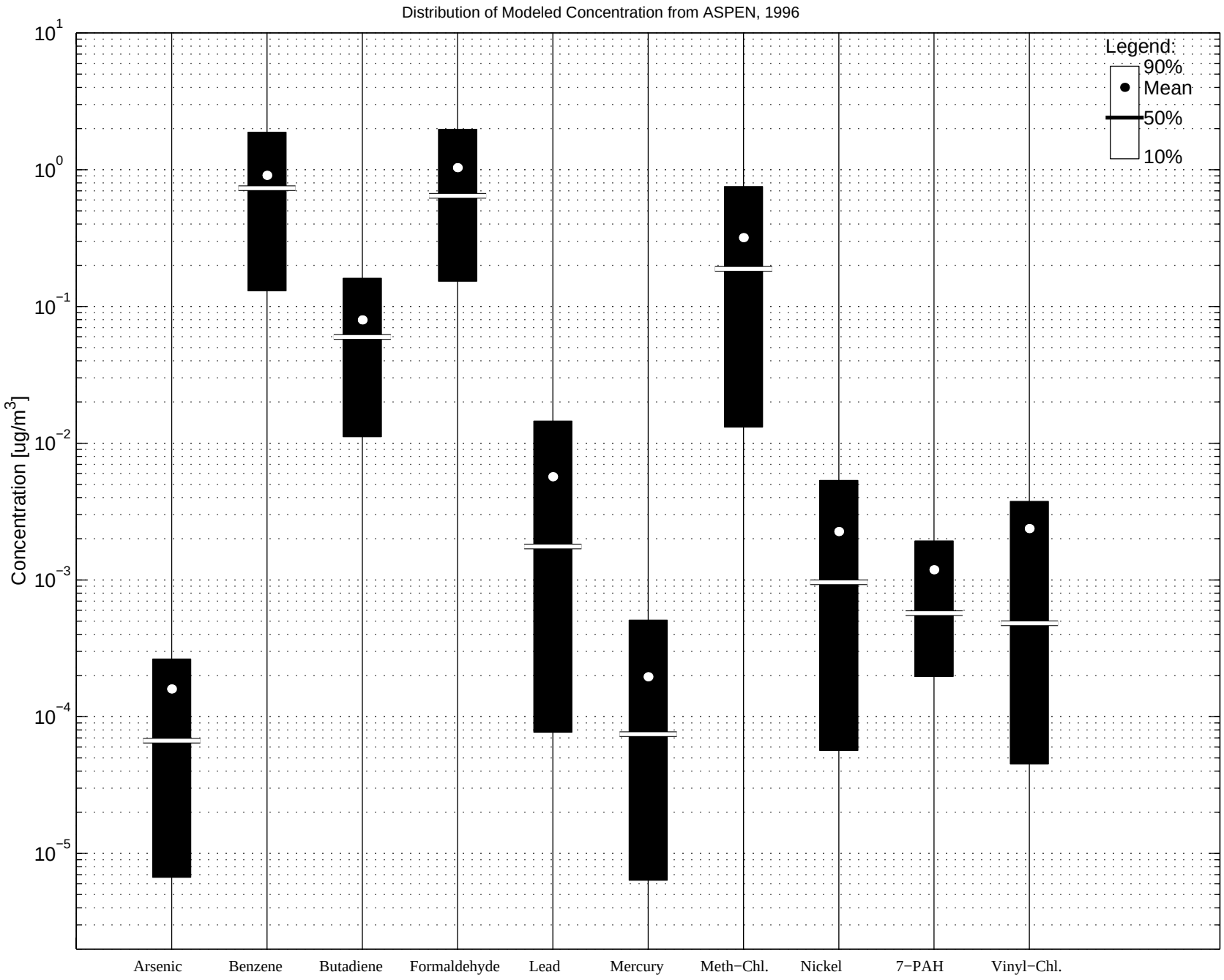


Figure 4-10. Annual Average Concentrations for Urban and Rural Census Tracts.

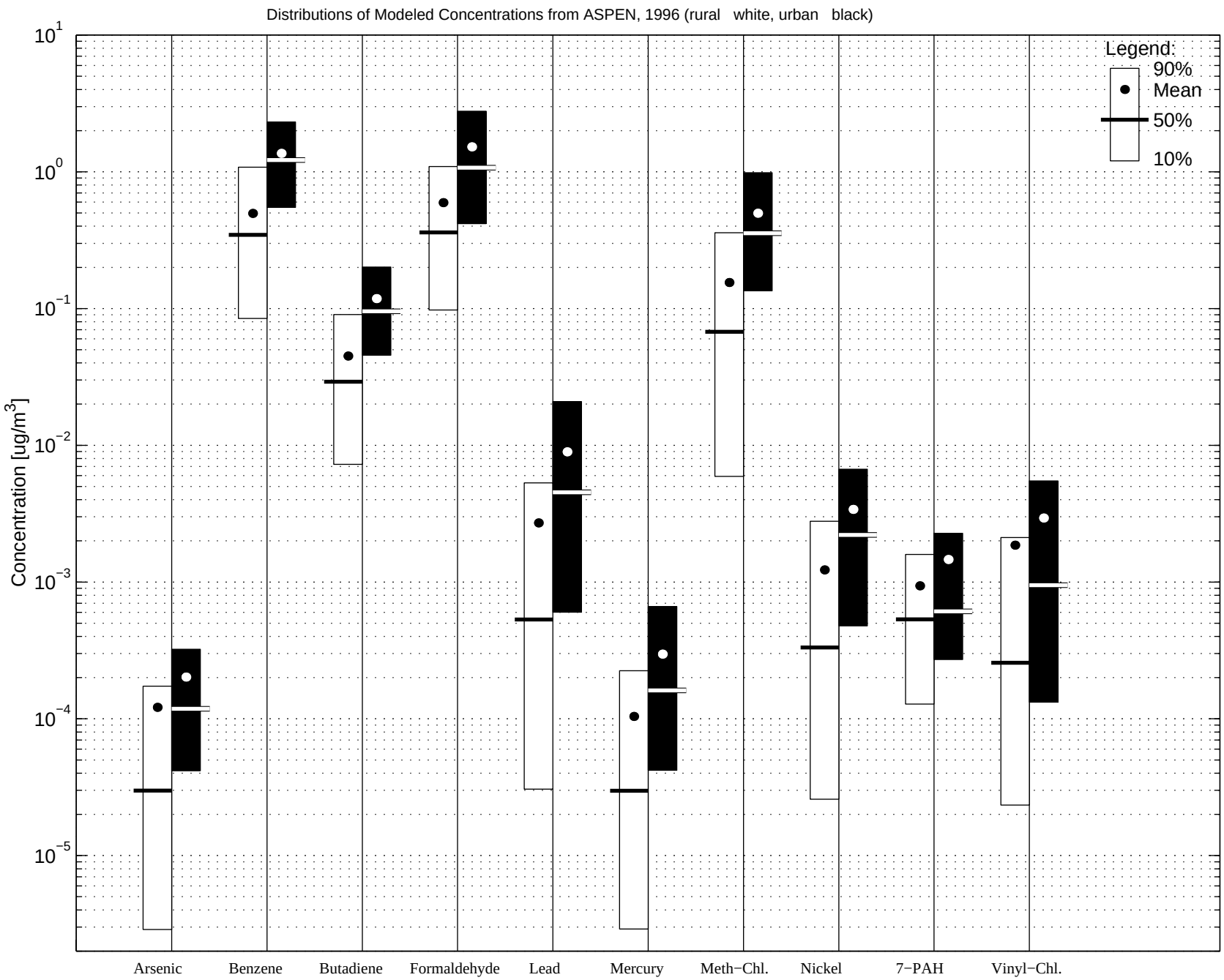


Figure 4-11. Relative contribution of major, area and mobile sources.

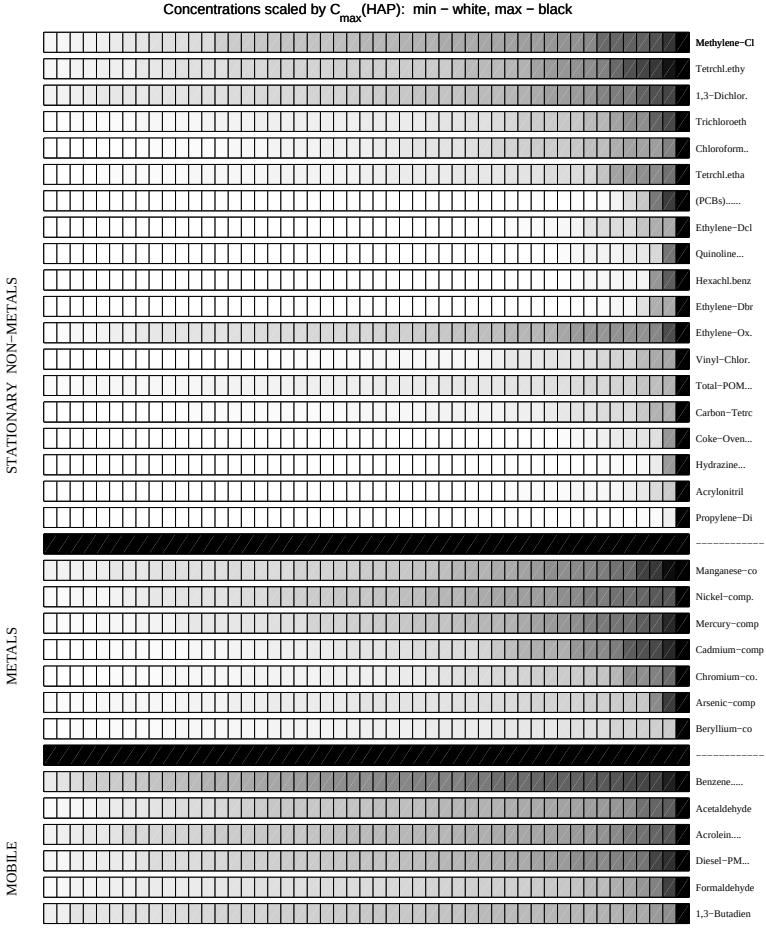
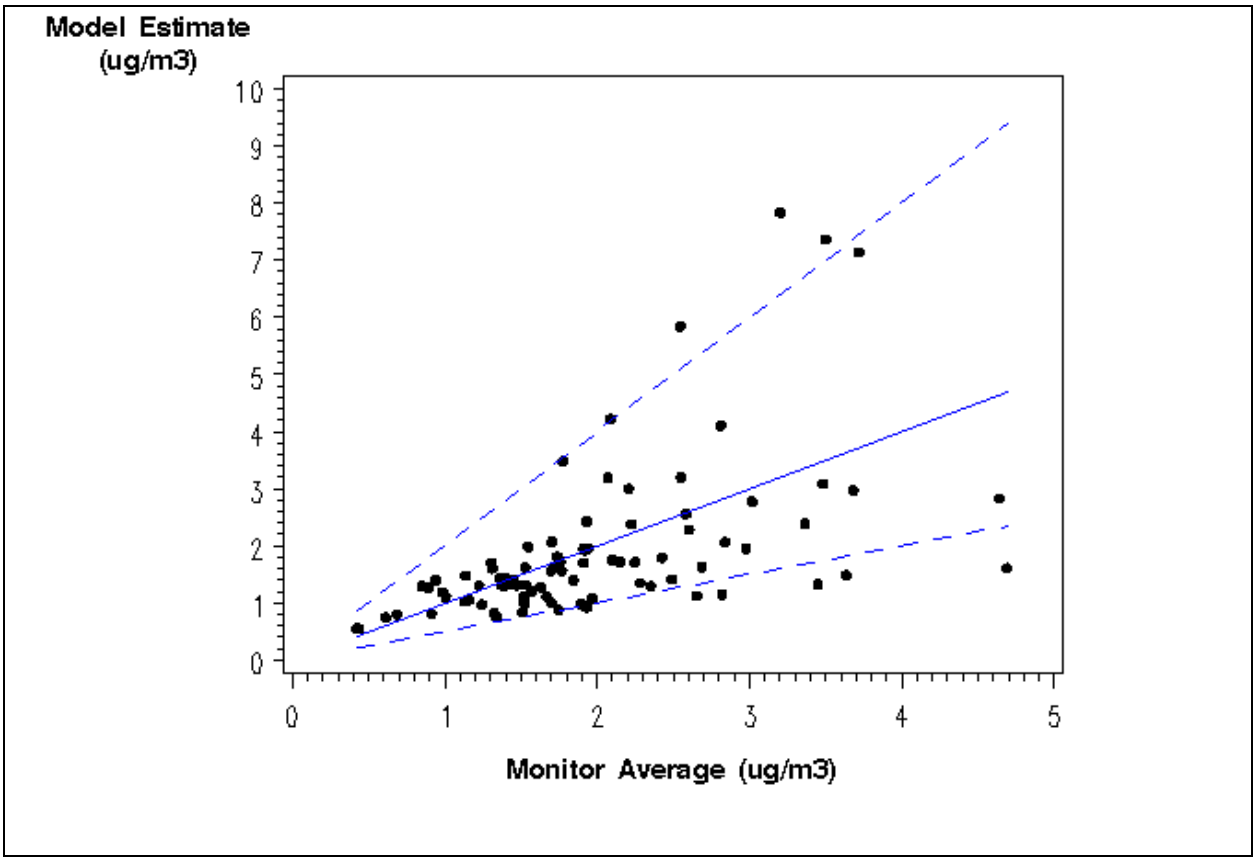
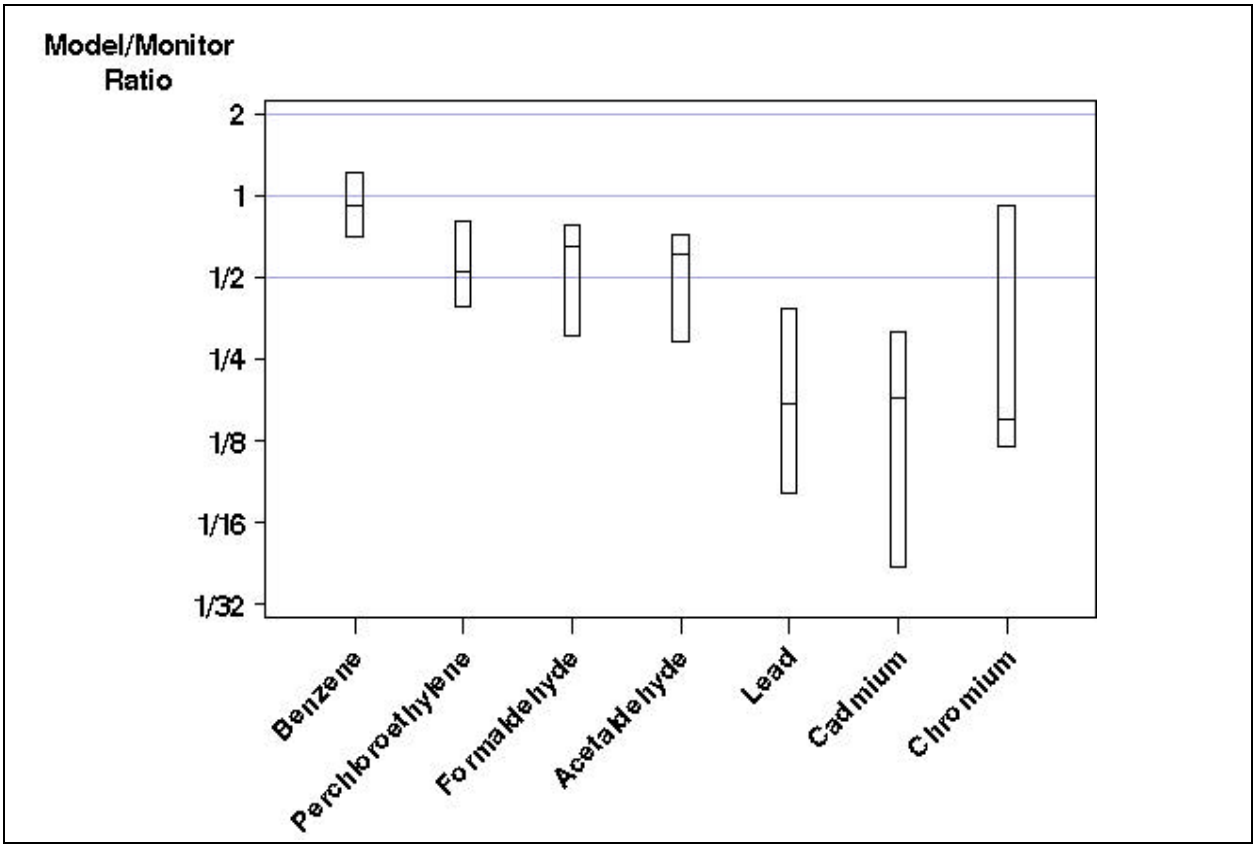


Figure 4-12. Model-to-monitor scatter plot for benzene.



