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EPA's Urban Area Source Research Program

- A Status Report on Preliminary Research -

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Acronyms and Abbreviations

BTX	Benzene, toluene, and xylene
CAA	Clean Air Act
EPA	Environmental Protection Agency
FIRE	Factor Information Retrieval system
HAPs	Hazardous Air Pollutants
L&E	Locating and Estimating documents
IARC	International Agency for Research on Cancer
LOAEL	Lowest-Observed-Adverse-Effect Level
MACT	Maximum Achievable Control Technology
PCBs	Polychlorinated biphenyl compounds
PIC	Products of Incomplete Combustion
POM	Polycyclic Organic Matter
RfC	Reference Concentrations
TEAM	Total Exposure Assessment Methods
TSDFs	Waste Treatment Storage and Disposal Facilities
VOCs	Volatile Organic Compounds

Executive Summary

The Clean Air Act (CAA) Amendments of 1990 require the Environmental Protection Agency (EPA) to develop an "Area Source Program" that includes both a Research Program and a National Strategy to "substantially reduce the public health risks posed by the release of hazardous air pollutants from area sources ...." The Research Program is to include three components: (a) characterization of the sources of hazardous air pollutants (HAPs), especially area sources, (b) characterization of the concentrations of HAPs to which people are exposed, and (c) consideration of public health risks from the emitted and transformed HAPs.

The Research Program is intended to support development of the National Strategy. The National Strategy must "identify not less than 30 hazardous air pollutants which, as the result of emissions from area sources, present the greatest threat to public health...." The National Strategy must then propose a strategy to control the sources of the identified pollutants. The strategy must also reduce the incidence of cancer attributable to exposure to HAPs by 75% or more.

This report deals with the Research Program and current research capability to characterize the Emission Sources, the Exposure Concentrations, and the Health Risks due to area source emissions of HAPs. These three areas are discussed in terms of the Environmental Health Paradigm. (See Figure E-1.) This paradigm provides a conceptual framework to describe both the three aspects of the Research Program and the process of risk assessment - risk management under the National Strategy.

There are two primary activities in the Environmental Health Paradigm: exposure assessment and effects assessment. Exposure Assessment evaluates how likely people are to come into contact with HAPs and determines how large their exposure is likely to be. Effects Assessment identifies what health effects are likely to occur once people are exposed to HAPs. In order to understand environmental health issues, it is necessary to have some knowledge about each component of the paradigm.

The current status of information needed for each of the components in the Environmental Health Paradigm for HAPs is discussed. The availability of data to assess the risks potentially posed by each of the 189 HAPs listed in the Clean Air Act was evaluated in three broad categories: (1) characterization of area sources, (2) characterization of exposure concentrations, and (3) characterization of probable health effects. The health effects data were characterized for both non-cancer effects and cancer. In general, a few HAPs in each category had a great deal of data, while many chemicals had little or no data.

Twenty HAPs were found to have "Fair or Better" data available in all three of the categories. (See Table E-1.) This list of chemicals does not identify the 30 or more "worst" HAPs; rather, the list simply identifies those HAPs with sufficient data to begin a risk assessment of either

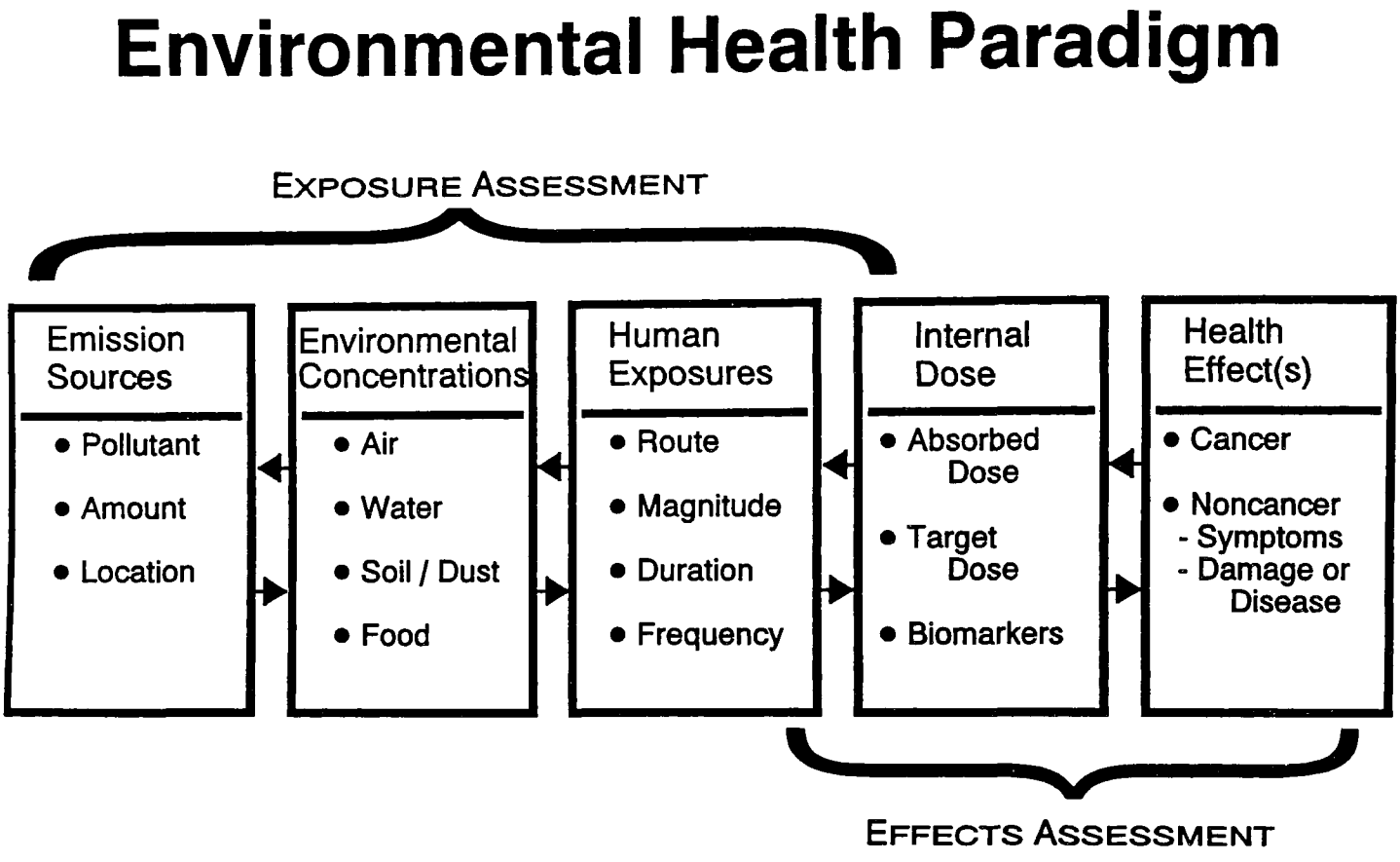

Figure E-1. The components of the Environmental Health Paradigm.

the cancer or noncancer effects due to exposure to that chemical. Another 20 HAPs are rated "Fair or Better" in two of the three required areas. Targeted research on this second group of HAPs could readily provide sufficient data to allow a risk assessment to be initiated. The 40 HAPs with the most complete available data are listed in the Table. The remaining 149 HAPs lacked important data in two or more of the categories. In addition to the 189 listed HAPs, other chemicals, such as those produced by atmospheric transformation, may also be of concern.

As a consequence of these data limitations, risk estimates for many of the chemicals known to be present in urban environments will be very uncertain. Research to overcome or address these data limitations will likely be both expensive and time-consuming. Data for selected chemicals, however, appear sufficient to assess risks and to develop control strategies as warranted.

Table E-1. The HAPs with the most extensive available data needed for evaluation of the Environmental Health Paradigm.

HAPs with data rated "Fair or Better" in the three areas: • Source Emissions • Ambient Concentrations and • Health Effects (Cancer or Noncancer)	HAPs with data rated "Fair or Better" in <u>two</u> of the following three areas: • Source Emissions • Ambient Concentrations and • Health Effects (Cancer or Noncancer)
Benzene	Acetaldehyde
1,3-Butadiene	DDE (p,p'-dichlorodiphenyldichloro-ethylene)
Carbon tetrachloride	1,4-Dichlorobenzene
Chloroform	Ethylbenzene
Ethylene dibromide	Ethylene oxide
Ethylene dichloride	Hexachlorobenzene
Formaldehyde	Hexane
Methylene chloride	Methyl bromide
Styrene	Methyl chloroform
Tetrachloroethylene	Pentachlorophenol
Toluene	Polychlorinated biphenyls
Trichloroethylene	Propylene dichloride
Vinyl chloride	2,3,7,8-Tetrachlorodibenzo-p-dioxin
Arsenic compounds	2,4,6-Trichlorophenol
Chromium compounds	Vinylidene chloride
Lead compounds	Xylenes (mixed isomers)
Manganese compounds	Antimony compounds
Mercury compounds	Beryllium compounds
Nickel compounds	Cadmium compounds
Selenium compounds	Polycyclic Organic Matter

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Section 1
Introduction

The purpose of this report is to summarize what is currently known about exposures to and risks from hazardous air pollutants (HAPs) that are emitted by "area" sources. The Clean Air Act (CAA) Amendments of 1990 require the Environmental Protection Agency (EPA) to develop an "Area Source Program" that includes both a National Strategy and a research program. The law also requires EPA to report the results of its preliminary research efforts. This report describes those preliminary research findings on area source emissions.

Section 112(k) of the CAA[1] mandates that EPA conduct an area source research program "after consultation with state and local air pollution control officials." The law specifies that the research program should contain at least three elements: (1) "ambient monitoring for a broad range of hazardous air pollutants ... in a representative number of urban locations;" (2) "analysis to characterize the sources" of hazardous air pollutants (HAPs), with a focus on area sources and their public health risks; and (3) "consideration of atmospheric transformation ... which can elevate public health risks."

The mandated research program is intended to provide the scientific basis for development of a comprehensive National Strategy to control emissions of HAPs from area sources. The National Strategy must be published by November, 1995, in a report to Congress. It must "identify not less than 30" HAPs that "present the greatest threat to public health in the largest number of urban areas." The strategy is to be fully implemented by the year 2000 and must provide guidelines for controlling the area source emissions of the 30 or more identified HAPs, while simultaneously ensuring a reduction of at least 75% in the "incidence of cancer attributable to exposure to hazardous air pollutants emitted by stationary sources ..., considering control of emissions of hazardous air pollutants from all stationary sources and resulting from measures implemented ... under [the CAA] or other laws."

The area source National Strategy is a key component of the Agency's overall approach to reducing exposure to and risk from HAPs. It is especially important because of the variety and number of sources that might be controlled under this strategy.

Traditionally, scientists and engineers have associated "area sources" with small, but numerous, sources that are likely to be found in any urban area — sources like gas stations, dry cleaners, auto repair shops, and even emissions from cars and trucks. However, the definition of an area source of HAPs in the CAA is different from the traditional meaning of the term. The CAA defines an "area source" as "any stationary source of hazardous air pollutants that is not a major source." In the CAA, a "major" source of HAPs is "any stationary source ... that emits or has the potential to emit considering controls, in the aggregate, 10 tons per year or more of any haz-

ardous air pollutant or 25 tons per year or more of any combination of hazardous air pollutants."[a] An "area source" of HAPs, as defined in the CAA, therefore, is any stationary source of HAPs that emits less than 10 tons per year of any single HAP and less than 25 tons per year of all of the HAPs emitted by that source.

Clearly, the definition of an "area source" of HAPs in the CAA is somewhat different from the traditional definition. Specifically, the definition in the legislation excludes motor vehicles and nonroad mobile sources (which are regulated elsewhere in the Act), while it does include small stationary sources, even though they may not be "numerous" in an urban area.

The National Strategy must address area sources as they are defined in the CAA, rather than the traditional definition. Throughout the remainder of this document, the term "area source" refers to the definition found in the CAA. Other documents, some of which are cited in this report, however, may use the traditional definition. Because the term "area source" may have different meanings in different documents (especially those that date from prior to the CAA Amendments of 1990), readers must be careful to understand what is included as an area source when evaluating other sources of information.

[a] Also note that the CAA defines a "major" source differently when dealing with volatile organic compounds (VOCs), pollutants that help produce ozone pollution. Throughout this document, the term "major source" refers to a major source of HAPs.

Section 2
Hazardous Air Pollutant Assessment

2.1 Overview

Ambient air pollution can contribute to the occurrence and/or aggravation of disease in urban and/or industrialized areas. Diseases associated with air pollution include respiratory diseases (*e.g.*, asthma, bronchitis, and emphysema) and cancer.[2, 3, 4] EPA has conducted a number of "screening" studies to begin to define the contribution of HAPs to this problem in the U.S.[b] The "screening" studies, which are discussed in Section 3, were intended to make broad comparisons of risks for program planning purposes. Such studies typically attempted to define exposures and risks from as many pollutants and sources as possible, although most studies included only 10 or fewer of the HAPs listed in the CAA. Because many assumptions about emissions, exposures, and health effects were commonly made in these studies, the results are generally viewed, at best, as crude approximations of the comparative risks posed to individuals and populations. While the results, typically expressed in terms of cancer risks or potential noncancer effects, are not viewed as representing absolute risks, they provide the best available estimates of the potential magnitude of the broad air toxics problem. Congress clearly considered the results of such screening studies to be relevant when legislating the Section 112(k) area source program, as evidenced by the extensive citations from various House and Senate Committee Reports containing the legislative history of the Clean Air Act Amendments.[5]

[b] Such studies have been conducted in Philadelphia, Baltimore, Kanawha Valley (WV), Los Angeles, Chicago, Santa Clara (CA), Baton Rouge, Phoenix, and a few other locations.

2.2 Environmental Health Paradigm

In order to assess the risks of HAPs, and to manage or control those risks, it is often helpful to consider the interrelated processes of exposure and effects assessment in a conceptual framework, or paradigm. Figure 2-1 illustrates one such paradigm that is especially useful for describing what is known about HAPs in urban air.[6]

Evaluation of potential health risks from exposure to environmental pollutants is composed of two primary activities that make up the Environmental Health Paradigm: exposure assessment and effects assessment. *Exposure Assessment* evaluates how likely people are to come into contact with HAPs and determines how large their exposure is likely to be. *Effects Assessment* identifies what health effects are likely to occur once people are exposed to HAPs. In order to understand environmental health issues, it is necessary to have some knowledge about each component of the paradigm — from *Emission Sources* through *Health Effects*.

Never will EPA have perfect and complete data about all aspects of the paradigm, yet critical decisions about the National Strategy must be

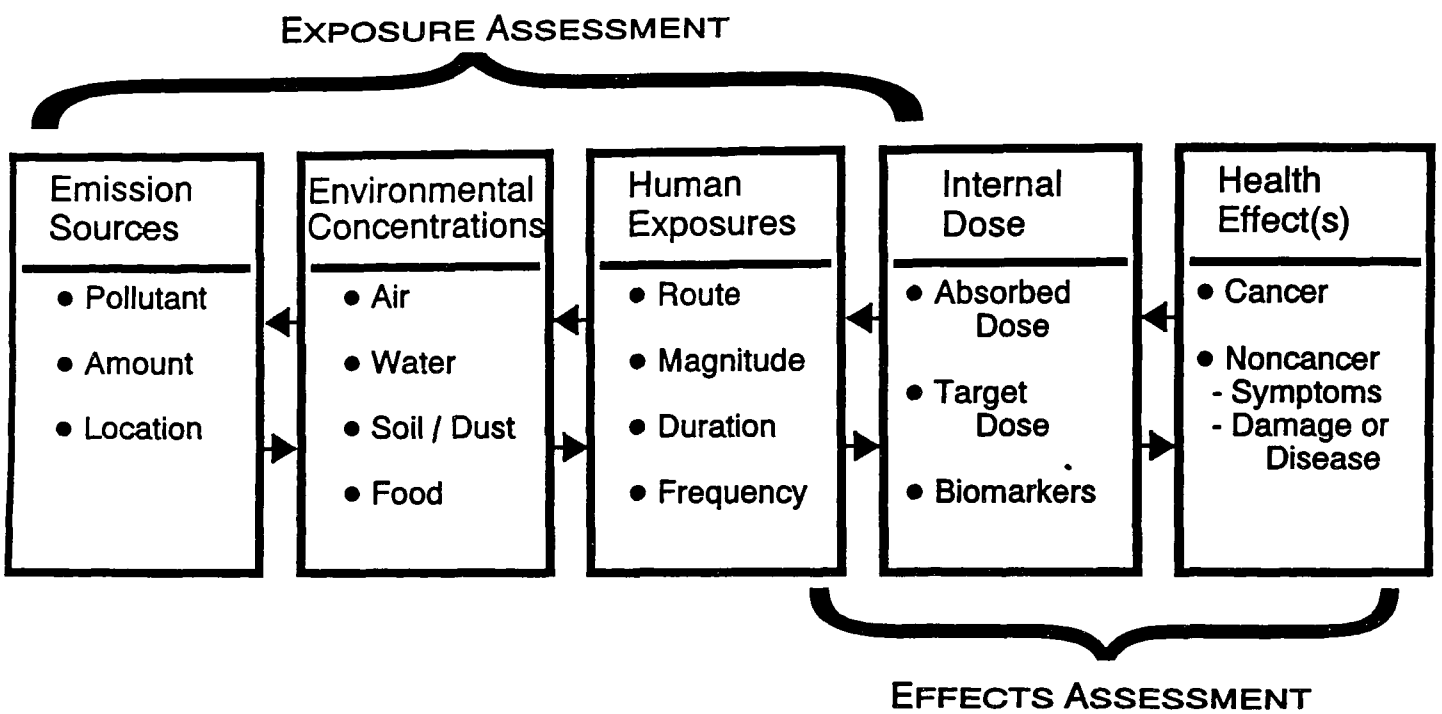


Figure 2-1. The components of the Environmental Health Paradigm and their relationship to Exposure Assessment and Effects Assessment.

made. Often, assumptions about one or more aspects of the Environmental Health Paradigm must be made in order to fill in the data gaps. In some situations, simplifying assumptions might not significantly affect the risk assessment. For other chemicals or locations, the need to make such assumptions might introduce large uncertainties into the assessment. The amount and quality of information needed to evaluate properly each component of the Environmental Health Paradigm will vary from case to case and chemical to chemical.

