Introduction to Air Toxics

Student Manual

APTI Course 400

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Notice

The information, procedures and legal interpretations set forth here are intended as a technical resource and training device to those involved with air toxics issues. This is not an official policy and standards document. The opinions and selections are those of the author and not necessarily those of the U.S. Environmental Protection Agency. Every attempt has been made to represent the present state of environmental knowledge as well as subject areas still under evaluation. Any mention of products or organizations does not constitute endorsement or recommendation by the United States Environmental Protection Agency.

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Table of Contents

Chapter 1  History of Air Toxics

1.1 Epidemiology and Toxicology ................................................................. 1-1
1.2 Legislative History of Air Toxics .............................................................. 1-6
1.3 The Risk Assessment Process ................................................................. 1-8
  1.3.1 Basics of the Risk Assessment Process ........................................... 1-8
  1.3.2 History of the Risk Assessment Process for Air Toxics ...................... 1-12
1.4 Emergency Planning and Community Right to Know Act ...................... 1-16
  1.4.1 Toxic Release Inventory ................................................................. 1-18
References ........................................................................................................ 1-20

Chapter 2  Regulation of Air Toxics

2.1 Pre-1990, Air Toxic Program ................................................................. 2-1
2.2 Post-1990, Air Toxic Program: An Overview .......................................... 2-2
2.3 Listing of Hazardous Air Pollutants ......................................................... 2-3
2.4 Source Category Listing ......................................................................... 2-6
2.5 Definition of Major and Area Source ...................................................... 2-6
2.6 MACT Standards .................................................................................... 2-11
  2.6.1 MACT Organization ......................................................................... 2-14
  2.6.2 MACT General Provision Rule ......................................................... 2-15
  2.6.3 The MACT Hammer ......................................................................... 2-15
  2.6.4 NESHAP (MACT) Rule Applicability .............................................. 2-16
  2.6.5 New Directions in Setting MACT Standards .................................... 2-18
2.7 Residual Risk Program ........................................................................... 2-19
2.8 Area Source Program ............................................................................. 2-20
2.9 Urban Air Toxics .................................................................................... 2-20
2.10 Mobile Source Air Toxic (MSAT) Program ........................................... 2-22
2.11 Mercury Emissions from Coal Fired Power Plants ................................. 2-24
2.12 Prevention of Accidental Releases ........................................................ 2-25
2.13 Great Waters Program .......................................................................... 2-27
2.14 State Air Toxic Programs ....................................................................... 2-28
2.15 Hazardous Air Pollution Monitoring Network ....................................... 2-29
2.16 Trends in Toxic Air Pollution ................................................................. 2-30
2.17 National Air Toxic Assessment (NATA) ............................................... 2-31
Chapter 3  Air Toxics: Chemicals, Sources, and Emission Inventories

3.1  Introduction ........................................................................................................ 3-1
3.2  Air Toxic Chemicals .......................................................................................... 3-1
  3.2.1  Chemical Abstract Registry Number ......................................................... 3-1
  3.2.2  Chemical Lists for Large Groups of Compounds ....................................... 3-2
  3.2.3  Three Main Groups of Air Toxics ............................................................... 3-2
  3.2.4  CAA Hazardous Air Pollutants ................................................................. 3-3
  3.2.5  CAA Criteria Air Pollutants ..................................................................... 3-4
  3.2.6  Toxic Release Inventory (TRI) Chemicals ................................................. 3-5
  3.2.7  Toxic Chemicals That Persist and Which Also May Bio-accumulate ........ 3-5
  3.2.8  Other Chemicals ....................................................................................... 3-8
3.3  Sources of HAPs ............................................................................................... 3-8
  3.3.1  Point Sources ............................................................................................ 3-10
  3.3.2  Nonpoint Sources .................................................................................... 3-10
  3.3.3  On-Road and Non-Road Mobile Sources ................................................. 3-11
  3.3.4  Sources Not Included in the NEI or TRI ................................................. 3-12
3.4  Emission Inventories ...................................................................................... 3-15
  3.4.1  National Emission Inventories (NEI) ....................................................... 3-15
  3.4.2  Toxic Release Inventory (TRI) ................................................................. 3-17
  3.4.3  State, Local, and Tribal Permit Files ....................................................... 3-18
  3.4.4  Regional Inventories ................................................................................. 3-18
3.5  Developing an Emission Inventory ................................................................ 3-19
  3.5.1  Planning ..................................................................................................... 3-19
  3.5.2  Gathering Information ............................................................................. 3-20
  3.5.3  Estimating Emissions ............................................................................. 3-20
  3.5.4  Compiling Data into a Database ............................................................... 3-23
  3.5.5  Data Augmentation ................................................................................. 3-23
  3.5.6  Quality Insurance/Quality Control ........................................................... 3-23
  3.5.7  Documentation ....................................................................................... 3-23
  3.5.8  Access to Data ......................................................................................... 3-24
References ............................................................................................................... 3-25
Chapter 4  Introduction to Risk Assessment

4.1 Risk .................................................................................................................. 4-1
4.2 Risk Assessment: Conceptual Model of Air Toxic Releases......................... 4-1
4.3 Four-Step, Risk Assessment Process ............................................................... 4-3
4.4 Framework for Risk Assessment .................................................................. 4-4
4.5 Tiered Approach for Risk Assessment ........................................................... 4-7
4.6 Risk Assessment and Risk Management ......................................................... 4-9
4.7 Community Involvement and Risk Communication ....................................... 4-11
  4.7.1 Community Involvement .......................................................................... 4-11
  4.7.2 Risk Communication .............................................................................. 4-12
4.8 Air Toxics Risk Assessment Library: 3 Volumes ......................................... 4-13
References ........................................................................................................... 4-19

Chapter 5  Dispersion, Transport, Fate, and Modeling of Air Toxics in the Atmosphere...

5.1 Introduction ...................................................................................................... 5-1
5.2 Dispersion and Atmospheric Transport of Air Pollution .............................. 5-3
  5.2.1 General Types of Air Pollution Releases ................................................. 5-3
  5.2.2 Source Characteristics that Affect Dispersion and Transport ............... 5-3
  5.2.3 Meteorological Factors Affecting Air Toxics Dispersion and Transport 5-5
  5.2.4 Physical Factors Affecting Air Toxics Dispersion and Transport .......... 5-8
5.3 Physical Process Removing Air Toxics .......................................................... 5-9
  5.3.1 Dry Deposition ....................................................................................... 5-9
  5.3.2 Wet Deposition ...................................................................................... 5-10
5.4 Chemical Reactions of Air Toxics in the Atmosphere ................................ 5-10
  5.4.1 Major Chemical Reactions of Air Toxics .............................................. 5-11
  5.4.2 Chemical Reactions that Result in the Secondary Formation of Pollutants5-11
5.5 Air Quality Models ......................................................................................... 5-12
  5.5.1 Types of Models ................................................................................... 5-13
  5.5.2 Screening vs. Refined Models ............................................................... 5-14
5.5.3 Data Required for Modeling .................................................................... 5-15
  5.5.4 Sources and Examples of Air Quality Models ...................................... 5-17
  5.5.5 Model or Monitor .................................................................................. 5-21
References.....5-23
Chapter 8  Risk Characterization of Air Toxics

8.1  Introduction ............................................................................................................. 8-1
8.2  Inhalation Cancer Risk ......................................................................................... 8-2
8.2.1  Cancer Risk from Exposure to Multiple Pollutants ........................................... 8-4
8.2.2  Descriptors of Cancer Risk .............................................................................. 8-5
8.3  Non-Cancer Hazard Estimates for Chronic Exposures ....................................... 8-7
8.3.1  Chronic Non-Cancer Hazard Estimates from Exposure to Multiple Pollutants ..................................................................................................................... 8-8
8.4  Quantifying Risk from Background Sources ....................................................... 8-10
8.5  Presenting Risk Results ....................................................................................... 8-10
8.6  Characterization of Assumptions, Limitations, and Uncertainty ......................... 8-12
References ..................................................................................................................... 8-15

Chapter 9  Toxic Torts: Risk Assessment in the Courtroom

9.1  Introduction ............................................................................................................. 9-1
9.2  Toxic Torts .............................................................................................................. 9-1
9.2.1  Plaintiff’s Burden of Proof .............................................................................. 9-2
9.2.2  Causation Components .................................................................................... 9-4
9.2.3  Special Causation Challenges ....................................................................... 9-4
9.2.4  Admissibility of Expert’s Opinion .................................................................. 9-5
9.2.5  Common Theories in Tort Liability .................................................................. 9-5
9.2.6  Special Toxic Tort Cases .................................................................................. 9-6
References ..................................................................................................................... 9-9

Chapter 10  Air Toxics Monitoring

10.1  Introduction ......................................................................................................... 10-1
10.2  EPA’s Air Toxic Monitoring Program ................................................................. 10-3
10.3  Reasons for Monitoring Air Toxics .................................................................... 10-3
10.4  Planning an Air Toxics Monitoring Program ..................................................... 10-4
10.4.1  Selecting Locations of Air Toxics Monitors ................................................. 10-6
10.4.2  Detection Limits and Limits of Quantification .............................................. 10-9
10.5  Air Toxics Monitoring Methods ......................................................................... 10-9
10.5.1  Organic and Inorganic Air Toxics ................................................................. 10-9
10.5.2  EPA’s Air Toxic Monitoring Methods ......................................................... 10-11
10.6  Determining the Types of Equipment and Samples ........................................... 10-17
12.9.4 Activated Carbon Injection .......................................................... 12-25
12.9.5 Mercury Controls for Coal-fired Utility Boilers ......................... 12-27
References .......................................................................................... 12-31

Appendix Summarized MACTs for Petroleum Refineries and Synthetic Organic
Chemical Manufacturing Industry

A.1 Summarized MACT for Petroleum Refineries, 40 CFR 63, Subpart CC .... A-1
A.2 Hazardous Organic NESHAP (HON) for the Synthetic Organic Chemical
Manufacturing Industry (SOCMI) 40 CFR 63, Subparts F, G, H .............. A-3
References .......................................................................................... A-5
Chapter 1: History of Air Toxics

1.1 Epidemiology and Toxicology

The roots of air toxic history are based in the sciences of epidemiology and toxicology. Epidemiology seeks to answer the question: What is causing this person (or these people) to experience a particular harmful effect? In short, adverse effects are observed and causes for them are sought. Toxicology, on the other hand, begins with a known or suspected cause of adverse health effects and seeks to discover the relationship between the amount of the toxin taken in (dose) and the degree of effect (response). The question that drives toxicology studies is: How much of this substance is needed to produce harmful effects? Therefore, causative factors are observed and critical effect levels are sought.

Early metallurgists, who transformed crude ores to metals, were probably observant enough to discover that some of the materials they worked with could also harm them. Early Greeks and Romans noticed that miners and metallurgists often developed particular diseases, and these conditions were attributed to toxic air they were breathing and the toxic metals they were handling.

In 1775, a British surgeon, Percival Pott, published his observations of high rates of cancers of the scrotum among London chimney sweeps. Pott attributed the cancers to the soot with which these workers came into contact. Pott knew nothing about the chemical composition of soot. The surgeon reached conclusions about the causal relationship between soot and scrotal cancer on several grounds, and that the occurrence of these cancers could be reduced if certain hygienic practices were followed to reduce the sweeps’ direct contact with soot. Pott’s observations can be said to be the first to establish a cause-effect relation between an environmental agent and cancer.

In 1854, a cholera epidemic broke out in London causing staggering mortality rates. Dr. John Snow studied the cause of this mysterious disease. Shortly before the 1854 epidemic, one of London’s water supplies had moved its intake on the River Thames to a point above the city where the water was noticeably cleaner. Quite by accident, this created a control test group, allowing Dr. Snow to perform a comparative study, and proving that none of the residences supplied by the new water source were affected by this new outbreak of cholera. Dr. Snow eliminated one possible source after another until discovering one commonality among all cholera deaths - the Broad Street well. When the well cover was removed, the water was found to be contaminated and the case was
closed. In the late 1800s, Dr. Snow’s efforts and other epidemiological studies help lead to the development of the germ theory of causation. This, in turn, fueled the search for antibiotics to fight germs.

History has shown that the general lack of knowledge about the toxic effects of certain chemicals in everyday practice can lead to the disregard of its devastating, unhealthy consequences. For example, as noted in Stelljes (2008) “Toxicology for Nontoxicologist,” aristocratic Romans and Greeks would typically coat their bronze and copper cooking pots and goblets with lead to prevent copper or other metals from being dissolved into the food or drink. Also, the flavor of poor wines was enhanced by adding the lead coatings to the goblets. Meanwhile, the common folk were unable to afford the great wine and expensive goblets and cooking pots. Historical accounts allude to abnormal behavior and health problems by emperors such as Nero, Claudius, and Caligula who possibly feasted to a great extent with lead-coated cookware. The ruling class could have been plagued by neurological diseases caused by lead ingestion. A recent study has shown that the Roman aristocracy consumed lead on an average of eight times what a highly-exposed city-dwelling American now consumes and some emperors may have consumed twenty times more than city-dwelling Americans.

On the other hand, some of the earliest written accounts of man provide evidence that the ancient Greeks and Romans were well aware of the poisonous properties of certain plants and metals. An example of this would be the famous case of the poisoning of Socrates with hemlock. “Toxicology” actually means “the study of poisons,” and this emerging science got a big boost from the rise of the “art” of poisoning. From the earliest times, poisons were used as political weapons, and their use became more widespread and more diverse as time passed. A poisoner was a well-respected member of society. Of course, along with the proliferation of toxic substances and their use came the need for effective antidotes for accidental poisonings. In 1312, the Jewish philosopher, Maimonides published “Poisons and Their Antidotes” in Arabic; this text synthesized all knowledge available at the time and served as a guide to physicians for several centuries. Both poisoners and “unpoisoners” were interested in discovering what amount of poison and antidotes were necessary and sufficient for the job. When experiments began to focus on pinpointing these critical levels of chemicals, toxicology was in full swing.

Paracelsus (1493-1541), a Swiss physician and alchemist, promoted theories of disease that were an odd mix of scientifically advanced notions and fanciful superstitions. Among today toxicologists, he is noted for his recognition that “All things are poison and nothing [is] without poison. Solely the dose determines that a thing is not a poison.” This remark decorates the beginning of most toxicology texts and holds true today.
Toxicology initially looked chiefly at how much of any substance could kill people and the antidotes to block these toxins. The use of poison gas during World Wars I and II had made researching the acute and immediate impacts of paramount importance. In 1927, J.W. Trevan developed the first test of a compound’s toxicity by taking a small group of animals and measuring the amount that could kill half of them quickly. This was called the lethal dose fifty or LD50. A typical test would be to administer a high dose of the toxin to 10 rats for about 14 days. In the group receiving the highest dose, perhaps 8 would die, 6 in the next highest group, 3 in the next, and 1 in the group receiving the lowest dose. By graphing the dose-response data, the LD50 would be determined as the dose that on average would kill 5 of the 10 rats (50%).

The LD50 has become a standard measure of a chemical’s acute toxicity and can be used to rank the potency of chemicals, but only at relatively high concentrations needed to cause death. There is a caveat in comparing LD50 potency. In Exhibit 1-1, the shapes of the dose-response curves are similar for each graph and the LD50 would be useful in defining potency; but in Exhibit 1-2, dissimilar shapes can result when the mechanism of action between the two chemicals is different. Chemical B is more potent at low doses than chemical A, but the opposite is true at higher doses. In this scenario, use of the LD50 would not be helpful in comparing potency.

**Exhibit 1-1: Sample Dose-Response Curves with Similar Shapes**
In 1950, the standard test for a toxicant was simple: if the exposed animal did not drop dead on the spot, the chemical studied was considered safe. Long term impacts of lower levels of exposure were unknown. Davis (2002) reveals in the book, “When Smoke Ran Like Water,” that after the Donora air pollution episode of 1948, Mary Amdur, a researcher at Harvard Medical School, began to study on what caused the sudden and unexplained deaths and illness in this small mill town. Philip Drinker and Amdur began research on the effects to the lungs caused by long-term exposure to sulfuric acid and particles. This research was funded directly by the American Smelting and Refining Company (ASARCO). They used guinea pigs because they breathe through their mouths, just like people, allowing pollutants to reach more deeply into their lungs. In 1952, Amdur found that the more acid in the air, the more damage to the lungs. The smaller the particles involved, the more deeply they penetrated, and the greater the impact. She found strikingly similar effects in both animals and people; heavy scarring and thickened linings deep inside the lower lung. This opened up a whole new world of research. Unfortunately, this was not the sort of work ASARCO thought it was funding. In 1953, after presenting her findings at a science association meeting, some heavy-handed efforts to suppress her research ensued. Her head professor, Drinker, ordered her to remove his name from their jointly prepared paper on the study that she presented. Drinker also insisted that she withdraw the paper from the publication in *Lancet*. When she refused, her position was eliminated in 1953, after ten years of working with the eminent Professor Drinker. The *Lancet* never published their paper, and it seemed to
have disappeared. Mary Amdur had opened up a whole new world of research. In 1989, the Society of Toxicology awarded her the Herbert Stokinger Award for outstanding contributions.

During the past 125 years, scientists created over 100,000 compounds not in nature. Following World War II, there was a dramatic expansion of the role of chemicals, especially man-made chemicals, in the United States. The vast majority of these chemicals have no toxicity information. A 1985 National Academy of Science report stated that there were more than 5 million chemicals in the United States, and we were exposed to about 60,000 to 70,000 of these chemicals. Of these exposed chemicals, 84% had minimal to no toxicity information, 14% had sufficient testing for a partial hazard assessment, and only 2% had sufficient testing for a complete hazard assessment.

Exhibit 1-3, developed from a 1984 publication from the National Resource Council, shows that proportionately more testing has been done on pesticides and drugs than most other chemicals. This is because of concerns uncovered from toxic tragedies and the increased public awareness to the dangers of toxic chemical exposure from drugs and pesticides usage.
One toxic drug tragedy happened when over 100 people died of acute kidney failure after ingesting the antibiotic, sulfanilamide, which had been improperly prepared by using diethylene glycol (today used in antifreeze). This resulted in the passage of the modern version of the Food, Drug, and Cosmetic Act in 1938.

Public awareness to the dangers of pesticides was increased with the publication of *Silent Spring* by Rachel Carson (1962), a biologist from Maryland. As a result of her book, the link between DDT and the eggshell thinning of bird eggs was scientifically documented which led to the banning of DDT in the United States in the early 1970s. (However, DDT is still being used today in developing countries because it is the most effective pesticide against rampant and troublesome diseases such as malaria.) *Silent Spring* was immensely popular and influential. Carson’s work almost single-handedly created modern society’s fears about synthetic chemicals in the environment and fostered renewed interest in the science of toxicology.

### 1.2 Legislative History of Air Toxics

By 1950, despite many positive changes in society and the economy, we still were unaware of the adverse effects of air pollution. Our growing middle class became concerned about air pollution mostly because of the Donora air pollution episode of 1948. An investigation of the Donora episode by the U.S. Department of Health Education and Welfare (HEW) resulted. HEW could produce no direct evidence of air pollution’s harm, but was instrumental in passage of the 1955 Air Pollution Control Act. Their awareness was stimulated mostly by the Donora air pollution episode of 1948. An investigation of the Donora episode by the U.S. Department of Health Education and Welfare (HEW) resulted. It could produce no direct evidence of air pollution’s harm, but was instrumental in the passage of the first federal legislation concerning air pollution: the 1955 Air Pollution Control Act. President Eisenhower signed this Act authorizing $5 million annually for five years to support federal research on the effects of air pollution and to provide states with technical assistance and training in air pollution. This was the first federal litigation concerning air pollution and was the beginning of federal involvement on a national level of our air pollution problem.

In 1970, the Clean Air Act (CAA) Amendments defined two principal types of air pollution for regulation: criteria air pollutants and hazardous air pollutants. (The term “criteria” pollutants originated in the Act from “criteria documents” by HEW in the late 1950s and 1960s, which studied such pollutants such as particulates, sulfur dioxide, etc.) The CAA Amendments, under Section 108, defined the term “criteria air pollutant” as those that “cause or contribute to air pollution that may reasonably be anticipated to endanger public health or welfare … the presence of which in the ambient air results
from numerous or diverse mobile or stationary sources.” The Environmental Protection Agency (EPA) set National Ambient Air Quality Standards (NAAQS) for criteria pollutants that required protection to the public health with an “adequate margin of safety.” This was a “health-based” standard that did not take implementation costs into consideration and protected people regardless of age or their health status.

The 1970 CAA Amendments also required, under Section 112, regulation of hazardous pollutants and defined a “hazardous air pollutant” (HAP) as those “air pollutants which may reasonably be anticipated to result in an increase in mortality or an increase in serious irreversible or incapacitating reversible, illness.” The EPA was required to list substances that met the definition of hazardous air pollutants and to publish national emission standards (NESHAP) providing an “ample margin of safety” to protect the public health from these HAPs. Once a HAP was listed, the EPA had six months to propose a NESHAP and schedule public hearings. After another six months, EPA was required to publish a final NESHAP or issue a determination that the substance was not a HAP. (The CAA and its subsequent amendments always address air toxics as hazardous air pollutants. The term “toxic” was not applied to air pollution until 1986 with the passage of the Emergency Planning and Community Right to Know Act.)

In the 1970s, President Nixon declared war on cancer. Therefore, EPA in the 70s & 80s, concentrated mainly on carcinogens when listing a hazardous air pollutant. But toxic effects of air pollution can also cause serious irreversible illnesses, including kidney damage, liver damage, nervous system damage, birth defects, developmental problems in children, etc. Between 1970 and 1990, EPA listed only eight HAPs: arsenic, asbestos, benzene, beryllium, radionuclides, vinyl chloride, coke oven emissions and mercury. All of these are carcinogens except mercury. The neurological effects of mercury on the central nervous system were known in 1970 and were a subject of public concern. This was the most likely reason that EPA listed the noncarcinogen.

EPA’s attempt to regulate HAPs under the 1970 CAA (and its 1977 Amendment) met with little success (issued only seven NESHAPs). Perhaps the greatest problem EPA had in listing HAPs was the difficulty in conducting the necessary risk analysis to determine at what level the emission limits would provide for an “ample margin of safety” protection to public health. In 1976, EPA’s position was that, in the absence of strong evidence to the contrary, it would assume that for a carcinogen there was no atmospheric concentration that would pose health risk. Thus, for carcinogens, EPA believed no emissions could be allowed. If EPA prohibited certain emissions, it would force closure of plants that emitted the pollutant. This would force the shutdown of whole or many parts of businesses. Thus EPA concluded that this approach was impractical. Therefore, EPA used a second approach that would determine what action is necessary to reduce the
risk while taking costs and technical feasibility into consideration. This “technology-based” approach would be used whenever emission prohibition would result in widespread industrial closure.

The EPA adopted its vinyl chloride NESHAP using this second approach. The National Resource Defense Council (NRDC) sued contending that EPA was not allowed to consider costs in setting the health-based standard under Section 112’s “ample margin of safety.” In **NRDC v. EPA** (1987), Judge Bork, of the U.S. Circuit Court of Appeals for the District of Columbia, found that cost could not be considered when establishing a “safe” level of exposure to toxic air pollutants under Section 112, but “safe” did not mean risk free. The court concluded that it is only in establishing a second step – the “ample margin of safety” step – that the EPA can consider cost and technological feasibility. Thus EPA was now mandated to take a two-step process in setting a NESHAP. The first step would determine what is “safe”. EPA’s decision on what is “safe” must be based only on human health effects while no cost or technical feasibility will be considered. The second step would determine “ample margin of safety.” Once EPA determines what a “safe” level is, it sets the regulation to allow less emission in which cost and technical feasibility can be considered. This will provide an “ample margin” beyond what is safe.

In 1989, EPA passed its new “risk policy.” What is “safe” would be exposure to a HAP that would not cause a maximum individual risk greater than 1 in 10,000. (Maximum individual risk is the estimated risk that a person living near a plant would have if exposed to the maximum [highest annual average] pollutant concentration for 70 years [a lifetime].) Risks between 1 in 10,000 and 1 in 1,000,000 are acceptable risk ranges that EPA can consider cost and technical feasibility to see if further emission reductions are needed. This would provide an “ample margin of safety” protection beyond what is “safe” and would protect the greatest number of persons possible to an individual lifetime risk no greater than 1 in 1,000,000 considering cost and technically feasibility. (Individual lifetime risk is the same as maximum individual risk except that it uses the average pollution concentration instead of maximum.)

EPA’s “ample margin of safety” protection is set at 1 in a million. The reason it was set so high is that exposure to HAPs are involuntary risks. A person does not voluntarily expose themselves to HAPs. An example of a voluntary risk is someone who drives a car all his life. His chances of dying in of a car accident are about 1 in 50; Very high, but these are risks that a person voluntarily exposed himself by driving.
1.3 The Risk Assessment Process

1.3.1 Basics of the Risk Assessment Process

**Risk Assessment:** It is the process used to estimate the increased risk of health problems in people who are exposed to different amounts of toxic substances. Risk assessment is a process made up of 4 steps: hazardous identification, dose-response assessment, exposure assessment, and risk characterization (Exhibit 1-4).

**Exhibit 1-4: The Four Step Risk Assessment Process**

- **Hazard Identification:**
  What health problems are caused by the pollutant?

- **Exposure Assessment:**
  How much of the pollutant do people inhale during a specific time period? How many people are exposed?

- **Dose Response Assessment:**
  What are the health problems at different exposures?

- **Risk Characterization:**
  What is the extra risk of health problems in the exposed population?

**Hazard Identification:** In hazard identification, scientists evaluate what health problems are caused by toxic air pollutants. All available information is evaluated about the effects of a possible toxic chemical to estimate the likelihood that it will cause health problems in humans. Health problems can include cancer, respiratory irritation, nervous system problems and birth defects. The better the evidence, the more certain the scientists can correlate toxic air pollutants with specific health problems. The amount, type, and quality of evidence are all important. The best type of evidence comes from human studies. Because human information is very limited for most toxic air pollutants,
scientists often conduct studies on laboratory animals, such as rats. Scientists can study a variety of health effects by exposing animals to pollutants at varied concentrations and for varied time periods. When relying on animal studies only, scientists need to be satisfied that health effects in humans are likely to be the same as those in the animals tested.

**Exposure Assessment:** Exposure assessment estimates the amount of a pollutant people inhale during a specific time period, as well as how many people are exposed. The first step in an exposure assessment is to decide the source(s) of the concerned pollutant. Once the identity and location of the source(s) are known, the next step is to determine the amounts of the toxic air pollutant released in a specific time period and how it moves away from the source(s). Scientists use either monitors or computer models to estimate the amount of pollutant released from the source and the amount of pollutant at different distances from the source. Monitors are used to sample the air and measure the amount of the pollutant present. Computer models use mathematical equations to represent the processes that occur when a facility releases a pollutant and also the movement of pollutants through the air. Factors such as distance from the source to exposed persons, wind speed and direction, and smokestack height (for factories) affect these estimates. The final step in an exposure assessment is to estimate the amounts each person inhales. To do this, scientists combine estimates of breathing rates and lifespan of an average person with estimates of the amount of pollutant in that person’s air.

**Dose-Response Assessment:** Dose-response answers the question of what are the health problems at different exposures? The dose-response relationship for a specific pollutant describes the association between exposure and the observed response (health effect). In other words, it estimates how different levels of exposure to a pollutant change the likelihood and severity of health effects. Just as in the hazard identification, scientists use results of animal and human studies to establish dose-response relationships.

In the absence of clear evidence to the contrary, EPA assumes that there are no exposures that have "zero risk" -- even a very low exposure to a cancer-causing pollutant can increase the risk of cancer. EPA also assumes that the relationship between dose and response is a straight line -- for each unit of increase in exposure (dose), there is an increase in cancer response (See Exhibit 1-5).
A dose may exist below the minimum health effect level for which no adverse effects occur. EPA typically assumes that at low dose exposure the body's natural protective mechanisms repair any damage caused by the pollutant, so there is no ill effect at low doses (See Exhibit 1-6). The point on the dose-response curve where no adverse effects begins is called the “no observed adverse effect level” (NOAEL).

**Risk Characterization:** Risk characterization addresses the question of what is the extra risk to health? Risk information is presented in different ways to illustrate how individuals or populations may be affected. Some of the most common risk measures are described here.

**Maximum Individual Lifetime Cancer Risks:** Combining the results of the exposure assessment and the dose-response assessment gives an estimate of the increased lifetime risk of cancer for an individual exposed to the maximum predicted long-term concentration.

\[
\text{Maximum Individual Lifetime Cancer Risk} = \text{Maximum Lifetime Exposure} \times \text{Dose-Response Relationship}
\]

**Population Risks:** Many people may be exposed to less than the maximum level. Depending on the amount of exposure, an individual's risk of cancer will vary. The population risk is usually expressed as the number of people estimated to be at various levels of risk. This is a distribution of population by individual risk levels.

**Cancer Incident Rates:** Refers to the expected increased incidence of cancer (that is, the number of new cases each year) for all people exposed to the pollutant. For example, the estimated cancer incident rate may be the number of new cancer cases per year expected among residents with the same cancer risk level.

**Non-cancer Hazards:** Health reference levels refer to exposure levels that will not cause significant hazards of non-cancer health effects. Long-term exposure, to levels below these levels, is assumed to produce no ill effects.
Health reference levels are an example of one index that government agencies use in characterizing non-cancer health risks. These levels are generally developed from exposure levels that do not produce ill effects in experimental animals. These exposure levels are adjusted to account for animal-human differences (such as breathing rate) and for underlying uncertainties (such as the difference in sensitivity between healthy adults and more sensitive people like children and the elderly).

Risk analysts then compare the health reference levels with the exposure estimates to determine how many people are exposed to concentrations higher than the health reference level. Some of these people might experience ill effects.

**Uncertainty in Risk Estimates:** Although scientists can estimate risks caused by toxic air pollutants in animals experimentally or in humans who have unusual exposures, converting these estimates to those expected in people under a wide range of conditions is difficult, and can be misleading.

By their nature, risk estimates cannot be completely accurate. The main problem is that scientists lack enough information on actual exposure and on how toxic air pollutants harm human cells. The exposure assessment often relies on computer models when the amount of pollutant getting from the source(s) to people can't be easily measured. Dose-response relationships often rely on assumptions about the effects of pollutants on cells for converting results of animal experiments at high doses to human exposures at low doses. When information is missing or uncertain, risk analysts generally make assumptions that tend to prevent them from underestimating the potential risk; that is, these assumptions provide a margin of safety for protection of human health.

### 1.3.2 History of the Risk Assessment Process for Air Toxics

Humans have always assessed risks associated with their activities and made decisions on those assessments. In the mid-seventeenth century, Blaise Pascal turned risk analysis into a science by calculating the probabilities associated with gambling. In the beginning of the twentieth century, actuaries were calculating risks and probabilities in issuing insurance premiums. With the advent of nuclear power, scientists expanded this process into a science called risk assessment.

In the 1970s public concerns of environmental carcinogens grew to new heights. As a result, EPA started to conduct human health risk assessments based largely from earlier scientific information and techniques used in calculating risks associated with radiation exposure. These types of risk assessments were difficult to develop because of a substantial lack of scientific understanding of many of the adverse effects resulting from
environmental exposures. EPA first used risk assessments while confronting potential hazards associated with pesticide use. For example, after considering available human and non-human toxicity data, EPA restricted domestic use of DDT and other pesticides, in part due to their cancer risks. EPA acknowledged that such risk-based regulations needed an appropriate scientific basis and began collecting cancer toxicity information on pesticides through administrative hearings and testimony. Summary documents from these hearings became known as the “Cancer Principles.” Criticism of these documents, which many inadvertently perceived to be formal Agency cancer risk assessment policy, led the Agency to develop the *Interim Risk Assessment Guidelines for Suspected Carcinogens* in 1976. Three years later, the Interagency Regulatory Liaison Group (a conglomeration of several federal agencies, including EPA) published additional cancer risk assessment guidelines. Concurrently, EPA used cancer risk assessment techniques in its toxic chemicals regulation under the 1976 Toxic Substances Control Act. By the end of EPA’s first decade in existence, the Agency used risk assessment techniques to develop water quality criteria protective of human health.

Throughout the 1980s, EPA increasingly utilized risk assessment to evaluate the potential for chemicals to cause non-cancer health effects and cancer risks. During the 1980s, the Agency used cancer risk assessment techniques in the development of national emission standards for air toxics such as vinyl chloride and benzene.

As EPA increased its use of risk assessment throughout the 1980s, the Agency’s inconsistent approach to risk assessment became apparent, largely due to a lack of standard guidance on the topic. To correct this problem, the Agency undertook administrative reforms and published several key guidelines and other policy documents.

In the 1980s, the emerging practice of federal-level risk assessment spurred Congress to commission a report from the National Research Council (NRC) of the National Academy of Sciences (NAS) on how the process was being used. The result was the landmark 1983 study, *Risk Assessment in the Federal Government: Managing the Process*. The document is often referred to as “The Red Book” because of its distinctive red cover. The Red Book acknowledged that regulatory agencies have differing statutory obligations that require some flexibility in both the risk assessment and risk management processes. The Red Book clarified what risk assessment and risk management are, and it separated the functions of risk assessment from the regulatory functions of making policy decisions (risk management). The Red Book also identified, for the first time, to the EPA the four step risk assessment process and recommended that EPA establish a Risk Assessment Council (RAC). In 1986, EPA established the RAC to oversee all aspects of the Agency’s risk assessment process. This group established EPA’s fundamental policies for conducting risk assessments and evaluating risk information. Also, in 1984,

After 1986, EPA began publishing an influential series of Agency-wide guidelines in the *Federal Register* identifying the recommended methods for assessing human health risks from environmental pollution. EPA did not intend for these guidelines, which cover both cancers and non-cancer hazards, to be static, and the Agency has revised the guidelines as new information and methods became available. EPA’s risk assessment guidelines are available at EPA’s “Risk Assessment Portal”: [http://www.epa.gov/risk/guidance.htm](http://www.epa.gov/risk/guidance.htm). The following is a current list of EPA’s major risk assessment guidelines:

- Carcinogen Risk Assessment (2005)
- Chemical Mixtures Risk Assessment (1986)
- Developmental Toxicity Risk Assessment (2000)
- Exposure Assessment (1992)
- Mutagenicity Risk Assessment (1986)
- Reproductive Toxicity Risk Assessment (1996)
- Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens (2005)

In the 1990 Clean Air Act (CAA) amendments, Section 112(o), Congress recognized the growing importance of quantitative risk assessment in the regulatory process by requiring EPA to enter into a contract with the NRC to evaluate the risk assessment methods EPA was using at the time. The NRC’s 1994 report, *Science and Judgment in Risk Assessment*, was prepared by the NRC’s Committee on Risk Assessment of Hazardous Air Pollutants. This report is now referred to as the “Blue Book.” In a sense, the Blue Book was a follow-up to the 1983 Red Book, but with a specific emphasis on EPA’s scientific methods. The NRC Committee observed that several themes were common to all elements of the risk assessment process and noted that these themes were usually the focal points for criticisms of individual risk assessments:

- The use of default assumptions
- Available data
- Uncertainty and variability
- Assessment of multiple chemical exposures, multiple routes of exposure, the potential for multiple adverse effects
Steps taken to validate the methodologies used throughout the risk assessment process

In the Blue Book, the NRC updated the risk assessment/risk management paradigm and also presented several recommendations for increasing the effectiveness and accuracy of EPA’s risk assessment and risk management process, particularly as it pertained to air toxics.

The 1990 CAA Amendments, Section 303, mandated the formation of a Presidential Commission on Risk Assessment and Risk Management (CRARM) in response to unresolved questions about EPA’s approach to assessing public health risks remaining after implementation of the maximum achievable control technology (MACT) program (i.e., technology based control). CRARM released its report, Risk Assessment and Risk Management in Regulatory Decision-Making, or the “White Book,” in two volumes in 1997. Volume I of the White Book focuses on the framework for environmental health risk management. Volume II addresses a variety of technical issues related to risk assessment and risk management, including a common metric for assessment of cancer and other effects, management of residual risks from air toxics, comparative risk, decision criteria, uncertainty analysis, and recommendations to specific agencies. The White Book also made several recommendations; one was that EPA should implement a tiered approach to managing residual risks after implementation of the 1990 CAA Amendment’s technology-based maximum achievable control technology (MACT) standards. A tiered approach is a process for a systematic, informed progression from a relatively simple to a more complex risk assessment approach.

The 1990 CAA Amendments, Section 112(f)(1), required EPA to investigate and report to Congress on a variety of topics pertaining to methods of calculating residual risks associated with air toxics emissions from stationary sources remaining after the implementation of MACT standards. As a result, in March of 1999, EPA released its Residual Risk Report to Congress. While the report did not specify a particular method for conducting risk assessments, it outlined a general framework for assessing residual risk and recommended a screening process utilizing CRARM’s 3-tiered approach to risk assessments.

Recently, EPA’s Office of Air Quality Planning and Standards developed methods and guidance for conducting facility-specific and community-scale air toxics assessment. This is shown on the EPA’s TTN web site as a three-volume risk assessment library for air toxics. Volume 1 provides the basics of risk assessment, risk management, and risk communication. It also provides information on additional special topics related to the
types of issues you may come up against while performing risk assessments. Volume 2 provides information on how to apply the information in Volume 1 to a specific source at a facility, or the facility as a whole. Volume 3 provides information on how to perform risk assessment at the community level, where folks are concerned about the cumulative impact of multiple facilities as well as mobile and possibly other sources.

Today, an important group within EPA with a risk assessment focus is the National Center for Environmental Assessment (NCEA). NCEA is a major component of the EPA’s Office of Research and Development and acts as EPA’s national resource center for human health and ecological risk assessment. NCEA conducts risk assessments, carries out research to improve the state-of-the-science of risk assessment, and provides guidance and support to risk assessors. Many of the critical Agency documents on risk assessment science and policy, as well as risk related databases such as the Integrated Risk Information System (IRIS), can be accessed through the NCEA website (www.epa.gov/ncea).

1.4 Emergency Planning and Community Right to Know Act (EPCRA)

One of world’s worst industrial disaster occurred in Bhopal, India in December 1984. Union Carbide accidentally released about thirty tons of methyl isocyanate (a light-weight chemical in a vapor state) into the city’s night air. The accident occurred over a matter of minutes. The toxic pesticide killed about 2,500 people and injured or permanently disabled 200,000 more. The deaths were mostly due to pulmonary edema (fluid in the lungs), which resulted from the direct irritation of airways in the lungs. Permanent disabilities included blindness from burning in the eyes.

A subsequent chemical release from a West Virginia facility sent about 100 people to the hospital. These two accidental releases drew attention to this toxic problem. The states started to develop their own toxic air programs because they were dissatisfied with EPA’s progress in its hazardous air program. Congress responded, in 1986, by passing the Emergency Planning and Community Right to Know Act (EPCRA). The primary goals of EPCRA are to provide the public access to information concerning hazardous chemicals present in the community and to use this information to adopt local emergency response plans in the event of a hazardous chemical release. EPCRA will achieve these goals via two mechanisms. First, it establishes the creation of state and local emergency planning bodies as well as the development and implementation of local emergency plans. Second, it requires certain facilities to provide detailed reports concerning the presence and health effects of specified chemicals and releases.
EPCRA establishes requirements for federal, state and local governments, Indian tribes, and industry regarding emergency planning and "Community Right-to-Know" reporting on hazardous and toxic chemicals. The Community Right-to-Know provisions help increase the public's knowledge and access to information on chemicals at individual facilities, their uses, and releases into the environment. States and communities, working with facilities, can use the information to improve chemical safety and protect public health and the environment.

EPCRA has four major provisions: emergency planning (Sections 301-303), emergency release notification (Section 304), hazardous chemical reporting requirements (Sections 311 and 312), and toxic chemical release inventory (Section 313). The emergency planning provision establishes the creation of state and local governmental emergency bodies, requires the local bodies to prepare “emergency response plans,” and requires that facilities that have an “extremely hazardous chemical” present over a threshold amount must report it. The emergency release notification provision requires that certain applicable facilities report “releases” immediately. The hazardous chemical reporting requirements provision require (Section 311) that facilities submit a list of their Material Safety Data Sheet chemicals (all hazardous chemicals under OSHA) present at their site (over threshold amounts), and require (Section 312) that facilities submit chemical inventories annually of all hazardous chemicals at their site. Regulations implementing EPCRA are codified in Title 40 of the Code of Federal Regulations, parts 350 to 372. EPA’s “Emergency Planning” web page gives an overview of EPCRA and explains its key provisions at http://www.epa.gov/emergencies/content/lawsregs/epcraoverview.htm.

The following are a list of EPCRA’s key provisions from EPA’s web site:

- **Sections 301 to 303. Emergency Planning:** Local governments are required to prepare chemical emergency response plans, and to review plans at least annually. State governments are required to oversee and coordinate local planning efforts. Facilities that maintain Extremely Hazardous Substances (EHSs) on-site in quantities greater than corresponding Threshold Planning Quantities (TPQs) must cooperate in emergency plan preparation.

- **Section 304. Emergency Notification:** Facilities must immediately report accidental releases of EHS chemicals and "hazardous substances" in quantities greater than corresponding Reportable Quantities (RQs) defined under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) to state and local officials. Information about accidental chemical releases must be available to the public.
- **Sections 311 and 312. Community Right-to-Know Requirements**: Facilities manufacturing, processing, or storing designated hazardous chemicals must make Material Safety Data Sheets (MSDSs) describing the properties and health effects of these chemicals available to state and local officials and local fire departments. Facilities must also report, to state and local officials and local fire departments, inventories of all on-site chemicals for which MSDSs exist. Information about chemical inventories at facilities and MSDSs must be available to the public.

- **Section 313. Toxics Release Inventory**: Facilities must complete and submit a Toxic Chemical Release Inventory Form annually for each of the more than 600 Toxic Release Inventory (TRI) chemicals that are manufactured or otherwise used above the applicable threshold quantities.

The chemicals covered by each of the sections are different, as are the quantities that trigger reporting. Exhibit 1-7 summarizes the chemicals and thresholds.

**Exhibit 1-7: EPCRA Chemicals and Reporting Thresholds**

<table>
<thead>
<tr>
<th>Chemicals Covered</th>
<th>Section 302</th>
<th>Section 304</th>
<th>Sections 311/312</th>
<th>Section 313</th>
</tr>
</thead>
<tbody>
<tr>
<td>356 extremely hazardous chemicals</td>
<td>&gt; 1,000 substances</td>
<td>50,000 products</td>
<td>650 toxic chemicals &amp; categories</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Thresholds</th>
<th>Section 302</th>
<th>Section 304</th>
<th>Sections 311/312</th>
<th>Section 313</th>
</tr>
</thead>
<tbody>
<tr>
<td>Threshold Planning Quantity (TPQ) 1-10,000 pounds on site at any one time</td>
<td>Reportable quantity, 1-50,000 pounds, released in a 24-hour period</td>
<td>TPQ or 500 pounds for Section 302 chemicals; 10,000 pounds on site at any one time for other chemicals</td>
<td>25,000 pounds/yr manufactured or processed; 10,000 pounds/yr used; certain persistent bio-accumulative toxics have lower thresholds</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reporting Requirements</th>
<th>Section 302</th>
<th>Section 304</th>
<th>Sections 311/312</th>
<th>Section 313</th>
</tr>
</thead>
<tbody>
<tr>
<td>One time notification to the state emergency response commissions (SERC)</td>
<td>Each time a release above reportable quantities occur, report to SERC &amp; local emergency planning commission (LEPC)</td>
<td>311: one time report to SERC &amp; LEPC, &amp; fire department 312: Annually to all of the above</td>
<td>Annually to EPA and the State</td>
<td></td>
</tr>
</tbody>
</table>
1.4.1 Toxic Release Inventory

EPCRA Section 313 (commonly referred to as the Toxics Release Inventory or TRI) requires certain facilities to complete a Toxic Chemical Release Inventory form annually for specified chemicals. This information is available to everyone. Citizens can obtain general TRI information by calling the EPA Right to Know Information Hotline. They also can obtain copies of the original reports companies submit from either the EPA or from state agencies.

EPA’s original TRI database provided substantial information in defining the scope of the nation’s air toxics problem. Of the original 189 chemicals listed in the 1990 CAA Amendments, 170 of these chemicals came from the TRI list. However, TRI reports are required only from the manufacturing sector of U.S. businesses, and there are many non-manufacturing sources of air pollution.

Manufacturing facilities that are required to report a TRI include a designated facility (by industrial sector: SIC codes), and employment of at least ten full time employees, and uses in excess of 10,000 pounds/year or manufactures or processes in excess of 25,000 pounds/year of a listed toxic chemical (650 chemicals), or lesser amounts of other persistent, bio-accumulative, and toxic chemicals (i.e., 0.1 grams/year of dioxin).

The Toxic Chemical Release Inventory form must be submitted to EPA and the State on July 1 and covers releases and other waste management of toxic chemicals that occurred during the preceding calendar year. One purpose of this reporting requirement is to inform the public and government officials about releases and other waste management of toxic chemicals. The following information is required on the form:

- Name, location, and type of business
- Whether the chemical is manufactured (including importation), processed, or otherwise used and the general categories of use of the chemical
- An estimate (in ranges) of the maximum amounts of the toxic chemical present at the facility at any time during the preceding year
- Quantity of the chemical entering the air, land, and water annually
- Off-site locations to which the facility transfers toxic chemicals in waste for recycling, energy recovery, treatment or disposal
- Waste treatment/disposal methods

The Toxics Release Inventory (TRI) is a database that contains detailed information on nearly 650 chemicals and chemical categories that applicable facilities manage through
disposal or other releases, recycling, energy recovery, or treatment. The TRI information collected is broken down between on-site disposal, releases, or other waste management methods and off-site transfers for disposal, releases or other waste management methods. On-site elimination of TRI chemicals includes disposals or releases to air, water, land, or underground injection. On-site waste management methods include recycling, energy recovery, or treatment. TRI chemicals can also be transferred off-site for disposal, release, or other waste management methods. Off-site elimination of TRI chemicals includes the same as on-site except for the addition of releases or disposals to publicly owned treatment facilities.

For 2006, 22,880 facilities reported to EPA’s TRI Program. They reported 4.25 billion pounds of on-site and off-site disposal or other releases of almost 650 toxic chemicals. Of these TRI chemicals, 33% were released into the air. Concerning on-site and off-site disposal/releases of these chemicals, almost 88% of the total was disposed/released on-site; 12% was sent off-site. Exhibit 1-8 shows that metal mining facilities were responsible for 29% of the 2006 TRI chemical disposal/releases, and electric utilities coming in a close second with 24%.

**Exhibit 1-8: Toxic Release Inventory, 2006**

![Pie chart showing disposal or other releases by category.](chart)

In 1988, the TRI showed that about 2.4 billion pounds of toxic chemicals were released into the air. In 1989, an EPA risk assessment showed that 2,700 cancer cases occur each year as a result of air exposure to EPCRA toxic pollutants. This information had a
significant effect on the public perception of the hazardous chemical problem and helped spur Congress to amend the CAA hazardous air pollution program in 1990.

For more information about the TRI program, visit EPA’s TRI web site at www.epa.gov/tri. At this site there is also a multitude of TRI training and assistant materials coupled with extensive TRI reporting aids.
References


Chapter 2: Regulation of Air Toxics

2.1 Pre-1990, Air Toxic Program

Although several major environmental laws were passed during the 1980s to regulate hazardous substances, hazardous waste, and toxic chemicals, there was little movement by the EPA on regulating hazardous air pollutants (HAPs) or air toxics. From 1970 to 1990, EPA listed only eight HAPs and established national emission standards for only seven of them (asbestos, benzene, beryllium, inorganic arsenic, mercury, radionuclides, and vinyl chloride). The reason was that EPA was involved in many legal, scientific, and policy debates over which pollutants to regulate and how stringently to regulate them.

Prior to 1990, the Clean Air Act (CAA) directed EPA to regulate toxic air pollutants based on the risks each pollutant posed to human health. Specifically, the Act directed EPA to:

- Identify all pollutants that caused “serious and irreversible illness or death,” and
- One year after identifying HAPs, EPA had to promulgate standards to reduce emissions of the HAPs to levels that provided an “ample margin of safety” protection for the public.

At that time, EPA’s problems in regulating HAPs were extensive. One of EPA’s problems was the unrealistic 1970 statutory timeframe of setting a national emission standard for hazard air pollutant (NESHAP). EPA was mandated to promulgate a NESHAP one year after listing a HAP. There was little available data on air toxics and EPA had problems in defining the “ample margin of safety” protection. Debates ensued on whether EPA had to reduce all risks, regardless of cost or technical feasibility in establishing an “ample margin of safety” protection. This could possibly be read (as the environmentalist did) to require the establishment of zero-risk level for emissions. Industry felt this was too harsh. This vagueness resulted in litigation which now demanded a risk, health-based approach in defining an emission standard for HAPs. As a result, EPA had a difficult time in implementing NESHAPs.

Another of EPA’s problems was that the risk analysis approach was controversial at that time. Controversies focused on risk assessment methods and assumptions, the amount of
health risk data needed to justify regulation, questions about the costs to industry and benefits to human health and the environment, and decisions about “how safe is safe.” During this time, EPA was still developing methods to assess risk. While EPA and the scientific community gained valuable knowledge about risk assessment methods through this work, the chemical-by-chemical regulatory approach—an approach based solely on risk—proved difficult.

### 2.2 Post 1990, Air Toxic Program: An Overview

In the 1990 Clean Air Act Amendments (CAAA), under Section 112, Congress completely changed the approach to hazardous air pollutants from a risk, health-based to a technology-based regulation. In these Amendments, Congress now defined a hazardous air pollutant as “any air pollutant listed in Section 112 (b).” In this Section, 189 substances were listed as hazardous air pollutants. EPA may add or delete chemicals to this list. CCA required EPA to publish a list of categories and subcategories of major and area sources of hazardous air pollutants. In July 1992, EPA published an initial list of 174 major source categories and 8 area source categories of hazardous air pollutants.

The 1990 CAAA also required EPA to pass technology-based emission standards (referred to as “maximum achievable control technology” [MACT] standards) for all major source categories (and area source categories it deemed warranted) according to a statutory schedule. The schedule required the source categories have MACT standards promulgated by the year 2000. By 2004, EPA passed all (96) MACTs standards.

In September 1992, EPA followed the source category list with a schedule for promulgation of emission standards, which specifies when each of the listed source categories will be regulated. If EPA fails to promulgate a MACT according to the source category schedule, then the “MACT hammer” applies: This hammer mandates that 18 months after the MACT due date, major sources must meet a MACT standard that is determined by the state on a case-by-case basis.

The 1990 CAAA air toxic program has two phases. In the first phase, EPA develops regulations - MACT standards - requiring sources to meet specific emissions limits that are based on emissions levels already being achieved by many similar sources in the country. In the second phase, EPA must establish additional requirements to control any “residual risk” that exists eight years after promulgation of MACT. The EPA will conduct a risk assessment to determine if any residual risk remains to the public health. If a risk remains, then EPA must pass an additional health-based standard that will provide an “ample margin of safety” to the public. These second-tier standards are known as “residual risk” standards. EPA completed development of its strategy for
addressing residual risks from air toxics in March of 1999 in its “Residual Risk Report to Congress.”

Also included in the 1990 CAAA are provisions that EPA study several specific topics. In accordance with this mandate, EPA published a number of reports to Congress (including the Mercury, Great Waters, and Utilities Reports to Congress) and continues to study these and other special topics. Additionally, EPA currently is refining its strategy for reducing risks in urban areas resulting from the emission of HAPs. EPA’s *Urban Air Toxics Strategy* released in August 1998 proposes to address the problems of cumulative exposures to air toxics in urban areas through an integrated approach that considers stationary and mobile sources of urban air toxics. These programs, in combination with the residual risk program, will provide a coordinated federal approach to address air toxics.

### 2.3 Listing of Hazardous Air Pollutants

To avoid controversy over which pollutants would be listed as HAPs, Congress included an initial list of 189 HAPs into the 1990 CAAA. EPA may add or delete (delist) pollutants from this list. Also, an individual can petition the EPA to add or delist a substance from the HAP list. The petitioner has the burden of showing adequate data to support their position. To add a substance to the list, EPA or the petitioner, must show that “emissions, ambient concentrations, bioaccumulation, or deposition of the air pollutant are known to cause, or may reasonably be anticipated to cause, adverse effects to human health or adverse environment effects.” To delist a substance, it must be shown that the substance may not reasonably be anticipated to cause “any adverse effects to human health or adverse environmental effects.” Since passage of these Amendments, EPA *and has delisted only two HAPs: caprolactam and methyl ethyl ketone* and has redefined the definition of glycol ethers (putting the current list of HAPs at 187). A listing of HAPs regulated under the CCA Section 112, is included in Exhibit 2-1. EPA also has a current list of all regulated HAPs on its Air Toxics Web page: http://www.epa.gov/ttn/atw/orig189.html.