Note: Most points fall within the factor of two wedge, and none are far outside the wedge.

Figure 4-13. Ratio box plot showing distribution of model/monitor ratios for each pollutant.



Note: The bottom of each box is the 25th percentile, the top is the 75th percentile, and the horizontal line in the middle is the median.

Figure 4-14. POM (Total) Exposure Concentration Distribution Among Cohorts in an Urban NY Census

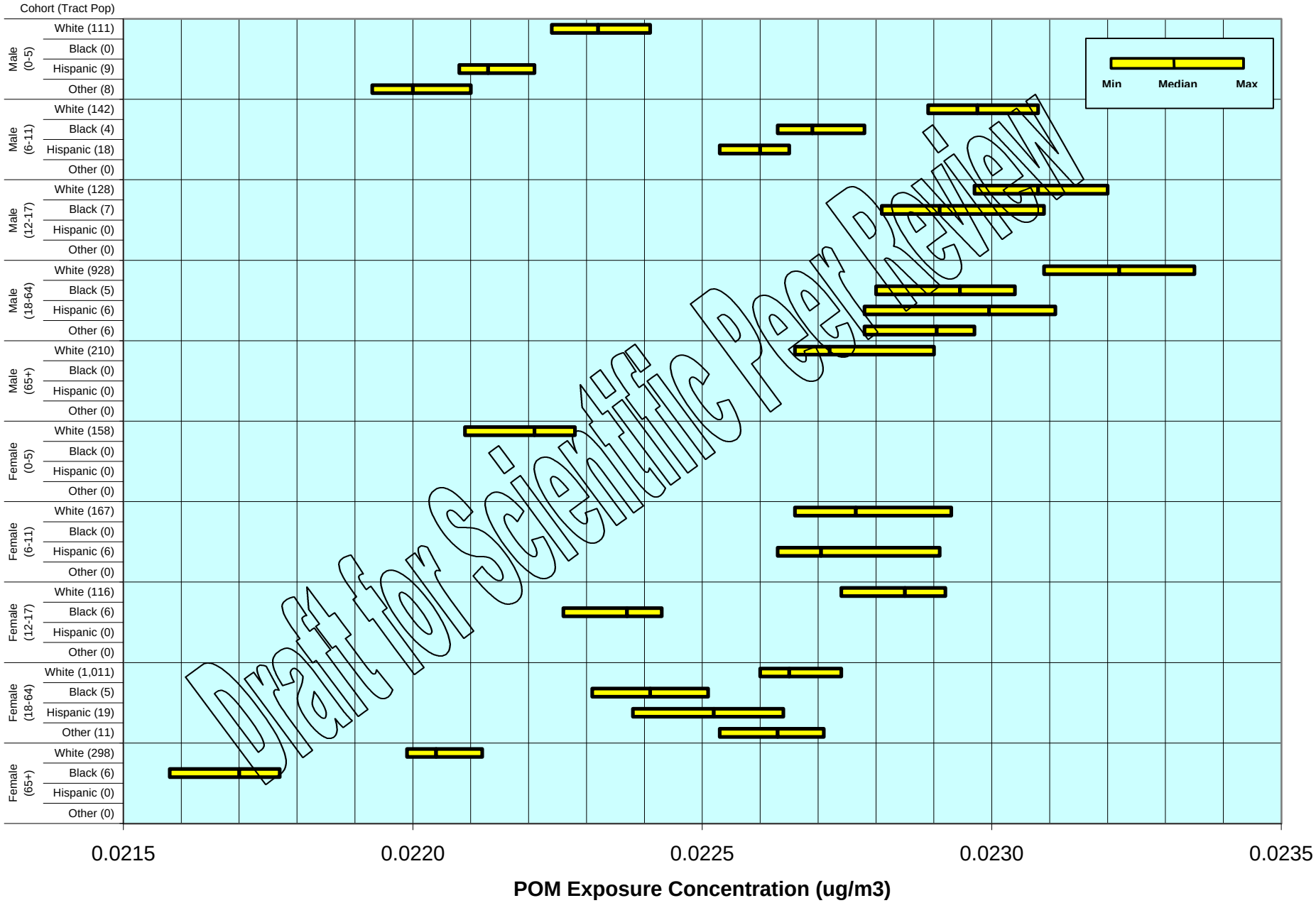


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Figure 4-15. Exposure Results - Summary Table Example (Partial Table)

1996 Modeled Exposure Concentrations for Acetaldehyde (CAS#75070)

EPA strongly cautions that these modeling results should not be used to draw conclusions about local concentrations or risk. The results are most meaningful when viewed at the state or national level; for smaller areas, the modeling becomes less certain. In addition, these results represent conditions in *1996* rather than current conditions.

- The exposure estimates presented below are representative of midrange estimates of population exposures. Due to a number of factors, some individuals may have substantially higher or lower exposures. It is important to note that the model, as applied on the national scale, is not designed to quantify these extreme values of individual exposures.
- Note that for certain chemicals, exposure pathways other than inhalation as well as indoor sources of air toxics may contribute substantially to total exposures of concern. This assessment does not address these other routes of exposure (i.e.,ingestion or dermal) or inhalation exposure resulting from indoor sources.
- The emissions used in this assessment do not reflect potentially significant emission reductions that have taken effect since 1996, including those from: 1) mobile source regulations which are being phased in over time; 2) many of the air toxics regulations EPA has issued for major industrial sources; 3) State or industry initiatives; and 4) any facility closures.
- Simplified modeling assumptions may introduce significant uncertainties into each component of the assessment. See the full discussion of these limitations.
- Because of these uncertainties, EPA will not use the results of this assessment to determine source-specific contributions or to set regulatory requirements. However, EPA expects to use these results to inform decisions about the priorities of the air toxics program as well as to guide the collection of additional data that could lead to regulatory decisions.

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				Estimated Annual Exposure Concentrations (Fg/m^3) for Acetaldehyde (CAS#75070)												
				Percentile Distribution of Exposure Concentrations Across Census Tracts								Contribution to Average from …				
State	County	FIPS	Urban or Rural	5th	10th	25th	Median	Average	75th	90th	95th	Major	Area and Other	Onroad Mobile	Nonroad Mobile	Estimated Background
National	**All**	**99999**	**-**	**3.09E-02**	**6.62E-02**	**1.67E-01**	**4.43E-01**	**6.56E-01**	**8.44E-01**	**1.34E+00**	**1.82E+00**	**5.23E-03**	**5.39E-02**	**3.89E-01**	**2.08E-01**	**0.00E+00**
National	**All Rural Counties**	**99998**	**R**	**1.15E-02**	**2.72E-02**	**5.87E-02**	**1.03E-01**	**1.58E-01**	**1.76E-01**	**2.73E-01**	**3.43E-01**	**6.02E-03**	**3.97E-02**	**8.37E-02**	**2.86E-02**	**0.00E+00**
National	**All Urban Counties**	**99998**	**U**	**6.69E-02**	**1.37E-01**	**3.07E-01**	**5.91E-01**	**7.66E-01**	**9.56E-01**	**1.48E+00**	**2.04E+00**	**5.05E-03**	**5.70E-02**	**4.56E-01**	**2.48E-01**	**0.00E+00**
Alabama	**State Total**	**01000**	**-**	**7.37E-02**	**9.38E-02**	**1.59E-01**	**2.97E-01**	**3.93E-01**	**5.32E-01**	**8.96E-01**	**1.02E+00**	**4.51E-03**	**4.53E-02**	**2.89E-01**	**5.42E-02**	**0.00E+00**
Alabama	State Rural Counties	01000	R	5.98E-02	6.92E-02	9.16E-02	1.31E-01	1.59E-01	1.94E-01	2.59E-01	3.31E-01	2.28E-03	3.24E-02	1.01E-01	2.40E-02	0.00E+00
Alabama	State Urban Counties	01000	U	1.43E-01	1.87E-01	2.79E-01	4.56E-01	5.04E-01	6.25E-01	9.81E-01	1.07E+00	5.57E-03	5.15E-02	3.78E-01	6.86E-02	0.00E+00
Alabama	Autauga County	01001	U	1.26E-01	1.39E-01	1.62E-01	2.90E-01	2.47E-01	3.09E-01	3.73E-01	3.74E-01	1.66E-02	4.52E-02	1.48E-01	3.68E-02	0.00E+00
Alabama	Baldwin County	01003	U	1.54E-01	1.68E-01	1.86E-01	2.16E-01	2.26E-01	2.66E-01	2.99E-01	3.00E-01	3.36E-03	3.60E-02	1.18E-01	6.88E-02	0.00E+00
Alabama	Barbour County	01005	U	5.67E-02	6.65E-02	8.22E-02	1.01E-01	1.19E-01	1.80E-01	1.85E-01	1.89E-01	1.38E-04	4.97E-02	4.21E-02	2.71E-02	0.00E+00
Alabama	Bibb County	01007	R	1.46E-01	1.51E-01	1.66E-01	2.28E-01	2.40E-01	3.04E-01	3.43E-01	3.56E-01	1.43E-07	4.71E-02	1.66E-01	2.70E-02	0.00E+00
Alabama	Blount County	01009	U	1.58E-01	1.64E-01	1.86E-01	2.32E-01	2.20E-01	2.48E-01	2.49E-01	2.50E-01	7.51E-06	2.71E-02	1.68E-01	2.53E-02	0.00E+00
Alabama	Bullock County	01011	R	8.40E-02	8.94E-02	1.06E-01	1.19E-01	1.19E-01	1.34E-01	1.52E-01	1.58E-01	4.75E-05	2.85E-02	3.49E-02	5.51E-02	0.00E+00
Alabama	Butler County	01013	R	5.08E-02	5.30E-02	6.60E-02	6.85E-02	1.02E-01	1.39E-01	1.66E-01	2.00E-01	0.00E+00	4.66E-02	4.45E-02	1.07E-02	0.00E+00
Alabama	Calhoun County	01015	R	2.17E-01	2.28E-01	2.65E-01	3.31E-01	3.05E-01	3.73E-01	4.09E-01	4.15E-01	2.27E-06	3.39E-02	2.28E-01	4.24E-02	0.00E+00
Alabama	Chambers County	01017	U	1.10E-01	1.15E-01	1.23E-01	1.78E-01	1.78E-01	2.02E-01	2.49E-01	2.60E-01	4.39E-07	6.00E-02	9.89E-02	1.94E-02	0.00E+00
Alabama	Cherokee County	01019	R	1.62E-01	1.62E-01	1.62E-01	1.70E-01	1.76E-01	1.87E-01	2.03E-01	2.08E-01	9.18E-04	3.36E-02	1.24E-01	1.72E-02	0.00E+00
Alabama	Chilton County	01021	R	1.10E-01	1.11E-01	1.18E-01	1.39E-01	1.44E-01	1.60E-01	1.70E-01	1.72E-01	7.54E-04	3.61E-02	8.80E-02	1.94E-02	0.00E+00
Alabama	Choctaw County	01023	R	7.88E-02	7.93E-02	8.07E-02	8.31E-02	8.41E-02	8.66E-02	8.98E-02	9.09E-02	6.39E-04	4.41E-02	3.33E-02	6.07E-03	0.00E+00
Alabama	Clarke County	01025	R	5.93E-02	6.06E-02	6.34E-02	6.75E-02	6.98E-02	7.52E-02	9.12E-02	9.84E-02	4.67E-03	3.15E-02	2.61E-02	7.60E-03	0.00E+00
Alabama	Clay County	01027	R	7.78E-02	7.84E-02	8.00E-02	8.31E-02	8.85E-02	9.14E-02	1.02E-01	1.06E-01	2.87E-04	2.93E-02	5.07E-02	8.21E-03	0.00E+00
Alabama	Cleburne County	01029	R	1.05E-01	1.10E-01	1.23E-01	1.31E-01	1.28E-01	1.35E-01	1.42E-01	1.44E-01	2.16E-05	3.16E-02	8.32E-02	1.31E-02	0.00E+00
Alabama	Coffee County	01031	U	8.71E-02	9.89E-02	1.18E-01	1.31E-01	1.51E-01	1.94E-01	2.00E-01	2.04E-01	5.50E-06	3.35E-02	7.45E-02	4.27E-02	0.00E+00
Alabama	Colbert County	01033	U	1.50E-01	1.51E-01	1.79E-01	2.95E-01	2.42E-01	3.55E-01	3.72E-01	3.74E-01	1.38E-03	2.74E-02	1.87E-01	2.60E-02	0.00E+00
Alabama	Conecuh County	01035	R	4.85E-02	5.00E-02	5.41E-02	5.89E-02	6.13E-02	6.45E-02	7.61E-02	8.11E-02	6.87E-05	3.22E-02	2.44E-02	4.68E-03	0.00E+00
Alabama	Coosa County	01037	R	9.49E-02	9.53E-02	9.67E-02	9.89E-02	1.04E-01	1.09E-01	1.15E-01	1.17E-01	7.62E-04	3.67E-02	5.56E-02	1.04E-02	0.00E+00
Alabama	Covington County	01039	R	5.76E-02	5.99E-02	6.58E-02	8.21E-02	8.68E-02	1.16E-01	1.28E-01	1.31E-01	0.00E+00	3.32E-02	4.08E-02	1.28E-02	0.00E+00
Alabama	Crenshaw County	01041	R	7.19E-02	7.76E-02	9.28E-02	1.17E-01	1.14E-01	1.31E-01	1.47E-01	1.54E-01	6.52E-05	2.65E-02	4.44E-02	4.25E-02	0.00E+00

Figure 4-16. 1996 Modeled Exposure Concentrations
Acetaldehyde - Statewide Concentration Distribution Estimates