To assess exposure thoroughly, one must characterize the *Emission Sources*, *Environmental Concentrations*, and *Human Exposure* factors. Knowledge of *Emission Sources* is needed to determine where, how much, and when HAPs are emitted. Critical information includes the types and amounts of pollutants released and the locations of the sources. Once the HAPs are emitted into the air, they are transported and transformed until some of them come into contact with humans. Information about *Environmental Concentrations* is necessary to determine the pollution levels to which people might be exposed. For a

comprehensive assessment, data are needed for all media through which exposure might occur, including air, water, soil, or food. The *Human Exposure* factors consider how people and pollutants come into contact with each other. The goal of the human exposure factor is to define the route, magnitude, duration, and frequency of the contact between humans and HAPs. Exposure is measured as the product of the pollutant concentration and the time during which people are exposed.

Human exposures to HAPs can occur through a variety of routes, in addition to the air that people breathe. Total exposure assessments include estimates for each route. HAPs can deposit out of the air to a variety of surfaces, eventually polluting water, soil, food, and objects around us. Indirect exposures to HAPs can also occur from the food and water people consume, and from the objects that humans touch. Although such indirect exposures can be extremely important in some cases,[c] this report will consider primarily exposures through the air people breathe.

The intent of the final components of the Environmental Health Paradigm is to identify the health hazards associated with HAPs and to define the relationships between exposure, target dose (the dose to the affected organs or biological systems), and health in human populations. This is also known as the exposure-response relationship. The overlap between *Exposure Assessment* and *Effects Assessment*, as shown in Figure 2-1, reflects the interrelationship of these two assessment activities.

For a health effect to occur, HAPs in ambient air first must actually get into the body. *Internal Dose* defines how much of the HAPs that one breathes (or ingests or contacts) actually gets into the body (absorbed dose), and how much gets to the specific organ(s) where they might cause damage (target dose). Significant biologic events resulting from this target dose can be used as measures of internal dose (biomarkers). Absorbed dose, target dose and resulting biomarkers are all critical links between human exposure and consequent health effects. Improving measures of these links improves the estimates of risks posed by HAPs.

Health Effects are often categorized into cancer and noncancer health effects. Historically, one basis for this categorization of health effects is the dichotomous nature of cancer (that is, either you have it or you don't) versus the wider variety of symptoms, damage, or disease associated with noncancer effects. For example, respiratory disorders resulting from exposure to HAPs can range from itching noses, coughing, shortness of breath, decreased capacity to inhale or exhale, bronchitis, increased asthma attacks, emphysema, pulmonary edema and death. More than one effect, like those listed, can often appear together, in varying degrees of severity. Effects in different organs or biological systems also can occur simultaneously. Consequently, *Effects Assessment* must often evaluate a complex set of health effects, with different patterns of affected organ systems and with widely different severity of effects. These patterns are often chemical-specific and change with exposure concentrations, durations, frequency of exposure, and with characteristics unique to the population that is exposed (for example, genetic or gender or age-related characteristics).

[c] Other routes of exposure may be very important in many cases. The Great Waters Program was authorized under Section 112(m) of the Clean Air Act because of deposition of toxic air pollutants to lakes and other bodies of water with subsequent entry into the food chain or drinking water and human exposure by ingestion. Recent National Academy of Science reports on lead discuss human exposure by ingestion of lead-containing particles deposited on food, as well as child ingestion of lead-containing dust. (See, for example, National Academy of Sciences, *Measuring Lead Exposure in Infants, Children, and Other Sensitive Populations*, National Academy Press, Washington, DC, 1993.)

Based primarily on laboratory animal studies and occupational observations, the health effects most commonly associated with HAPs exposures are cancer, developmental and reproductive disorders (for example, retarded development in children or birth defects), neurotoxicity, and short-term and long-term pulmonary disorders.[7, 8]

The components of the Environmental Health Paradigm also provide a reasonable way to summarize the current understanding of HAPs in urban air. The following discussions will focus on the *Exposure Assessment* and the *Effects Assessment*.

2.2.1 Exposure Assessment

The first component of the Environmental Health Paradigm is Exposure Assessment. In this section, we consider each of the components of Exposure Assessment:

- Emission Sources
- Environmental Concentrations
- Human Exposure.

2.2.1.1 Emission Sources

Reliable data on emissions of HAPs from area sources are limited. Most previous studies of emissions in urban areas have focused primarily on criteria pollutants or their precursors, such as volatile organic compounds (VOCs), particulate matter, sulfur oxides, and nitrogen oxides, not on the 189 chemicals listed as HAPs. Furthermore, previous studies focused primarily on *all types* of sources (major point sources, mobile sources, and area sources), not just area sources. Emissions (*e.g.*, tons of pollutant per year) from area sources may have not been included in such studies, and even if they were included, the data may not allow a complete and accurate emissions inventory to be assembled.

Deficiencies in emissions data might involve any of the various aspects of Emission Source characterization, including describing the type of pollutants, quantifying how much of the HAP is released, or locating the sources geographically. Data available under the Toxic Release Inventory are very useful in locating potential releases of HAPs from many major sources, but similar data are not available for the smaller area sources. Nonetheless, some area sources of HAPs are well defined, and a great deal of data are available for area sources like residential wood combustion, dry cleaners, and publicly-owned treatment works. Aside from such sources, however, emission inventories have traditionally focused mostly on major sources of VOC emissions (some of which are also HAPs) or on sources of criteria pollutants (for example, sulfur oxides, particulate matter, and nitrogen oxides). In many cases the exact HAPs and the concentrations that are emitted from small sources are not well known. In many inventories, emissions from small area sources are not located or measured precisely, but are estimated from indirect measures like the number of people in an area, the number of cars, and the quantity of solvent sold.

Efforts are underway to reduce the uncertainties in emissions inventories for a number of important HAPs. EPA is continuing to develop improved tools for use in developing HAP emission inventories. "Locating and Estimating" ("L&E") reports are available for more than 30 HAPs. These reports contain pollutant-specific information on industrial processes, emission factors (*e.g.*, pounds of pollutant emitted per ton of fuel burned), source test methods, and in the recently updated reports, national inventories, including emission estimates for point, area and mobile sources. Thirteen "L&E" reports were developed or upgraded in fiscal year 1993, and seven additional updated reports are anticipated for 1994. In addition, the Factor Information REtrieval system (FIRE) contains evaluated emis-

sion factors for both criteria pollutants and HAPs. FIRE is updated periodically and now contains 9700 rated emission factors, of which approximately 4000 factors are available for 29 of the listed HAPs.

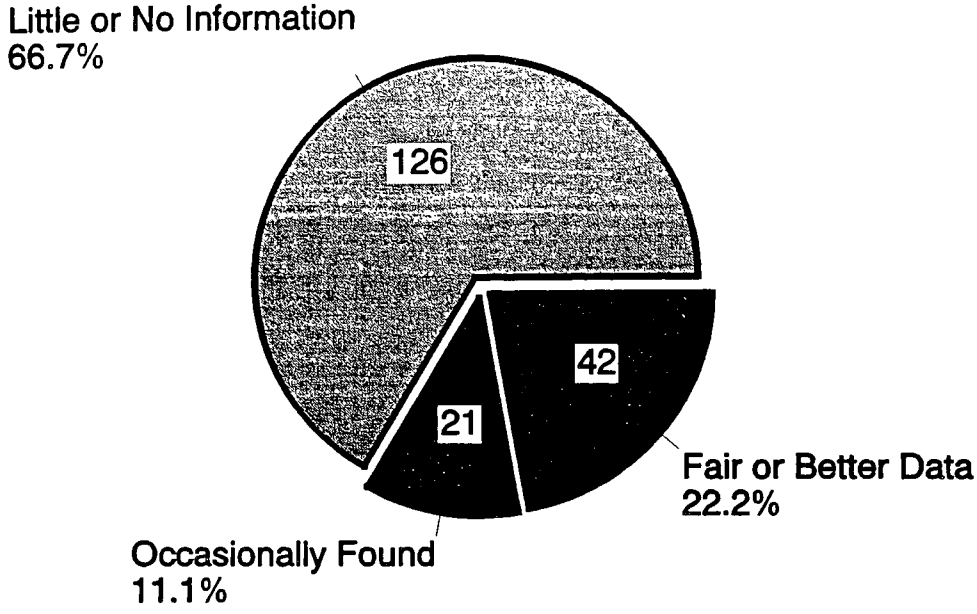
Even when data on emissions are available, there are still uncertainties involved in extrapolating the data to other locations or to other operational conditions. To ensure the development of reliable emission factors, one must measure the emissions at a variety of sources in a specific category and must collect sufficient data on plant operations, processes, and conditions. Obtaining reliable emission factors is expensive even when the source is not difficult to test and reliable measurement techniques are available. Under California's Assembly Bill 2588 program (the "Hot Spots" Program), many producers of HAPs are required to conduct such tests at their own expense. EPA has used the data from California to extract more than 1500 HAP emission factors, and is implementing a project to obtain source test data from other state and local agencies.

A number of state or local air pollution control agencies have voluntarily developed inventories[9] of HAP emission sources over the last decade, despite the lack of a federal requirement for HAP emission inventories. Without specific guidance about such inventories, the state and local agencies have chosen to include different HAPs in their inventories and to use a wide variety of methods to estimate emissions. Consequently, there is often very little consistency between the available inventories. Nonetheless, efforts are underway both at the federal and state levels to overcome some of the shortcomings found in the inventories and to reduce the inconsistencies. As mentioned above, the California "Hot Spots" Program has proven to be very productive in providing better emissions data. In addition, the eight member states of the Great Lakes Commission are working together to develop a regional emissions inventory for mobile, area, and point source emissions of 49 HAPs. Additional data on source emissions should become available as states implement the permit programs as required by the Clean Air Act Amendments of 1990.

On a national scale, EPA has also supported national HAP emission inventories for fourteen HAPs (and related species) in 1993. These HAPs included mercury, alkylated lead, hexachlorobenzene, POM, polychlorinated biphenyls (PCBs), tetrachlorodibenzodioxin, tetrachlorodibenzofuran, benzene, 1,3-butadiene, carbon tetrachloride, tetrachloroethylene, trichloroethylene, methylene chloride, and formaldehyde. These national inventories include estimates for mobile, area and point sources and are allocated to the county level. Although such inventories do not precisely locate all sources of HAPs, they can still provide valuable information for estimating urban emissions of HAPs.

Efforts to assemble emission inventories have been identified for a variety of urban sources, including area sources, for more than 60 HAPs, but fewer than 20 of the HAPs appear with regularity (that is, in 50% or more of studies) in the detailed inventories compiled by state and local agencies.[10] Emissions of other HAPs can be estimated on the basis of national inventories, or might be computed from available emission factors. Figure 2-2 illustrates the availability of emissions data for the 189 listed HAPs. Forty-two HAPs (seventeen HAPs that appear in 50% or more of the state and local inventories, together with an additional twenty-five HAPs that appear in the FIRE data base or that are included in national inventories) are categorized as "Fair or Better." HAPs that appear infrequently (less than 50% of the time) in detailed inventories are listed as "Occasionally Found." There are little or no emissions data for more than 120 HAPs.

Availability of Source Emissions Data For the 189 Listed HAPs**



** Based on frequency of inclusion in state or local inventories, data availability in the FIRE data base, or the availability of a national inventory.

Figure 2-2. Summary of the available data on emissions of HAPs from all source types. (Table A-1, Appendix A, categorizes the data for each of the 189 HAPs.)

Two major approaches can be used to identify how much of urban pollution comes from the area sources: dispersion modeling and source apportionment.

If the emissions from all sources are well known, the contribution from area sources to ambient concentrations of HAPs can be estimated. The estimates for area sources may then be compared with the contributions from all other types of sources, through dispersion modeling. Dispersion models describe how the emissions mix in the atmosphere and are distributed throughout the urban area. However, there are serious shortcomings in the current emission inventories for urban areas with regard to area source emissions of HAPs, as previously noted. These shortcomings bring into question the reliability and accuracy of the dispersion modeling approach.

The second approach, source apportionment, uses ambient monitoring data to estimate how much of the pollution came from each source. This approach works best when each source (or source category) contributes substantially to the total pollution in a unique and distinctive way.

Such is not the case, however, for many sources. For example, benzene, toluene, and xylene (often referred to jointly as BTX) are frequently the HAPs with the highest concentrations in urban air. It would be very useful to know what fraction of BTX in air was due to area sources. Two recent apportionment studies[11, 12] found that 85–95% of the BTX in urban air came from mobile sources. With such a large and dominant source, apportionment of the small remaining fraction of BTX from area sources will prove very difficult to assign to specific area sources or source categories.

While both dispersion modeling and source apportionment methods have their limitations, they can be used together to complement the strengths and weaknesses of each approach.

It is important to understand the impact of area sources on human exposure and risk, even if their emissions are small compared to the total quantities emitted by all sources. Exposure and risk is not necessarily proportional to the magnitude of the emissions. This is especially true if the area sources (and other sources, like indoor sources or sources from personal habits) are much more closely linked with human activities, because such sources could still dominate the resulting risk since they could contribute disproportionally to human exposure.

2.2.1.2 Environmental Concentrations

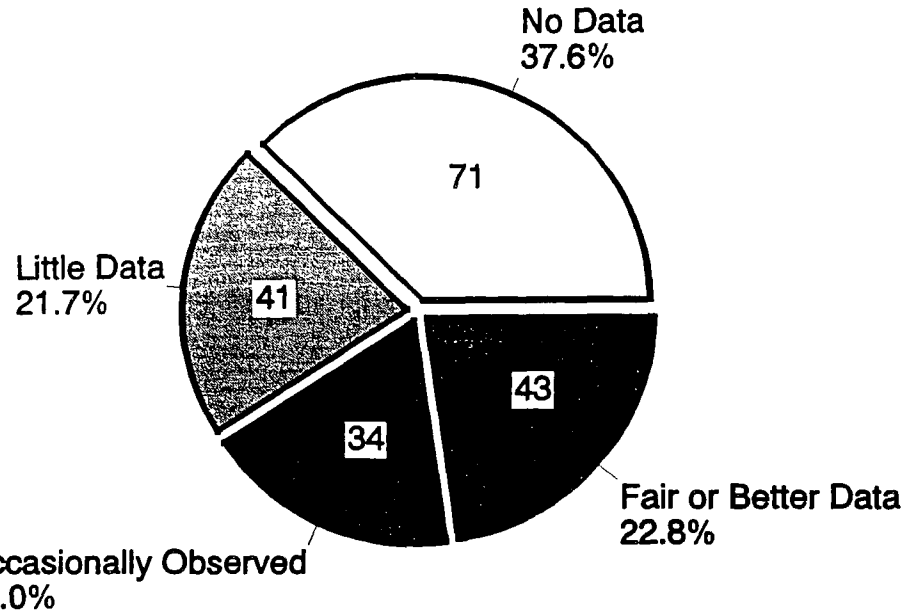
The availability of data on ambient outdoor concentrations of the 189 HAPs is highly uneven (Figure 2-3). The ambient outdoor concentrations result from emissions from all types of sources, including point, area, and mobile sources. The figure plots the total number of HAPs that have been measured versus the number of times they have been measured in outdoor air in populated areas.[13] There are little or no ambient measurement data (fewer than 100 observations) for nearly two-thirds (112) of the HAPs, while a few chemicals — notably benzene, toluene, and the three xylene isomers — have each been measured many thousands of times. For 71 of the 189 HAPs (38%), there are no ambient measurements at all. The 43 HAPs with "Fair or Better" data all have more than 1000 observations. (An "observation" is one or more measurements at the same location within a 24 hour period.) Clearly, there is little or no information about a large number of HAPs, but a great deal of information about a smaller number of HAPs.