There is some overlap between HAPs and criteria pollutants. This is important because many programs aimed at the reduction of particulate matter (PM) and volatile organic compounds (VOCs) will have a beneficial effect on air toxics (the reverse also being true). For example, ozone, which is formed by the interaction of NOx, VOC, and sunlight, will be reduced when a HAP VOC is removed as a result of a MACT reduction. Another example would be a program that reduces PM. Since PM is comprised of many chemicals, some which may contain various HAPs, a reduction in PM will also reduce the specific HAP that is in its makeup.
<table>
<thead>
<tr>
<th>CAS Number</th>
<th>Chemical Name</th>
<th>Chemical Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>75070</td>
<td>Acetaldehyde</td>
<td>334883 Diazomethane</td>
</tr>
<tr>
<td>60355</td>
<td>Acetamide</td>
<td>132649 Dibenzofuran</td>
</tr>
<tr>
<td>75058</td>
<td>Acetonitrile</td>
<td>96128 1,2-Dibromo-3-chloropropane</td>
</tr>
<tr>
<td>98862</td>
<td>Acetophenone</td>
<td>84742 Dibutylphthalate</td>
</tr>
<tr>
<td>53963</td>
<td>2-Acetylaminofluorene</td>
<td>106467 1,4-Dichlorobenzene(p)</td>
</tr>
<tr>
<td>107028</td>
<td>Acrolein</td>
<td>91941 3,3-Dichlorobenzidine</td>
</tr>
<tr>
<td>79061</td>
<td>Acrylamide</td>
<td>53963 3,3-Dichloropropene</td>
</tr>
<tr>
<td>79107</td>
<td>Acrylic acid</td>
<td>62737 Dichlorvos</td>
</tr>
<tr>
<td>107131</td>
<td>Acrylonitrile</td>
<td>111422 Diethanolamine</td>
</tr>
<tr>
<td>107051</td>
<td>Allyl chloride</td>
<td>121697 N,N-Diethyl aniline (N,N-Dimethylaniline)</td>
</tr>
<tr>
<td>92671</td>
<td>4-Aminobiphenyl</td>
<td>64675 Diethyl sulfate</td>
</tr>
<tr>
<td>62533</td>
<td>Aniline</td>
<td>119904 3,3-Dimethoxybenzidine</td>
</tr>
<tr>
<td>90040</td>
<td>o-Anisidine</td>
<td>60117 Dimethyl aminoazobenzene</td>
</tr>
<tr>
<td>1332214</td>
<td>Asbestos</td>
<td>79447 Dimethyl carbamoyl chloride</td>
</tr>
<tr>
<td>71432</td>
<td>Benzene (including benzene from gasoline)</td>
<td>68122 Dimethyl formamide</td>
</tr>
<tr>
<td>92875</td>
<td>Benzidine</td>
<td>57147 1,1-Dimethyl hydrazine</td>
</tr>
<tr>
<td>98077</td>
<td>Benztetrachloride</td>
<td>131113 Dimethyl phthalate</td>
</tr>
<tr>
<td>100447</td>
<td>Benzyl chloride</td>
<td>77781 Dimethyl sulfate</td>
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<tr>
<td>92524</td>
<td>Biphenyl</td>
<td>534521 4,6-Dinitro-o-cresol, and salts</td>
</tr>
<tr>
<td>117817</td>
<td>Bis(2-ethylhexyl)phthalate (DEHP)</td>
<td>51285 2,4-Dinitrophenol</td>
</tr>
<tr>
<td>542881</td>
<td>Bis(chloromethyl)ether</td>
<td>121142 2,4-Dinitrotoluene</td>
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<tr>
<td>75252</td>
<td>Bromoform</td>
<td>123911 1,4-Dioxane (1,4-Diethyleneoxide)</td>
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<tr>
<td>106990</td>
<td>1,3-Butadiene</td>
<td>122667 1,2-Diphenyldihydrazine</td>
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<td>156627</td>
<td>Calcium cyanamide</td>
<td>106898 Epichlorohydrin (1-Chloro-2,3-epoxypropane)</td>
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<td>105602</td>
<td>Caprolactam (Delisted)</td>
<td>106887 1,2-Epoxybutane</td>
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<td>Captan</td>
<td>140885 Ethyl acrylate</td>
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<td>63252</td>
<td>Carbaryl</td>
<td>100414 Ethyl benzene</td>
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<td>75150</td>
<td>Carbon disulfide</td>
<td>51796 Ethyl carbamate (Urethane)</td>
</tr>
<tr>
<td>56235</td>
<td>Carbon tetrachloride</td>
<td>75003 Ethyl chloride (Chloroethane)</td>
</tr>
<tr>
<td>463581</td>
<td>Carbonyl sulfide</td>
<td>106934 Ethylene dibromide (Dibromoethane)</td>
</tr>
<tr>
<td>120809</td>
<td>Catechol</td>
<td>107062 Ethylene dichloride (1,2-Dichloroethane)</td>
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<tr>
<td>133904</td>
<td>Chloramben</td>
<td>107211 Ethylene glycol</td>
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<tr>
<td>57749</td>
<td>Chlor dane</td>
<td>151564 Ethylene imine (Aziridine)</td>
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<tr>
<td>7782505</td>
<td>Chlorine</td>
<td>75218 Ethylene oxide</td>
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<tr>
<td>79118</td>
<td>Chloroacetic acid</td>
<td>96457 Ethylene thiourea</td>
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<tr>
<td>532274</td>
<td>2-Chloroacetophenone</td>
<td>75334 Ethyldiene dichloride (1,1-Dichloroethane)</td>
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<td>108907</td>
<td>Chlorobenzene</td>
<td>50000 Formaldehyde</td>
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<td>510156</td>
<td>Chlorobenzilate</td>
<td>76448 Heptachlor</td>
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<td>67663</td>
<td>Chloroform</td>
<td>118741 Hexachlorobenzene</td>
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<td>107302</td>
<td>Chloromethyl methyl ether</td>
<td>87683 Hexachlorobutadiene</td>
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<td>126998</td>
<td>Chloroprene</td>
<td>77474 Hexachlorocyclopentadiene</td>
</tr>
<tr>
<td>1319773</td>
<td>Cresols/Cresylic acid (isomers and mixture)</td>
<td>67721 Hexachloroethane</td>
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<tr>
<td>95487</td>
<td>o-Cresol</td>
<td>822060 Hexamethylene-1,6-diisocyanate</td>
</tr>
<tr>
<td>108394</td>
<td>m-Cresol</td>
<td>680319 Hexamethylphosphoramide</td>
</tr>
<tr>
<td>106445</td>
<td>p-Cresol</td>
<td>110543 Hexane</td>
</tr>
<tr>
<td>98828</td>
<td>Cumene</td>
<td>302012 Hydrazine</td>
</tr>
<tr>
<td>94757</td>
<td>2,4-D, salts and esters</td>
<td>7647010 Hydrochloric acid</td>
</tr>
<tr>
<td>3547044</td>
<td>DDE</td>
<td>7664393 Hydrogen fluoride (Hydrofluoric acid)</td>
</tr>
</tbody>
</table>
### Introduction to Air Toxics

<table>
<thead>
<tr>
<th>Code</th>
<th>Compound</th>
<th>Code</th>
<th>Compound</th>
</tr>
</thead>
<tbody>
<tr>
<td>7783064</td>
<td>Hydrogen sulfide <em>(Removed from list in 1991 – listed because of clerical error)</em></td>
<td>91225</td>
<td>Quinoline</td>
</tr>
<tr>
<td>123319</td>
<td>Hydroquinone</td>
<td>106514</td>
<td>Quinone</td>
</tr>
<tr>
<td>78591</td>
<td>Isophorone</td>
<td>100425</td>
<td>Styrene</td>
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<td>58899</td>
<td>Lindane (all isomers)</td>
<td>96093</td>
<td>Styrene oxide</td>
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<td>108316</td>
<td>Maleic anhydride</td>
<td>1746016</td>
<td>2,3,7,8-Tetrachlorodibenzo-p-dioxin</td>
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<tr>
<td>67561</td>
<td>Methanol</td>
<td>79345</td>
<td>1,1,2,2-Tetrachloroethane</td>
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<tr>
<td>72435</td>
<td>Methoxychlor</td>
<td>127184</td>
<td>Tetrachloroethylene (Perchloroethylene)</td>
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<tr>
<td>74839</td>
<td>Methyl bromide (Bromomethane)</td>
<td>7550450</td>
<td>Titanium tetraoxide</td>
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<tr>
<td>74873</td>
<td>Methyl chloride (Chloromethane)</td>
<td>108883</td>
<td>Toluene</td>
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<tr>
<td>71556</td>
<td>Methyl chloroform (1,1,1-Trichloroethane)</td>
<td>95807</td>
<td>2,4-Toluene diamine</td>
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<td>78933</td>
<td>Methyl ethyl ketone (2-Butanone) <em>(Delisted)</em></td>
<td>584849</td>
<td>2,4-Toluene diisocyanate</td>
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<tr>
<td>60344</td>
<td>Methyl hydrazine</td>
<td>95534</td>
<td>o-Toluidine</td>
</tr>
<tr>
<td>74884</td>
<td>Methyl iodide (Iodomethane)</td>
<td>8001352</td>
<td>Toxaphene (chlorinated camphene)</td>
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<tr>
<td>108101</td>
<td>Methyl isobutyl ketone (Hexone)</td>
<td>120821</td>
<td>1,2,4-Trichlorobenzene</td>
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<tr>
<td>624839</td>
<td>Methyl isocyanate</td>
<td>79005</td>
<td>1,1,2-Trichloroethane</td>
</tr>
<tr>
<td>80626</td>
<td>Methyl methacrylate</td>
<td>79016</td>
<td>Trichloroethylene</td>
</tr>
<tr>
<td>1634044</td>
<td>Methyl tert butyl ether</td>
<td>95954</td>
<td>2,4,5-Trichlorophenol</td>
</tr>
<tr>
<td>101144</td>
<td>4,4-Methylene bis(2-chloroaniline)</td>
<td>88082</td>
<td>2,4,6-Trichlorophenol</td>
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<tr>
<td>75092</td>
<td>Methylene chloride (Dichloromethane)</td>
<td>121448</td>
<td>Triethylamine</td>
</tr>
<tr>
<td>101688</td>
<td>Methylene diphenyl diisocyanate (MDI)</td>
<td>1582098</td>
<td>Trifluralin</td>
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<tr>
<td>101779</td>
<td>4,4’-Methylene diisocyanate</td>
<td>540841</td>
<td>2,2,4,Trimethylpentane</td>
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<td>91203</td>
<td>Naphthalene</td>
<td>108054</td>
<td>Vinyl acetate</td>
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<td>98953</td>
<td>Nitrobenzene</td>
<td>593602</td>
<td>Vinyl bromide</td>
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<td>92933</td>
<td>4-Nitrobenzenyl</td>
<td>75014</td>
<td>Vinyl chloride</td>
</tr>
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<td>100027</td>
<td>4-Nitrophenol</td>
<td>75534</td>
<td>Vinilidene chloride (1,1-Dichloroethylene)</td>
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<td>79469</td>
<td>2-Nitropropane</td>
<td>1330207</td>
<td>Xylenes (isomers and mixture)</td>
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<td>684935</td>
<td>N-Nitroso-N-methylurea</td>
<td>95476</td>
<td>m-Xylenes</td>
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<tr>
<td>62759</td>
<td>N-Nitrosodimethylamine</td>
<td>108383</td>
<td>p-Xylenes</td>
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<tr>
<td>59892</td>
<td>N-Nitrosomorpholine</td>
<td>106423</td>
<td>p-Xylenes</td>
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<tr>
<td>56382</td>
<td>Parathion</td>
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<td>Antimony Compounds</td>
</tr>
<tr>
<td>82688</td>
<td>Pentachloronitrobenzene (Quintobenzene)</td>
<td>0</td>
<td>Arsenic Compounds <em>(inorganic including arsine)</em></td>
</tr>
<tr>
<td>87865</td>
<td>Pentachlorophenol</td>
<td>0</td>
<td>Beryllium Compounds</td>
</tr>
<tr>
<td>108952</td>
<td>Phenol</td>
<td>0</td>
<td>Cadmium Compounds</td>
</tr>
<tr>
<td>106503</td>
<td>p-Phenylenediamine</td>
<td>0</td>
<td>Chromium Compounds</td>
</tr>
<tr>
<td>75445</td>
<td>Phosgene</td>
<td>0</td>
<td>Cobalt Compounds</td>
</tr>
<tr>
<td>7803512</td>
<td>Phosphine</td>
<td>0</td>
<td>Coke Oven Emissions</td>
</tr>
<tr>
<td>7723140</td>
<td>Phosphorus</td>
<td>0</td>
<td>Cyanide Compounds <em>(inorganic including arsine)</em></td>
</tr>
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<td>85449</td>
<td>Phthalic anhydride</td>
<td>0</td>
<td>Glycol ethers2</td>
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<tr>
<td>1336363</td>
<td>Polychlorinated biphenyls <em>(Aroclors)</em></td>
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<td>Lead Compounds</td>
</tr>
<tr>
<td>1120714</td>
<td>1,3-Propane sulfone</td>
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<td>Manganese Compounds</td>
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<tr>
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<td>beta-Propiolactone</td>
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<td>Mercury Compounds</td>
</tr>
<tr>
<td>123386</td>
<td>Propionaldehyde</td>
<td>0</td>
<td>Fine mineral fibers3</td>
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<tr>
<td>114261</td>
<td>Propoxur (Baygon)</td>
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<td>Nickel Compounds</td>
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<tr>
<td>78875</td>
<td>Propylene dichloride (1,2-Dichloropropane)</td>
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<td>Polycyclic Organic Matter <em>(inorganic including arsine)</em></td>
</tr>
<tr>
<td>75569</td>
<td>Propylene oxide</td>
<td>0</td>
<td>Radionuclides <em>(including radon)</em></td>
</tr>
<tr>
<td>75558</td>
<td>1,2-Propylenimine <em>(2-Methyl aziridine)</em></td>
<td>0</td>
<td>Selenium Compounds</td>
</tr>
</tbody>
</table>
2.4 Source Category Listing

The 1990 CAAA required EPA to publish a list of categories of major and area sources of HAPs. Although not defined in the Act, a “category” of sources is a group of sources that emit listed HAPs and have some common features. For example, the synthetic organic chemical manufacturing industry (SOCMI) was grouped as one category. The common feature among SOCMI plants was that they would produce, or use in their production, many common chemicals that were all organic listed HAPs (the MACT for the SOCMI category regulates 131 listed HAPs). Another example would be dry cleaners. All dry cleaners that use perchloroethylene (PCE) in their dry cleaning operations, a listed HAP, are considered one source category.

In July 1992, EPA published an initial list of 174 major source categories and 8 area source categories. The 1990 CAAA allowed the EPA to delist a category from this list, and for the public to petition the EPA to delist a category. For a carcinogenic HAP category, the determination was exposure to the HAP would cause no greater than one in a million cancer cases. For a non-carcinogenic HAP category, the determination was based on its adverse health or environmental impact. The EPA can add a category to the list, at any time, according to the same criteria used in making the original list. More information about the source category list can be found at EPA’s Air Toxics web page.

2.5 Definition of Major and Area Source

Under the CAAA Section 112, HAP program, a “major source” is defined as any stationary source or group of stationary sources that are contiguous and under common control that has the potential to emit considering controls 10 tons per year or more of any listed HAP or 25 tons per year or more of any combination of listed HAPs. (EPA may establish a lesser quantity of pollutant emissions for the definition of major source based on various characteristics of the pollutants being emitted – including potency, persistence, potential for bioaccumulation, or other relevant factors.)

An “area source” is any stationary source of HAPs that is not a major source.

A “stationary source,” under CAAA Section 112, HAP program, is defined as “any building, structure, facility, or installation which emits any air pollution.” It has the same meaning as in CAAA Section 111 New Source Performance Standards (NSPS) (“source” can be as small as an emission unit or as large as the entire facility). The Standard Industrial Classification (SIC)-code definition for “major source” (all the pollutant-emitting activities which belong to the same industrial grouping) and the functional relationship of the emissions units, both often relied on in Prevention of Significance

2-6
Deterioration (PSD) and New Source Review (NSR) programs, do not apply to “major sources” under Section 112.

Also, unlike PSD and NSR programs where the EPA must conduct rule making to include a source’s fugitive emissions in calculating major source status, under CAAA Section 112, the EPA concluded that fugitive emissions are included in the major source definition. Fugitive emissions from chemical plants and petroleum refineries are mostly associated with equipment leaks from certain emission units (i.e., valves, pumps, connectors, flanges, compressors, pressure relief valves, etc.). EPA has developed emission factors associated with equipment leaks at petroleum facilities and chemical plants. The EPA document “Protocol for Equipment Leak Emission Factors” is available at http://www.epa.gov/ttn/chief/efdocs/equiplks.pdf. The factors presented in this document can be used for estimating emissions of VOC HAPs by using the percentage of the given air toxic to the total VOC emission rate.

**Example of Fugitive Emission Calculation:** A chemical manufacturing facility has 145 valves in active light liquid VOC service. The material is 3% benzene. To calculate fugitive benzene emissions from valve leaks:

- The VOC emission factor from Table 2-1 in the “Protocol for Equipment Leak Emission Factors” is 0.00597 kg/hr/valve (0.0132 lb/hr/valve).

- VOC emissions would be calculated as:
  145 valves x 0.0132 lb/hr/valve = 1.914 lb/hr

- Benzene emissions would be calculated as:
  1.914 lb/hr x 3% = 0.057 lb/hr

- Annual benzene emissions would be calculated as:
  0.057 lb/hr x 8,760 hr/yr = 499 lb/yr = 0.25 tons/yr
  (Note that 8,760 hours per year is equivalent to 24-hours per day and 365 days per year.)

“Contiguous” (located at a common site) and common control (same ownership) under CAAA Section 112 have the same meaning as PSD and NSR programs.

“Potential to emit” under CAAA Section 112 is similar to the language in PSD and NSR programs. Potential to emit (PTE) is the maximum design capacity of the source after pollution controls and restrictions on hours of operation or type and amount of material.
Introduction to Air Toxics

combusted or processed. PTE refers to the amount of a certain pollutant that a facility could release into the air operating at its maximum capacity (even if the facility never actually emitted that amount). A facility’s maximum capacity is based on the amount of input material that can be used and production rates. Usually it should be assumed that the process can operate for 24-hours per day, 365 days per year. PTE, however, takes into account the design limitations of the facility, as well as any controls or limits on operations imposed in the facility’s permit.

A common type of design limitation might involve setting operating rates or operating time limits for certain equipment. For example, in a paint spraying booth at an auto-body shop, there is a limitation on the number of vehicles that can be painted and dried in a given amount of time based on the time it takes to perform each task (i.e., preparation of the painted surface, paint drying, etc.). In estimating the PTE it would not have to be assumed that the paint spraying equipment operates every hour throughout the year; however, it would have to be assumed the business operated every hour throughout the year.

Other examples would include permit limits or conditions that can be applied to reduce the PTE. Some examples would be:

- The use of pollution control devices
- Restriction on the rate or time of operation of the facility or processes
- Limitations on the amounts of raw material or fuel used

According to the EPA, limits on PTE must be “practically enforceable” (means permit written in such a manner as to provide for effective operational controls) and “federally enforceable” (limit must effectively be approved in a state implementation plan). Federal enforceability was subject to litigation in 1995 and, as a result, EPA has been using its “transition policy” for interpretation of “federally enforceable.”

Under the 1990 CAAA, “major sources” of hazardous air pollutants are required to meet MACT limitations. Also, with the advent of these Amendments, “major sources” of HAPs are required to obtain a Title V permit. Therefore, EPA’s CAAA Section 112 program, which would show a source’s compliance with applicable MACT regulations, is accomplished when the HAP source applies for and secures a Title V operating permit.
At present, EPA follows its “once-in-always-in” policy. This policy states that a major source cannot become an area source if it reduces HAP emissions below 10 tons/year for a single HAP or 25 tons/year combined (May 16, 1995 EPA policy memo). On January 3, 2007, EPA proposed a rule that would eliminate the “once-in-always-in” policy by allowing a major source that reduces its emissions under the major source requirements to become an area source. As of the date of this writing, no final rule has been promulgated.

**Example of a Major Source Calculation:** The following example shows how to determine “major source” status of a facility. Larry’s Printing Company, Curly’s Chemical Company, and Moe’s Wood Furniture Company are all owned by Lou’s Recreational Products Company and are located in the same industrial complex, but separated by a street and railroad tracks. Each of Lou’s three companies manufactures different goods and has no relationship to each other except for common ownership.

The first question asked, is what companies make up the stationary “source?” Regardless that the Printing, Chemical, and Wood Furniture companies all have different SIC codes and are not functionally related, this does not stop them from being the same stationary source under CAAA Section 112, HAP program. All three companies are contiguous (even though they are not adjacent, they are in same industrial complex), and all the companies are under common ownership by Lou’s Recreational Products. Therefore, the Printing, Chemical, and Wood Furniture companies are all part of the same stationary source.

The next question asked, is this stationary source a “major” source? A major source has the PTE considering controls at least 10 tons per year of any individual HAP or at least 25 tons per year of the total HAPs. To answer this question, all the HAP emissions from the source must be calculated to see if they are over these threshold limits.

The following information about the emission and operating conditions of all three companies will be used in making this determination:

**Printing Co:**
- Blanket wash solvent: emissions = 2 tons toluene/yr
- Fountain solution: emissions = 1 ton ethylene glycol/yr.
Chemical Co:
- Reactor controlled by a scrubber (90% efficient):
  - 60 tons styrene/yr = uncontrolled emissions
  - 6 tons styrene/yr = emissions after federal enforceable scrubber
  - 2 tons styrene/yr = fugitive emissions
- Storage tank: 4 tons toluene/yr = uncontrolled emissions

Wood Furniture Co:
- Coating line:
  - 9 tons toluene/yr = maximum emission running 24 hours a day - 7 days a week.
  - 3 tons toluene/yr = actual emissions - by limiting hours of operation to one shift (8 hrs.) a day in a federally enforceable operating permit.

All of the above emission amounts in italics are the PTE from the individual emission units. The Chemical Company’s reactor stack exit has a PTE 3 tons styrene per year (not 30 tons styrene per year) because these are the emissions “after” the control device. The reactor also has 2 tons styrene per year of “fugitive emissions” that must be added into the PTE. The Furniture Company lowered its PTE (from 9 tons toluene per year to 3 tons toluene per year) by changing its hours of operation to only one shift a day. Both the control device and the changing of operating hours must be “federal enforceable” (usually in a state operating permit that has federal oversight).

The next step is to calculate the total emission for the individual HAPs at the source and the total emissions for all HAPs at the source. Exhibit 2-2 calculates all these “potential to emit” HAP emissions. This Exhibit shows that the individual total HAP emissions of styrene, toluene and ethylene glycol are all below the major source threshold of 10 tons per year and also shows that the total of all HAP emissions from the source are below the major source threshold of 25 tons per year. Therefore, Lou’s Recreation Products Company is not a “major” source under CAAA Section 112. This results in the Company being an “area” source and not subject to Title V permitting.
Exhibit 2-2: Calculation of the Potential to Emit (PTE)

<table>
<thead>
<tr>
<th>HAP</th>
<th>Facility</th>
<th>Emission Unit</th>
<th>PTE(tons/yr)</th>
<th>Major(tons/yr)</th>
</tr>
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<tbody>
<tr>
<td>Styrene</td>
<td>Chemical Co.</td>
<td>Reactor</td>
<td>6.0</td>
<td></td>
</tr>
<tr>
<td>Styrene</td>
<td>Chemical Co.</td>
<td>Fugitive emissions</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td><strong>Total styrene</strong></td>
<td></td>
<td></td>
<td><strong>8.0</strong></td>
<td><strong>&lt; 10</strong></td>
</tr>
<tr>
<td>Toluene</td>
<td>Printing Co.</td>
<td>Wash solvent</td>
<td>2.0</td>
<td></td>
</tr>
<tr>
<td>Toluene</td>
<td>Chemical Co.</td>
<td>Storage tank</td>
<td>4.0</td>
<td></td>
</tr>
<tr>
<td>Toluene</td>
<td>Furniture Co.</td>
<td>Coating line</td>
<td>3.0</td>
<td></td>
</tr>
<tr>
<td><strong>Total toluene</strong></td>
<td></td>
<td></td>
<td><strong>9.0</strong></td>
<td><strong>&lt; 10</strong></td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td>Printing Co.</td>
<td>Fountain solution</td>
<td>1.0</td>
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<tr>
<td><strong>Total Eth. glycol</strong></td>
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<td></td>
<td><strong>1.0</strong></td>
<td><strong>&lt; 10</strong></td>
</tr>
<tr>
<td><strong>Total HAP</strong></td>
<td></td>
<td></td>
<td><strong>18.0</strong></td>
<td><strong>&lt; 25</strong></td>
</tr>
</tbody>
</table>

2.6 MACT Standards

Under 1990 CAAA Section 112, NESHAPs are now established as technology-based standards based on MACT, taking into consideration the cost of achieving such emission reduction. Congress realized the shortcomings of its previous, risk-based decision framework for stationary sources and therefore adopted this new strategy in 1990. Specifically, Congress revised Section 112 of the Act to mandate a more practical approach to reducing emissions of toxic air pollutants. This approach required that EPA identify categories of stationary sources that emit large amounts of HAPs and then develop pollution reduction regulations – called MACT standards - for those sources.

The MACT standards adopted by EPA are technology-based (not risk-based), which means EPA requires emission reductions based on an evaluation of the emission reductions that the best-performing similar sources are already achieving. Specifically, when developing a MACT standard for a particular source category, EPA looks at the level of emissions already being achieved by the best-performing similar sources through clean processes, control devices, work practices, or other methods. The CAA specifies baselines (often referred to as the “MACT floors”) for the new standards. At a minimum, a MACT standard must achieve, throughout the industry, a level of emissions control that is at least equivalent to the MACT floor. EPA can establish a more stringent standard after considering cost, non-air quality and environmental impacts, and energy requirements (Section 112(d)(2) of the CAAA).
The “MACT floors” specified in the CAAA are different for existing sources and new sources. For existing sources, the MACT floor must equal the average emissions limitations achieved by the best-performing 12 percent of sources in that source category, if there are 30 or more existing sources. If there are fewer than 30 existing sources, then the MACT floor must equal the average emissions limitation achieved by the best-performing five sources in the category. For new sources, the MACT floor must equal the level of emissions control achieved in practice by the best-controlled similar source. A new source is any source that is constructed or modified after the date of the proposed MACT. New sources must immediately comply with MACT. Existing sources have up to three years to comply.

EPA has issued MACT standards for a variety of industrial source categories, including chemical plants, oil refineries, aerospace manufacturers, and steel mills, and smaller sources; i.e., dry cleaners, commercial sterilizers, secondary lead smelters, and chromium electroplating facilities.

Exhibit 2-3 is an example of a NESHAP (MACT) standard: The 1993 NESHAP for Dry Cleaning Facilities (40 CFR 63 Subpart M).

### Exhibit 2-3 Requirements of PCE Dry Cleaning NESHAP (MACT): 1993

<table>
<thead>
<tr>
<th>Requirement</th>
<th>Small Area Source</th>
<th>Large Area Source</th>
<th>Major Source</th>
</tr>
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<tr>
<td><strong>Applicability</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dry Cleaning Facilities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>with:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Only Dry-to-Dry Machines</td>
<td>Consuming less than:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>140 gallons PCE/yr.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Only Transfer Machines</td>
<td>200 gallons PCE/yr.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Both Dry-to-Dry and Transfer Machines</td>
<td>140 gallons PCE/yr.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Process Vent Controls:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Existing Facilities</td>
<td>None</td>
<td>Refrigerated condenser (or equivalent)</td>
<td>Refrigerated condenser and small carbon adsorber (or equivalent)</td>
</tr>
<tr>
<td>New Facilities</td>
<td>Refrigerated condenser (or equivalent)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Carbon adsorbers installed on existing machines before 9/22/93 can remain</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Fugitive Controls:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Existing Facilities</td>
<td>- Leak detection/repair</td>
<td>Transfer machine systems are contained inside a room enclosure</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Store all PCE solvent &amp; waste in sealed containers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>New Facilities</td>
<td>- Leak detection/repair</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Store PCE solvent &amp; waste in sealed containers</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- No new transfer machine systems allowed</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
The first section of the NESHAP addresses applicability. It breaks dry cleaning facilities into small area sources, large area sources, and major sources. Any dry cleaning facility that is smaller than a small area source (i.e., it consumes less than 140 gallons per year of perchloroethylene (PCE) in a dry-to-dry machine) does not have to comply with this NESHAP. Other larger area and major sources have to comply. According to this NESHAP, in lieu of measuring a facility’s PTE (at least 10 tons PCE per year) it is considered “major” if the facility consumes a listed amount of PCE per year (see Exhibit 2-3 for quantities). Interestingly, a 2006 EPA fact sheet states that there are only 12 major source dry cleaners in the United States.

Also shown in Exhibit 2-3, the NESHAP requirements are more stringent depending on the size of source and if it is new or existing. For example, small existing sources are not required to have a process vent control; whereas, small new sources must use a refrigerated condenser as process vent control device. On new sources, the NESHAP required that small and large sources utilize only a refrigerated condenser control device whereas, a major source utilizes both the refrigerated condenser and carbon absorber as control devices on the process vents.

In 2006, EPA passed the residual risk standard for dry cleaning facilities which strengthened air toxic requirements for dry cleaning facilities and is incorporated in the Dry Cleaning NESHAP (40 CFR 63 Subpart M). It required the elimination of all
transfer machines because it was considered the highest-emitting type of dry cleaning equipment. It also required the elimination of all PCE dry-cleaning machines at residential buildings by December 21, 2020.

All NESHAP (MACT) standards passed under the 1990 CAAA Section 112 program are promulgated by the EPA and codified at 40 CFR Part 63 (usually written as 40 CFR 63) by source category. For example, the NESHAP (MACT) for dry cleaning operations is at 40 CFR 63 Subpart M; the NESHAP (MACT) for petroleum refineries is at 40 CFR 63 Subpart CC. All the pre-1990 NESHAPs are codified at 40 CFR Part 61. For example, the NESHAP for asbestos is at 40 CFR 61 Subpart M; the NESHAP for benzene storage vessels is at 40 CFR 61 Subpart Y.

MACT, Residual Risk and Area Source control standards are now commonly called NESHAPs; because when a 1990 CAAA Section 112, NESHAP is passed, it may regulate both area sources and major sources of HAPs. The 1990 CAA defines a MACT standard as only regulating major sources of HAPs. The portion of the NESHAP that is applicable to “major sources” is the MACT portion. For example, the Dry-Cleaner NESHAP and the Chromium Electroplating NESHAP regulate both area and major sources. Whereas, the Petroleum Refinery NESHAP, Organic Liquids Distribution (OLD) NESHAP, and Hazardous Organic NESHAP (HON) regulate only major sources and, therefore, are entirely MACT standards. All the NESHAP (MACT) standards are available on EPA’s Air Toxic web site at (www.epa.gov/ttn/atw/mactfnlalph.html). Concerning pre-1990 NESHAPs, only recent amendments to the Asbestos and Benzene Waste Operations NESHAPs are available at this site.

2.6.1 MACT Organization

A NESHAP (MACT) is usually organized into several sections. The first sections address applicability, determinations, and definitions. The next sections cover the emission standards and/or work practice standards (i.e., required control equipment, equipment leak detection, and maintenance and inspection plans). The final sections address test methods and compliance procedures, monitoring requirements, and recordkeeping and reporting requirements. For example, the Gasoline Distribution Facilities MACT (40 CFR 63 Subpart R) is organized as follows:

- Section 63.420 - **Applicability**: Applies to Bulk Gasoline Terminals (BGT) or Pipeline Breakout Stations (PBS) that are a major source. The BGT and the PBS are the “affected sources” for this MACT. Also required for each of these affected sources are screen tests equations (factors are based on size and gasoline throughput of facility)
• Section 63.421 – Definitions

• Section 63.422 – Standards: loading racks – (this MACT regulates the loading racks [emission units] from only the PBS affected source)

• Section 63.423 – Standards: storage vessels – (this MACT regulates the storage vessels [emission units] from both affected sources: PBS & BGT) The standards apply only to gasoline storage vessels having a capacity greater than or equal to 75 meters cubed (19,813 gallons) and storing gasoline. The standards are more stringent for new sources (built after February 8, 1994) than for existing sources.

• Section 63.424 – Test methods and procedures

• Section 63.425 – Alternative means of emission limitations

• Section 63.426 – Continuous monitoring

• Section 63.427 – Reporting and recordkeeping

• Section 63.428 – Delegation of authority

2.6.2 MACT General Provisions Rule

In 1994 the EPA passed the General Provisions Rule (40 CFR Part 63, Subpart A). The purpose of the Rule is to eliminate the need to repeat general information and requirements for each emission standard. The General Provisions Rule provides procedures and criteria to help in the implementation of a NESHAP (MACT). If there is a conflict between the “general provisions” and the specific NESHAP (MACT) requirements, the specific requirements apply. The general provisions cover the following:

• Applicability and definitions
• Prohibited activities and circumvention
• Construction and reconstruction
• Compliance with standards and maintenance requirements
• Performance testing requirements
• Monitoring and notification requirements
Introduction to Air Toxics

- Recordkeeping and reporting requirements
- Control device requirements

2.6.3 The MACT Hammer

The MACT hammer provision is codified at CAAA Section 112(j). This provision mandates that if EPA fails to pass a MACT standard within 18 months of the scheduled regulatory deadline, owners and operators of major sources of HAP emissions are required to obtain an equivalent emission limitation by permit (Title V permit). The “equivalent emission limitation” must be at least as stringent as the MACT standard that EPA would have promulgated under the CAA. As a result of the hammer falling, the burden shifts to the states to develop case-by-case MACT standards to reduce adverse effects of air toxics. In 2000, the MACT hammer fell when EPA failed to promulgate 32 MACT standards from about 60 source categories. Before the states had to address the issue, the EPA passed all the required MACTs.

Recently, the U.S. Federal Courts have vacated several MACT standards:

- Brick and Structural Clay Products Manufacturing, 40 CFR Part 63, Subpart JJJJJ, *Sierra Club v. EPA*, 479 F. 3d 875 (D.C. Cir 2007);

- Clay Ceramics Manufacturing, 40 CFR Part 63, Subpart KKKKK, *Sierra Club v. EPA*, 479 F. 3d 875 (D.C. Cir 2007); and


Additionally, the EPA passed the Clean Air Mercury Rule to substitute as a MACT for mercury emissions from coal-fired and oil-fired power plants, source category. This rule has subsequently been vacated, *New Jersey v. EPA*, 517 F. 3d 574 (D.C. Cir 2008). Therefore, at present, there is no MACT regulation of coal-and oil-fired power plants under CAAA Section 112.

The problem is that states have received no guidance from the EPA regarding how EPA interprets the requirements of CAA Section 112(j) to apply when MACT standards are vacated. Since EPA has not addressed the consequences of these court decisions in either rulemaking or guidance, these decisions have resulted in confusion about how and when states must implement the MACT hammer provisions. If EPA has failed to
promulgate a MACT, then states are obligated to provide case-by-case MACT for major sources of hazardous air pollutants under CAAA Section 112(j). At issue is whether CAAA Section 112(j) applies when EPA has passed the MACT, but the MACT is subsequently vacated. If states decide to handle the vacated MACT by using a case-by-case MACT, there is guidance available at the National Association of Clean Air Agencies (NACAA) web page: http://www.4cleanair.org. NACAA has the Mercury Model Rule and Boiler Model Permit Guidance information available.

2.6.4 NESHAP (MACT) Rule Applicability

The first step in rule applicability of a NESHAP (MACT) is to determine whether the facility (plant site) is a major source of HAPs. The second step is to determine which individual source categories at the plant site are subject to an applicable MACT rule. The third step is to identify the plant site’s process equipment (emission points) to the appropriate source category. Each source category is regulated by a specific NESHAP (MACT) rule. Each NESHAP (MACT) rule will define what “affected sources” and emission points it regulates. There should be no overlaps of NESHAP (MACT) rules for an emission point. The purpose of this determination is to discover what applicable emission points are regulated by which NESHAP (MACT) rule.

While “major source” status is based on the entire plant site (source), a NESHAP (MACT) rule applies only to the “affected source” as defined in the rule. The general provisions define the term “affected source” to designate specific source or group of emission units that are subject to a particular Section 112 standard. This term is similar to the “affected facility” under Section 111. The particular “affected source” for each standard will be defined in the standard. For example, in the Gasoline Distribution Facilities MACT (40 CFR 63 Subpart R), there are two defined “affected sources”: BGT and PBS. There are three regulated emission units which have control requirements: storage vessels (for both BGT and PBS), loading racks (for BGT only), and equipment leaks from all gasoline equipment during the loading of gasoline cargo (for both BGT and PBS).

Example of Rule Applicability to a Plant Site: Delta Petroleum Corporation is a petroleum refinery and chemical company that produces petroleum distillates and petrochemicals. In addition, petroleum refining requires a large amount of heat which is generated with numerous process heaters and boilers. The first step is to determine whether the facility (plant site) is a major source of HAPs. Delta Petroleum is a “major” source of HAPs because it has a potential to emit 100 tons per year of total HAPs (which is more than the 25 tons per year limit for total HAPs). The second step is to determine which individual source categories at the plant site are subject to an applicable MACT
rule. The following is a list of source categories and applicable MACTs at Delta Petroleum plant site:

- Petroleum refinery (40 CFR 63 Subpart CC)
- Fluid catalytic cracking (FCC) (40 CFR 63 Subpart UUU)
- Hazardous organic NESHAP (HON) (40 CFR 63 Subpart F, G, H)
- Gasoline distribution (40 CFR 63 Subpart R)
- Organic liquid distribution (OLD) (40 CFR 63 Subpart EEEE)
- Industrial boilers/process heaters (40 CFR 63 Subpart DDDDD) *Vacated*
- Industrial cooling towers (40 CFR 63 Subpart Q)
- Benzene waste operations (40 CFR 61 Subpart FF)

The third step is to identify the plant site’s process equipment (*emission points*) to the appropriate source category. This step would be very long and complicated. It entails listing all the “affected sources” and emission units at the plant site and assigning them to a particular NESHAP (MACT). Exhibit 2-4 is a summary of the NESHAP (MACT) applicability to the “affected sources” at the plant site:
### Exhibit 2-4  Delta Petroleum List of Affected Sources and NESHAP Applicability

<table>
<thead>
<tr>
<th>“Affected Sources” at Plant Site</th>
<th>Applicability of NESHAP (MACT)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Petroleum refinery: miscellaneous process vents; storage vessels; wastewater treatment facilities; cooling towers; equipment leaks; marine vessel loading; and gasoline loading racks</td>
<td>All petroleum refinery “affected sources” emission units are regulated by the Petroleum Refinery MACT.</td>
</tr>
<tr>
<td>HON: process vents, storage vessels, transfer racks, wastewater treatment facilities, and equipment leak components</td>
<td>Only 2 storage vessels are regulated by HON. These vessels store liquid organic HAPs that are listed in the HON. The remaining HON emission units are exempt from HON because they are petroleum refinery process units.</td>
</tr>
<tr>
<td>Bulk gasoline terminal emission units: storage tanks, loading racks and equipment leaks components</td>
<td>Gasoline Distribution MACT does not apply because these emission unit are already regulated under Petroleum Refinery MACT</td>
</tr>
<tr>
<td>OLD: Storage vessels, transfer racks, transport vehicles, containers, and equipment leak components (applies only to storing of organic liquids listed in OLD MACT Table 1 – no gasoline)</td>
<td>OLD MACT is not applicable because the emission units are already regulated under Refinery MACT and remaining units do not handle material with a Table 1 HAP &gt; 5%</td>
</tr>
<tr>
<td>Boilers and process heaters</td>
<td>Since the Industrial Boiler &amp; Process Heater MACT has been vacated – the state should set regulation on a case-by-case basis (hammer?)</td>
</tr>
<tr>
<td>Cooling towers</td>
<td>Cooling Tower NESHAP is not applicable because the towers do not use chromium-based treatment of chemicals</td>
</tr>
<tr>
<td>FCC unit, catalytic reformer units, and the sulfur recovery units</td>
<td>These sulfur removal emission units are regulated by NESHAP for Petroleum Refinery FCC, Catalytic Reformer Units, and the Sulfur Plant Units</td>
</tr>
<tr>
<td>Benzene waste operations (tanks, treatment, etc.)</td>
<td>Regulated by Benzene Waste Operation NESHAP</td>
</tr>
</tbody>
</table>

### 2.6.5 New Directions in Setting MACT standards

Since the tragic chemical release in Bhopal, India, EPA and industry have been looking at more nontraditional ways (besides “end of stack” approaches) to reduce air toxics. As a result, the 1990 CAAA directed EPA to look at a wide variety of emission reduction mechanism to be included in a MACT standard. Examples of these novel concepts in MACTs are as follows:

- Dictates the raw material products a source may use in the production process or how the productions unit should be designed to minimize HAP emissions (i.e., dry cleaner rule banned transfer machines for new sources.)
- Use of emission averaging: (i.e., over control one emission point in order to under-control another emission point covered by the same MACT)

- Use of the predominant MACT concept: (If a facility is covered by multiple categorical MACTS, you may choose predominant MACT across all units.)

- Incorporate pollution prevention: (EPA can prohibit a particular HAP, i.e., prohibited the use of chromium based water treatment in cooling towers.)

2.7 Residual Risk Program

Because the MACT standards are technology based rather than health based, the 1990 CAAA provide for a second phase of regulatory controls aimed at protecting public health with an “ample margin of safety.” After a risk assessment to see if there are any remaining risks from a MACT, EPA is required to pass a residual risk, health-based standard providing “ample margin of safety” protection within eight years after promulgation of the MACT. In March 2007 (72 FR 14,734) EPA described its residual risk analysis. EPA will use its 1989 “risk policy” (used in creating the Benzene NESHAP) for residual risk determinations. For carcinogens, EPA will establish residual risk standards for cancer risks greater than one in 10,000. For cancer risks less than one in a million, EPA will not set a residual risk standard. For cancer risks in-between, EPA will consider costs, technical feasibility, location near a facility, and other factors in deciding whether to set a residual risk standard. For non-cancer risks, EPA will look at target organ hazard information in deciding whether to issue a residual risk standard.

Risk and Technology Review (RTR) is a combined effort to evaluate both risk and technology as required by the CAA after the application of maximum achievable control technology (MACT) standards. The CAAA Section 112 required EPA to complete a Report to Congress that includes a discussion of methods the EPA would use to evaluate the risks remaining after the application of MACT standards. These are known as residual risks. EPA published the “Residual Risk Report to Congress” in March 1999 in which EPA reported to Congress on its residual risk framework and included a discussion of its methods, data, and tools. CAAA Section 112(f)(2) directs EPA to conduct risk assessments on each source category subject to MACT standards, and to determine if additional standards are needed to reduce residual risks. CAAA Section 112(d)(6) requires EPA to review and revise the MACT standards, as necessary, taking into account developments in practices, processes, and control technologies.

In November, 2006, EPA published its RTR Assessment Plan which combines risk and technology review for several industrial sectors into single regulatory actions (more
efficient). RTR is divided into Phases, and further divided into Groups within these Phases. RTR Phase I consists of the first 8 separate residual risk standards, covering 14 source categories, completed through April 2007. Phase I, residual risk standards were completed the old way, one industrial sector at a time. RTR Phase II streamlines the residual risk standard development process by grouping together MACT source categories with compliance dates of 2002 and earlier. RTR Phase II is divided into Groups 1, 2, and 3. The MACT source categories and corresponding EPA contacts for RTR Phase I and Phase II, Groups 1 and 2 are listed on EPA’s web site: www.epa.gov/ttn/atw/rrisk/rtrpg.html. Group 3 information will be added later.

2.8 Area Source Program

An “area source” is any stationary source of HAPs that is not a major source. Area sources may be divided in affected area sources and unaffected area sources. An affected area source would be a facility that is not a major source, but is subject to a relevant emission standard NESHAP (MACT) that regulates major sources in that source category. Examples of affected area sources would be dry cleaners and chromium electroplating. An unaffected area source would be a facility that is not a major source, and is not subject to the NESHAP (MACT) that regulates major sources in that category. Examples of unaffected area sources would be large appliances surface coating operations, petroleum refineries, and organic liquid distribution facilities.

Congress provided in Section 112(d)(5) an alternative technology-based standard for area sources. These standards provide for the use of “generally available control technology” (GACT) or management practices. These standards do not require any type of “floor analysis” or minimum control requirements. The EPA does not have to issue residual risk standards for these source categories.

2.9 Urban Air Toxics

Air toxic emissions occur throughout the United States, but the highest concentration of sources occurs primarily in urban areas. The 1990 CAAA Section 112(c)(3) requires sources representing 90% of the area sources with emissions of the 30 HAPs presenting the greatest risk to public health to be regulated by 2000. This required EPA to:

- List at least 30 HAPs that pose the greatest potential health threat in urban areas. (EPA identified a list of 33 HAPs in their Integrated Air Toxic Strategy – see Exhibit 2-5.)
List area source categories. (EPA identified 70 area source categories.)

Pass control standards for these urban HAPs by November, 2000.

Many area sources will be regulated under this Urban Air Toxics Strategy. By June, 2007 EPA issued control standards for only 28 of 70 area source categories, very much behind the required November, 2000 due date. As a result, in March 2006 the Court directed EPA to issue emission standards for four area sources by December 2006 and continue issuing standards every six months until June 15, 2009 (50 area sources in total). A list of area source standards under the urban air toxic strategy are listed at: www.epa.gov/ttn/atw/area/arearules.html.

CAAA Section 112(k)(3) overlapped Section 112(c)(3) because both required EPA to list at least 30 HAPs that cause the greatest threat to public health from urban area sources. EPA developed the 1999 Integrated Urban Air Toxics Strategy to address the Section 112(k)(3) and Section 112(c)(3) overlapping requirements. This Strategy regulates 33 HAPs in urban settings by looking at significant stationary, mobile, and indoor sources of these HAPs. The goals of the strategy are as follows:

- 75% reduction (from 1993 to 2010) in cancer from HAP stationary sources.
- Reduce HAP public health risk from area sources.
- Address disproportionate impacts of HAPs across urban areas.

The Air Toxics Strategy (previously known as the “Urban Air Toxics Strategy” now sometimes referred to as the “Integrated Air Toxics Strategy”) is required by the CAAA of 1990, provisions at 112(c) and 112(k). The EPA also weaves in CAAA provisions at 202(l) regarding mobile source toxics into an integrated strategy aimed at reducing air toxics emissions. Information on EPA’s Integrated Air Toxic Strategy can be found: www.epa.gov/ttn/atw/urban/urbanpg.html.

Exhibit 2-5 is a list of the 33 priority toxic air pollutants for the Integrated Air Toxic Strategy (urban air toxics HAPs list). In 1999, EPA estimated that these 33 air toxics represent approximately 20 percent of national air toxics emissions, but they are believed to be the most important air toxics contributing to potential health risks in urban areas.
**Exhibit 2-5: Urban Air Toxics HAPs**

<table>
<thead>
<tr>
<th>Acetaldehyde</th>
<th>Dioxin</th>
<th>Methylene chloride</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acrolein</td>
<td>1, 2-dibromoethane</td>
<td>Nickel compounds</td>
</tr>
<tr>
<td>Acrylonitrile</td>
<td>Propylene dichloride</td>
<td>Polychlorinated biphenyls</td>
</tr>
<tr>
<td>Arsenic compounds</td>
<td>1, 3-dichloropropene</td>
<td>Perchloroethylene</td>
</tr>
<tr>
<td>Benzene</td>
<td>Ethylene dichloride</td>
<td>Polycyclic organic matter</td>
</tr>
<tr>
<td>Beryllium compounds</td>
<td>Ethylene oxide</td>
<td>Quinoline</td>
</tr>
<tr>
<td>1,3-Butadiene</td>
<td>Formaldehyde</td>
<td>Tetrachloroethylene</td>
</tr>
<tr>
<td>Cadmium compounds</td>
<td>Hexachlorobenzene</td>
<td>Trichloroethylene</td>
</tr>
<tr>
<td>Carbon tetrachloride</td>
<td>Hydrazine</td>
<td>Vinyl chloride</td>
</tr>
<tr>
<td>Chloroform</td>
<td>Lead compounds</td>
<td></td>
</tr>
<tr>
<td>Chromium compounds</td>
<td>Manganese compounds</td>
<td></td>
</tr>
<tr>
<td>Coke oven emissions</td>
<td>Mercury compounds</td>
<td></td>
</tr>
</tbody>
</table>

According to an EPA 1999 urban air toxics brochure, distribution of the emissions of the 33 urban air toxics among the various source types shows that the least amount of air toxics are from major sources, whereas the most amounts are from area sources and mobile sources of air toxics. The breakdown is as follows:

- 40% Area sources of air toxics
- 40% Mobile sources of air toxics
- 20% Major sources of air toxics

### 2.10 Mobile Source Air Toxic (MSAT) Program

Mobile sources is a term used to describe a wide variety of vehicles, engines, and equipment that generate air pollution and that move, or can be moved, from place to place. Mobile sources pollute the air through combustion and fuel evaporation. These emissions contribute greatly to air pollution nationwide and are the primary cause of air pollution in many urban areas. EPA will use the Integrated Air Toxic Strategy plus the MSAT Rule (described below) to regulate HAPs from mobile sources.

Animal studies have shown that diesel exhausts are more carcinogenic and mutagenic than gasoline exhaust. A 1999 EPA report showed that diesel exhaust is a likely human carcinogen (risks are difficult to quantify). While EPA’s 2001 Diesel Rule regulates only the sulfur content of fuel, it does not regulate HAPs from diesel exhaust.
The many vehicle and fuel changes in the last 25 years have reduced air toxic emissions from highway vehicles. For example, the removal of lead from gasoline has essentially eliminated on-road mobile source emissions of this highly toxic substance in the United States. New cars using reformulated gasoline are capable of emitting 90% less air toxics on a per-mile basis than the uncontrolled models of 1970.

In addition to achieving air toxics emissions reductions as a result of actions aimed at reductions in criteria pollutants, the 1990 CAAA Amendments contain provisions specific to air toxics. These amendments direct EPA to address emissions of air toxics from motor vehicles and their fuels. Specifically, Section 202(l) of the Clean Air Act instructs EPA to:

• *Study* the need for and feasibility of controlling emissions of toxic air pollutants associated with motor vehicles and their fuels. This Section identifies benzene, 1,3-butadiene, and formaldehyde for particular consideration. EPA completed this study in 1993 and updated it in 1999.

• *Set standards* for HAPs from motor vehicles, their fuels, or both. These standards are to be promulgated under Section 202(a) or Section 211(c) of the Act and must address at least benzene and formaldehyde. EPA is to base these standards on available technology, taking into account existing standards, costs, noise, energy, safety factors, and lead time. EPA promulgated the *Mobile Source Air Toxic (MSAT) Rule* in accordance with CAAA Section 202(l) on March 29, 2001 (66 FR 17230). In this rule EPA has:

  • Identified 21 mobile source air toxics (MSATs) (see Exhibit 2-6). Twenty of these are also listed as HAPs in CAA Section 112(b); the remaining one (diesel PM and diesel exhaust organic gases) is a mixture that includes many HAPs, and

  • Established toxic emission performance for gasoline refineries.

In February 2007, under Section 202(l), EPA finalized a rule to reduce mobile source air toxics. The rule requires refineries to lower the benzene content in gasoline to 0.62% (today it’s at 1.0%) and also mandates the reduction of non-methane hydrocarbon exhausts from cars when operating cold. For more information on EPA’s MSAT Program visit EPA’s Air Toxic web site: www.epa.gov/otaq/toxic.html.
Exhibit 2-6: List of Mobile Source Air Toxics

- acetaldehyde
- acrolein
- arsenic compounds
- benzene
- 1,3-butadiene
- chromium compounds
- diesel particulate matter and diesel exhaust organic gases (DPM + DEOG)
- dioxin/furans
- ethylbenzene
- formaldehyde
- n-hexane
- lead compounds
- manganese compounds
- mercury compounds
- methyl tertiary butyl ether (MTBE)
- naphthalene
- nickel compounds
- polycyclic organic matter (POM)
- styrene
- toluene
- xylene

2.11 Mercury Emissions from Coal Fired Power Plants

Mercury compounds are one of the 188 HAPs. They are of concern because they persist in the environment and bio-accumulate in food, and are associated with serious health and environmental effects, including neurological impacts in infants. Coal-fired electric utility plants are the largest air emission sources of mercury in the U.S. (responsible for approximately 40 percent of 1999 emissions). Resultant mercury concentrations in air are usually low and of little direct concern. However, when mercury enters surface waters, biological processes transform it to a highly toxic form that accumulates in fish, which can result in large exposures to fish consumers (including people).

The 1990 CAA Amendments required EPA to study and report on mercury emissions. This included the sources of mercury emissions, the possible controls of these emissions, and the types of environmental and health impacts resulting from mercury exposure. In the ensuing 1997 Mercury Report to Congress, EPA listed coal-fired utilities as the primary source of mercury emissions and that mercury control technology for utility boilers was in the research stage.

The 1990 CAAA Amendments also required EPA to study and report on HAPs from fossil fuel power plants. EPA’s 1998 and 1999 reports stated that mercury from coal-fired power plants is the HAP of greatest concern to public health. The reports also stated that other HAPs needing further study are dioxins, arsenic, and nickel.
As a result of these studies, President G.W. Bush proposed his “Clear Skies Initiative” Bill that called for 70% reduction in mercury emissions from power plants by 2018. The long compliance date was justified by EPA’s reduction of mercury emissions by 45% (see Exhibit 2-7 from EPA’s web site), and the time needed to develop mercury emission control technology. The Bill never passed Congress, but the EPA implemented the program in 2005 by passing the Clean Air Mercury Rule (CAMR) which required 70% reduction in mercury emissions from coal-fired power plants (from years 1999 to 2018). This reduction would be achieved through a national mercury emissions “cap and trade” program for power plants. Many states and environmental organizations have opposed the Rule because of possible mercury “hot spots” that can be created as a result of the trading program. In 2007, 23 states were in the process of pursuing their own mercury power plant MACT. Litigation resulted and in February 2008 the D.C. Circuit Court vacated CAMR because the Rule failed to satisfy EPA’s CAAA Section 112 requirement to regulate mercury as a HAP (i.e., requires “floor analysis” of mercury emissions from power plants for development of a MACT). The Court never addressed the legality of EPA’s HAP “cap and trade” program which was also challenged.

Exhibit 2-7

Mercury Emissions Have Dropped 45% Since 1990

2.12 Prevention of Accidental Releases

The tragic chemical release that killed thousands of people in Bhopal, India in 1984 served as primary impetus for various programs. EPCRA in 1986 was enacted to provide the public with information about the amounts of hazardous chemicals present and discharged from fixed-site facilities. The law establishes the infrastructure of SERCs and LEPCs (see Chapter 1) to develop
emergency response plans for each community and fosters chemical emergency management dialogue between industry and local communities.

In 1990, Congress took additional measures to protect communities from hazardous chemicals by including accident prevention and emergency preparedness measures in the CAAA of 1990. Section 112(r) of the CAAA authorizes EPA to create regulations that prevent and prepare for accidental releases of any hazardous pollutants defined by this Act. Facilities that store or handle extremely hazardous substances over a threshold limit must have submitted to EPA, by June 1999, a risk management plan (RMP) for each hazardous substance used. On January 31, 1994, EPA promulgated a list of these extremely hazardous substances with their threshold limits (40 CFR 68.130). On June 20, 1996, EPA issued the final rules implementing the Accidental Release Program. The core of Section 112(r) regulations are codified at 40 CFR Part 68. CAAA Section 112(r)(7) provides that the RMP must include:

- A hazardous assessment including an estimate of the hazardous effects of a release and the facility’s history of releases.
- A program to prevent accidental releases of the regulated hazardous substances.
- A response program providing for specific actions to be taken if there is an accidental release.

Exhibit 2-8 (from 1999 EPA Chemical Safety document) shows the types of facilities regulated by the Accidental Release Program. Propane retailers and users make up 44% of all risk management plans.
Exhibit 2-8: Types of Facilities Regulated by the Accidental Release Program

Exhibit 2-9 (from same EPA document) shows information on hazard assessment provided in a RMP. This contains information on (1) a worst case toxic release, (2) an alternative toxic release, (3) a worst-case flammable release, or (4) an alternative flammable release. Worst-case and alternative release scenarios identify the area and population that may face a hazard if these events occur. The media and concerned parties can use graphic representations to display areas that may be in danger of this event. For more information on RMP see: www.epa.gov/emergencies/content/rmp/index.html.
2.13 Great Waters Program

Pursuant to Section 112(m) of the CAAA, EPA, in conjunction with the National Oceanic and Atmospheric Administration (NOAA), has issued three reports (1994, 1997, & 2000) to Congress on the deposition of air toxics and the resulting effects on the Great Lakes, Chesapeake Bay, Lake Champlain, and certain other coastal waters, collectively known as the Great Waters. In addition to EPA and NOAA, other international, national, regional, and local organizations also contribute to the body of science relevant to the Great Waters program and are engaged in activities that seek to reduce sources and quantities of pollution to the Great Waters. These activities focus on 15 pollutants of concern, including certain pesticides, metal compounds, chlorinated organic compounds, and nitrogen compounds. These pollutants enter the air in a variety of ways, including direct emission from industries and natural sources, and “re-emission” from soil and water. The Agency selected pollutants of concern due to their persistence, potential to bio-accumulate, and/or potential for adverse impacts to the Great Waters. The three congressional reports suggest that deposition to the great waters is constant or declining. They also suggest that because of long-range atmospheric transport, it’s difficult to determine emission sources. The most recent Great Water Report to Congress is available at: www.epa.gov/airprogm/oar/oaqps/gr8water/.
2.14 State Air Toxic Programs

In the absence of a strong federal air toxics program prior to passage of the CAA Amendments of 1990, many state and some local agencies began to respond to the air toxics problem by developing their own programs. Many states in the U.S. currently have an air toxics control program in place addressing at a minimum, new sources of toxic air pollutants. Some have their own regulations that allow them to actively control air toxics emissions to a level protective of human health; others rely on comprehensive policies or authority provided to implement the federal program. Some state and local programs are risk-based, while others are technology-based.

The state and local programs have progressed in protecting the health of their people and their environment from exposure to air toxics. A successful comprehensive air toxics program will integrate the residual risk and other federal programs with state and local programs and strengthens those existing programs. Program integration will involve interactive sharing of expertise, data, analyses, and methodologies. Additionally, state and local authorities may complement the federal program by addressing local risk issues that may not be effectively addressed nationally.

CAAA Section 112(l) allows state and local programs to be implemented rather than other applicable Section 112 standards. HAP programs can be delegated in any of the following ways:

- States may substitute a state rule that is no less stringent for an EPA industry-specific rule.
- States may substitute an approved state air toxic program that is no less stringent than a federal Section 112 program.
- EPA may delegate to state authority the power to implement a federal Section 112 program.