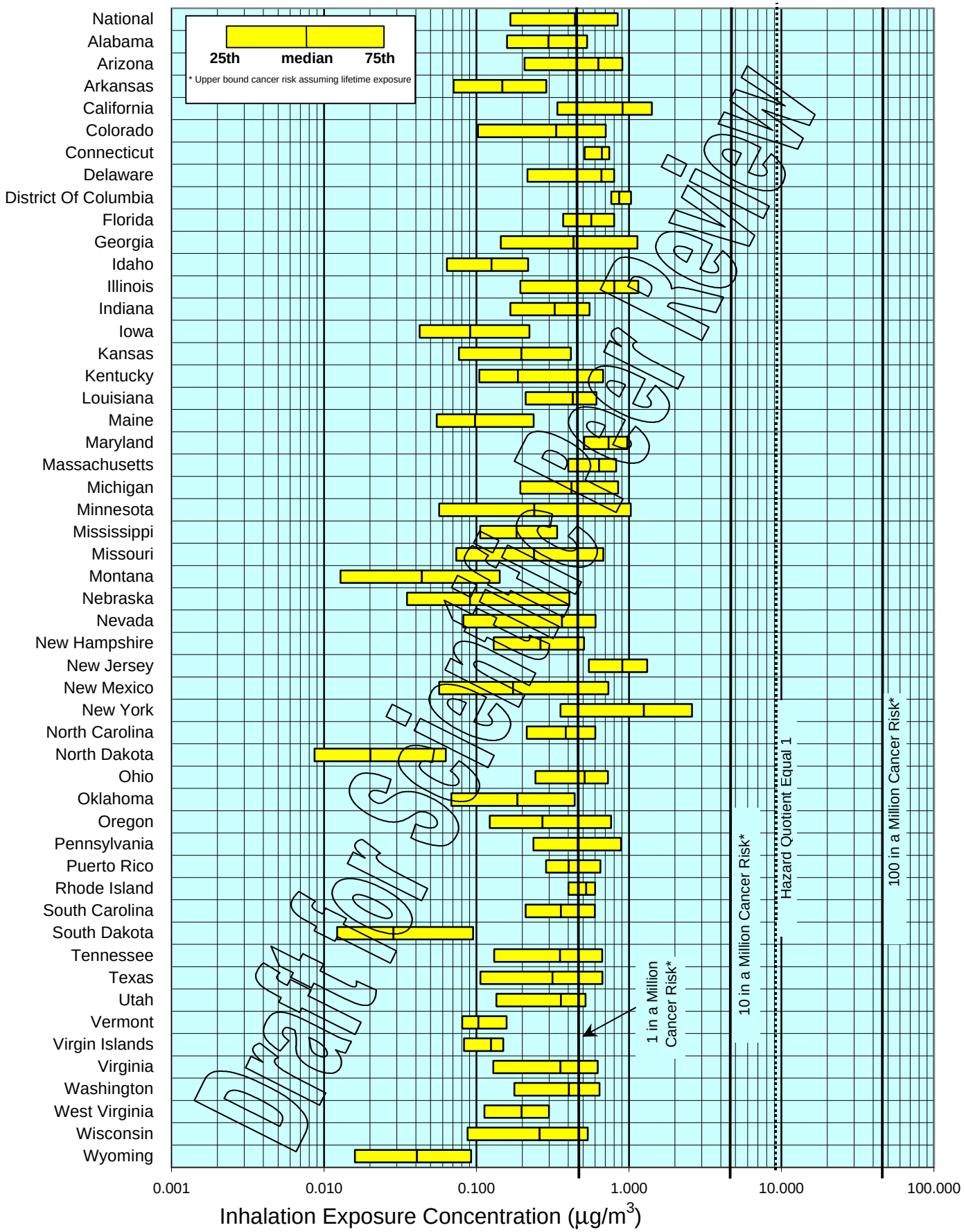

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Figure 4-17. 1996 Modeled Exposure Concentrations
Acetaldehyde - Statewide Source Sector Contribution Estimates

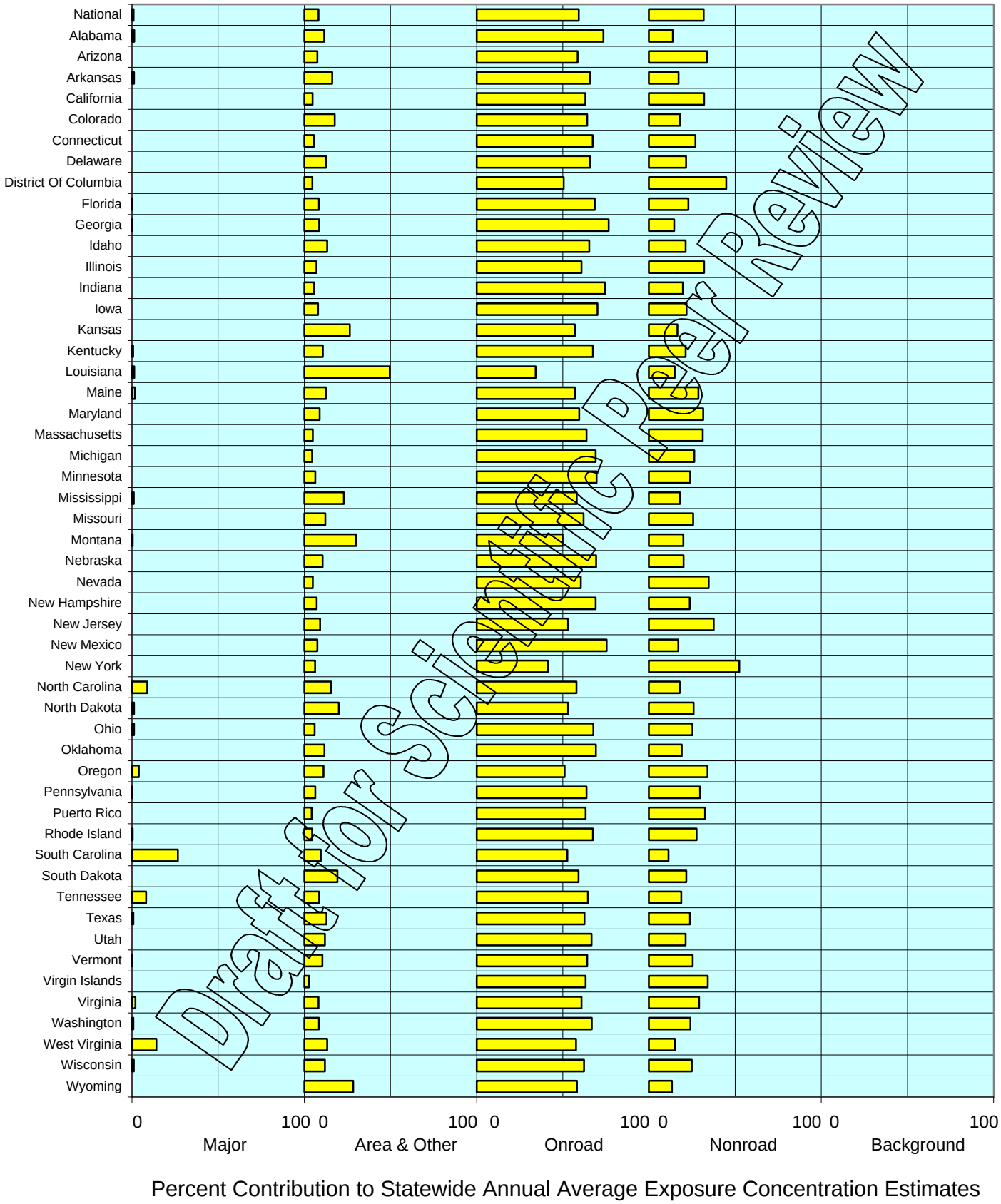

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Figure 4-18. 1996 Modeled Median Exposure Concentrations for North Carolina

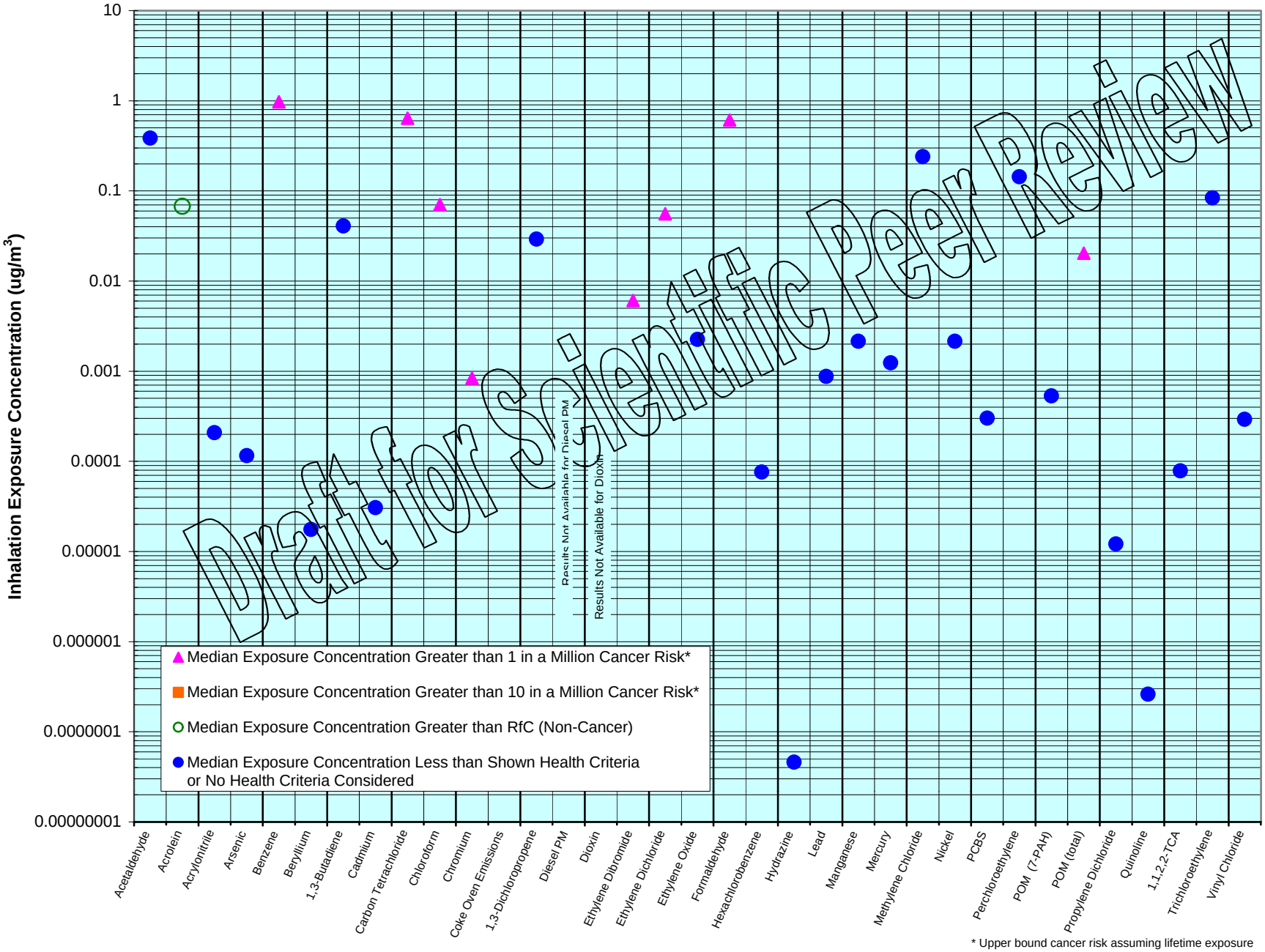

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Figure 4-19. Example State Exposure Concentration Map

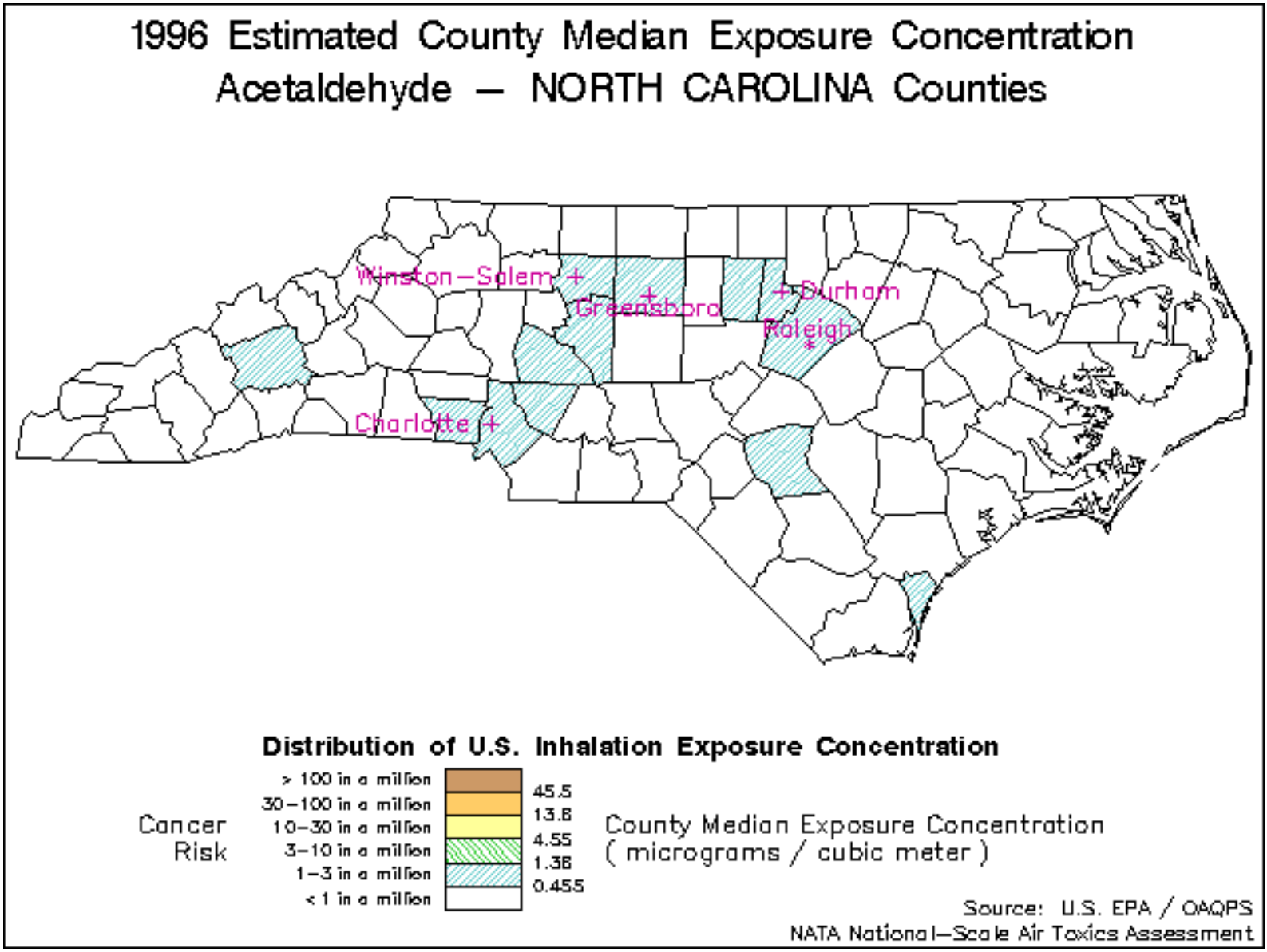


Figure 4-20. Benzene Exposure Variability within a County.