The same conclusion (very little data for most HAPs; considerable data for some HAPs) also extends to the number of cities for which ambient outdoor concentration data are available. Nearly two-thirds of the listed HAPs have been measured at fewer than 5 cities or towns, while BTX data are available for more than one hundred cities. Figure 2-4 illustrates just how few HAPs have been measured at an adequate number of cities. Additionally, the data are often available only for short periods of time — a few days or weeks — while special studies were underway. Long-term collection of data on HAPs is available for only a very few cities.

When two-thirds of the designated HAPs have been measured only a few times and at only a few cities, the "representativeness" of the ambient outdoor data becomes an important issue. Even the data that are available are of inconsistent quality and duration. When large data gaps exist, either in space or time, it is very difficult to estimate human exposures and potential health effects reliably, or to identify trends in order to characterize the impacts of regulatory programs.

Table 2-1 lists typical outdoor concentrations of a few HAPs[13] that are among the best-studied in terms of health effects. As discussed earlier, the actual ambient outdoor measurements are often variable; nevertheless, these concentrations

Availability of Ambient Outdoor Concentration Data For the 189 Listed HAPs



The categories are based on the number of reported observations, as described in the text. HAPs with No data have 0 observations; HAPs with "Little Data" have <100 observations. The "occasionally observed" HAPs have 100-1000 observations, and HAPs with "Fair or Better Data" have been observed more than 1000 times.

Figure 2-3. Summary of available data on ambient outdoor concentrations of HAPs. (Table A-1, Appendix A, categorizes the available data for each of the 189 HAPs.)

are typical of the reported data. Median concentrations, in micrograms per cubic meter ($\mu g/m^3$), are listed in the table. The median is the middle of the distribution of observed concentrations: half of the time, the measured concentrations were larger than those listed, and half of the time, the concentrations were reported to be smaller. "Average" concentrations are not given since an arithmetic average can sometimes be misleading, especially if there are a few very large concentration measurements or if there are many observations with concentrations too small to measure accurately. There are major differences between the number of times and number of locations in which the various chemicals have been measured.

2.2.1.3 Human Exposures

To develop the National Strategy to minimize adverse health effects from area source emissions of HAPs, it is necessary to consider the actual human exposure to the HAPs, not merely the ambient concentrations. The following text describes what is currently known about the distribution of HAPs across urban areas and about the impact of outdoor air on indoor air and personal exposures.

Cities and Towns with Ambient Outdoor Data For the 189 Listed HAPs

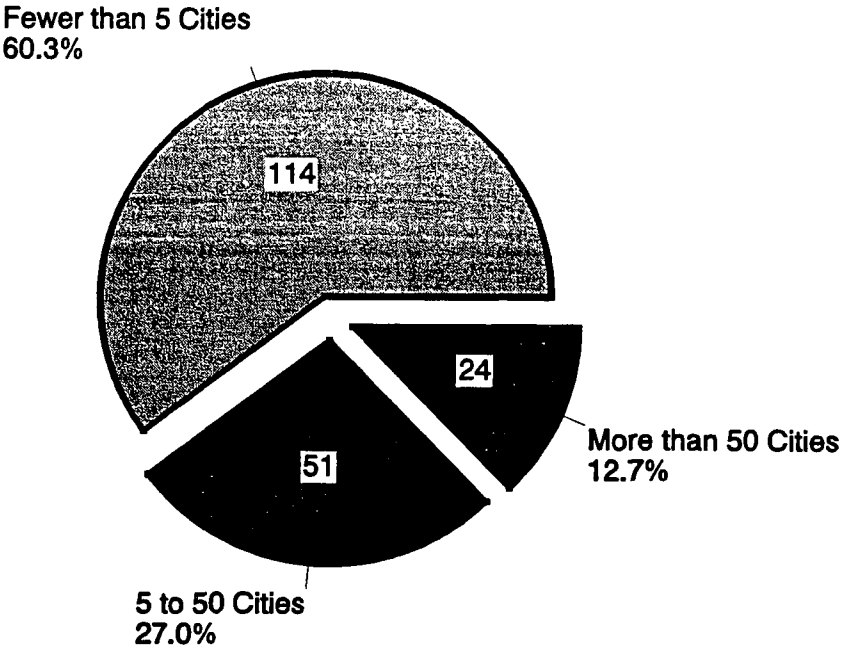


Figure 2-4. Summary of the number of HAPs that have been measured in a variety of U.S. cities or towns.

Distribution

To estimate human exposure to HAPs one must know how widespread are the concentrations of urban air pollutants. If area sources are uniformly and widely distributed across an urban area, one would expect the concentrations of the emissions to be relatively consistent across the community, although this is not always true. Only a few studies have included simultaneous measurements of pollutants at different sites across an urban area. One recent study, focusing on the particle-bound pollutants from residential wood burning and from automobiles, found the concentration of fine particles from these sources to be relatively consistent across an urban area.[14] Other studies measuring gaseous HAPs and other vapor-phase pollutants have found that a few gaseous pollutants appear to have relatively constant concentrations[15] across distances as large as 10 km, implying that the sources of those pollutants are widely and uniformly distributed throughout the community.[11] However, in the same study, a larger group of gaseous pollutants were reason-

Table 2-1. Typical median ambient outdoor concentrations of some of the 189 listed HAPs. Concentrations are in micrograms per cubic meter ($\mu g/m^3$) of air.

Chemical Abstract System Number	Chemical Name	Median Concentration $\mu g/m^3$	Number of Observations*	Number of Cities with Ambient Data
71-43-2	Benzene	5	>8600	172
108-88-8	Toluene	9	>6500	159
95-47-6 108-38-3 106-42-3	Xylenes (o-, m-, p- isomers)	2 to 4	>5700	> 130
75-09-2	Methylene chloride	0.5	> 3400	86
67-66-3	Chloroform	0.2	> 4900	135
106-99-0	1,3-Butadiene	0.4	> 1900	66
71-55-6	Methyl chloroform	2	> 4900	155
56-23-5	Carbon Tetrachloride	0.8	> 6300	149
50-00-0	Formaldehyde	3	> 2500	75
Not applicable	Chromium compounds	0.003	> 1800	> 192
57-74-9	Chlordane	0.02	> 345	> 8

* An observation is one or more measurements taken within the same 24-hour day.

ably constant only across distances of about 1 km, while still other pollutants were even more variable.

Impact of Outdoor Air

People typically spend more than 80% of their time indoors,[16] so any analysis of the health effects from exposure to area sources must assess the penetration of the area-source pollutants from outdoors to indoors. Many of the volatile HAPs are stable chemicals that do not react quickly with other chemicals in the environment. Such stable gaseous pollutants can easily penetrate indoors with little or no loss of concentration. The instantaneous indoor and outdoor concentrations can be different, however, due to delays caused by the rate at which outdoor air enters the building — the air exchange rate.[d]

For time periods longer than a few hours, the average indoor concentration of stable gaseous pollutants generated by outdoor sources (including area sources) is identical to the outdoor concentration adjacent to the house (for example, "on the front porch.")[17]

Some HAPs (for example, most POMs) are not volatile vapors; instead, these HAPs are attached to small particles in the air that people breathe. Non-volatile HAPs that are emitted by chemical or combustion processes are often bound

[d] Conversely, reactive pollutants, for example ozone, are readily destroyed as they penetrate indoors resulting in indoor concentrations that are generally less than outdoor concentrations. Few of the listed HAPs are expected to be so reactive.

to "fine" particles (less than 2.5 micrometers in diameter). Such particles are only partially removed as the air penetrates indoors. The number of particles that successfully penetrate indoors is roughly proportional to the air exchange rate. Thus, the more air brought indoors, the more the concentration of particles in the indoor air is like the concentration of particles outdoors. For many buildings, the air exchange rate is large enough to permit about 50%-90% of the outdoor fine particles to penetrate indoors successfully. Non-volatile HAPs found on particles that are generated by mechanical processes (like dust kicked-up by automotive traffic, wind-blown dust, and construction projects) are usually bound to larger particles that are much less likely to penetrate indoors.[17]

Finally, indoor sources, workplace sources, and personal activities can provide additional exposures to HAPs, beyond those due to the outdoor sources. The outdoor sources provide a baseline of exposure to HAPs, on top of which indoor sources, workplace sources, and personal activities add additional exposures. If such indoor, workplace, or personal sources are large, they can dominate the total exposure calculation for those exposed individuals. These sources must be taken into account when determining the total human exposure to HAPs.

2.2.1.4 Complicating Factors

There are a number of factors that make Exposure Assessment a difficult and complex task. Two factors that make identifying and characterizing the urban area sources of HAPs difficult are: the complexity of urban air pollution, and uncertainty in defining area sources.

Urban air is a complex mixture of thousands of chemicals. These chemicals come from a wide variety of sources, including major point sources, area sources, mobile sources, and natural sources. Examples of natural sources of HAPs include forest fires, plant decay, and weathering of minerals containing heavy metals. The objective of the urban area source research program is to characterize the exposures and health risks due to *area* sources in support of the mandated National Strategy. But once the pollutants from the area sources have mixed with those from major point sources, mobile sources, and natural sources, it is extremely difficult to identify how much of a specific pollutant came from just the area sources.

Even the definition of an area source under Section 112 adds a complicating factor. For purposes of the HAP National Strategy, area sources also include point sources that do not meet the requirements to be classified as major sources. These "non-major point" sources have not traditionally been considered as area sources, and were not previously included in efforts to characterize area sources. "Major" sources are defined as part of the Maximum Achievable Control Technology (MACT) standard setting process under Section 112(d): sources that do not meet the requirements for MACT standards are by default "non-major point" sources, or area sources. (Some source categories that include individual sources that are likely not to meet the definition of a major source are: bulk liquid (*e.g.*, gasoline) terminals, electric arc furnaces/stainless steel mini-mills, wood furniture manufacturing, secondary lead smelters, *etc.*) The final MACT standards are not scheduled for promulgation until November 2000. Additional area sources might be added for consideration, long after the National Strategy has had to go into effect.

Other factors make characterization of ambient outdoor concentrations of HAPs a difficult undertaking. For example, measurement methods are not available for many HAPs, and natural reactions in the atmosphere can either destroy or produce HAPs.

Measurement Methods

One reason for the lack of data on both emissions and environmental concentrations of many of the HAPs is that there are often no reliable methods to collect and measure these chemicals. Measurements at the source and in ambient air are often made under distinctive conditions that make such measurements difficult. For example, source measurements often have high concentrations of contaminants and harsh conditions that make sampling and analysis difficult: ambient samples contain very small amounts of the species of interest and must be concentrated to be detected reliably. Validated source sampling methods exist for only 87 of the HAPs. In ambient air, there is one group of HAPs where there is a particularly noteworthy lack of data. These compounds, nitrogenated or oxygenated organics, are often referred to as "polar" organics, and they comprise 89 of the 189 HAPs. Only about one-third of these polar organics have actually been measured in ambient air.

Atmospheric Transformation

Another difficulty with evaluating HAPs in urban air is atmospheric transformation. Natural atmospheric events cause chemical reactions that can both destroy and create HAPs. These transformation processes will eventually break down and remove some of the HAPs from the air. Conversely, transformation processes might convert non-hazardous pollutants into dangerous products (or even transform HAPs into products that are more hazardous than the original HAPs.) The HAPs formaldehyde, acetaldehyde, acetone, and acrolein, for example, are all produced in significant quantities in urban air[18] by the atmospheric transformation of many organic compounds — including many compounds not on the list of HAPs. In other words, transformation processes can produce a HAP even when one was not emitted. This is similar to the situation with ground-level ozone, which is produced primarily through transformation of other pollutants, even though it is not directly emitted.

Many of the most important of these atmospheric transformation processes involve sunlight. Sunlight, shining on polluted urban air, sets into motion a complex series of chemical reactions that convert the directly emitted pollutants into an even more complex "soup." It is not possible to identify all of the chemicals in the resulting product mixture, but studies over the last decade suggest that the sunlight-transformed mixture might be even more hazardous than the originally emitted pollutants. As an indicator of this potential for increased hazard, the bacterial mutagenicity — the ability to cause changes in the genetic material of bacteria — of the transformed mixture is often much greater than that of the original pollutants. This increase in mutagenicity is especially true for the gaseous products, which are likely to be the partially-oxygenated or -nitrogenated transformation products of the emitted chemicals. Figure 2-5 shows the dramatic increases in bacterial mutagenicity brought about by sunlight in two complex pollutant mixtures that are often found in urban air, namely wood smoke and automobile exhaust.[19,20]

The data in Figure 2-5 are from smog chamber simulations of atmospheric reactions, but at concentrations higher than those normally found in the environment: such simulations are necessary, since the mutagenicity tests are not sufficiently sensitive to measure such changes in actual urban air. Indirect evidence, however, suggests such transformation effects do occur in ambient outdoor urban air. A variety of simulations by researchers around the world, involving many of the pollutants commonly found in urban air, have demonstrated several important facts about the mutagenic products of atmospheric transformation:

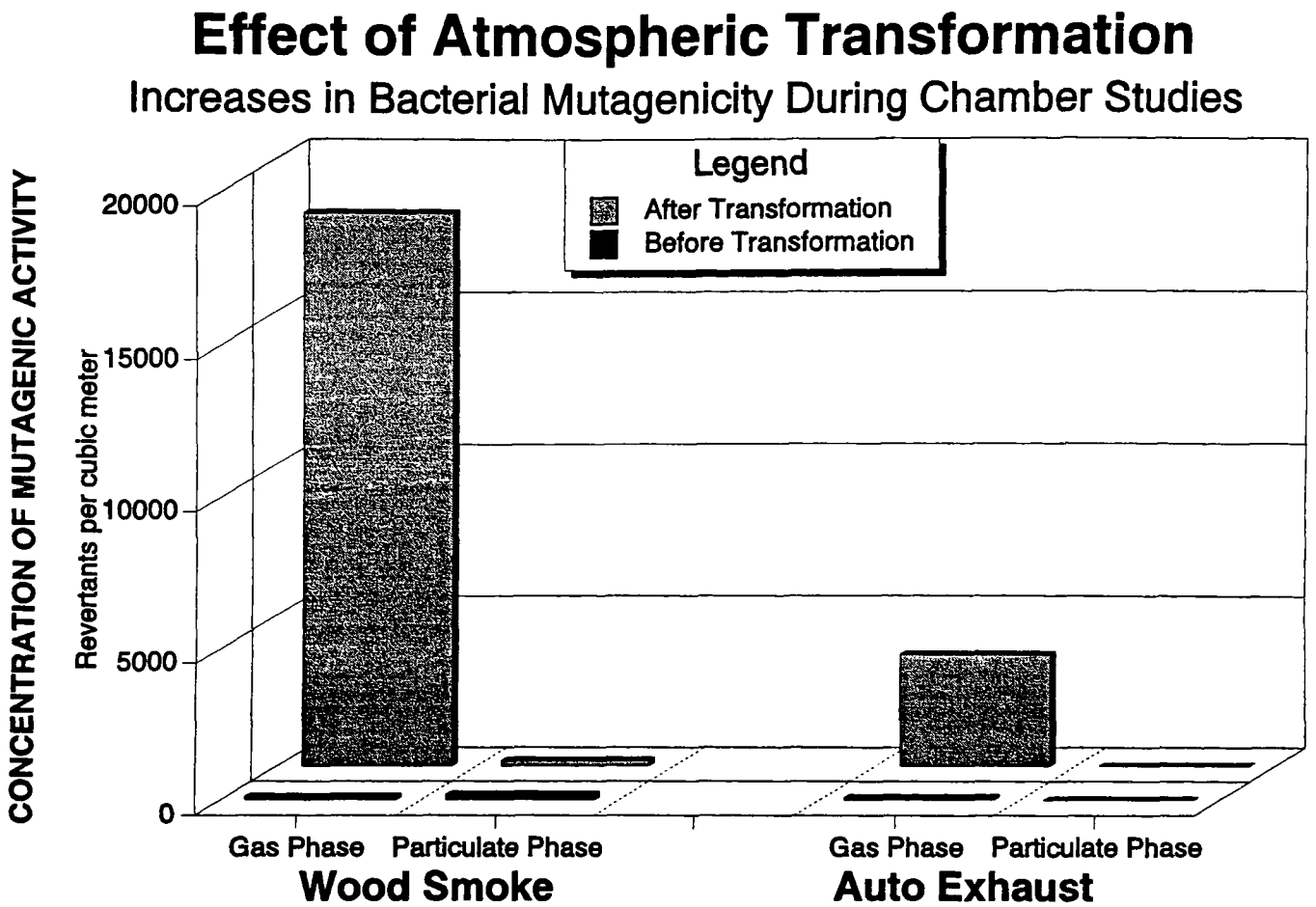


Figure 2-5. Effects of photochemical reactions on the mutagenicity of wood smoke and auto exhaust, two common pollutant sources in populated areas. (Mutagenicity was measured using two different bacterial reversion assays.)

- Sunlight transforms many, but not all, urban pollutants into both gaseous and particle-bound mutagenic products.

- The gaseous mutagenic transformation products are persistent: in the laboratory simulations, they are stable in the air for hours after they are produced. If they are produced and are stable under ambient conditions, then exposures can occur over large areas and for long times.