2.15 Hazardous Air Pollution Monitoring Network

EPA does not maintain an extensive air monitoring network for air toxics as they do for criteria pollutants. Exhibit 2-11 (from EPA’s 2008 Trends Report) shows ambient monitoring locations for air toxics sites in 2007. The majority of these sites are located in or near densely populated areas.
In 2003, working with its state and local partners, EPA launched the implementation of the National Air Toxics Trends Station (NATTS) program, a national monitoring network for toxic air pollutants. The central goal of the NATTS network is to assess trends in high-risk air toxics such as benzene, formaldehyde, 1,3-butadiene, acrolein, and chromium. The initial 23 stations were established between 2003 and 2005; two stations were added in 2007 and two more in 2008 for a total of 27 NATTS sites. In addition, the list of pollutants monitored was expanded to include polycyclic aromatic hydrocarbons (PAHs), of which naphthalene is the most prevalent.

In addition to the NATTS program, about 300 air toxics monitoring sites are currently collecting data to help air pollution control agencies track toxic air pollutant levels in various locations around the country. State and local air quality agencies operate these sites to address specific concerns such as areas of elevated concentrations or “hot spots,” environmental justice concerns, and/or public complaints. Some state and local agencies use EPA’s sampling and analysis support such as the Urban Air Toxics Monitoring Program (UATMP). Air toxics monitoring is generally most prevalent in California, Texas, and the eastern U.S. and reflects a tendency to monitor in densely populated areas. Most sampling is conducted on a 1-in-6-day schedule for a
24-hour period. For the latest information about national air toxics monitoring, visit: www.epa.gov/ttn/amtic.

2.16 Trends in Toxic Air Pollution

EPA compiles an air toxics inventory as part of the National Emissions Inventory (NEI) to estimate and track national emissions trends for the 187 toxic air pollutants regulated under the Clean Air Act. Exhibit 2-11 (from EPA’s 2008 Trends Report) shows the emissions of toxic air pollutants divided among the four types of sources, based on 2002 estimates.

Exhibit 2-11: Contribution by Source Sector to National Air Toxic Emissions, 2002

Nationwide, air toxics emissions decreased approximately 35 percent between the 1990-1993 baseline and 2002 as shown in Exhibit 2-12 (from EPA’s 2008 Trends Report). Major and on-road mobile sources showed the greatest emission reductions (67 and 47 percent, respectively), while emissions from both area and non-road mobile sources increased over this period (26 and 15 percent, respectively).
Although changes in how EPA compiled the national inventory over time may account for some differences, EPA and state regulations, as well as voluntary reductions by industry, have clearly achieved large reductions in total toxic emissions.

2.17 National Air Toxic Assessment (NATA)

Monitoring data are limited for most toxic air pollutants. Because ambient monitoring data is so limited for toxic air pollutants, EPA frequently relies on ambient modeling studies to better define trends in toxic air pollutants. One such modeling study, the National-Scale Air Toxic Assessment (NATA), is a nationwide study of ambient levels, inhalation exposures, and health risks associated with emissions of 177 toxic air pollutants (a subset of the Clean Air Act’s list of 187 toxic air pollutants). EPA developed the NATA in 2002 as a state-of-the-science screening tool. NATA assessments estimate the risk of cancer and other serious health effects from inhaling air toxics. Assessments include estimates of cancer and non-cancer health effects based on chronic exposure from outdoor sources, including assessments of non-cancer health effects for diesel PM. The first NATA assessment data was based on the 1996 National Emission Inventory (NEI) air toxic emissions. EPA’s second NATA (and most recent) is based on 1999 NEI air toxic emissions.

Exhibit 2-13 (from EPA’s 2008 Trends Report) shows the estimated lifetime cancer risk across the continental U.S. by county based on 2002 NATA model estimates. The national average cancer risk level in 2002 was 36 in a million. Many urban areas as well as transportation...
corridors show a risk above the national average. From a national perspective, benzene is the most significant toxic air pollutant for which cancer risk could be estimated, contributing over 30 percent of the average individual cancer risk identified in the 2002 assessment. Though not included in the figure, exposure to diesel exhaust is also widespread. EPA has not adopted specific risk estimates for diesel exhaust but has concluded that diesel exhaust is a likely human carcinogen and ranks with the other substances that the national-scale assessment suggests pose the greatest relative risk to human health. For more information about EPA’s National Air Toxics Assessment, visit http://www.epa.gov/ttn/atw/natamain/.

**Exhibit 2-13: Estimated County-Level Cancer Risk from the 2002 NATA.**

(Darker colors show greater cancer risk associated with toxic air pollutants.)

The purpose of these national scale assessments is to gain a better understanding of the air toxics problem. Specifically, the goals for these assessments are to assist in:

1. Identifying air toxics of greatest potential concern in terms of contribution to population cancer and other health risks.

2. Characterizing the relative contributions of various types of emissions sources to air toxics concentrations and population exposures.
3. Setting priorities for collection of additional air toxics data and research to improve estimates of overall concentrations and public health impacts.

4. Tracking trends in modeled ambient air toxics concentrations over time.

5. Measuring progress toward meeting goals for inhalation risk reduction from ambient air toxics.

2.18 Conclusion

In June, 2006, the Government Accounting Office (GAO) Report to Congress, “Clean Air Act: EPA Should Improve the Management of Its Air Toxics Program,” analyzed EPA’s progress on its Air Toxic Program. The report concluded that EPA was behind schedule in passing its MACT and area source regulations because EPA put a lower priority on its HAP program compared to other air programs. The report suggested that five states and local programs could enhance the federal Section 112 program (Wisconsin, Oregon, California, New Jersey, and Louisville, Kentucky). Several of these states use modeling and monitoring to identify chemicals, areas, and facilities of concern for regulations; whereas, the EPA concentrates on large stationary sources. New Jersey regulates smaller facilities than those required by the federal MACT standards. Wisconsin lists 535 HAPs and subject facilities to regulation if it emits over a threshold amounts (i.e., emissions less than one pound of HAPs per year - depending on the HAP toxicity).

Regardless of criticism of this Report, EPA has made significant progress in its fight in reducing HAP emissions in this country. Exhibit 2-9 (from EPA’s January 2008, Air Status and Trends Report) shows EPA had reduced HAPs in the United States from over 7 million tons per year in 1990 to less than 5 million tons per year in 2002. EPA had accomplished this by focusing on reducing emissions from major sources of MACT (from about 2.4 million tons per year in 1990 - 1993 to about 0.4 million tons per year). Exhibit 2-9 also shows a significant increase of emissions from area sources over the same time period. In 2002, EPA was behind on passing the required area source control regulations. Since 2007, EPA has been making greater strides in promulgating area source standards.
References


Chapter 3: Air Toxics: Chemicals, Sources and Emission Inventories

3.1 Introduction

This chapter addresses air toxic chemicals, the sources of air toxics, and two of EPA prepared emission inventories: National Emission Inventory (NEI) and the Toxic Release Inventory (TRI). The NEI is developed from input from numerous state, local, and tribal (S/L/T) air agencies. NEI data are used for air quality modeling, regional strategy development, regulation setting, air toxic risk assessment, and tracking trends in emissions over time. EPCRA Section 313 requires EPA and states to annually collect data on releases and transfers of listed toxic chemicals from certain industrial facilities and make the data available to the public in the TRI. Also discussed is the process for developing emission inventories. (Note: Selections from Chapters 2, 4, and 7 of EPA’s “Air Toxics Risk Assessment Reference Library, Volume I Technical Resource Manual” have been edited and are used in this Chapter.)

3.2 Air Toxic Chemicals

The term “air toxics” is a generic term that could conceivably encompass literally anything in the air that poses harm to people or the environment. This Manual uses the term “air toxics” in this general sense. Thus, while the focus of most air toxics risk assessments will be on the 188 chemicals and chemical compounds listed as HAPs in the Clean Air Act (CAA) Section 112(b), some assessment teams may wish to have a broader focus. The use of the term “air toxics” in this general sense is meant to provide for this flexibility.

The various lists that are the focus of this Handbook were all derived directly from the Clean Air Act, the Emergency Planning and Community Right to Know Act (Toxic Release Inventory chemicals), or a specific EPA initiative (i.e., the persistent, bio-accumulative, toxic [PBT] list of chemicals. This chapter will provide more detailed information related to the chemicals on each of those lists. A thorough understanding of the different types of chemicals that may be of interest for an assessment, as well as the nuances of the various ways chemicals are written into those lists, will be important for the risk assessment team to comprehend before the assessment begins in earnest.
3.2.1 Chemical Abstract Registry Number

Lists of toxic chemicals commonly provide the chemical identity by both a name and a unique identifying number called a Chemical Abstract Registry Number. However, most chemicals have multiple synonyms (sometime dozens). Fortunately, every unique chemical has only one unique Chemical Abstract Service (CAS) number, and one can always refer to this unique number to identify the compound in question. For example, toluene and methylbenzene are synonyms for the same compound (which is normally referred to as toluene). However, there is only one CAS number for the compound: 108-88-3. No matter where one is in the world or what name is attached to a chemical, there is unanimity of identity through the CAS numbering system.

3.2.2 Chemical Lists for Large Groups of Compounds

Some of the entries on chemical lists are for large groups of compounds and not just one single substance. For example, one of the HAPs is listed in the CAA as “polychlorinated biphenyls” and is most commonly referred to as PCBs. This listing is not for one single substance but, rather, for any one or a mixture of any of the 209 possible chemicals that are themselves PCBs. As another example, the pesticide “2,4-D” is written into the list of HAPs as “2,4-D (salts and esters).” This listing includes any possible salt of 2,4-D and any possible ester of 2,4-D. Another example, the lead compound listing includes any compound known to exist in or be emitted to the environment that contains a lead molecule as part of the compound’s molecular structure (a potentially huge number of possibilities). Another important group of chemicals is called “POM” for polycyclic organic matter. This includes organic compounds with more than one benzene ring, and which have a boiling point greater than or equal to 100° C (i.e., polycyclic aromatic hydrocarbons (PAHs) such as benzo(a)pyrene).

3.2.3 Three Main Groups of Air Toxics

In general, all air toxics can be broadly categorized into three main groups, organic chemicals, inorganic chemicals, and organometallic compounds.

Organic chemical compounds are composed of carbon in combination with other elements such as hydrogen, oxygen, nitrogen, phosphorous, chlorine, and sulfur (not including carbonic acid or ammonium carbonate). Organic compounds can generally be split into two different groups (volatile organic compounds [VOCs] and semi-volatile organic compounds [SVOCs]), based on their propensity to evaporate.
VOCs are organic chemicals that have a high vapor pressure and tend to have low water solubility. Simply put, VOCs have a high propensity to evaporate and remain airborne. Many VOCs are human-made chemicals that are used and produced in the manufacture of paints, pharmaceuticals, and refrigerants, as industrial solvents, such as trichloroethylene, or produced as by-products, such as chloroform produced by chlorination in water treatment. VOCs are often also components of petroleum fuels (i.e., benzene), hydraulic fluids, paint thinners, and dry cleaning agents.

SVOCs are organic chemicals that have a lower vapor pressure than VOCs and, thus, have a lower propensity to evaporate from the liquid or solid form. Once airborne, they also tend to more readily condense out of the gas phase. Examples of SVOCs include most organic pesticides (i.e., chlordane), and certain components of petroleum, such as polycyclic aromatic hydrocarbons.

The *inorganic chemicals group* includes all substances that do not contain carbon and includes a wide array of substances such as:

- Metals (i.e., mercury, lead, and cadmium) and their various salts (e.g., mercury chloride)
- Halogens (i.e., chlorine and bromine)
- Inorganic bases (i.e., ammonia)
- Inorganic acids (i.e., hydrogen chloride, sulfuric acid)

The *organometallic compounds group* is comprised of compounds that are both organic and metallic in nature. The alkyl lead compounds that were added to gasoline to enhance its properties can be used for illustration. “Alkyl” refers to the organic portion of a compound which is attached to the inorganic metal lead. The result is a so-called “organometallic” material, a hybrid of both metallic and organic.

An understanding of the general characteristics of organic chemicals, inorganic chemicals and organometallic compounds will aid in planning a risk assessment and developing an appropriate analysis strategy. For example, most VOCs tend to remain airborne and also do not tend to bio-accumulate to the same extent as some of the non-volatile chemicals. Thus, if an assessment was being planned to evaluate the impact of a source from which only VOCs were released, it becomes less likely that a multi-pathway risk analysis will be necessary (since VOCs do not tend to migrate into soil or water and do not tend to bio-accumulate as strongly in living tissue).
3.2.4 CAA Hazardous Air Pollutants

The HAPs are a group of 187 specific chemicals and chemical compounds and are identified in Section 112(b) of the CAA. HAPs are pollutants known to cause or suspected of causing cancer or other serious human health effects or ecosystem damage. They include individual organic and inorganic compounds and pollutant groups closely related by chemical structure (i.e., arsenic compounds, cyanide compounds, glycol ethers, polycyclic organic matter) or emission sources (i.e., coke oven emissions).

Discussions of “air toxics risk assessment,” generally mean assessments of risks associated with one or more of the HAPs. This is largely because of the CAAA listing of 187 HAPs and its requirement under Section 112(f)(2) (Residual Risk) that EPA assess the risks associated with HAPs that remain after the application of the Maximum Achievable Control Technology (MACT) standards (Section 112(d) of the Act). However, given that this is a relatively short list of chemicals, many communities may want to go beyond this list when assessing risk. It is for this reason, that assessors and other stakeholders must clearly identify why they are conducting an “air toxics” risk assessment, and what they want to include in that assessment.

In its Integrated Urban Air Toxics Strategy, EPA identified a subset of 33 HAPs as those posing the greatest risk in urban areas (see Chapter 2). These 33 HAPs were selected based on a number of factors, including toxicity-weighted emissions, monitoring data, past air quality modeling analysis, and a review of existing risk assessment literature. Also, EPA recently concluded that diesel exhaust is likely to be carcinogenic to humans by inhalation at environmental levels of exposure. Diesel exhaust is addressed in several regulatory actions and diesel particulate matter plus diesel organic gases are listed by EPA as a mobile source air toxic.

3.2.5 CAA Criteria Air Pollutants

The “criteria air pollutants” are six substances regulated pursuant to Title I of the CAA, for which “criteria documents” were developed by the EPA prior to national standard setting decisions. There are already national ambient air quality standards (NAAQS) in place for each of these pollutants as well as established regulatory programs and activities in place to meet those standards. However, they are discussed here because there is some crossover between the realm of HAPs and criteria pollutants. The more important crossover issues are discussed below.
Particulate matter: NAAQS have been established for particles with an aerodynamic diameter less than or equal to 10 microns (called PM10) and particulate matter with an aerodynamic diameter less than or equal to 2.5 microns (called PM2.5). PM can be made up of as little as one or a few, or many hundreds of individual chemicals. In many cases (and depending on the source of the PM), any number of specifically listed HAPs may be a part of the PM makeup. It is for this reason that risk assessors may opt to evaluate the composition of PM and to include the identified chemicals in risk calculations.

Ozone and other criteria pollutants: Certain other criteria pollutants are not specifically listed as HAPs, but HAPs may lead to their formation or they may lead to HAP formation. For example, ozone is produced by the interaction of certain VOCs, oxides of nitrogen (called NO$_x$), and sunlight. As noted previous in Chapter 2, many of the HAPs are VOCs and may play a role in ozone formation. In contrast, sulfur dioxide is a criteria pollutant that can be transformed in the environment into sulfuric acid which, in turn, may become part of a listed HAP (i.e., cadmium sulfate). In general, the criteria pollutants (ozone, nitrogen dioxide, sulfur dioxide, carbon monoxide) are not usually considered in air toxics risk assessments.

3.2.6 Toxic Release Inventory (TRI) Chemicals

Data on TRI chemicals are reported pursuant to Section 313 of the Emergency Planning and Community Right-To-Know Act (EPCRA) of 1986 and Section 6607 of the Pollution Prevention Act of 1990 (PPA). EPCRA and the PPA are intended to inform communities and citizens about chemical hazards in their areas. EPA and states are required to collect data annually on releases (to each environmental medium) and waste management methods (i.e., recycling) of certain toxic chemicals from industrial facilities, and to make the data available to the public in the TRI. EPCRA Section 313(d) permits EPA to list or delist chemicals based on certain criteria. In a 1994 rulemaking, EPA added 286 chemical categories to the TRI chemical list. The TRI chemicals are listed in 40 CFR Section 372.65, and information about the 667 currently-listed TRI chemicals is provided online.

The utility of the TRI for air toxics risk assessment is two-fold. First, it provides a broader perspective of industrial emissions than the HAP list because it includes information on air releases of many hundreds of additional chemicals. Second, accessing TRI information is extremely quick and easy. Using the TRI Explorer search engine (http://www.epa.gov/tri/tridata/index.htm), one may quickly identify the location of emissions sources and the identity and quantity of chemicals released to the air.
3.2.7 Toxic Chemicals That Persist and Which Also May Bio-accumulate

Some toxic compounds have the ability to persist in the environment for long periods of time and may also have the ability to build up in the food chain to levels that are harmful to human health and the environment. For example, releases of metals from a source may deposit out of the air onto the ground where they remain in surface soils for long periods of time. Children playing in the area may ingest this contaminated dirt through hand-to-mouth behaviors. The chemicals in the dirt may also be taken up into plants through the roots and accumulate in foraging animals.

EPA’s challenge in reducing risks from this category of toxic air pollutants stems from this ability to transfer from air, to sediments, water, land, and food; to linger for long periods of time in the environment; and for some substances, their ability to travel long distances. Many of these chemicals (i.e., DDT) have been banned for use in the U.S. As such, there should be no active air emissions of these chemicals (although releases into the air are still possible (i.e., by re-suspension of previously contaminated soil). However some, such as mercury, are still in use today. A number of lists of these persistent and bio-accumulative chemicals have been developed through international and internal EPA efforts (see Exhibit 3-1). A number of the HAPs appear on one or more of these lists.

Exhibit 3-1 “Lists” of Toxic Chemicals that Persist and Also May Bio-accumulate

<table>
<thead>
<tr>
<th>LRTAP chemicals</th>
<th>Great Lakes Priority Substances</th>
</tr>
</thead>
<tbody>
<tr>
<td>• aldrin</td>
<td>In keeping with the obligations of the Great Lakes Water Quality Agreement, Canada and the United States on April 7, 1997, signed the “Great Lakes Bi-national Toxics Strategy: Canada-United States Strategy for the Virtual Elimination of Persistent Toxic Substances in the Great Lakes” (<a href="http://www.epa.gov/lrtap2pg.htm">http://www.epa.gov/lrtap2pg.htm</a>). This Strategy seeks percentage reductions in targeted persistent toxic substances so as to protect and ensure the health and integrity of the Great Lakes ecosystem. The list of “Level 1” substances is identical to EPA’s priority PBT pollutants.</td>
</tr>
<tr>
<td>• chlordane</td>
<td></td>
</tr>
<tr>
<td>• endrin</td>
<td></td>
</tr>
<tr>
<td>• kepone (chlordecone)</td>
<td></td>
</tr>
<tr>
<td>• toxaphene</td>
<td></td>
</tr>
<tr>
<td>• heptachlor</td>
<td></td>
</tr>
<tr>
<td>• mercury</td>
<td></td>
</tr>
<tr>
<td>• dichlorodiphenyltrichloroethane (DDT)</td>
<td></td>
</tr>
<tr>
<td>• lindanexdioxins (polychlorinated dibenzo-p-dioxins)</td>
<td></td>
</tr>
<tr>
<td>• furans (polychlorinated dibenzofurans)</td>
<td></td>
</tr>
<tr>
<td>• cadmium</td>
<td></td>
</tr>
<tr>
<td>• dieldrin</td>
<td></td>
</tr>
<tr>
<td>• hexabromobiphenyl</td>
<td></td>
</tr>
<tr>
<td>• mirex</td>
<td></td>
</tr>
<tr>
<td>• hexachlorobenzene</td>
<td></td>
</tr>
<tr>
<td>• lead</td>
<td></td>
</tr>
<tr>
<td>• polychlorinated biphenyls (PCBs)</td>
<td></td>
</tr>
<tr>
<td>• polycyclic aromatic hydrocarbons</td>
<td></td>
</tr>
<tr>
<td>• hexachlorobenzene</td>
<td></td>
</tr>
</tbody>
</table>
Exhibit 3-1 (continued)

**PBT Chemicals:** EPA has identified the following priority persistent, bio-accumulative, and toxic (PBT) chemicals and has developed the PBT program to address the cross-media issues associated with these chemicals (http://www.epa.gov/opptintr/pbt/).

- aldrin/dieldrin
- benzo(a)pyrene
- chlordane
- DDT
- dichlorodiphenyldichloroethylene (DDE)
- hexachlorobenzene
- alkyl-lead
- mercury and its compounds
- mirex
- octachlorostyrene
- dichlorodiphenyldichloroethane (DDD)
- PCBs
- dioxins and furans
- toxaphene

**Great Waters Pollutants of Concern:** The 1990 CAA Amendments established research and reporting requirements related to the deposition of hazardous air pollutants to the Great Lakes, Lake Champlain, Chesapeake Bay, and certain other “Great Waters.” The Program has identified the following pollutants of concern (http://www.epa.gov/airprogm/oar/oaqps/gr8water/index.html).

- cadmium and cadmium compounds
- DDT/DDE
- hexachlorobenzene
- lindane ((-hexachlorocyclohexane)
- mercury and mercury compounds
- polycyclic organic matter
- tetrachlorodibenzo furan (furans)
- nitrogen compounds
- chlordane
- dieldrin
- "-hexachlorocyclohexane
- lead and lead compounds
- PCBs
- tetrachlorodibenzo-p-dioxin (dioxins)
- toxaphene

**TRI PBT chemicals:** EPA has published two final rules that lowered the Toxics Release Inventory (TRI) reporting thresholds for certain persistent bio-accumulative and toxic (PBT) chemicals and added certain other PBT chemicals to the TRI list of toxic chemicals (http://www.epa.gov/tri/lawsandregs/pbt/pbtrule.htm). The following PBT chemicals are subject to reporting at lowered thresholds:

- dioxin and dioxin-like compounds
- mercury compounds
- aldrin
- chlordane
- hexachlorobenzene
- lead
- methoxychlor
- pendimethalin
- PCBs
- toxaphene
- lead compounds
- polycyclic aromatic compounds
- benzo(g,h,i)perylene
- heptachlor
- isodrin
- mercury
- octachlorostyrene
- pentachlorobenzene
- tetrabromobisphenol A
- trifluralin
Exposure to persistent and bio-accumulative air toxics through a pathway other than inhalation of contaminated air is termed an *indirect exposure pathway* because contact with the chemical occurs in a medium that is not the original medium to which the chemical was released (i.e., air). In contrast, a *direct exposure pathway* is one in which contact occurs with the chemical in the medium to which it was originally released. Then exposure of a person to a chemical (or chemicals) occurs through more than one pathway, a *multi-pathway analysis* may be considered.

### 3.2.8 Other Chemicals

The chemicals included in the various lists of air toxics described above – HAPs, criteria pollutants, TRI chemicals, and toxic chemicals that persist and which also may bio-accumulate – do not represent all of the chemicals potentially emitted to air in a given place. EPA is required to maintain an inventory, known as the “Toxic Substances Control Act (TSCA) Inventory,” of each chemical substance which may be legally manufactured, processed, or imported in the U.S. The TSCA inventory currently contains over 75,000 chemicals (see EPA Compliance web page: “enforcement programs” at [http://www.epa.gov/compliance/civil/programs/tscia/](http://www.epa.gov/compliance/civil/programs/tscia/). At best, we have the capability to assess only a few hundred in detail. It is important to keep in mind that the ability to evaluate air toxics releases is limited by current technology, the lack of toxicity information for all but a relatively small number of chemicals.

### 3.3 Sources of HAPs

People exposed to HAPs at sufficient concentrations and for a sufficient duration of time may have an increased chance of developing cancer or experiencing other serious health effects. These health effects can include damage to the immune system, as well as neurological, reproductive (e.g., reduced fertility), developmental, respiratory, and other health effects. In addition to exposure from breathing air toxics, some HAPs such as mercury compounds can deposit onto soils or surface waters, where they can be taken up by plants and animals. Like humans, ecological systems may experience adverse health problems if exposed to sufficient quantities of HAPs over time.

Hazardous air pollutants, or air toxics, are emitted from thousands of sources across the nation, or they are formed through atmospheric reactions of directly emitted substances. Most air toxics originate from man-made sources, including mobile sources (i.e., cars, trucks, construction equipment) and stationary sources (i.e., factories, refineries, power plants), as well as indoor sources (i.e., some building materials and cleaning solvents). Some air toxics are also released from natural sources such as volcanic eruptions and forest fires. (See Exhibit 3-2.)
Exhibit 3-2: Sources of Air Toxics

Air toxics have been placed into several major groupings that track EPA’s programs and emissions inventories. These major groupings are listed as:

- Point sources
- Nonpoint sources
- On-road mobile sources
- Non-road mobile sources
- Indoor sources
- Natural sources
- Exempt sources

Exhibit 3-3 shows that some differences in terminology exist between the CAA and the National Emission Inventory (NEI). For example, a CAA area source could be considered a point source under NEI if it has location coordinates reported. Also, CAA nonpoint sources are considered area sources under NEI.
### Exhibit 3-3: Terminology Related to Groupings of Source Types

<table>
<thead>
<tr>
<th>Source Type</th>
<th>How Defined in CAA</th>
<th>How Reported in NEI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Point Source - Major</td>
<td>Point Source - Major</td>
<td>Point Source</td>
</tr>
<tr>
<td>Point Source - Area</td>
<td>Point Source - Area</td>
<td>Point Source if location coordinates reported</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Area Source if coordinates are not reported</td>
</tr>
<tr>
<td>Nonpoint Source</td>
<td>Nonpoint Source</td>
<td>Area Source</td>
</tr>
<tr>
<td>Mobile Source – on road</td>
<td>Mobile Source – on road</td>
<td>Modeled</td>
</tr>
<tr>
<td>Mobile Source – non-road</td>
<td>Mobile Source – non-road</td>
<td>Modeled or estimated</td>
</tr>
</tbody>
</table>

#### 3.3.1 Point Sources

Point sources of air toxics are stationary sources (i.e., sources that remain in one place) that can be located on a map. A large facility that houses an industrial process is an example of a point source – the facility and its emission release points (i.e., stacks, vents, fugitive emissions from valves) are stationary, and the emission rates of air toxics can be characterized, either through direct measurements, such as stack monitoring, or indirect methods, such as engineering estimates based on throughput, process information, and other data. The CAA divides point sources into two main categories primarily on the basis of annual emissions rates:

- **Major sources** are defined in Section 112(a)(1) as “any source or group of stationary sources located within a contiguous area and under common control that emits or has the potential to emit, considering controls, in the aggregate, 10 tons per year (tpy) or more of any hazardous air pollutant or 25 tpy or more of any combination of hazardous air pollutants.”

- **Area sources** are defined in Section 112(a)(2) as “any stationary source of hazardous air pollutants that is not a major source. For purposes of Section 112, the term ‘area source’ shall not include motor vehicles or non-road vehicles subject to regulation under Title II.” Examples of area sources include dry cleaners, gas stations, chrome electroplaters, and print shops. Though emissions from individual area sources may be relatively insignificant in human health terms, collectively their emissions can be quite significant, particularly where large numbers of sources are located in heavily populated areas.
Nonpoint Sources

The term nonpoint sources refer to smaller and more diffuse sources within a relatively small geographic area. In the context of EPA’s National Emission Inventory (NEI), nonpoint sources of air toxics are stationary sources for which emissions estimates are provided as an aggregate amount of emissions for all similar sources within a specific local geographic area, such as counties or cities, rather than on a facility- or source-specific basis.

Area sources may be reported as either point or nonpoint sources in the NEI. If a state or local agency reports an area source emission as a point source, then the NEI retains the area source emission as a point source. The NEI does not aggregate point area sources as nonpoint sources, and EPA has taken steps to avoid “double-counting” of emissions in the point and nonpoint source inventories. For example, in the Denver area, the State of Colorado inventories dry cleaners and service stations as point sources. The NEI contains point sources estimates for these two categories in the six county area of Denver and the NEI does not contain nonpoint estimates for these two categories. Dry cleaners and service station emissions are contained in the NEI nonpoint inventory for the other fifty counties on Colorado.

Additional NEI nonpoint sources that contribute to air pollution are agricultural activities, residential trash and yard-waste burning, wood stoves and fireplaces, releases from spills and other accidents, and volatilization and re-suspension of pollutants from contaminated sites. Examples of agricultural activities contributing to air pollution are biomass burning (i.e., for land clearing) and the application of fertilizers and pesticides. The open burning of forests (including wildfires) is also categorized as nonpoint sources. (Note that forest fires are generally considered for the purposes of the NEI to be an anthropogenic source of air toxics because they are assumed to be directly or indirectly, for purposes of the NEI, caused by man.)

On-Road and Non-Road Mobile Sources

Mobile sources pollute the air with fuel combustion products and evaporated fuel. These sources contribute greatly to air pollution nationwide and are the primary cause of air pollution in many urban areas. Section 202(l) of the CAAA gives EPA the authority to regulate air toxics from motor vehicles. Based on 1996 National Toxics Inventory (NTI) data (the NTI is the former name of the air toxics portion of the current NEI), mobile sources contributed 2.3 million tons per year or about half of all air toxics emissions in the U.S. Mobile sources emit hundreds of air pollutants – for example, exhaust and evaporative emissions from mobile sources contain more than 700 compounds. EPA’s
Final Rule, Control of Emissions of Hazardous Air Pollutants from Mobile Sources, commonly known as the “Mobile Source Air Toxics” (MSAT) rule, identified 21 compounds as HAPs emitted by mobile sources (see Chapter 2). All of these compounds except diesel PM and diesel exhaust organic gases (DPM + DEOG) are included on the CAAA Section 112 HAPs list. Although some mobile source air toxics are TRI chemicals, mobile sources are not generally subject to TRI reporting. Other mobile source regulations address emissions of criteria pollutants and their precursors, including carbon monoxide (CO), nitrogen dioxide (NO$_2$), PM, VOCs, and sulfur dioxide (SO$_2$). These criteria air pollutant control programs for mobile sources have and will continue to result in substantial reduction of HAP releases.

Mobile sources include a wide variety of vehicles, engines, and equipment that generate air pollution and that move, or can be moved, from place to place. In the NEI, EPA divides mobile sources into two broad categories. On-road mobile sources include motorized vehicles that are normally operated on public roadways for transportation of passengers or freight. This includes passenger cars, motorcycles, minivans, sport-utility vehicles, light-duty trucks, heavy-duty trucks, and buses. Non-road mobile sources, (sometimes also called “off-road”) include aircraft, commercial marine vessels (CMVs), locomotives, and other non-road engines and equipment. The other non-road engines and equipment included in NEI comprise a diverse list of portable equipment, such as lawn and garden equipment; construction equipment; engines used in recreational activities; and portable industrial, commercial, and agricultural engines.

### 3.3.4 Sources Not Included in the NEI or TRI

In addition to the four primary categories used in compiling the NEI, five other sources of air toxics which are not captured by either the NEI or TRI are described below: Indoor sources, natural sources, secondary formation of air toxics, exempt sources, and international transport.

#### 3.3.4.1 Indoor Sources

The importance of indoor air exposures to the total risk from air toxics is a relatively new finding. The contribution of indoor sources was not really recognized until the early 1980s when EPA performed the Total Exposure Assessment Methodology (TEAM) studies, which showed that the indoor concentrations of some air toxics can be significantly higher than outdoor concentrations. Since that time, numerous studies have confirmed that finding. In addition, the fact that Americans spend about 90 percent of their times indoors makes these exposures even more important.

There are many potential sources of indoor air toxics in any home or building. These sources include combustion sources such as oil, gas, kerosene, coal, wood, and tobacco...
products; building materials and furnishings as diverse as deteriorated, asbestos-containing insulation, and cabinetry or furniture made of certain pressed wood products; products for household cleaning and maintenance (i.e., pesticides), personal care, or hobbies; and outdoor sources such as radon and other air pollution that penetrate into the indoor space. The major indoor pollutants of concern are:

- Radon
- Environmental Tobacco Smoke
- Biological pollutants (i.e., pollen, mold animal dander, and fungi)
- Carbon Monoxide
- Nitrogen Dioxide
- Volatile Organic Compounds
- Respirable particles
- Formaldehyde
- Pesticides
- Asbestos
- Lead

Although EPA does not regulate indoor air pollution levels, it does take a proactive approach. The Agency provides a broad range of information about indoor air-related risks, as well as the steps to reduce them, through the use of public awareness campaigns, guidance document dissemination, training course delivery, the operation of several linked hotlines and clearinghouses, and other outreach efforts. Useful resources on indoor air quality from the Agency are also available online. EPA’s activities to reduce exposures to indoor air toxics are many and include publishing guidelines about radon testing and result interpretation; persuading parents and caregivers of young children not to smoke indoors; and providing information to homeowners, school administrators, and office managers on the proper use of products and materials indoors, including appropriate maintenance and ventilation.

To prioritize activities for other chemicals typically found in indoor air, EPA’s Office of Radiation and Indoor Air (ORIA) is sponsoring a screening-level, risk-based analysis, which is currently in draft form and being revised. Some of the chemicals that may be of concern in indoor air, based on the draft ranking, are provided in Exhibit 3-4. However, it should be noted that the final results of this analysis may be significantly different. It should also be noted that, because monitoring data were only available for 112 chemicals and only 59 chemicals could be ranked, many chemicals found indoors might rank higher, given more complete information.
3.3.4.2 **Natural Sources:** Natural processes are significant sources of some air pollutants, including VOCs, NO\(_X\), O\(_3\), PM and other pollutants (Exhibit 3-5). Natural pollutants contribute significantly to air pollution. For example, biogenic emission estimates for the United States were 28.2 million tons of VOC and 1.53 million tons of NO\(_X\) in 1997.

### Exhibit: 3-5: Categories of Natural Sources

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples of Emissions</th>
<th>Sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>Geological</td>
<td>• Sulfuric, hydrofluoric &amp; hydrochloric acids</td>
<td>• Volcanic gas</td>
</tr>
<tr>
<td></td>
<td>• Radon</td>
<td>• Radioactive decay of rocks</td>
</tr>
<tr>
<td></td>
<td>• Nitrogen Dioxide</td>
<td>• Soils, lightning</td>
</tr>
<tr>
<td>Biogenic</td>
<td>• Ammonia</td>
<td>• Animal wastes</td>
</tr>
<tr>
<td></td>
<td>• Methane</td>
<td>• Animal wastes, plant decay</td>
</tr>
<tr>
<td></td>
<td>• VOCs</td>
<td>• Vegetation</td>
</tr>
<tr>
<td>Marine</td>
<td>• Dimethyl sulfide, ammonia, chlorides, sulfates, alkyl halides, nitrous oxides</td>
<td>• Sea spray released by breaking waves</td>
</tr>
</tbody>
</table>

3.3.4.3 **Formation of Secondary Pollutants:** Some air pollutants, in addition to being directly emitted to the atmosphere by identifiable sources, are generated in the atmosphere by the chemical transformation of precursor compounds (a process called *secondary formation*). For example, under some meteorological conditions up to 90 percent of ambient formaldehyde originates from secondary formation from a variety of precursor compounds in the presence of light (i.e., via a *photochemical reaction*). Some of the precursor compounds include isoprene (an organic compound released from trees), and isobutene.

The NEI and other emission inventories generally do not include estimates of pollutants formed through secondary formation – only the initially emitted species are included. Because the formation of secondary pollutants depends on the meteorological conditions
and the presence or absence of other compounds and/or light, a model that incorporates chemical transformation algorithms is required to estimate how much secondary product is formed from precursor compounds once they enter the atmosphere.

3.3.4.4 Small Sources, Exempt Sources and International Transport of Air Toxics:
Many air toxics sources, usually relatively small ones, may not be covered or are exempt from various emissions control, reporting, and other requirements, and in some cases the number or stringency of requirements is tiered according to source size or other criteria. For example, air pollution regulations for municipal waste combustors (MWCs) promulgated pursuant to Section 129 of the CAA include separate rules for large MWCs (i.e., with capacities greater than 250 tons per day) and small MWCs (i.e., with capacities between 35 and 250 tons per day). However, there are no rules for MWCs with capacities less than 35 tons per day.

Other miscellaneous sources of air pollution (i.e., agricultural and residential burning) are controlled primarily by other state and local requirements. However, EPA conducts research, provides information, and pursues other non-regulatory means of addressing some of these pollution sources.

Certain air toxics may be transported over long distances, sometimes over international borders. International sources may be an important contributor to local pollutant levels in some areas. For example, studies have shown that long-range transport from many regions of the globe is a significant source of “persistent organic pollutants” chemicals to the Great Lakes, and that mitigation efforts are going to be needed both in the U.S. and globally to address potential sources.

In summary, emissions from small sources, exempt sources, and international transport of air toxics are not included in a NEI or TRI.

3.4 Emission Inventories
An emission inventory is a comprehensive listing, by source, of the air pollutant emissions within a specific geographic area in a specific time period. The primary emission inventory for HAPs and criteria pollutants is EPA’s National Emission Inventory (NEI). EPA’s Toxic Release Inventory (TRI) is a second inventory that has some utility for planning and scoping an air toxics risk assessment, but is of limited use for risk assessment because of the nature of the way the data are reported. In addition to the NEI and the TRI, state, local and tribes (S/L/T) air agency permit files and, in some instances, S/L/T and regional inventories that have been developed, but not submitted to the NEI, can also provide information on the location, identity, magnitude, and source
characteristics of air toxics releases. This section will describe the NEI, the TRI, S/L/T permit files, and regional inventories. The next section will discuss the process of developing an emissions inventory.

### 3.4.1 National Emission Inventory (NEI)

EPA’s Office of Air and Radiation compiles and maintains the National Emissions Inventory (NEI) that includes quantitative data on anthropogenic emissions of criteria pollutants and HAPs and characteristics of the sources of these air toxics. It includes point, non-point, and mobile sources for all 50 states, Washington, D.C., and U.S. territories.

Previously, emissions of criteria pollutants and HAPs were tracked separately by EPA in databases that preceded the NEI. Criteria pollutant emissions data for 1985 through 1998 are available in the National Emission Trends (NET) database. Hazardous air pollutant (HAP) emissions data are available for 1993 and 1996 in the National Toxics Inventory (NTI) database. For 1999 (the most recent year for which data are available), criteria and HAP emissions data have been prepared separately but in a more integrated fashion. The final version of both the criteria and HAP inventories (for 1999) are available at [http://www.epa.gov/ttn/chief/net/1999inventory.html](http://www.epa.gov/ttn/chief/net/1999inventory.html). EPA plans to update the NEI every three years.

The NEI inventories are developed by EPA’s Emission Factors and Inventories Group with input from S/L/T agencies, industry, and a number of EPA offices. In some cases, if a S/L/T agency does not submit data, EPA may use data from an earlier year and “grow” the emissions (i.e., for criteria stationary sources) or use only data available from other sources (i.e., HAP collected by EPA as part of the development of emission standards, or data submitted by sources under the Toxics Release Inventory program).

An important fact to keep in mind about the NEI is that it includes data on HAPs from both small and large stationary sources and both on- and off-road mobile sources. Equally important, it is much more likely to include the data necessary for modeling. Information such as stack height, emission rate, and temperature are critical to developing reasonably accurate estimates of human exposure in the areas surrounding a source. It is for this reason that the NEI can be of more use than other databases.

#### 3.4.1.1 NEI for HAPs – Point Sources:

For the NEI for HAP emissions from point sources, S/L/T agencies are asked to supply HAP emission inventory data to EPA. If they do not provide HAP emission inventory data to EPA, then EPA prepares default emission inventory data (this has been done for the 1993, 1996, and 1999 inventory
years). The target area for the NEI includes every state and territory in the United States and every county within a state. The pollutants inventoried included all 188 HAPs identified in Section 112(b) of the CAAA. Some S/L/T agencies collect information on more than just these HAPs, but only the 188 are included in the HAP NEI.

### 3.4.1.2 NEI for HAPs and Criteria Pollutants – Nonpoint Sources:

Much of the nonpoint source data in NEI for HAPs was initially compiled as a national-level inventory. National-level emission estimates are spatially allocated to the county-level using a number of allocation factors, such as population and employment within certain industries. For example, aggregate amounts of dry cleaner emissions for a county might be estimated from the number of people living within a county. For HAPs, EPA uses MACT data and S/L/T data, where available.

### 3.4.1.3 On-road Mobile Sources:

In the final Version (V 3.0) of the 1999 NEI, EPA used the most recent version of the MOBILE6 model to calculate emission factors for criteria pollutants and 36 HAPs. On-road emissions inventories for CO, NO\textsubscript{x}, VOC, PM\textsubscript{10}, PM\textsubscript{2.5}, SO\textsubscript{2}, NH\textsubscript{3}, and the 36 HAPs are calculated by multiplying an appropriate emission factor in grams emitted per mile by the corresponding vehicle miles traveled (VMT) in millions of miles, and then converting the product to units of tons of emissions.

### 3.4.1.4 Non-road Mobile Sources:

To develop this component of the NEI, data were compiled on criteria and HAP emissions data for aircraft, commercial marine vessels, and locomotives. HAP emissions for other non-road engines operating in the United States were estimated using the latest non-road model. S/L/T data are used when provided.

### 3.4.2 Toxic Release Inventory (TRI)

The Toxics Release Inventory (TRI) is a publicly available EPA database that contains information about releases and other waste management activities reported annually by covered industry groups as well as federal facilities for over 650 toxic chemicals (see [http://www.epa.gov/tri/](http://www.epa.gov/tri/)). This inventory was established under the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA) and expanded by the Pollution Prevention Act of 1990. (For more information on TRI, see Chapter 1 of this Manual.) EPCRA’s primary purpose is to inform communities and citizens of chemical hazard in their community.

The list of TRI pollutants is organized differently than the list of HAPs in CAAA Section 112, causing some complications in interpreting emission data. It is difficult to correctly relate some of the SIC codes (under which TRI emissions are grouped) to specific air emission processes. Because quantities are only reported if a statutory threshold is met, a
facility may report emissions for one year but not the next, even though the facility is still in operation. Similarly, individual pollutants may not be reported consistently from year to year due to the thresholds that apply to individual pollutants (i.e., a facility may report releases of 10 pollutants one year and releases of only five pollutants the next year because the others dropped below the reporting threshold).

Furthermore, for some facilities, it is possible that, for a variety of releases, the data included for a facility’s emissions in the TRI do not match the same data reported to the NEI, indicating a potential problem with either or both data sets. The risk assessor should apply care and discretion when using TRI information to estimate exposures and risk from individual facilities. Ultimately, the TRI provides information about the location, identity, and amount of air toxics emissions in a community. However, due to the nature of the way the data are developed and reported, TRI data should generally be considered a source of limited information about a facility and should not be used in risk assessments involving modeling (as noted above, S/L/T and NEI data are more likely to be useful for modeling). For robust analysis, it should generally be considered a starting point, not an end.

### 3.4.3 State, Local, and Tribal Permit Files

Most stationary sources (especially large sources) are subject to one or more emissions limitation standards to control criteria emissions and/or HAP emissions. These sources are usually subject to a Title V operating permit that will include all of the operating and emissions limit requirements subject to that facility. In addition, they may be subject to additional S/L/T regulations. Operating permits require routine reporting to confirm that the operating conditions and emission limits are being met. Frequently, these reports are based on some kind of monitoring information. In most cases, actual release rates are reported. Therefore, permit compliance reports represent an excellent source of information that will provide the actual release rate directly through continuous emissions monitoring, or they will provide sufficient information to estimate the release rates with a fairly high level of reliability.

Unfortunately, using the permit compliance system is not always an attractive source for data on release rates that are suitable for risk assessment activities. EPA does not maintain a central database of Title V permit or compliance information. Therefore, gathering data from the permit program can be a time-consuming task if many sources are needed for the risk analysis.

In general, EPA has made an effort to include permit data in the NEI database (via data submissions from S/L/T offices) where appropriate and has taken steps to review the
data. In many cases, it may be more reasonable to consult the NEI prior to attempting to gather release rates directly from permit files.

### 3.4.4 Regional Inventories

Several regional organizations provide emission data specific to their geographic area of concern. For example, the Great Lakes Commission (a partnership among EPA, the eight Great Lakes states, and the province of Ontario, Canada), with funding from EPA and the Great Lakes Protection Fund, have developed the Regional Air Pollutant Inventory Development System (RAPIDS). This ongoing initiative seeks to provide researchers and policy makers with detailed, basin-wide data on the source and emission levels of toxic contaminants. Originally focused on 49 toxic air pollutants, the inventory database has been expanded to include 82 toxic air pollutants which have been identified as significant contributors to the contamination of the Great Lakes. RAPIDS uses the FIRE database to estimate emissions for both point and nonpoint sources. The software may be downloaded from [http://www.glc.org/air/rapids/](http://www.glc.org/air/rapids/).

Additionally, EPA provides funding to five regional planning organizations throughout the U.S. to address regional haze and visibility impairment issues. These organizations exist to evaluate technical information to better understand how their states and tribes impact national park and wilderness areas (Class I areas under the CAA) across the country and to then pursue the development of regional strategies to reduce emissions of PM and other pollutants contributing to regional haze. To this end, each regional planning organization assesses its member states’ emission inventories, and some provide funding through EPA for the development of regional emission inventories. Information regarding regional emission inventorying activities may be found at the organizations’ respective websites as listed below:

- Midwest Regional Planning Organization (Midwest RPO) – [http://64.27.125.175/](http://64.27.125.175/)

### 3.5 Developing an Emission Inventory

The process of developing an emissions inventory is described in detail in Chapter 7 of EPA’s “Air Toxics Risk Assessment Reference Library, Volume I Technical Resource
There are eight steps for developing an emissions inventory: (1) planning, (2) gathering information, (3) estimating emissions, (4) compiling data into a database, (5) data augmentation, (6) quality control/quality assurance, (7) documentation, and (8) access to data.

3.5.1 Planning

Planning is the first stage in developing an emission inventory. The inventory Preparation Plan (IPP) is developed during the planning stage and is the overarching guidance document for the entire emission inventory development process. The following are a list of the steps used in developing an IPP.

- Identify the end-uses of the inventory
- Determine Data Quality Objectives
- Define the inventory to be created
- Select an inventory data management and reporting system
- Summarize data reporting and documentation
- Establish QA/QC procedures
- Determine staffing and resource requirements
- Develop a schedule
- Identify partners and develop a communication plan

3.5.2 Gathering Information

The next step in the development of an emission inventory is to gather the relevant information from existing sources. The information gathered should, at a minimum, include applicable pollutants, their sources, and emissions data (i.e., chemicals, emissions rates over time). If air quality modeling will be a part of the exposure assessment, the emissions inventory will need to include all of the source term data required by the model(s) to be used (e.g., latitude and longitude coordinates for each source, building size and shape for assessing downwash, chemical speciation).

A comprehensive information search may include guidance documents, existing emissions data, preliminary screening studies, emission factors, models, source characterization documents, and activity data references. A good starting point in this search is EPA’s “Handbook for Air Toxics Emission Inventory Development,” EPA-454/B-98-002 (available at EPA’s Air Toxic web page).
3.5.3 Estimating Emissions

After gathering data from existing information sources, the analyst estimates the emissions to be reported in the inventory. In making these emission estimates, the analyst will consider the following five methods:

- Direct Measurement
- Emission Estimate Models
- Emission Factors
- Mass Balance
- Engineering Judgment

Direct Measurement: Direct measurement of source-specific emission rates is relatively infrequent except for certain permitted facilities with specific monitoring requirements written into their permits. For example, source monitoring is typically available for large point source releases at facilities covered under the Title IV emissions tracking system associated with the acid rain control program. Various state and local permitting programs may also require intermittent or continuous monitoring depending on the nature of the process.

In some instances, source testing is required as part of the process of obtaining a permit. For example, a hazardous waste incinerator must do stack testing during trial burns to ensure that the incineration units and air pollution control equipment meet the limits established in the permit before full operation is allowed to begin. Subsequent to full operation, the facility will usually be required to perform continuous monitoring of stack emissions to ensure continued compliance.

Emission Estimation Models: Specific emission measurements are generally the best and most accurate method to quantify emissions; however, source data are not always available and/or practical to obtain. As an alternative, emission estimation software and accompanying models may be used to generate emissions data. Emission estimation models are used when a large number of complex calculations must be undertaken in order to estimate a given emission or when a combination of parameters has been identified that affect emissions but individually do not provide a direct correlation. EPA provides a variety of approved models that can be used to determine point, nonpoint, and
mobile source emissions based on a variety of known input parameters. Some of these emission estimation models are:

- CHEMDAT8
- WATER9
- LANDGEM (Landfill Gas Emission Model)
- TANKS
- MOBILE6 Vehicle Emission Modeling Software
- NONROAD Model

**Emission Factors:** Emission factors are constants that assessors can use to relate release rates to the amount of specific activities that occur at a source. An emission factor is typically represented as a mass of chemical released per unit of activity. For example, releases from a coal-burning combustion device are represented as pounds of pollutant emitted per BTU coal burned. Depending on the emission source, there may be a lot of emissions testing data, just one or two measurements (the usual case), or none.

Frequently, emission factors contain an associated confidence level by species, which assists in determining the appropriate emission factor. Thus, the use of the emission factor for any specific source may over- or under-predict actual release rates. In some cases, accurate measurements of the activity rates are not available and estimates of activity rates can also contribute uncertainty to the release rate estimate for any particular source type. An example is for individual motor vehicles; this source model estimates an average emission factor for a fleet of vehicles in a particular location. Modeling approaches for traffic activity estimate the total amount of miles driven by vehicle class. Finally, multiplying the emissions factor by the number of vehicle-miles driven produces the total emissions. Thus, any individual motor vehicle may have a release rate significantly far removed from the average, but when averaged across the fleet, the release rate provides a more reliable estimate.

EPA suggests emission factors for criteria pollutants and HAPs in its national database, Factor Information Retrieval System (FIRE), which includes emission factors from EPA documents (such as *Compilation of Air Pollutant Emission Factors* (AP-42) and the *Locating and Estimating Air Emission* series) factors derived from state-reported test data, and factors taken from literature searches. FIRE is available for download at [http://www.epa.gov/ttn/chief/software/fire/](http://www.epa.gov/ttn/chief/software/fire/).

**Mass Balance:** Assessors can use the mass balance approach in complex processes in which a known amount of air toxics material is introduced to a process, and at the end of
the process, a known amount of air toxics material is still retained in the final product. The difference between the two represents the production release. Engineering estimates can then suggest into what medium the process released the air toxic (i.e., to air or water, or as solid waste).

As an example, consider the use of a VOC as a carrier medium for a solid (i.e., paint particles). In this surface-coating situation, the organic solvent that suspends the solids makes application of the coating possible. Once the mixture is exposed to the air, the solvent evaporates, leaving the solid coating film on the object. Mass balance techniques in this type of application may assume that 100 percent of the solvent is released to the air through evaporation. Other mass balance estimates may assume that some stable amount of the solvent is retained in the product that is shipped to customers. Mass balance estimates also may need to consider how much of the solvent is recycled at various stages in its life-cycle.

*Engineering Judgment:* With engineering judgment, users can estimate emission releases through engineering and operational observations about a process. For example, if a certain process must be operated at a set temperature and pressure to achieve the ideal result, engineers who understand the history of the process can often estimate how the release rate actually varies under changing operational conditions. Engineering judgment is a less desirable approach for estimating releases than actual measurements; however, it is often used because of a lack of any better information or options (i.e., it may not be possible to measure all fugitive leaks at a large facility with thousands of joints and valves).

### 3.5.4 Compiling Data Into a Database

After estimating the applicable emissions from each source, the analyst compiles the data into the inventory database, based on the data management system delineated in the Inventory Preparation Plan. Three elements of data compilation are of note for a risk assessment: selection of production rates, unusual conditions, and how emissions are quantified for risk assessment purposes.

### 3.5.5 Data Augmentation

If previous efforts at estimating emissions fail to obtain data to assemble an emissions inventory of sufficient quality or to provide the necessary inputs for an emissions model, the next step would be data augmentation. The analyst now identifies any missing information, most notably emission data, vent parameters, and location coordinates.
3.5.6 Quality Insurance/Quality Control

Quality assurance and quality control (QA/QC) procedures are vital to the validity of the emissions inventory and ensure that the modeling input parameters derived from the inventory are of specified quality. The quality assurance plan (QAP) for the emissions inventory is usually a part of the quality assurance project plan (QAPP) for the overall risk assessment that is developed during problem formulation. The QAP documents the procedures of the QA/QC elements of the emissions inventory. Quality control measures include:

- Technical reviews
- Use of approved standardized procedures for emissions calculations
- Data verification procedures
- Completeness checks
- Consistency checks
- Accuracy checks
- Reasonableness tests

3.5.7 Documentation

Documentation is the next step in developing an emission inventory. The key documents to be compiled into a final written report include:

- Inventory Preparation Plan (IPP)
- Quality Assurance Plan (QAP)
- Methods
- Assumptions
- Raw data (database)
- Calculations

3.5.8 Access to Data

The risk manager generally ensures appropriate access to the data compiled in the emission inventory. A key part of the planning and scoping process for the risk assessment is determining who needs access to the emissions data and how they will access the data. If it is necessary to report the results of the emission inventory to the EPA as part of S/L/T agency’s responsibilities under the Consolidated Emission Reporting Rule, data preparation and submission procedures prepared by EPA for HAP data should be followed.
References


Chapter 4: Introduction to Risk Assessment

4.1 Risk

“Risk” is the probability that some harmful event will occur. EPA considers “risk” to be the chance of harmful effects to human health or to ecological systems resulting from exposure to an environmental stressor. A stressor is any physical, chemical, or biological entity that can induce an adverse response. Stressors may adversely affect specific natural resources or entire ecosystems, including plants and animals, as well as the environment with which they interact.

The risks associated with human endeavors are usually categorized as voluntary or involuntary risks. Voluntary risks are taken by people on their own free will. Examples include risks associated with recreational sports, flying a private aircraft, or driving an automobile. Involuntary risks are imposed because of circumstances beyond our control. Examples would include using elevators in tall buildings, traveling in commercial aircraft, or being exposed to a hazardous air pollutant. Risks associated with the environment are considered involuntary and the public expects risks to be no larger than those associated with naturally-occurring events (i.e., being struck by lightning).

How is risk expressed? Because it is a probability, risk is expressed as a fraction, without units. It could be expressed as 0 (there is no risk of the event occurring) to 1.0 (absolute certainty that the risk event will occur). Values between 0 and 1.0 represent the probability that a risk will occur. For example, a lifetime cancer risk of 1 in 10,000 means that one of every 10,000 people exposed to the particular carcinogen will develop cancer over a lifetime. This risk would be expressed as 0.0001 or just $10^{-4}$.

The following simple mathematical formula describes the basis for human health risk assessment. Specifically, the likelihood that injury or disease may occur from exposure to air toxics can be described as a function of two separate, but related, things – an estimate of exposure to a chemical and an estimate of the toxic properties of the chemical (see equation below). This general equation is important to understand and keep in mind since the exact equations used to develop risk estimates are derived from it.

Potential for Injury or Disease (i.e., the “Risk”) = $f$ (metric of exposure, metric of toxicity)
Introduction to Air Toxics

4.2 Risk Assessment: Conceptual Model of Air Toxic Releases

EPA uses risk assessment to characterize the nature and magnitude of health risks to humans and ecological receptors (i.e., birds, fish, wildlife) from chemical contaminants and/or other stressors that may be present in the environment. The overall goal of risk assessment is to establish chemical concentrations that can be present in the environment without putting humans or ecological species at risk. There are two types of risk assessment related to chemical exposure: human health and ecological. Although the overall goals are the same, there are some differences in how the process is conducted. This manual will concentrate on human health risk assessment.