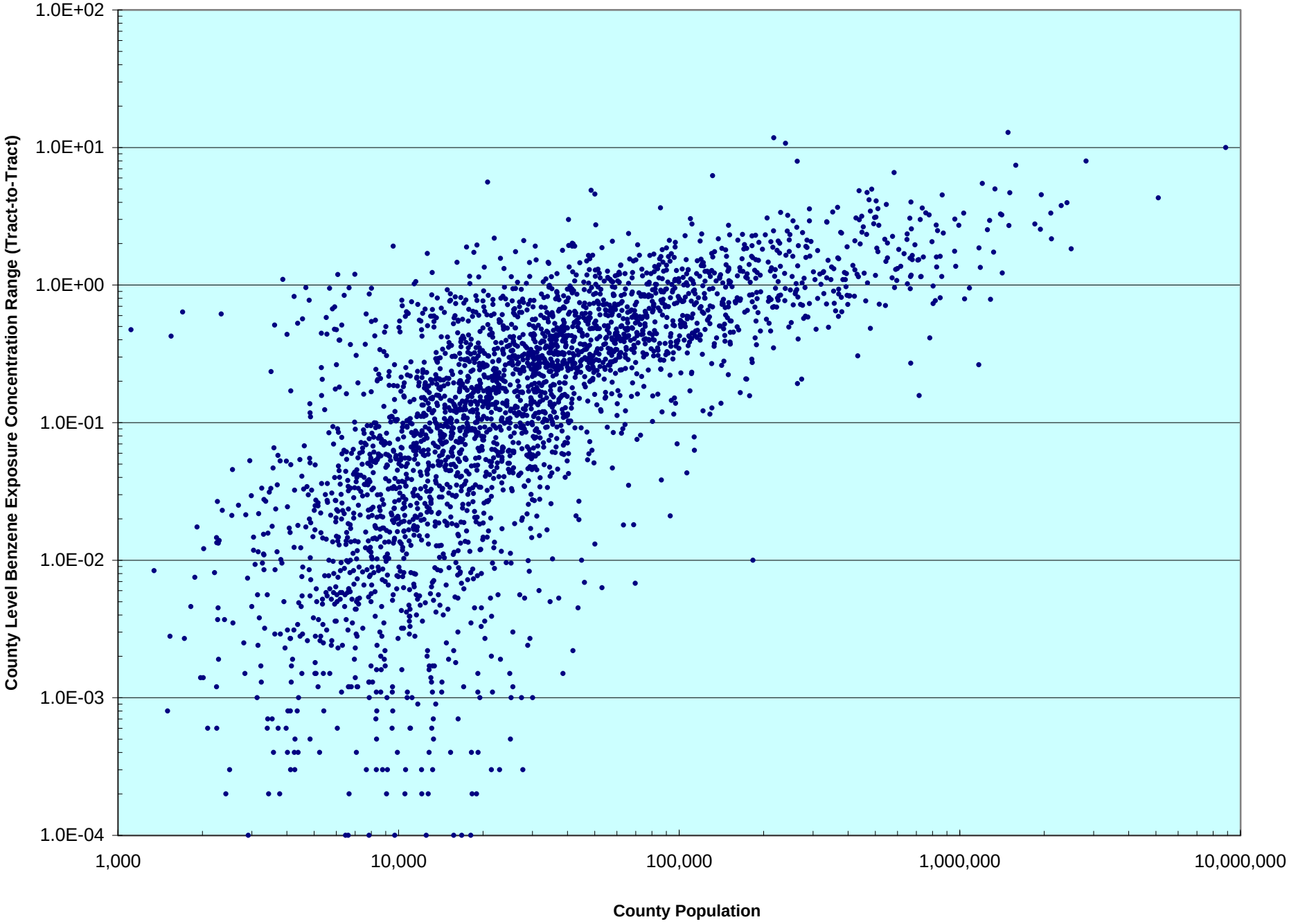
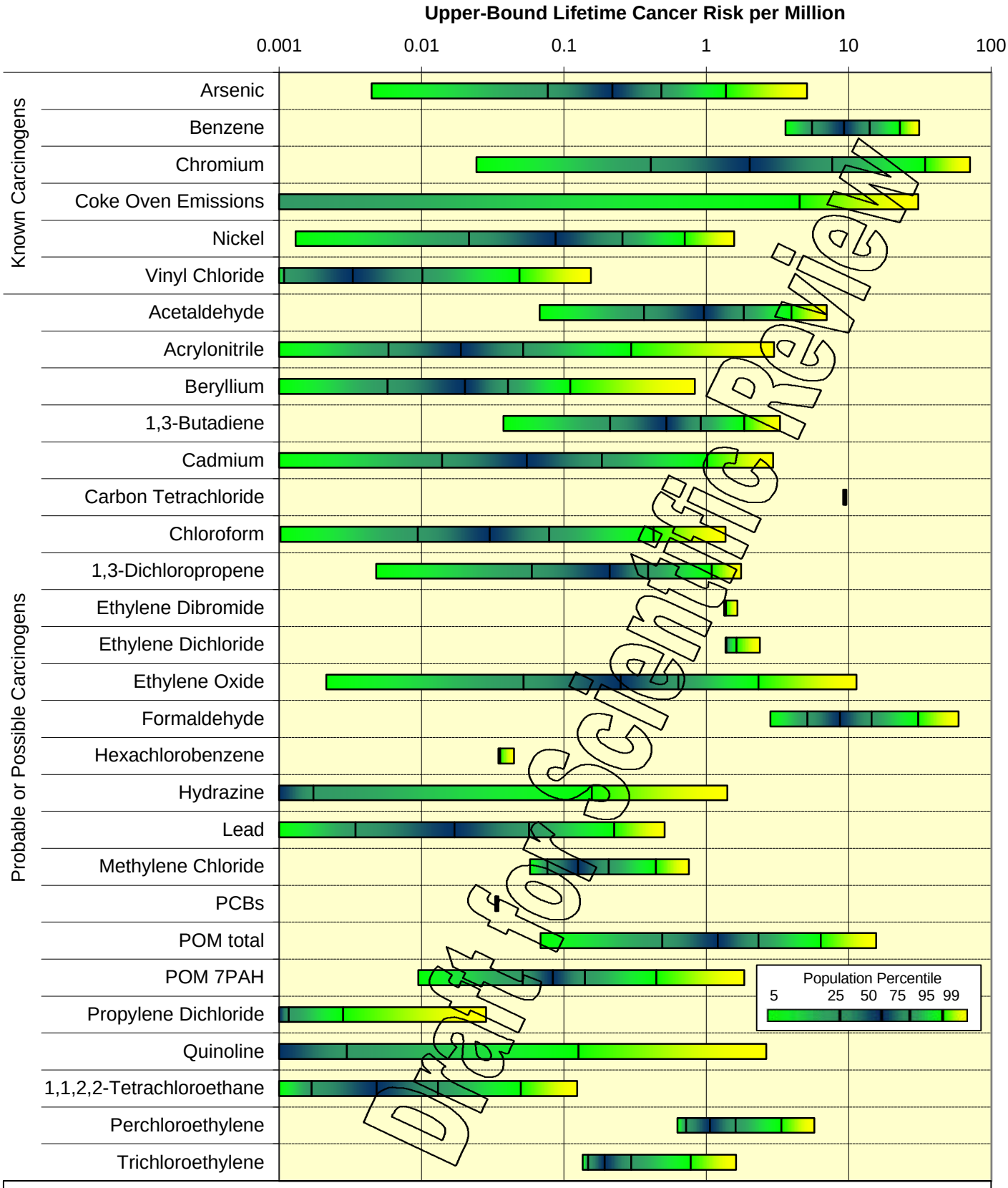


Figure 5-1. 1996 Risk Characterization

Distribution of lifetime cancer risk for the US population, based on 1996 exposure* to all source sectors and background combined.

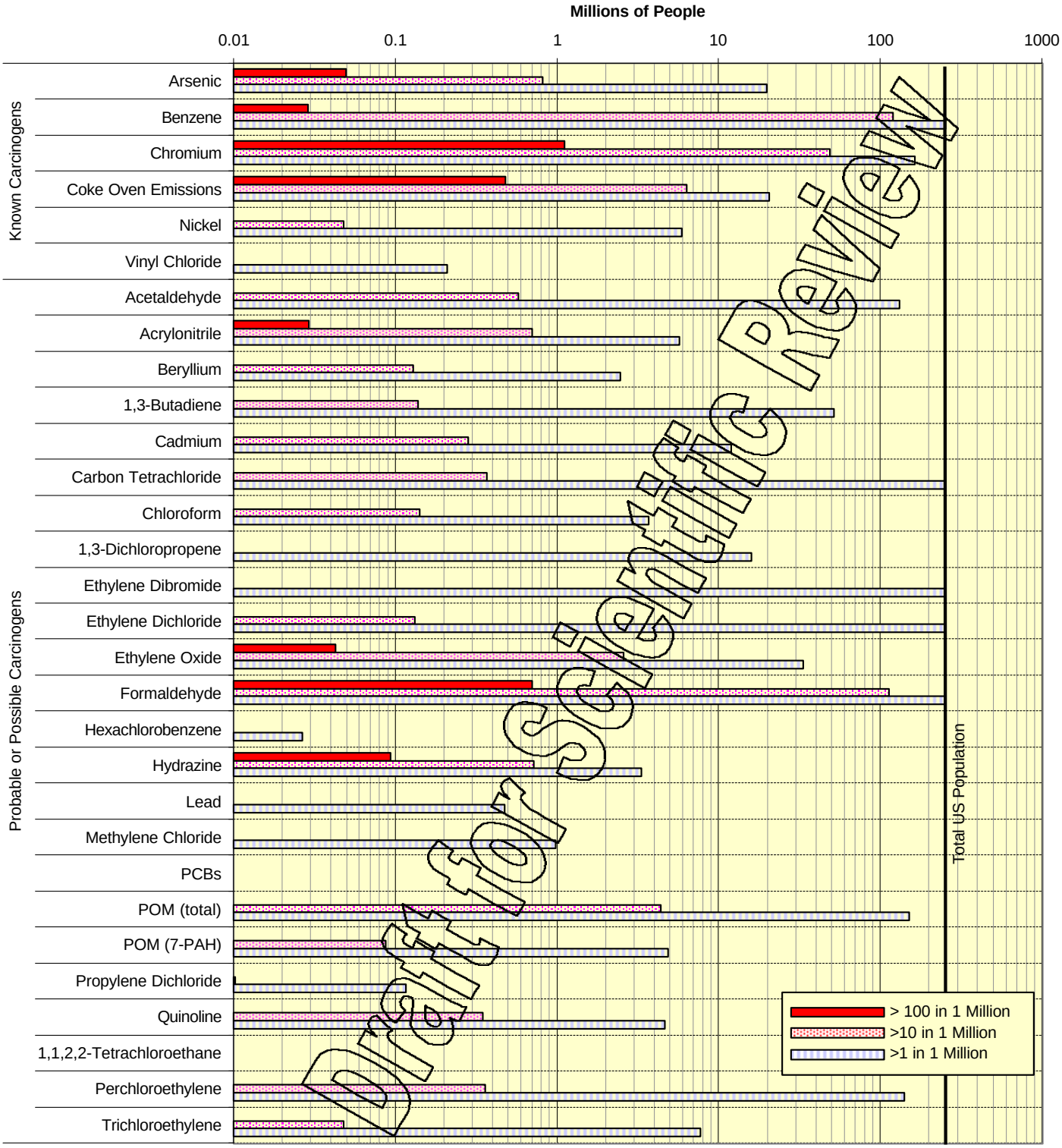


Draft for Scientific Review

* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated cancer risk for some pollutants. See additional information on the following page.

Figure 5-2. 1996 Risk Characterization

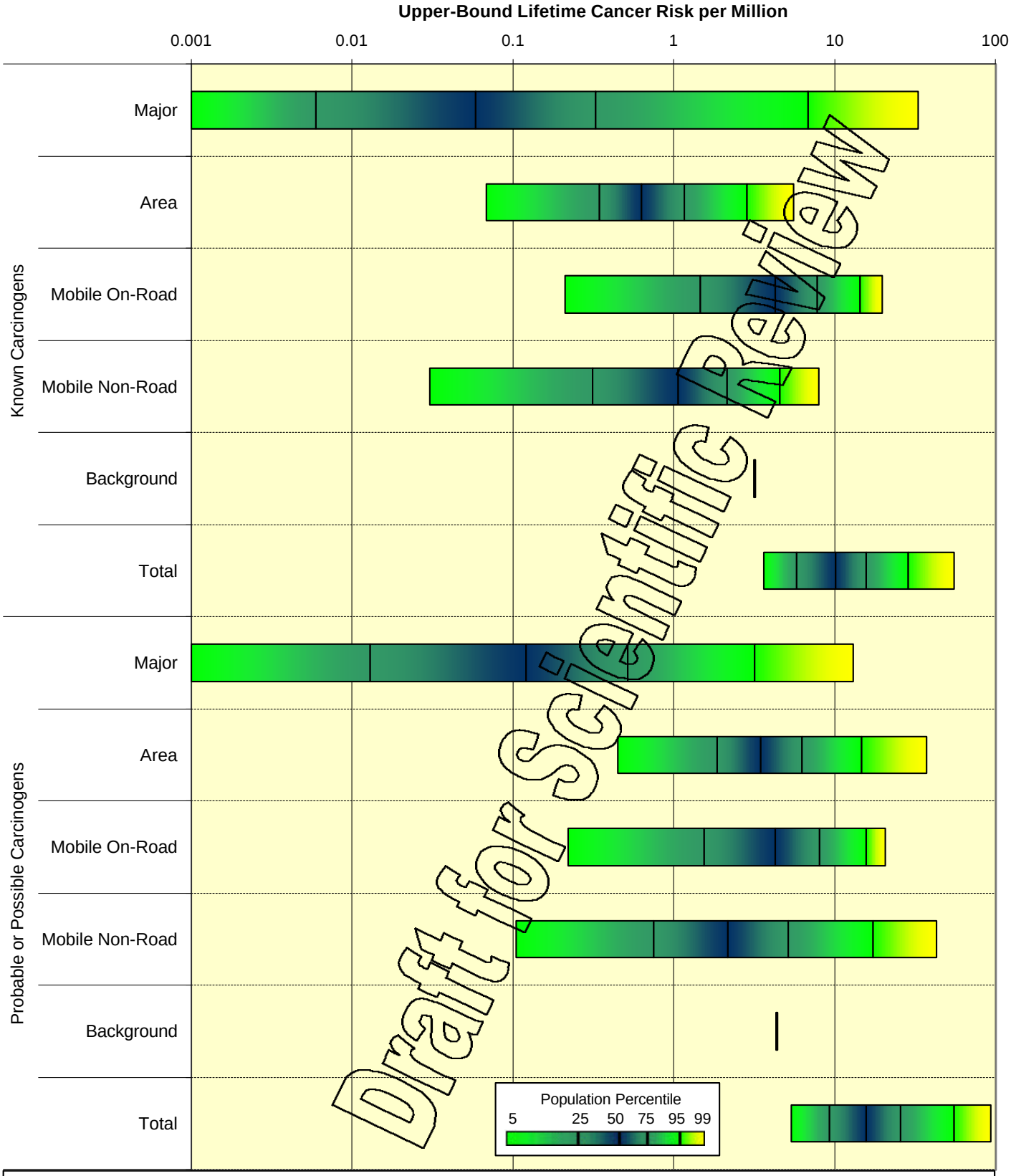
Population whose 1996 exposure* exceeded set cancer risk levels based on all source sectors and background.



* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated cancer risk for some pollutants. See additional information on the following page.

Figure 5-3. 1996 Risk Characterization

Distribution of lifetime cancer risk for the US population, based on 1996 exposure* to multiple carcinogens.

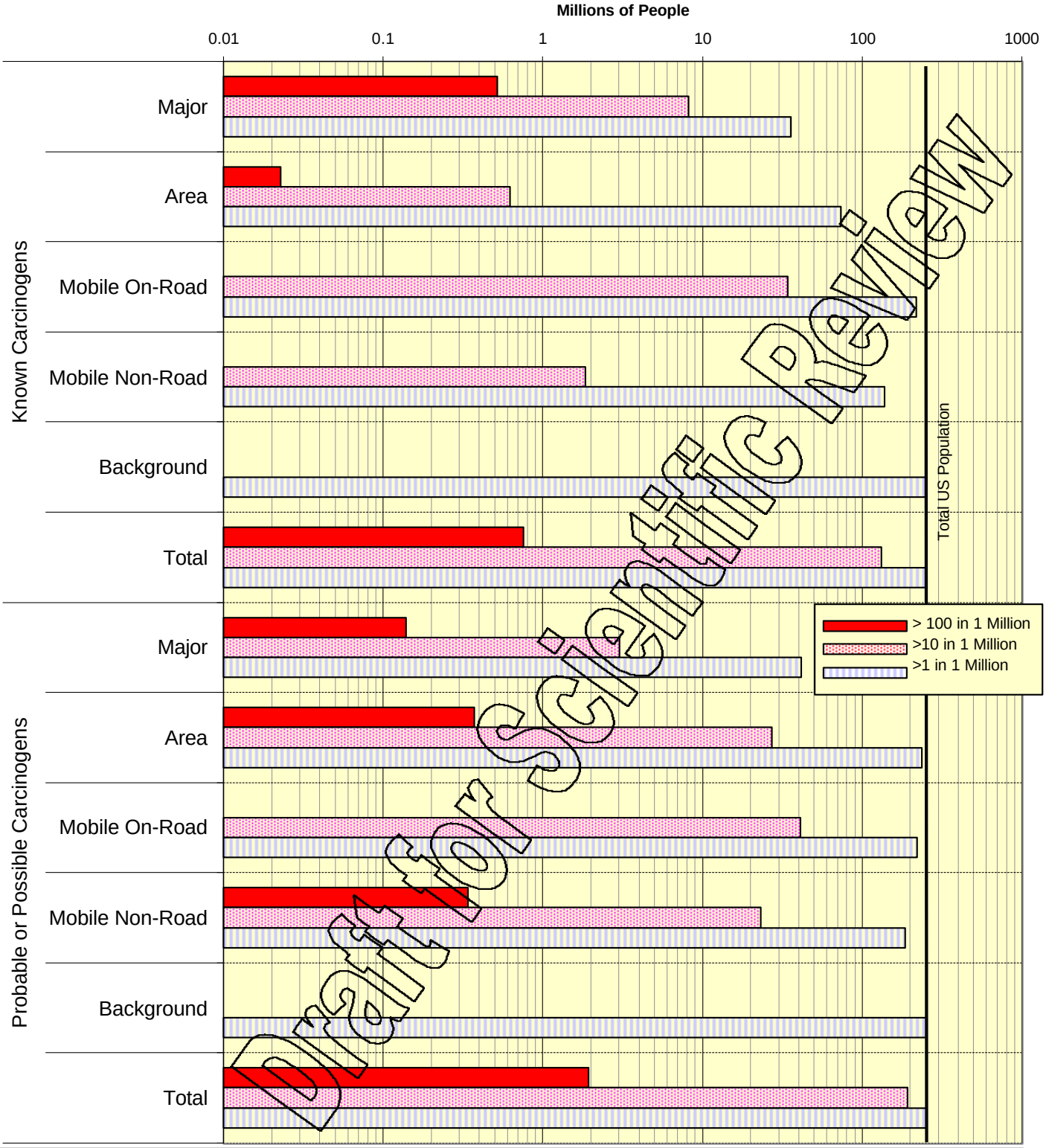


Draft for Scientific Review

* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated cancer risk for some pollutants. See additional information on the following page.

Figure 5-4. 1996 Risk Characterization

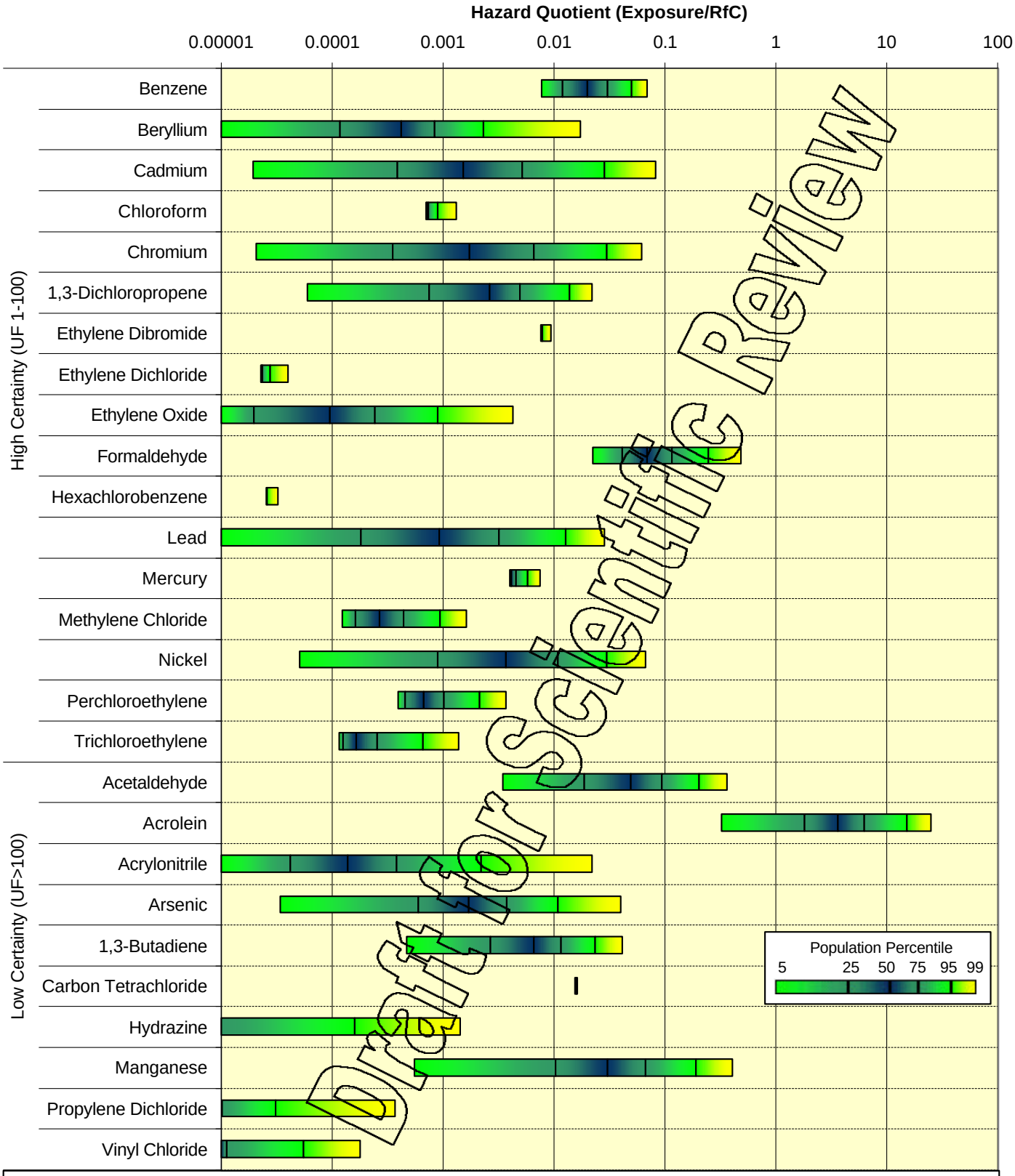
Population whose 1996 exposure* exceeded set risk levels of risk for carcinogens combined.



* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated cancer risk for some pollutants. See additional information on the following page.

Figure 5-5. 1996 Risk Characterization

Distribution of non-cancer hazard quotient for the US population, based on 1996 exposure* to all source sectors and background combined.

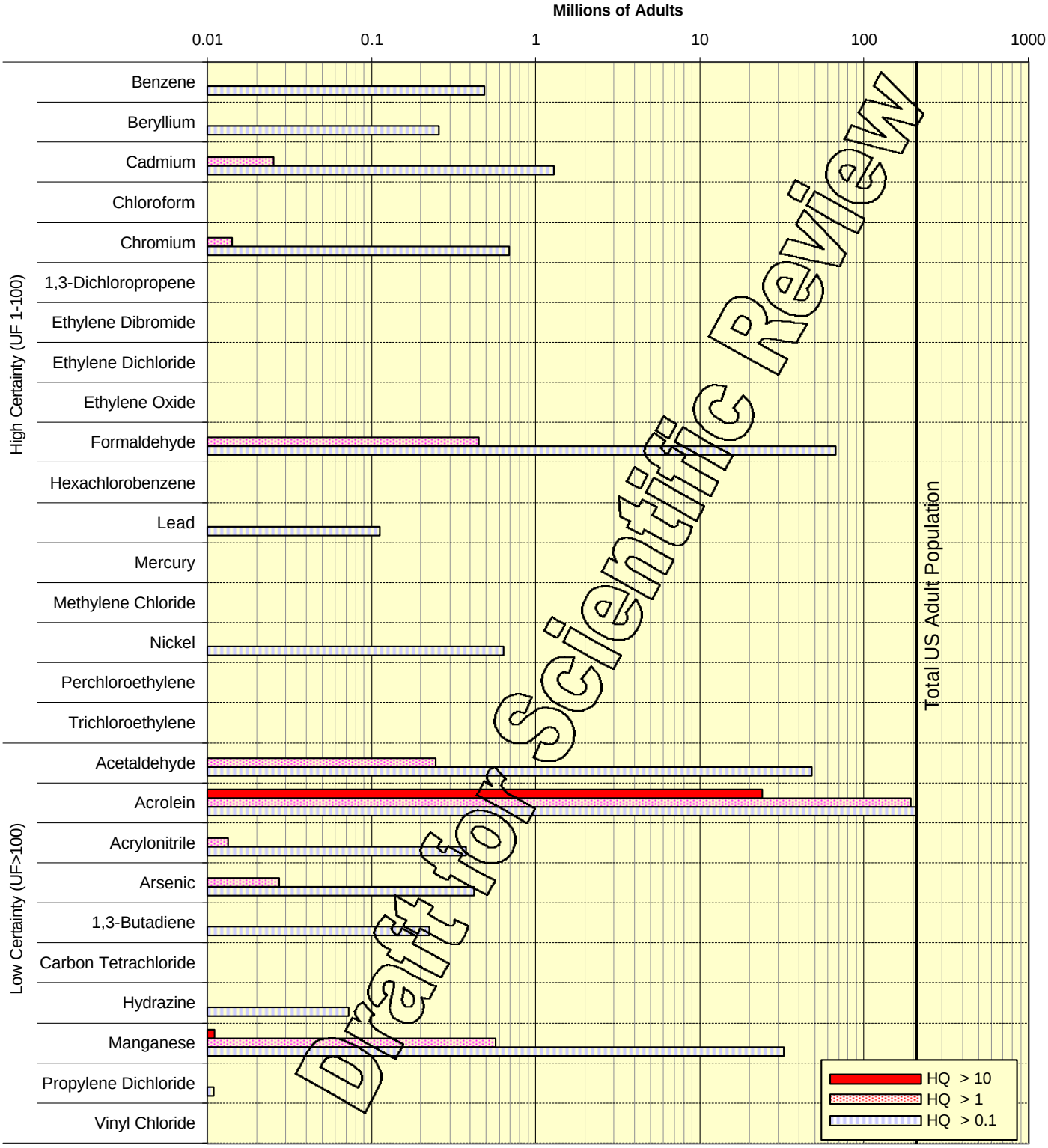


Draft for Scientific Review

* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated non-cancer risk for some pollutants. See additional information on the following page.

Figure 5-6. 1996 Risk Characterization

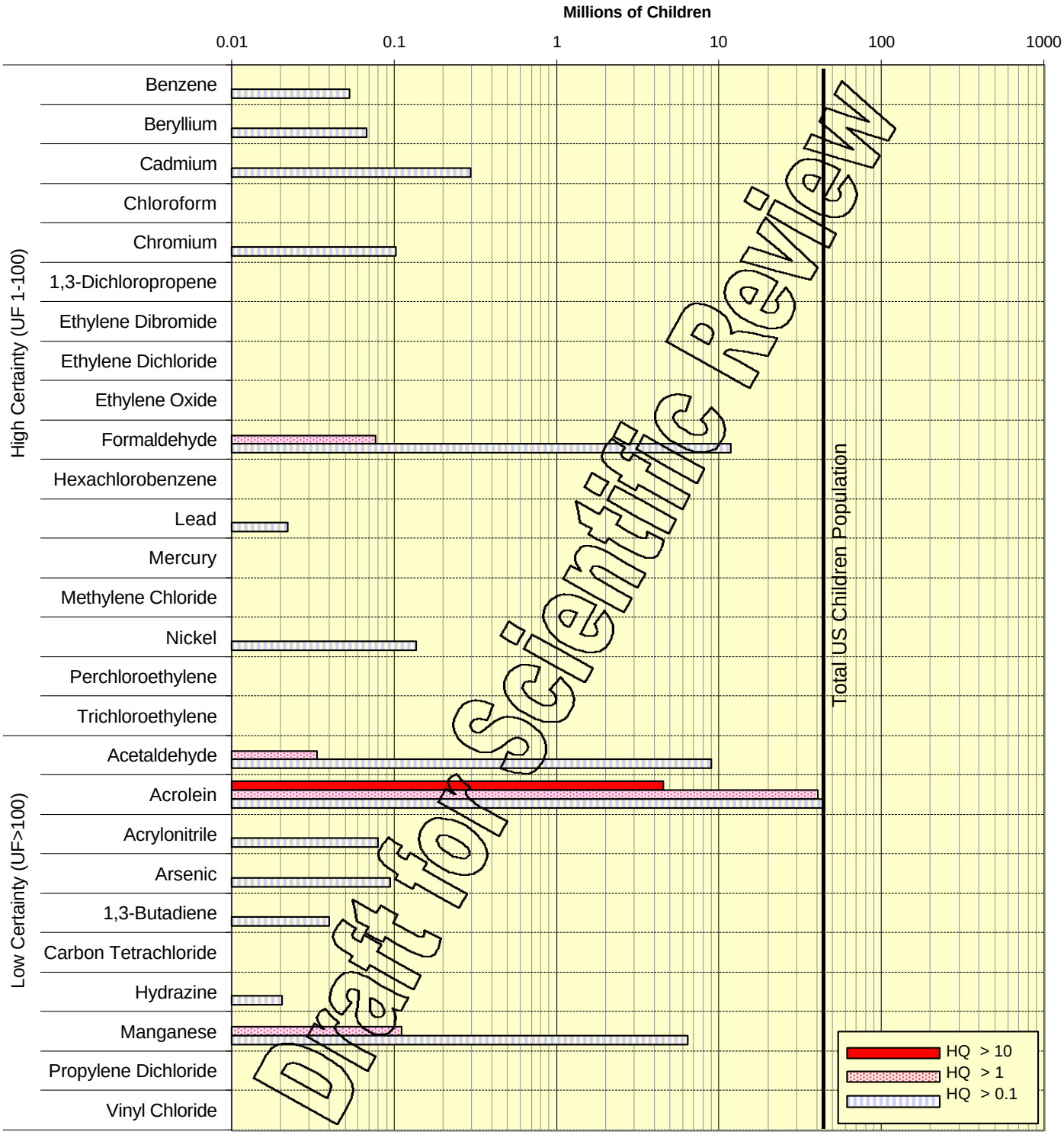
Adult population whose 1996 exposure* exceeded set non-cancer hazard quotient levels based on all source sectors and background combined.



* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated non-cancer risk for some pollutants. See additional information on the following page.

Figure 5-7. 1996 Risk Characterization

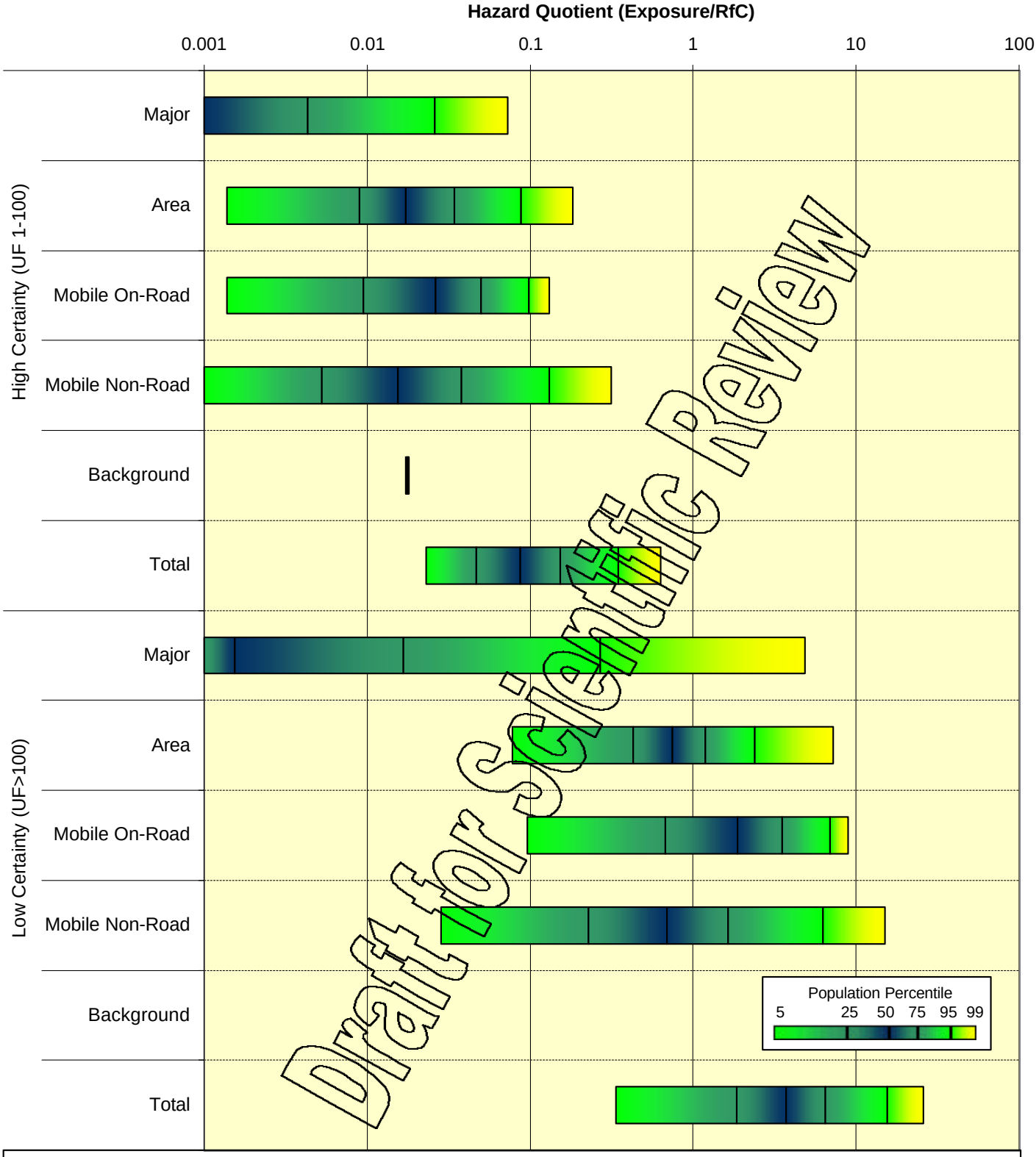
Children population whose 1996 exposure* exceeded set non-cancer hazard quotient levels based on all source sectors and background combined.



* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated non-cancer risk for some pollutants. See additional information on the following page.

Figure 5-8. 1996 Risk Characterization

Distribution of non-cancer target organ-specific hazard index (TOSHI) for effects to the respiratory system, based on 1996 multiple-pollutant exposure* to adults in the US population.



* Results are based on inhalation exposure to outdoor sources only. Although these results assume continuous exposure to 1996 levels of air toxics over a lifetime, current and planned control programs are expected to substantially reduce these exposures and associated non-cancer risk for some pollutants. See additional information on the following page.

Figure 5-9. *Illustration*: Distribution of monitor-to-model ratios for stable gases, developed from ratios for benzene and perchloroethylene.

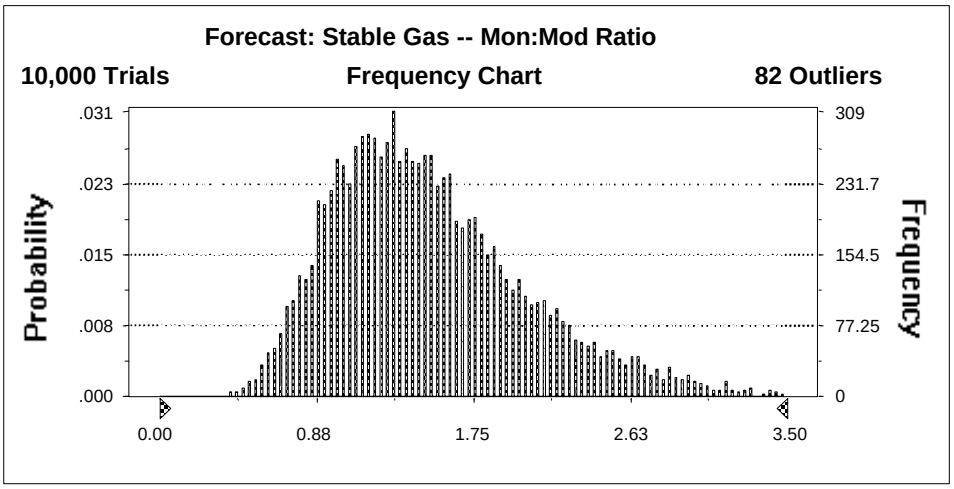


Figure 5-10. *Illustration:* Distribution of monitor-to-model ratios for reactive gases, developed from ratios for formaldehyde and acetaldehyde

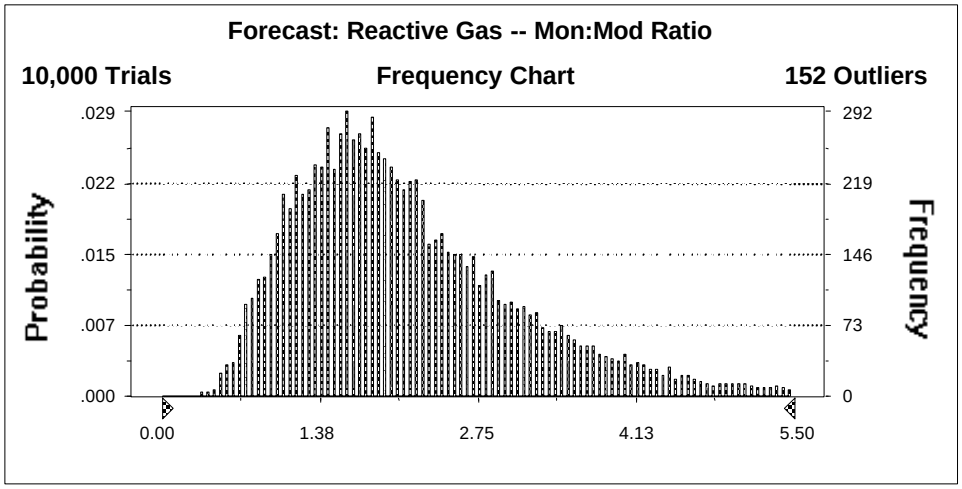


Figure 5-11. *Illustration:* Distribution of monitor-to-model ratios for particulate species, developed from ratios for lead, chromium, and cadmium.

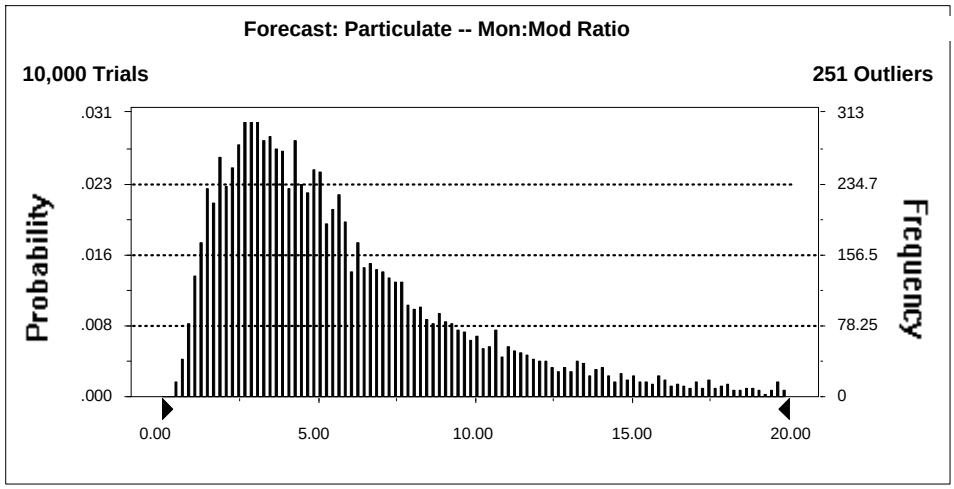


Figure 5-12. *Illustration:* Distribution of ambient-to-personal concentration ratios for ozone, assumed to apply for "typical" gaseous pollutants.

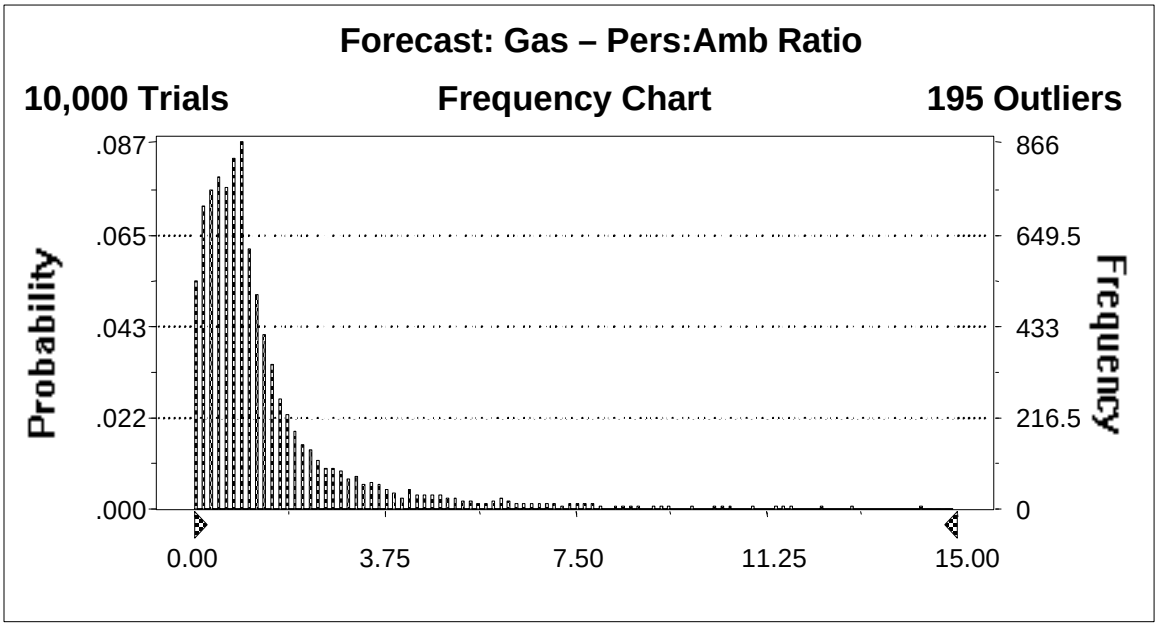


Figure 5-13. *Illustration:* Distribution of ambient-to-personal concentration ratios for particulate matter, assumed to apply for "typical" particulate pollutants.

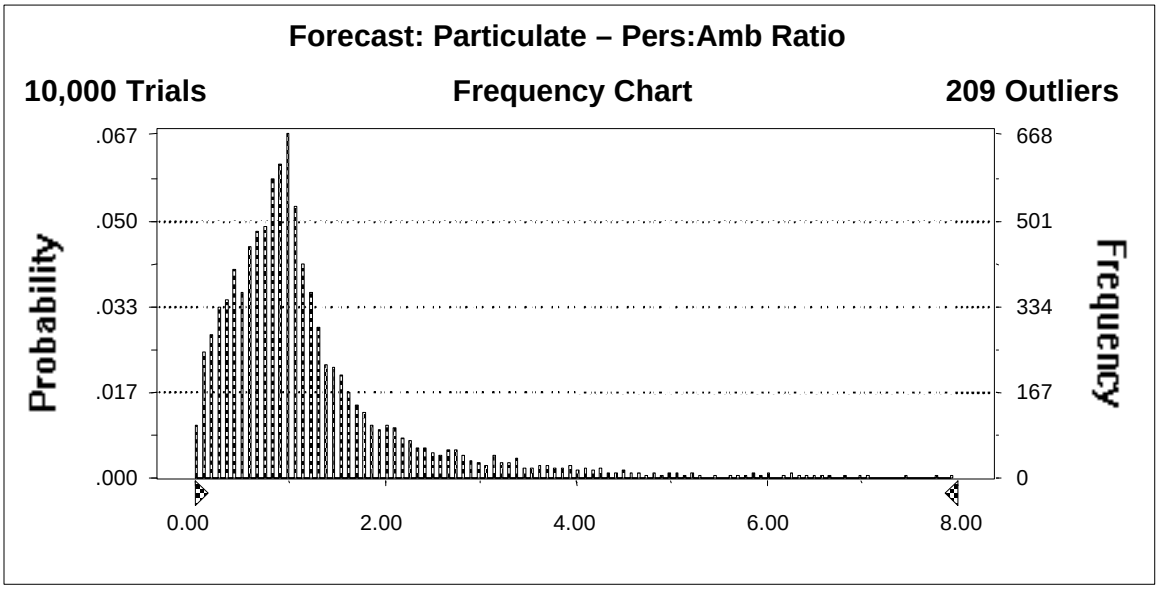


Figure 5-14. *Illustration:* Uncertainty and variability surrounding the URE for benzene, in terms of the ratio between the estimated URE and the "true" URE.