- About 90% (by mass) of organic chemicals in urban air are gaseous, with only about 10% bound to particles. In the laboratory simulations, the total mutagenicity of the gaseous transformation products in the air greatly exceeded the total mutagenicity of the particle-bound products in the same volume of air. The relative risk from gaseous mutagens versus particle-bound mutagens is unknown.

These data on mutagenicity taken together cause concern about the potential impact of atmo-

spheric transformation on cancer risks in urban areas. If transformation of non-hazardous air pollutants can cause a substantial cancer risk in urban areas, it will make it difficult to develop a National Strategy that can reduce cancer risks by 75%, as required by law.

Exposure Variabilities

One factor that complicates efforts to estimate human exposure is the fact that people and air pollutants move around throughout the day. What people do, and where they are, and when they are at a specific location all affect their exposure. Available exposure or concentration data often do not describe well the extremes in exposure, either very large or very small exposures. People who live very close to a source (for example, in an apartment above a dry cleaning business, or near industrial or gasoline-handling facilities) can be exposed to abnormally high concentrations of specific HAPs. In addition, both people and air pollutants move about during the day. As people move in and out of polluted areas, their exposures can change. Time-activity patterns are descriptions of: 1) where people are throughout the day, 2) how long they remain in each location, and 3) what activities they are doing that can influence exposure (for example, jogging in a park will cause a person to inhale more air and more pollutants than will reading a book on a bench in the same park). Clearly, where a person is during the day and what he or she is doing can significantly affect that person's exposure to HAPs. Only with information on the time-activity patterns of the population relative to the sources of HAPs is it possible to characterize accurately the exposures of people at the high end of the range of exposures — the very people who are most likely to be at risk. Some studies, like the Total Exposure Assessment Methodology (TEAM) studies[21, 22] or the planned National Human Exposure Assessment Survey (NHEXAS),[23] have a statistical approach that is designed to measure a wide range of exposures. Such studies are extending the understanding of the range of potential human exposures, but such statistically based studies are very expensive to conduct and difficult to analyze.

2.2.2 Effects Assessment

The second major aspect of the Environmental Health Paradigm is Effects Assessment. Effects Assessment is concerned with what happens to human health once someone is exposed to HAPs. There are three components of Effects Assessment: Human Exposure, Internal Dose, and Health Effects. Since Human Exposure is also a part of Exposure Assessment and was described earlier, the following describes the remaining two components of Effects Assessment:

- Internal Dose
- Health Effect(s).

2.2.2.1 Internal Dose

The term "Internal Dose" is often used to convey a variety of concepts. In the current context it means the estimation of the amount of HAP that enters the body and reaches an organ or system where it might cause damage to human health. Ambient air concentrations of HAPs have often been used as surrogates for Internal Dose. However, this practice can result in either over- or under-estimations of risk. Ambient concentrations are not always reliable indicators of internal dose because biological and biochemical processes, such as absorption into the body, distribution in the body, metabolism, and excretion, all affect how much of the HAP concentration in the air actually reaches the organs or physiological systems where the pollutants might cause damage. For particle-bound HAPs, even the physical characteristics of the pollution may be important. Particle size and the nature of the particles on which the HAPs are carried may strongly influence the location in the body where the HAPs are deposited, the mechanism by which adverse ef-

fects may occur, the distribution of the pollutant within the body, and the internal persistence of the pollutants. It is important, therefore, to estimate Internal Dose as precisely as possible. The more accurate this estimation, the more accurate will be the assessment of potential HAP risks.

The use of Internal Dose is particularly valuable when human risk estimates are derived from animal laboratory experiments or occupational studies. (HAP risk assessments are almost always derived from these types of data [see discussion of extrapolation of health effects data in the discussion of complicating factors that follows]). New techniques are now being developed that allow for better estimates of Internal Dose. Some of these techniques are: measurements of biological and biochemical processes (pharmacokinetics); use of alternative and more relevant surrogates (biomarkers) of Internal Dose; and actual measurement of the HAPs at the affected tissue (molecular dosimetry). Scientific groups such as the National Academy of Sciences and EPA's Science Advisory Board have encouraged the use of improved estimates of Internal Dose in risk assessments. Unfortunately, reliable information on Internal Dose is currently available for only a few HAPs, and development of such information is currently expensive, slow, and laborious. Through experience with available methods, and through research to improve methodology, the costs to obtain better estimates of Internal Dose will, undoubtedly, decline over time, and improved estimates will become more and more available.

2.2.2.2 Health Effects

There are some toxicity data available for each of the 189 HAPs. In almost no case, however, are data available on all of the most important health effects: cancer, developmental and reproductive disorders (birth defects), neurotoxicity, and acute (short-term) and chronic (long-term) pulmonary effects. Moreover, the quality of the available studies is highly variable. Some studies are barely adequate, others are excellent. Another problem is the lack of data on toxicity associated with exposure by inhalation. Much of data on health effects comes from tests involving only ingestion of the HAP (commonly called oral exposure). However, it is known that differences in the route of exposure can produce major differences in the character and extent of toxicity. Relying on only ingestion data alone generally results in large uncertainties in the prediction of health effects.

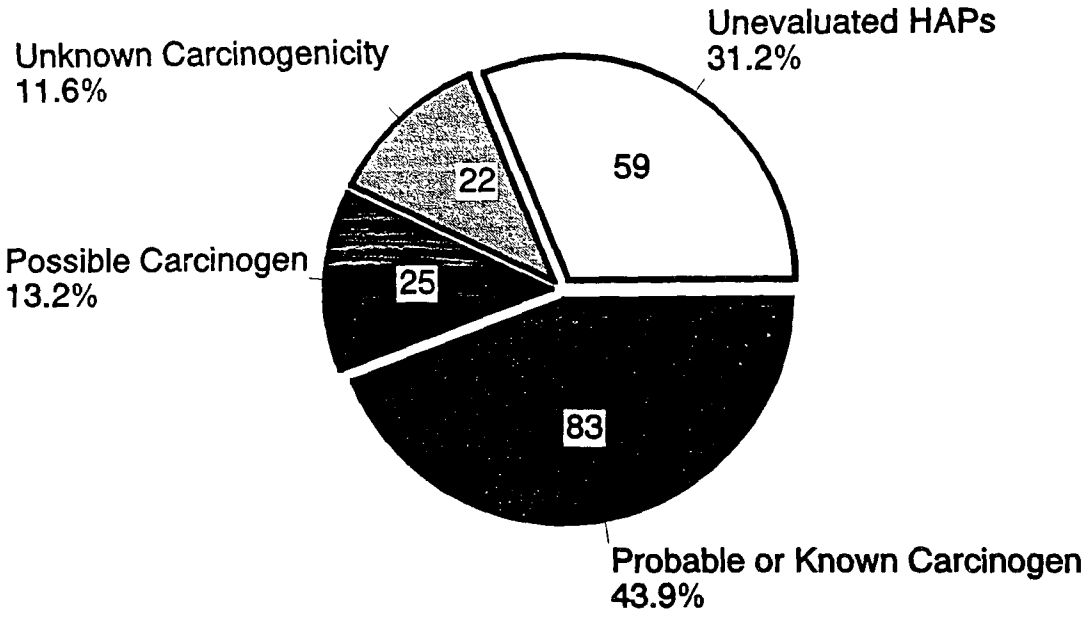
Cancer

A serious possible health effect of HAPs is their potential to cause cancer. More than 100 of the 189 HAPs have sufficient data to assess their ability to cause cancer qualitatively:[24] even for these chemicals, however, a quantitative estimate of the dose-response relationship (potency) is not always possible. Chemicals are classified based on a variety of factors such as the quality of the studies, the number of studies, and the species reported to have chemically induced cancer. Both human and animal data are considered. Eighty-three of the listed HAPs are considered to be "probable" or known human carcinogens. Another 25 HAPs are considered "possible" human carcinogens. (See the classification definitions in the glossary. *N.B.*, EPA is revising its guidelines for carcinogen risk assessment and the definitions are expected to change.) Twenty-two of the HAPs lack sufficient data for a classification, while the remaining 59 of the HAPs have not been evaluated for carcinogenicity.[c] The carcinogenicity data are illustrated in Figure 2-6.

Noncancer Effects

For noncancer health effects, a Reference Concentration (RfC) is used to estimate an exposure concentration that is not harmful. An RfC is an estimate, based on a single critical effect, of

Availability of Carcinogenicity Data For the 189 Listed HAPs



IARC carcinogens and Class A & B carcinogens are listed as "Probable or Known." Class C chemicals are listed as "Possible." Class D chemicals are classified as "Unknown." The remaining chemicals have not been evaluated.

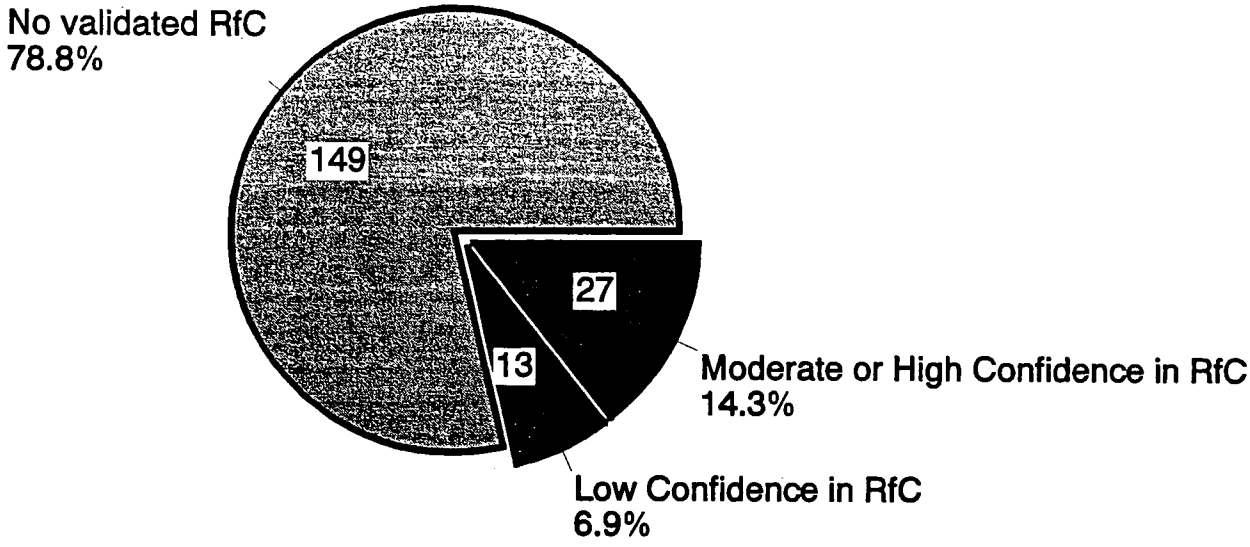
Figure 2-6. Evidence of carcinogenicity of the HAPs. (Table A-1, Appendix A, categorizes the data for each of the 189 HAPs.)

the concentration (with uncertainty spanning a factor of 10) that could be inhaled for a lifetime with no adverse health effects. Only 40 of the 189 HAPs have sufficient data to support estimation of an RfC. Confidence levels for an RfC vary from unknown to low to high as illustrated in Figure 2-7. Only five of the RfCs have "high" confidence. Most of the 149 HAPs without a validated RfC have not been studied for chronic inhalation effects at all.

Some of the chemicals on the list of 189 HAPs are also of concern to EPA because of their potential to cause serious, immediate health effects if people are exposed to very large concen-

[e] Some of the listed HAPs are actually groups of compounds, and data may exist on several different chemicals within a single HAP definition. For example, nickel compounds are generally considered to be "possible" carcinogens, but nickel subsulfide and nickel refinery dust are "known" human carcinogens. In such case, the HAP (nickel compounds) was classified at the higher risk level ("Probable or Known") for tabulation in this report. Similarly, Polycyclic Organic Matter (POM) was classified as a "probable" carcinogen on the basis of some specific compounds (for example, benzo(a)pyrene) that often occur in POM.

Availability of Noncancer Effects Data For the 189 Listed HAPs



- Based on the level of confidence placed on the calculated Reference Concentration (RfC), the concentration that could safely be inhaled for a lifetime with no harmful noncancer health effect.

Figure 2-7. Availability of validated Reference Concentrations (RfCs) for the 189 listed HAPs. RfCs are available for more chemicals, but several are grouped under a single listed HAP. (See Table A-1 for data on each chemical.)

trations. Exposures to large concentrations might occur, for example, after an industrial accident that releases large quantities of a chemical. An accepted method for describing the relationship between dose of pollutants and the biological effects (the dose-response) due to large, short-term exposures to these chemicals is currently under development by EPA. Data on short-term (acute) effects are critical to EPA's Accidental Release Program which is also mandated in the Clean Air Act.

2.2.2.3 Complicating Factors

Several factors make Effects Assessment — evaluation of health effects from exposures to HAPs in urban air — a very difficult task. Three complicating factors are discussed below: (1) extrapolation of health effects data, (2) exposure to complex mixtures of environmental pollutants, and (3) the fact that chemicals other than those on the list of 189 HAPs can pose hazards to human health.

Extrapolation

Most of the HAPs data available must be interpreted in some manner in order to assess public health risks. The most common types of interpretations involve the following: 1) using animal effects data to predict effects that might occur in humans; 2) using effects data collected at relatively high exposure concentrations to predict effects that might occur at lower exposure concentrations; and 3) using effects data collected with certain exposure durations and patterns to predict the effects that might occur with different exposure durations and patterns. These interpretations are often called animal-to-human, high-to-low dose, and across-exposure-scenario extrapolations. These types of extrapolations are often difficult to perform with a great degree of certainty. Limited data on which to base extrapolations increase the uncertainty.

Biological or biochemical processes might differ between laboratory animals and humans. Consequently, responses to the same ambient exposures can also differ. Similarly, biological and biochemical processes in healthy adult male workers can differ from important segments of the general population, such as children and the elderly. Also, exposure concentrations in animal experiments and occupational studies are likely to be higher than environmental exposures. Exposure durations and patterns are also often different. These disparities can differentially affect biological and biochemical processes, and consequently, Internal Dose. With careful study and estimation of Internal Dose, many of these differences can be understood and quantified.

Many of the uncertainties in risk assessment are unavoidable, given the current state of knowledge and the need to assess public health risks from HAPs. Particular types of scientific information, however, can improve analyses and reduce some uncertainties. In particular, reduction in uncertainties can occur via better estimation of dose to the affected organ (through such methods as evaluation of pharmacokinetics, biomarkers, and molecular dosimetry), and understanding what causes HAPs to have a toxic effect (the mechanisms of action). The size of the effort that will be required to gather these types of data, for even just the most important HAPs, is substantial.

Complex Mixtures

Complex mixtures confound the evaluation of the HAP problem in urban air. Urban air is a mixture of many pollutants, and little is known about the effects of exposure to mixtures of chemicals. Usually, effects assessments deal with only one chemical at a time. Sometimes, however, the effects of simple mixtures are assessed by adding together the anticipated effects from exposure to each individual compound. This additivity approach is normally only used when the anticipated effects are similar for the various chemicals in the mixture. When dealing with complex mixtures (like those found in urban air) and with many different potential health effects, scientists are reluctant simply to add together all of the anticipated individual health effects. They are reluctant because the interactions of mixtures on health are not well understood. Because of the complexity of the interactions, the total effect of the mixture might be very different than the simple sum of the individual effects. Additional research is currently being conducted to develop methods that will allow assessment of the effects of exposure to complex mixtures.

Chemicals Not on the List of 189 HAPs

Another important uncertainty in evaluating urban air is that chemicals, other than the 189 listed HAPS, might be shown to be more important air pollutants in the future. Thousands of individual chemicals, representing almost every known chemical class, are expected to be present

in urban air. There is recent evidence from studies of complex mixtures of urban air particles and gases that the major contributors to the mutagenicity of urban air are chemicals that have not yet been identified. These as yet unidentified chemicals might be produced by atmospheric transformation of organic pollutants emitted by a variety of sources. Specifically, new bioassay-directed chemical identification techniques have identified polar organic chemicals (for example, hydroxylated- and nitrated-aromatic hydrocarbons) in urban air that appear to arise from atmospheric transformation.[25]

Table 2-2 categorizes the almost 3000 chemicals that have been detected in ambient air: it also notes the number of chemicals in each category that have been evaluated in cancer biological assays and the number that have been found to be carcinogenic.[26] Several important points can be seen from this table: 1) only 10% of the chemicals detected in air have been screened in short-term genotoxic tests for their ability to cause cancer; 2) of the approximately 300 chemicals that have been screened, roughly 22% were found to be carcinogenic in the laboratory animal studies;[f] and 3) most evaluation has been focused on a few pollutant categories.[g] Consequently the contribution of many categories of chemicals as airborne carcinogens cannot be estimated. Further, it should be noted that this analysis does not consider the wide range of toxic effects needed for a full evaluation of the potential hazard. The development of data on this broad range of substances is almost certainly not warranted. Further analysis is needed to target specific chemicals for further evaluation.

[f] This percentage of positive cancer results must be interpreted with caution. Candidates for carcinogenicity testing often can be identified based on short-term mutagenic assays or other assays that detect genetic changes. Consequently, the chemicals selected for long-term cancer bioassays are more likely to be positive than randomly selected chemicals.