The purpose of a human health air toxics risk assessment is to attempt to understand public health risks potentially associated with exposures to particular pollutants emitted into the air from sources of interest. Exhibit 4-1 presents a simple illustration of the overall real-world process that is investigated through the use of risk assessment. This picture is a conceptual model of how air toxic releases may pose risks to people. It is a conceptual model because it provides a picture (or “model”) of our “concept” of what may happen in the real world when toxic chemicals are released to the air.

Exhibit 4-1 tracks the release of air toxics from the source to the human endpoint of interest. This exhibit also illustrates how air toxics risk assessments usually focuses, at a minimum, on the inhalation of contaminated air. However, for a small subset of air toxics, the risk assessment also may need to address ingestion or dermal contact with soils, water, or food that have become contaminated with chemicals that have deposited out of the air.
Starting at the upper left hand side of this diagram, air toxics are released from one or more sources (i.e., factories, cars/trucks, small businesses, forest fires) to the air and begin to disperse by the wind away from the point of release. Once released, the chemical may remain airborne, convert into a different substance, and/or deposit out of the air onto soils, water, or plants. People may be exposed to air toxics by breathing contaminated air (inhalation) or through ingestion of chemicals that can accumulate in soils, sediments, and foods (the latter process is called bioaccumulation). People also can be exposed to deposited chemicals via skin (dermal) contact; however, this tends to be a less important risk factor than ingestion or inhalation. Inhalation, ingestion, and dermal absorption are called the routes of exposure.

This description of what happens to an air toxic once it is released into the air is called fate and transport analysis. “Transport” evaluates how an air toxic physically moves (i.e., is transported) through the environment. “Fate” describes what ultimately happens to the chemical after it is released to the air (i.e., what is the “fate” of the chemical in the environment). The results of a fate and transport analysis are an estimate of the
concentration of the air toxic in the air, soil, water, and/or food at the point of contact by a person. The exposure assessment is the process of evaluating how human contact with the contaminated media occurs.

Once an exposure occurs, the air toxics can enter the body and exert an effect at the point of entry (the “portal of entry”) or move via the bloodstream to other target organs or tissues. The action of a pollutant on a target organ can result in a variety of harmful effects, including cancer, respiratory effects, birth defects, and reproductive and neurological disorders. An overall risk assessment process evaluates what people are exposed to, how the exposure occurs, and, when combined with information about the toxic properties of the chemicals in question, estimates the likelihood that the exposure will result in injury or disease.

4.3 Four-Step, Risk Assessment Process

In addition to a conceptual model, there is a need for a defined process to quantify relationships among the conceptual model components in order to generate numeric risk estimates. Risk assessment is that process. The 1983 National Resource Commission (NRC) report, “Risk Assessment in the Federal Government: Managing the Process,” defined risk assessment as a process in which information is analyzed to determine if an environmental hazard might cause harm to exposed persons and ecosystems. The NRC report also described, for the first time, the following four-step paradigm for risk assessment that continues to serve as EPA’s model for human health risk assessments:

Hazard Identification: The first step in a risk assessment is to determine whether the pollutants of concern can be causally linked to the health effects in question (cancer and/or non-cancer). Factors such as the route of exposure, the type and quality of the effects, the biological plausibility of findings, the consistency of findings across studies, and the potential for bioaccumulation all contribute to the strength of the hazard identification statement.

Dose-response Assessment: This step is the quantitative characterization of the relationship between the concentration, exposure, or dose of a pollutant and the resultant health effects. When adequate data exist, the typical end product of the dose-response assessment for non-cancer effects is the identification of a sub-threshold dose or exposure level that humans could experience daily for a lifetime without appreciable probability of ill effect. For cancer, the typical goal of this step is estimation of a full dose-response curve for low exposures.
**Exposure Assessment:** EPA’s “Guidelines for Exposure Assessment,” published in 1992, provide the framework for this step. An exposure assessment for air toxics has four major components: (1) emissions characterization, (2) environmental fate and transport analysis, (3) characterization of the study population, and (4) exposure characterization for both inhalation and non-inhalation pathways.

**Risk Characterization:** In this step all the information from the previous steps is integrated to describe the outcome of the analysis and where the uncertainty and variability in the results are described. EPA’s 1995 “Guidance for Risk Characterization” is the foundation for this step of the process.

### 4.4 Framework for Risk Assessment

Using knowledge gained from past risk assessments, information from other regulatory agencies, and guidance from Reports such as the NRC and CRARM reports, the Agency developed a general framework for risk assessment, and Exhibit 4-2 shows the framework for a human health risk assessment. It includes the following four components (or steps):

1. Planning and scoping (data evaluation)
2. Exposure assessment analysis
3. Toxicity assessment analysis
4. Risk characterization

#### Exhibit 4-2

<table>
<thead>
<tr>
<th>Planning and Scoping</th>
<th>Toxicity Assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Exposure Assessment</td>
<td></td>
</tr>
<tr>
<td>Who is exposed?</td>
<td>Is a chemical toxic?</td>
</tr>
<tr>
<td>What chemicals are they exposed to?</td>
<td>What is the relationship between the dose of a chemical and the response that results?</td>
</tr>
<tr>
<td>How does the exposure occur?</td>
<td></td>
</tr>
<tr>
<td>Risk Characterization</td>
<td></td>
</tr>
<tr>
<td>What is the likelihood that the exposure will result in an adverse health effect?</td>
<td></td>
</tr>
<tr>
<td>How sure are we that our answers are correct?</td>
<td></td>
</tr>
</tbody>
</table>

**Planning and Scoping:** A human health risk assessment should begin with proper planning and scoping. Good planning and scoping clearly articulates the assessment...
questions, states the quantity and quality of data needed to answer those questions; provides in-depth discussion of how assessors will do the analysis, outlines timing and resource considerations, as well as product and documentation requirements, and identifies who will participate in the overall process from start to finish and what their roles will be. The planning and scoping team generally makes initial decisions about the scope of the risk assessment (i.e., size of the study area, what emission sources and chemicals are to be considered), the appropriate level of detail and documentation; trade-offs between depth and breadth in the analysis, quality assurance and quality control requirements, analytical approaches to be used (modeling vs. monitoring), and the staff and monetary resources to commit.

**Analysis Phase:** The analysis phase is the process in which analysts apply risk assessment approaches to evaluate the problem at hand. It consists of two main components: exposure assessment and toxicity assessment.

An exposure assessment is conducted to characterize the potentially-exposed population, the chemicals of potential concern, identify exposure pathways and routes of exposure, and estimate the exposure. This includes estimating or measuring concentrations of air toxics in the environment and evaluating how nearby populations interact with the contaminated media.

In the exposure assessment, the risk assessment team will refine the initial conceptual model by providing detailed information about the study area (i.e., physical description, meteorology, source locations and detailed characteristics, population demographics and locations, the exposure pathways under study). The exposure assessment also is the analytic step in which the magnitude, frequency, and duration of human exposures are quantified. For example, one of the main outcomes of an air toxics exposure assessment is an estimate of the exposure concentration of air toxics in the air at the point where human contact occurs. Assessors usually estimate this value with either a computer program (a model) or by physically taking samples of air and measuring air toxics concentrations in a laboratory (a monitor). When there are concerns about exposure pathways other than inhalation, assessors may use different models or monitoring strategies to estimate or measure concentrations of air toxics in soil, water, or foods.

The toxicity assessment component of the risk assessment process considers: (1) the types of adverse health effects associated with exposure to the chemicals in question, and (2) the relationship between the amount of exposure and resulting response. Toxicity assessment for air toxics generally consists of two steps:
Hazard identification is the process of determining whether exposure to a chemical can cause an adverse health effect, as well as the nature and strength of the evidence of causation and circumstances in which these effects occur (e.g., inhalation/ingestion, repeated exposure over a long period /single exposure over a short period, etc.).

Dose-response assessment is the process of quantitatively characterizing the relationship between the dose of the contaminant and the incidence of adverse health effects in the exposed population. As information on dose at the site in the body where the response occurs is rarely available, various factors and models are used to predict the dose metric from estimates of exposure (the inhalation exposure concentration or oral intake). From this quantitative dose-response relationship, toxicity values are derived for use in risk characterization. Most toxicity assessments are based on studies in which toxicologists expose animals to chemicals in a laboratory and extrapolate the results to humans. For some chemicals, information from actual human exposures is available (usually from workplace exposure studies).

Risk Characterization: The risk characterization summarizes and combines outputs of the exposure and toxicity assessments to characterize risk, both in quantitative (numerical) expressions and qualitative (descriptive) statements. Chemical-specific exposure-response information is mathematically combined with modeled or monitored contaminant levels and other information regarding how exposure occurs to provide numerical expressions that represent the likelihood that the exposure may cause an adverse health outcome. The risk characterization also includes a thorough uncertainty analysis for each step of the entire risk assessment process in order to provide the risk manager with an understanding of which elements of the assessment are most uncertain, the magnitude and direction of the effect (higher or lower) that the various uncertainties have on the risk estimates and in some cases, a quantitative analysis of uncertainty. Often the uncertainty analysis is a narrative that reflects the assessor’s best professional judgment. Other analyses, however, may require a more quantitative approach to evaluating uncertainty.

4.5 Tiered Approach for Risk Assessment

EPA cannot perform a time and resource-intensive risk assessment for every situation and EPA decision. Consequently, for each risk assessment, EPA selects an approach that is consistent with the nature and scope of the decision being made. The appropriate approach depends on the needs of the decision maker and/or the role that risk
information plays in the decision, balancing uncertainty, and resources. Even when using the best models and data, uncertainty is still inherent in the process. Given that uncertainty is inherent, there is a continuing tension between improving our understanding in order to make a decision and the reality of limited resources to perform the analysis and the desire for timely decision making. Exhibit 4-3 illustrates this risk assessment continuum and the balance of resources and uncertainty as the assessment becomes more complex. It also illustrates that risk assessment can be performed with low levels of data and relatively little effort to develop conservative estimates of risk. Depending on the outcome and the needs of the risk manager, higher levels of analysis may be performed.

**Exhibit 4-3**

<table>
<thead>
<tr>
<th>The Risk Assessment Continuum: Tiered Approaches to the Process</th>
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<tbody>
<tr>
<td><strong>SCREENING</strong></td>
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<tr>
<td><strong>MORE REFINED</strong></td>
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<td></td>
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<tr>
<td>Complete study-specific data, no assumptions; higher cost, lower uncertainty</td>
</tr>
</tbody>
</table>

Note: As one moves up the risk assessment continuum, the data needs and costs also rise. However, the quality of the result should also rise as well.

This risk assessment continuum utilizes a tiered approach depicting three tiers of analysis (see Exhibit 4-4). Each successive tier represents more complete characterization of variability and/or uncertainty as well as a corresponding increase in complexity and resource requirements.

- **Tier 1** is represented as a relatively simple, screening-level analysis using conservative exposure assumptions (i.e., receptors are located in the area with the highest estimated concentrations) and relatively simple modeling (i.e., a model that requires few inputs, most of which can be “generic,” yet conservative).
- **Tier 2** is represented as an intermediate-level analysis using more realistic exposure assumptions (e.g., use of actual receptor locations) and more detailed modeling (i.e., a model that requires additional site-specific inputs).

- **Tier 3** is represented as an advanced analysis using probabilistic techniques such as Monte Carlo analysis (see Part VII of “Air Toxics Risk Assessment Reference Library, Volume I Technical Resource Manual” for a discussion of these techniques) and more detailed and/or intensive modeling.

**Exhibit 4-4: Generalized Representation of the Tiered Risk Assessment Concept**

While the tiered risk assessment concept usually contains three tiers of complexity, these three tiers are best thought of as points along a spectrum of increasing complexity and detail in the risk assessment. The important focus is the specific ways in which a given risk assessment is refined in successive iterations, rather than whether or not it would be considered Tier 1, 2, or 3. Exhibit 4-4 is illustrative of the concept of tiered approaches.

### 4.6 Risk Assessment and Risk Management

This section introduces risk management, focusing on its role in addressing the risks that air toxics pose. Specifically, **risk management** refers to the regulatory and other actions taken to limit or control exposures to a chemical. **Risk assessment**, on the other hand, is a tool used to support risk management decisions by providing quantitative and qualitative expressions of risk, along with attendant uncertainties. Specifically, the risk
assessment conveys a quantitative and qualitative description of the types of impacts that may occur from exposure to an air toxic, the likelihood that these impacts will occur given existing conditions, and the uncertainties surrounding the analysis. Risk management considers these principle factors along with a variety of additional information (which may include the cost of reducing emissions or exposures, the statutory authority to take regulatory actions, and the acceptability of control options) to reach a final decision. Additional information includes:

a. Economic factors – the costs and benefits of risk and risk mitigation alternatives

b. Laws and legal decisions – the framework that prohibits or requires some actions

c. Social factors – attributes of individuals or populations that may affect their susceptibility to risks from a particular stressor

d. Political factors – interactions among and between different branches and levels of government and the citizens they represent

e. Technological factors – the feasibility, impact, and range of risk management options

f. Public factors – the attitudes and values of individuals and societies with respect to environmental quality, environmental risk, and risk management

The National Academy of Sciences and others stress the distinction between risk assessment and risk management, while they also stress the integration of the two efforts (see Exhibit 4-5). Risk assessments are often designed and conducted with awareness of the risk management options available to decision-makers and the social, economic, and political context in which those decisions are made. Likewise, periodically reviewing the risk management options during the risk assessment effort ensures that the results of the risk assessment will provide meaningful input into the decision-making process. Ultimately, the risk assessors should be aware of risk management goals; however, the fundamental science performed in the risk assessment should be impartial and based on the factual base of information to the extent possible.
Two general categories of risk management decisions are relevant to air toxics: emissions control and sitting. Emissions control decisions may involve “command-and-control” decisions (i.e., emissions limits) or incentives (i.e., tax credits for reduced emissions). EPA’s preference is to encourage pollution prevention whenever feasible. Emissions control decisions are most likely to involve formal risk assessments. These decisions involve where to locate industrial facilities, businesses, waste disposal facilities, and transportation routes. Siting decisions are typically made by governments through mechanisms such as zoning, deed restrictions and other property controls and, in some cases, regulation. Many of these decision-making processes include public involvement in which citizens may seek to influence the final decision. Siting decisions may increasingly involve air toxics risk assessments.

4.7 Community Involvement and Risk Communication

4.7.1 Community Involvement: Community involvement can be an important aspect of the risk assessment and risk management process. Participation of local stakeholders, at various levels and in various forms, can help ensure a better understanding of the risk
assessment results and will promote buy-in to the selected risk reduction strategies. Encouraging and facilitating community involvement also is sometimes required by law.

When performing an air toxics risk assessment in a particular geographic area, the community is often thought of as the people who live within the area of impact of air toxic sources. However, other parties in the area, such as local industry may also consider themselves part of the community.

In addition to the people who actually live and work in an area, a number of other stakeholders may also have a stake in the community’s concerns (e.g., local officials, health professionals, and local media). It is often helpful, when dealing with a community, to keep in mind that many different people (not just the people who live there) may have an interest in the risk assessment and management work being undertaken.

As noted above, many laws recognize and accommodate the idea that government decisions should be open to citizen input before a decision is finalized. This is realized through the required public meetings and public comment periods associated with many government actions. For example, the Clean Air Act (CAA) has a number of requirements to provide an opportunity for the public to review and comment on Agency proposals. In some cases, the public is brought in at an even earlier stage.

When possible, risk assessors and risk managers should consider including the public early in the process. Doing so can lead to some very positive benefits. For example, if the community participates early on and throughout the process, they will be in a better position to understand what assessors and risk managers are doing, and there is a better chance that they will believe that the work being done is in their best interest. The process works best when the community appreciates that assessors and managers are working with them and respecting their input (keeping them informed and involved). Ultimately, a community that is involved early on in the process may be more willing to support the risk assessment process and results. This may, in turn, foster the development of risk reduction strategies the community as a whole can live with and have a stake in.

In addition to fostering the trust and acceptance of the community, there are many other positive reasons for early and ongoing involvement. For example, important unrecognized sources of emissions and exposure pathways may be identified through the community involvement process. Ultimately, it is important to recognize that community
members know their community and understand the types of solutions that will be most accepted – after all, they live there.

4.7.2 Risk Communication: Risk communication involves making risk assessment and risk management information understandable to the layperson, including community groups, environmentalists, lawyers, and the media. It allows decision makers to communicate with various interested parties about the nature and level of risk, and about risk reduction strategies to reduce the risk. The purpose of risk communication is to help in planning of the risk assessment and to convey the results of the risk assessment in a way that effectively supports risk management decisions.

Good risk communication strategies are a fundamental aspect of developing trust among various stakeholders and the community and are often considered an important first step before conducting the risk assessment. Involving the community, establishing and maintaining relationships, and networking with other partners (i.e., agencies, organizations, officials, and the media) are key elements in a risk communication strategy. Tailoring communications to the cultural diversity of the community may help establish the trust necessary to complete a risk assessment that meets all stakeholder and community needs.

Interested parties typically want to know if something is safe, not how uncertain the risk may be. If people perceive themselves to be at risk, their perception is unlikely to change even if they are not being exposed or harmed. Elements that affect risk perception include experience, culture, level of education, outrage factors, who is affected/how they are affected (equal treatment), and the level of control exercised on the event. Many successful risk communication efforts have had one major thing in common – a portrayal that puts the calculated exposure risks from an assessment in perspective, with risk ranges the public can easily relate to and understand.

Several general statements can be made concerning the average person’s perception of risk. For example, voluntary risks are accepted more readily than those that are imposed. An individual will accept a high risk if he voluntarily exposed himself to the high risk activity. A risk resulting from a chemical exposure is not a voluntary risk and, therefore, not well accepted. Another example would be the controllability of risk. If the risk from taking an over-the-counter drug is the same as that as being exposed to a toxic air pollutant, the ability to control our exposure to some degree makes the risk seem lower. A final example is that natural risks seem more acceptable than artificial risks. Risks from a natural disaster (lightning, tornadoes, earthquakes, floods) are more acceptable to a person than an artificial risk like air pollution, regardless if the quantitative risk.
numbers are the same. These are just a few examples of the predictable impact on the public perception of risk to be considered in risk communication.

Risk communication strategies also consider whether the listener understands how to use the information in forming opinions, making decisions, and taking actions. When risks are calculated for air toxics and the risk results are presented the community may be unfamiliar with quantitative risk data and what it means for them. In order to prevent panic and to encourage participation in and buy-in of risk management decisions, risk communication strategies are developed to reassure the community and explain the potential risks and uncertainties in a clear and honest way.

The media can be a primary source of information on risks to the public. Effective news media relations have many benefits, complementing other communication efforts. What people observe in news coverage can lend credibility to agencies associated with air toxics risk assessment, and can help to make it a familiar topic for public discussion. News coverage can inform people about air toxics issues. Skill in media relations can help risk communications avoid or dispel rumors, respond to criticism, defuse controversy, and even turn adversity to advantage.

The best approach to the media, as with the public, is open and honest information tailored to the needs of each type of media, such as graphics and other visual aids, and provides background material via fact sheets, press kits, and lists of experts. Establishing an information center also can be an effective way to make materials available to the news media and to the general public.

4.8 Air Toxics Risk Assessment Library: 3 Volumes


The primary purpose of Volume 1 is to provide, in one place, descriptions of the major methods and technical tools commonly used to perform air toxics risk assessments. Specifically, the manual attempts to cover all common basic technical approaches used to evaluate: how people in a particular city or neighborhood may be exposed; what chemicals they may be exposed to and at what levels, how toxic those chemicals are, and how likely it is that the exposures may result in adverse health outcomes. Topics include
uncertainty and variability, basic toxicology and dose-response relationships, air toxics monitoring and modeling, emissions inventory development, and risk characterization. The manual also discusses approaches for using the results of a risk assessment in the risk management decision-making process. Links to more detailed references on each subject are presented, along with EPA contacts. Additionally, EPA’s Fate, Exposure, and Risk Analysis (FERA) web site http://www.epa.gov/tnn/fera/ provides up-to-date tools for air toxics risk assessment, including computer models, databases, and other information used by EPA and others for air pollutant human exposure modeling, multimedia modeling, and risk.

Volume 1 is divided into six parts, each comprised of three or more chapters. A number of Appendices provide more detailed reference materials.

- **Part I (Background) - Introduction** to air toxics risk assessment and is divided into four chapters:

  - Chapter 1 - Introduction to the Volume 1 manual.
  - Chapter 2 - Overview of the CAA and major regulations, programs, and initiatives that relates to air toxics risk reduction.
  - Chapter 3 - Overview of risk assessment and the risk-based decision making framework, including an introduction to tiered approaches to risk assessment.
  - Chapter 4 - Set of chemical pollutants that are the focus of the Volume 1 manual, the general categories of air toxics sources, and the primary emissions inventories (which contain information on the nature and magnitude of emissions released from various sources).

- **Part II (Human Health Risk Assessment: Inhalation)** - Methods and tools used to evaluate risks to human health via the inhalation pathway, it is divided into nine chapters.

  - Chapter 5 - Overview of the inhalation risk assessment process, discusses the initial planning and scoping process that needs to be completed before the risk assessment begins, and describes the exposure assessment, which usually comprises the bulk of the effort for most air toxics risk assessments.
  - Chapter 6 - Problem formulation phase which results in the development of the conceptual model and analysis plan for the risk assessment.
  - Chapter 7 - How to develop an emissions inventory for the risk assessment.
  - Chapter 8 - Factors that affect the movement and, in some cases, chemical transformation and the fate and transport of chemicals in the atmosphere.
– Chapter 9 - Computer modeling to predict the movement, fate, and transport of air toxics in the atmosphere and the major computer models commonly used for this purpose.
– Chapter 10 - Monitoring methods are commonly used to measure ambient concentrations of air toxics in the atmosphere.
– Chapter 11 - Estimating exposure concentrations for inhalation analyses, including exposure modeling.
– Chapter 12 - Toxicity assessment for air toxics.
– Chapter 13 - Completing the risk characterization, including uncertainty analysis and how to present the results of the risk assessment.

• Part III (Human Health Risk Assessment: Multi-pathway) - Methods and tools used to evaluate risks to human health when air toxics that are highly persistent or bioaccumulative are present in emissions. Multi-pathway risk assessment is to evaluates the potential exposures associated with ingesting soil, food, and water contaminated with chemicals after deposition from the atmosphere to surfaces, such as soils and surface waters. This Part is divided into nine chapters:

  – Chapter 14 - Multi-pathway risk assessment process, discusses the initial planning and scoping process that needs to be completed before the risk assessment begins, and describes the multi-pathway exposure assessment.
  – Chapter 15 - Problem formulation for the multi-pathway risk assessment.
  – Chapter 16 - Developing an emissions inventory for the multi-pathway risk assessment.
  – Chapter 17 - Factors that affect the movement and, in some cases, chemical transformation of air toxics in soil, water, sediment, and biota.
  – Chapter 18 - Computer modeling used to predict the movement, fate, and transport of toxics in soil, water, sediment, and biota and the major multimedia computer models commonly used by risk assessors.
  – Chapter 19 - Monitoring methods used to measure ambient concentrations of air toxics in soil, water, sediment, and biota.
  – Chapter 20 - Summary of the process and assumptions used to estimate chemical intake rates – the key measure of exposure used to assess ingestion risks – including exposure modeling.
  – Chapter 21 - Toxicity assessment for air toxics that are persistent and which may have a high potential to bio-accumulate in food chains.
  – Chapter 22 - How to complete the risk characterization for the multi-pathway risk assessment, including uncertainty analysis and how to present the results of the risk assessment.
• **Part IV (Ecological Risk Assessment)** - Methods and tools used to evaluate risks to ecological receptors (e.g., birds, mammals, plants, and ecological communities) due to exposure to air toxics. This Part is divided into four chapters.

  – Chapter 23 - Ecological risk assessment process and discusses the initial planning and scoping process to be completed before the risk assessment begins.
  – Chapter 24 - Characterizing exposure for the ecological risk assessment.
  – Chapter 25 - Characterizing ecological effects, including development of the stressor-response profile.
  – Chapter 26 - Risk characterization for the ecological risk assessment, including the analysis of uncertainty, and how to present the results of the ecological risk assessment.

• **Part V (Risk-Based Decision Making)** - Process by which the information from the risk assessment can be used to inform risk management decisions and two important aspects of that process. This Part is divided into three chapters.

  – Chapter 27 - Risk management process, including the types of decisions that may need to be made and how the risk assessment informs the decision-making process.
  – Chapter 28 - Stakeholder involvement in the risk assessment and management process and provides information for developing and implementing a stakeholder involvement plan.
  – Chapter 29 - Developing and implementing a risk communication strategy for the community and the media to understand the risk assessment results and use in the decision-making process.

• **Part VI (Special Topics)** - Three tools or procedures that may be used as part of performing or reporting a risk assessment.

  – Chapter 30 - Process by which public health agencies may evaluate the public health implications posed by the emissions from air toxic sources in a community. The public health assessment is a complementary process to risk assessment.
  – Chapter 31 - Risk assessment aimed at describing risks as a distribution (or range) of potential outcomes.
  – Chapter 32 - Geographical Information System (GIS) tools in the process of conducting risk assessments and reporting results.

• **Glossary** defines key terms and acronyms.

• **Appendix A** - All HAPs along with their status as a Toxics Release Inventory (TRI) chemical, a Section 112(k) high priority urban toxic, and a Mobile Source Air Toxic.
Introduction to Air Toxics

- **Appendix B** - Agencies and organizations that oversee air toxics regulations.

- **Appendix C** - Dose-response values for cancer and non-cancer effects for all HAPs.

- **Appendix D** - Process by which the persistent, bioaccumulative HAP compounds (PB-HAPs) were selected.

- **Appendix E** - CAA designated air toxics Source Categories, including the most common HAPs in emissions, typical industries, and applicable maximum achievable control technology (MACT) standards.

- **Appendix F** - Specific pollutants and compound groups included in the 1999 National Emissions Inventory (NEI) along with their Chemical Abstract Services (CAS) numbers.

- **Appendix G** - Meteorology as it relates to the movement of air toxics in the atmosphere and information on sources of meteorological data for modeling air toxics dispersal and transport.

- **Appendix H** - Evaluating and reducing a monitoring data set (e.g., air, water, soil sample results) into a grouping of data useable for exposure evaluation.

- **Appendix I** - How a reduced monitoring data set, developed by the methods in Appendix H, may be used to estimate exposure concentrations.

- **Appendix J** - Available air toxics monitoring methods.

- **Appendix K** - Equations for calculating concentrations of PB-HAPs in non-air media (e.g., soil, food, water).

**The Relationship of Volume 1 to Volumes 2 and 3:** Volume 1 is the first volume of a three-volume set. **Volume 1: Technical Resource Manual** discusses the overall air toxics risk assessment process and the basic technical tools needed to perform these analyses. The manual addresses both human health and ecological analyses. It also provides a basic overview of the process of managing and communicating risk assessment results. Other evaluations (such as the public health assessment process) are described to give risk assessors, risk managers, and other stakeholders a more holistic understanding of the many issues that may come into play when evaluating the potential impact of air toxics on human health and the environment.
Volume 2: Facility-Specific Assessment builds on the technical tools described in Volume 1 by providing an example set of tools and procedures that may be used for source-specific or facility-specific risk assessments, including tiered approaches to source- or facility-specific risk analysis.

Volume 3: Community-Level Assessment builds on the information presented in Volume 1 to describe to communities how they can evaluate and reduce air toxics risks at the local level. The volume will include information on screening level and more detailed analytical approaches, how to balance the need for assessment versus the need for action, and how to identify and prioritize risk reduction options and measure success. Since community concerns and issues are often not related solely to air toxics, the document will also present readily available information on additional multimedia risk factors that may affect communities and strategies to reduce those risks. The document will provide additional, focused information on stakeholder involvement, communicating information in a community-based setting, and resources and methodologies that may play a role in the overall process.
References


Chapter 5: Dispersion, Transport, Fate, and Modeling of Air Toxics in the Atmosphere

5.1 Introduction

Dispersion, transport, and fate are the links between release of air toxics from a source and its contact with a receptor at an exposure point. Analysis of the links involves obtaining the release characteristics of air toxics at the source and following it to the receptor, taking into account physical/topographical/meteorological factors and physical and chemical changes that the air toxics may undergo. A variety of mathematical models, each with specific data needs, has been or is under development to describe the transport and fate of pollutants released to the atmosphere. Any available air toxic monitoring data can be used to check the validity of these modeled concentration estimates.

Dispersion, transport, and fate of air pollutants in the atmosphere: Dispersion (applied to air toxics releases) means to spread or distribute from a source with (generally) a decrease in concentration with distance from the source. Dispersion is affected by a number of factors including characteristics of the source, the pollutants, and ambient atmospheric conditions. Transport refers to the processes (i.e., winds) that carry or cause pollutants to move from one location to another, with or without a change in concentration, but with no change in chemical identity. Fate refers to a transformation either through chemical or biological mediation, to where a pollutant ultimately ends up (i.e., air, soil, water, fish tissue), and how long it persists in the environment.

In the Detailed Air Toxic Risk Assessment Process (see Exhibit 5-1), the pollutants of interest and their sources and emission rates are first defined in the exposure assessment process, and then continues with the estimation of the pollutants’ fate and transport. This step describes how the pollutant is transported, dispersed, and transformed over the area of interest. Initially, the fate of the emitted pollutants is largely determined by the source release characteristics. After pollutants are released to the atmosphere, their transport, dispersion, and transformation are governed by meteorological principles, terrain characteristics, wet and dry deposition rates, and certain chemical properties of the HAP (such as aqueous solubility, vapor pressure, air-water partition coefficient [i.e., Henry’s Law constant], molecular diffusivity, phase partition coefficient, melting point, and adsorptivity). For a limited subset of HAPs, it is important to consider deposition from air to soil, vegetation, or water-bodies; for others, such deposition is not important. The HAP’s reactivity and persistence will influence its fate as well and can be important factors in estimating exposure for certain pollutants. Additionally, secondary
transformation products of some HAPs may need to be identified for consideration in the risk assessment process. High quality, representative meteorological information and local topography are crucial to a valid exposure assessment for air toxics.

Many studies indicate that a limited number of pollutants emitted into the atmosphere (i.e., mercury) are passed to humans or wildlife through non-inhalation pathways; for example, a HAP depositing from the air onto the soil, followed by ingestion of the soil by a child or by biota in an ecosystem. Exhibit 5-2 is an example of the conceptual model diagram for an ecological risk scenario involving multi-pathway exposure to HAPs. For a limited subset of HAPs, greater human and ecological exposures to the HAP occur through non-inhalation exposures than through inhalation exposures. These HAPs typically are persistent in the environment, have a strong tendency to bio-accumulate, and exhibit moderate to high toxicity.
5.2 Dispersion and Atmospheric Transport of Air Pollution

Several characteristics of the source can affect the movement of air toxics while they are still close to the source (i.e., source height, gas exit temperature). Once air toxics are transported beyond the immediate vicinity of the source, atmospheric and meteorological factors (particularly wind speed and direction) govern air toxics dispersion and transport. This section describes how the movement of air toxics is affected by source characteristics, chemical properties, and atmospheric processes.

5.2.1 General Types of Air Pollution Releases

The distinction between the major types of air pollution releases is not always clear, and the same air toxic can often be released in more than one way from a single source or process. The following terms are routinely used to describe or categorize emissions at a facility:

**Stack or Vent Emissions:** These emissions are how most people envision air pollution. Stacks and vents include “smokestacks” that emit combustion products from fuel or waste combustion, as well as vents that carry air toxics away from people or industrial processes. The major characteristics that stack and vent emissions share are that the release is intentional, they remove airborne materials from specific locations or processes, and they channel the releases through specifically designed dedicated structures. Often, stack and vent releases involve the active “pumping” of pollutant-laden air to the external atmosphere by using fans or the “draft” associated with the tendency of hot gases to rise rapidly through cooler, denser air. A 2001 Toxic Release Inventory
(TRI) report shows that 86% of emissions from large industrial sources are from their stacks and vents.

**Fugitive Emissions:** “Fugitive” emissions are uncontrolled air pollutant releases that “escape” from physical, chemical, or industrial processes and activities, and do not travel through stacks or vents. Examples include dust or vapors that are generated by the transfer of bulk cargo (i.e., coal, gravel, occasionally organic liquids) from one container to another (i.e., from a tank or hopper car to a storage silo, tank, or bin). Another example includes leaks from joints and valves at industrial facilities and evaporative emissions of fuel from mobile sources.

### 5.2.2 Source Characteristics that Affect Dispersion and Transport

EPA and others have developed several air “dispersion” models to predict the often-complex behavior of air pollution releases and take into account a number of characteristics of the source and pollutants released. Available air pollution dispersion models differ in the ways in which they use these characteristics, as described below:

**Release Rate and Volume:** The rate of release (exit velocity) strongly influences the behavior of the pollutant plume as it moves through the atmosphere. In the case of stack and vent releases, sources can release pollutants as pure vapors, as dilute solutions of vapor in air or other gases, or as suspended particles. Large volumes are often released at a relatively high velocity from stacks or vents, which serves to drive pollutants higher in the atmosphere. Air quality models often calculate volume from data on exit area and exit velocity. In the case of fugitive releases or volatilization, the “volume” of release has less meaning, and often does not receive explicit consideration in fate and transport modeling.

**Concentration:** Concentration (the mass of pollutant per unit volume of released gases) is the other half of the equation that determines the amount (mass) of pollutant released.

**Temperature:** The temperature of a plume emitted from a stack or vent influences the dispersion and transport of pollutants. A plume that is warmer than the surrounding air will generally rise, which tends to increase the distance over which pollutants will be transported. The combination of temperature and vertical velocity of stack emissions combine to affect the height to which the plume will rise and the layer of the atmosphere in which it will initially be transported. The plume also affects the physical form of pollutants, with less volatile pollutants condensing faster from cooler plumes.
**Height:** Pollutants may also be released into the atmosphere at different heights, and the height of release can strongly affect dispersion and transport. Greater release heights generally result in increased pollutant dilution in the atmosphere, lower ground-level concentrations, and a greater distance to peak ground-level concentrations. Release height also is important in evaluating local effects on air transport, such as building downwash.

**Timing and Duration:** Multiplying the release duration by the release rate produces the total mass of pollutant released. The timing of release relative to specific meteorological conditions determines the particular dispersion and transport of pollutants.

**Physical Form:** The physical form of pollutant releases greatly affects the dispersion, transport and chemical reactions that pollutants undergo. Generally, pollutants are characterized as being *vapors* (not bound to particles, but existing as single molecules or very small aggregates “dissolved” in air – also called gaseous), *particle-bound* (reversibly absorbed or condensed onto the surface of particles), or *particulate* (irreversibly incorporated into airborne particles). Sources can emit chemical pollutants in the vapor phase at relatively high temperatures, and these pollutants can condense into or onto particulates as the emitted gases cool in the atmosphere. Sources generally emit most metals (with the important exception of mercury) as particles in the atmosphere.

**Particle Size:** When sources release pollutants as particles (or when released as gases, if these pollutants condense into particles or absorb onto the surface of existing particles), the rate of pollutant removal from the atmosphere to surfaces depends upon particle size. The typical size of particles that different activities and processes emit into the air can vary by many orders of magnitude (powers of ten). As the size of particles increases, the rate at which particles fall due to gravity (the settling velocity) increases. Thus, fine particles (approximate diameter less than a few microns) may remain suspended in air indefinitely, but particles larger than about 20 microns in diameter settle rapidly and may not transport far from sources of release.

For purposes of air toxics risk assessment, particles less than 10 microns in diameter are of primary concern because they are small enough to be taken into and deposited in the lung after inhalation. These particles are divided into two size ranges: “fine” particles less than 2.5 micrometers in size (PM2.5), and “coarse” particles covering the range from 2.5 to 10 micrometers in diameter (PM10).
5.2.3 Meteorological Factors Affecting Air Toxics Dispersion and Transport

Whereas large-scale pressure systems control the prevailing meteorology of a region, local meteorological factors such as horizontal airflow (wind speed and direction) and vertical air movement (atmospheric stability) affect the dispersion and transport of air toxics.

5.2.3.1 Plume Rise: Pollutants enter the atmosphere in a number of different ways. One method that has received much attention is pollution released from stacks (point sources). Stacks release pollutants high enough above the earth’s surface that pollutants can sufficiently disperse in the atmosphere before reaching the ground. The air space the stack pollutant occupies can be described as a plume. As the plume travels, it spreads and disperses, reducing ambient pollutant concentrations even though the cross-sectional mass of the plume remains the same. Eventually, the plume may intercept the ground. The combination of emission velocity, emission temperature, vertical air movement, and horizontal airflow all influences how high a plume will rise and how fast and far it travels. Another factor is wind meander (i.e., changes in wind direction during light wind speed conditions), which can cause the plume to deviate in the horizontal direction due to turbulence and wind fluctuation.

This plume rise allows air toxics emitted in this stack stream to be lofted higher in the atmosphere. Since the plume is higher in the atmosphere where the winds are generally stronger, the plume will generally disperse more before it reaches ground level. Plume rise depends on the stack’s physical characteristics and on the effluent’s exit temperature and velocity. The condition of the atmosphere (i.e., the vertical profile of the winds and temperature) along the path of the plume also determines how far the plume rises in the atmosphere (see Exhibit 5-3).

\[ \text{Plume height} = \text{Hs} + \Delta H \]

*Physical release height (Hs)
  - From a stack
  - From an area/volume source
  - From the ground

*Plume rise (\( \Delta H \))
  - Exit velocity
  - Stack temperature
  - Wind speed

Exhibit 5-3
5.2.3.2 Horizontal Motion of the Atmosphere (Wind Speed and Direction): The initial velocity of the plume (stack exit velocity) reduces quickly as the plume entrains ambient air and acquires horizontal momentum from the wind. This momentum causes the plume to bend over. The greater the wind speed, the more horizontal momentum the plume acquires. Wind speed usually increases with height above the earth’s surface. Therefore, as the plume continues upward the stronger winds tilt the plume even further. This process continues until the plume may appear to be horizontal to the ground.

It is common to consider wind speed and wind direction as separate variables. Wind speed determines the amount of initial dilution experienced by air toxics released into the atmosphere.

Wind speed also influences the height to which the toxics will rise after being released from an elevated source - as wind increases, the air toxics are kept lower to the ground, allowing them to impact the ground at shorter distances downwind.

Wind direction for meteorological purposes is defined as the direction from which the wind is blowing. However, wind direction has both horizontal and vertical components that can be measured with a bi-directional wind vane or an anemometer.

Wind roses are often used to graphically depict the prevailing wind direction of an area. The wind rose depicts the relative frequency of wind direction, typically on a 16-point compass, with north, east, south, and west directions going clockwise. Each ring on the wind rose represents a frequency of the total. A wind rose illustration is for a given locality or area and for a selected time period, typically a year or longer. In Exhibit 5-4, the length of the bars indicates the percentage of the year the wind blows in each of 16 directions and the colors indicate the percent of time at that wind speed for each of these directions.
5.2.3.3 Vertical Air Motion and Atmospheric Stability: When air is displaced vertically, atmospheric behavior is a function of atmospheric stability. Stability is a measure of whether pollutants emitted will convectively rise and disperse, or build up in concentration near the surface. A stable atmosphere resists vertical motion, and air that is displaced vertically in a stable atmosphere tends to return to its original position. This atmospheric characteristic determines the ability of the atmosphere to disperse pollutants. To understand atmospheric stability and the role it plays in pollution dispersion, it is important to understand the mechanics of the atmosphere as they relate to vertical atmospheric motion.

The degree of stability of the atmosphere is determined by the temperature difference between an air parcel and the surrounding air. This difference can cause the parcel to rise or fall. There are three general categories of atmospheric stability: stable, unstable and neutral. When conditions are extremely stable, cooler air near the surface is trapped by a layer of warmer air above it: this is called an inversion.

- In stable conditions, vertical movement tends not to occur. Stable conditions occur at night when there is little or no wind. Air that is lifted vertically will
remain cooler, and therefore denser than the surrounding air. Once the lifting force is removed, the air that has been lifted will return to its original position.

- **Neutral** conditions ("well mixed") neither encourage nor discourage air movement. Neutral stability occurs on windy days or when there is cloud cover allowing neither strong heating nor cooling of the earth’s surface. Air lifted vertically will generally remain at the lifted height.

- **In unstable** conditions, the air parcel tends to move upward or downward and to continue that movement. Unstable conditions most commonly develop on sunny days with low wind speeds where strong solar radiation is present. The earth rapidly absorbs heat and transfers some of it to the surface air layer. As warm air rises, cooler air moves underneath. The cooler air, in turn, may be heated by the earth’s surface and begin to rise. Under such conditions, vertical motion in both directions is enhanced, and considerable vertical mixing occurs.

- **Inversions** occur whenever warm over-runs cold air and "traps" the cold air beneath. During an inversion, air temperature increases with altitude. Within these inversions there is little air motion, and the air becomes relatively stagnant. High air toxic concentrations can occur within inversions due to the limited amount of mixing between the "trapped" air and the surrounding atmosphere. Inversions allow no vertical air motion and can limit the volume of air into which emissions are dispersed, even from tall stacks. Plumes emitted into air layers that are experiencing an inversion do not disperse very much as they are transported in the wind.

### 5.2.4 Physical Factors Affecting Air Toxics Dispersion and Transport

Due to configuration of the stack or adjacent buildings, the plume may not rise freely into the atmosphere. The way in which the wind moves around adjacent buildings and the stack can force the plume toward the ground instead of allowing it to rise in the atmosphere. Exhibit 5-5 shows how a structure can disturb the horizontal flow of the wind (indicated by dashed line) and cause a plume to get "down-washed" to the ground quickly. Stack-tip *downwash* can occur where the ratio of the stack exit velocity to horizontal wind speed is small. In this case, low pressure in the wake of the stack may cause the plume to draw downward behind the stack. Pollutant plume rise reduces when this occurs and elevates pollutant concentrations immediately downwind of the source. As air moves over and around buildings and other structures, it forms turbulent wakes. Depending upon the stack height, it may be possible for the plume to be pulled down into
this wake area. The reduction in plume height is known as aerodynamic or building downwash.

Exhibit 5-5

5.3 Physical Processes Removing Air Toxics

A number of important physical processes (processes that do not alter the chemical nature of pollutants) affect how air toxics move in and out of the atmosphere. In particular, this section discusses how gravity and precipitation remove air toxics from the atmosphere. The process through which particulates fall (or settle) to the surface in the absence of precipitation is known as dry deposition, and the removal of pollutants from the air through precipitation events is called wet deposition.

5.3.1 Dry Deposition

As the previous section noted, dry deposition is the settling of particles due to gravity. The maximum speed at which a particle will fall in still air is known as the settling velocity (settling rate). A particle’s settling velocity is a function of its size, density, and shape. Larger, denser particles settle more rapidly, and particles with more irregular shape settle more slowly. For particles smaller than a few microns in diameter (fine and ultrafine particles), the gravitational settling rate is so slow that other forces, such as local air currents and collisions with gas molecules, tend to offset it. Thus, in the absence of other removal mechanisms (i.e., condensation and/or aggregation to form larger
particles, wet deposition), particles in this size range tend to remain suspended in the air for long periods of time. Depending on the conditions, fine particles may persist in the atmosphere for days or weeks and travel hundreds or thousands of miles from their source.

At the other extreme, coarse dust particles (> 50 microns in diameter), such as those generated while handling materials, have large settling velocities. Under normal conditions, such particles generated near the ground will deposit on the surface within a few seconds or minutes, generally within less than a kilometer of the source. Particles in between these two extremes in the size distribution will settle at intermediate velocities, and will distribute at intermediate distances from their sources.

In typical air dispersion models, the modeler must specify a particle size distribution, classifying what proportions of the emitted particles are within particular size ranges. In initial screening level analyses of pollutant levels in air, users assume that particulate settling does not occur. This is conservative relative to the air concentration - if the amount deposited to the ground is the key issue of concern, then a high removal rate from the atmosphere would be “conservative.” Users can assess dry deposition for low-volatility pollutants that partition out of the air primarily onto airborne particles in the same fashion as non-volatile particulates. Volatile chemicals that exist primarily in the vapor phase have negligible settling velocities, and modelers generally need not consider dry deposition for these pollutants. In the case of pollutants whose vapor-particle partitioning is unclear, it is common to run air dispersion models assuming a range of partitioning behavior from fully particle-bound to fully vapor phase.

5.3.2 Wet Deposition

Wet deposition involves the “washing out” of pollutants from the atmosphere through precipitation events (including rain, snow, and, in some cases, hail). Wet deposition affects both particulate and vapor-phase pollutants. For larger particles and vapor phase pollutants that are soluble in water, precipitation is very efficient at removing pollutants from the air and depositing them on the earth’s surface. Wet deposition may be less efficient at removing fine particulates, and has limited effect on the levels of gaseous pollutants with high Henry’s Law constants (indicating low solubility in water compared to vapor pressure). Because wet deposition depends on the occurrence of precipitation events, it is best characterized over long periods (i.e., seasons or years). The relative importance of precipitation in removing pollutants from the air depends on the climatic conditions in the areas affected by pollution.
5.4 Chemical Reactions of Air Toxics in the Atmosphere

In addition to deposition, chemical reactions may occur that reduce air toxics concentrations. Air toxics may be destroyed through the action of sunlight, through reactions with atmospheric chemical pollutants, or through a combination of these pathways. In estimating the ambient air concentration associated with air toxics releases, it is therefore necessary to consider chemical reactions as well as deposition. As will be discussed in the next section, not all chemical reactions result in the destruction of air toxics, or their conversion to less harmful products.

Potentially harmful pollutants may also be formed as a result of atmospheric chemical reactions (a process that is called secondary production or secondary formation). This section, however, focuses on atmospheric chemical reactions which are known or believed to destroy air toxics (i.e., resulting in less toxic forms).

5.4.1 Major Chemical Reactions of Air Toxics: Generally, organic compounds are much more susceptible to chemical reactions in the atmosphere than metals or other inorganic contaminants. The major chemical reactions undergone by organic chemicals in the atmosphere include:

- Photolysis (destruction by sunlight alone)
- Reactions with the hydroxyl radical (OH•)
- Reactions with the nitrate radical (NO3•)
- Reaction with ozone (O3)

Often these reactions occur in combination with reactions that are strongly affected by sunlight.

While reaction rates vary widely for pollutants, under typical atmospheric conditions, reactions with the hydroxyl radical are the most rapid, and account for a large portion of pollutant degradation during daylight hours. Reactions with the nitrate radical occur primarily during the night, and reactions with ozone occur both day and night. Except in the case of a few pollutants, “pure” photolysis is a relatively minor reaction process. The relative importance of these reactions is dependent not only on climatic factors (i.e., duration and intensity of sunlight), but also on the overall concentrations of pollution present. For example, high levels of nitrogen oxide (NOx) emissions and emissions of VOCs increase the levels of nitrate radicals and ozone in the atmosphere, thereby increasing reaction rates for subsequent reactions where these species are involved.
5.4.2 Chemical Reactions that Result in the Secondary Formation of Pollutants:
As noted previously, not all chemical reactions result in the destruction of pollutants or in reaction products that are of less concern than the pollutants from which they derive. In some cases, the immediate reaction products result in more toxic and/or more persistent products than the chemicals that were originally released into the atmosphere.

Examples of large-scale chemical reactions that result in products that can be hazardous to health include the generation of acid particulate through photo-oxidation after the release of sulfur dioxide (SO₂) and NOₓ from combustion sources (i.e., to make sulfuric acid and nitric acid), and the formation of ozone and photochemical oxidant in areas with high levels of NOx and volatile organic emissions. In addition, there are many reactions of specific organic pollutants that generate air toxics of concern, as Exhibit 5-6 shows. The extent to which these reactions are important at any given location depends, of course, on the emissions and resulting concentrations of the precursor materials. In addition, many of these reactions are catalyzed directly and indirectly by sunlight, so weather and climatic factors are important in judging the importance of secondary formation. While it is difficult to generalize, the secondary formation of formaldehyde and acrolein are thought to be important in many regions of the country with significant industrial and mobile source emissions.

Exhibit 5-6: Examples of Secondary Pollutants

<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Formed From</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetaldehyde</td>
<td>propene, 2-butene</td>
</tr>
<tr>
<td>acrolein</td>
<td>1,3-butadiene</td>
</tr>
<tr>
<td>carbonyl sulfide</td>
<td>carbon disulfide</td>
</tr>
<tr>
<td>o-cresol</td>
<td>toluene</td>
</tr>
<tr>
<td>formaldehyde</td>
<td>ethene, propene</td>
</tr>
<tr>
<td>hydrogen chloride</td>
<td>nitric acid, chlorinated organics</td>
</tr>
<tr>
<td>methylethyl ketone</td>
<td>butane, branched alkenes</td>
</tr>
<tr>
<td>N–nitroso-N–methylurea</td>
<td>N–methylurea</td>
</tr>
<tr>
<td>N–nitrosodiethylamine</td>
<td>dimethylamine</td>
</tr>
<tr>
<td>N–nitrosomorpholine</td>
<td>morpholine</td>
</tr>
<tr>
<td>phosgene</td>
<td>chlorinated solvents</td>
</tr>
<tr>
<td>propionaldehyde</td>
<td>1-butene</td>
</tr>
</tbody>
</table>

Source: Rosenbaum et al., 1998
5.5 Air Quality Models

Air quality models use mathematical and numerical techniques to simulate the physical and chemical processes that affect air pollutants as they disperse and react in the atmosphere. Based on inputs of meteorological data and source information like emission rates and stack height, these models are designed to characterize primary pollutants that are emitted directly into the atmosphere and, in some cases, secondary pollutants that are formed as a result of complex chemical reactions within the atmosphere. These models are important to our air quality management system because they are widely used by agencies tasked with controlling air pollution to both identify source contributions to air quality problems and assist in the design of effective strategies to reduce harmful air pollutants.

The model chosen must be appropriate for the intended application, which may vary among estimates of short-term peak concentrations immediately adjacent to a facility, long-term concentrations over a city-wide area, or deposition over hundreds or even thousands of miles. Any available HAP monitoring data can be used either to check the validity of modeled concentration estimates or as a primary or supplemental source of information for the exposure assessment itself.

Air quality models provide estimates of ambient air concentrations at specific points distant from the source(s) being modeled. These are either predetermined within the model or selected by the analyst. In the simplest models (i.e., SCREEN3), the points are laid out along a vector (straight line) from the source. Many other models use a grid system to calculate ambient concentrations at specific exposure points at specified “nodes.” The model does not always automatically provide an estimate of concentration at every desired location, and extrapolation to desired locations is often required.

5.5.1 Types of Models: In general, air quality models can be categorized as one of two types: steady-state and non-steady state models. The movement of mass away from the source is modeled in both types of models. The steady-state model assumes that no variations occur over a certain time period (typically, one-hour); the non steady-state allows time-varying changes, but this capability imposes the need for additional model inputs, increased computation resources, and increased model formulation complexities.

• Steady-state models assume no time-varying processes occur over the period of interest. Hence, material released travels infinitely in only one direction over the time period (i.e., one hour). Often, these models assume that the material is distributed normally (also termed a “Gaussian distribution”) and are thus called “Gaussian plume” models. The steady-state model typically uses meteorological information obtained near
the source and assumes it holds true throughout the modeling region (i.e., a 50-kilometer radius). Wind direction, wind speed, and atmospheric stability are used to predict concentrations. This type of model is most widely used for stationary sources and for non-reactive pollutants (although models can take into account deposition and simple linear decay). The models are least applicable in areas with rapid time-varying conditions, over spatially varying terrain and land use, over large spatial scales (> 50 km), and where complex atmospheric chemistry takes place.

- **Non-steady state models** can simulate the effects of time- and space- varying meteorological conditions on pollutant transport, transformation, and removal. The modeling region is typically divided into grid cells, and the model simulates movement of pollutants between cells by taking into account advection, degradation, and other physical and chemical processes. These models are often used for chemically reactive pollutants or where there is complex topography or meteorology (i.e., complex sea breeze circulation). They require complex wind flow characterization and other detailed meteorological information for dispersion. For chemical transformation, they require information on the important chemical compounds as well as chemical kinetics to properly characterize the transformation and removal of air toxics. These models often take the form of grid models with the calculation of the physical and chemical processes taking place at each grid location. Other model types include “puff models,” which use a series of overlapping puffs to represent emissions. The calculations of the physical and chemical processes are made for each “puff.”

Another type of non-steady state model, the atmospheric trajectory model, uses meteorological data and mathematical equations to simulate transport in the atmosphere. The position of a parcel of air with time are calculated based on externally provided meteorological data such as wind speed and direction, temperature, humidity, and pressure. Model results depend on the spatial and temporal resolution of the meteorological data used, and also on the complexity of the model itself. Simpler models may deal with only two-dimensional transport by winds assuming the material emitted into the parcel stays at the same level, while more complex models may include 3-dimensional chemical and thermodynamic processes such as aerosol formation, convection, and turbulent diffusion.

### 5.5.2 Screening vs. Refined Models:

The overall accuracy and precision of results determined by a model is generally proportional to the complexity of the model, which in turn affects input data requirements and overall resources.

- **Screening-level models** are designed to provide conservative (i.e., high) estimates, and are useful for applications such as identifying facilities and/or air toxics that appear likely
to contribute the greatest risk among a group of sources and chemicals released. Data requirements are generally low (i.e., emission rates, some stack parameters), and running the models is generally easy and requires few resources.

- **Refined models** take into account more complex chemical behavior and a greater degree of site-specific information, generally producing more accurate results. Data requirements are higher (i.e., site-specific meteorology, terrain, chemistry data), and application of more refined models may require expert judgment in developing model inputs and setting model options. Some models can be used both as a screening model and refined model if additional site-specific information is used in the application.

The selection of a model for a specific application depends on a number of factors, including:

- The nature of the pollutant (i.e., gaseous, particulate, reactive, inert)
- The meteorological and topographic complexities of the area of concern
- The complexity of the distribution of sources
- The spatial scale and temporal resolution required for the analysis
- The level of detail and accuracy desired for the study and the amount of uncertainty that the analyst/risk manager is willing to accept
- The technical expertise of user

For example, steady-state models are not considered appropriate for downwind distances beyond a 50 km range, primarily because the steady-state wind speed and direction over that distance become unrealistic over the typical one-hour simulation period. This is especially true where complex terrain or meteorology is present.

Because screening models are applied with fewer resources and data to provide conservative estimates of concentrations, screening models are often applied prior to any refined modeling in order to narrow the set of sources or air toxics to be modeled. Such an iterative approach is generally recommended by EPA, where screening results are used to generate a subset of potentially higher-risk sources or chemicals for more refined assessment. General guidance on screening-level modeling has been published by EPA. Additional guidance on air modeling is incorporated into EPA’s Guideline on Air Quality Models.

Risk assessors generally work out the development of a modeling protocol to be used in the assessment during the planning/scoping and problem formulation phase of the assessment. Providing this protocol will help establish the modeling approach for not
only review and comment by interested parties up front, but will help to establish technical credibility and provide for consensus building among all interested parties.

5.5.3 Data Required for Modeling: Meteorology, terrain, and emissions data are processed and used as primary input data for air quality models. Depending on the level of refinement of the model, the required input data for an air quality model will include (but not necessarily be limited to) the following parameters:

- **Emission rate**: the rate at which emissions are released into the atmosphere.

- **Physical/chemical characteristics of emissions**: For some models, the phase of emission must be specified (i.e., gas, particulate, or semi-volatile). For chemicals present as PM or as semi-volatile substances, particle size distribution and fraction of particle phase as a function of temperature, for each chemical, may be necessary inputs. In some cases, information may only be available on the basis of total volatile organic compounds or total particulates.

- **Type of release point**:
  - *Point sources* (modeling sense) are releases from stacks and isolated vents and typically have plume rise associated with the release.
  
  - *Area sources* (modeling sense) are sources which are usually low level or ground level releases with no plume rise (i.e., fugitive emissions from the summary of equipment leaks across a facility; uncontrolled emissions that escape from the windows along a building wall; releases of dust from a road or work site; slag dumps; storage ponds). If a large number of sources are to be modeled, a common approach is to spread these sources uniformly across the modeling domain if no appropriate spatial surrogate is available. Alternatively, these sources may be allocated based on spatial surrogates. Typical examples include census tract population and commercial, residential and industrial land-uses.