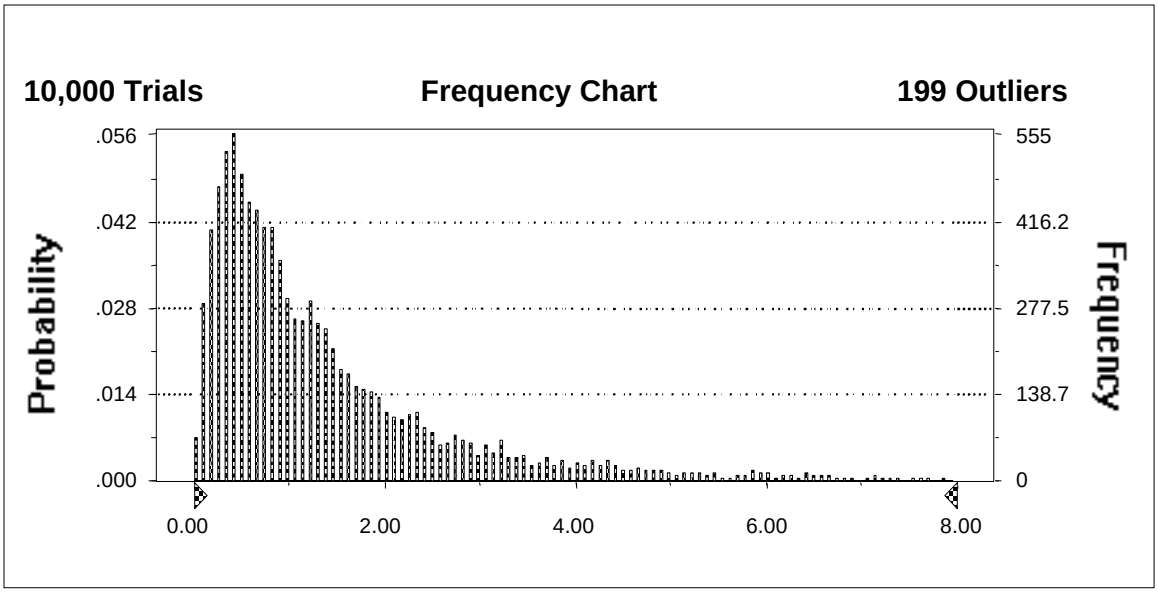


Figure 5-15. *Illustration:* Uncertainty and variability surrounding a typical RfC, in terms of the ratio between the estimated RfC and the "true" RfC.

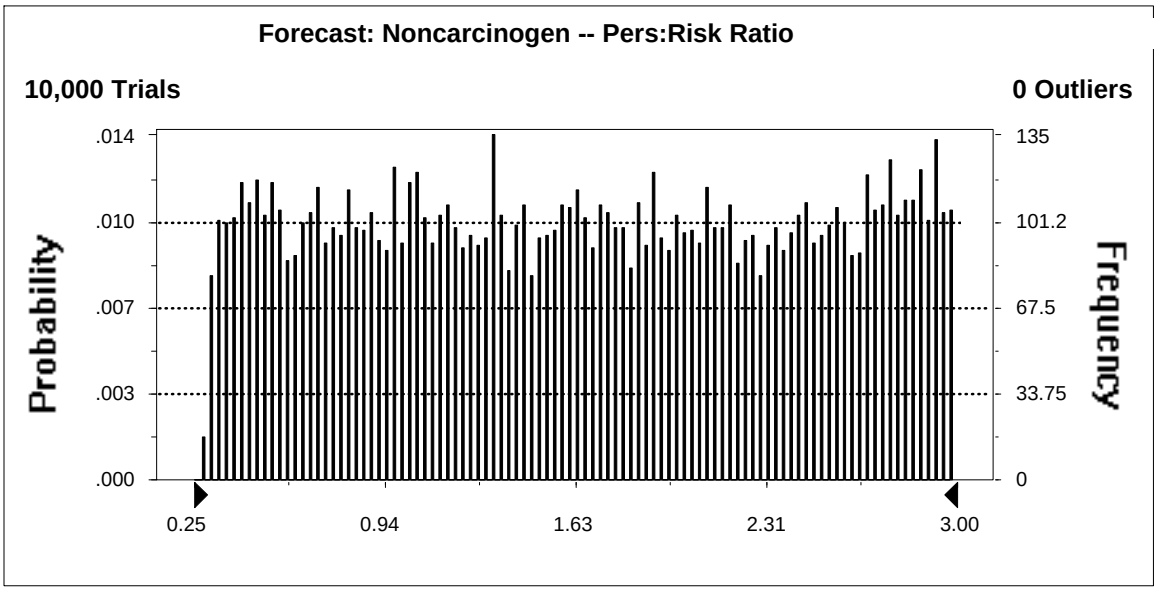


Figure 5-16. Cancer – risk ratio for stable gas.

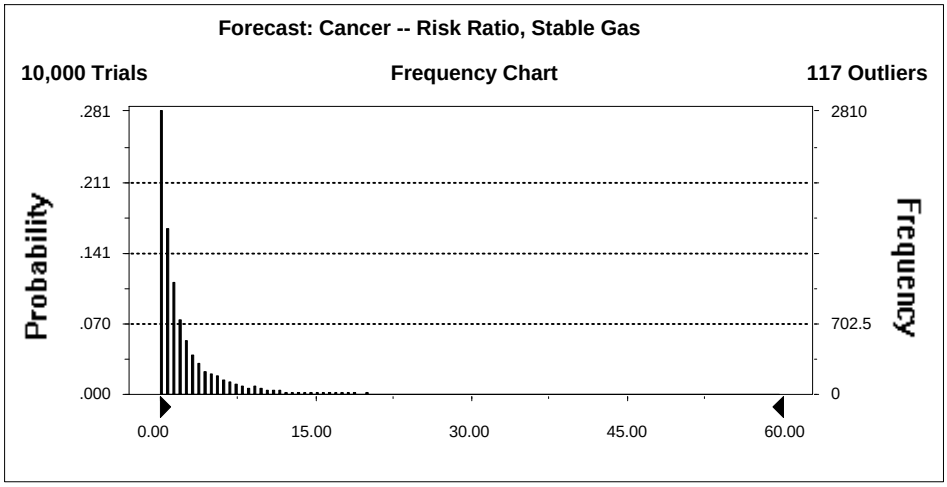


Figure 5-17. Cancer – risk ratio for reactive gas

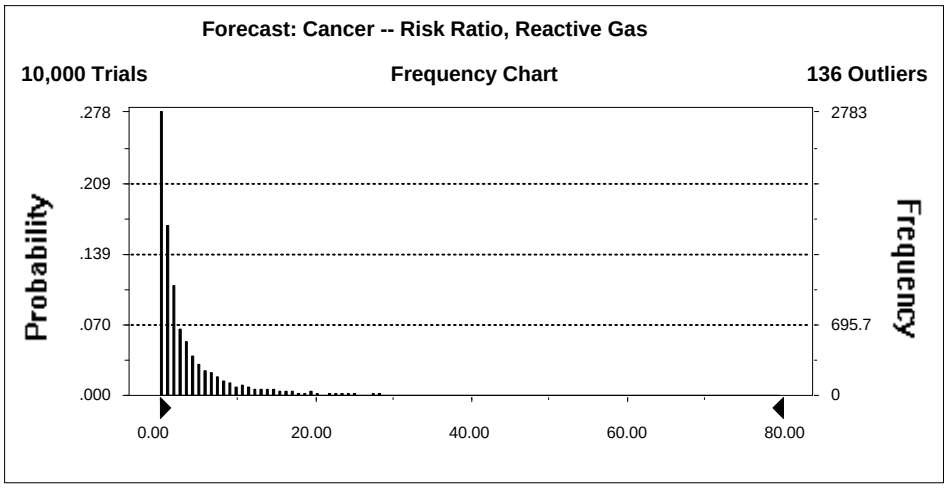


Figure5-18. Cancer – risk ratio for particulate

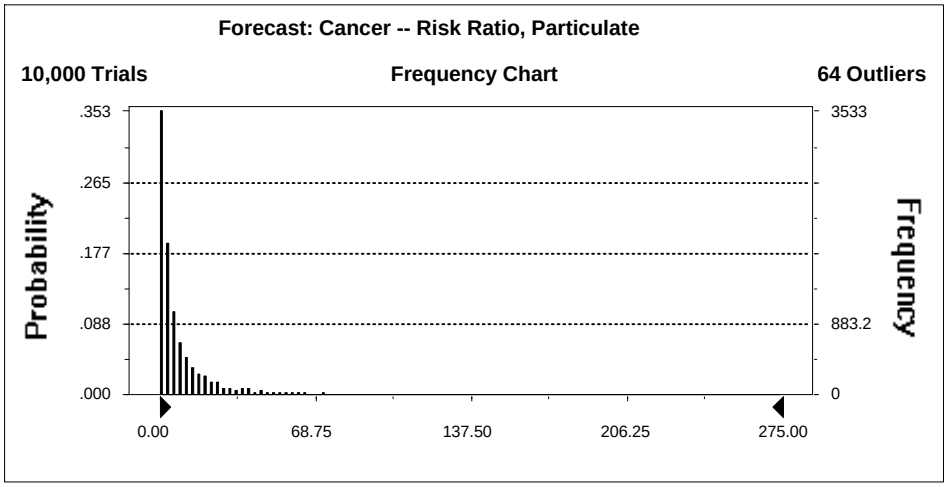


Figure 5-19. Noncancer – risk ratio for stable gas

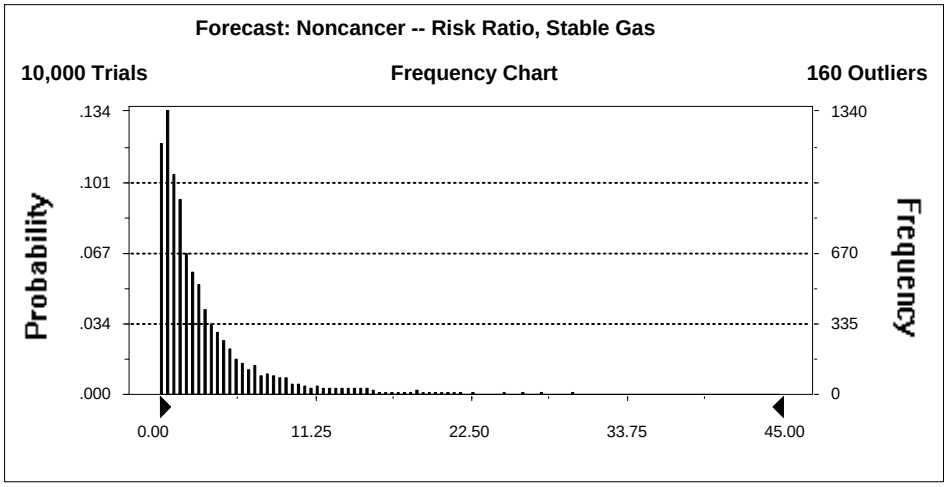


Figure 5-20. Noncancer – risk ratio for reactive gas.

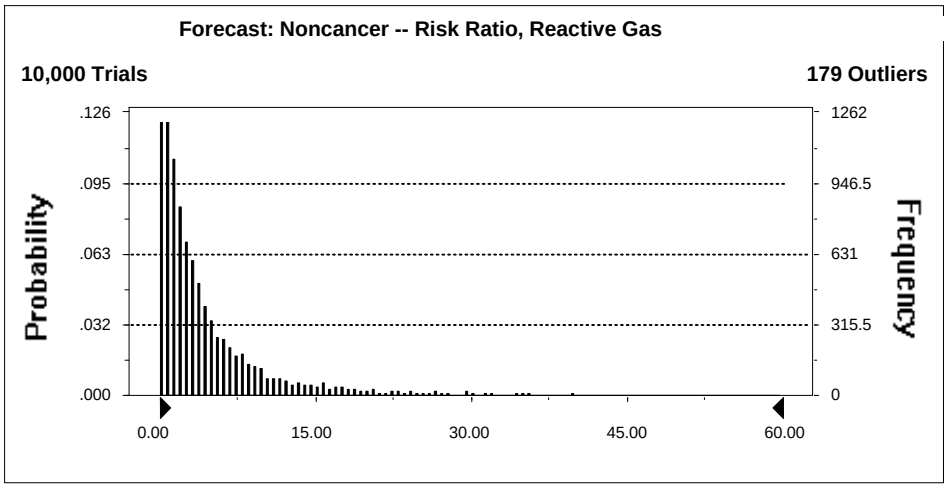
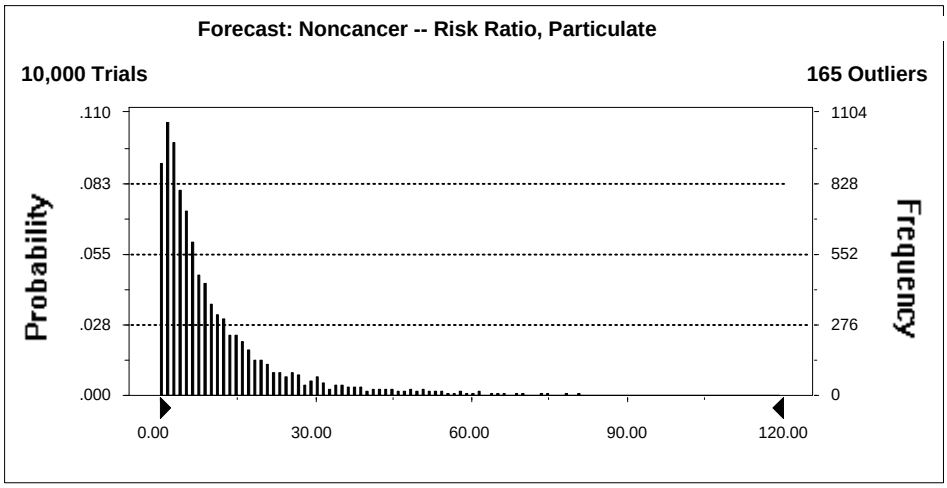


Figure 5-21. Noncancer – risk ratio for particulate.



Appendix A

Summary of July 2000 Peer Review of the Draft Document "Planning and Scoping the Initial National-Scale Assessment: An Element of the EPA National Air Toxics Program"

(Provided in electronic format on Appendices CD)

Appendix A - Summary

Summary of July 2000 Peer Review of the Draft Document "Planning and Scoping the Initial National-Scale Assessment: An Element of the EPA National Air Toxics Program"

In July 2000, six non-U.S. EPA scientists completed a peer review of the "Planning and Scoping the Initial National-Scale Assessment: An Element of the EPA National Air Toxics Program'". The reviewers were asked to focus their review on the main body of the planning and scoping document as well as the supporting technical information. Reviewers were asked to consider the appropriateness of approaches used to (1) process the State-derived National Toxics Inventory for dispersion modeling, (2) estimate ambient concentrations using the Assessment System for Population Exposure Nationwide (ASPEN) model, (3) estimate human inhalation exposures using the Hazardous Air Pollutant Exposure Model version 4 (HAPEM4), and (4) estimate, aggregate, and interpret associated cancer and non-cancer risks. Specific charge questions, the reviewer's comments as well as the EPA's response to these comments are contained in this appendix

Appendix B

HAPEM4 Documentation

(Provided in electronic format on Appendices CD)

Appendix B - Summary

HAPEM4 Documentation

This appendix contains detailed documentation for the HAPEM4 exposure model. Two items are include "The HAPEM4 User's Guide" and the "Development of Microenvironmental Factors for the HAPEM4 in Support of the National Air Toxics Assessment (NATA)"

The HAPEM4 User's Guide contains:

Chapter 1 - Provides a brief introduction to HAPEM4 modeling fundamentals including a brief history of the development of HAPEM4.