[g] Some categories of chemicals (for example, hydrocarbons, nitrogen-containing organics and halogenated organics) are relatively well tested. Other categories of chemicals, like ketones and carboxylic acids and their derivatives, are commonly detected in ambient air but have not been extensively evaluated.

Table 2-2. Occurrence and biological test results indicating carcinogenicity of airborne chemicals, for the 2,827 chemicals that have been reported to exist in the air.

Category	Number of Air Pollutants Identified In Each Category	Number of Pollutants that have Been Screened For Genotoxic Effects [a]	Number of Pollutants that have been Found Positive in Genotoxic Tests	Number of Chemicals Found to Cause Cancer in Laboratory Animals [b]
Inorganics	260	30	5	4
Hydrocarbons	729	51	12	19
Ethers	44	3	1	0
Alcohols	233	28	1	0
Ketones	227	11	0	0
Aldehydes	108	6	4	1
Carboxylic Acid Derivatives	219	6	0	2
Carboxylic Acids	174	5	0	0
Heterocyclic Oxygen Compounds	93	16	4	7
Nitrogen-Containing Organics	384	59	22	12
Sulfur-Containing Organics	99	4	1	1
Halogen-Containing Organics	216	71	16	21
Organometallic Compounds	41	13	6	0
GRAND TOTALS	**2,827**	**303**	**72**	**67**

[a] Short-term mutagenic or other genotoxic tests.

[b] Does not include all human carcinogens.

Data are compiled from Graedel, Hawkins and Claxton. *Atmospheric Chemical Compounds: Sources, Occurrence, and Bioassay*, Academic Press, Inc., Orlando, FL. 1986.

Section 3
Previous Assessments

The results from many previous screening studies have been compiled and presented in a 1990 report entitled *Cancer Risk From Outdoor Exposure to Air Toxics*[27]. This report provides a "snapshot" of the current understanding of the air toxics problem. It included emissions from all source types, not just area sources, including motor vehicle emissions. The report estimates that exposure to hazardous air pollutants from all source types accounts for as many as 1000–3000 cancer deaths each year in the U.S.[h]

Figures 3-1 and 3-2, adapted from the *Cancer Risk From Outdoor Exposure to Air Toxics* report, show the HAPs and source categories associated with the estimated HAPs-related cancer risks, respectively. Figure 3-1 shows that products of incomplete combustion (PIC), 1,3-butadiene, hexavalent chromium, and benzene account for more than half of the overall cancer risk among the pollutants evaluated.[i, 28] ("PIC" refers to a group of chemicals generated when fuels are only partially burned. PIC includes the HAP listed as polycyclic organic matter, or POM.) Results from such screening studies suggest that a handful of source categories — such as motor vehicles, chrome electroplaters, waste treatment storage and disposal facilities (TSDFs), woodstoves and fireplaces, asbestos demolition, and gasoline marketing — account for a majority of HAPs-related cancer risks (see Figure 3-2) in these screening studies. Lifetime cancer risk to individuals living in urban areas, aside from those risks obviously associated with major sources of HAPs, typically range from 1 in 100,000 (10^{-5}) to 1 in 1000 (10^{-3}). These figures demonstrate the relative importance of controlling non-major sources of HAPs in urban areas.

Other cancer screening studies not covered in *Cancer Risk From Outdoor Exposure to Air Toxics* generally suggest similar results; while data are available for only a small number of sources and pollutants, a relatively small subset of these generally account for most of the currently estimated HAPs-related cancer risk. The comparative rankings of sources and pollutants in each study vary, depending on what cities, sources, and pollutants are included in the analysis, and on methodological differences in the risk assessments.

[h] Please note the use of the term "as many as." The risk factors used to derive the estimates of possible cancer deaths in the cited report are "upper bound estimates." Such estimates are highly uncertain. The actual human risks are not known and are expected to be lower than the "upper bound" estimates used in the report.

[i] The actual risk estimates will change as new and better data are obtained. Indeed, a recent update (*Motor Vehicle-Related Air Toxics Study*, EPA 420-R-93-005, April, 1993) of mobile source risks suggests that the relative roles of PIC and 1,3-butadiene may be reversed. This assessment found the risk from PIC from all urban sources may be less than that shown and the risk from 1,3-butadiene may be greater than that found in the *Cancer Risk From Outdoor Exposure to Air Toxics* report and discussed in this section.

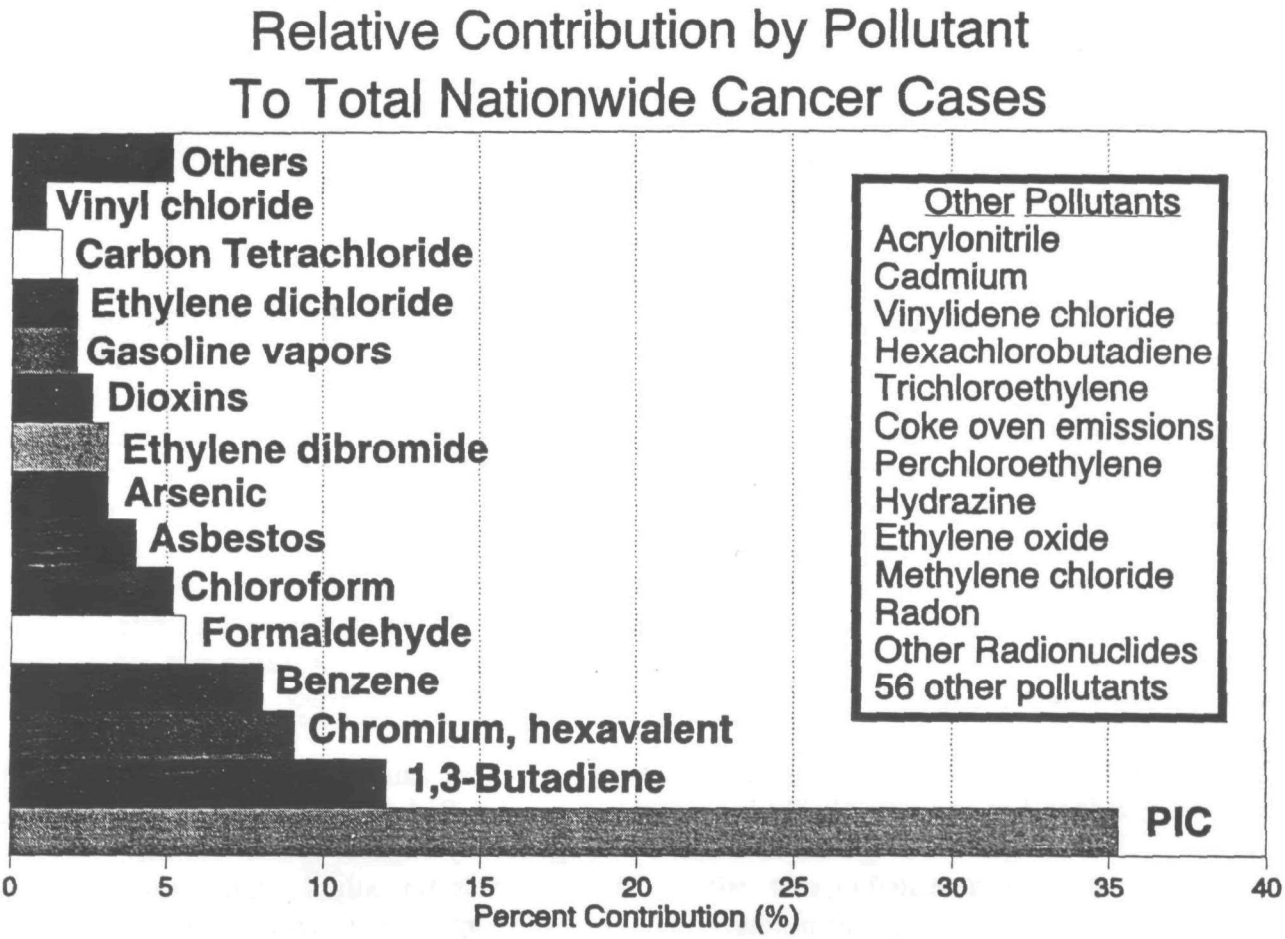


Figure 3-1. Relative contribution of various hazardous air pollutants to the estimate of nationwide cancer cases (from *Cancer Risk From Outdoor Exposure to Air Toxics*).

Very few screening studies have examined health effects other than cancer. One such effort, however, found that noncancer effects[8, 29] would likely be expected to occur in exposed urban populations.

The study attempted to estimate the potential noncancer effects of urban air pollutants, not just the listed HAPs. The study considered pollutants from all types of sources (not just area sources). Outdoor air monitoring data or computer-modeled estimates of ambient outdoor concentrations were used to examine potential exposures to air pollutants. Monitoring or modeling estimates of ambient outdoor concentrations were available for 334 air pollutants.[j]

[j] The average annual concentrations of 40 chemicals were modeled, based on estimated emissions data that were provided by more than 3500 individual commercial and industrial facilities across the U.S. Measured outdoor concentrations, of varying reliability and completeness, were available for more than 300 volatile organic chemicals at more than 1000 sites in 310 cities, and for 6 trace metals in more than two million samples from more than 1500 U.S. cities.

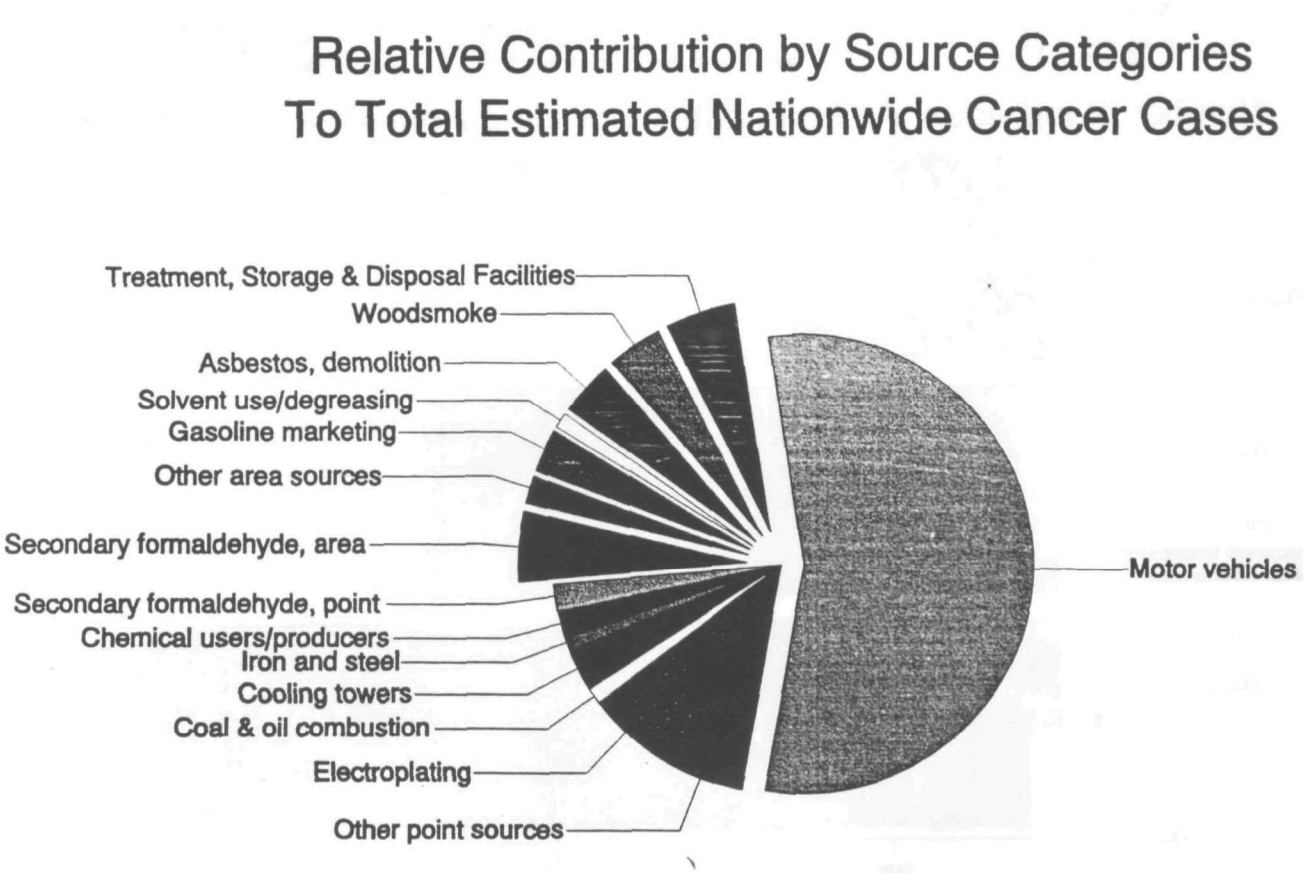


Figure 3-2. Relative contribution by source to the estimate of nationwide cancer cases per year caused by all sources, as reported in *Cancer Risk From Outdoor Exposure to Air Toxics*.

Of the 334 air pollutants with estimated ambient outdoor concentrations, information on potential noncancer health effects were available for 143 chemicals. For these pollutants, the estimated outdoor concentrations were compared to the lowest-observed-adverse-effect level (LOAEL) and to a health reference level.[k] Concentrations of 54 of the 143 pollutants exceeded the health reference level at one or more sites; more than 20 pollutants exceeded the health reference levels at more than 25% of the sites studied. Figure 3-3 shows the number of chemicals that exceeded these levels. The data are grouped according to whether the noncancer health effect was acute or chronic, and whether the estimated concentration

[k] A LOAEL is the lowest dose or exposure level at which an adverse effect has been reported in the health literature, typically from studies conducted in laboratory animals. A health reference level is the LOAEL divided by appropriate uncertainty factors to account for intra- and inter-species variability. The goal is to establish an exposure level below which the population is not expected to be affected at some unspecified level of frequency (risk). Health reference levels differ from Reference Concentrations (RfCs), which will be discussed later in this document, in that health reference levels receive much less review and validation than do RfCs.

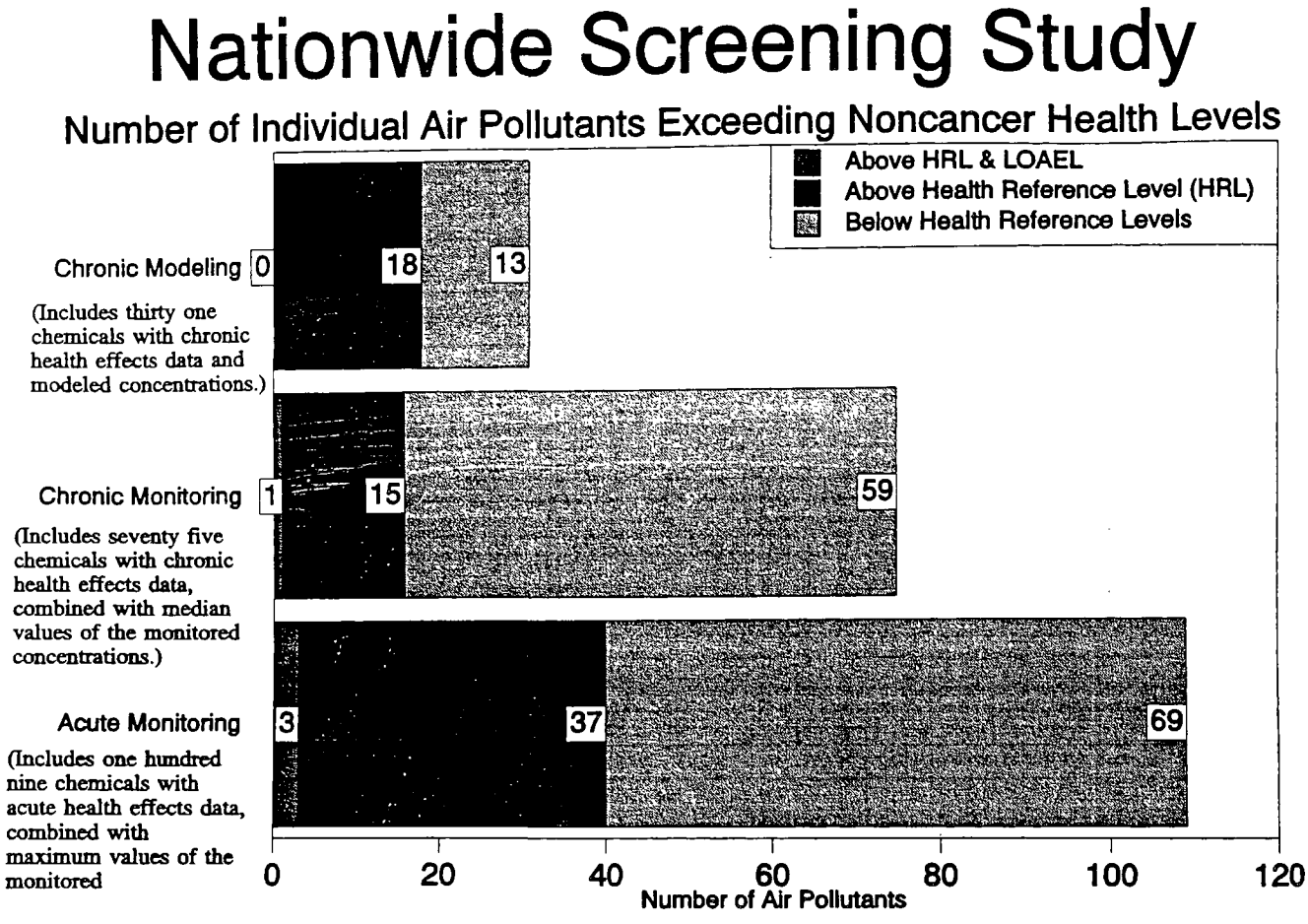


Figure 3-3. Results of a screening study to identify air pollutants with potential noncancer health effects.

was modeled or measured. An estimated 50 million persons lived within 10 km of monitored sites or within 2 km of facilities where modeled concentrations of one or more chemicals exceeded the health reference level. For the LOAEL, the comparable population estimate was 19 million persons. The data in Figure 3-3 are for individual air pollutants: typically, however, several pollutants were present in each area studied, but the effects of simultaneous exposure to multiple pollutants were not considered. This screening study concluded that exposure to air pollutants may pose risks of respiratory, neurologic, and reproductive systems effects and a risk for adverse developmental effects, for both individual chemicals, and chemical mixtures.