  - *Volume sources* are releases that are modeled as emanating from a 3-dimensional volume (such as a box). Examples include releases from conveyor belts or the collective releases from the gas pumps at service stations. Volume sources differ from area sources in that they have a vertical dimension to their release. Like area sources, they do not have plume rise.
- **Line sources** are releases that are modeled as emanating from a two-dimensional area. Examples include rail lines and roadway segments. Line sources differ from area sources in that they have aspect ratios (length to width) much higher than 10:1. Like area sources, they do not have plume rise.

- **Specialized release types** include multiple parallel release lines that result in increased buoyant dispersion (i.e., coke ovens, aluminum smelters); dense gas release; and exothermic gas release, jet-plume release and horizontal venting that may be defined and modeled using special techniques or models depending on the characteristics of the emission source.

- **Release point parameters**: The user may need to specify the physical characteristics of the release point. Key parameters may include release height above ground level (i.e., stack height, average height of fugitive emissions), area of the release point (for point sources, stack diameter; for area sources, length and width of the area across which releases occur), other stack parameters of the release stream for point sources that can alter the effective release height (includes temperature, stack orientation, and exit velocity), and facility/building dimensions (downwash).

- **Information on the surrounding land-use and terrain heights**: For dispersion models, classification of the surrounding area as urban or rural is usually required (this classification can affect the rate of dispersion). In addition, more refined modeling that takes into account complex terrain (i.e., ground surfaces higher than release height elevation) will require terrain elevation data.

- **Chemical-specific data**: If transformation removal is being modeled, rates of transformation or removal for the chemicals being modeled are required.

- **Boundary or background concentrations**: Ideally, emissions from modeled source(s) are responsible for the modeled concentrations. However, background concentrations, or boundary conditions in the case of grid models, may be important contributors to the total concentrations. This is particularly relevant where modeled concentrations are compared to observed concentrations.

**5.5.4 Sources and Examples of Air Quality Models**: Dispersion models are used to support a variety of air toxics regulations. Several models, user's guides, and guidance documents are available for these purposes from EPA’s [Support Center for Regulatory Air Models (SCRAM) website](http://www.epa.gov/ttn/scram/).
A variety of models are available for air toxics risk assessments, with some models having been designed for specific air toxics application. The SCRAM website provides detailed information regarding individual models, including software/code for each model, user’s manuals, and other support documentation.

The extent to which a specific air dispersion model is suitable for the evaluation of air toxic source impacts depends upon several factors, such as the nature of the pollutant (e.g., gaseous, particulate, reactive, inert), the meteorological and topographic complexities of the area, the complexity of the source distribution, the spatial scale and resolution required for the analysis, and the level of detail and accuracy required for the analysis. For example, steady-state Gaussian plume models are not considered appropriate for downwind distances outside of the 0.1 km to 50 km range. Because of the assumption in Gaussian models of a steady wind speed and direction over the entire modeling domain for each hour, a > 50 km distance may be inappropriately long in many areas, especially where complex terrain or meteorology is present. In such cases, a non-steady state model would be more appropriate.

Finer scale models, such as CAL3QHC and CALINE4, are most typically applied to exposure studies from mobile sources. The UAM-TOX and CMAQ models are examples of models which can simulate photo-chemically active air toxic species, including secondary formation of pollutants like formaldehyde. Because the complex secondary formation processes are nonlinear and can occur at locations distant from the emission source, these models are designed to be applied to an exhaustive set of sources over a large region, rather than to individual facilities or small groups of facilities. The models more typically applied to single or multiple facilities include SCREEN3, ISCST3, ISCLT3, AERMOD, ASPEN, CALPUFF, and UAM-TOX. Brief descriptions of these models are provided below.

**SCREEN3**

- Screening-level Gaussian dispersion model that estimates an hourly maximum ambient concentration based on an average, constant emission rate (concentration results can be scaled up to annual average using simple conversion factors as specified in EPA guidance); results are not direction-specific (i.e., wind direction is not taken into account).

- Data requirements are relatively low; uses site-specific facility data (i.e., stack height, diameter, flow rate, downwash); does not use site-specific meteorology data.
• Data processing requirements are low; easy to use for quick assessment of a single facility.

• Model does not estimate deposition rates.

**Industrial Source Complex - Short Term (ISCST3)**

• Gaussian dispersion model (more advanced than SCREEN3); estimates average annual ambient concentration by modeling hourly emissions, and meteorology includes removal effects for wet and dry deposition flux for any locations specified by the user.

• Data requirements are higher than for SCREEN3; requires hourly, site-specific, processed meteorological data, physical characteristics of emissions, and terrain information. Model can accommodate variable emission rates.

• More expertise is required to use model (compared to SCREEN3); user should possess specific technical and computer skills.

**Industrial Source Complex - Long Term (ISCLT3)**

• Similar to ISCST3, but uses seasonal frequency distribution of meteorological inputs rather than hourly data; runs more rapidly than ISCST3, but can only produce concentrations averaged over a relatively long period of time; not considered as accurate as ISCST3.

• Unlike ISCST3, it cannot simulate wet deposition or complex terrain (terrain higher than the stack height).

**AMS/EPA Regulatory Model (AERMOD)**

• Replacement model for ISCST3 using new or improved algorithms on the parameterization of the earth’s boundary layer turbulence and state-of-the-science dispersion modeling; deposition algorithms should be available soon.

• Like ISCST3, is a Gaussian formulated model.

• Similar to ISCST3, but includes dispersion algorithm for both convective and stable boundary layers and allows plume penetration into elevated inversions.

• Incorporates new algorithms for building downwash.
• Unlike ISCST3, it simulates vertical profiles for wind, turbulence, and temperature.

• No wet or dry deposition (although planned future improvement).

• Requires surface characteristics as inputs (i.e., surface roughness), which allow user to differentiate between different types of terrain.

ASPEN

• A Gaussian dispersion model used to estimate toxic air pollutant concentrations over a large scale domain from regional to continental scale. (This is the model used for NATA risk characterization analyses.)

• Employs a dispersion algorithm similar to ISCLT3.

• However, unlike ISCLT3, it includes treatment of wet deposition for particles, and more detailed treatment of chemical transformation than ISCLT3 or ISCST3, although less detailed than UAM-Tox.

• In contrast to ISCLT3, ASPEN can utilize meteorological information from several locations, and includes a simplified treatment of secondary formation of gaseous air toxics.

CALPUFF

• A Gaussian puff model designed for long-range transport (> 50km) assessment, but may also be applied for near-source in situations with complex meteorology. A puff represents a continuous plume as a number of discrete packets of pollutant material.

• Has all the functional capabilities of ISCST3, but also includes capabilities for including 3-dimensional wind fields, vertical wind shear, and overwater effects.

• Not as extensively evaluated and tested as ISCST3 model.
• Requires a substantially higher level of air quality modeling expertise to use the model (compared to ISCST3).
UAM-TOX (Urban Air-shed Model - Toxics Version)

- A three-dimensional, grid-type model used to model pollutants in urban areas. Derived from the Urban Air-shed Model (UAM), and designed to calculate ozone concentrations under short-term, episodic conditions lasting three to four days resulting from emissions of oxides of nitrogen, volatile organic compounds, and carbon monoxide.

- Simulates the most photo-chemically active air toxics (i.e., acetaldehyde, 1,3-butadiene, and formaldehyde), as well as secondary formation of acetaldehyde and formaldehyde, tracking primary and secondary fractions separately.

- Requires a substantially higher level of air quality modeling expertise to use this model (compared to ISCST3).

Exhibit 5-7 identifies some common applications for these air quality models. Exhibit 5-8 provides an overview of the key physical processes simulated in the most widely used air quality models oriented toward assessment of risks from facilities.

**Exhibit 5-7 Typical Applications for Common Dispersion Models**

<table>
<thead>
<tr>
<th>Averaging Period</th>
<th>Terrain Type</th>
<th>Single Source</th>
<th>Multiple Sources</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Rural</td>
<td>Urban</td>
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<td>Screening Models</td>
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<tr>
<td>Short Term (1-24 hour average)</td>
<td>Simple</td>
<td>SCREEN3</td>
<td>SCREEN3</td>
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<tr>
<td></td>
<td>Complex</td>
<td>SCREEN3, ISCST3</td>
<td>SCREEN3, ISCST3</td>
</tr>
<tr>
<td>Long Term (Monthly-Annual)</td>
<td>Simple</td>
<td>ISCLT3</td>
<td>ISCLT3</td>
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<tr>
<td></td>
<td>Complex</td>
<td>ISCT3</td>
<td>ISCT3</td>
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<tr>
<td>Refined Models</td>
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<tr>
<td>Short Term (1-24 hour average)</td>
<td>Simple</td>
<td>ISCST3, AERMOD</td>
<td>ISCST3, AERMOD</td>
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<tr>
<td></td>
<td>Complex</td>
<td>AERMOD, CALPUFF</td>
<td>AERMOD, CALPUFF</td>
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<tr>
<td>Long Term (Monthly-Annual)</td>
<td>Simple</td>
<td>ISCST3, AERMOD</td>
<td>ISCST3, AERMOD</td>
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<tr>
<td></td>
<td>Complex</td>
<td>CALPUFF, AERMOD</td>
<td>CALPUFF, AERMOD</td>
</tr>
</tbody>
</table>
5.5.5 Model or Monitor? Ultimately, the choice of whether to monitor ambient air concentrations of air toxics or model (or both) depends on the goals of the assessment, the exposure setting, other specific project circumstances (i.e., many communities want monitoring as part of a risk assessment), and the assessing entity. For example, to understand the exposure an actual individual receives as they move about their daily activities, personal monitoring is the best option because it reflects the pattern of this movement. However, such studies are rarely done outside of research settings. As another example, compliance with a permitted release rate may also require monitoring as the preferred method of measurement.

Air toxics risk assessments, however, tend to examine potential exposures to hazardous air toxics for a relatively large number of people over relatively large geographic areas (i.e., a neighborhood or urban area, county, or larger). In these instances, the risk managers and analysts must carefully use their planning and scoping activities to develop
the questions they want to answer and to identify the types of data they will need to answer those questions. For some questions and data needs, monitoring is the preferred tool. For others, modeling is better. In general, most air toxics risk assessments will benefit from some combination of both modeling and monitoring to provide the depth and breadth of information that will be necessary to answer the assessment questions (see Exhibit 5-9).

Exhibit 5-9: Hypothetical Example of a Combined Modeling and Monitoring Program

This figure illustrates a hypothetical set of isopleths for annual average air concentrations that a dispersion model predicted, assuming a single source (black dot) near the center of the geographic region. Note that the model predicts the point of maximal exposure to be somewhere within the area bounded by grid points 2, 4, S1, and S3, based on the existing information on release rate, wind direction, and effective release height. In this hypothetical example, a monitoring station was used to measure ambient concentrations as a means of evaluating the model predictions. Note that the monitoring location is not in the area of estimated highest concentration and, therefore, might not provide a better estimate of maximum exposure.

Most air toxics risk assessments that evaluate exposures to populations receiving impacts from one or more sources should consider using modeling as their primary tool to evaluate and characterize exposures and risks. In certain instances, assessors may use monitoring as the primary tool to evaluate exposure concentrations for potentially exposed populations. The utility of modeling for neighborhood and larger scale analyses is that it provides a better picture of the variation of exposure conditions over the assessment area domain (i.e., modeling provides spatial resolution) and allows a more
straightforward approach to source allocation (i.e., what portion of the risk is caused by each of the modeled sources). Therefore, modeling is the most common approach for estimating exposure concentrations in air toxics risk assessment. Monitoring is often used as a secondary tool to provide input data to the models and validate model results.
References


Chapter 6: Exposure Assessment of Air Toxics

6.1 Introduction

An exposure assessment has four major components: emission characterization (description of the source and a quantification of the rate of emissions of an air toxic from the source), environmental fate and transport (how the released air toxics is transported, dispersed, and transformed from the source to the exposed receptor population), characterization of the study population (i.e., location, behavior, age and other characteristics of the study population) and exposure characterization (the spatial integration of the air toxics concentration with the study population to characterize exposure). The exposure assessment box in Exhibit 6-1 outlines these four components and shows how the exposure assessment procedure fits into the detailed risk assessment process.
Chapter 5 discussed the first two components of an exposure assessment: emission characterization and environmental fate and transport. This chapter discusses the last two components: characterization of the study population and exposure characterization. An exposure assessment concludes with a quantification of air toxic concentrations at a point external to the receptor population (or at a target organ of the receptor), and a statement of the uncertainty associated with the quantification.

This chapter also discusses an overview of the exposure assessment process. Exposure assessment is generally the most multifaceted and time-consuming part of an air toxics risk assessment. EPA has developed general guidance for exposure assessment in its 1992 “Guidelines for Exposure Assessment.” This guidance document is the key reference document for the exposure assessment portion of the risk assessment. Exposure assessment helps identify and evaluate a population receiving exposure to a toxic agent, and describe its composition and size, as well as the type, magnitude, frequency, route and duration of exposure. In other words, an exposure assessment is that part of the risk assessment that identifies:

- Who is potentially exposed to toxic chemicals
- What toxics they may be exposed to and
- How they may be exposed to those chemicals (amount, pattern, and route)

### 6.2 Components of an Exposure Assessment

The nature and complexity of the components within the exposure assessment are often functions of the particular risk management question (or other purpose) to be addressed. Simple screening analyses that rely on conservative default assumptions may be sufficient to rule out the need for further analyses or action. On the other hand, a more detailed exposure analysis may be needed to determine the necessity for emission controls, particularly when the application of those controls is associated with large economic consequences. Indeed, the exposure assessment raises and addresses many of the risk assessment’s difficult and critical policy questions. As illustrated in Exhibit 6-1, the exposure assessment includes the following steps:

- **Characterization of the exposure setting**, including the physical environment, the scale of the study area, important sources and chemicals, and potentially exposed populations and population characteristics (i.e., demographics). Most of this information is collected and organized during the problem formulation portion of the risk assessment.
• **Identification of exposure pathways**, including sources and mechanism of release, exposure points and routes of exposure, and transport media. Again, most of this information is collected and organized during problem formulation.

• **Quantification of exposure**, including an *evaluation of uncertainty* and *preparation of documentation*. Quantification of exposure includes three general steps: characterization of emissions (discussed in Chapters 3 and 5), evaluation of chemical fate and transport (discussed in Chapter 5), and estimation of exposure concentrations (EC) (discussed in this chapter along with exposure modeling, evaluation of uncertainty, and preparation of documentation).

### 6.3 Exposure, Route of Exposure, and Exposure Pathway

#### 6.3.1 Exposure

Exposure assessment is the overall process of evaluating who receives exposure to toxic chemicals, what those chemicals are, and how the exposure occurs. In “*Guidelines for Exposure Assessment,*” EPA established a specific definition of *human exposure*. It is defined as contact with a chemical at the visible external boundary of a person, including skin and openings into the body such as mouth, punctures in the skin, and nostrils. This definition of exposure does not describe the contact of a chemical with the actual exchange boundaries in the body where absorption into the bloodstream can take place, such as the linings of the lung or digestive tract. (One exception to this is chemical contact with skin or punctures in the skin; in this case, the location of the exposure and the exchange boundary are one in the same.) Other than dermal exposure, chemicals must be physically taken into the body by ingestion or inhalation (a process called *intake*) before they can contact an exchange boundary and be taken into the bloodstream (a process called *uptake*).

#### 6.3.2 Routes of Exposure

The term *route of exposure* is used to describe the different ways a chemical enters the body. The three main routes of exposure are *inhalation, ingestion*, and absorbing a chemical through the skin (*dermal*). For inhalation risk assessments, we are only concerned with the inhalation route of exposure. The dermal and ingestion routes of exposure are generally only relevant to chemicals that persist and which also may bio-accumulate (i.e., the persistent, bio-accumulative HAP [PB-HAP] compounds).
Some chemicals can cause harm in the part of the body where individuals take them in (i.e., in the respiratory system for inhaled chemicals or in the digestive tract for ingested chemicals). This is called a portal of entry effect because the adverse effect occurs at the place (i.e., the “portal”) where the chemical enters the body. Other chemicals have to be taken into and distributed by the circulatory system to cause a harmful effect at a point distant from their portal of entry into the body. Such effects are called systemic effects because they have the potential to act at points throughout the system. As a chemical moves through the body, it may be metabolized (possibly to a more toxic entity); stored in the body; and/or eliminated in urine, feces, sweat, nails/hair, or exhaled breath.

### 6.3.3 Exposure Pathway

Pathway analysis is a concept that is linked strongly to environmental fate and transport. The exposure pathway is the course that a toxic chemical takes from its source to the exposed receptor. An exposure pathway describes a unique mechanism by which an individual or population is exposed to air toxics at, or originating from, a source or group of sources. Once released from the sources, air toxics begin to disperse by the wind away from the point of release and may remain airborne; convert into a different substance; and/or deposit out of the air onto soils, water, or plants. People may be exposed to air toxics by breathing contaminated outdoor and/or indoor air (inhalation); ingestion (for the small number of air toxics that can accumulate in soils, sediments, and foods – a process called bioaccumulation); and skin (dermal) contact with deposited air toxics (Exhibit 6-2). Air toxics risk assessments always evaluate the inhalation exposure pathway. However, when sources release chemicals that persist and which also may bioaccumulate, analysis of non-inhalation pathways may also be necessary.
An exposure pathway consists of four elements:

- A source and mechanism of chemical release
- One or more environmental media (i.e., air, water, soil) in which the chemical is transported from the source
- A point of potential human contact with the contaminated medium (referred to as the exposure point)
- An exposure route (i.e., inhalation, ingestion) at the contact or exposure point

6.3.4 Multi-pathway Exposure

The multi-pathway exposure assessment covers a broader scope and may be more complex than the direct inhalation exposure pathway assessment. Exposure pathways to be evaluated include multiple media (soil, water, sediment, and biota) and exposure routes in addition to inhalation. The multi-pathway exposure assessment focuses on two general categories of ingestion pathways: incidental ingestion and food chain. Incidental ingestion pathways consider exposures that may occur from ingestion of soils or surface water while an individual is engaged in other activities (i.e., ingestion of soil while gardening or playing outside; ingestion of surface water while swimming). Food chain
Introduction to Air Toxics

pathways consider exposures that may occur if PB-HAP compounds accumulate in the food and water that people consume.

The focus of the multi-pathway assessment is on ingestion pathways. Other exposure pathways may be important for particular risk assessments, including dermal exposures (i.e., direct contact with contaminated soils, surface waters, or surface water sediments during outside activities such as gardening or swimming); re-suspension of dust (i.e., from wind blowing across contaminated soils, or agricultural activities such as tilling) and subsequent inhalation of the dust particles; and ingestion of contaminated groundwater. However, EPA does not have sufficient experience with multi-pathway air toxics risk assessments to identify the circumstances for which exposures via these additional pathways may represent a potential concern.

6.3.5 Ingestion – Chemical Intake Rate

In contrast to the inhalation assessment, in which the quantitative metric of exposure is the ambient air concentration at the exposure point, ingestion exposures are quantified using the chemical intake rate – the amount of chemical ingested per unit time – generally expressed in units of milligrams of chemical per kilogram of body weight per day. The fundamental equation for dietary intake and ingestion pathways in general is given as:

\[ I = \frac{EC \times CR}{BW} \times \frac{EF \times ED}{AT} \]

Where

- \( I \) = Chemical intake rate, expressed in units of mg/kg-day. For evaluating exposure to non-carcinogens, the intake is referred to as average daily dose (ADD); for evaluating exposure to carcinogenic compounds, the intake is referred to as lifetime average daily dose (LADD).

- \( EC \) = Exposure concentration of the chemical in the medium of concern for the time period being analyzed, expressed in units of mg/kg for soil and food or mg/L for surface water or beverages (including milk).

- \( CR \) = Consumption rate, the amount of contaminated medium consumed per unit of time, event, or other measure (i.e., kg/day for soil and food; L/day for water).

- \( EF \) = Exposure frequency (number of days exposed per year).
Introduction to Air Toxics

- \( ED = \) Exposure duration (number of years exposed).

- \( BW = \) Average body weight of the receptor over the exposure period (kg).

- \( AT = \) Averaging time, the period over which exposure is averaged (days). For carcinogens, the averaging time is usually 25,550 days, based on an assumed lifetime exposure of 70 years; for non-carcinogens, averaging time equals \( ED \) (years) multiplied by 365 days per year.

6.4 Estimating Inhalation Exposure Concentration

Unless persistent bio-accumulative hazardous air pollutants (PB-HAPs) are present in source emissions, most air toxics risk assessments will only estimate inhalation exposure concentrations. Limiting the exposure assessment this way is possible because the dose-response values that characterize inhalation risk (i.e., reference concentrations, inhalation cancer unit risk estimates – see Chapter 7) take into consideration the complex physical and pharmacokinetic processes that influence how the chemical reaches the target organ, which may be a region of the respiratory tract or a remote site. Specifically, other than exposure modeling to account for things like time in different microenvironments and microenvironment concentrations, no adjustment for other exposure parameters (i.e., body weight and inhalation rate) are warranted. For multi-pathway risk assessments, however, where ingestion intake rate is the exposure parameter, it will be necessary to consider parameters such as body weight and contact rate (i.e., amount of soil ingested, fish eaten) as shown in the above equation for ingestion chemical intake rate.

Assessors determine human exposure to an environmental pollutant via inhalation by estimating the concentration of that pollutant in the ambient air and the contact of an individual with that air (along with the characteristics of the contact). Because concentrations in the air vary over space and time, it is important to know where and how long people spend their time in relation to the contaminated air under study. Through air quality modeling and monitoring, the ambient concentrations of pollutants in air can be estimated geographically and temporally. Through the use of exposure modeling, estimates of exposure via the inhalation route can be adjusted from modeling data to take into account the demographics of people in the study area and the time they may spend in various microenvironments.

For the inhalation route of exposure, the metric of exposure is the concentration of the chemical in the air the population of interest is breathing over the period of interest. This concentration is called the exposure concentration (EC) and is the primary quantitative
output of the inhalation exposure assessment. This metric is intended to represent the time weighted average exposure(s) to the population(s) of interest during the exposure period. (Note: Exposure models are often also applied to better reflect how different people interact with contaminated air. In other words, the air quality model evaluates how chemicals move and change in the environment. The *exposure model* evaluates how different types of people interact with the resulting contaminated air - with the result that the EC is refined to provide more realistic estimates of exposure. A discussion of exposure modeling is provided below.)

### 6.4.1 Two General Approaches to Derive Exposure Concentrations

There are two general ways to derive the EC for a given risk assessment: *General Air Quality Assessment* and *Exposure Modeling* (Exhibit 6-3). Both may incorporate the results of air quality modeling and/or monitoring efforts.

#### Exhibit 6-3: Two General Ways to Estimate Inhalation Exposure Concentrations

- **General Air Quality Assessment (Ambient Air Concentrations as a Surrogate):** For *screening-level evaluations*, assessors use the concentrations of air toxics generated at each modeling node or the concentrations determined by a monitor (if modeling is not performed) as surrogates of the inhalation exposure concentrations for the populations in the study locations. In such a screening level assessment, the default assumption is that the populations of interest are breathing outdoor air continuously at the modeled or monitor location. This is

- **Exposure Modeling:***
believed to be a conservative assumption since indoor air concentrations of air toxics are expected to be the same or lower than the outdoor concentrations (when the indoor concentrations are produced solely by inflow from outside air).

- **Exposure Modeling (Assessment Using Microenvironment Concept):** More comprehensive inhalation exposure assessments combine estimates of ambient pollutant concentrations (i.e., from air quality models) with information about the population of interest, including the types of people present (i.e., ethnicity, age, sex), time spent in different microenvironments, and microenvironment concentrations. The assessment objective is to identify a representative estimate of the pollutant concentration in the inhaled air in each microenvironment and combine it with an estimate of the time spent in different microenvironments (and the activities within these microenvironments) throughout the daily routine of different groups of people with similar attributes (called cohorts).

### 6.4.2 Types of Exposure Time Frames

People living in the vicinity of one or multiple air toxics sources have the potential to receive exposure to emitted chemicals many different ways. For example, they might be exposed occasionally, but to very high concentrations (i.e., when an accident occurs that releases large amounts of chemicals to the air in a very short amount of time). On the other hand, they might receive exposure quite often (or even continuously) to low levels that would likely go unnoticed. Air toxics inhalation exposure assessments usually focus on two of these different types of possible exposure scenarios:

- **Chronic exposure** refers to situations in which the exposure occurs repeatedly over a long period of time (usually years to lifetime). If there is substantial variation in exposure concentration during segments of the chronic period, it may be appropriate to evaluate the segments separately using the appropriate dose-response values.

- **Sub-chronic exposure** refers to situations in which the exposure occurs repeatedly over a period of time that ranges between acute and chronic exposures. (As toxicity values are less widely available for this duration, it is less routinely assessed than the others. For air toxics assessments, this exposure period is not commonly assessed.)

- **Acute exposure** refers to situations in which the exposure occurs over a short period of time (usually minutes, hours, or a day) and usually at relatively high
concentrations. The averaging times commonly used to represent acute exposures concentrations (i.e., acute ECs) are a 24-hour average, a one-hour average, or a 15-minute average.

The EC values the assessor develops to represent acute and chronic exposures should match the assumptions built into the dose-response values that the assessor uses to characterize risk. For example, it would be inappropriate to compare a one-week average exposure concentration to a one-hour acute dose-response value. For chronic exposures, the scale of time-weighted averaging performed to develop the exposure estimate should be generally similar to that used in developing the dose-response value.

6.4.3 Common Ways to Estimate Exposure Concentrations

Risk assessors commonly use several different ways to estimate exposure concentrations. Some ways are used primarily for screening-level (Tier 1) assessments; others are used primarily for more refined assessments. Exhibit 6-4 illustrates several different ways to estimate exposure concentrations when ambient air concentrations are used as surrogates.

- **Monitoring locations**: Sites where air monitors are located provide a direct measure of ambient air concentrations at those locations. However, these locations may or may not be representative of ambient air concentrations in other parts of the study area. If monitors are not located where people live, the monitoring results may not be of much value for the risk assessment other than to check the accuracy of modeling. Monitoring results may be used as inputs to exposure modeling.

- **Point of maximum modeled concentration**: This is the modeling node where the maximum modeled ambient air concentration occurs, regardless of whether there is a person there or not. This generally provides a conservative estimate of exposure and could be used as the EC in a screening-level evaluation. This point can be used to provide an estimate of “high-end” exposure to the risk manager because, although no one may actually be living there at the present, someone might move there in the future. This point may be referred to as the point of the “maximum exposed individual (MEI).”

- **Point of maximum modeled concentration at an actual receptor location**: This is the modeling node where the maximum ambient air concentration occurs to an actual person in the area of impact, usually at an actual residence. To identify this point precisely, it is necessary to know detailed information about the location of
actual people in the study area. As with the point of maximum modeled concentration above, this point can be used to provide an estimate of “high-end” exposure to the risk manager (in this case, based on current actual exposures). This point may be referred to as the point of the “maximum individual risk (MIR).”

Exhibit 6-4: Map A

Exhibit 6-4: Map B
In this hypothetical example, the risk assessors have modeled a release of a volatile organic HAP from a facility using a computerized air quality model, and the ambient air concentration is used as a surrogate for the Exposure Concentration (EC).

Knowing only the information displayed in the first version of the map (A), it is difficult to say much about exposure since we do not know where the people are in relation to the facility or the area of impact. To remedy this, our next step is to obtain demographic data (usually from the Census Bureau) and overlay it on the above map. Performing this analysis and redrawing the map gives map (B).

In the second version of the map (B), we have included the census tract boundaries (dotted lines) and we also know from study area reconnaissance that there is an uninhabited national forest to the west of the facility, a farmer directly to the north, and a small town in the northeast. (Note that the town, Smallville, actually can be further subdivided into smaller census blocks; however, they are not shown here to keep the picture simple.) Now that we have a better idea of where people are in relation to the facility (and the area of impact caused by the VOC release), we are in a better position to start making some statements about how people are exposed.

- **Census tract/block internal point**: The U.S. Census Bureau provides information about populations in geographic units called census tracts, which are subdivided into block groups/enumeration districts and blocks. In cases where there is only limited information about the census tract (e.g., nothing is known other than the number of people living within the tract), the Census Bureau’s “internal point” (sometimes referred to as a centroid) for the tract typically is used as the point of exposure for all the population in the tract. The Census Bureau provides an internal point for each of its geographic subdivisions (i.e., tracts, blocks, and block groups).

- **Population-based approaches**: Exposures may be evaluated by tracking individual members of a population and their inhalation through time and space. Such analyses may incorporate a user-specified number of simulated individuals or population groups (cohorts) to represent the population in the study area. A cohort is defined here as a group of people within a population with the same demographic variables who are assumed to have similar exposures. In this approach, the exposure analysis process consists of relating chemical concentrations in air (outdoor and/or indoor) and tracking the movement of a population cohort through locations where chemical exposure can occur according to a specific activity pattern. Population-based analysis is generally accomplished using exposure models.

- **Personal monitoring**: Exposures may be estimated directly by placing monitors on individuals, which allows collection of more detailed information specific to
the exposure pattern for that individual. Such monitors are referred to as *personal monitors* because they provide information on exposure to that individual, rather than to the general area in which an individual might be moving.

### 6.5 Exposure Modeling

Exposure modeling uses the ambient air concentration estimates along with information about the population of interest and information on how the pollutant concentration can vary in different microenvironments to derive estimates of exposure concentration over the period of exposure. Information on human exposure modeling for air toxics can be found on EPA’s Fate, Exposure, and Risk Assessment (FERA) website: [http://www.epa.gov/ttn/fera/](http://www.epa.gov/ttn/fera/).

Air quality modeling estimates how contaminated the air is in the different locations within a study area. Exposure modeling simulates how different types of people interact differently with that contaminated air to derive integrated (i.e., time weighted) estimates of their exposure for the duration of interest. The estimation of population exposure is a very difficult task because it requires information on the activity patterns of the population as well as information on the air toxics concentrations (indoor and outdoor) to which that population is exposed. Although several databases have been developed to characterize activity patterns, various sources of variability (i.e., among individuals and geographical regions) introduce uncertainty.

This section focuses on exposure models to evaluate inhalation exposures. Exposure models are also available for other routes of exposure as well (i.e., a model may be employed to track patterns of food and drinking water consumption across a population). These indirect pathway exposure models are not discussed in the Manual, but information on these models can be found in EPA’s *Air Toxic Risk Assessment Reference Library*.

#### 6.5.1 Inhalation Exposure Modeling

Inhalation exposure is characterized by the pollutant concentration in the air (i.e., the exposure concentration) reaching an individual’s nostrils and/or mouth (in units of $\mu g/m^3$). Estimates of air concentrations from modeling or monitoring can be used in inhalation exposure modeling. When derived from monitoring measurements, exposure concentrations are an aggregate of the contributions from all emissions sources impacting the monitor. When derived from modeling studies, the estimated exposure concentrations reflect only the sources that were included in the modeling exercise.
Because air pollutant concentrations vary over time and space, inhalation exposure models combine information on human activity patterns and micro-environmental concentrations to estimate exposure concentrations. *Activity patterns* are defined by an individual’s or cohort’s allocation of time spent in different activities in various microenvironments and various geographic locations. A *microenvironment* is a defined space that can be treated as a well-characterized, relatively homogeneous location with respect to pollutant concentration for a specified time period (i.e., rooms in homes, restaurants, schools, offices; inside vehicles; outdoors).

A common exposure model for inhalation that combines information on microenvironment concentrations and activity patterns calculates a *time-weighted average of all exposures* from the different microenvironments in which a person spends time during the period of interest.

\[
EC_A = \frac{1}{T} \left( \sum_j C_j \times t_j \right)
\]

Where:

- \( EC_A \) = the adjusted average inhalation exposure concentration (\( \mu g/m^3 \)),
- \( T \) = total averaging time (\( T = 3 t_j \); years),
- \( C_j \) = the average concentration for microenvironment \( j \) (\( \mu g/m^3 \)), and
- \( t_j \) = time spent in the microenvironment \( j \) (years).

Note that the two critical parameters that need to be evaluated in this equation are the concentration of a chemical in a microenvironment and the amount of time spent in that microenvironment. Exhibit 6-5 presents a simple example.
Exhibit 6-5: i.e., How to Estimate Exposure Concentrations (EC) for Exposure Modeling

The following exposure profile has been developed for one year (which represents, for example, the 30 years of “work”) for a representative individual within the population of interest:

<table>
<thead>
<tr>
<th>Duration Spent in Each Microenvironment (% year)</th>
<th>Average Concentration of Pollutant A In Each Microenvironment ($\mu g/m^3$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>10 = outside</td>
<td>80</td>
</tr>
<tr>
<td>50 = at work</td>
<td>20</td>
</tr>
<tr>
<td>40 = inside home</td>
<td>10</td>
</tr>
</tbody>
</table>

The EC for that individual is calculated as:

$$EC = (0.1 \times 80) + (0.5 \times 20) + (0.4 \times 10) = 22 \mu g/m^3$$

6.5.2 Sources of Data for Human Activity for Exposure Assessments

Numerous EPA and related databases provide information useful for conducting exposure assessments, including information on activity pattern and demographic information useful for inhalation exposure modeling. Types of information included are human activity surveys, standard values for physiological processes and consumption of food and water, measured exposure data, health status surveys and measurements, nutrition surveys, and data on the spatial distribution of populations.

This section provides several of the more notable information sources, some of which are important for inhalation exposure modeling, and some of which are important for modeling exposures through pathways other than inhalation (i.e., ingestion of contaminated fish, soil, and groundwater).

- **EPA Consolidated Human Activity Database (CHAD):** CHAD contains data obtained from human activity studies that were performed at city, state, and national levels. CHAD is intended to provide input data for exposure/intake dose modeling and/or statistical analysis. CHAD is a master database providing access to other human activity databases using a consistent format. Demographic fields include personal characteristics (age, gender, ethnicity, and weight), social characteristics (education, occupation, and income), residential location (state, county, zipcode) and housing characteristics (heating fuel, cooking fuel).

- **EPA Exposure Factors Handbook:** The Exposure Factors Handbook provides a statistical summary of the available data on various parameters and variables used...
in assessing human exposure. This Handbook is used by risk assessors who need to obtain data on standard factors to calculate human exposure to toxic chemicals. These factors include human activity factors and residential characteristics. Recommended values are for the general population and also for various segments of the population who may have characteristics different from the general population. Included are full discussions of the issues that assessors may want to consider in deciding how to use these data and exposure parameter recommendations.

- **EPA Human Exposure Database System (HEDS):** HEDS is a web-enabled data repository for human exposure studies. Its mission is to provide data sets, documents, and metadata for human exposure studies that can be easily accessed and understood by a diverse set of users. HEDS provides only data and accompanying documentation from research studies; it does not provide interpretations. It allows a user to download documents for review or data sets for analysis on their own computer system. Currently contained in HEDS are various components of the National Human Exposure Assessment Survey (NHEXAS).

- **National Human Exposure Assessment Survey (NHEXAS):** The National Human Exposure Assessment Survey was developed by US EPA’s Office of Research and Development (ORD) in the 1990’s to provide information about multimedia and multi-pathway population exposure to chemicals of various types. Phase I consists of demonstration/scoping studies using probability-based sampling designs. Volunteer participants were randomly selected from several areas of the U.S. These studies included personal exposure, residential concentrations, and biomarker measurements. The Arizona study measured metals, pesticides, and VOCs. The Maryland study measured metals, pesticides, and polycyclic aromatic hydrocarbons (PAHs). The Region 5 study, conducted in Ohio, Michigan, Illinois, Indiana, Wisconsin, and Minnesota, measured metals and VOCs. Researchers worked with the participants to measure the level of chemicals in the air they breathed, in the foods and beverages they consumed (including drinking water), in the soil and dust around their homes, and in their blood and urine. Participants completed questionnaires to help identify possible sources of chemical exposure.

- **CDC National Health and Nutrition Examination Survey (NHANES):** NHANES is a survey conducted by the National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention. This survey has been designed to collect information about the health and diet of people in the United
States. NHANES is unique in that it combines a home interview with health tests that are done in a Mobile Examination Center. The current NHANES is eighth in a series of national examination studies conducted since 1960. The results of these surveys are compiled in databases and summarized in a variety of tables and reports.

- **U.S. Census Data:** The U.S. Census provides data on the spatial distribution of population and population subgroups at several geographic levels: national, state, county, tract, block group and block. Examples of useful spatially-resolved data for exposure assessment include: population by age, gender, and ethnic group; house heating fuel use; estimated travel time to work by various modes of transportation; and levels of employment in various industries.

- **LandScan USA:** LandScan is a high resolution population distribution database for the continental U.S. LandScan uses satellite imagery in population distribution modeling to produce population distribution data at a much finer resolution than previously available. LandScan 1998 and 2000 have a grid cell size of 30 seconds (<1 kilometer) and use census data in combination with many other geospatial data, such as land use/cover, topography, slope, roads, and nighttime lights, in order to improve the estimation and prediction of the spatial distribution of residential populations. LandScan updates use a much smaller grid cell size of 3 seconds (<100 meters). LandScan will be very useful for exposure modeling, environmental justice studies, and other types of risk assessments.

### 6.5.3 Examples of Inhalation Exposure Models

Some of the important characteristics that vary among the models include:

- **Ambient concentrations**
  - Modeling or monitoring estimates
  - Time scales (i.e., averaging time)

- **Exposure concentration time scale**
  - Time increment for calculations (i.e., by minute, hourly, seasonally, annually)
  - Averaging time for reporting (i.e., hourly, annually)
Introduction to Air Toxics

- Spatial scale
  - Geographic resolution of predictions (i.e., Census tracts, Census blocks, grids)
  - Potential size of modeling domain (i.e., neighborhood, county, nation)

- Population activity data
  - Type (i.e., time in microenvironments, commuting locations, food and water ingestion rates)
  - Temporal resolution (i.e., by minute, hourly, seasonally, annually)
  - Area specific resolution (i.e., national or regional)
  - Demographic resolution (i.e., by age, gender, or ethnic group)

- Framework
  - Deterministic: inputs and outputs are characterized as point estimates
  - Stochastic or probabilistic: inputs and outputs are characterized as distributions representing variability and/or uncertainty; Monte Carlo techniques are used to randomly select input values from the distributions for repeated simulations.

The following provides brief descriptions of some of the recently developed inhalation exposure models. The features of each model described are summarized in Exhibit 6-6.

<table>
<thead>
<tr>
<th>Model</th>
<th>Population Activity Data</th>
<th>Source of Ambient Concentrations</th>
<th>Spatial Resolution</th>
<th>Framework</th>
</tr>
</thead>
<tbody>
<tr>
<td>HEM-3</td>
<td>None (screening model)</td>
<td>ISCST3</td>
<td>Census blocks (additional points can be specified)</td>
<td>Deterministic</td>
</tr>
<tr>
<td>HAPEM</td>
<td>Micro-environment time/sequence, Commuting</td>
<td>External model or monitoring data</td>
<td>Census tract</td>
<td>Stochastic</td>
</tr>
<tr>
<td>TRIM.Expo (a.k.a. APEX)</td>
<td>Micro-environment time/sequence, Commuting</td>
<td>External model or monitoring data</td>
<td>Depends on resolution of air quality and demographic inputs</td>
<td>Stochastic</td>
</tr>
<tr>
<td>CPIEM</td>
<td>Micro-environment time/sequence, Commuting</td>
<td>External model or monitoring data</td>
<td>User-specified for the selection of activity patterns (i.e., state, region)</td>
<td>Stochastic</td>
</tr>
</tbody>
</table>
**Human Exposure Model (HEM):** The Human Exposure Model (HEM) is used primarily for performing risk assessments for major point sources (usually producers or large users of specified chemicals) of air toxics. The HEM only addresses the inhalation pathway of exposure, and is designed to predict risks associated with emitted chemicals in the ambient air (i.e., in the vicinity of an emitting facility but beyond the facility's property boundary). The HEM provides ambient air concentrations, as surrogates for lifetime exposure, for use with unit risk estimates and inhalation reference concentrations to produce estimates of cancer risk and non-cancer hazard, respectively, for the air toxics modeled.

The HEM contains (1) an atmospheric dispersion model, the Industrial Source Complex Model, with included meteorological data, and (2) U.S. Bureau of Census population data at the Census block level. The model utilizes 2000 Census data. The model estimates the magnitude and distribution of ambient air concentrations in the vicinity of each source. The model is generally used to estimate these concentrations within a radial distance of 50 kilometers (30.8 miles) from the source.

The HEM is available in two versions: HEM-Screen and HEM-3.

- **HEM-Screen** can generate chronic cancer risk and hazard estimates for multiple facilities nationwide in one run. This model uses a simplified version of the Industrial Source Complex Model (dispersion model). Several simplifications and assumptions are built in to HEM-Screen, and user-supplied data requirements are relatively low. For these reasons, HEM-Screen may be more appropriate for lower-tier or screening-level assessments involving a large number of facilities.

- **HEM-3** generates chronic cancer risk and chronic and acute hazard estimates for one facility at a time. This model uses the Industrial Source Complex Model (short-term), Version 3 (ISCST3), or the AERMOD dispersion model. Data requirements are somewhat higher for HEM-3 compared to HEM-Screen; however, the results are typically more refined because ISCST3 provides several additional dispersion modeling options.

**The Hazardous Air Pollutant Exposure Model (HAPEM5):** The latest version of EPA’s Hazardous Air Pollutant Exposure Model (HAPEM5) is a stochastic screening-level inhalation exposure model appropriate for assessing average long-term (annual) exposures of the general population, or a specific sub-population, over spatial scales ranging from urban to national ([http://www.epa.gov/ttn/fera/human_hapem.html](http://www.epa.gov/ttn/fera/human_hapem.html)). This application requires a moderate level of computer modeling skills.
HAPEM5 uses the general approach of tracking representatives of specified demographic groups as they move among 37 indoor, in-vehicle, and outdoor microenvironments and among geographic locations. The estimated pollutant concentrations in each microenvironment visited are combined into a time-weighted average concentration, which is assigned to members of the demographic group (the cohorts). HAPEM5 uses five primary sources of information: population data from the U.S. Census, population activity data from CHAD, commuting data developed by the Bureau of the Census, user supplied air quality data either from measurements or an air dispersion model, and micro-environmental factors data.

The previous version of HAPEM5 (namely, HAPEM4), was used in the NATA national scale assessment of the 1996 NEI to develop estimates of risk, by census tract, for each of the 33 HAPs (http://www.epa.gov/ttn/amtic/netamap.html). Specifically, HAPEM4 was used to predict population exposure for each of 10 demographic groups in each tract.

**Total Risk Integrated Methodology Exposure Event Model (TRIM.ExpoInhalation), also known as Air Pollutants Exposure Model (APEX):** The Air Pollutants Exposure Model (APEX) comprises the inhalation portion of the TRIM exposure module, TRIM.Expo (http://www.epa.gov/ttn/fera/human_apex.html). TRIM.Expo (a.k.a. APEX) uses a personal profile approach rather than a cohort simulation approach. That is, individuals are selected for simulation by selecting combinations of demographic characteristics and finding an activity pattern to match it, rather than directly selecting an activity pattern. If the selection probabilities for the demographic characteristics are the same as within the population to be simulated, this approach will provide a representative sample of that population’s activity patterns without the need for post-simulation weighting of results.

The current version (APEX3, available on the web) includes a number of useful features including automatic site selection from large (i.e., national) databases, a series of new output tables providing summary statistics, and a thoroughly reorganized method of describing microenvironments and their parameters.

**California Population Indoor Exposure Model (CPIEM):** The CPIEM is a stochastic inhalation exposure model developed for the California Air Resources Board’s (ARB’s) Indoor Program to evaluate indoor exposures for the general California population as well as certain sub-populations. CPIEM combines indoor air concentration distributions with Californians’ location and activity information to produce exposure and dose distributions for different types of indoor environments.
The temporal resolution and averaging time are user-selected from the options of 1-hour, 8-hour, 12-hour, and 24-hour. The spatial resolution and modeling domain similarly are specified by the user according to county, state region, or the entire state. Although outdoor concentrations may be included in the application, the focus is on indoor exposures and indoor emission sources.

6.6 Personal Monitoring

An alternative to “roof top” monitors is to place monitors directly on individuals, which allows collection of more detailed information specific to the exposure pattern for that individual. Such monitors are referred to as personal monitors because they provide information on exposure to that individual, rather than to the general area in which an individual might be moving. An advantage is that personal monitors reflect the time-varying concentrations (unless they are integrating monitors) an individual experiences as he or she moves about through various activities. Personal monitors have seen increasing use in recent years due to two factors: they are more readily available, reliable, and cheaper than in the past, and there is growing evidence that personal exposures may at times be correlated poorly with average values derived for larger geographic areas.

Personal monitors are available in two types:

- **Active monitors** use a small air pump to draw air through a filter, packed tube, or similar device. They can be both continuous and integrated. Such a personal exposure monitor is available to measure PM10 and PM2.5 in air. The pump and battery pack are worn in a bag, while the filter can be located essentially anywhere on the body. Combinations of impactor and denuder filter packs are available to sample both aerosols and gases such as SO2, NH3, and HNO3. Different coating materials on the diffuser tube can be used to collect different gases.

- **Passive monitors** rely on sorption, entrapment, etc., driven largely by diffusion. They are primarily integrated sampling devices, giving an estimate of average exposure over the sampling period. Examples include diffusion tubes, badges, and detector tubes. Diffusion badges currently are available for measurement of NO2, O3, SO2, CO and formaldehyde. Organic vapors can be measured in passive devices using activated charcoal badges, although the range of compounds, aside from organics, that can be sampled in this way is small.
In general, air toxics risk assessments that rely on monitoring to characterize exposure will generally not rely on personal monitoring because of the highly complex and resource intensive nature of this technique, and because personal monitoring and its findings are currently more geared toward basic research.

### 6.7 Evaluating Uncertainty

Uncertainty includes the assumptions and unknown factors inherent in the exposure assessment. Discussing uncertainty places the risk estimates in proper perspective. Specific uncertainties associated with the chemical monitoring data, fate and transport models, and the input data (especially emissions inventory data) that assessors use to estimate exposure concentrations usually account for the bulk of uncertainty within the assessment. Exposure models also contribute to the overall uncertainty in exposure assessment. The assessor needs to understand the extent to which variability and uncertainty are considered in all the fate and transport and exposure models that are used.

The assessor should be familiar with the extent to which the various components of the exposure assessment can and do accommodate uncertainty and variability analyses. In addition, it is important to consider the compatibility of models in the various steps in the exposure assessment (emissions, transport, etc.) with regard to addressing important sources of uncertainty. Once the capabilities and data requirements of the various models are known, the assessor should consider the appropriate level of detail for addressing uncertainty in specific variables, and approaches for integrating uncertainty analyses across the models.

### 6.8 Presenting the Results of an Exposure Assessment

The summary of exposure assessment for air toxics consists of presenting the exposure concentrations (ECs) for each chemical of potential concern (COPC) with the duration of exposure for the populations of interest, as well as characterizing salient features of the study population(s), particularly those that may be influencing their exposure and resultant risk (i.e., size and proximity to sources and/or locations of highest ambient concentrations). The assumptions used to develop these estimates should also be presented and discussed. In addition to the summary tables, it is useful to show sample calculations for each pathway to aid in the review of the calculations. (If exposure modeling is used, a thorough discussion with sample calculations is usually also provided.)
References


USEPA web site, “Fate, Exposure, and Risk Assessment (FERA)” (Last accessed March 2009), (Available at http://www.epa.gov/ttn/fera/).
Chapter 7: Toxicity Assessment of Air Toxics

7.1 Introduction

The purpose of the **toxicity assessment** is to weigh available evidence regarding the potential for toxicity in exposed individuals (hazard identification) and to quantify the toxicity by deriving an appropriate dose-response value (dose-response assessment). Toxicity assessment is the second part of the general risk equation and is a two-step process: hazard identification and dose-response assessment. **Hazard identification** determines whether exposure to a chemical *can cause adverse health effects* (i.e., cancer, birth defects, etc.) *and/or environmental effects* (i.e., reduced visibility, damage to buildings, climate change, etc.), and also looks at the *strength of evidence of causation and circumstances* that cause these effects (i.e., long-term vs. short-term exposure, animal vs. human data, inhalation vs. ingestion route of exposure, etc.). **Dose response assessment** establishes a *quantitative relationship* between the *dose* of the contaminant and the incidence of *adverse health effects* (cancer and non-cancer) in the exposed population. The combination of toxicity assessment and exposure assessment will determine risk and the likelihood of toxic effects (see Exhibit 7-1).

**Exhibit 7-1: The Detailed Risk Assessment Process**
Although the toxicity assessment is an integral and important part of the overall air toxics risk assessment, it is usually accomplished prior to the risk assessment. EPA has completed this toxicity assessment for many HAPs and has made available the resulting toxicity information and dose-response values, which have undergone extensive peer review (see EPA web site for this information: http://www.epa.gov/ttn/atw/toxsource/summary.html).

In most air toxics risk assessments, the development or evaluation of new toxicological data will not generally be required. However, it is important for analysts to understand how the available toxicity data were developed in order to both select and use toxicity values appropriately and to be able to describe their associated uncertainties in the resulting risk assessment. A basic understanding of toxicity assessment will also aid in identifying and interpreting the results of a risk analysis and help communicate the risk assessment to interested parties and stakeholders.

7.2 Hazardous Identification

The hazard identification step has two parts. First, it reviews and analyzes toxicity data to see if exposure to a chemical can cause particular health (or environmental) effects, and second it weights the strength of evidence and circumstances that the chemical causes various toxic effects. Health effects resulting from exposure to air toxics at sufficient concentrations and durations include increased chance of getting cancer, damage to the immune system, as well as neurological, reproductive (reduced fertility), developmental (birth defects), respiratory, and other health problems. The strength of the hazard identification statement will be determined by contributing factors such as the route of exposure, the type and quality of the effects, the biological plausibility of findings, the consistency of findings across studies, and the potential for bioaccumulation.

7.2.1 Sources of Information for Hazard Identification

The first part of hazard identification is to determine whether the pollutants of concern are causally linked to adverse health effects. (This Manual will not be discussing adverse environmental effects.) Evidence is gathered from a variety of sources regarding the potential for an air toxic to cause adverse health effects in humans. These may include human data, experimental animal studies, and supporting information such as in-vitro laboratory tests (typically a cell culture from an animal or plant, or a tissue from an organism grown in the laboratory). The source of data affects the overall uncertainties in the resulting human dose-response values, as discussed below.
• **Human data.** Human toxicity data associated with exposures to air toxics may be located in *epidemiological studies, controlled exposure studies, or studies of accidental exposures*. Epidemiology is the study of the distribution and determinants of disease or health status in a population. Well-conducted epidemiological studies that show a positive association between exposure to a chemical and adverse health effects often provide evidence about human health effects associated with chronic exposures. Such data, however, are available only for a limited number of air toxics. Epidemiological data also are very difficult to interpret, because the number of exposed individuals may be small, the incidence of effects may be low, doses are usually not well-characterized, and there may be complicating factors such as simultaneous exposure to multiple chemicals and heterogeneity among the exposed group in terms of age, sex, diet, and other factors. Controlled exposure studies provide stronger evidence, since both the exposure duration and exposure concentrations are more accurately known. However, such studies with humans are generally limited to acute exposure durations. Studies reporting health effects associated with accidental exposures may be helpful, although exposure concentrations to air toxics may be high, and effects may be acute rather than chronic. Also note that small sample size is often a significant limitation to interpreting controlled and accidental exposure studies.

• **Animal data.** The toxicity database for most air toxics is drawn from experiments conducted on non-human mammals such as rats, mice, rabbits, guinea pigs, hamsters, dogs, or monkeys. The underlying assumption is that the susceptibility of humans and these animals to the effects of the chemicals is broadly similar because we share many common biological attributes (i.e., similar organs, similar and, in some cases, identical metabolic processes). However, some observations in animals may be of uncertain relevance to humans (i.e., if tumors are observed in an animal experiment, but the organ in which the tumor is formed does not exist in humans). Also, it is necessary to adjust the results from animal studies to humans due to differences in body mass, anatomy, metabolic rate, and other species-specific factors. This is why derivation of dose-response values from animal studies requires considerable expertise.

Animal testing can be described based on different exposure times to the toxic pollutant. Generally accepted descriptors are as follows:

• *Acute tests:* study the adverse effects after daily or continuous exposure for less than 14 days. These tests are usually relatively short in duration, but high in concentration.
- **Sub-chronic tests**: study the adverse effects after daily or continuous exposure from about one week up to 10% of the animal’s lifetime.
- **Chronic tests**: study the adverse effects (commonly tumor formation for carcinogens) after daily or continuous exposure over at least 10% of the animal’s lifetime. These tests are usually long in duration, but relatively low in concentration.

**Supporting data.** Metabolic, pharmacokinetic, and genotoxicity studies are sometimes used to infer the likelihood of adverse effects in humans. Metabolic studies on absorption, distribution, metabolism, and elimination can provide information about the mechanisms of toxicity associated with a particular chemical in humans. In physiologically based pharmacokinetic (PBPK) models, the body is subdivided into a series of anatomical or physiological “compartments” that represent specific organs or lumped tissue and organ groups, and the behavior of the chemical is modeled in each compartment. Genotoxicity studies provide evidence of the effects of chemicals on chromosomal material, and may demonstrate evidence of gene mutations, abnormalities of chromosomal structure, or changes in DNA replication or repair. Data on a chemical’s pharmacokinetics, genotoxicity, and possible mode of action can be used to refine a toxicity assessment. In some cases, computer models using structure-activity relationships (i.e., predictions of toxicological activity based on analysis of chemical structure) also may be used as supporting evidence. EPA considers these types of data to be supportive, not definitive, evidence of a chemical’s toxicity.

### 7.2.2 Weight of Evidence (Classification Scheme): Cancer Effects

A major determination made during the hazard identification step concerns the potential of a chemical to cause cancer in humans. This determination, which involves considering (or weighing) all the available evidence, is called the weight of evidence determination. This determination is complicated by possible inadequacies of the published studies, as well as differences in body processes between people and laboratory animals. EPA’s *Guidelines for Carcinogen Risk Assessment* guide scientists in interpreting available studies to assess the potential human carcinogenicity of environmental pollutants. EPA’s carcinogen risk assessment guidelines were first published in 1986 and created a classification scheme from A to E classifying the strength of evidence. The weight of evidence classification scheme from EPA’s 1986 *Guidelines for Carcinogen Risk Assessment* is as follows:

- **Group A**: *Human Carcinogen* (sufficient evidence of carcinogenicity in humans)
• **Group B1:** *Probable Human Carcinogen* (limited evidence of carcinogenicity in humans)

• **Group B2:** *Probable Human Carcinogen* (sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)

• **Group C:** *Possible Human Carcinogen* (limited evidence of carcinogenicity in animals with inadequate or lack of human data)

• **Group D:** *Not Classifiable as to Human Carcinogenicity* (inadequate or no evidence)

• **Group E:** *Evidence of Non-carcinogenicity for Humans* (no evidence of carcinogenicity in adequate studies)

Many existing carcinogen assessments were developed pursuant to EPA’s 1986 *Guidelines for Carcinogen Risk Assessment*, which used a simple but less informative weight of evidence system. In March of 2005, EPA replaced the 1986 Guidelines with the 2005 *Guidelines for Carcinogen Risk Assessment*. The 2005 Guidelines place particular importance on the consideration of a chemical’s “mode of action” and emphasize an analysis of the available data with regard to the key events inherent in how exposure to a chemical results in cancer. Evidence considered includes tumor findings, or lack thereof, in humans and laboratory animals; an agent’s chemical and physical properties; its structure-activity relationships (SARs) as compared with other carcinogenic agents; and studies addressing potential carcinogenic processes and mode(s) of action, either *in vivo* or *in vitro*.