Chapter 2 - Provides an overview of the various components of HAPEM4 and basic i information needed to run the model.

Chapter 3 - Provides a description of the format, data, and options for each of HAPEM4 input files.

Chapter 4 - Provides a description of the format and data associated with each of HAPEM4 output files.

Chapter 5 - Provides a description of the purpose and operations, inputs, and outputs, including a brief description of the computer code, for each of HAPEM4 computer programs.

Chapter 6 - References.

The Microenvironmental Factors Report contains detailed information on:

- Defining the HAPEM4 ME Factors
- Calculating HAPEM4 ME Concentrations and Estimation of Proximity Factors
- Estimating Ambient HAP Concentrations Using ASPEN
- Literature Search
- Grouping HAPs and Microenvironments
- A complete listing of the ME factors for the Urban 33 pollutants
- ME Factors for Diesel PM (Provided as an addendum to the Report)
- The Use of a Linear Model for the Initial NATA Assessment

Appendix C
EMS-HAP User's Guide

(Provided in electronic format on Appendices CD)

Appendix C - Summary

The Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP) User Guide Synopsis

1.1 What is EMS-HAP?

The Emissions Modeling System for Hazardous Air Pollutants (EMS-HAP) is a series of computer programs that process emission inventory data for subsequent air quality modeling. EMS-HAP accomplishes two goals.

1. It processes an emission inventory, such as the 1996 National Toxics Inventory, for use in the Assessment System for Population Exposure Nationwide (ASPEN) dispersion model.

2. It allows you to estimate future emissions resulting from user-designed emission reduction scenarios and growth.

To accomplish the first goal, EMS-HAP:

- quality assures point source inventory location and stack parameter data and defaults missing or erroneous data where possible,
- groups individual pollutant species (e.g. lead oxide, lead chromate into lead compounds),
- facilitates the selection of pollutants and pollutant groups for modeling,
- spatially allocates area and mobile source emissions from the county level to the census tract level using surrogates such as industrial land or roadway miles,
- allocates aircraft emissions to airport locations,
- temporally allocates annual emission values to average hourly values based on the type of source, and,
- produces emission files formatted for direct input into the ASPEN model.

To accomplish the second goal, EMS-HAP adjusts point, area and mobile emissions to account for growth and emission reductions resulting from user-designed scenarios such as the implementation of the Maximum Achievable Control Technology (MACT) standards.

The U.S. Environmental Protection Agency's Office of Air Quality Planning and Standards (EPA/OAQPS), developed EMS-HAP to facilitate multiple runs of ASPEN and to analyze emission reduction scenarios. ASPEN can be used to estimate annual average ambient air quality concentrations of multiple pollutants emitted from a large number of sources at a large scale (i.e. nationwide) as part of a national air toxics assessment.

Although we tailored EMS-HAP to process the1996 National Toxics Inventory (NTI), you can use it for any emission inventory following the instructions in this guide. The 1996 NTI is the first comprehensive model-ready national inventory of toxics, containing facility-specific estimates of hazardous air pollutants (HAPs).

While other emission models, such as EMS-95 and EPS 2.0, are available, they do not address the details of the 1996 NTI or the input requirements of the ASPEN model.

1.2 How do I use the EMS-HAP User's Guide?

This guide describes the programs that comprise EMS-HAP, and gives instructions on how to use them to create ASPEN emission input files for base year or projected year inventories of your choice. This manual is not specific to any one input inventory. For example, you are not limited to using the 1996 NTI to run EMS-HAP. You need only make sure your input inventory meets the requirements described within each program.

This guide also provides information on how we used EMS-HAP to process 1996 emissions data for a national screening study.

We present the programs in the order you may choose to use them. Chapter 2 describes the aircraft emissions processing program. Chapters 3 through 7 describe the point source processing programs. Chapters 8 through 10 describe the programs for area and mobile source processing. Each chapter describes the function of the program, how to run the program, all required ancillary input files and emission inventory data requirements, and how to evaluate the output to determine if the data were processed successfully. In this guide, all ancillary SAS data files are named without their extension, since SAS files extension names vary with system and engine type. All programs are also named without their extension.

Appendix A presents the file formats of the ancillary input files. Appendix B contains sample batch files for running the EMS-HAP programs. Appendix C discusses preparation of the point source component of the 1996 NTI for input into EMS-HAP. Appendix D presents the methodologies used to prepare emission input files for the ASPEN model for a national air toxics assessment. Appendix D also discusses how we developed the key ancillary input files, such as the spatial allocation factor files, provided with EMS-HAP. The ancillary files provided with EMS-HAP are those we used to produce the 1996 ASPEN modeling inventory.

A separate user's guide is available for the ASPEN model. Users familiar with ASPEN model input requirements will have a better understanding of EMS-HAP.

Appendix D

Development of the Emission Inventory

(Provided in electronic format on Appendices CD)

Appendix D - Summary

Development of the Emission Inventory

This appendix includes a technical paper describing the development of the 1996 National Toxic Inventory (NTI). The following is the abstract for this paper:

The 1990 amendments to the Clean Air Act (CAA) established the need for a comprehensive hazardous air pollutant (HAP) emissions inventory effort that can be used to track progress by the Environmental Protection Agency (EPA) over time in reducing HAPs in ambient air. To estimate risk and HAP emission reductions, the EPA compiled the 1996 National Toxics Inventory (NTI) to provide a model-ready emissions inventory.

The 1996 NTI contains estimates of facility-specific HAP emissions and their source-specific parameters necessary for modeling such as location and facility characteristics (stack height, exit velocity, temperature, etc.). Complete source category coverage is needed for modeling, and the NTI contains estimates of emissions from major, area, and mobile source categories. Compiling this huge amount of data presents a significant challenge to EPA. To compile the data, the EPA first solicited HAP emissions data from states, and 36 states, Puerto Rico and the Virgin Islands delivered HAP emissions inventories to the EPA. These state data varied in completeness, format, and quality. The EPA evaluated the state data and supplemented it with data gathered while developing Maximum Achievable Control Technology (MACT) standards and with Toxic Release Inventory (TRI) data. Then the EPA estimated emissions for other states and for sources not included in the state data to produce a complete model-ready national 1996 inventory. The EPA released the draft 1996 NTI for external comment and received revisions from 42 states, industry, and other organizations. The EPA released the final 1996 NTI in June 2000. This paper discusses the compilation of the 1996 NTI in order to evaluate the success of EPA's national air toxics program and presents summary emissions data from the 1996 NTI.

In addition to the paper include in Appendix D, the following are reference files that provide further detail on the inventory development. These documents are avaiable at http://www.epa.gov/ttn/chief/ei_guide.html#airtoxics

- Documentation for the 1996 Base Year National Toxics Inventory for Aircraft Sources
- Documentation for the 1996 Base Year National Toxics Inventory for Area Sources
- Documentation for the 1996 Base Year National Toxics Inventory for Commercial Marine Vessel and Locomotive Mobile Sources
- Documentation for the 1996 Base Year National Toxics Inventory for Nonroad Vehicle and Equipment Mobile Sources
- Documentation for the 1996 Base Year National Toxics Inventory for Onroad Sources
- Documentation for the 1996 Base Year National Toxics Inventory for Point Sources

Appendix E

ASPEN User's Guide

(Provided in electronic format on Appendices CD)

Appendix E - Summary

This appendix contains the **" User's Guide for the Assessment System for Population Exposure Nationwide Model (ASPEN, Version 1.1"**

This user's guide provides documentation for the Assessment System for Population Exposure Nationwide (ASPEN, Version 1.1), referred to hereafter as ASPEN. It includes a technical description of the ASPEN algorithms, user instructions for running the model and a tutorial for getting started. The ASPEN model consists of a dispersion and mapping module. The dispersion module is a Guassian formulation for estimating ambient annual average concentrations at a set of fixed receptors within the vicinity of the emission source. The mapping module produces a concentration at each census tract. Input data needed are emissions data, meteorological data and census tract data.

Appendix F

Estimation of Background Concentrations for Diesel Particulate Matter

(Provided in electronic format on Appendices CD)

Appendix F - Summary

Estimation of Background Concentrations for Diesel Particulate Matter

This appendix contains the calculations utilized to determine the background concentrations for Diesel Particulate Matter. Background concentrations are an essential part of the total air quality concentration to be considered in determining source impacts. Background air quality includes pollutant concentrations due to: 1) natural sources; 2) nearby sources that are unidentified in the inventory; and 3) long range transport into the modeling domain. Typically, monitored air quality data should be used to establish background concentrations.

Appendix G

Health Effects Information Used in Cancer and Noncancer Risk Characterization for the NATA 1996 National-Scale Assessment

(Provided in electronic format on Appendices CD)

Appendix G - Summary

Health Effects Information Used In Cancer and Noncancer Risk Characterization for the NATA 1996 National-Scale Assessment

This appendix contains the hazard identification and dose-response assessment information for the NATA national-scale assessment. The criteria for selection of the information was obtained from various sources and prioritized according to (1) applicability, (2) conceptual consistency with EPA risk assessment guidelines, and (3) level of review received. The prioritization process was aimed at incorporating into our assessment the best-available science with respect to dose-response information. The following sources were used:

- US Environmental Protection Agency (EPA)
- Agency for Toxic Substances and Disease Registry (ATSDR)
- California Environmental Protection Agency (CalEPA)
- International Agency for Research on Cancer (IARC)

Appendix H

Estimating Carcinogenic Potency for Mixtures of Polycyclic Organic Matter (POM) for the 1996 National-Scale Assessment

(Provided in electronic format on Appendices CD)

Appendix H - Summary
Estimating Carcinogenic Potency for Mixtures of Polycyclic Organic Matter (POM) for the 1996 National-Scale Assessment

The polycyclic organic matter (POM) category within the Clean Air Act's section 112(b) list of hazardous air pollutants comprises a broad spectrum of compounds having widely varying toxic potencies. Because all these compounds have been listed as a single category under the Act, the 1996 National Toxics Inventory (NTI) also records them only as a group for the great majority of sources, usually in terms of total polynuclear aromatic hydrocarbons (PAH – one type of POM) or total POM. Most of these entries do not include information on the method used to estimate the emission rate.

For this reason, the NTI data could not support modeling of individual POM compounds for the initial national-scale assessment. The alternative-modeling modeling POM as a group – was a significant simplifying step because different types of emission sources may be expected to produce different characteristic mixtures of POM compounds. These different mixtures have the potential to vary substantially in toxic potency per unit mass. The method to aggregate these pollutants for the national scale assessment is presented in this appendix**.**

Appendix I

Model-to-Monitor Comparison Methods

(Provided in electronic format on Appendices CD)

Appendix I - Summary

Protocol for Model-to-Monitor Comparisons for the Initial National Scale Assessment

This appendix provides the protocol for "Model-to-Monitor" comparisons for national scale Assessment (Protocol) that was reviewed by the EPA Science Advisory Board (SAB) on August 18, 2000. The SAB review of the Protocol is available on the internet at: http://www.epa.gov/sab/ec0015.pdf

Not all of the methods described in the Protocol were used in the model-to-monitor comparison included in the NATA Report; some of the methods that were used were modified; and, some new methods were introduced. The decision to diverge somewhat from the methods described in the Protocol was based on the recommendations of the SAB, as well as our own judgment.

Appendix J

Comparison of ASPEN Results to Monitored Concentrations

(Provided in electronic format on Appendices CD)

Appendix J - Summary

Initial National-Scale Assessment
Comparison of ASPEN Modeling System Results to Monitored Concentrations

This appendix describes the results of a model-to-monitor comparison we conducted for subset of the 33 urban HAPs. We view this comparison as a evaluation of ASPEN and he inputs that go into ASPEN, including: emissions data from various sources including the National Toxics Inventory (NTI), the Emissions Modeling System for Hazardous Air pollutants (EMS-HAP), and meteorological data. For most of the pollutants evaluated, we found that ASPEN estimates tended to be lower than the monitor averages at the exact locations of the monitors. In general it appears that ASPEN is underestimating monitor-based HAP concentrations.

Possible reasons for ASPEN to underestimate HAP concentrations include:

1) The National Toxics Inventory (NTI) missing specific emissions sources (for many of the sources in the NTI some of the emissions parameters are defaulted or missing)
2) Emission rates being underestimated in many locations. We believe the ASPEN model itself is contributing in only a minor way to the underestimation. This is mainly due to output from the antecedents of the ASPEN model comparing favorably to monitoring data in cases where the emissions and meteorology were accurately characterized and the monitors took more frequent readings. In simulations we ran, the ASPEN's estimates compared favorably to the estimates derived from a more meticulous model.
3) Monitor siting may have also contributed to the underestimation. Sites are normally situated to find peak pollutant concentrations, which implies that errors in the characterization of sources would tend to make the model underestimate the monitor values.
4) Finally, we are not sure of the accuracy of the monitor averages, which have their own sources of uncertainty.

Our results suggest that the model estimates are uncertain on a local scale (i.e., at the census tract level). We believe that the model estimates are more reliably interpreted as being a value likely to be found within 30 km of the census tract location.

Appendix K

HAPEM4 Results

(Provided in electronic format on Appendices CD)

Appendix K - Summary

HAPEM4 Result Charts

This appendix presents the graphical results of the HAPEM4 exposure modeling Included are:

- Pollutant specific exposure concentration charts
- Pollutant specific percent contribution charts
- State summary exposure concentration estimate charts

In addition, the "Page 2" referred to on each chart has been provided at end of the appendix.

Appendix L

Risk Characterization Results

(Provided in electronic format on Appendices CD)

Appendix L - Summary

Risk Characterization Charts

This appendix contains the risk and population summary charts for carcinogens as well as noncarcinogens. Charts are also summarized by source sector (i.e., major, area, mobile onroad, mobile off-road, background). A set of the Key Risk Assumptions and Limitations is also provided.

TECHNICAL REPORT DATA

(Please read Instructions on reverse before completing)

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16. ABSTRACT

The document describes the EPA's National-Scale Air Toxics Assessment, based on emissions data for 1996. The national-scale assessment is a nationwide study of potential inhalation exposures and health risks associated with 32 hazardous air pollutants (i.e., air toxics) and diesel particulate matter, based on 1996 data. This initial national-scale assessment is one component of the National Air Toxics Assessment (NATA), the technical support component of EPA's National Air Toxics Program. The purpose of the national-scale assessment if to gain a better understanding of the air toxics problem in the U.S. The document explains the methodologies used for the initial national-scale assessment and provides a summary of the results. The document also presents the risk characterization, which includes an uncertainty and variability analysis, and provides an overall summary of results and recommendations for future actions.

17. KEY WORDS AND DOCUMENT ANALYSIS

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