None of the screening studies performed to date claim to demonstrate actual cause-and-effect relationships between routine emissions of HAPs (or their resulting exposures) and an observed disease or other health effect. As noted previously, the risk factors used in such screening studies are "upper bound estimates" and are highly uncertain. The actual human risks are not known and are expected to be lower than the

"upper bound" estimates derived in such screening studies. The screening studies are useful, however, for comparing the relative ranking of the potential risks due to different pollutants and sources.

Section 4
Research Needs

The limited information currently available suggests that area sources contribute to air pollution that can potentially damage public health. In addition, atmospheric transformation products formed from area source emissions might also contribute to health risks. Only a limited amount of credible data is available with which to characterize the risks posed by exposures to urban air. Determining exposures, identifying the sources of those exposures, estimating the likely resulting health impacts, and identifying needed controls are very complex tasks. The scope and the complexity of these tasks make it necessary to identify the most critical research needs. Identification of the critical research needs provides a framework for systematically gathering information about urban HAPs over the coming decade to support development and implementation of the National Strategy for area sources.

To assess and manage efficiently the risks associated with HAPs from area sources, data from each compartment of the Environmental Health Paradigm are needed. Consequently, the research needs are organized and presented using this paradigm.

4.1 Research on Exposure Assessment

The discussion of Exposure Assessment research needs will address the key research questions related to Emission Sources, Environmental Concentrations, and Human Exposures.

Emission Sources

The key research needs for characterizing emission sources of HAPs are organized around the following questions:

- Which area sources emit HAPs, and how much do they emit?
- What are the most important sources and pollutants for which detailed emissions data must be developed?
- What are the most reasonable approaches for reducing emissions?

To assist with implementation of the National Strategy, data on the feasibility of pollution prevention or of adding emission controls must also be addressed.

Which area sources emit HAPs, and how much do they emit?

Given the limited availability of high-quality data on emissions of HAPs from many area sources, research is needed to identify the specific types of stationary sources that meet the definition of an "area source" and to characterize which HAPs they emit and in what quantities. Such data are critical to identifying the 30 or more "worst" HAPs, as required by the CAA. Research into methods to measure the emitted HAPs is fundamental to increasing our knowledge of area source emissions.

What are the most important sources and pollutants for which detailed emissions data must be developed?

The 30 or more "worst" HAPs have not yet been identified. Once the chemicals are specified, the task of identifying the area sources accounting for 90% of the area source emissions of each of the identified compounds becomes critical. In order to identify those area sources, detailed emission factors and emission estimation techniques will need to be developed.

What are the most logical approaches for reducing emissions from area sources, in terms of potential benefit, technical feasibility, costs, and impacts of other control programs?

Currently, the best approaches have not been determined. Pollution prevention approaches must be explored, while taking into account current data to define the achievable level of control and the costs of control. Other emission control programs, notably efforts to limit precursors of ozone (some of which are also HAPs; others of which might produce HAPs through transformation processes, *etc.*), can indirectly benefit the National Strategy, and the benefits from those programs must also be considered.

Environmental Concentrations

The key research issues under the Environmental Concentrations component deal with collecting the ambient data, considering the impacts of atmospheric transformation, and developing methods to make use of the ambient monitoring data. The key research questions are:

- What are the concentrations of HAPs from area sources?
- How does atmospheric transformation increase public risks?
- How can monitoring and modeling best be used to assess the effectiveness of the National Strategy?

What are the concentrations of HAPs from area sources, both from direct emissions and as secondary products, to which people are exposed?

Research is needed to develop methods to measure not only the listed HAPs, but the myriad potentially harmful chemicals present in urban air. Data are also needed to assess just how much monitoring is needed (for example, number of cities needed to provide a "representative" sample, the number of sites per city and the distances between sites, and the frequency of sample collection) to characterize the urban levels to which people are exposed.

How does atmospheric transformation increase public risks?

Research is needed to determine if the mutagenic transformation products formed in urban air are actually a hazard to human health, and if so, to identify the specific transformation products and any other necessary precursors that are responsible for the potential elevated risks. Only then can reasonable steps be taken to mitigate or prevent the exposure to and risk from these transformation products.

How can ambient monitoring best be used with available modeling methods (including emissions modeling, dispersion modeling, and source apportionment modeling) to demonstrate the effectiveness of the National Strategy (as required in the CAA)?

Critical components of this research are: 1) defining how to use ambient outdoor monitoring data to establish a "baseline" (the concentrations existing before the National Strategy

is implemented) and to determine concentration trends to measure the effectiveness of the National Strategy; 2) identifying the other factors (like wind speed, wind direction and the mixing depth, source emission profiles, and the distribution of sources throughout the urban area) that must be measured in order to derive an estimate of total area source emissions from the measured ambient outdoor concentrations; 3) developing data analysis methods to allow the trend in area source emissions to be determined despite "noise" from natural variations (like those caused by year to year changes in weather) and from the trends of point sources and mobile sources; and 4) determining if ambient outdoor data indicate that all area sources of the controlled HAPs have been recognized (that is, do the ambient concentrations reconcile with EPA's understanding of the emission sources?)

Human Exposures

The key research questions for Human Exposures are:

- What are the human exposures to HAPs?
- What are the routes of exposure?

What is the distribution of human exposures to the various HAPs? By what route, and how effectively, do the HAPs reach humans?

Data are needed to define how people's activities and the concentration of the HAPs vary with time and to characterize how that variation will affect the distribution of exposures. Research is also needed to define those circumstances that will lead to high exposures and high potential risks, including research to identify the chemicals and circumstances that make indirect exposures important.

4.2 Research on Effects Assessment

As with Exposure Assessment, there is a need for more research into Effects Assessment. Two areas that need additional research are Internal Dose and Health Effects.

Internal Dose and Health Effects

Critical issues facing health effects researchers in trying to define the potential human health effects of hazardous air pollutant emissions from area sources are:

- How can the most substantial hazards from HAPs be identified?
- How can health risks be estimated reliably?

How can the most substantial hazards from HAPs be identified?

Hazard identification research is needed to develop, refine, and validate methods for identifying chemicals and agents that pose potential human hazards. Faster, more accurate, less expensive, and more reliable techniques are needed to determine cause and effect relationships between environmental pollutants and adverse health outcomes than the methods that are currently available. Batteries of test methods designed to evaluate potential hazards comprehensively also need to be validated. A comprehensive program to collect toxicity data also is needed. Efforts should include evaluation of realistic scenarios for concentrations and exposures.

Additionally, field studies that evaluate the biological effects of exposure to urban air pollution are needed. These field studies should combine short-term methods developed in the laboratory to screen for problem chemicals, mixtures, and/or sources, and longer-

term studies to describe in more detail the hazards of urban air pollutant exposures.

How can health risks be estimated reliably?

Improved methods are needed to link ambient exposures to internal dose. Efforts in this area should include development and validation of biological markers for exposure, effects, and susceptibility in human populations; and improvement in pharmacokinetic models. These models use physiological and biochemical data to estimate internal doses resulting from external exposures. These efforts improve the confidence in extrapolation of animal data to humans and from the high doses used in laboratory studies to the lower doses more typical of human exposures.

Dose-response research is needed to develop biologically based dose-response models that elucidate: 1) the relationship between exposure concentration (or, the applied dose) and the dose at the site of toxics action (that is, the target dose) and 2) the basic biological mechanisms responsible for the observed effects. Understanding of underlying biological mechanisms is crucial to the accurate extrapolation of research results (for example, extrapolation of results from animals to humans, from high- to low-dose, and from "across-exposure scenario" effects.) These models estimate the type and extent of biological damage resulting from doses to the affected tissues, which, when coupled with exposure data, provides estimates of public health risks.

In addition, because HAPs in the environment never occur alone, predictive models for risk assessment of complex mixtures of HAPs are needed: the most urgent needs include techniques to compare potencies of various mixtures, to understand the mechanisms of chemical interactions in complex mixtures, to identify the most critical components leading to biological activity in complex mixtures, to determine the quantity of the biologically active components that reach susceptible organs or tissues in exposed people, and to develop biological markers of exposure and effects. These efforts are necessary to enable evaluation of the risks from environmental mixtures of pollutants.

Lastly, research in environmental epidemiology is needed to assess the impact of exposure to HAPs on the general population and to establish the link between environmental exposures and human health effects. Identification of appropriate biomarkers of exposure and effects are likely to be necessary to make such studies feasible for many pollutants.

Section 5
Summary of Preliminary Findings

Much of the discussion in this report has been framed around the Environmental Health Paradigm. Without at least some understanding of each component in the paradigm, it is impossible to develop reliable risk assessments. Adequate data exist only for a few HAPs. *Screening studies, like those referred to in Section 3, are helpful for outlining the potential dimensions of the urban HAP problem, but these studies are often based on incomplete, inadequate, and unreliable data. From a strictly scientific perspective, such studies are suggestive; however, they might not be sufficiently comprehensive or reliable to use for identifying the "worst" HAPs from area sources, or to use as the basis for the National Strategy.* In the following discussion, the summary of preliminary findings on what is currently known about HAPs from area sources in urban areas is organized according to the components of the Environmental Health Paradigm.

Emission Sources

- A total of 42 HAPs appear to have "Fair or Better" emissions data for *all* (not just area) sources. (Seventeen HAPs are regularly included in the available urban area emission inventories; an additional twenty-five HAPs either have national inventories or have validated emission factors in the FIRE data base.) Detailed area source information in most urban area HAP emission inventories is limited. Much of the data (including data available under Title III of the Superfund Amendments and Reauthorization Act) is considered to be incomplete, out-of-date, or limited in scope and application. More than 120 HAPs have little or no validated source emissions data.

- Emission factors, source activity data, and other emission estimation techniques are of questionable quality or are currently unavailable for a number of area sources of HAPs.

Environmental Concentrations

- There are no measurements of the air concentrations of almost 40% of the listed HAPs. Another 20% of the HAPs have very little monitoring data. For a few compounds, there are considerable monitoring data collected at a variety of locations. The ability to measure the HAPs is severely limited by the lack of methods to collect and analyze many of the listed chemicals.

- Atmospheric transformations complicate exposure assessment because they can increase or decrease the environmental concentrations of the listed HAPs. In addition, sunlight causes reactions among pollutants in urban air that can produce a variety of products, some of which are potentially even more harmful than the original pollutants. HAPs might be formed from non-hazardous precursors, some of which are emitted in large amounts into urban air.

Human Exposures

- Outdoor sources of HAPs form the baseline for human exposure, on top of which HAPs from indoor, workplace, and personal use sources add additional exposures. For some of the HAPs, such interior sources may be very commonplace and may frequently increase interior concentrations substantially above outdoor concentrations. For many of the gaseous HAPs, the indoor concentrations due to outdoor sources are equal to the outdoor concentrations. For other HAPs, the indoor concentrations attributable to outdoor sources are expected to be somewhat less because of physical or chemical losses as the HAPs are transported indoors. For HAPs attached to fine particles in the air, the indoor concentrations from outdoor sources are expected to be *50-90% of the outdoor concentrations.*

- Available human exposure data often do not describe well those situations that can lead to very high exposures to area source emissions (for example, living above a dry cleaning establishment or adjacent to a gas station).

Internal Dose

- Estimating the amount of HAP that reaches affected or susceptible organ(s) and causes damage to health is important in understanding the relationship between exposures to HAPs and the nature and magnitude of potential public health effects. This is particularly true when risk estimates are based on extrapolated information. Current methods and data for estimating internal dose are often crude. Good information exists only for a few HAPs.

Health Effects

- There are some health effects data available for each of the 189 HAPs. In almost no case, however, are there data available on all of the most important health effects: cancer, developmental and reproductive effects, neurotoxicity, and short-term and long-term pulmonary effects. The quality of the available data varies, ranging from inadequate to excellent.

- The evaluation of the cancer-causing potential of the HAPs is more complete than for other health effects. Also, reference concentrations (RfCs) for noncancer health effects have been developed for 40 of the listed HAPs. Values of the cancer risk estimates and of the RfCs are likely to change as new and better data become available.

- Only 10% of nearly 3,000 chemicals that can exist as air pollutants have been tested for genotoxicity or carcinogenicity. The number of chemicals tested for noncancer effects is even smaller. The development of data on this broad range of substances is almost certainly not warranted. Further analysis is needed to target specific chemicals for further evaluation.

- People are exposed to mixtures of many pollutants simultaneously, not just one pollutant at a time. Yet, how these mixtures of pollutants interact to affect human health is only poorly understood.

Synopsis

The availability of data on the 189 HAPs that are needed to do a complete environmental health assessment is illustrated in Figure 5-1. It reveals that very little is known about many of the HAPs, while significant amounts of information exist for a few chemicals. The same data are given for each of the 189 listed HAPs in Table A-1, found in the Appendix. A review of Table A-1 reveals that 20 chemicals have enough data to merit "Fair or Better" classifications in Source Emissions

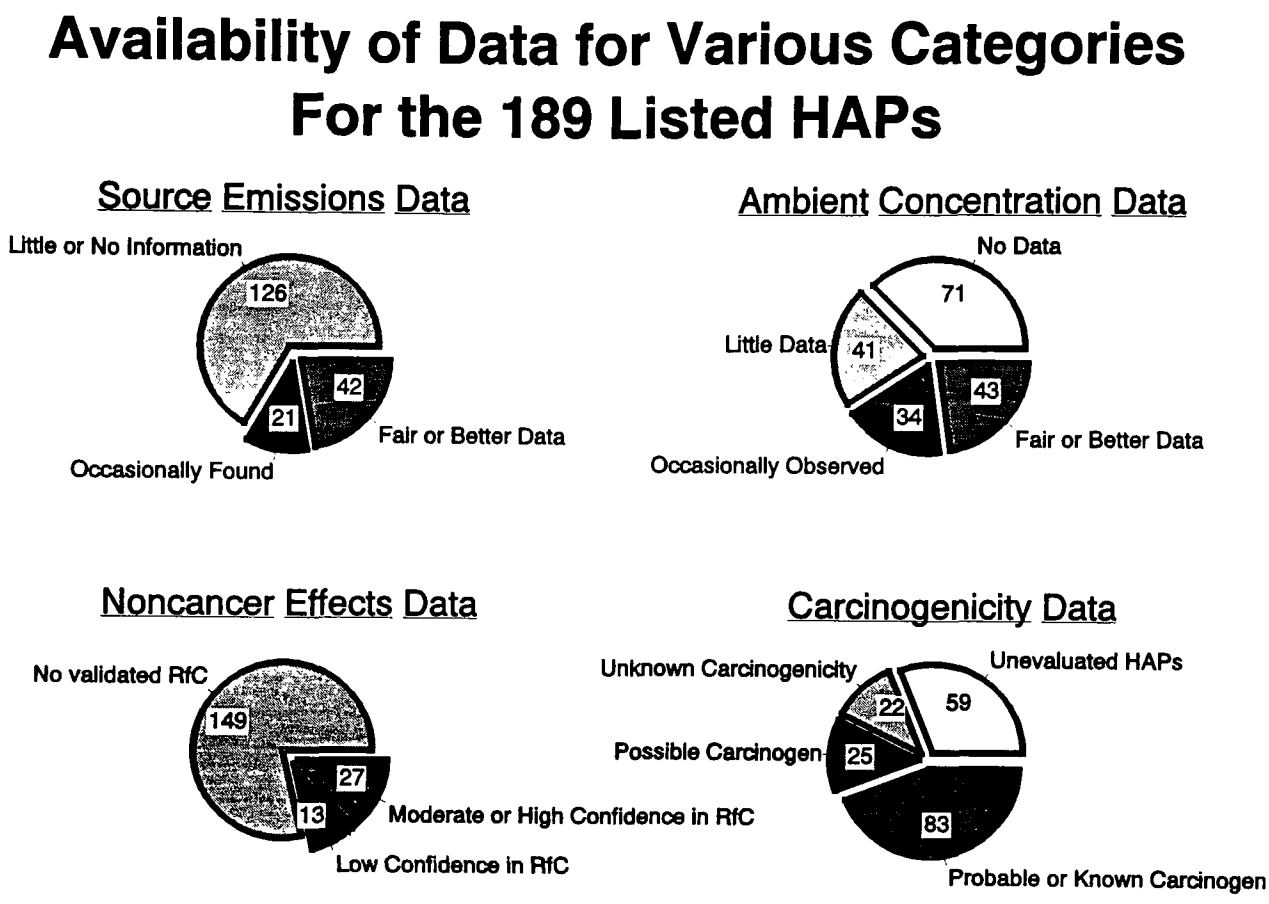


Figure 5-1. Summary of the available data on the 189 listed HAPs. (Table A-1, Appendix A, categorizes the data for each of the 189 HAPs.)