To express conclusions about the weight of evidence for human carcinogenic potential, the 2005 Guidelines call for a complete characterization in a weight of evidence narrative. To give some measure of consistency in an otherwise free-form narrative, five hazard descriptors are recommended as part of the hazard assessment narrative ("Carcinogenic to Humans," “Likely to Be Carcinogenic to Humans,” “Suggestive Evidence of Carcinogenic Potential,” “Inadequate Information to Assess Carcinogenic Potential,” and “Not Likely to Be Carcinogenic to Humans."). The weight of evidence determination now includes one of these descriptors, and is accompanied by additional text that more completely summarizes EPA’s interpretation of the evidence. These hazard descriptors are described below in the new weight of evidence classification scheme from EPA’s 2005 *Guidelines for Carcinogen Risk Assessment*:
- **Carcinogenic to Humans:** The Guidelines recommend this descriptor when there is convincing epidemiologic evidence demonstrating causality between human exposure and cancer, or exceptionally when there is strong epidemiological evidence, extensive animal evidence, knowledge of the mode of action, and information that the mode of action is anticipated to occur in humans and progress to tumors.

- ** Likely to be Carcinogenic to Humans:** The Guidelines recommend this descriptor when the available tumor effects and other key data are adequate to demonstrate carcinogenic potential to humans, but does not reach the weight-of-evidence for the descriptor "carcinogenic to humans."

- **Suggestive Evidence of Carcinogenic Potential:** The Guidelines recommend this descriptor when the evidence from human or animal data is suggestive of carcinogenicity, which raises a concern for carcinogenic effects but is judged not sufficient for a stronger conclusion.

- **Inadequate Information to Assess Carcinogenic Potential:** The Guidelines recommend this descriptor when available data are judged inadequate to perform an assessment.

- **Not Likely to be Carcinogenic to Humans:** The Guidelines recommend this descriptor when the available data are considered robust for deciding that there is no basis for human hazard concern.

Information on how the Agency is implementing the 2005 *Cancer Guidelines* and *Supplemental Guidance* is available on the Agency’s web site (see [www.epa.gov/cancerguidelines](http://www.epa.gov/cancerguidelines) and [http://www.epa.gov/osa/spc/cancer.htm](http://www.epa.gov/osa/spc/cancer.htm)).

### 7.2.3 Weight of Evidence (Narrative Approach): Non-Cancer Effects

In large part due to the wide variety of endpoints, hazard identification procedures for non-cancer effects are less formally described in EPA guidance than procedures for the identification of carcinogens. The EPA has published guidelines for assessing several specific types of non-cancer effects, and rather than specifying risk assessment methodology, these non-cancer guidelines tend to focus on the proper conduct of testing and the appropriate toxicological interpretation of results of the commonly performed
Introduction to Air Toxics

The guidance for hazard identification decisions is fairly general. A more comprehensive discussion of hazard identification and the evaluation of the underlying database for non-cancer effects are included in the EPA document “A Review of the Reference Dose and Reference Concentration Process” (2002).

For assessment of chronic toxic effects other than cancer, EPA’s general approach to hazard identification is to review the health effects literature and characterize its strengths and weaknesses, using primarily a narrative approach rather than a formal classification scheme. The narrative description discusses factors such as the methodological strengths and weaknesses of individual studies, the time period over which the studies were conducted (i.e., chronic vs. sub-chronic), routes of exposure, and possible biological mechanisms.

7.2.3.1 Identification of Critical Effects – Non-Cancer Endpoints: As part of the characterization of the available information on non-cancer health effects, the targets of chemical toxicity within the body are identified, along with what have been termed “critical effects” associated with the toxicity. A critical effect is described as “either the adverse effect that first appears in the dose scale as dose is increased, or as a known precursor to the first adverse effect.” Note that not all observed effects in toxicity studies are considered adverse effects.

7.3 Dose Response Assessment

A dose-response assessment is the process of quantitatively evaluating toxicity information, characterizing the relationship between the dose of the contaminant received (or the inhalation exposure concentration, for inhalation assessments) and the incidence of adverse health effects in the exposed subjects (which may be animal or human) and then, as appropriate, extrapolating these results to human populations. Depending on the type of effect and the chemical, there are two types of dose-response values that traditionally may be derived: predictive cancer risk estimates, such as the inhalation unit risk estimate (IUR), and predictive non-cancer estimates, such as the reference concentration (RfC). Both types of dose-response values may be developed for the same chemical, as appropriate.

Inhalation Unit Risk (IUR): The upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent via inhalation per μg/m³ over a lifetime. The interpretation of the IUR would be as follows: if IUR = 2 × 10⁻⁶ μg/m³, not more than 2 excess tumors are expected to develop per 1,000,000 people if exposed continuously for a
lifetime to 1 μg of the chemical per cubic meter of inhaled air. The number of expected tumors is likely to be less; it may even be none.

**Reference Concentration (RfC):** An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subpopulations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. RfC is generally used in EPA’s non-cancer health assessments.

The relationship of dose to response can be illustrated as a graph called a *dose-response curve*. Exhibit 7-1 is a simple example of a dose-response curve for graded responses of a specific physiological parameter to increasing exposure. The output of the dose-response assessment is the relationship between *dose* (the level of exposure) and the resulting *response* (the increased incidence and/or severity of adverse effects).

![Exhibit 7-1: Dose-Response Curve](image)

An important aspect of dose-response relationships is whether the available evidence suggests the existence of a threshold. For many types of toxic responses, there is a *threshold dose* or dose rate below which there are thought to be no adverse effects from exposure to the chemical (see Exhibit 7-2). The human body has defenses against many toxic agents. Cells in human organs, especially in the liver and kidneys, break down many chemicals into less toxic substances that can be eliminated from the body in urine and feces. In this way, the human body can withstand some chemical exposure (at doses below the threshold) and still remain healthy.
Depending on whether a substance causes cancer and whether its dose-response curve is thought to have a threshold, EPA may use either of two approaches in a dose-response assessment. One approach produces a predictive estimate (inhalation cancer risk estimate), and the other produces a reference value (RfC). Historically, the use of a predictive estimate has been limited to cancer assessment. That is, dose-response assessments for cancer have been expressed as predictive cancer risk estimates based on an assumption that any amount of exposure poses some risk. Exhibit 7-3 displays a non-threshold dose for carcinogens: showing no exposure is without risk. Assessments of effects other than cancer usually have been expressed as reference values at or below which no harm is expected (threshold). Many substances have been assessed both ways: the first for cancer and the second for adverse effects other than cancer. While this use of predictive estimates for cancer and reference values for other effects is still the practice for the vast majority of chemicals, EPA now recognizes that there are chemicals for which the data support an alternate approach.
Epidemiological and toxicological data on air toxics typically result from exposure levels that are high relative to environmental levels; therefore low-dose extrapolation (prediction) is necessary to derive an applicable dose-response value. Low-dose extrapolation requires either information or assumptions about the type of dose-response curve likely under low dose situations. Confidence in the toxicity levels is indicated for non-carcinogens by applying uncertainty and modifying factors and by discussion of the confidence level. Confidence for carcinogens is indicated by EPA’s weight of evidence evaluations.

### 7.4 Dose-Response Assessment for Cancer Effects

The inhalation cancer dose-response value derived by linear extrapolation is the *Inhalation Unit Risk (IUR)* (also known as *cancer potency*). It is presented as an upper-bound estimate of the excess cancer risk resulting from a lifetime (assumed 70-year) of continuous exposure to an agent at a concentration of 1 μg/m³ in air. As illustrated in Exhibit 7-4, IUR is the slope of the dose-response line. Risk is the product of the slope and the estimated exposure.

\[
\text{Risk} = EC \times \text{IUR}, \quad \text{where}
\]

\[
\text{EC} = \text{lifetime estimate of continuous inhalation exposure to an individual air toxic.}
\]

\[
\text{IUR} = \text{the corresponding inhalation unit risk estimate for that air toxic.}
\]
7.4.1 Derivation of Inhalation Unit Risk (IUR) for Cancer Effects

Exhibit 7-5 shows the process for deriving a quantitative dose-response estimate for cancer (a cancer slope factor or IUR).
It involves the following three steps:

1. Determination of the *Point of Departure (POD)* from an animal study;

2. *Duration adjustment* of the POD to continuous exposure;

3. *Interspecies extrapolation* of the POD into its corresponding Human Equivalent Concentration (POD\(_{\text{HEC}}\)); and

4. *Extrapolation from the POD\(_{\text{HEC}}\)* to derive the carcinogenic potency estimates (IUR).

### 7.4.1.1 Determination of Point of Departure (POD):

Dose-response assessment for cancer and other effects begins with identification of the point of departure (an exposure concentration or intake) from the experimental data. This point, while within the range of observation, is the point from which extrapolation begins, either for the purposes of deriving a cancer risk estimate (the IUR) or a RfC for non-cancer health effects.

The POD may be the traditional *no observed adverse effect level (NOAEL)*, *lowest observed adverse effect level (LOAEL)*, or *benchmark concentration (BMC)*. EPA has recommended the use of the BMC approach, where possible, because the traditional use of the LOAEL or NOAEL in determining the POD has long been recognized as having several limitations (and generally is not used in dose-response for cancer effects).

The BMC approach is an alternate way of determining the point of departure for low-dose extrapolation. It can be used in cancer and non-cancer risk assessment as the starting point for linear low-dose extrapolation. BMC methods involve fitting various mathematical models for dose-response to reported data and using the different results to select a BMC that is associated with a predetermined benchmark response, such as a 10 percent increase in the incidence of a particular lesion or a 10 percent decrease in body weight gain. EPA has developed the Benchmark Dose Software (BMDS) to facilitate these operations. BMDS currently offers 16 different mathematical models that can be fit to the laboratory data.

It is likely that there will continue to be situations that are not amenable to BMC modeling and for which a NOAEL or LOAEL approach should be used. In some cases,
there may be a combination of benchmark doses and NOAELs to be considered in the assessment of a particular agent.

7.4.1.2 Duration adjustment of the POD to continuous exposure: Because inhalation toxicity studies typically involve discontinuous exposures (i.e., animal studies routinely involve inhalation exposures of 6 hours per day, 5 days per week), the POD will usually need to be extrapolated to a continuous exposure scenario. This duration adjustment step is essential in interpreting inhalation studies, but is not routinely necessary for the interpretation of oral exposures. Operationally, this is accomplished by applying a concentration-duration product (or $C \times t$ product) for both the number of hours in a daily exposure period and the number of days per week that the exposures are performed. For example, for a POD of 100 mg/m$^3$ derived from an animal study in which animals are exposed by inhalation for 6 hours per day, 5 days per week, the adjustment to a continuous exposure concentration would consider both hours per day and days per week:

$$100 \text{ mg/m}^3 \times (6/24) \times (5/7) = 18 \text{ mg/m}^3$$

Thus, 18 mg/m$^3$ is the POD concentration adjusted for continuous exposure versus 100 mg/m$^3$ unadjusted.

7.4.1.3 Interspecies extrapolation of the POD into its corresponding Human Equivalent Concentration (POD$_{HEC}$): After duration adjustment, the POD is converted into a human equivalent concentration (HEC) from the experimental animal dose. This extrapolation or adjustment compensates for interspecies differences (between humans and animals tested) such as size, life spans, and pharmacokinetics (what the body does with the chemical once it’s inside the body). Some examples of pharmacokinetics differences include metabolic (how the body converts the chemical to a less toxic substance), excretion and distribution to storage sites (i.e., fat, bones, etc.), and absorption rate (mainly in lung and small intestines).

The interspecies extrapolation may be done using default methods specific to the particular chemical class of concern or more refined methods such as physiologically-based pharmacokinetic (PBPK) modeling. The Agency’s inhalation dosimetry methodology provides a recommended hierarchy, as well as default generalized procedures for deriving dosimetric adjustment factors (DAFs) for this extrapolation. Application of DAFs to an animal exposure value yields an estimate of the corresponding concentration relevant to humans (the HEC).

7.4.1.4 Extrapolation from the POD$_{HEC}$ to derive the carcinogenic potency estimates (IUR): Observable cancer rates in laboratory or human occupational epidemiologic
studies tend to be several orders of magnitude higher than cancer risk levels that society is willing to tolerate from involuntary chemical exposures. To obtain observable results, laboratory studies need to be extrapolation from the POD_{HEC} to lower doses is usually necessary. This extrapolation is performed consistent with the mode of action, if adequately supported. A linear extrapolation is generally appropriate when the evidence supports a mode of action of gene mutation due to direct DNA reactivity or another mode of action that is thought to be linear in the low dose region. For linear extrapolation, a straight line is drawn from the point of departure to the origin, and the risk at any concentration is determined by interpolation along that line. A linear mode of action also will serve as a default when available evidence is not sufficient to support a nonlinear extrapolation procedure, even if there is no evidence for DNA reactivity.

An assumption of nonlinearity is used when there is sufficient evidence to support a nonlinear mode of action. A nonlinear mode of action could involve a dose-response pattern in which the response falls much more quickly than linearly with dose, but still indicating risk at low doses.

For linear extrapolation, a straight line is drawn from the point of departure expressed as a human equivalent dose to the origin (i.e., zero incremental dose, zero incremental response) to give an incremental probability dose unit. EPA’s 2005 Guidelines for Carcinogen Risk Assessment recommends the use of the lowest effective dose (concentration) using a 10 percent human response level (LEC_{10}) (as estimated by the lower one-sided confidence limit on the benchmark concentration [or BMCL_{10}]) as the POD for linear extrapolation. The LEC_{10} is the lower 95 percent limit on a dose (exposure concentration) that is estimated to cause a 10 percent response. This approach is to draw a straight line between the estimated POD_{LEC10}, and to the graphs origin (see Exhibit 7-6). This linear extrapolation creates a straight line whose slope is the Inhalation Unit Risk (IUR).
7.4.2 Derivation of Inhalation Reference Concentration (RfC) for Non-Cancer Effects

The inhalation reference concentration (RfC) is defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive sub-populations) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC is expressed as a chronic exposure level to the chemical in ambient air (in units of milligrams of the substance per cubic meter of air, or mg/m$^3$). This value is usually derived for use with effects other than cancer. But when a chemical’s carcinogenicity has been shown to be associated with a nonlinear mode of action, a reference concentration may be derived for use with all effects, including cancer.

Inherent in the derivation of a reference concentration is the recognition of an exposure level likely to be without an appreciable risk of adverse effects (sub-threshold level for adverse effects). The objective of this type of dose-response assessment, then, is to
estimate that exposure level for humans. The RfC is derived after a thorough review of the health effects database for an individual chemical and identification of the most sensitive and relevant endpoint (the “critical effect”) along with the principal studies demonstrating that endpoint. In addition to an analysis of the study data available for the chemical, risk assessors also use uncertainty factors to account for differences in sensitivity between humans and laboratory animals, the possibility of heightened sensitivity of some population groups (i.e., people with respiratory disease, very young children, the aged), and any limitations of the database.

The first part of this type of assessment, which involves a careful qualitative and quantitative analysis of the study data, parallels that performed for linear cancer dose-response assessment (i.e., derivation of the point of departure in terms of a human equivalent concentration [POD_{HEC}]). The latter part of this type of assessment involves the application of uncertainty factors to address limitations of the data used.

### 7.4.2.1 Derivation of the Point of Departure in Terms of a Human Equivalent Concentration (POD_{HEC}):

Derivation of the RfC begins with identification of the critical effect (the adverse effect that appears at the lowest dose) from the available human and animal study data. Using the dose-response relationship for the critical effect, toxicologists identify the POD from the experimental data. This exposure concentration (in terms of its human equivalent) which marks the boundary between the range of observation and that of extrapolation, is the point from which extrapolation begins for derivation of a RfC. The POD may be derived from benchmark concentration (BMC) modeling. If the data do not meet requirements for benchmark modeling, the POD is derived by the use of a statistical analysis to identify the no-observed-adverse-effect-level, or NOAEL, defined as the highest dose level administered to laboratory animals that did not cause statistically or biologically significant observable adverse effects after chronic (usually lifetime) exposure in the studied population. In some cases, a lowest-observed adverse-effect level (LOAEL) is used in the absence of a NOAEL.

The BMC approach is an alternative to the NOAEL approach as a way to identify a dose associated with a given level of response, or a dose without appreciable effect based on experimental data. The BMC approach fits a dose-response curve to the data in the observed experimental range. A lower bound on the dose causing some specified level of risk above background (i.e., 10 percent) is calculated, and this dose value is used as a point of departure (POD) for the application of uncertainty factors (UF)s in place of the experimental NOAEL or LOAEL. In either case, the POD is transformed into a continuous inhalation exposure (i.e., from an intermittent animal exposure, 6 hours/day, 5
days/week) and then into a human equivalent concentration using dosimetric methods (as described in Section 7.4.1).

Exhibit 7-7 (example of a non-linear approach) is an overview of development of an inhalation reference concentration (RfC). The LOAEL_{HEC} and NOAEL_{HEC} are illustrated with the application of uncertainty or modifying factors to derive the human health-protective RfC. (Note: NOEL is the no observed effect level and does not measure an adverse effect.)

**Exhibit 7-7: Dose-Response Curves for Liver Toxicity, Tremors, and Enzyme Changes**

7.4.2.2 Application of Uncertainty Factors (UF)s: The RfC is an estimate derived from the PODHEC for the critical effect (based on either a BMCLHEC, NOAELHEC or LOAELHEC) by consistent application of uncertainty factors (UF)s. The UF{s} are applied to account for recognized uncertainties in the use of the available data to estimate an exposure concentration appropriate to the assumed human scenario. The general formula for deriving an RfC from a PODHEC is:

\[
RfC \, (\text{mg/m}^3) = \frac{POD_{HEC} \, (\text{mg/m}^3)}{UF}
\]
A UF of 10, 3 or 1 is applied in each of the following extrapolations used to derive the RfC (see EPA’s 1994 document “Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry.”)

- **Animal to human**: Extrapolation from valid results of long-term studies on laboratory animals when results of studies of human exposure are not available or are inadequate. Intended to account for the uncertainty in extrapolating laboratory animal data to the case of average healthy humans.

- **Human to sensitive human populations**: Extrapolation of valid experimental results for studies using prolonged exposure to average healthy humans. Intended to account for the variation in sensitivity among the members of the human population.

- **Sub-chronic to chronic**: Extrapolation from less than chronic exposure results on laboratory animals or humans when there are no useful long-term human data. Intended to account for the uncertainty in extrapolating from less than chronic NOAELs to chronic NOAELs.

- **LOAEL to NOAEL**: Derivation from a LOAEL instead of a NOAEL. Intended to account for the uncertainty in extrapolating from LOAELs to NOAELs. Incomplete to complete database. Extrapolation from valid results in laboratory animals when the data are “incomplete.” Intended to account for the inability of any single laboratory animal study to adequately address all possible adverse outcomes in humans.

- **Incomplete to complete data**: Extrapolation from valid results in laboratory animals when the data are “incomplete.” Intended to account for the inability of any single laboratory animal study to adequately address all possible adverse outcomes in humans.

The UF values are generally an order of magnitude (10), although incorporation of dosimetry adjustments or other information may result in the use of reduced UF values for RfCs (3 or 1). The composite UF applied to an RfC will vary in magnitude depending on the number of uncertainties involved; however, an RfC will not be derived when use of the data involves more than four areas of extrapolation. The composite UF when four factors are used generally is reduced from 10,000 to 3,000 in recognition of the lack of independence and the conservatism of these factors.
Because of this procedure to address the lack of information on the translation from experimental data to a human scenario, the resulting RfC for many HAPs is on the order of 100 to 300 times lower than the NOAEL actually observed in the animal testing (see Exhibit 7-7). This reflects the lowering of the RfC to address the uncertainties in the extrapolations mentioned above. For those HAPs that have had their effects well documented in human studies, the RfC may be much closer to the highest concentration at which an adverse effect was not observed (i.e., within a factor of 3 to 10).

### Exhibit 7-7: Examples of the Use of Uncertainty Factors in Deriving RfCs

<table>
<thead>
<tr>
<th>RfC from NOAEL</th>
<th>RfC from LOAEL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Example: Diesel Engine Emissions</td>
<td>Example: Toluene</td>
</tr>
<tr>
<td><strong>Toxicity data:</strong></td>
<td><strong>Toxicity data:</strong></td>
</tr>
<tr>
<td>144 μg chemical/m$^3$ air (NOAELHEC from chronic rodent study)</td>
<td>119 mg chemical/m$^3$ air (LOAELHEC from chronic occupational study)</td>
</tr>
<tr>
<td><strong>Uncertainty factors:</strong> 3 x 10 = 30</td>
<td><strong>Uncertainty factors:</strong> 10 x 10 x 3 = 300</td>
</tr>
<tr>
<td>3 = animal-to-human extrapolation</td>
<td>10 = human to sensitive human subpopulations</td>
</tr>
<tr>
<td>10 = human to sensitive human subpopulations</td>
<td>10 = LOAEL-to-NOAEL extrapolation</td>
</tr>
<tr>
<td>RfC = 144/30 = 4.8 μg/m$^3$ = 0.005 mg/m$^3$</td>
<td>3 = database deficiencies</td>
</tr>
<tr>
<td></td>
<td>RfC = 119/300 mg/m$^3$ = 0.4 mg/m$^3$</td>
</tr>
</tbody>
</table>

NOAELHEC = No-Observed-Adverse-Effect Level (Human Equivalent Concentration)
LOAELHEC = Lowest-Observed-Adverse-Effect Level (Human Equivalent Concentration)

*Source: EPA’s IRIS database [http://www.epa.gov/IRIS/](http://www.epa.gov/IRIS/)*

In some of the older Integrated Risk Information System (IRIS) assessments a “modifying factor” may have been applied in addition to the traditional uncertainty factors. It had been used with professional judgment when it was determined that another uncertainty factor was needed; its magnitude depended upon the professional assessment of scientific uncertainties of the study and database not explicitly treated via the other uncertainty factors. The 2002 Agency review of the RfC process (“A Review of the Reference Dose and Reference Concentration Process,” Risk Assessment Forum, Washington, D.C., 2002), however, recommended against continued use of the modifying factor. It was felt that the traditional factors could account for any remaining uncertainties.

### 7.5 Sources of Toxicity Data

As stated earlier, EPA has completed toxicity assessment for many HAPs and has made available the resulting toxicity information and dose-response values, which have
undergone extensive peer review (see: http://www.epa.gov/ttn/atw/toxsource/summary.html). This site compiles assessments from various sources for many of the 187 substances listed as hazardous air pollutants ("air toxics") under the Clean Air Act Amendments of 1990. Many of the below web sources (including IRIS) of chronic dose-response can be accessed through this EPA web site.

- **EPA’s Integrated Risk Information System (IRIS) Database:** For chronic non-cancer criteria (i.e., RfC) and cancer criteria (i.e., IUR) of toxic chemicals the preferred source of information is IRIS. Dose-response assessments that have achieved full intra-agency consensus are incorporated in IRIS, which is regularly updated and available on-line (www.epa.gov/iris). All IRIS assessments since 1996 also have undergone independent external peer review. In the past, dose-response assessments for some substances were prepared by the EPA Office of Research and Development, but were never submitted for EPA consensus.

- **Health Effects Assessment Summary Tables (HEAST):** Although the values in HEAST have undergone some review and have the concurrence of individual Agency program offices, they have not had enough review to be recognized as Agency-wide consensus information. In addition, since HEAST has not been updated since 1997, other sources described here are, for many chemicals, more reliable.

- **Agency for Toxic Substances and Disease Registry (ATSDR):** ATSDR, which is part of the US Department of Health and Human Services, develops and publishes Minimum Risk Levels (MRLs) for many toxic substances. The MRL is defined as an estimate of daily human exposure to a substance that is likely to be without an appreciable risk of adverse effects (other than cancer) over a specified duration of exposure. ATSDR describes MRLs as substance-specific estimates to be used by health assessors to select environmental contaminants for further evaluation. The MRL data undergo a rigorous review process, including internal ATSDR review, peer reviews, and public comment periods. ATSDR Minimal Risk Levels (MRLs) for Hazardous Substances is available at http://www.atsdr.cdc.gov/mrls.html.

- **California Environmental Protection Agency (CalEPA):** The CalEPA Office of Environmental Health Hazard Assessment (OEHHA) has developed dose-response assessments for many substances, based both on carcinogenicity and health effects other than cancer. The process for developing these assessments is
similar to that used by EPA to develop IRIS values and includes significant external scientific peer review. The non-cancer information includes inhalation health risk guidance values expressed as chronic inhalation reference exposure levels (RELs). CalEPA defines the REL as a concentration level at (or below) which no health effects are anticipated, a concept that is substantially similar to EPA’s approach to non-cancer dose-response assessment. CalEPA’s dose response assessments for carcinogens and noncarcinogens are available on-line at: http://www.oehha.ca.gov/air/hot_spots/index.html.

- **Health Effects Notebook for Hazardous Air Pollutants:** the EPA has compiled fact sheets for the 188 CAA hazardous air pollutants and makes them available on the Air Toxics website (http://www.epa.gov/ttn/atw/hapindex.html). This collection is called the Health Effects Notebook for Hazardous Air Pollutants, and provides for these HAPs a summary of available information in the following categories: hazard summary, physical properties, uses, sources and potential exposure, and health hazard information. These fact sheets are useful for describing hazards associated with the 188 HAPs.

- **EPA’s National Center for Environmental Assessment (NCEA):** provides guidance and risk assessments aimed at protecting human health and the environment. This guidance presents critical analyses and summaries of scientific consensus, vetted through a rigorous peer review process, on the risks of pollutants to human health and the natural environment (site available at http://www.epa.gov/ncea).
References


Chapter 8: Risk Characterization of Air Toxics

8.1 Introduction

The final step in the risk assessment process is risk characterization. This step combines the information from the exposure assessment and the toxicity assessment to provide a quantitative estimate of potential cancer risk and/or hazard for other adverse effects, along with a statement of confidence about the data and methods used. Quantitative statements of risk estimates are presented in the context of uncertainties and limitations in the underlying data and methodology. The evaluation of the assumptions and uncertainties inherent in the risk assessment is critical in order to place the risk estimates in proper perspective so that risk managers understand the strengths and limitations of the risk assessment. The risk manager will take the risk characterization information (along with legal, social, economic, political, and public health factors) into consideration in making a risk management decision concerning exposure to air toxics (see Exhibit 8-1).

Risk assessors purposefully use the term risk to mean the statistical probability of developing cancer over a lifetime (even if exposure only occurs over a portion of that lifetime). Non-cancer “risks,” on the other hand, are not expressed as a statistical probability of developing a disease. Rather they are expressed as a simple comparison (called the “hazard quotient”) of the exposure concentration to a reference concentration associated with the observable adverse health effects. To help make this distinction, the potential harm from exposure to carcinogens is called “risk” and the potential harm from non-carcinogens is called “hazard.”
In risk characterization, cancer risks are presented separately from non-cancer hazards. Estimated cancer risks and non-cancer hazards are generally developed for each chemical to which people are exposed in the study area and each exposure pathway through which exposure can occur. The results are then summed in a specific way to provide total estimates of risk and hazard (i.e., the ingestion risk estimates are first added across all ingestion pathways and then added to inhalation risk estimates to calculate total risk – then the process is repeated for all non-cancer risks). The general steps involved in risk characterization are:

1. Organize outputs of inhalation exposure and toxicity assessments.

2. Derive (data are available on-line at EPA’s Integrated Risk Information System [IRIS] database for most of the CAA 187 air toxics) inhalation cancer risk estimates and non-cancer hazard quotients for each pollutant in each pathway for each type of receptor being studied.

3. Derive cumulative inhalation cancer risk estimates and non-cancer hazards for each receptor for all chemicals in a pathway and then across pathways.

4. Identify key features and assumptions of exposure and toxicity assessments.

5. Assess and characterize key uncertainties and variability associated with the assessment.

6. Consider additional relevant information (i.e., related studies).

The risk characterization for multi-pathway risk assessments is more complicated than that for only a single pathway (i.e., inhalation). For this reason, and because our concern is mainly with air toxics, this chapter will focus on the inhalation pathway only.

8.2 Inhalation Cancer Risk

For inhalation exposures, chronic cancer risks for individual air toxics are typically estimated by multiplying the estimate of long-term exposure concentration (EC) by the
corresponding inhalation unit risk (IUR) for each pollutant to estimate the potential incremental cancer risk for an individual:

\[ \text{Risk} = \text{ECL} \times \text{IUR} \]

where:

- **Risk** = Cancer risk to an individual (expressed as an upper-bound risk of contracting cancer over a lifetime);
- **ECL** = Estimate of long-term inhalation exposure concentration for a specific air toxic; and
- **IUR** = the corresponding inhalation unit risk estimate for that air toxic.

Performing the estimate in this way provides an estimate of the probability of developing cancer over a lifetime due to the exposure in question. Because of the way this equation is written, the underlying presumption is that a person is exposed continuously to the ECL for their full lifetime (usually assumed to be 70 years). The ECL is an estimate of this long-term exposure even though it is probably based on one year’s worth of monitoring data or a modeling run that covers only one year’s worth of time.

Estimates of cancer risk are usually expressed as a statistical probability represented in scientific notation as a negative exponent of 10. For example, an additional risk of contracting cancer of one chance in 10,000 (or one additional person in 10,000) is written as \(1 \times 10^{-4}\) (or 1E-04). This means that for every 10,000 people that are exposed, *in the way that we have presumed*, one of those people may develop cancer over their lifetime. Likewise, a risk of one person in one million is written \(1 \times 10^{-6}\) (or 1E-06) and a risk or one in one hundred thousand is written \(1 \times 10^{-5}\) (or 1E-05).

Because IURs are typically upper-bound estimates, actual risks may be lower than predicted, and the true value of the risk is unknown and may be as low as zero. These statistical projections of hypothetical risk are intended as screening tools for risk managers and cannot make realistic predictions of biological effects. Such risk estimates also cannot be used to determine whether someone who already has cancer because of a past exposure.

Risks for cancer are generally expressed as *individual risks* (i.e., the risk borne by an individual in a larger exposed population). The number of people in the population who have the same risk level (*population risk*) may also be provided (i.e., 1,000,000 people in the exposed population under study have a risk of \(1 \times 10^{-6}\), 2,495 have a risk of \(1 \times 10^{-5}\), and 300 have a risk of \(1 \times 10^{-4}\)). It is also possible to calculate the number of cases of cancer expected over a 70-year period by multiplying the cancer risk to an individual by the
number of individuals. This calculation yields an estimate of predicted cancer incidence rates. For example if 700,000 people are exposed to a carcinogen (IUR is \(1 \times 10^{-5}\)), how many new cancer cases will result per year? The following calculation yields:

- \(700,000\) (population size) x \([1 \times 10^{-5} \text{ (unit risk)}]\) x [1 yr. (exposure duration)] ÷ 70 yr. (averaging time)] = up to 0.01 new cancer cases per year

Exhibit 8-2 is a simple example of inhalation cancer risk calculations for two different chemicals. Chemical A has the higher cancer risk (0.002) than chemical B (0.0001). Therefore, at first glance it would seem that the exposure to chemical A is more toxic than chemical B. But, chemical A is only a “possible carcinogen,” whereas chemical is a “known human carcinogen.” All of this should be discussed in the risk characterization portion of the risk assessment.

Exhibit 8-2: Example of Inhalation Cancer Risk for Two Chemicals

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Exposure Concentration</th>
<th>IUR</th>
<th>Class</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1 µg/m³</td>
<td>(2 \times 10^{-3})</td>
<td>“C” Possible Carcinogen</td>
<td>(0.002)</td>
</tr>
<tr>
<td>B</td>
<td>5 µg/m³</td>
<td>(2 \times 10^{-5})</td>
<td>“A” Known Human Carcinogen</td>
<td>(0.0001)</td>
</tr>
</tbody>
</table>

8.2.1 Cancer Risk from Exposure to Multiple Pollutants

People may receive exposure to multiple chemicals, rather than a single chemical, at the same time. The concurrent exposure to multiple carcinogens may occur through the same pathway or across several pathways. With a few exceptions (i.e., coke oven emissions), cancer dose-response values (i.e., IURs) are usually available only for individual compounds within a mixture.
The following equation estimates the predicted cumulative incremental individual cancer risk from multiple substances, and assumes an additive effect from simultaneous exposures to several carcinogens:

\[
\text{Risk}_T = \text{Risk}_1 + \text{Risk}_2 + \ldots + \text{Risk}_i \quad \text{where:}
\]

\[
\text{Risk}_T = \text{total cumulative individual pathway-specific cancer risk (expressed as an upper-bound risk of contracting cancer over a lifetime); and}
\]

\[
\text{Risk}_i = \text{individual risk estimate for the } i^{th} \text{ substance in the inhalation pathway.}
\]

In screening-level assessments of carcinogens, for which there is an assumption of a linear dose-response, the cancer risks predicted for individual chemicals may be added to estimate cumulative cancer risk. This approach assumes that the risks associated with individual chemicals in the mixture are additive. In more refined assessments, the chemicals under assessment may be evaluated to determine whether effects from multiple chemicals are synergistic (greater than additive) or antagonistic (less than additive), although sufficient data for this evaluation are usually lacking. In those cases where IURs are available for a chemical mixture of concern, risk characterization can be conducted on the mixture using the same procedures as for a single compound. When more than one pathway is involved, the pathway specific risks are generally summed first, and then summed across pathways.

Exhibit 8-3 is an example calculation to estimate cancer risk from exposure to multiple chemicals (hypothetical). In this example, a Tier 1 modeling analysis was performed to estimate risk to the maximum exposed individual, assumed to reside at the point of maximum concentration for ABC Factory. Four HAPs were potentially of concern: benzene, dichloroethyl ether, formaldehyde, and cadmium compounds. Cancer risk estimates were obtained for each HAP by multiplying the estimated annual average EC by the IUR for each HAP. The resulting upper bound cancer risk estimates ranged from \(2 \times 10^{-6}\) (benzene, formaldehyde) to \(8 \times 10^{-4}\) (dichloroethyl ether). The cancer risk estimates for each HAP were summed to obtain an estimate of total inhalation cancer risk (\(9 \times 10^{-4}\)). Note that 97 percent of the estimated total risk results from dichloroethyl ether, and that more than 99 percent results from dichloroethyl ether and cadmium compounds. In this hypothetical example, the risk assessor would need to decide which HAPs to carry to higher tiers by weighing the small proportion of risk posed by benzene and formaldehyde against the fact that these risks nevertheless exceeded one in one million.
Exhibit 8-3: Example - Estimation of Cancer Risk from Exposure of Multiple Chemicals

<table>
<thead>
<tr>
<th>HAP</th>
<th>EC $\mu g/m^3$</th>
<th>IUR $1/(\mu g/m^3)$</th>
<th>Cancer Risk $\mu g/m^3$</th>
<th>% of Total Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>0.3</td>
<td>$7.8 \times 10^{-6}$</td>
<td>$2 \times 10^{-6}$</td>
<td>&lt; 1%</td>
</tr>
<tr>
<td>Dichloroethyl Ether</td>
<td>2.5</td>
<td>$3.3 \times 10^{-4}$</td>
<td>$8 \times 10^{-4}$</td>
<td>97%</td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>0.2</td>
<td>$1.3 \times 10^{-4}$</td>
<td>$2 \times 10^{-6}$</td>
<td>&lt; 1%</td>
</tr>
<tr>
<td>Cadmium compounds</td>
<td>0.1</td>
<td>$1.8 \times 10^{-3}$</td>
<td>$1 \times 10^{-4}$</td>
<td>2 %</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td></td>
<td>$9 \times 10^{-4}$</td>
<td></td>
</tr>
</tbody>
</table>

8.2.2 Descriptors of Cancer Risk

One of the important data quality objectives for risk characterization is the need to present multiple descriptors of risk, given the likely distribution (distributions are more useful than point estimates) of exposure for the study area population. Except where these descriptors clearly do not apply, all Agency risk assessments are expected to address or provide descriptions of risk to: (1) individuals exposed at average levels and those in the high-end portions of the risk distribution; (2) the exposed population as a whole; and (3) important subgroups of the population such as highly susceptible groups or individuals (i.e., children), if known.

**Individual Risk** (*central tendency* and *high-end* estimates of individual risk and hazard): Such measures are intended to give a sense of the risks posed to a typical individual in the exposed community as well as more highly exposed individuals.

- **Central Tendency Estimates**: Central tendency estimates are intended to give a characterization of risk for the typical individual in the population. This is usually either based on the arithmetic mean risk (average estimate) or the median risk (median estimate). One way to do this is to rank order all the risk values calculated across all modeling nodes in the study area and use the 50th percentile value as the measure of central tendency. Another method is to identify the arithmetic average of all calculated risks. There is no prescribed way of representing the “average person” and risk managers will often find it helpful to see several different ways of representing central tendency.

- **High-end**: High end estimates are intended to estimate the risk that is expected to occur in the upper range of the distribution (i.e., risk above about the 90th percentile of the population distribution). For example, the maximum exposed individual (MEI) risk or maximum individual risk (MIR) might be used to estimate high-end risks. The maximum individual risk (MIR) represents the
highest estimated risk to an exposed individual in areas that people are believed to occupy. The *maximum exposed individual (MEI)* represents the highest estimated risk to an exposed individual, regardless of whether people are expected to occupy that area.

**Population Risk** *(the number of people at different risk and hazard levels):* These measures are particularly important for risk managers because they answer the broad question “are many people at high risk, or only a few.” For example, the analyst might decide to select risk bins (i.e., Bin 1 includes all people with a risk below 1E-06, Bin 2 includes all people with a risk of 1E-06 to 1E-05, etc.) and determine the numbers of people in each bin.

**Sensitive or Susceptible Subpopulations:** Risk predictions for sensitive subpopulations are a subset of population risks. Sensitive subpopulations consist of a specific set of individuals who are particularly susceptible to adverse health effects because of physiological (i.e., age, gender, pre-existing conditions), socioeconomic (i.e., nutrition), or demographic variables, or significantly greater levels of exposure. Subpopulations can be defined using age, race, gender, and other factors. If enough information is available, a quantitative risk estimate for a subpopulation can be developed. If not, then any qualitative information about subpopulations gathered during hazard identification should be summarized as part of the risk characterization. It may not be necessary or possible to do a quantitative risk assessment on each subpopulation. For instance, where there are many sensitive population groups for a given pollutant, it may be sufficient to estimate risks for the most sensitive group, with the idea that as long as they are protected by the associated risk management action, other groups may be protected adequately.

### 8.3 Non-Cancer Hazard Estimates for Chronic Exposures

Risk assessors derive estimates of chronic non-cancer hazard for each HAP by combining the applicable exposure concentration (EC) and reference concentration (RfC) for the HAP to obtain the chronic Hazard Quotient (HQ) for the HAP using the following equation:

\[
HQ = \frac{EC_C}{RfC}
\]

where:

- **HQ** = the chronic hazard quotient for an individual HAP
- **EC_C** = estimate of continuous inhalation exposure to that HAP
- **RfC** = the corresponding reference concentration for that HAP
When calculating an HQ, it is very important to make sure that the EC and RfC are expressed in the same units. Modeled results (EC) are usually expressed in units of μg/m$^3$, while RfCs (i.e., from IRIS) are usually expressed in units of mg/m$^3$. (Note that 1 mg/m$^3$ is equal to 1,000 μg/m$^3$.)

In screening inhalation risk assessments, which are routinely built around a particular year’s estimate of emissions, the exposure estimate is usually based on an assumption of continuous long-term exposure using an annual average as the estimate of exposure concentration. A more refined assessment (i.e., by use of an exposure model) may generate an estimate of a more realistic exposure (i.e., by the application of an exposure model or refined emissions estimates over the longer time period).

Based on the definition of the RfC, an HQ less than or equal to one indicates that adverse non-cancer effects are not likely to occur, and thus can be considered to have negligible hazard. Unlike cancer risks, however, HQs greater than one are not statistical probabilities of harm occurring. Instead, they are a simple statement of whether (and by how much) an exposure concentration exceeds the RfC. Moreover, the level of concern does not increase linearly or to the same extent as HQs increase above one for different chemicals. This is because RfCs do not have equal accuracy or precision and are generally not based on the same severity of effect. Thus, we can only say that with exposures increasingly greater than the RfC, (i.e., HQs increasingly greater than 1), the potential for adverse effects increases, but we do not know by how much. For example, an HQ of 100 does not mean that the hazard is 10 times greater than an HQ of 10. Also an HQ of 10 for one substance may not have the same meaning (in terms of hazard) as another substance resulting in the same HQ.

**8.3.1 Chronic Non-Cancer Hazard Estimates from Exposure to Multiple Pollutants**

Non-cancer health effects data are usually available only for individual HAPs within a mixture. In these cases, the individual HQs can be summed together to calculate a multi-pollutant hazard index (HI) using the following formula:

$$HI = HQ_1 + HQ_2 + ... + HQ_i$$

where:

- **HI** = chronic hazard index
- **HQ** = chronic hazard quotient for the $i^{th}$ HAP
For screening-level assessments, a simple HI may first be calculated for all HAPs. This approach is based on the assumption that even when individual pollutant levels are lower than the corresponding reference levels, some pollutants may work together such that their potential for harm is additive and the combined exposure to the group of chemicals poses greater likelihood of harm. Some groups of chemicals can also behave antagonistically, such that combined exposure poses less likelihood of harm, or synergistically, such that combined exposure poses harm in greater than additive manner. Where the overall HI exceeds the criterion of interest, a more refined analysis is warranted.

Exhibit 8-4 is an example calculation to estimate chronic non-cancer hazard (hypothetical). In this example, a Tier 1 modeling analysis was performed to estimate chronic non-cancer hazard to the maximum exposed individual, assumed to reside at the point of maximum concentration for ABC Factory. Four HAPs were potentially of concern: benzene, dichloroethyl ether, formaldehyde, and cadmium compounds. Non-cancer hazard estimates were obtained for each HAP by dividing the estimated Exposure Concentration (EC) by the Inhalation Reference Concentration (RfC) for each HAP (note that the EC is expressed in units of mg/m3 for this analysis). The resulting Hazard quotient (HQ) estimates ranged from 1×10^{-3} (formaldehyde) to 1 (cadmium compounds). Note that no RfC was available for dichloroethyl ether. The HQs for each HAP were summed to obtain an estimate of the Hazard Index (HI) of 1. Note that cadmium compounds account for 95 percent of the HI, suggesting that the other HAPs may not need further consideration (although this determination should be made in consideration of all relevant information, including uncertainties such as confidence in the exposure concentration and uncertainty factors used to derive each RfC).

**Exhibit 8-4: Example - Hazard Index Calculation for Chronic Non-cancer Hazards**

<table>
<thead>
<tr>
<th>HAP</th>
<th>EC (mg/m³)</th>
<th>RfC (mg/m³)</th>
<th>HQ</th>
<th>% of HI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene</td>
<td>6 × 10^{-4}</td>
<td>6 × 10^{-2}</td>
<td>1 × 10^{-2}</td>
<td>1 %</td>
</tr>
<tr>
<td>Dichloroethyl Ether</td>
<td>5 × 10^{-3}</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Formaldehyde</td>
<td>4 × 10^{-4}</td>
<td>1 × 10^{-2}</td>
<td>4 × 10^{-2}</td>
<td>4 %</td>
</tr>
<tr>
<td>Cadmium compounds</td>
<td>2 × 10^{-3}</td>
<td>2 × 10^{-3}</td>
<td>1</td>
<td>95 %</td>
</tr>
<tr>
<td>Hazard Index (HI)</td>
<td>2 × 10^{-3}</td>
<td></td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Although the HI approach encompassing all chemicals in a mixture is commonly used for a screening-level study, it is important to note that application of the HI equation to compounds that may produce different effects, or that act by different toxicological mechanisms, could overestimate the potential for effects. Consequently, it is more
appropriate to calculate a separate HI for each endpoint of concern for which mechanisms of action are known to be similar. EPA guidance documents suggest subgrouping pollutant specific HQs by toxicological similarity of the pollutants for subsequent calculations; that is, to calculate a target-organ-specific-hazard index (TOSHI) for each sub-grouping of pollutants. This calculation allows for a more appropriate estimate of overall hazard.

Segregation of hazard indices by effect and mechanism of action can be complex and time-consuming because it is necessary to identify all the major effects and target organism for each chemical and then to classify the chemicals according to target organ(s) or mechanism of action. For example, the chemical may cause liver damage at an EC of 20 μg/m³ and neurotoxicity at an EC of 50 μg/m³. This analysis is not simple and a toxicologist with familiarity in developing TOSHI is best suited to perform this function. If the segregation is not carefully done, an underestimate of true hazard could result.

Acute HQs are developed in the same manner as chronic HQs, with the caveat that the exposure duration associated with the exposure concentration should match the exposure duration embodied in the acute toxicity value. Whereas, summing chronic HQs to a total hazard index is a relatively straightforward exercise, the issues related to developing acute HI are more subtle and complex. A toxicologist familiar with acute exposure and risk analysis should be consulted to perform this process.

8.4 Quantifying Risk from Background Sources

In some cases, it may be appropriate to quantify background concentrations of the air toxics of concern. For example, background concentrations may be a critical element in determining the need for further reductions of emissions from a particular source. Background concentrations are the levels of contaminants that would be present in the absence of contaminant releases from the source(s) under evaluation. Background concentrations may occur naturally in the environment or originate from other human sources (i.e., an industrial area upwind from the sources of concern).

A detailed analysis of background concentrations typically would require extensive data gathering and modeling beyond that required for the incremental risk analysis. For example, numerous nearby (and possibly distant) air toxics sources of varying types would need to be characterized in sufficient detail to support release and exposure modeling. The data needs for assessment of background concentrations may differ depending on what will be done with the data. For example, if the question is simply “what is the risk to the population in a specific place,” then an assessment of background
may be unnecessary (monitoring data in the study area may be all that is required). On the other hand, if the question is “what is the risk and what can we do about it,” then knowledge of how much risk is contributed from both local and background sources may be necessary. If the risk is unacceptably high, but most of the risk is background in nature, there may be no appropriate risk reduction strategy (especially in regard to local sources).

In general, the most appropriate way to evaluate the contribution of background concentrations to the risk estimate is to simply compare the risk attributable to known or estimated (i.e., through monitoring) background concentrations in a bar chart against the risk attributable to the source(s) being evaluated. It generally is not appropriate to subtract background concentrations from monitored values.

8.5 Presenting Risk Results

Different graphical presentations can help to effectively convey the risk characterization results to the risk management team members and others in ways particularly suited to the goals and purpose for the overall analysis. Pie charts, bar charts, tabular formats, and other methods that show risk contributions of different sources can be used. Presentation using graphical interfaced systems (GIS) formats is particularly useful.

For example, the RAIMI Risk-MAP tool can be used to depict both the risk across the study area as a whole or can zoom in to display what is predicted at smaller geographic scales. Exhibit 8-5 illustrates how an analyst has used this tool to focus on one specific neighborhood (Greenbriar) for emphasis. The dots represent the modeling nodes across the neighborhood and the risk results have been highlighted in a box to the side. For this neighborhood, the analyst has decided to display the average risk (i.e., the average risk and hazard across all the modeling nodes) along with relevant demographic data. The analyst could have chosen to display information for this neighborhood in a number of other ways, including information about risk variation across the modeling nodes (i.e., highest to lowest) or providing risk estimates for different segments of the population (i.e., if an exposure model has been used). The way in which the analyst chooses to display the information will depend on the message that is trying to be communicated.
Another important method for displaying risk is graphic presentation of risk “isopleths” to represent study area potential risk gradients. However, analysts need to carefully consider how to select “breaks” in the data (i.e., what risk value they will use to show contour lines) since it is easy to create different impressions about the meaning of the data depending on the way the data breaks are chosen. When using this type of presentation format it is particularly important to clarify there is no risk without the presence of people and a completed exposure pathway. In other words, depicting an isopleth implies risk at every point within the contour lines. It is only when people are present and contacting contaminated air, however, that risk is actually a possibility.

An example of a figure depicting risk isopleths from several modeled sources is provided in Exhibit 8-6. This hypothetical example illustrates cancer risk isopleths from the combined impact (all air toxics, all sources) of study-area stationary sources (major and area sources). The mobile sources were modeled two different ways. The study-area secondary roads were modeled by allocating mobile emissions uniformly across the study area. This allows the addition of the secondary road impacts to the overall cumulative risk. However, by allocating the emissions evenly over the entire study area, the detail of impacts in the immediate vicinity of any particular secondary road is lost. The major highway in the lower part of the figure, on the other hand, was modeled as a “linked source” (i.e., breaking the length of the highway up into short segments [links] and
modeling each segment as an individual source). This allows the analyst to provide additional detail about the risk posed in the immediate vicinity of that one roadway.

**Exhibit 8-6: Example of Cancer Risk Isopleths from All Air Toxic Sources Combined**

8.6 Characterization of Assumptions, Limitations, and Uncertainty

In the final part of the risk characterization, risk assessors commonly present estimates of health risk in the context of uncertainties and limitations in the data and methodology. Exposure estimates and assumptions, toxicity estimates and assumptions, and the assessment of uncertainty are usually discussed. Additionally, information relevant to the public health context of the estimated risks is presented.

The risk characterization document should allow the risk manager and the public to know why risk was assessed the way it was, by clearly summarizing the available data and its analysis, uncertainties, alternative analyses, and the choices made. A good risk characterization will state the scope of the assessment, express results clearly, articulate major assumptions and uncertainties, identify reasonable alternative interpretations, and separate scientific conclusions from science policy judgments. EPA’s “Policy for Risk Characterization” calls for the explanation of the choices made to be highly visible.
The goal of risk characterization is to clearly communicate the key findings and their strengths and limitations so that decision-makers can put the risk results into context with other information critical to evaluating risk management options (i.e., economics, social values, public perception, and policies). The risk characterization will provide a means of placing the numerical estimates of risk and hazard in the context of what is known and what is not about the potential exposures.

During the course of a risk assessment, a number of assumptions may have been made and used in the development and analysis of the conceptual model, particularly when significant data gaps exist that require a parameter value for the risk assessment to proceed. For example, meteorological data for a specific neighborhood may not have been available so analysts decided to use data from a nearby airport instead. Based on an understanding of the local meteorology, the analysts may have assumed that the airport data was sufficiently representative of the study area to use without question. All major assumptions made throughout the analysis should be thoroughly documented. Readers of the final report should be able to understand why an assumption had to be made, how it was made, why the assumption was appropriate for the analysis at hand, and the potential influence of the assumption on the final risk estimates.

At the end of the risk characterization, the assessors will have developed both quantitative and qualitative expressions of risk. It is important for the analysts to carefully articulate any important limitations associated with those values. For example, if the risk characterization is performed at the county-level, the results should only be used to make statements about risks at the county-level (i.e., it might be inappropriate to try and extrapolate the results to a finer geographic resolution). As another example, if small, diffuse sources are evaluated in the aggregate, then it might not be possible to draw any conclusions about individual sources in specific locations.

The final discussion of the risk characterization results must place the numerical estimates of risk in the context of the uncertainties inherent in the analysis. Uncertainty, within the context of the risk assessment process, is defined as “a lack of knowledge about specific factors, parameters, or models.” When applied to the results of a risk assessment, the term “uncertainty” refers to the lack of accuracy in the risk estimate due to unknown values or unavoidable errors in the input assumptions, models and parameter values. Accordingly, one of the key purposes of uncertainty analysis is to provide an understanding of where the estimate of exposure and risk falls within the range of possible values. The discussion should include:

- Level of confidence in the quantitative toxicity information used to estimate risks
• Presentation of qualitative information on the toxicity of substances not included in the quantitative assessment

• Level of confidence in the exposure estimates for key exposure pathways and related exposure parameter assumptions

• Major factors reducing certainty in the results and the significance of these uncertainties (e.g., adding individual risk estimates for several substances or across multiple exposure pathways)

• Possible graphical presentation of key parameter and risk uncertainties
References


USEPA “Air Toxics Risk Assessment Reference Library, Volume III Community-Scale Assessment” (Chapter 6), EPA 452/K-06-001C, April 2006.


Chapter 9: Toxic Torts: Risk Assessment in the Courtroom

9.1 Introduction

In toxic tort cases, lawyers must explain to the court or jury the causal link between his client’s exposure to a toxic chemical and the adverse health effect that his client has suffered. These lawyers will now stand at the intersection of tort law and science. He must research (with the help of experts) generally-accepted scientific knowledge about the causal relationship between the toxic chemical exposure and the specific medical condition that has harmed his client. The lawyer and his experts will study any available epidemiological evidence. They may conduct a risk assessment or study available risk assessment data to help them establish the causal relationship between a toxic chemical and harm suffered.

9.2 Toxic Torts

What are toxic torts? Toxic torts involve some claim of harm, physical or psychological, caused by exposure to a substance. The substance is usually a toxic chemical. Veterans of the Vietnam War were exposed to Agent Orange, a toxic herbicide which contained low levels of dioxins and was used to clear forests in Vietnam. During the early 1980s, many of these veterans filed toxic tort lawsuits against the U.S. for their serious range of diseases that resulted from this exposure.

During the later part of the 20th century, asbestos litigation became the predominant toxic tort in the United States. During this century, asbestos was commonly used in many manufactured products. According to “Toxic Tort Litigation,” “by 1970, asbestos was found in theater-curtains, ironing board covers, floor and ceiling tile, wallboard, joint compound, cement pipe, gaskets, spray-on insulation products, brake linings, gas mask filters, protective clothing for welders, hair dryers, and even in the “micronite” filters once attached to Kent cigarettes.” At this time, asbestos-related diseases were starting to become evident. Workers in shipyards during World War II, workers in the post war construction boom, and workers at varying industrial facilities had all been exposed asbestos in their occupations. These workers now were filing toxic tort claims against manufactures that used asbestos in their products.

Toxic torts are very different from other forms of tort claims. This is because of the characteristics of toxic torts. Toxic torts have at least four of the following common characteristics:
- Large number of plaintiffs and defendants (but serious injuries to a single plaintiff are not uncommon)

- Difficulty in identifying the source or product causing the harm and qualifying the portion of harm from each source (i.e., multiple air toxics emitted from one plant or many plants; numerous toxic chemicals from multiple sources contaminating a drinking water supply)

- Use of complex litigation procedures (i.e., the court may bifurcate the case forcing plaintiff to demonstrate evidence of exposure and causation before the jury [or court] gets to see the severity of plaintiff’s injury and possibly even before plaintiff initiates discovery); and

- Reliance on scientific concepts to resolve causation issues (i.e., the need for experts in epidemiology, hydrology and toxicology are common to translate complex scientific concepts).

9.2.1 Plaintiff’s Burden of Proof

In a toxic tort case, plaintiff has the burden to prove that he was harmed, that the exposure to the toxic chemical caused his harm, and that defendant(s) was liable for creating the exposure. A toxic tort plaintiff has several hurdles to overcome in his burden of proof. For example, plaintiff’s harm may be a serious injury that has been manifested, but the level of his exposure to the toxic chemical may be unverifiable. Or maybe, the exposure to the toxic chemical is known, but the injury has not yet been manifested because of a long latency period between the injury and exposure (i.e., exposure to asbestos). To resolve this latter problem, most state courts and all federal courts use the “discovery rule.” This rule will toll the statute of limitations until plaintiff’s injury has been discovered (manifested).

Causation (that exposure to a toxic chemical caused plaintiff’s harm) is the most difficult and complex element of plaintiff’s burden of proof. Causation is the major battleground in toxic tort litigation. Proving that defendant(s) was liable for plaintiff’s exposure to a toxic chemical is also difficult. Plaintiff must identify all liable defendants, show how they are liable for plaintiff’s exposures, and choose a theory of liability (i.e., negligence, nuisance, strict liability).

An example of the difficulty plaintiffs will encounter in their burden of proof is shown in the following Woburn, Massachusetts toxic tort lawsuit. In this case, drinking wells contaminated with trichloroethylene (TCE) caused leukemia in twelve children (among other adverse health
Local attention about a toxic problem in Woburn was first publicly noted by Charles Ryan, a *Daily Times* reporter. He completed a story concerning a study of cancer mortality by the state’s department of public health. The story stated that in Woburn, “deaths from all cancers had increased by 17 percent during a five-year period in the mid-1970s. The incident of leukemia in particular, and to a lesser extent kidney cancers, was alarming.” A subsequent Harvard Health Study suggested that water from Woburn Wells G and H were linked to a variety of adverse health effects including a high rate of childhood leukemia. Plaintiffs’ attorney, Jan Schlichtmann, was delighted and yet disturbed with the Study. The Study did not address the biological causes of leukemia. “It showed only that those children who drank from the Wells G and H were more likely to get leukemia than those children who did not.” Schlichtmann now had an epidemiological study, but knew he had a significant “general” causation problem. He had to prove that TCE was a cause of childhood leukemia. At that time, EPA had listed TCE as “probable” carcinogen on the basis of animal studies only (no human studies). Courts are reluctant to find causation on animal studies alone. There was an available epidemiological study conducted on thirty dry-cleaning workers (exposed to TCE & perchloroethylene at their workplace) that showed significant increases in several different types of cancer including five leukemia cases. This study and the Harvard study helped reduce plaintiff’s “general” causation problem and avoid a summary judgment for defendant.

Schlichtmann also had a “specific” causation problem. He had to establish a relationship between how much (exposure and dose) of the TCE was needed to cause plaintiffs’ leukemia deaths. There was a discrepancy between the severity of their symptoms and the low levels of exposure. To account for this, Schlichtmann and his toxicologist looked at possible “routes of exposure” that the families could have been exposed to TCE. The families could have been exposed to TCE in three different ways: by drinking the water (ingestion), by breathing the vapor (inhalation by breathing vaporized TCE when taking showers), and by absorbing TCE through the skin when bathing. When he took all these “routes of exposure” into consideration, he found that “those levels of exposure were … not so low after all.”

Schlichtmann had more than causation problems. He had to identify the sources of the contaminants and prove they were liable for the TCE exposure that caused the leukemia cluster in Woburn. Through discovery Schlichtmann identified two deep-pocket defendants, W.R.
Grace and Riley Tannery, a Division of Beatrice Foods, as the source of TCE groundwater contamination. Schlichtmann had to prove that the chemicals (mainly TCE) were dumped or released from Grace and Beatrice and had actually gotten to the Woburn wells. He had to obtain records and witnesses that would testify to the use and dumping of TCE by the defendants (this was a major weakness in his case). Schlichtmann hired engineers that “drilled thirty new monitoring wells … to plot the underground flow of the contaminants.” The study of the underground flow was a TCE “fate and transport” study. It would show the movement of TCE in the groundwater. This was very expensive to accomplish, but was needed to prove that the TCE in Wells G and H was the result of defendants’ dumping.

9.2.2 Causation Components

In all toxic tort cases there are three fundamental causation components: exposure and dose, general causation, and legal causation.

- **Exposure and dose:** The magnitude and duration of plaintiff’s exposure and that defendant(s) is the source of this exposure must be identified. Also, the actual dose received by plaintiff needs to be identified. Remember, that just because a person is exposed to a toxic chemical does not mean they will actually have a dose in their body. The human body has defenses against toxic agents (i.e., liver and kidneys break down chemicals to less toxic form).

- **General Causation:** Plaintiff must prove that exposure to a particular toxic chemical is capable of causing the identified harm in plaintiff. This is where the generally accepted scientific knowledge between exposure and harm must be shown. Epidemiological studies and dose-response animal studies are thoroughly researched and utilized with the help of an expert in toxicology.

- **Specific Causation:** Plaintiff must prove how much of the toxic chemical was plaintiff exposed to and for how long.

9.2.3 Special Causation Challenges

*Long latency periods* make it difficult for plaintiff to establish a connection between exposure and harm suffered. Sometimes in toxic tort cases there is a long latency period from exposure to manifestation of plaintiff’s injury. Examples would be lung cancer from cigarette smoking and asbestosis from asbestos exposure. Earlier plaintiffs were denied their day in court because they
did not discover their disease before the statute of limitations barred their action. Today most courts have responded with the “discovery rule” (discussed earlier).

Another causation problem is when plaintiff’s injury can be caused by exposure to other chemicals in which defendant is not liable. An example of this took place in the Woburn case during the depositions of Woburn family plaintiffs. For three weeks the defendants’ attorneys questioned the families “about their use of more than five hundred brand-name household products – cleaning agents and detergents, rug shampoos, cosmetics, nail-polish removers, insect repellents, paints, lawn fertilizers, cold remedies, cough syrups, herbal tea, coffee, and even peanut butter. To Schlichtmann, the strategy … was obvious. These five hundred items all allegedly contained a known or suspected carcinogen.”

A final causation problem is that there is only a little hard scientific data linking toxic exposure to human injury. According to “Toxic Tort Litigation,” in toxic tort cases the injury is to humans not animals. For this reason, it should be human scientific studies not simply dose-response animal studies or in vitro studies that are used to establish the “general” causation connection.

9.2.4 Admissibility of Expert’s Opinion

In 1923, the standard for admissibility of scientific evidence in federal courts was established by a federal appellate court decision in Frye vs. U.S. The “Frye” Rule stated that scientific evidence must be “generally accepted” in the scientific community. Before 1993, federal courts were liberal toward admissibility of an expert’s opinion on scientific evidence and left it up to a jury to sort it out. Expert opinions were allowed with no scientific consensus by professional publications or an expert’s peers. Juries were making conclusions on unresolved scientific issues based on pioneered opinions. This all changed in 1993 as a result of the Supreme Court decision in Daubert vs. Merrell Dow Pharmaceuticals. As a result of Daubert, the trial judge is now a “gatekeeper” and must assess reliability of the expert’s testimony to determine admissibility of expert opinion on scientific evidence. The judge must consider the following four factors in determining the reliability of a particular scientific theory or technique:

1. Testability (whether the scientific theory or technique is capable of being repeated and verified)

2. Whether the scientific opinion has been published after peer review
3. The known or potential error rate of the technique

4. Whether the opinion has been generally accepted in the relevant scientific community

9.2.5 Common Theories in Tort Liability

Well-established tort theories are applied to modern toxic torts in a strained endeavor. Some of these common theories of tort liability are negligence, nuisance, trespass, and strict liability.

- **Negligence**: To state a negligence claim, a plaintiff must show that defendant had a “duty” to conform to certain standard of conduct, and that defendant violated this duty which caused plaintiff to be harmed. (i.e., a chemical plant has a duty to prevent releases of toxic chemicals that can harm nearby residents and plant workers.)

- **Nuisance**: Defendant’s action caused an “unreasonable interference” with plaintiff’s use and enjoyment of his land. A mere sensory effect is sufficient (i.e., taste and odor of MTBE in plaintiff’s water is actionable).

- **Trespass**: Defendant causes an invasion to plaintiff’s land. (i.e., In Martin vs. Reynolds Metals Company, 1959, defendant released fluoride particles in the air causing plaintiff’s cattle to die. The court held that even though the particles were invisible, defendant was liable.)

- **Strict Liability**: Defendant’s use of “abnormally dangerous activity” causes harm to plaintiff regardless that defendant used the utmost care to prevent the harm. The defendant is strictly liable for his conduct without any proof of fault required; (i.e., in 1957, the Louisiana Supreme Court imposed strict liability for property damage caused by aerial spraying of herbicides and the resulting drifting of chemicals.) In 1963, the California Supreme Court extended “strict liability” to a seller of a defective product for a product-related injury (now used in asbestos cases).

9.2.6 Special Toxic Tort Cases

**Asbestos**: Exposure to asbestos can cause asbestosis, mesothelioma, and lung cancer. To establish lung cancer it is usually necessary to have preexisting asbestosis (i.e., in many “smoking lung cancer” cases, where plaintiff did not have asbestosis, juries found that cigarettes were the cause - not asbestos). The latency period between exposure and asbestos-type diseases can be 10 to 40 years - depending on exposure & sensitivity of the individual. This resulted in
early asbestos cases being barred by the statute of limitations. Until 1960s, workers compensation was the principle remedy for occupational asbestos exposure, consequentially causing plaintiffs to receive inadequate compensation for their injuries. In 1969, the courts started to allow strict liability actions in asbestos cases, thereby causing the floodgates to asbestos litigation to swing open.

Between 1940 & 1979 up to 27.5 million Americans worked in occupations where substantial asbestos exposures were common. Proliferation of asbestos litigation continued into the 21st century, and Congress was not taking any action to shut the floodgates. By 2001, more than 600,000 people filed asbestos lawsuits, 6,000 companies have been named as defendants, and 60 companies have filed bankruptcy (Johns-Manville in 1982). Defendants and their insurers had paid approximately 54 billion dollars to resolve claims through 2000. Claimants received 21 billion dollars (most of this money went to non-functionally impaired plaintiffs). As a result of defendants’ financial loss, 138,000 jobs were not created. To date, Congress has failed to take any action to resolve the proliferation of asbestos litigation. In 2005, Senator Spector sponsored a bill that would take asbestos injury claims out of court and create a 140 billion dollar trust fund. The bill did not pass because of the lack of consensus over fundamental aspects of the bill.

*Mold:* Although the majority of molds are harmless (over 100,000 types), molds have the potential to cause health problems. Molds produce allergens (substances that can cause allergic reactions), irritants, and in some cases, potentially toxic substances called mycotoxins. (Extreme exposure to very high levels of mycotoxins can lead to neurological problems and in some cases death; fortunately, such exposures, are extremely rare even in residences with serious mold problems.) Harmful mold exposure can cause nausea, fatigue, sore throat, asthma, and other respiratory difficulties.

There are two main types of mold cases: those concerning *property damage* and those for *personal injury*. Plaintiff can use numerous liability theories, such as breach of contract, breach of warranty (i.e., construction cases), or negligence (i.e., landlord has a duty to maintain a safe premise). In proving his case, plaintiff must show that the amount and location of mold resulted in exposure to cause plaintiff’s adverse health effects. When comparing mold litigation to asbestos litigation, mold will never rise to the level of concern as asbestos. Unlike asbestos, mold is not scientifically linked to a clearly mold-caused disease and rarely causes irreversible injury and/or death. Also, asbestos defendants are usually large manufacturers with “deep pocket,” whereas, mold defendants are simply landlords or builders. Concerning issuance coverage issues, today many insurance policies will exclude mold claims.
Methyl-tertiary-butyl-ether (MTBE): MTBE is a fuel additive used to raise fuel octane rating or as a fuel oxygenate. The 1990 CAAA required the use of oxygenated fuels for motor vehicles in carbon monoxide non-attainment areas. MTBE and ethanol are commonly used as fuel oxygenates. The CAAA also required the use of reformulated gas (RFG) in severe ozone non-attainment areas (required oxygen content in fuel greater than 2%). Problems occur when drinking water supplies (groundwater) becomes contaminated with MTBE from leaking underground storage tanks. Defendants are usually the refineries or distributors of fuel containing MTBE (viewed as defective product). Concerning the health effects, EPA has rated MTBE as a “possible human carcinogen” based on animal inhalation studies. In an advisory document, EPA has stated that there is little likelihood that MTBE concentrations between 20 & 40 micrograms per liter would cause an adverse effect in drinking water. MTBE is highly soluble and when it has contaminated water, it has a strong taste and smell. Thus, even small amounts of MTBE in water make the water undrinkable. Today, twenty-three states have banned or restricted MTBE as a fuel additive in motor vehicles. The U.S Energy Policy Act of 2005 does not ban MTBE, but will reduce its demand by eliminating the CAAA, reformulated gas requirement (that it must contain at least 2% oxygen by weight) and mandating the increase usage of renewable fuels like ethanol.
References


Chapter 10: Air Toxics Monitoring

10.1 Introduction

In environmental investigations, the term “monitoring” describes the collection of actual samples of environmental media and then subjecting those samples (usually) to chemical analysis to determine the identity and concentration of the various pollutants in the sample. A distinction may be made between sampling (i.e., stack testing) and monitoring (i.e., for ambient concentrations). Ambient monitoring samples at relatively low pollution concentrations and is used to measure pollution concentration in the ambient air. Whereas, stack testing samples at relatively high pollution concentrations and is used to measure pollutant concentration in the source’s stack exit gases.

Chapter 10 outlines EPA’s Air Toxics Monitoring Program, explains reasons for monitoring air toxics, gives insight on how to plan an air toxics monitoring program (while explaining important air monitoring terminology), discusses factors to consider in locating air monitors, and summarizes monitoring “methods” for air toxics that are available on EPA’s Ambient Monitoring Technology Center (AMTIC) web site. (Note: Selections from Chapters 10 of EPA’s “Air Toxics Risk Assessment Reference Library, Volume I Technical Resource Manual” have been edited and are used in this Chapter.)

10.2 EPA’s Air Toxics Monitoring Program

Since the passage of the 1990 Clean Air Act Amendments, there are now 187 hazardous air pollutants (HAPs) that need to be monitored. Because of the impracticality of monitoring all these HAPs and the prohibitive costs, EPA has begun monitoring the air toxics that pose the most risk to human health in its new Air Toxics Monitoring Program.

The Clean Air Act (CAA) does not require a national air toxics monitoring network, but EPA and state and local agencies have recognized such a network is needed. The Urban Air Toxic Monitoring Program (UATMP) was initiated by EPA in 1987 to meet the increasing need for information on air toxics. The program was intended to allow participating agencies to screen air samples for concentrations of air toxics that could potentially result in adverse human health effects. The program has allowed the identification of compounds that are prevalent in ambient air and the identification of emission sources likely contributing to existing concentration levels. Since 2000, EPA has significantly increased its ambient air toxics monitoring efforts and funding to establish a national network and support state and local agencies’ monitoring activities. In
2004, EPA began awarding grants to state and local agencies to conduct short-term, local-scale monitoring projects. Also in 2004, EPA published “National Air Toxics Monitoring Strategy” that describes the structure of the national air toxics monitoring program, including its history, status, and expected products. At the start of the program, EPA’s focus was on “nationwide pervasive” priority pollutants. In recent years, EPA has initiated local scale monitoring studies to address potential air toxics problem areas. EPA’s air toxics monitoring program is structured into four groups:

- National level
- Local scale
- Persistent Bio-accumulative Toxics (PBTs)
- “Other” EPA-specific monitoring programs existing prior to this Program

**National Air Toxics Trends System (NATTS):** The NATTS network was created to generate long-term ambient air toxics concentration data at specific fixed sites across the country. The NATTS Pilot program was developed and implemented during 2001 and 2002, leading to the development and initial implementation of the NATTS network during 2003 and 2004. The (NATTS) program is a network of monitoring stations at 27 urban or rural locations across the country (see Exhibit 2-10 in Chapter 2 for locations). The trends sites are evaluated regularly to assess their effectiveness in characterizing trends and assessing concentration levels. If a given site is determined to no longer be useful for trends (or other) purposes, then it may be discontinued or relocated.

Because of the large number and variety of the 188 hazardous air pollutants (HAPs) specified in the CAA, it is not practical to measure all 188 HAPs at all locations. It was decided to begin by evaluating the List of 33 Urban HAPs. From this List, candidates for the NATTS Program were selected and are presented in Exhibit 10-1 (20 HAPs listed). Six of the 20 entries must be monitored from the initiation of NATTS because these entries are the major risk drivers based on a relative ranking performed by EPA. The remaining 14 entries must be reported to NATTS if the corresponding methods are being conducted at the site. These HAPs can be grouped into several general categories, which include volatile organic compounds (VOCs), metals, aldehydes, and semi-volatile organic compounds (SVOCs).
### Exhibit 10-1: NATTS Monitored HAPs (*major risk driven HAPs*)

<table>
<thead>
<tr>
<th>VOCs</th>
<th>Metals</th>
<th>Aldehydes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,3-butadiene *&lt;br&gt;carbon tetrachloride&lt;br&gt;chloroform&lt;br&gt;1,2-dichloropropene&lt;br&gt;methylene chloride&lt;br&gt;tetrachloroethylene&lt;br&gt;trichloroethylene&lt;br&gt;vinyl chloride&lt;br&gt;benzene *</td>
<td>Arsenic *&lt;br&gt;beryllium&lt;br&gt;cadmium&lt;br&gt;hexavalent chromium *&lt;br&gt;chromium (and compounds)&lt;br&gt;lead&lt;br&gt;manganese&lt;br&gt;nickel</td>
<td>Acrolein *&lt;br&gt;Formaldehyde *&lt;br&gt;Acetaldehyde</td>
</tr>
</tbody>
</table>

### Local-Scale Monitoring Studies: These studies complement the NATTS by allowing flexible approaches to address a wide range of air toxics issues. They are intended to probe potential problem areas throughout the nation that may require subsequent attention with respect to more dedicated monitoring and aggressive emission mitigation strategies. In some instances, these studies will be used to better characterize impacts of diesel emissions, or to define spatial concentration patterns throughout an area that simply are not achievable with a single NATTS site. Local scale monitoring studies are short term (less than 2 years) and have more flexible study requirements to go beyond the scope of the NATTS. Local-level studies provide information of urban/local interest that is not achievable with a single monitoring site at a city. For example, these studies may address specific source categories or better characterize pollutant levels associated with different locations in a metropolitan area.

### Persistent Bio-accumulative Toxics (PBTs) Monitoring Program: This program primarily consists of deposition monitoring, not ambient air monitoring. Several monitoring programs operated by various federal agencies have been established to measure the presence of toxics in various media (i.e., water, fish tissue). These toxics impact human health through multiple exposure pathways, with exposure through ingestion generally presenting a greater health risk than exposure from inhalation. Programs that monitor atmospheric deposition of PBTs include:

1. The National Atmospheric Deposition Program – Mercury Deposition Network (NADP – MDN), a multi-agency program with approximately 90 monitoring sites
2. The Integrated Atmospheric Deposition Network (IADN), a partnership between EPA and Canada, which is measuring PBTs in the Great Lakes Region
3. The National Dioxin Air Monitoring Network (NDAMN), a 30-site research program
EPA Regional air toxics monitoring activities that existed prior to NATTS: These programs will continue. Based on information provided by state and local air pollution control agencies across the country, air toxics monitoring data are being collected at over 300 locations for a number of compounds (see Exhibit 2-10 in Chapter 2 for locations). These sites are currently collecting data to help air pollution control agencies track toxic air pollutant levels in various locations around the country. State, local, and tribal air quality agencies operate these sites for various different purposes: some to address concerns such as areas of elevated concentrations, or “hot spots,” assessment of trends; characterization of air toxic levels, investigation of source-specific (compliance related) issues; and support of risk assessments.

10.3 Reasons for Monitoring Air Toxics

The following are a list of reasons for monitoring air toxics:

- Identify and estimate current exposures to ambient concentrations of air toxics (outdoor and/or indoor) at a specific location of concern (i.e., a school or neighborhood). For example, air toxics monitoring can be used to evaluate the impacts of a specific source on a nearby receptor.

- Validate the predictions of a model in specified circumstances (i.e., validate that the location of highest exposure predicted by the model is correct, which increases confidence that a maximally exposed subpopulation has been identified – may be difficult to do without a very dense monitoring network).

- Track trends in air quality levels (i.e., to determine whether air pollution programs have generally been effective at reducing exposures).

- Identify gaps in emissions inventories (i.e., monitoring identifies an airborne chemical that is not reported in existing emissions inventories) or close gaps that might be present in existing data (i.e., concentrations of specific air toxics in specific releases).

- Determine compliance with air toxics legal requirements (i.e., permit limits at a factory, emissions limitations on motor vehicles).

- Gather data in support of enforcement actions.
10.4 Planning an Air Toxics Monitoring Program

Planning an air toxics monitoring program involves a step-wise integration of sampling protocols with data quality criteria and data analysis processes that are consistent with the study-specific, conceptual model (CM), quality assurance project plan (QAPP), and data quality objectives (DQO) processes.

A QAPP is part of the overall risk assessment analysis plan that ensures the quality of data used in decisions. Generally included in the data quality program is the DQO process, which establishes the criteria that must be met if data are to meet the needs of a decision-maker (i.e., it establishes the error bounds on data, which are related in turn to the uncertainties a decision-maker can tolerate in reaching a defensible decision). The QAPP specifies precisely how to collect and analyze the data to meet the goals established by the DQO process. The QAPP establishes specific procedures that assessors follow to meet DQOs. These DQOs include procedures for identifying reliable methods, choosing sample locations and frequencies, handling samples, calibration of equipment, recording and archiving of data, and analysis of the data.

The following lists the steps for planning an air toxics monitoring program:

1. **Understand the problem.** Assessors may design monitoring programs to support a number of different types of management decisions. For risk assessments, the CM can focus participants’ understanding of both the scope and the breadth of the problem that the sampling and analysis are to address. The most important questions to answer immediately are: whether assessors will use monitoring results to characterize exposure and risk, whether they will use results to evaluate air quality model performance and look for gaps in the emissions inventory, or whether they will use results for both reasons. These are critical questions for participants to answer, because the data needs can be drastically different, depending on how the assessors will use the monitoring data.

2. **Identify existing data.** Sampling and analysis for risk assessment may not be necessary if the information to be developed is already available from other sources and meets the quality requirements for decision making.

3. **Itemize data needs.** Where existing data are insufficient to answer the study-specific questions, it will be necessary to obtain new data through monitoring. Potential data needs include: filling gaps in emissions inventory data; providing input data for models and validating modeling results; generating new data to more fully characterize exposures in areas, populations, or pathways; establishing trends over time; or supplementing a
body of data to increase their quality for the risk management decision. The process for itemizing data needs includes articulating critical decision criteria (which may drive data quality needs and/or selection of specific methods), applying these criteria to determine areas where existing data are insufficient, and identifying the manner in which new data can supplement existing data to meet the decision criteria.

4. **Define data quality needs.** The reliability (i.e., accuracy and precision) of monitoring results must be adequate to meet the needs of the risk management decision. However, given finite resources, even well-designed studies may not be able to achieve all quality criteria. That limitation makes it important to determine which criteria are essential for addressing the study-specific, decision problem and for focusing resources on meeting (and not necessarily exceeding) those criteria.

The DQO process determines general data quality objectives to meet specific needs. This process can be informed by a sensitivity analysis to determine which aspects of a monitoring program will require the greatest attention and resources. A common approach is to consider all aspects of sample and data handling from collection to data report writing, as these affect the confidence with which decisions can be made through the introduction of random or systemic errors. A number of factors affect data quality, including bias related to sampling error (i.e., taking only a single sample at one location, which may or may not be representative of actual ambient concentrations) and relative precision related to analysis methods.

5. **Select monitoring methods to meet data quality needs.** The choice of monitoring method depends on the scale of the assessment, specific contaminant(s) to be analyzed, the sampling time over which the result is derived (i.e., a sample collected over 15 minutes versus a sample collected over 24 hours), the decision criteria or other reporting limit needs, and the resources available. Methodologies include the sampling methods and techniques, sampling program design (i.e., sampling frequency, coverage, and density), as well as analytical methods.

6. **Develop systems to ensure that data meet decision requirements.** Setting the objectives and selecting sampling and methods capable of meeting the DQOs are the prelude to determining whether and to what degree the data may support risk management decisions. Having collected and analyzed the data, it will be necessary to determine whether decisions can now be made with the desired confidence. For example, the actual data collected must be assessed for quality and compared against any decision criteria such as toxicity dose-response values. Where the quality is insufficient to support
the decision (i.e., insufficient to determine whether the benchmark is or is not exceeded), the previous steps may need to be re-assessed.

7. **Develop documentation.** The QAPP and other planning documents must record the results of the environmental data collection design process. Information to be documented includes the assumptions, findings, outliers, biases, data confidences, and other factors that are critical to implementation, as well as evaluation and eventual interpretation of the data collected. Data collected and analyzed is often reviewed thoroughly to ensure they are adequate to support decisions; sufficient documentation allows such a review.

### 10.4.1 Selecting Locations of Air Toxics Monitors

Determining the location of an air toxics monitor depends on a number of factors, including the specific purpose of the monitoring (i.e., confirm modeled concentrations at a specific location, estimate background concentrations), meteorological and terrain constraints, and the relative magnitude and location of the source(s) of concern versus other emissions sources that might contribute to measured air concentrations. For example, locations too close to a source may underestimate exposure concentrations if the plume has not yet reached ground level where people can come into contact with the contaminant. Locations too far from the source may also underestimate exposure concentrations for large groups of people due to the dispersion that takes place between the point of touch-down of the plume and the point of monitoring.

EPA’s *“Quality Assurance Handbook for Air Pollution Measurement Systems”* provides a set of consistent QA practices that will improve the quality of the nation’s ambient air quality monitoring data and ensure comparability among sites across the nation. Although these practices were developed specifically for criteria air pollutants, they provide useful guidance for air toxics risk assessments. The following factors are usually considered when locating monitors:

- **Perform measurements at locations that are representative of exposure.** Determining the location will depend on whether the goal is to quantify exposures in general, or exposures to the maximally-exposed individual. In the latter case, locations too close to a source may underestimate exposure if the plume has not yet reached ground level where people can come into contact with the contaminant. Locations too far from the source may also underestimate exposure to large groups of people due to the dispersion that takes place between the point of touch-down of the plume and the point of monitoring.
**Take into account shielding and concentrating effects.** Buildings, hills, and trees can have shielding and concentrating effects. These effects may cause assessors to underestimate exposure if either measurement sites are shielded from normal air flow or if these same structures produce high concentrations downwind due to metrolological effects. Unless there is a pattern of movement of people that make sites near buildings and other structures of particular interest, assessors should perform measurements away from the influence of these structures. It is particularly important to locate monitors away from such structures if the goal is to locate sources, as the flow patterns for air are highly complex near these structures, greatly complicating the ability to identify the source location from monitoring data.

**Be aware that sources of air toxics from mobile sources (cars, trucks, etc.) can complicate measurements of ambient air concentrations produced by stationary sources.** For the estimates of exposures from stationary sources, it may be preferable to make measurements at locations away from roads. Monitoring should occur at distances ranging from 3 to 61 meters from a major traffic artery. These roads provide, in a sense, a “background” level, or noise, above which the source must rise to create a discernible signal. Of course, if total ambient exposure from all sources is to be estimated, and the exposed population spends a significant fraction of time near roads, this factor may be captured by selecting a sample of sites near those roads.

**Make sure that the heights of monitoring and sampling devices are consistent with the breathing zones of people when public exposures are being evaluated.** This is generally between 1 and 2 meters (the lower end being for children and the upper end for adults). While less important for highly dispersed gases (i.e., gases with high diffusion coefficients), this consideration can be important for heavy gases and particulates, which produce significant vertical gradients of concentration.

**Keep in mind that background concentrations can be difficult to determine.** It is important to estimate background concentrations as accurately as possible at the location of measurement. Unfortunately, even background levels can vary dramatically over time and over a geographic area, and so assessors should exercise caution in using past studies and studies from other geographic areas in establishing background for a measurement location. Meteorological and pollutant source information must also be carefully considered in selecting an appropriate background monitoring location. The location must not be near major sources of the contaminant, or in the predominant downwind direction of those sources. *Background monitors* should be placed in the predominant...
upwind direction (in relation to sources) in the assessment area to measure the concentrations of the chemicals of potential concern in air that is moving into the assessment area. Background monitoring results should not be subtracted from assessment area monitoring results because of the uncertainties in the background monitor as a truly representative measure of long-term ambient background concentrations. Instead, EPA recommends bar charts that compare contemporaneous concentrations of a chemical in a background monitor to the same chemical at assessment area monitors; these charts provide a sense of the potential influence of background concentrations on the assessment area.

Establishing sampling locations: Sampling may be purposive, random, or systematic. Purposive sampling refers to locating the monitor at a particular location because that location is of special interest. While such sampling can be useful to address specialized questions (such as the impacts of a specific source, or the reliability of model results), they generally are less useful for risk assessment purposes, and care should be taken when averaging the results along with results from the other forms of sampling. Random sampling involves selecting monitoring locations in a random and unbiased manner, with no correlation between locations (other than, perhaps, the fact that they are all in a defined region). Assessors could establish locations by creating a grid, and then randomly selecting the two coordinates (x and y) in that grid. Random sampling has the advantage of well established and relatively easy to apply statistical methods for evaluating results, but runs the risk of missing some “hot spots” of exposure. Systematic sampling involves establishing a grid and placing monitors systematically on the grid nodes. This ensures that sampling is uniform across an area, although statistical analysis is more complex because the samples are not truly random.

10.4.2 Detection Limits and Limits of Quantification

The detection limit is the minimum concentration that an analyst can reliably expect to find (i.e., detect) in a sample, if it is present. For any given method (i.e., the method to analyze for volatile organic compounds [VOCs] in air), this limit is established in each lab for each instrument and is called the method detection limit or MDL. An MDL of 1μg/m³, indicates that a field sample that contains 1 μg/m³ or below of contaminant will probably not be detected by the instrument in question. The limit of quantitation (LOQ), on the other hand, is the minimum concentration for which the analyst can reliably say that the substance is present in the sample and at a specific concentration within some pre-established limits of precision and accuracy. If the limit of quantitation is 2 μg/m³, then measurement results above 2 μg/m³ may be reported as not only indicating the presence of the substance in the sample, but as indicating the specific concentration measured (i.e., positive identification, certain concentration). Measurements
between the MDL and the LOQ, indicate the presence of the substance in the sample, but analysts can only make an estimate of the concentration (i.e., certain identification, uncertain concentration). NOTE: It is common (but incorrect) to refer to the quantitation limit as the detection limit. The LOQ, practical quantitation limit (PQL), estimated quantitation limit (EQL), and sample quantitation limit (SQL) are all limits of quantitation, not detection. Thus, when one says “benzene was not detected at a detection limit of 5 μg/m³,” this most likely actually means “benzene was not detected; the limit of quantitation was 5 μg/m³.” Likewise, when a lab reports a measurement as “<5 μg/m³,” this most likely means “not detected; the limit of quantitation was 5 μg/m³.” There is much confusion on this point and analysts must clarify with the laboratory exactly what they mean in their lab reports (and what the analyst needs to have reported to them for their risk assessment activities). For air toxics risk assessments, the MDL is largely irrelevant for purposes of estimating exposure and the limit of quantitation is the critical information that needs to be reported.

When selecting the appropriate monitoring or sampling methods for the air toxic(s) to be measured, it is important that the methods selected have the sensitivity needed to monitor at concentrations likely to be of health and/or regulatory concern. At a minimum, the PQL or SQL should be below any relevant health benchmarks (i.e., the human health dose-response values discussed in Chapter 7).

10.5 Air Toxic Monitoring Methods

10.5.1 Organic and Inorganic Air Toxics

The CAA’s 187 HAPs can be divided into two main groups: organic and inorganic compounds. They can be further divided by degree of volatility. The volatility of chemical varies with vapor pressure and varies directly with temperature (vapor pressure [in mm Hg at 25 degrees Celsius (C)] and boiling point [(in degrees C)]). The volatility classification for toxic organic compounds is as follows:

- Very Volatile Organic Compounds (VVOC)
- Volatile Organic Compounds (VOC)
- Semi-volatile Organic Compounds (SVOC)
- Nonvolatile Organic Compounds (NVOC)
The volatility classification for toxic inorganic compounds is as follows:

- Very Volatile Inorganic Compounds (VVINC)
- Volatile Inorganic Compounds (VINC)
- Semi-volatile Inorganic Compounds (SVINC)
- Nonvolatile Inorganic Compounds (NVINC)

The majority of the 187 HAPs are volatile and semi-volatile compounds; most of the remainder are metal compounds. Exhibit 10-2 shows the amount of HAPs in each volatility classifications, and also also shows the range of vapor pressures for each volatility classification.

<table>
<thead>
<tr>
<th>Volatility Class</th>
<th>Number of HAPs</th>
<th>Range of Vapor Pressure (mm Hg at 25 degrees C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VVONC</td>
<td>15</td>
<td>&gt; 380</td>
</tr>
<tr>
<td>VVINC</td>
<td>6</td>
<td>&gt; 380</td>
</tr>
<tr>
<td>VONC</td>
<td>82</td>
<td>0.1 to 380</td>
</tr>
<tr>
<td>VINC</td>
<td>3</td>
<td>0.1 to 380</td>
</tr>
<tr>
<td>SVONC</td>
<td>64</td>
<td>$10^{-1}$ to $10^{-7}$</td>
</tr>
<tr>
<td>SVINC</td>
<td>2</td>
<td>$10^{-1}$ to $10^{-7}$</td>
</tr>
<tr>
<td>NVONC</td>
<td>5</td>
<td>&lt; $10^{-7}$</td>
</tr>
<tr>
<td>NVINC</td>
<td>12</td>
<td>&lt; $10^{-7}$</td>
</tr>
</tbody>
</table>

Examples of HAPs in VVOC class and their vapor pressure at 25 degrees C are:
- Acetaldehyde 952 mm Hg
- Formaldehyde 2,700 mm Hg

Examples of HAPs in VVINC class and their vapor pressure at 25 degrees C are:
- Chlorine 4,000 mm Hg
- Phosphine 2,000 mm Hg

Examples of HAPs in VOC class and their vapor pressure at 25 degrees C are:
- Benzene 76 mm Hg
- Xylene 5 mm Hg
Examples of HAPs in VINC class and their vapor pressure at 25 degrees C are:
- Hydrazine 16 mm Hg
- Hydrochloric acid 23 mm Hg

Examples of HAPs in SVOC class and their vapor pressure at 25 degrees C are:
- Benzidine $10^{-5}$ mm Hg
- Captan $10^{-6}$ mm Hg

Examples of HAPs in SVINC class and their vapor pressure at 25 degrees C are:
- Phosphorus $10^{2}$ mm Hg
- Mercury Compounds $10^{-3}$ mm Hg

Examples of HAPs in NVOC class and their vapor pressure at 25 degrees C are:
- 3,3'-Dimethoxybenzidine $10^{-13}$ mm Hg
- 4,4'-Methylenedianiline $10^{-10}$ mm Hg

Examples of HAPs in NVINC class and their vapor pressure at 25 degrees C are:
- Asbestos Very Low
- Cadmium Compounds Very Low

10.5.2 EPA’s Air Toxic Monitoring Methods

The choice of monitoring method depends on the specific air toxic(s) to be analyzed, the objective of the monitoring, the time over which a result is to apply, and available resources. It is important to note here that there do not currently exist valid methods for a large number of chemicals that may be of interest; for methods that do exist, the achievable sensitivity may not match the data quality objectives.

Many of the EPA’s original ambient monitoring methods for air toxics were extracted from industrial hygiene indoor air quality methods: the National Institute of Occupational Health (NIOSH) and the Occupational Safety and Health Administration (OSHA). Concerning the CAA’s 187 HAPs, EPA has developed 34 monitoring methods that can be used for most of these air toxics. (There still are significant problems because the analytic method may not be sensitive enough to detect down to levels of concern needed.) These monitoring methods include everything from the sample collection devices to analytical laboratory methods. EPA’s 34 air toxic monitoring methods can be found on EPA’s Ambient Monitoring Technology Information Center (AMTIC) website (http://www.epa.gov/ttn/amtic/airtox.html). According to “Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air,”
These methods are provided only for consideration by the user for whatever potential applications for which they may be deemed appropriate. In particular, these methods are not intended to be associated with any specific regulatory monitoring purpose and are specifically offered with no endorsement for fitness or recommendation for any particular application.” Of EPA’s 34 air monitoring methods, 17 are for “toxic organic” (TO), and 17 are for “toxic inorganic” (IO).

10.5.2.1 Toxic Organic Pollutants Monitoring Methods: EPA’s set of 17 TO standardized methods are for the determination of volatile, semi-volatile, and selected toxic organic pollutants in the air. These methods are identified as TO 1 through 17. The following is a short description of these 17 toxic organic monitoring methods:

- **TO-1**: Method for the Determination of Volatile Organic Compounds (VOCs) in Ambient Air using Tenax® Adsorption and Gas Chromatography/Mass Spectrometry (GC/MS)
- **TO-2**: Method for the Determination of VOCs in Ambient Air by Carbon Molecular Sieve Adsorption and Gas Chromatography/Mass Spectrometry (GC/MS)
- **TO-3**: Method for the Determination of VOCs in Ambient Air using Cryogenic Pre-concentration Techniques and Gas Chromatography with Flame Ionization and Electron Capture Detection
- **TO-4**: Determination of Pesticides and Polychlorinated Biphenyls in Ambient Air Using High Volume Polyurethane Foam (PUF) Sampling Followed by Gas Chromatographic/Multi-Detector Detection (GC/MD)
- **TO-5**: Determination of Aldehydes and Ketones in Ambient Air Using High Performance Liquid Chromatography (HPLC)
- **TO-6**: Determination of Phosgene in Ambient Air Using High Performance Liquid Chromatography (HPLC)
- **TO-7**: Method for the Determination of nitrosodimethylamine (NDMA) in Ambient Air Using Gas Chromatography
- **TO-8**: Method for the Determination of Phenol and Methylphenols (Cresols) in Ambient Air Using High Performance Liquid Chromatography
• **TO-9A**: Determination of Polychlorinated, Polybrominated, and Brominated/Chlorinated Dibenzo-p-Dioxins and Dibenzofurans in Ambient Air

• **TO-10A**: Determination of Pesticides and Polychlorinated Biphenyls in Ambient Air Using Low Volume Polyurethane Foam (PUF) Sampling Followed by Gas Chromatographic/Multi-Detector Detection (GC/MD)

• **TO-11A**: Determination of Formaldehyde in Ambient Air using Adsorbent Cartridge Followed by High Performance Liquid Chromatography (HPLC)

• **TO-12**: Method for the Determination of Non-methane Organic Compounds (NMOC) in Ambient Air Using Cryogenic Pre-concentration and Direct Flame Ionization Detection (PDFID)

• **TO-13A**: Determination of Polycyclic Aromatic Hydrocarbons (PAHs) in Ambient Air Using Gas Chromatography/Mass Spectrometry (GC/MS)

• **TO-14A**: Determination of VOCs in Air Using Specially-Prepared Canisters with Subsequent Analysis by Gas Chromatography

• **TO-15**: Determination of VOCs in Air Collected in Specially-Prepared Canisters and Analyzed by Gas Chromatography/Mass Spectrometry (GC/MS)

• **TO-16**: Long-Path Open-Path Fourier Transform Infrared Monitoring of Atmospheric Gases

• **TO-17**: Determination of VOCs in Air Using Active Sampling on to Sorbent Tubes

The HAP’s boiling point and/or vapor pressure will determine the applicable TO monitoring method to be used. The following is a breakdown of TO-1 thru TO-17 according to volatility and boiling point and/or chemical.

**For Volatile Organic Toxics:**

TO-1: 80 degrees C to 200 degrees C  
TO-2: -15 degrees C to 120 degrees C  
TO-3: 10 degrees C to 200 degrees C  
TO-12: Non-Methane Organic Compounds (NMOC)
TO-14A: -158 degrees C to 170 degrees C
TO-15: -50 degrees C to 170 degrees C
TO-16: 80 degrees C to 200 degrees C
TO-17: -158 degrees C to 200 degrees C

For Semi-Volatile Toxic Organic Compounds:

TO-4A: Pesticides and PCBs
TO-9A: Dioxins
TO-10A: Pesticides
TO-13A: Semi-Volatiles

For Specific Toxic Organic Compounds:

TO-5: Aldehydes and Ketones
TO-6: Phosgene
TO-7: Nitrosodime-thylamine
TO-8: Cresols and Phenols
TO-11A: Formaldehyde

10.5.2.2 Toxic Inorganic Pollutants Monitoring Methods: EPA has also developed a set of 17 standardized methods for the determination of selected toxic inorganic pollutants in the air. The following is a description of these 17 toxic inorganic monitoring methods:

Chapter IO-1 Continuous Measurement of PM10 Suspended Particulate Matter (SPM) in Ambient Air

- Method IO-1.1 Determination of PM10 in Ambient Air Using the Andersen Continuous Beta Attenuation Monitor
- Method IO-1.2 Determination of PM10 in Ambient Air Using the Thermo Environmental Inc. (formerly Wedding and Associates) Continuous Beta Attenuation Monitor
- Method IO-1.3 Determination of PM10 in Ambient Air Using a Continuous Rupprecht and Patashnick (R&P) TEOM® Particle Monitor
Chapter IO-2 Integrated Sampling of Suspended Particulate Matter (SPM) In Ambient Air

- Method IO-2.1 Sampling of Ambient Air for Total Suspended Particulate Matter (SPM) and PM10 Using High Volume (HV) Sampler
- Method IO-2.2 Sampling of Ambient Air for PM10 Using an Andersen Dichotomous Sampler
- Method IO-2.3 Sampling of Ambient Air for PM10 Concentration Using the Rupprecht and Patashnick (R&P) Low Volume Partisol® Sampler
- Method IO-2.4 Calculations for Standard Volume

Chapter IO-3 Chemical Species Analysis of Filter-Collected Suspended Particulate Matter

- Method IO-3.1 Selection, Preparation and Extraction of Filter Material
- Method IO-3.2 Determination of Metals in Ambient Particulate Matter Using Atomic Absorption (AA) Spectroscopy
- Method IO-3.3 Determination of Metals in Ambient Particulate Matter Using X-Ray Fluorescence (XRF) Spectroscopy
- Method IO-3.4 Determination of Metals in Ambient Particulate Matter Using Inductively Coupled Plasma (ICP) Spectroscopy
- Method IO-3.5 Determination of Metals in Ambient Particulate Matter Using Inductively Coupled Plasma/Mass Spectrometry (ICP/MS)
- Method IO-3.6 Determination of Metals in Ambient Particulate Matter Using Proton Induced X-Ray Emission (PIXE) Spectroscopy
- Method IO-3.7 Determination of Metals in Ambient Particulate Matter Using Neutron Activation Analysis (NAA) Gamma Spectrometry
Chapter IO-4 Determination of Reactive Acidic and Basic Gases and Strong Acidity of Atmospheric Fine Particles in Ambient Air Using the Annual Denuder Technology

- Method IO-4.1 Determination of the Strong Acidity of Atmospheric Fine Particles (2.5Fm)
- Method IO-4.2 Determination of Reactive Acidic and Basic Gases and Strong Acidity of Atmospheric Fine Particles

Chapter IO-5 Sampling and Analysis for Atmospheric Mercury

- Method IO-5 Sampling and Analysis for Vapor and Particle Phase Mercury in Ambient Air Utilizing Cold Vapor Atomic Fluorescence Spectrometry (CVAFS)

The 17 specific methods, covering a variety of inorganic pollutants and compounds, are categorized into 5 chapters. Nearly all the procedures have considerable flexibility and assume that the analyst has a substantial air monitoring background and expertise. Consequently, users are responsible for preparing a specific standard operating procedure (SOP) when incorporating the Inorganic Compendium methods into their air monitoring program.

Deficiencies in EPA Monitoring Methods (from EPA’s 2004 “Air Toxics Risk Assessment Reference Library, Volume I Technical Resource Manual”): EPA has not developed monitoring methods for all air toxic compounds. Potential deficiencies in particular monitoring methods include:

- Quantity limits are not low enough relative to environmental levels and/or health benchmarks
- Lack of available standards for monitoring protocols (i.e., standards developed by the National Institute of Science and Technology [NIST])
- Methods are not practical or easy to implement
- Compound stability is so poor that the compound degrades significantly between the time it is collected and the time it is analyzed, resulting in poor to no recovery at the time of analysis
• Recover efficiencies are too low, resulting in poor precision and/or quantity limits that are not low enough for use relative to health benchmarks

• Methods have not been sufficiently tested in the laboratory and field

• Methods are not producing results that are comparable to established methods

• Poor reliability

The deficiencies noted in Exhibit 10-3 are particularly important and have been identified by EPA as needing methodology development.

Exhibit 10-3: Identified Deficiencies in Available Monitoring Methods

<table>
<thead>
<tr>
<th>Compound</th>
<th>Candidate Method</th>
<th>Deficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,3-butadiene</td>
<td>TO-14A/15</td>
<td>sensitivity issue; false highs</td>
</tr>
<tr>
<td>1,2-dibromoethane</td>
<td>TO-14A/15</td>
<td>NIST standard needed; recovery problems</td>
</tr>
<tr>
<td>1,2-dichloroethane</td>
<td>TO-15</td>
<td>NIST standard needed</td>
</tr>
<tr>
<td>Acrylonitrile</td>
<td>TO-14A/15</td>
<td>NIST standard needed; recovery problems</td>
</tr>
<tr>
<td>ethylene oxide</td>
<td>None</td>
<td>poor storage stability</td>
</tr>
<tr>
<td>1,1,2,2-tetrachloroethane</td>
<td>TO-15</td>
<td>NIST standard needed</td>
</tr>
<tr>
<td>arsenic and compounds</td>
<td>IO-3</td>
<td>sensitivity issues; filter contamination; resource intensive</td>
</tr>
<tr>
<td>Beryllium and compounds</td>
<td>None</td>
<td>resource intensive; sensitivity issue</td>
</tr>
<tr>
<td>mercury and compounds</td>
<td>IO-5</td>
<td>requires special equipment</td>
</tr>
<tr>
<td>Acrolein</td>
<td>None</td>
<td>TO-11A results in unstable derivative; poor recovery</td>
</tr>
<tr>
<td>2,3,7,8-tetrachlorodibenzo-p-dioxin</td>
<td>TO-9</td>
<td>resource intensive</td>
</tr>
</tbody>
</table>

Because they present a similar challenge, EPA has targeted several VOCs for programs to improve monitoring capabilities (Exhibit 10-4).
Exhibit 10-4: VOC Compounds Needing Improved Monitoring Methods

<table>
<thead>
<tr>
<th>Vinyl chloride</th>
<th>Carbon tetrachloride</th>
<th>Tetrachloroethylene</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,2-dichloroethene</td>
<td>1,2-dichloropropane</td>
<td>1,1,2,2-tetrachloroethane</td>
</tr>
<tr>
<td>Dichloromethane</td>
<td>Trichloroethene</td>
<td>Hexachlorobutadiene</td>
</tr>
<tr>
<td>Chloroform</td>
<td>cis- and trans-1,3-dichloropropene</td>
<td>Acrylonitrile</td>
</tr>
<tr>
<td>1,2-dichloroethane</td>
<td>1,1,2-dichloroethane</td>
<td>1,3-butadiene</td>
</tr>
<tr>
<td>Benzene</td>
<td>1,2-dibromoethane</td>
<td>Ethylene oxide</td>
</tr>
</tbody>
</table>

In addition, both diesel exhaust (a complex mixture), acrolein, and arsenic require additional method development to yield accurate, reliable, and field-tested monitoring methods.

10.6 Determining the Types of Equipment and Samples

The particular monitoring method (which depends on the compound being sampled) will state what type of equipment for collecting a sample of air for pollution analysis needs to be used. These types of equipment fall into three broad categories related to the time scale over which concentration will be averaged:

- **Grab samples** provide a quasi-instantaneous measurement of a concentration. They generally are obtained in the field usually over a period of 24 hours or less and then returned to the laboratory for analysis. The sampling may be automated, allowing a time-series of samples to be drawn, but all samples still are generally returned to the laboratory for analysis.

- **Continuous monitors** provide a time series of measurements in the field, with a stream of data at selected intervals (i.e., once each 24 hours). These monitors may be fully automated versions of grab sampling, taking samples at a set interval but then analyzing the samples internally rather than returning to the lab. An alternative is continuous flow monitors, that draw ambient air through a chamber and analyzes it in real time (i.e., the semi-continuous formaldehyde monitor developed by the EPA, which runs through one complete cycle of sampling and analysis in 10 minutes).

- **Time-integrated samples** are collected over an extended period of time. Only the total pollutant collected is measured, and so only the average concentration during the sampling period can be determined. As with grab samples, these measurements generally are obtained in the field and returned to a laboratory for analysis.
Monitoring methods/systems can also be divided into a different set of categories based on the method of collection:

- **Integrated air sampling devices** use a pump to draw air continuously into the sample chamber, over a reactive medium, or through a filter during a prescribed period of time; the sample is returned to the laboratory for analysis. Integrated air sampling methods are the predominant type of monitoring employed for air toxics networks. For metal and carbonyl air toxics, this collection device consists of some type of filter or reactive material that collects the air toxics. In the case of VOC air toxics, the sample is collected in a canister. The pump can be programmed to collect air for a pre-set period of time (i.e., 1 hour to 24 hours). The collected samples are then sent to a laboratory for analysis.

- **Direct-read monitors** draw air through a measurement system and provide a direct reading of the concentration without returning samples to the lab.

- **Automated monitoring systems** collect samples, perform the analysis, and report results at regular intervals in the field.

- **Air deposition monitors** rely on deposition properties of compounds (i.e., particulates), and may consist of active and/or passive, wet and/or dry sampling methods.

- **Passive monitors** allow the compound to diffuse into contact with an active material; these generally are analyzed in the lab, although some indicate the presence of a compound by a color change.

- **Grab sampling devices** use an essentially instantaneous sampling method, such as an evacuated chamber into which ambient air is allowed to enter at a fixed rate; the sample collected is returned to the laboratory for analysis.
References


Chapter 11: Source Sampling of Air Toxics

11.1 Introduction

A stack test, also referred to in EPA regulations as a performance or source test, measures the amount of a specific regulated pollutant, pollutants, or surrogates being emitted; demonstrates the capture efficiency of a capture system; or determines the destruction or removal efficiency of a control device used to reduce emissions at facilities subject to the requirements of the Clean Air Act (CAA). Stack testing is an important tool used to determine a facility’s compliance with emission limits, or capture or control efficiencies established pursuant to the CAA. Stack testing can also be used to assist in the establishment of emissions standards, calculate emission factors (i.e., AP-42), and for screening tests that will provide a preliminary indication of levels of a pollutant.

Stack sampling methods were originally developed for criteria pollutants and approved by the EPA. These methods were developed to demonstrate compliance with new source performance standards. The need for measurement of hazardous air pollutants (HAPs) evolved whenever a national emission standard for hazardous air pollutant (NESHAP) was promulgated. This caused EPA to make modifications to their criteria pollutant stack sampling methods by combining these methods with analytical procedures for the measurement of hazardous substances found in other media (i.e., solid waste) and from sources such as Occupational Safety and Health Administration (OSHA) and National Institute of Occupational Safety and Health (NIOSH).

Stack testing methods are prescribed by EPA. Reference methods (RM) exist for a wide-variety of criteria and hazardous air pollutants. The methods specify detailed procedures to be followed in conducting the stack tests. Stack testing is highly technical and should be conducted by trained and qualified personnel only. More details on stack testing are available at EPA’s Emission Measurement Center (EMC) web page http://www.epa.gov/ttn/emc/.

11.2 Categories of Stack Test Methods

The following categories of stack test methods are published on the EPA’s EMC website at www.epa.gov/ttn/emc/tmethods.html. A fundamental component of the EMC web site is to provide information regarding relevant methods and procedures for emission testing and monitoring. EMC presents these methods into four different categories (A, B, C, D). The categories are based on the legal status of the methods with regard to their application under federally enforceable regulations.
Each category is explained below and each includes stipulations for use of the methods included therein. Further provisions regarding the use of a specific method are contained within the applicability statement of the method. The guidance provided by these categories and methods is meant to supplement, not override the stationary source emission measurement requirements of Title 40 of the Codes of Federal Regulations (CFR) Parts 51, 60, 61, and 63.

**Category A: Methods Proposed or Promulgated in the Federal Register (FR)**

These methods have been proposed or promulgated in the Federal Register and codified in the Code of Federal Regulations (CFR). The EMC is responsible for methods that have method numbers between 1 and the 300's. These methods have between 1 and 3 digits with no decimal points.

- Method numbers or Performance Specifications between 1 and 100 are for New Source Performance Standards (NSPSs). These methods are found in 40 CFR Part 60, Appendix A. In this series, there are presently 76 promulgated methods, the following is a list of some of these methods:
  - 5/5A-I: Particulate matter
  - 12: Inorganic lead
  - 18: VOCs by gas chromatograph (GC)
  - 25: Gaseous non-methane organic emissions
  - 23: Dioxin and Furan
  - 29: Metals from stationary sources
  - 30: Mercury from power plants

- Method numbers in the 100 series are for the (pre-1990 CAA Amendments) National Emission Standards for Hazardous Air Pollutants (NESHAPs). These methods are found in 40 CFR Part 61, Appendix B. In this series, there are presently 16 promulgated methods, the following is a list of some of these methods:
  - 101/101A: Mercury in air streams
  - 102: Mercury in hydrogen streams
  - 104: Revised Beryllium screening
  - 106: Vinyl Chloride
  - 108 A, B, C: Arsenic in ore samples
  - 110 Benzene
• Method numbers in the 200 series are example State Implementation Plans (SIPs). These methods are found in 40 CFR Part 51, Appendix M. In this series, there are presently 13 promulgated methods, the following is a list of some of these methods:
  - 201/201A: PM10
  - 206: Ammonia

• Method numbers in the 300 series are for the (post-1990 CAA Amendments) Maximum Achievable Control Technology (MACT) standards. These methods are found in 40 CFR Part 63, Appendix A. In this series, there are presently 25 promulgated methods, the following is a list of some of these methods:
  - 301 Data validation
  - 302 Gas chromatograph
  - 303 Coke oven doors
  - 304 A, B: Biodegradation rate
  - 305: Compound specific liquid waste
  - 306: Hexavalent chromium
  - 307: VOC for solvent degreasers
  - 308: Methanol
  - 315: HAPs surrogates
  - 316: Formaldehyde

All these methods (except for the 200 series) are directly cited by specific regulations for determining compliance under 40 CFR Parts 60, 61, and 63, and may be used by sources for application as cited by the regulations and the method itself without further EPA approval. The 200 series methods are promulgated to facilitate incorporation in applicable State Implementation Plans. All these methods include quality control and quality assurance procedures that must be met. EPA staff can provide technical support on all methods in this category.

Category B: Source Category Approved Alternative Methods

These methods are approved alternatives to the methods required by 40 CFR Parts 60, 61 and 63 as described by the General Provisions of the corresponding Parts and as further explained in a Federal Register notice dated January 30, 2007. As such, the methods may be used by sources for determining compliance with the requirements of these Parts per their specified applicability provisions without further EPA approval. The EPA Administrator, or designee (currently the
group leader of the Measurement Technology Group in the Air Quality Assessment Division of the Office of Air Quality Planning and Standards), has approved these methods for the specified applications; this approval has been documented through an official EPA letter. These methods include quality control and quality assurance procedures that must be met. The EPA staff may not necessarily be the technical experts on these methods.

Presently, there are 57 alternative methods. An example of an alternative method would be Alternative Method 39 (Alt-039) for sludge drying or sludge incineration facilities affected under the NESHAP for mercury in 40 CFR Part 61, Subpart E: you can use Method 29 (Metals Emissions from Stationary Sources) in lieu of Method 101A (Determination of Particulate and Gaseous Mercury Emissions from Sewage Sludge Incinerators).

Category C: Other Methods

This category includes test methods which have not yet been subject to the Federal rulemaking process. There are presently 28 other test methods listed. Each of these methods, as well as the available technical documentation supporting them, have been reviewed by the EMC staff and have been found to be potentially useful to the emission measurement community. The types of technical information reviewed include field and laboratory validation studies; results of collaborative testing; articles from peer-reviewed journals; peer-review comments; and quality assurance (QA) and quality control (QC) procedures in the method itself. The EPA strongly encourages the submission of additional supporting field and laboratory data as well as comments in regard to these methods.

These methods may be considered for use in federally enforceable state and local programs (i.e., Title V permits, State Implementation Plans [SIPs]) provided they are subject to an EPA Regional SIP approval process or permit veto opportunity and public notice with the opportunity for comment. The methods may also be considered to be candidates to be alternative methods to meet Federal requirements under 40 CFR Parts 60, 61, and 63. However, they must be approved as alternatives under 60.8, 61.13, or 63.7(f) before a source may use them for this purpose. Consideration of a method’s applicability for a particular purpose should be based on the stated applicability as well as the supporting technical information outlined in the table. The methods are available for application without EPA oversight for other non-EPA program uses including state permitting programs and scientific and engineering applications.

As many of these methods are submitted by parties outside the Agency, the EPA staff may not necessarily be the technical experts on these methods. Therefore, technical support from EPA for these methods is limited, but the table contains contact information for the developers so that
you may contact them directly. Also, be aware that these methods are subject to change based on the review of additional validation studies or on public comment as a part of adoption as a Federal test method, the Title V permitting process, or inclusion in a SIP.