Data, Ambient Concentration Data, and in one of the Health Effects areas, either Noncancer Health Effects or Cancer Health Effects. This list of chemicals does not identify the 30 or more "worst" HAPs; rather, the list simply identifies those HAPs with sufficient data to begin a risk assessment of either the cancer or noncancer effects due to exposure to that chemical. Another 20 HAPs are rated "Fair or Better" in two of the three required areas. Targeted research on this second group of HAPs could readily provide sufficient data to allow a risk assessment to be initiated. The 40 HAPs with the most complete available data are listed in Table 5-1.

Continuing research will undoubtedly improve the scientific understanding of human exposures and health effects from increasing numbers of HAPs.

Table 5-1. The HAPs with the most extensive available data needed for a risk assessment.

HAPs with data rated "Fair or Better" in the three areas: • Source Emissions • Ambient Concentrations and • Health Effects (Cancer or Noncancer)	HAPs with data rated "Fair or Better" in <u>two</u> of the following three areas: • Source Emissions • Ambient Concentrations and • Health Effects (Cancer or Noncancer)
Benzene	Acetaldehyde
1,3-Butadiene	DDE (p,p'-dichlorodiphenyldichloro-ethylene)
Carbon tetrachloride	1,4-Dichlorobenzene
Chloroform	Ethylbenzene
Ethylene dibromide	Ethylene oxide
Ethylene dichloride	Hexachlorobenzene
Formaldehyde	Hexane
Methylene chloride	Methyl bromide
Styrene	Methyl chloroform
Tetrachloroethylene	Pentachlorophenol
Toluene	Polychlorinated biphenyls
Trichloroethylene	Propylene dichloride
Vinyl chloride	2,3,7,8-Tetrachlorodibenzo-p-dioxin
Arsenic compounds	2,4,6-Trichlorophenol
Chromium compounds	Vinylidene chloride
Lead compounds	Xylenes (mixed isomers)
Manganese compounds	Antimony compounds
Mercury compounds	Beryllium compounds
Nickel compounds	Cadmium compounds
Selenium compounds	Polycyclic Organic Matter

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Appendix

Table A-1 provides a listing of the available data for each of the 189 listed HAPs. The characterizations are consistent with those used in the text of the report. The shaded chemicals are the 20 HAPs identified in Table 5-1 as having "Fair or Better" data in three categories: Emission Sources, Environmental Concentrations, and Health Effects (either Cancer Effects or Noncancer Effects).

Meaning of the Symbols Used in the Table:

Source Emissions Data	Blank	Seldom included in emissions inventories (Little or no data)
	✓	Occasionally included in emissions inventories (*i.e.*, included in 10% or more, but less than half, of emission inventories studied)
	✓✓	Routinely included in emissions inventories (*i.e.*, in 50% or more of the case studies), or national inventory is available, or emission factors included in FIRE data base
Ambient Concentration Data	Blank	Little or no (71 HAPs) ambient data available
	✓	Between 100 and 1000 observations
	✓✓	More than 1000 observations
Noncancer Health Effects Data	Blank	No validated RfC available
	✓	RfC available, but "low" confidence
	✓✓	RfC available, "moderate" or "high" confidence
Cancer Health Effects Data	Blank	Unclassified (59 HAPs) or Class D (22 HAPs)
	✓	Class C carcinogen or IARC Class 2B chemical
	✓✓	Class A or B carcinogen or IARC Class 1 or Class 2A chemical

Table A-1. Availability of data on the 189 listed HAPs.

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
1	Acetaldehyde		✓✓	✓	✓✓
2	Acetamide				✓
3	Acetonitrile				
4	Acetophenone				
5	2-Acetylaminofluorene				✓✓
6	Acrolein			✓✓	✓
7	Acrylamide	✓			✓✓
8	Acrylic acid			✓✓	
9	Acrylonitrile	✓	✓	✓✓	✓✓
10	Allyl chloride	✓	✓	✓	✓
11	4-Aminobiphenyl				✓✓
12	Aniline			✓	✓✓
13	o-Anisidine				✓
14	Asbestos	✓	✓		✓✓
15	Benzene	✓✓	✓✓		✓✓
16	Benzidine				✓✓
17	Benzotrichloride				✓✓
18	Benzyl chloride	✓	✓		✓✓
19	Biphenyl				
20	Bis(2-ethylhexyl)phthalate	✓			✓✓
21	Bis(chloromethyl)ether				✓✓

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
22	Bromoform	✓	✓		✓✓
23	1,3-Butadiene	✓✓	✓✓		✓✓
24	Calcium cyanamide				
25	Caprolactam				
26	Captan		✓		
27	Carbaryl		✓		
28	Carbon disulfide				
29	Carbon tetrachloride	✓✓	✓✓		✓✓
30	Carbonyl sulfide				
31	Catechol				
32	Chloramben				
33	Chlordane		✓		✓✓
34	Chlorine				
35	Chloroacetic acid				
36	2-Chloroacetophenone			✓	
37	Chlorobenzene		✓✓		
38	Chlorobenzilate				✓✓
39	Chloroform	✓✓	✓✓		✓✓
40	Chloromethyl methyl ether				✓✓
41	Chloroprene		✓	✓✓	
42	Cresols (isomers and mixture)				✓
43	o-Cresol				✓
44	m-Cresol				✓
45	p-Cresol				✓

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
46	Cumene		✓✓		
47	2,4-D, salts & esters		✓✓		
48	DDE (72-55-9: p,p'-dichlorodiphenyldichloroethylene)		✓✓		✓✓
49	Diazomethane				
50	Dibenzofuran	✓✓			
51	1,2-Dibromo-3-chloropropane			✓✓	✓✓
52	Dibutyl phthalate	✓✓			
53	1,4-Dichlorobenzene	✓	✓✓	✓✓	✓✓
54	3,3'-Dichlorobenzidine				✓✓
55	Dichloroethyl ether				✓✓
56	1,3-Dichloropropene		✓	✓✓	✓✓
57	Dichlorvos		✓	✓✓	✓✓
58	Diethanolamine	✓			
59	N,N-Dimethyl aniline (also, diethyl)				
60	Diethyl sulfate				✓✓
61	3.3'-Dimethoxybenzidine				✓✓
62	4-Dimethylaminoazobenzene				✓✓
63	3,3'-Dimethylbenzidine				✓✓
64	Dimethylcarbamoyl chloride				✓✓
65	Dimethylformamide			✓✓	✓
66	1,1-Dimethylhydrazine				✓✓
67	Dimethyl phthalate				
68	Dimethyl sulfate				✓✓

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
69	4,6-Dinitro-o-cresol & salts				
70	2,4-Dinitrophenol				
71	2,4-Dinitrotoluene				✓✓
72	1,4-Dioxane		✓		✓✓
73	1,2-Diphenylhydrazine				✓✓
74	Epichlorohydrin	✓		✓✓	✓✓
75	1,2-Epoxybutane			✓✓	
76	Ethyl acrylate	✓			✓✓
77	Ethylbenzene	✓✓	✓✓	✓	
78	Ethyl carbamate				✓✓
79	Ethyl chloride		✓	✓✓	
80	Ethylene dibromide	✓✓	✓✓	✓✓	✓✓
81	Ethylene dichloride	✓✓	✓✓		✓✓
82	Ethylene glycol				
83	Ethylenimine				✓✓
84	Ethylene oxide	✓✓			✓✓
85	Ethylenethiourea				✓✓
86	Ethylidene dichloride		✓✓		✓
87	Formaldehyde	✓✓	✓✓		✓✓
88	Heptachlor		✓		✓✓
89	Hexachlorobenzene	✓✓	✓		✓✓
90	Hexachloro-1,3-butadiene	✓✓	✓		✓
91	Hexachlorocyclopentadiene				

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
92	Hexachloroethane	✓✓			✓
93	Hexamethylene-1,6-diisocyanate			✓✓	
94	Hexamethylphosphoramide			✓	✓
95	Hexane		✓✓	✓✓	
96	Hydrazine				✓✓
97	Hydrochloric acid	✓✓		✓	
98	Hydrogen fluoride	✓✓			
99	Hydroquinone				
100	Isophorone				✓
101	Lindane		✓		✓✓
102	Maleic anhydride				
103	Methanol				
104	Methoxychlor		✓		
105	Methyl bromide	✓	✓✓	✓✓	
106	Methyl chloride	✓	✓✓		✓
107	Methyl chloroform	✓✓	✓✓		
108	Methyl ethyl ketone	✓✓	✓	✓	
109	Methyl hydrazine			✓✓	✓✓
110	Methyl iodide		✓	✓	✓
111	Methyl isobutyl ketone				
112	Methyl isocyanate				
113	Methyl methacrylate				
114	Methyl tert-butyl ether		✓	✓✓	
115	4-4'-Methylenebis(2-chloroaniline)				✓✓

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
116	Methylene chloride	✓✓	✓✓		✓✓
117	Methylene diphenyl diisocyanate			✓✓	
118	4,4'-Methylenedianiline	✓			
119	Naphthalene				
120	Nitrobenzene	✓	✓		
121	4-Nitrobiphenyl				
122	4-Nitrophenol				
123	2-Nitropropane			✓	✓✓
124	N-Nitroso-N-methylurea				✓✓
125	N-Nitrosodimethylamine	✓			✓✓
126	N-Nitrosomorpholine	✓			✓
127	Parathion		✓		✓
128	Pentachloronitrobenzene				✓
129	Pentachlorophenol	✓✓			✓✓
130	Phenol	✓✓			
131	p-Phenylenediamine				
132	Phosgene				
133	Phosphine				
134	Phosphorus				
135	Phthalic anhydride				
136	Polychlorinated biphenyls	✓✓	✓		✓✓
137	1,3-Propane sultone				✓✓
138	beta-Propiolactone				✓✓
139	Propionaldehyde		✓		

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
140	Propoxur		✓		✓✓
141	Propylene dichloride	✓	✓✓	✓✓	✓✓
142	Propylene oxide	✓		✓✓	✓✓
143	1,2-Propylenimine				
144	Quinoline				
145	Quinone				
146	Styrene	✓✓	✓✓	✓✓	✓
147	Styrene oxide				✓✓
148	2,3,7,8-Tetrachlorodibenzo-p-dioxin	✓✓	✓		✓✓
149	1,1,2,2-Tetrachloroethane		✓✓		✓
150	Tetrachloroethylene	✓✓	✓✓		✓✓
151	Titanium tetrachloride				
152	Toluene	✓✓	✓✓	✓✓	
153	2,4-Toluenediamine				✓✓
154	Toluene-2,4-diisocyanate				
155	o-Toluidine				✓✓
156	Toxaphene		✓		✓✓
157	1,2,4-Trichlorobenzene		✓		
158	1,1,2-Trichloroethane		✓✓		✓
159	Trichloroethylene	✓✓	✓✓		✓✓
160	2,4,5-Trichlorophenol	✓✓			
161	2,4,6-Trichlorophenol	✓✓			✓✓
162	Triethylamine			✓	
163	Trifluralin		✓		✓

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
164	2,2,4-Trimethylpentane		✓✓		
165	Vinyl acetate			✓✓	✓
166	Vinyl bromide			✓	✓✓
167	Vinyl chloride	✓✓	✓✓		✓✓
168	Vinylidene chloride	✓✓	✓✓		✓
169	Xylenes (mixed isomers)	✓✓	✓✓		
170	o-Xylene		✓✓		
171	m-Xylene		✓✓		
172	p-Xylene		✓✓		
173	Antimony Compounds		✓✓		✓✓
174	Arsenic Compounds (inorganic, including arsine)	✓✓	✓✓	✓✓	✓✓
175	Beryllium Compounds	✓✓	✓		✓✓
176	Cadmium Compounds	✓✓	✓		✓✓
177	Chromium Compounds	✓✓	✓✓		✓✓
178	Cobalt Compounds		✓✓		
179	Coke Oven Emissions	✓			✓✓
180	Cyanide Compounds				
181	Glycol Ethers (various)	✓		✓	
182	Lead Compounds	✓✓	✓✓		✓✓
183	Manganese Compounds	✓✓	✓✓	✓✓	
184	Mercury Compounds	✓✓	✓✓	✓✓	
185	Fine Mineral Fibers		✓		
186	Nickel Compounds (Subsulfide/Carbaryl)	✓✓	✓✓		✓✓

No.	Chemical Name	Frequency of Occurrence		Health Effects Data	
		Source Emissions Data	Ambient Concentration Data	Noncancer	Cancer
187	Polycyclic Organic Matter (various PAHs)	✓✓	✓		✓✓
188	Radionuclides				
189	Selenium Compounds (sulfide, disulfide or other compounds)	✓✓	✓✓		✓✓

Table A-2 identifies the types of health effects, other than cancer (referred to as noncancer effects), that have been reported for the listed HAPs. The table presents data for only those HAPs that have produced effects in humans or animals by inhalation exposure.

SYSTEM or HEALTH EFFECT	EXPOSURE DURATION		
	ACUTE	SUBCHRONIC	CHRONIC
Bone	0	6	2
Cardiovascular	52	30	6
Death	56	15	1
Dermal	33	21	3
Reproductive/ Developmental	7	54	13
Endocrine/Exocrine	24	24	8
Ocular	96	44	7
Gastrointestinal	63	31	7
Hematopoietic	38	49	16
Hepatic	51	69	27
Immunologic	14	24	5
Multiple	1	0	1
Neurologic/Behavioral	107	74	20
Olfactory	8	15	6
Pancreatic	1	0	1
Renal	47	49	23
Respiratory	114	78	30
Spleen	3	18	11
Systemic	48	71	27
LD 50	65	0	0
Total Number of HAPs Showing an Effect	142	122	57

Table A-2. Number of hazardous air pollutants that have been reported to produce health effects in humans or animals by inhalation exposure.

Glossary

Term	Definition
Accidental release	Emissions resulting from an unpredicted failure of a system due to which some harm results.
Accuracy	The quality of being free from error. The degree of accuracy is a measure of the uncertainty in identifying the true measure of a quantity at the level of precision of the scale used for quantity.
Acute effects	Toxic effects of a substance which become manifest after only a short period of exposure of a duration measured in minutes, hours, or days.
Adverse health effects	An undesirable antagonistic consequence to human health due to some causative agent.
Air quality modeling	A mathematical representation of pollutant concentrations and their distribution in the atmosphere based upon assumptions or simulations of pollutant emissions, meteorological dispersion and transport, chemical and physical reactions, etc.
Air toxics	An expression commonly used to refer to hazardous air pollutants — often used interchangeably with "hazardous air pollutants." Any air pollutant (excluding those pollutants for which ambient criteria do exist, namely ozone, sulfur dioxide, carbon monoxide, nitrogen oxides, lead, and particulate matter) that may cause any of a wide range of potential harmful effects.
Ambient air	The surrounding or encompassing atmosphere. In the context of pollution monitoring, ambient air is often erroneously used to refer only to "outdoor" air, even though indoor air is "ambient" to a person who is indoors.
Ambient concentration	The concentration of a chemical (usually, a pollutant) in the atmosphere surrounding humans or other potentially affected receptors.
Ambient measurement	Measurement of a chemical (pollutant) found in the atmosphere surrounding humans or other receptors, any potentially affected species or ecosystem.
Ambient monitoring	Measuring the concentrations of pollutants or other species in ambient air.

Term	Definition
Area source of hazardous air pollutants	A stationary source which annually releases to the atmosphere, or has the potential to release considering controls, less than 10 tons of a single hazardous air pollutant listed in the Clean Air Act or less than 25 tons of a mixture of these pollutants. The term "area source" shall not include motor vehicles or nonroad vehicles subject to regulation under Title II of the Clean Air Act.
Area Source National Strategy	The National Strategy mandated in Section 112(k) of the Clean Air Act. By November 1995, EPA must "prepare and transmit to Congress a comprehensive strategy to control emissions of hazardous air pollutants from area sources in urban areas." The strategy shall "identify not less than 30 hazardous air pollutants which, as the result of emissions from area sources, present the greatest threat to public health in the largest number of urban areas." The strategy also shall "identify the source categories or subcategories" emitting the 30 or more hazardous air pollutants and "shall assure that sources accounting for 90 per centum or more of the aggregate emissions of each of the 30 identified hazardous air pollutants are subject to [emission] standards." "The strategy shall achieve a reduction in the incidence of cancer attributable to exposure to hazardous air pollutants emitted by stationary sources of not less than 75 per centum, considering control of emissions of hazardous air pollutants from all stationary sources and resulting from measures implemented ... under [the Clean Air Act] or other laws."
Atmospheric transformation	The chemical reactions in the atmosphere, many of which occur naturally and are unavoidable, that change (transform) one substance in the air into a different chemical or chemicals; or the physical processes (like washout into rain water or adsorption onto particles) that change the form of the chemical in the atmosphere and affect its distribution in the environment.
Bacterial mutagenicity	Refers to the use of bacteria to assess the mutagenic potential of pollutants.
Bioassay	Determination of the biological activity or potency of a substance by testing its effect on an organism. As used in this report, a test for carcinogenicity in laboratory animals (generally, rats and mice) that includes near-lifelong exposure to the agent (pollutant) under test. The term is used interchangeably with "animal test."

Term	Definition
Bioassay-directed chemical identification techniques	A combination of chemical and physical separation and identification methods with short-term biological tests in order to identify the chemicals in a complex mixture of pollutants that have a potential biological effect.
Biomarkers	Surrogates or indicators of biological exposure, dose, or effect. Part of the considerations under the Internal Dose component of the Environmental Health Paradigm.
Cancer risk	The risk of developing cancer.
Carcinogen	A substance or agent that tends to produce cancer in living organisms.
Carcinogenicity	The ability of a substance or agent to produce cancer.
Characterizing emission sources	Describing the pollutants emitted by a source, including the chemical composition, the quantity emitted as a function of time, and the location and relevant operational parameters of the source.
Chronic effects	Toxic effects of a substance that become manifest after prolonged or repeated exposures of a duration measured in weeks, months, or years.
Data base	Available, relevant raw information about the subject of concern.
Developmental disorders or effects	One type of noncancer health effects of concern; impairment of the normal development of a fetus, infant, or child, including developmental retardation and birth defects.
Direct emissions	Emissions of a pollutant that come directly from the source, without having to be produced by transformations.
Distribution of human exposures	A mathematical representation or other characterization of the range of exposures that people have to a pollutant.
Dose	The amount of a substance administered to an animal or human, usually measured in mg/kg of body weight, mg/m^2 of body surface area, or parts per million in the food, drinking water, or inhaled air. Dose, or target dose, is often used to refer to the quantity of the agent that reaches an affected organ of interest.
Dose-response relationship	The functional relationship between the amount of a substance at the affected organ and the lethality, morbidity, or level of health effect produced.

Term	Definition
Effects Assessment	Identification of the health effects that are likely to occur once humans (or ecosystems) are exposed to HAPs (or other pollutants).
Emission	The releasing of pollutant(s) to the atmosphere by a source or source category.
Emission estimation techniques	A method of estimating pollutant emissions from a particular source or category of sources. Such methods include the use of emission factors and activity data for the source or source category, as well as statistical approaches using surrogate data (e.g., census information) to estimate emissions in a specific geographic area.
Emission factor	An emission factor is a measure of the quantity of HAP that is emitted per unit quantity of a source activity (for example, pounds of HAPs per barrel of crude oil processed). Ideally, the source "activity" will represent the operations that lead to emissions (for example, how many barrels of crude oil are processed in a day). The product of the emission factor and the source activity is used to estimate the mass of HAP emitted. An emission factor is an average value which relates the quantity of a pollutant released to the atmosphere by a source (e.g., chemical process, fuel combustion) to the activity associated with release of that pollutant. It is usually expressed as the weight of pollutant per unit weight, volume, distance, or duration of the activity that emits the pollutant (e.g., kg of particulate matter per Mg of coal burned). To estimate emissions of a pollutant from a source, the emission factor for that source/pollutant is typically multiplied by the corresponding source activity level.
Emission source	A commercial, individual/residential, industrial, or institutional activity or process that releases pollutants to the atmosphere. These can be stationary (at a fixed geographic location) or mobile (e.g., automobiles).
Emission Sources	One of the components of the Environmental Health Paradigm: it includes evaluation of the pollutants emitted by the sources, including identification of the chemical emitted, the amount emitted, and the location of the source and the emission points and their characteristics.

Term	Definition
Environmental Concentrations	A component of the Environmental Health Paradigm: it includes evaluation of the concentrations of the pollutants in all environmental compartments and media, as appropriate, including indoor and outdoor air, water, soil, and food.
Environmental fate	The disposition of substance in the environment, including a description of the distribution between various media (air, water, soil).
Environmental Health Paradigm	A conceptual framework with which to organize and relate all of the aspects or considerations needed to characterize how pollutants from a source reach a human (or other receptor) and cause an effect. Understanding the linkages between the components of the paradigm also helps with evaluation of environmental management options. The paradigm includes evaluation of Emission Sources, Environmental Concentrations, Human Exposures, Internal Dose, and Health Effect(s).
Epidemiology	The study of the causes of diseases by identifying personal and environmental characteristics common to those contracting the disease. The sum of the factors controlling the presence or absence of a disease or pathogen.
Estimation	The assignment or derivation of outcome values and/or probability measures to a postulated event; a rough or approximate calculation; a numerical value obtained from a statistical sample and assigned to a population parameter.
Exposure	The coming into contact of humans (or ecosystems) with pollutants; exposure is measured as the product of concentration of the pollutant and the time of the exposure.
Exposure Assessment	Evaluation of how people are likely to come into contact with HAPs (or other pollutants) and the determination of how large the exposure is likely to be; the measurement or estimation of the magnitude, frequency, duration, and route of exposure to a hazardous substance or situation, and the size, nature, and classes of the exposed population.
Extrapolation (*e.g.*, across-exposure-scenario, animal-to-human, high-to-low dose)	To project, extend, or expand known or observed data to an area not known or observed. In the context of hazardous air pollutants, extrapolation is used to predict the following: responses in humans from animal data; low-dose responses from high-dose responses; and responses from one specific hazardous air pollutant exposure scenario to another different exposure scenario.

Term	Definition
Factor Information Retrieval (FIRE)	An EPA supported and published data base of information on emission factors of various sources.
Genotoxic	Possessing the ability to produce harmful effects in the genetic makeup of an organism.
Great Waters Program	A research and assessment program being conducted by EPA in response to Section 112(m) of the Clean Air Act, entitled "Atmospheric Deposition to Great Lakes and Coastal Waters."
Hazard	A source of risk (danger, peril, threat) that does not necessarily imply potential for occurrence. A hazard produces risk only if an exposure pathway exists and if exposures create the possibility of adverse consequences.
Hazardous air pollutant	An airborne substance whose effect on man or animals is potentially large but undefined since an exposure pathway may or may not exist; the 189 chemicals, or groups of chemicals, in the initial list of hazardous air pollutants found in Section 112(b) of the Clean Air Act; an air toxic.
Health Effect(s)	One of the components of the Environmental Health Paradigm: it includes characterization of the potential health effects due to exposure, including cancer effects, noncancer effects, any observable damage of disease or symptoms of adverse effects.
Human Carcinogen	A classification given to a chemical when there is sufficient evidence from epidemiologic studies to support a causal association between exposure to the agents and cancer.
Human Exposures	One of the components of the Environmental Health Paradigm: it involves evaluation of the route, magnitude, duration and frequency of exposure; the interaction of humans with a pollutant or other physical parameter. Exposure is measured as the product of concentration and time.
Incidence	The number of new cases of a disease, usually expressed as a rate; typically, the number of new cases of a disease occurring in a population during a specified period of time divided by the number of persons exposed to risk of developing the disease during that period of time. The incidence rate is a direct estimate of the probability of developing a disease during a specified period of time.

Term	Definition
Internal dose	One of the components of the Environmental Health Paradigm: it involves identification of the quantity (dose) of pollutant that is absorbed (the absorbed dose), the quantity that reaches the affected organ where it may have an effect (the target dose), and biological indicators (biomarkers) of exposure and effects.
LD_{50} (lethal dose for a 50% death rate)	A calculated dose of a substance that is expected to cause the death of 50% of an entire defined experimental population within a specified length of time.
Locating and Estimating Reports	A series of documents issued by EPA to compile available information on sources and emissions of substances which may be toxic at certain concentrations in the ambient air.
Major source of hazardous air pollutants	A stationary source or group of stationary sources located within a contiguous area and under common control that annually releases to the atmosphere, or has the potential to release considering controls, 10 tons or more of a single hazardous air pollutant listed in the Clean Air Act or 25 tons or more of a mixture of these pollutants.
Margin of safety	A factor added to an estimated risk level for purposes of increasing the probability that a standard based on the resultant level will provide increased protection to the general population and individual members from harmful effects of a given substance.
Mechanism(s) of action	The underlying cause of disorder or disease; the specific physical, chemical, and/or biological events caused by HAP exposure that are necessary for development of the resulting symptoms, disorder, or disease.
Mechanistic data	Data describing or pertaining to mechanisms of action.
Median	The value in an ordered set of values (that is, ambient concentration measurements arranged from lowest to highest) in the middle, with the number of values (measurements) that are larger than the median being equal to the number of values (measurements) that are smaller than the median.
Metabolism	The sum of the physical and chemical process in an organism by which its material substance is produced, maintained, and destroyed, and by which energy is made available.
Mobile sources	Sources of emissions that can move, like automobiles, trucks, planes, boats, and trains.

Term	Definition
Model	A simplified representation of a system or phenomenon, as in the sciences or economics, with any hypotheses required to describe the system or explain the phenomenon, often mathematically; a system of postulates, data, and inferences presented as a mathematical description of an entity or state of affairs; a representation of reality; a description or analogy used to help visualize something (*e.g.*, air pollution patterns across a city) that can not be directly observed.
Molecular dosimetry	Characterization of the quantity of a chemical reaching an affected organ at a molecular level.
Monitoring (of pollutants)	Periodic or continuous sampling and analysis to determine the level of pollution or other characteristics.
Mutagen	A substance possessing the ability to induce heritable mutations in living organisms.
Mutagenic	Having the characteristic of being a mutagen.
Mutagenicity	The quality of being mutagenic. Mutagenicity is often measured using short-term bioassays in which changes to the genetic code of bacteria are identified.
Mutagenic products	Products of atmospheric transformation that are mutagenic.
Mutation	A departure from being like the parent in one or more heritable characteristics, due to a change in a gene or chromosome.
Neurotoxicity	The degree to which a substance is toxic to nerve tissues; one of the noncancer health effects of concern in development of the Area Source National Strategy.
Noncancer health effect	A health effect other than the development of cancer. Section 112(k) of the Clean Air Act lists a number of noncancer health effects to be considered under the Area Source program, including "mutagenicity, teratogenicity, neurotoxicity, reproductive dysfunction and other acute and chronic effects including the role of such pollutants as precursors of ozone or acid aerosol formation."
Noncancer risks	The risk of developing a noncancer health effect.
Oral exposure data	Data on health effects developed from animal tests in which the exposure to the pollutant is through ingestion.

Term	Definition
Particulate matter	Solid or liquid particles suspended in the atmosphere; a form of pollution for which maximum allowable concentrations in the air have been established through legislation and regulation.
Pharmacokinetic models	Models that describe the fate of pharmacological substances in the body, including absorption, distribution, metabolism, and elimination; dose-response models based on the principle that biological effects are the result of biochemical interaction between foreign substances or their metabolites and parts of the body.
Photochemical process	Chemical reactions initiated by the absorption of light. Formation of ozone and other manifestations of "smog" are the result of a long series of atmospheric reactions that are started by the absorption of light by chemicals in the air and the resultant production of highly reactive molecular fragments.
Point source	A stationary source of pollutants, where the location of the source and its emissions of pollutants can be specified.
Possible human carcinogen	A classification given to a chemical when there is limited evidence of carcinogenicity in animals in the absence of human data.
Potency	The efficacy, effectiveness, or strength of a chemical to cause a toxicologic response.
Probable human carcinogen	A classification given to a chemical when there is limited evidence of human carcinogenicity based on epidemiologic studies or sufficient evidence of carcinogenicity based on animal studies.
Products of incomplete combustion (PIC)	All of the products other than water and carbon dioxide that are produced when an organic fuel, like gasoline, fuel oil, or wood, is burned; commonly, PIC is used to refer to a complex mixture of non-volatile and semi-volatile organic chemicals, many of which are polycyclic organic compounds, associated with particulate emissions that occur whenever a fuel is burned incompletely.
Pulmonary effects, acute and chronic	Adverse health effects involving the lungs and due to short-term exposures to high concentrations of pollutants (acute) or to long-term exposures to lower concentrations of pollutants (chronic). One of the noncancer health effects listed in Section 112(k) of the Clean Air Act.

Term	Definition
Quantification	The assignment of a number to an entity; a method for determining a number to be assigned to an entity; the act of determining, indicating, or expressing the quantity of an item.
Range of values	Evaluation of an uncertain outcome by estimation of maximal and minimal values.
Reference concentrations (RfCs)	An estimate, with uncertainty spanning a factor of 10, of the concentration that could be inhaled for a lifetime with no adverse health effects.
Reliability	The probability that a system will perform its required functions under conditions for a specified operating time.
Revertants	A measure of mutagenicity in a short-term bioassay using bacteria. Specifically, mutant strains of bacteria are exposed to pollutants, and only those bacteria that mutate back, or "revert," to their original genetic coding are able to survive and produce colonies.
Risk	The probability of uncertain, undesirable consequences or outcomes; having a chance of injury or loss.
Risk, absolute	A quantifiable estimate of a risk, based upon measurable and observable data or statistics, without major assumptions or upper-limit estimates.
Risk, comparative	An evaluation of the ranking of risks from a variety of causes in relationship to each other.
Risk assessment	The process of quantifying the level of risk associated with some situation or action.
Risk assessment method	A systematic procedure or mode of inquiry that may be employed as part of a risk assessment.
Risk estimation	The process of characterizing uncertainty (i.e., quantification of probabilities) and consequence values for risk.
Superfund Amendments and Reauthorization Act (SARA) Title III	Title III of the Superfund Amendments and Reauthorization Act of 1986, also known as the "Emergency Planning and Community Right-to-Know Act of 1986," (EPCRA) which requires a periodic (annual) inventory of toxic chemicals used, manufactured, or processed in quantities above specified threshold amounts at facilities in the U.S. [See Toxic Release Inventory.]

Term	Definition
Screening (studies or hazards)	A preliminary process of hazard identification whereby a standardized procedure is applied to classify products, processes, phenomena, or persons with respect to their hazard potential.
Secondary products	The products that are produced from the first, or primary, products; specifically, photochemical reactions in polluted air produce primary products which themselves react further to produce the secondary products, many of which are the more stable products normally associated with smog.
Short-term test	Tests that take less time to complete than do other types of bioassays. Many short-term tests measure the biological interactions between the agent under test and deoxyribonucleic acid (DNA). Agents that have effects in short-term tests are generally considered more likely to be health hazards than those that have no effect.
Smog (photochemical smog)	Air pollution containing ozone and other reactive compounds formed by the action of sunlight on nitrogen oxides and hydrocarbons (or other organic precursors).
Smog chamber	An experimental apparatus used to simulate the production of photochemical "smog"; often a large, Teflon-lined enclosure or bag, surrounded by lights that represent the sun's radiation or open to natural sunlight, with connections for inserting and withdrawing samples of pollutants.
Source category	A grouping of individual sources for consideration together because of similarities in emissions, manufacturing processes, or other factors.
Stationary source	A source of pollutants in a fixed position, the location of which can be specified.
Target dose	The amount of HAP that directly impinges on tissues or organs and induces a significant or toxic effect. Part of the considerations under the Internal Dose component of the Environmental Health Paradigm.
Technology	The tangible products of the application of scientific knowledge.
Teratogenicity	Production or induction of malformations or monstrosities, especially of a developing embryo or fetus. One of the noncancer health effects listed in Section 112(k) of the Clean Air Act.

Term	Definition
Threshold	A discontinuous change of state of a parameter as its measure increases. One condition exists below the discontinuity, and a different one above it. In context of toxicity and this report, exposures above a threshold produce effects, whereas exposures below threshold do not produce effects.
Toxicity	Inherent ability of a substance to adversely affect living organisms.
Toxic Release Inventory (TRI)	The TRI is an inventory of releases to air, water, and soil, or transfers to treatment facilities of 322 toxic chemicals. The TRI was mandated by the Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986 (also known as Title III of the Superfund Amendment and Reauthorization Act). Manufacturing facilities that produce, import, or process 50,000 pounds or more per year, or facilities that use 10,000 pounds or more per year of the 322 chemicals specified in the EPCRA must report their emissions annually to EPA. [See SARA Title III.]
Toxic substance	A substance of which exposure to humans or animals results in deleterious effects.
Uncertainty	A situation where there are a number of possible outcomes and one does not know which of them has occurred or will occur; indeterminacy; unpredictability; indefiniteness.
Urban areas	Areas in a city or town; areas that are city-like.
Volatile organic compounds	Two major definitions are common: (1) Under the regulatory control program to limit production of ozone pollution, VOCs are organic chemicals, usually hydrocarbons, that produce ozone at a rate greater than ethane; (2) In a scientific sense, VOCs are chemicals containing carbon that evaporate so readily that they exist in the air as vapors.