**Category D: Historic Conditional Methods**

This category includes methods that were categorized as conditional test methods before EMC’s method categories were revised. Because some of these methods have been cited in state rules and permits under their Conditional Test Method (CTM) designation, EMC has created a category for them and called it "Historic Conditional Methods." This category is closed and no new methods will be added to it.

These CTMs have been evaluated by the EPA and may be applicable to one or more categories of stationary sources. The EPA confidence in a method included in this category is based upon review of various technical information including, but not limited to, field and laboratory validation studies; EPA understanding of the most significant quality assurance (QA) and quality control (QC) issues; and EPA confirmation that the method addresses these QA/QC issues sufficiently to identify when the method may not be acquiring representative data. The method's QA/QC procedures are required as a condition of applicability.

CTMs in Category D may be used by state and local programs in conjunction with federally enforceable programs (e.g., Title V permits, State Implementation Plans [SIP]) provided they are subject to an EPA Regional SIP approval process or permit veto opportunity and public notice and opportunity for comment. They are available for application without EPA oversight for other non-EPA program uses including state permitting programs and scientific and engineering applications.

The methods in Category D, however, must be approved as alternatives before a source may use them to meet Federal requirements under 40 CFR Part 60, 61, and 63. The EPA encourages the submission of additional field and laboratory data to further support a method. The EPA staff may not necessarily be the technical experts on these methods. These methods are potentially subject to change based on the review of additional validation studies or on public comment as a part of adoption as a Federal test method (Category A).

**11.3 Alternative Stack Test Methods**

Many of the stack test methods for criteria pollutants were combined with analytical methods for hazardous materials to establish sampling methods for HAPs. For example Method 0010, also
known as Modified Method 5 and the semi-volatile organic sampling train, was developed under Resource Conservation Recovery Act (RCRA) for determining destruction and removal efficiency of hazardous waste incinerators for semi-volatile principal organic hazardous compounds (POHCs) in the hazardous waste feed. (For the purpose of this method, semi-volatile organic compounds are defined as compounds with boiling points greater than 100°C, but less than 500°C. For other methods, they may be defined differently.)

The EPA publication SW-846, entitled “Test Methods for Evaluating Solid Waste, Physical/Chemical Methods,” is Waste's official compendium of analytical and sampling methods that have been evaluated and approved for use in complying with the RCRA regulations. SW-846 functions primarily as a guidance document setting forth acceptable, although not required, methods for the regulated and regulatory communities to use in responding to RCRA-related sampling and analysis requirements.

SW-846 is a multi-volume document that changes over time as new information and data are developed. It has been issued by EPA since 1980 and is currently in its third edition. Advances in analytical instrumentation and techniques are continually reviewed by EPA and incorporated into periodic updates to SW-846 to support changes in the regulatory program and to improve method performance and cost effectiveness. To date, EPA has finalized Updates I, II, IIA, IIB, III, IIIA, IIIB, IVA and IVB to the SW-846 manual, and the updated and fully integrated manual contains approximately 3500 pages.

The following are air toxic stack test methods listed in SW-846:

- 0010: Semi-volatiles
- 0011: Aldehydes and ketones
- 0020: Source assessment sampling train
- 0023: Dixon and Furons
- 0030/31/40: Volatile organics
- 0050/51: HCL/CL2
- 0060: Multi-metals
- 0061: Hexavelent chromium
- 0100: Formaldehyde in indoor air
- 0200: Source assessment sampling train

As part of the Technical Studies Program, National Council for Air and Stream Improvement (NCASI) sometimes develops methods for analytical parameters for which no rigorously tested or validated methods are available. Once developed, these NCASI methods are frequently used for
other program applications and to respond to regulatory and member company requests. The methods are compiled into a single manual to facilitate their distribution and use. Examples of a couple of their air toxic sampling methods (issued in 2005) are:

- IM/CAN/WP-99.02: Canister source sampling methods for selected HAPs from wood products facilities
- CI/SG/PULP-94.03: Methanol emissions from pulp mills
- ISS-FP-A-105.01: Selected aldehydes, ketones, and polar compounds

Reference methods should be used whenever possible. Use of an equivalent method (i.e., Categories B, C, & D) is acceptable. The acceptability of alternative methods (i.e., EPA SW-846, NCASI, etc.) that have not been proven equivalent will be evaluated by the regulating agency on a case-by-case basis.

11.4 Testing of Volatile Organic Compounds

The majority of CAA Section 112 HAPs are volatile organic compounds (VOC) with physical and chemical properties ranging from those of formaldehyde found in the gas phase to polynuclear aromatic hydrocarbons (PAHs) which may be absorbed on particle surfaces. The range of volatility of air toxic VOCs requires a variety of sampling techniques (i.e., Methods 18, 25, 25A, etc).

Testing for volatile organic compounds is often confusing for a variety of reasons. According to Winberry (APTI 468) the following are some of the reasons:

- There is no straightforward way to measure the VOC emissions since there is no way to separate VOCs by vapor pressure.
- All of the reference methods for organic compounds have inherent limitations that restrict their applicability, and no one method can satisfy characterization of organic emissions from an industrial source.

Pennsylvania Department of Environmental Protection, “Source Testing Manual” (Revision 3.3), provides a general scheme for the selection of a VOC test method (see Exhibit 11-1). This scheme can be used to select an appropriate sampling technique. The selection scheme does not address all of the limitations for a given reference method nor does it list all of the possibilities.
Exhibit 11-1 follows two different paths, one for speciated (single component concentration) VOCs and the other for non-speciated (combined organic gas emission) VOCs. When speciation is not required, the following Federal Register Methods may be used:

- **Method 18** (VOC by gas chromatograph): Based on separating components of a gas mixture in a gas chromatograph column and measuring separated components with suitable detector (i.e., flame ionization detector [FID]). Applicable to VOC concentrations greater than one ppm in the sampled gas.

- **Method 25** (non-methane organic compounds) applies to the measurement of VOCs as total gaseous non-methane organics, condensable and non-condensable, as carbon in source emissions. (All compounds are converted to methane before measuring with a FID.)

- **Method 25A** (organic concentration using a FID): This method is applicable to the measurement of total gaseous organic concentration of vapors consisting primarily of alkanes, alkenes, and/or aromatic hydrocarbons. Results are expressed in terms of volume concentration of propane (or other appropriate organic calibration gas) or in terms of carbon. (All compounds are measured directly as a whole with a FID.)

- **Method 25B** (organic concentration using an infrared analyzer) and

- **Method 25C** (non-methane organic compounds from landfills).
For speciated VOCs, the following alternative methods (from SW-846) may be used:

- **Method 0010** for semi-volatile organics: In this method the sample is collected in a sampling train that is similar to Federal Register Method 5 for particulates. In method 0010, a high efficiency glass filter is used to collect organic-laden particulates, a packed bed of porous polymeric resin (XAD-2™) serves to adsorb semi-volatile organic species, and a series of water-filled impingers may collect some semi-volatile organics that pass through the filter and sorbent.

- **Method 0011** is used for aldehydes and ketones.

- **Method 0030** is used for VOCs (compounds with boiling points less than 100 °C but normally above 30 °C).
The following definitions are used in measurements of organic compounds:

- **Volatile Organic Compounds (VOCs):** An organic compound that participates in atmospheric photochemical reactions; that is, an organic compound other than those that the Administrator of the EPA designates as having negligible photochemical reactivity. The exempted compounds are listed in 40 CFR §51.100(s)(1). VOCs usually have high vapor pressures (greater than 0.1 mm Hg).

- **Semi-volatile Organic Compounds (SVOC):** This definition can vary depending on the test method. Usually SVOCs are organic compounds with vapor pressure between 0.1 and $10^{-7}$ mm Hg.

- **Total Organic Compounds (TOCs):** The sum of all volatile organic compounds and all exempted compounds listed in 40 CFR§51.100(s)(1).

- **Total Hydrocarbons (THCs):** The subset of total organic compounds containing only carbon and hydrogen.

- **Total Non-Methane Organic Compounds (TNMOCs):** The sum of all volatile organic compounds and all exempted compounds listed in 40 CFR§51.100(s)(1), except methane.

These terms are used in air toxic regulations. For example, in the Petroleum Refinery MACT (40 CFR § 63.643(a)(2)) Miscellaneous Process Vents Provisions, reduction of organic HAPs “compliance can be determined by measuring either organic HAPs or TOCs using the procedures in § 63.643.” This MACT (and the HON MACT also) lets you measure the total organic compounds in the exhaust to see if it exceeds a defined limitation. Identifying individual organic HAP amounts would not be necessary.

### 11.5 Phases of a Stack Test

A stack test usually consists of three phases, the: (1) protocol review; (2) test observation, and (3) final report review. Each phase is conducted to ensure that the test is appropriate, accurate, and valid. Generally, a facility hires a consultant to conduct the stack test. The consultant prepares a protocol describing the pollutants to be tested, the test methods they plan to use, and the expected operating conditions at the facility. The protocol should be made available to regulatory personnel (EPA, state or local officials) for their review prior to the test.
Once approved, a test date is scheduled. Whenever possible, regulatory personnel should be present during enough of the testing to ensure the test is valid. Depending on the type of test, samples may be sent to a laboratory for analysis. After the test results have been analyzed, the consultant issues a final report to the facility. Regulatory personnel should review the report to determine whether the test was accurate, complete and valid. If the facility fails the test, the regulatory agency should take appropriate enforcement action to ensure the facility is brought into compliance. If the test is determined to be invalid, the regulatory agency should require retesting.

11.6 Detection Limits

In stack testing, non-detect or below detection levels are often encountered. The “limit of detection” is the smallest amount of a substance that an analytical method can reliably distinguish from zero. More formally, it is the minimum concentration or amount of a target analyte that produces a signal the tester can distinguish, at a specified confidence level, from the signal produced by a blank. The “limit of quantification” is the minimum concentration or amount of an analyte that a method can measure with a specified degree of precision.

Each laboratory should report its limit of detection, identify its procedure for measuring the limit of detection, and label results below the detection limit as "below detection limit (BDL)." The tester must report the experimentally measured limit of detection and the procedure used to measure it whether or not individual results are above or below the detection limit. If the user averages results of samples from the same "test," where some results are BDL and other results are above the detection limit, then the user should substitute the estimated detection limit for the BDL results. The user should then report the average as "equal to or less than" the averaged result. If all results are BDL, the user should report the average as BDL also.

11.7 EMC Software

EPA’s EMC web site contains databases and other supporting software for method technology, not excluding additional software supporting other emission measurement issues. Examples include the Test Method Storage and Retrieval software, PC Nomograph program, Manual Emission Testing Cost Model PC program, and CEM cost estimation & methods spreadsheet programs.

In 2007, EMC added the latest of its emission measurement software: the Electronic Reporting Tool (ERT). ERT, a Microsoft Access desktop application, is an electronic alternative for paper reports documenting nineteen of EPA’s emissions measurement methods for stationary sources.
The ERT replaces the time-intensive manual preparation and transcription of stationary source emissions test plans and reports currently performed by contractors for emissions sources and the time-intensive manual quality assurance evaluations and documentation performed by State agencies. The ERT provides a format that:

- Highlights the need to document the key information and procedures required by the existing EPA Federal Test Methods;
- Facilitates coordination among the source, the test contractor, and the regulatory agency in planning and preparing for the emissions test;
- Provides for consistent criteria to quantitatively characterize the quality of the data collected during the emissions test;
- Provides for future capabilities to electronically exchange information in the reports with facility, state or federal data systems.

11.8 Continuous Emission Monitors

The air toxic sampling that has been discussed up to this point is called manual sampling and is useful for short-term compliance demonstrations and emission screening. Some instrumental analytical techniques have been applied to source emission measurements including some air toxics. Many of these instrumental methods require sample extraction much like a manual method but the analysis is conducted on-site with a compound specific detector and an electronic data reduction device. These instrumental methods are intended for short-term (i.e., less than twenty-four hours) operation in a manner similar to that for the manual methods.

Instrumental measurement techniques can be modified to operate on a continuous basis to collect emission data for long-term data averaging. Continuous emission monitoring systems (CEMs) have been applied to emissions from major stationary sources for over thirty years. Two types of CEMs are commonly used: extractive CEMs and in-situ CEMs. Extractive CEMs draw a sample from a stack, condition the sample gas (i.e., remove particulate matter and moisture), and analyze for the specific compounds of interest. In-situ CEMs provide a measure of target compounds in the stack without sample extraction or conditioning. The components of in-situ CEMs commonly include a light or radiation source, a detector, and a data reduction device mounted on the stack. Since in-situ CEMs allow analysis of the sample in its normally-occurring condition, their results may be more representative than those for extracted CEMs.
A majority of the listed HAPs are VOCs. These VOC concentrations are detected using analyzer methods such as flame ionization detection (FID), photo-ionization detection (PID), or non-dispersive infrared (NDIR) absorption. These VOC analyzers do not specifically identify VOCs nor do they respond equally to all VOCs. They only provide a measure of the relative VOC concentration of the mixture of compounds. Although VOC analyzers can be used for air toxics measurements when consistent proportions of multiple air toxics are in the exhaust stream and they generate equal instrumental responses, the best application for a VOC CEMs for air toxics determination is for an exhaust gas stream that contains a single air toxic and no other interfering substances.

The Emission Measurement Center (EMC) has been investigating the application of an innovative new technology, Fourier Transform Infared Spectroscopy (FTIR), to emissions monitoring. The FTIR technology shows promise since it has the capability to measure more than 100 of the 189 Hazardous Air Pollutants (HAPs) listed in Title III of the CAA Amendments of 1990. Upon passage of these Amendments, measurement methods existed for only 40 of the HAPs. The FTIR has the capability of measuring multiple compounds simultaneously, thus providing an advantage over current measurement methods which measure only one or several HAPs; FTIR can provide a distinct cost advantage since it can be used to replace several traditional methods (cost savings can vary depending on the number of compounds present).

The FTIR approach provides for collection of the entire infrared spectrum in a few seconds. FTIR equipment includes a standard interferometer, associated optical support, and a computer system. A typical extractive FTIR system includes path length of about twelve meters achieved using multiple IR beam reflections in a contained sample cell. Path length for in-situ CEMs without multiplication through reflective techniques is limited to the width of the stack. Sensitivity at path lengths across a stack is limited to about one to five parts per million, and this may not be sufficient for some air toxics measurement situations.
References


Chapter 12: Air Toxics Controls for Stationary Sources

12.1 Introduction

This chapter describes available control technologies for stationary sources. Because the majority of air toxics are gaseous volatile organic compounds (VOCs), the focus of this Chapter is on control technologies for gases. (These sections are adopted from Crowder’s 2002, “Sources and Control of Volatile Organic Compounds” Student Manual and EPA’s “Control Technologies for Hazardous Air Pollutants” Handbook.) This chapter also describes, in a cursory fashion, the various control technologies for particulate emissions. Finally, this chapter will describe the different control technologies for various sources of mercury emissions (including municipal waste combustion and medical waste incineration) with a special emphasis on the available control technologies for coal-fired utility boilers.

12.1.1 Introduction to Control Techniques for Gaseous Emissions

Control devices for gaseous emissions can be of two types: combustion and recovery. The combustion devices discussed in this Chapter include thermal incinerators, catalytic incinerators, flares, and boilers/process heaters. Applicable recovery devices include condensers, adsorbers, and absorbers. Combustion devices are the more commonly applied control device, since they are capable of high removal (i.e., destruction) efficiencies for almost any type of organic vapor HAP although carbon adsorbers are also quite popular. Combustion devices serve as a complete control technique; in that, they destroy rather than collect pollutants. With carbon adsorbers and condensers, the VOC HAP must be dealt with after collection. The removal efficiencies of the recovery techniques generally depend on the physical and chemical characteristics of the HAP under consideration as well as the emission stream characteristics. Applicability of the control techniques depends more on the specific emission stream under consideration than on the particular source category (i.e., degreasing vs. surface coating in solvent usage operations source category). Thus, selection of applicable control techniques for point source emissions is made on the basis of stream-specific characteristics and desired control efficiency. The key emission stream characteristics and HAP characteristics that affect the applicability of each control technique are identified in Exhibit 12-1, and limiting values for each of these characteristics are presented. Matching the specific characteristics of the stream under consideration with the corresponding values in Exhibit 12-1 will help the user to identify those techniques that can potentially be used to control the emission stream. The list of potentially applicable control techniques will then be narrowed further depending on the capability of the applicable control devices to achieve the required performance levels. The expected emission reduction from the
application of each control technique on the basis of the total VOC concentration in the emission stream is identified in Exhibit 12-2. Very little data regarding control device removal efficiency for specific HAPs are available. Therefore, without actual source test data for a specific emission stream and control system, HAP removal efficiency is assumed to equal total VOC removal efficiency.

**Exhibit 12-1: Emission Stream and HAP Characteristics for Selecting Control Devices**  
(Source: EPA’s “Control Technologies for Hazardous Air Pollutants” Handbook)

<table>
<thead>
<tr>
<th>Control Device</th>
<th>HAP/Organic Contents (ppmv)</th>
<th>Heat Content (Btu/scf)</th>
<th>Flow Rate (scfm)</th>
<th>HAP Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thermal Incineration</td>
<td>&gt;20; (&lt; 25% of LEL)</td>
<td>&lt;50,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Catalytic Incineration</td>
<td>50 – 10,000 (&lt; 25% of LEL)</td>
<td>&lt;50,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Flare</td>
<td>300</td>
<td>&lt;2,000,000</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Boiler/Process Heater</td>
<td>150</td>
<td>Steady</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Carbon Adsorber</td>
<td>700 – 10,000</td>
<td>300 – 200,000</td>
<td></td>
<td>Must have adsorbent properties</td>
</tr>
<tr>
<td>Absorber</td>
<td>250 – 10,000</td>
<td>1,000 – 100,000</td>
<td></td>
<td>Must be water soluble</td>
</tr>
<tr>
<td>Condenser</td>
<td>5,000 – 10,000</td>
<td>&lt;2,000</td>
<td></td>
<td>Vapor pressure &gt; 10 mm Hg (at stp)</td>
</tr>
</tbody>
</table>

**Exhibit 12-2: Approximate Percent Reduction for Add-On Control Devices**  
(Source: EPA’s “Control Technologies for Hazardous Air Pollutants” Handbook)
12.2 Incineration

In vapor incineration processes, organic contaminants are removed from a gas stream by oxidizing them to other compounds. If the organic compounds are composed of carbon and hydrogen, then the products of that oxidation are carbon dioxide and water vapor. However, if the organic materials contain chlorine, fluorine or sulfur, then hydrochloric acid vapor, hydrofluoric acid vapor, sulfur dioxide or other compounds may be formed, requiring absorbers downstream of the incinerator in order to reduce the emission of these compounds. In general, incinerators are capable of destruction efficiencies greater than 95%. Some incinerator designs have destruction efficiencies greater than 99%.

There are three common types of incineration processes: flares, thermal oxidizers, and catalytic oxidizers. Flares are usually used for gas streams that have an organic vapor concentration greater than two to three times the lower explosive limit. The lower explosive limit, or LEL, is the level of organic vapor concentration at which oxidation will be self-supporting if a source of ignition is provided. Thermal oxidizers are used for contaminated gas streams that have an organic vapor concentration that is generally less than 50% of the LEL and usually less than 25%. Catalytic oxidizers are used for gas streams that have concentrations less than 25% of the LEL.

12.2.1 Flares

A typical flare consists of a system that first collects the waste gases and then passes them through a knockout drum to remove any liquids before entering the flare stack. Flame arrestors are placed between the knockout drum and the flare stack to prevent flashback of flames into the collection system. The flare stack is a hollow pipe that may extend to heights above 150 feet. The flare tip is at the top of the stack, where the waste gases are ignited by pilot flames. If the gases do not have sufficient heat content, fuel will be added to the gas stream. This is called a fired or endothermic flare.

The flare tip is designed to provide good mixing characteristics over a range of gas flow rates while maintaining smokeless combustion. Steam jets are one of the most effective ways to mix air with the waste gases. An example of a flare tip with steam injection is shown in Exhibit 12-3. In addition to increasing turbulence, steam injection reduces the partial pressure of the contaminant gases, reducing polymerization of higher molecular weight organic compounds in the waste gas stream. The steam also reacts with the waste gases to produce oxygenated compounds that burn at lower temperatures. For these reasons, steam is often used for waste
gases that are difficult to burn. Compounds with a hydrogen-to-carbon ratio less than 0.3 will usually require steam injection.

Flares can be used for controlling almost any VOC emission stream. They can be designed and operated to handle fluctuations in emission VOC content, inert content, and flow rate. Flaring is generally considered a control option when the heating value of the emission stream cannot be recovered because of uncertain or intermittent flow as in process upsets or emergencies. If the waste gas to be flared does not have sufficient heating value to sustain combustion, auxiliary fuel may be required. Based on studies conducted by EPA, 98 percent destruction efficiency can be achieved by steam-assisted flares when controlling emission streams with heat contents greater than 300 Btu/scf.

Exhibit 12-3: Flare Tip
(Source: “Sources and Control of Volatile Organic Compounds” Student Manual)

12.2.2 Thermal Oxidizers

Thermal incinerators are used to control a wide variety of continuous emission streams containing VOCs. Compared to the other techniques, thermal incineration has broad applicability; that is, it is much less dependent on HAP characteristics and emission stream characteristics. Destruction efficiencies up to 99 + percent are achievable with thermal incineration. Although they accommodate minor fluctuations in flow, thermal incinerators are
Introduction to Air Toxics

not well suited to streams with highly variable flow because the reduced residence time and poor mixing during increased flow conditions decreases the completeness of combustion. This causes the combustion chamber temperature to fall, thus decreasing the destruction efficiency.

The thermal oxidizer consists of a refractory brick-lined chamber that has one or more gas- or oil-fired burners. The contaminated gas stream does not usually pass through the burner itself, unless a portion of the gas stream is used to provide the oxygen needed to support combustion of the fuel. Instead, the burners are used to heat the gas stream to the temperature necessary to oxidize the organic contaminants. That temperature is based primarily on the auto-ignition temperature of the most difficult to-destroy compound in the gas stream.

Auto-ignition temperatures for most organic compounds range from 750°F to 1,000°F. Operation at temperatures near the auto-ignition value will result in destruction of the contaminants; however, the carbon in the compounds will be left primarily as CO, even if sufficient oxygen is available. To fully oxidize the carbon to CO₂, incineration should be conducted at a minimum temperature of 1,300°F. Most thermal oxidizers operate at temperatures of 1,300°F to 1,800°F.

The combustion chamber is sized to provide sufficient residence time to complete the oxidation reactions. The time needed for destruction depends on the temperature and mixing within the chamber, with shorter times required at higher temperatures. Typical residence times are 0.3 to 0.5 seconds, but may exceed 1 second. Residence time is typically calculated by dividing the volume of the combustion chamber by the actual volumetric flow rate through the combustion chamber. However, it should be recognized that not all of the combustion chamber may be at the effective oxidation temperature. Thus, residence times calculated in this manner should be somewhat higher than the minimum desired value.

Exhibit 12-4 contains theoretical combustion chamber temperatures for 99.99 percent destruction efficiencies for various HAP compounds with a residence time of one second.
Exhibit 12-4: Theoretical Combustion Chamber Temperatures Required for 99.99% Destruction Efficiencies
(Source: EPA’s “Control Technologies for Hazardous Air Pollutants” Handbook)

<table>
<thead>
<tr>
<th>HAP Compound</th>
<th>Combustion Temperature (°F)</th>
<th>Residence Time (seconds)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acrylonitrile</td>
<td>1,344</td>
<td>1</td>
</tr>
<tr>
<td>Allyl chloride</td>
<td>1,276</td>
<td>1</td>
</tr>
<tr>
<td>Benzene</td>
<td>1,350</td>
<td>1</td>
</tr>
<tr>
<td>Chlorobenzene</td>
<td>1,407</td>
<td>1</td>
</tr>
<tr>
<td>Methyl chloride</td>
<td>1,596</td>
<td>1</td>
</tr>
<tr>
<td>Toluene</td>
<td>1,341</td>
<td>1</td>
</tr>
<tr>
<td>Vinyl chloride</td>
<td>1,369</td>
<td>1</td>
</tr>
</tbody>
</table>

Two types of thermal incinerators are commonly used. The thermal recuperative type uses a conventional heat exchanger to heat the incoming emission stream. The thermal regenerative type uses ceramic beds to heat the incoming stream.

12.2.2.1 Recuperative Thermal Oxidizer: Because of fuel costs, most incinerators use heat exchangers to recover some of the heat from the treated gas stream. That heat is usually used to pre-heat the contaminated gas stream before it enters the incinerator. One type of heat exchanger is the recuperator, and incinerators that use them are sometimes called recuperative thermal oxidizers. The most common recuperator is shell-and-tube heat exchanger. These devices have a shell that encloses numerous small tubes. The contaminated gas stream flows through these tubes, while the treated gas stream flows through the shell across the tubes. Approximately 50% to 60% of the heat from the treated gas stream is transferred through the tubes to the waste gas stream. An example of a recuperative thermal oxidizer is shown in Exhibit 12-5.

Exhibit 12-5: Double Pass Recuperative Heat Exchanger
(Source: “Sources and Control of Volatile Organic Compounds” Student Manual)
Because of the presence of the heat exchanger, particle concentrations in the inlet gas stream must be minimized. Particulate matter can foul the inside surfaces of the tubes, reducing the thermal efficiency. It can also increase the resistance to flow through the heat exchanger, reducing the fan’s ability to move volume through the system, potentially causing fugitive emissions at the source. Some recuperative units include clean-out ports and access hatches to facilitate cleaning.

12.2.2.2 Regenerative Thermal Oxidizers: Another type of heat exchanger is the regenerator, and incinerators that use them are sometimes called regenerative thermal oxidizers. A regenerator consists of a set of refractory beds that store heat. Heat recovery is achieved by passing the waste gas stream through a packed ceramic bed at the inlet to the incinerator that was previously heated, with the gases exiting the incinerator. It is common to use at least three beds in a regenerative system. One bed is used to pre-heat the waste gas stream, one bed is used to store heat from the treated gas stream, and one bed is in a purge cycle. The purge cycle is needed to prevent emission spikes each time the gas flow is reversed. A set of gas flow dampers is used to switch the inlet, outlet and purge gas streams to the appropriate beds. This unit has three parallel beds with an overhead refractory-lined combustion chamber equipped with two gas-fired burners.

Regenerative heat exchangers have much higher heat recovery efficiencies than recuperative units, recovering as much as 95% of the heat from the treated gas stream. Because of the high inlet gas temperatures created by the heat recovery, burner fuel is typically required only if the organic vapor concentration is very low. At moderate to-high concentrations, the heating value of the organic compounds may be sufficient to maintain the necessary temperatures.

12.2.2.3 Process Heaters and Boilers: An alternative to installing a thermal oxidizer is to incinerate the contaminated gas stream in a process heater or boiler. This avoids the capital cost of new equipment. The contaminated gas stream is typically injected close to the main burners, or it may be used as part of the combustion air supplied to the burners. Process heaters and boilers are normally designed to operate with combustion chamber temperatures greater than 1,800°F and flue gas residence times in excess of 1 to 2 seconds. These conditions are similar to those of conventional thermal oxidizers.

There are some limitations in the use of a process heater or boiler as an incinerator. The boiler must operate within about 40% to 100% of the design load in order to provide the temperatures necessary for organic vapor destruction. The waste gas stream should be only a small portion of the total gas flow through the unit. If the volume of additional gas is large, maximum recommended velocities through the unit would be exceeded, potentially lowering the
destruction efficiency of the system. Finally, there should be nothing in the waste gas stream or its combustion products that would present a corrosion problem or cause deposits on heat transfer surfaces.

12.2.3 Catalytic Oxidizers

In order to minimize fuel consumption, catalytic oxidizers are designed with both recuperative and regenerative heat exchangers. Since the advantages and limitations of the recuperators and regenerators are identical to those used on thermal oxidizers, this section will discuss only the characteristics of catalytic oxidation.

A catalyst is a substance that accelerates a chemical reaction without undergoing a change itself. The typical catalysts used in incineration are noble metal oxides of platinum, palladium or rhodium that are deposited as a thin layer on a high surface area material, such as alumina, that is bonded to a support structure. Base metal oxides, such as vanadium pentoxide, titanium dioxide or manganese dioxide, may also be used. The support structure is arranged in a matrix that provides high geometric surface area, low pressure drop, and uniform flow. Structures providing these characteristics include honeycombs, grids, and mesh pads.

After passing through the heat transfer section, the contaminated gas stream is heated to the required temperature by a gas-fired burner and then passed through the catalyst material, called the bed. The catalyst causes the oxidation reaction to occur at much lower temperatures than would be required for thermal oxidation. This is the principal advantage of catalytic oxidation. Because of the lower operating temperature, less fuel is required for heating, and refractory-lined combustion chambers are usually not needed. In some cases, it is possible for the catalytic oxidizer to operate without supplemental fuel, except during start-up.

The inlet temperature to the catalyst bed is generally about 100°F above the catalytic auto-ignition temperature of the most difficult-to-destroy compound in the gas stream. Most organic compounds have catalytic auto-ignition temperatures of about 400°F to 500°F, giving a typical minimum inlet temperature of about 500°F to 600°F. For each 1% LEL concentration that is oxidized in the catalyst bed, the gas stream temperature will increase about 27°F. For a 25% LEL gas stream concentration, the total temperature increase would be about 675°F, bringing the outlet temperature from the catalyst bed to about 1,175°F to 1,275°F. This is very close to the temperature that can cause damage to most catalyst materials. For this reason, catalytic oxidizers are usually used only on gas streams with an organic vapor concentration less than 25% of the LEL.
The quantity of catalyst is often described in terms of the space velocity. Space velocity is calculated by dividing the standard volumetric flow rate by catalyst bed volume and is essentially the reciprocal of residence time at standard conditions. Typical space velocities for noble metal catalysts are 30,000 to 40,000 per hour. For the less active base metal catalyst, space velocities are typically 10,000 to 15,000 per hour.

The primary disadvantages of catalytic oxidizers are the cost of the catalyst and performance problems related to physical and chemical deterioration of the catalyst material. Catalytic oxidizers usually cannot be used effectively on waste gas streams with high concentrations of liquid or solid particles. These particles deposit on the catalyst surface, blocking access for the organic compounds. This is termed fouling. When low concentrations of particles are present, periodically cleaning the catalyst can restore more than 90% of its activity.

Certain metals can react or alloy with the catalyst, permanently deactivating it. This is called catalyst poisoning. Fast-acting poisons include mercury, phosphorus, arsenic, antimony, and bismuth. Slow-acting poisons include lead, zinc, and tin. At temperatures above about 1,000°F, even copper and iron are capable of alloying with platinum, reducing its activity.

Some materials (principally sulfur and halogen compounds) have a high adsorptive affinity for some catalytic surfaces, reducing the active sites available to the organic compounds. This is termed masking. Although removing the masking agent will return catalyst activity to normal, the better approach is to use a catalyst that is not masked by compounds in the gas stream. Vanadium pentoxide and titanium dioxide, for example, are not masked by sulfur compounds.

High temperatures can also result in catalyst deactivation. Loss of activity because of high temperature is called thermal aging. This results in a rapid loss of catalyst activity and is caused primarily by the sintering of the catalyst support material. Sintering involves the merging of the support material ridges. As this occurs, catalyst is trapped below the surface of the support material and is no longer available to the organic compounds. Extremely high temperatures can cause volatilization of the catalyst material.

12.3 Carbon Adsorption

Adsorption is a surface phenomenon where VOCs are selectively adsorbed on the surface of such materials as activated carbon, silica gel, or alumina. Activated carbon is the most widely-used adsorbent. Adsorption systems using silica gel or alumina are less likely to be encountered in air pollution control and are not discussed in this manual.
Adsorbed VOCs are removed from the carbon bed by heating to a sufficiently high temperature (usually via steam) or by reducing the pressure to a sufficiently low value (vacuum desorption). During desorption, about 3 to 5 percent of organics desorbed on virgin-activated carbon is adsorbed so strongly that it cannot be desorbed during regeneration.

Carbon adsorption is commonly employed as a pollution control and/or a solvent recovery technique. It is applied to dilute mixtures of VOC and air. Removal efficiencies of 95 to 99 percent can be achieved using carbon adsorption. The maximum practical inlet concentration is usually about 10,000 parts per million by volume (ppmv) but virtually all applications will have significantly lower concentrations. The inlet concentrations are typically limited by the adsorption capacity of the carbon bed or safety problems posed by high bed temperatures produced by heat of adsorption and presence of flammable vapors. Outlet concentrations around 50 ppmv can be routinely achieved with state-of-the-art systems; concentrations as low as 10 to 20 ppmv can be achieved with some compounds. In contrast to incineration methods whereby the VOCs are destroyed, carbon adsorption provides a favorable control alternative when the VOCs in the emission stream are valuable.

The amount of material adsorbed by activated carbon is termed its retention and is expressed in weight percent or as the ratio of pounds of organic contaminant adsorbed per 100 pounds of carbon. Saturated activated carbon will generally hold 20 to 40 pounds of contaminant per 100 pounds of carbon. The most common method of expressing saturation capacity data is the isotherm. The isotherm is a constant temperature plot of the adsorbent capacity at saturation versus the concentration of the contaminant in the gas stream, usually expressed in partial pressure. Exhibit 12-6 is an example of an isotherm for carbon tetrachloride adsorption onto activated carbon.

**Exhibit 12-6: Adsorption Isotherm for Carbon Tetrachloride on Activated Carbon**
(Source: “Sources and Control of Volatile Organic Compounds” Student Manual)
The ability of activated carbon to retain organic contaminants is influenced by a number of parameters. One of these is temperature. At lower temperatures, because of the lower vibrational frequency of the molecules, the retention is high. As temperature increases, it becomes increasingly difficult to keep the contaminant molecules on the surface and retention goes down. Because of this, carbon adsorbers are usually operated at temperatures no higher than about 125°F.

Retention is also influenced by the absolute pressure of the system. At higher pressures, retention is higher because the molecules in the gas stream are more closely spaced, making it more difficult for contaminant molecules to escape from the surface and move back into the gas stream. As the pressure is reduced, the wider spacing of the gas stream molecules makes it more difficult to keep contaminant molecules on the surface, and retention goes down.

Similarly, the concentration or partial pressure of the contaminant molecules in the gas stream also influences the retention. At higher concentrations, more contaminant molecules are moving toward the surface, making it more difficult for molecules on the surface to escape, and retention is increased. Likewise, at lower concentrations it is easier for molecules on the surface to escape and retention is reduced. Even though retention is higher for higher concentration gas streams, this does not mean that less carbon is needed. At higher concentrations, there is more material to be collected, generally requiring larger amounts of carbon.

Retention is affected by the molecular weight of contaminant molecules, because of variations in their vibrational frequency. Small molecules with high vibrational frequencies are difficult to retain on the carbon surface. Generally, molecules with molecular weights less than about 45 are not good candidates for adsorption. As the molecular weight of molecules increases, retention increases. However, molecules with a molecular weight greater than about 200 are also not good candidates for adsorption. Although they are readily retained by the activated carbon, they are more difficult to remove during regeneration. Also, contaminants that can react with the surface, like organic acids, aldehydes and some ketones, are not good candidates for carbon adsorption.

Moisture in the gas stream can also affect retention. Although water molecules are polar, they will adsorb onto activated carbon, competing with the organic contaminant for adsorption sites. This generally becomes a concern when the relative humidity of the gas stream is greater than about 50%. The designer usually counteracts this effect by using more total carbon when the relative humidity is high.

Adsorption is also affected by the presence of liquid or solid particles in the gas stream. As the gas stream wiggles through the bed of carbon granules, any particles in the gas stream would be
collected on the surface of the granules, blocking access to the high surface area in the internal pore structure. Accumulated PM can also cause poor flow distribution, reducing collection efficiency. As a result, any PM in the gas stream must be removed before entering the adsorber.

A carbon adsorber system may have difficulties when controlling an emission stream containing ketones (i.e., acetone, methyl ethyl ketone). Ketones exothermically polymerize on the carbon bed, clogging the pores on the surface of the carbon which reduces the effective amount of carbon contained in the vessel. This in turn, decreases the system efficiency. If a carbon adsorber system is used to control ketones, the reader should be aware of this potential problem.

The most common industrial adsorber is the fixed-bed design that regenerates on site. An example of this collector is shown in Exhibit 12-7. The activated carbon will be contained in two or more vessels or modules, so that one of them can be taken off line for regeneration while the others continue to collect the contaminants. The carbon beds in these modules are typically 1.5 to 4 feet thick. The lower dimension is set to reduce channelization of the gas flow that can occur in thinner beds. The upper dimension is set because of the energy required to move the gas stream through thicker beds. The contaminated gas stream flows down through the operating modules to mitigate fines formed from attrition of the carbon being introduced into the cleaned gas stream.

**Exhibit 12-7: Three-Bed Regenerative System**
(Source: “Sources and Control of Volatile Organic Compounds” Student Manual)
When a module is put back on line after regeneration, the carbon will contain a small quantity of contaminant that was not practical to remove during regeneration. This is called the heel and is generally 3 to 5 weight percent or 3 to 5 pounds of contaminant per 100 pounds of carbon. Since regeneration is conducted with an upward flow, this heel is left as an essentially saturated layer at the top of the carbon bed.

When the gas flow enters this vessel, a mass transfer zone, or MTZ, is formed (see Exhibit 12-8). This is the zone of active adsorption and is generally 2 to 4 inches thick. As adsorption proceeds, the MTZ moves down the bed leaving behind it saturated carbon. When the MTZ reaches the end of the bed, the module is taken off line for regeneration. If the MTZ is allowed to exit the bed, a condition called breakthrough occurs and the outlet concentration rises rapidly.

The quantity of organic material that can be retained by the carbon bed during each adsorption cycle is the difference between the capacity at breakthrough and the heel capacity. This is termed working capacity and is conservatively estimated to be about half of the saturation capacity or, typically, about 10 to 20 pounds of contaminant per 100 pounds of carbon.

There are two ways to decide when to take a module off line for regeneration. The most effective way is to use an analyzer in the outlet gas stream that takes the module off line when breakthrough occurs. This allows for full utilization of the carbon bed, but requires the purchase, operation, and calibration of an analyzer. It is the most expensive option.

Exhibit 12-8: Mass Transfer Zone Movement
(Source: “Sources and Control of Volatile Organic Compounds” Student Manual)
12.4 Absorbers (Scrubbers: i.e. Packed Towers)

Packed bed absorbers are the most common absorbers used for gas removal. Packed columns disperse the scrubbing liquid over packing material, which provides a large surface area for gas-liquid contact. The most common packed bed absorber is the counter-current flow tower shown in Exhibit 12-9. The gas stream being treated enters the bottom of the tower and flows upward through the bed of packing material. Liquid is introduced at the top of the packed bed by sprays or weirs and flows downward over the packing material, resulting in the highest theoretically achievable efficiency. The most dilute gas is put into contact with the least saturated absorbing liquor. Accordingly, the maximum concentration difference between the gas phase contaminants and the dissolved concentration of the contaminant in the liquid is at the top of the packed bed. This concentration difference provides a driving force for continued absorption.

Exhibit 12-9: Counter-Current Packed Tower
(Source: “Control of Gaseous Emissions” Student Manual)

Absorption is widely used as a raw material and/or a product recovery technique in separation and purification of gaseous streams containing high concentrations of VOCs. As an emission control technique, it is much more commonly employed for inorganic vapors (e.g., hydrogen
sulfide, chlorides, etc.) than for organic vapors. Using absorption as the primary control technique for organic vapor, HAPs is subject to several limitations and problems as discussed below.

The suitability of absorption for controlling organic vapor emissions is determined by several factors; most of these factors will depend on the specific HAP in question. For example, the most important factor is the availability of a suitable solvent. The pollutant in question should be readily soluble in the solvent for effective absorption rates and the spent solvent should be easily regenerated or disposed of in an environmentally acceptable manner.

Another factor that affects the suitability of absorption for organic vapor emissions control is the availability of vapor/liquid equilibrium data for the specific HAP/solvent system in question. Such data are necessary for design of absorber systems. For uncommon HAPs, these data are not readily available.

Another consideration involved in the application of absorption as a control technique is disposal of the absorber effluent (i.e., used solvent). If the absorber effluent containing the organic compounds is discharged to the sewer, pond, etc., the air pollution problem is merely being transformed into a water pollution problem. In solvent recovery, used organic solvents are typically stripped (reverse of absorption) and recycled to the absorber for economic reasons. However, in HAP control applications, stripping requirements will often be very expensive because the residual organic concentrations in the solvent must be extremely low for it to be suitable for reuse. In organic vapor HAP control applications, low outlet concentrations will typically be required. Trying to meet such requirements with absorption alone will lead to impractically tall absorption towers, long contact times, and high liquid-gas ratios that may not be cost effective.

Factors which may affect inorganic vapor absorption are similar to those for organic vapor absorption. Typical solvents used by industry for inorganic vapor control include water, sodium hydroxide solutions, amyl alcohol, ethanolamine, weak acid solutions, and hypochlorite solutions. Exhibit 12-10 summarizes the reported efficiencies for various inorganic vapors employing absorption as the control method. Water is the ideal solvent for inorganic vapor control by absorption. It offers distinct advantages over other solvents, the main one being its low cost. It is typically used on a once-through basis and then discharged to a waste-water treatment system. The effluent may require pH adjustment to precipitate metals and other HAPs as hydroxides or salts; these are typically less toxic and can be more easily disposed of.
Control efficiencies for absorbers vary from 50 to greater than 95 percent. Efficiency depends on the selected solvent, the contact surface area (absorber size), and the temperature. The applicability of absorption to vent streams is dependent on the availability of a suitable solvent, and the solubility of the VOC in the solvent. If a VOC cannot be easily desorbed from the solvent, then absorption is less viable. Absorption is usually considered for streams with a VOC concentration above 200 to 300 ppmv.

Scrubbers are used downstream of combustion devices to control emissions of halogens and halogen halides formed during combustion. The typical scrubbing solvents used are water or a caustic solution. Either plate or packed bed scrubbers can be used, and these scrubbers can have countercurrent or crosscurrent flow. The type and orientation of the scrubber used depends on liquid and gas flow rates. Scrubber efficiencies for removal of halogens and halogen halides will vary depending on the type of scrubber and the type of solvent used, and the equilibrium relationship between the gas and liquid. However, most systems can achieve efficiencies from 90 percent to greater than 99 percent.

### 12.5 Condensers

In condensation processes, gaseous contaminants are removed from a gas stream by causing them to change to a liquid. In general, this can be accomplished by increasing the pressure or reducing the temperature or by a combination of both. However, because of the cost of operating and maintaining compression equipment, most condensers for air pollution control use temperature reduction.

The ultimate efficiency of a condenser depends on the operating temperature. As a vapor-laden gas stream is cooled, the molecules slow down and crowd together so closely that weak electrostatic forces between the molecules cause them to condense. The point at which this occurs is termed the *dew point temperature*. With further reduction in temperature, the vapor pressure of the condensing compound decreases. The lower the vapor pressure, the lower the
concentration of the contaminant in the gas stream. Condensation systems generally operate at efficiencies greater than 90%.

The two most common types of condensers used are surface and contact condensers. Surface condensers are usually shell-and-tube heat exchangers (see Exhibit 12-11). The coolant typically flows through the tubes and the vapors condense on the shell outside the tubes. The condensed vapors form a film on the cool tubes and are drained to a collection tank for storage or disposal. In contrast to surface condensers where the coolant does not contact either the vapors or the condensate, in contact condensers, the vapor mixture is cooled by spraying a cool liquid directly into the gas stream.

Condensers are widely used as raw material and/or product recovery devices. They are frequently applied as preliminary air pollution control devices for removing VOC contaminants from emission streams prior to other control devices such as incinerators, adsorbers, or absorbers.

Condensers are also used by themselves for controlling emission streams containing high VOC concentrations (usually >5,000 ppmv). In these cases, removal efficiencies obtained by condensers can range from 50 to 90 percent although removal efficiencies at the higher end of this scale usually require HAP concentrations of around 10,000 ppmv or greater. The removal efficiency of a condenser is highly dependent on the emission stream characteristics including the nature of the HAP in question (vapor pressure-temperature relationship) and HAP concentration, and the type of coolant used. Note that a condenser cannot lower the inlet VOC concentration to levels below the saturation concentration (or vapor pressure) at the coolant temperature. When water, the most commonly used coolant, is employed, the saturation conditions represent high outlet concentrations. For example, condenser outlet VOC concentrations are often limited to above 10,000 to 20,000 ppmv due to the saturation conditions of most of the organic compounds at the temperature of the cooling water. Therefore, it is not possible for condensation with water as the coolant to achieve the low outlet concentrations that would be required in HAP control applications.
Removal efficiencies around 90 percent may be achieved if lower temperatures than those possible with cooling water are employed, but this is generally only true if the HAP concentration is very high (e.g. >10,000-20,000 ppmv). These low temperatures can be obtained with coolants such as chilled water, brine solutions, or chlorofluorocarbons. These refrigerated condenser systems are often sold as packaged units. However, for extremely low outlet HAP concentrations, condensation will usually be infeasible.

Depending on the type of condenser used, there may be potential problems associated with the disposal of the spent coolant. Therefore, using contact condensers that generate such effluents for controlling HAP emissions is not recommended.

Flow rates up to about 2,000 standard cubic foot per minute (scfm) can be considered as representative of the typical range for condensers used as emission control devices. Condensers for emission streams with flow rates above 2,000 scfm and containing high concentrations of noncondensibles will require prohibitively large heat transfer areas.

The temperature of the outlet steam is a fundamental indicator of performance for a condenser system. Therefore, continuous monitoring of this parameter is recommended for control of HAPs.
12.6 Bioreactors

Bio-reaction systems are sorption processes that use aerobic microorganisms to consume the organic compounds. They are used primarily for gas streams with a low organic vapor concentration and where the compounds are soluble in water and biodegradable. The main classes of bioreactors are bio-filters, bio-trickling filters, and bio-scrubbers. The classes of compounds that can be removed in these systems include alcohols, aldehydes, esters, ketones, and many aromatic hydrocarbons. Removal efficiencies range from 60% to more than 99%.

12.6.1 Bio-filters

Bio-filters use a fixed microbial population in a stationary water phase. The microorganisms grow in a bio-film on the surface of a medium, or they are suspended in the water phase surrounding the medium. The filter medium consists of relatively inert materials, such as compost and peat, which provide a large surface area and additional nutrient supply. As the gas stream passes through the bed, the contaminants sorb into the bio-film and onto the filter medium where they are degraded.

Bio-filter beds are 3 to 5 feet deep and have a void volume of about 50% and water content of about 60% by weight. They usually have some sort of water addition to control moisture and add nutrients. Often, this involves only humidification of the gas stream before it enters the bio-filter. However, if humidification proves inadequate, direct irrigation of the bed may be necessary. The operating temperature of the bed is 60°F to 85°F and the pH of the support material is typically 6 to 8. Compounds containing sulfur or nitrogen can generate acidic byproducts during biodegradation, requiring alkali addition to maintain the pH level. Gas stream residence time in the bed ranges from 15 to 60 seconds.

Bio-filters are relatively inexpensive to construct and to operate, they have a low pressure drop, and they produce no further waste streams. However, they have a large space requirement, the moisture and pH can be difficult to control, and PM may clog the medium.

12.6.2 Bio-trickling Filters

Bio-trickling filters have a fixed microbial population in a flowing water phase. Prior to biodegradation, the contaminants are absorbed from the gas stream into a water phase that flows over an inorganic packing material. Microbes fixed to the packing and suspended in the water degrade the absorbed contaminants as they pass through the reactor. Bio-trickling filters can be operated concurrently or counter-currently and are governed by many of the same operating...
conditions as bio-filters. Nutrients, acids, or bases may be added to the re-circulated water to regulate the environment for optimal pollutant removal.

Bio-trickling filters are more complex and expensive to construct and operate than bio-filters, but they are better able to treat acid-producing contaminants. They are prone to clogging by biomass and produce a waste stream that requires further processing.

12.6.3 Bio-scrubbers

Bio-scrubbers have a suspended microbial population in a flowing water phase. Absorption takes place in typical wet scrubbing equipment, such as spray towers, plate towers or packed beds. The effluent from the wet scrubber is then transferred to a separate aerated vessel, where degradation of the contaminants is performed by suspended microbes. As with the bio-trickling filter, the re-circulated water allows nutrients, acids, or bases to be added to maintain an environment conducive to optimal pollutant removal. They are less prone to clogging by biomass, but produce a waste stream that requires further processing.

12.7 Control Techniques for Inorganic Vapor Emissions

Inorganic vapors make up only a small portion of the total HAPs emitted to the atmosphere. Inorganic HAP vapors typically include gases such as ammonia, hydrogen sulfide, carbonyl sulfide, carbon disulfide, metals with hydride and carbonyl complexes, chloride, oxychloride, and cyanide.

In many cases, although the inorganic HAPs are emitted as vapors at the emission source, they may condense when passing through various ducts and form particulates. Prior to discharge to the atmosphere, these particulates are typically controlled by particle control devices.

Only a limited number of control methods are applicable to inorganic vapor emissions from stationary sources. The two most commonly used control methods are absorption (scrubbing) and adsorption. Absorption is the most widely used and accepted method for inorganic vapor control. Although combustion can be used for some inorganic HAPs (i.e., hydrogen sulfide, carbonyl sulfide, nickel carbonyl), typical combustion methods such as thermal and catalytic incineration are generally not applied. In some cases, for example, in controlling hydrogen sulfide emissions from gas wells and gas processing, flares are used.

Applicability of absorption and adsorption as control methods depends on the individual emission stream characteristics. The removal efficiencies that can be achieved will be
determined by the physical and chemical properties of the HAP under consideration. Other factors (i.e., waste disposal, auxiliary equipment requirements), while not necessarily affecting the technical feasibility of the control device, may affect the decision to use that particular control method.

### 12.8 Control Techniques for Particulate Emissions

Three types of control devices applicable to particulate laden emission streams from point sources: fabric filters (baghouses), electrostatic precipitators (ESP), and venturi scrubbers. The control efficiencies and applicability of these devices depend on the physical and/or chemical/electrical properties of the airborne PM under consideration. Selection of the control devices themselves depends on the specific stream characteristics (i.e., particle size, temperature, corrosiveness, resistivity, and moisture content) and the parameters (i.e., required collection efficiency) that affect the applicability of each control device. Brief descriptions of each of these control devices appear in the subsections that follow.

#### 12.8.1 Baghouses

Fabric Filters, or baghouses, are an efficient means of separating PM matter entrained in a gaseous stream. A fabric filter unit consists of one or more isolated compartments containing rows of fabric bags in the form of round, flat, or shaped tubes, or pleated cartridges. Particle laden gas passes up (usually) along the surface of the bags then radially through the fabric. Particles are retained on the upstream face of the bags, and the cleaned gas stream is vented to the atmosphere. The filter is operated cyclically, alternating between relatively long periods of filtering and short periods of cleaning. During cleaning, dust that has accumulated on the bags is removed from the fabric surface and deposited in a hopper for subsequent disposal.

Fabric filters collect particles with sizes ranging from submicron to several hundred microns in diameter at efficiencies generally in excess of 99 or 99.9 percent. The layer of dust, or dust cake, collected on the fabric is primarily responsible for such high efficiency. The cake is a barrier with tortuous pores that trap particles as they travel through the cake. Gas temperatures up to about 500°F, with surges to about 550°F, can be accommodated routinely in some configurations. Most of the energy used to operate the system appears as pressure drops across the bags, associated hardware, and ducting. Typical values of system pressure drop range from about 5 to 20 inches of water. Fabric filters are used where high efficiency particle collection is required. Limitations are imposed by gas characteristics (temperature and corrosivity) and particle characteristics (primarily stickiness) that affect the fabric or its operation and that cannot be economically accommodated.
Important process variables include particle characteristics, gas characteristics, and fabric properties. The most important design parameter is the air- or gas-to-cloth ratio (the amount of gas in ft³/min that penetrates one ft² of fabric) and the usual operating parameter of interest is pressure drop across the filter system. The major operating feature of fabric filters that distinguishes them from other gas filters is the ability to renew the filtering surface periodically by cleaning. Fabric filters are usually made of woven or (more commonly) needle punched felts sewn to the desired shape, mounted in a plenum with special hardware, and used across a wide range of dust concentrations.

Another type of fabric filter developed in the 1970s and 1980s is the electrostatically enhanced filter. Pilot plant baghouses employing this technology have shown substantially lower pressure drops than conventional filter designs. Further, some cost analyses have shown that electrostatically enhanced baghouses could have lower lifetime costs than convention baghouses.

12.8.2 Electrostatic Precipitators

An electrostatic precipitator (ESP) is a particle control device that uses electrical forces to move the particles out of the flowing gas stream and onto collector plates. The particles are given an electrical charge by forcing them to pass through a corona, a region in which gaseous ions flow. The electrical field that forces the charged particles to the walls comes from electrodes maintained at high voltage in the center of the flow lane.

Once the particles are collected on the plates, they must be removed from the plates without re-entraining them into the gas stream. This is usually accomplished by knocking them loose from the plates, allowing the collected layer of particles to slide down into a hopper from which they are evacuated. Some precipitators remove the particles by intermittent or continuous washing with water.

ESP’s are less sensitive to particle size than the other two particle control devices and in fact can control submicron particles quite well. The electrical resistivity of the particles influences the drift velocity, or the attraction between the particles and the collection plate. A high resistivity will cause a low drift velocity which may decrease the overall collection deficiency. A low resistivity indicates that it is difficult to chart the particles and will tend to decrease the collection efficiency, all other things being equal.
12.8.3 Venturi Scrubbers

A venturi scrubber has a “converging-diverging” flow channel. In this type of system the cross-sectional area of the channel decreases then increases along the length of the channel. The narrowest area is referred to as the “throat.” In the converging section, the decrease in area causes the waste gas velocity and turbulence to increase. The scrubbing liquid is injected into the scrubber slightly upstream of the throat or directly into the throat section. The scrubbing liquid is atomized by the turbulence in the throat, improving gas-liquid contact. The gas-liquid mixture then decelerates as it moves through the diverging section, causing additional particle-droplet impacts and agglomeration of the droplets. The liquid droplets are then separated from the gas stream in an entrainment section, usually consisting of a cyclonic separator and mist eliminator.

Venturi scrubbers are more expensive than spray tower, cyclonic, or tray tower scrubbers, but collection efficiencies for fine PM are higher. High gas velocities and turbulence in the venturi throat result in high collection efficiencies, ranging from 70% to 99% for particles larger than 1 μm in diameter and greater than 50% for submicron particles. Increasing the pressure drop in a venturi scrubber increases the efficiency, but the system’s energy demand also increases leading to greater operational costs.

12.9 Control Technologies for Mercury Emissions

With the exception of mercury, most metals have sufficiently low vapor pressures at typical air pollution control device (APCD) operating temperatures that condensation onto PM is possible. Mercury, on the other hand, has a high vapor pressure at typical APCD operating temperatures, and collection by PM control devices is highly variable. Factors that enhance mercury control are low temperature in the APCD system (less than 150 °C [300 to 400 °F]), the presence of an effective mercury sorbent and a method to collect the sorbent. In general, high levels of carbon in the flyash enhance mercury sorption onto PM. The ash is then removed by the PM control device. Additionally, the presence of hydrogen chloride (HCl) in the flue gas stream can result in the formation of mercuric chloride (HgCl₂), which is readily adsorbed onto carbon-containing PM. Conversely, sulfur dioxide (SO₂) in flue gas can act as a reducing agent to convert oxidized mercury to elemental mercury, which is more difficult to collect.
Introduction to Air Toxics

Some of the most common add-on controls to reduce mercury emissions include:

- Carbon filter beds
- Wet scrubbing
- Selenium filters
- Activated carbon injection

12.9.1 Carbon Filter Beds

Carbon filter beds have been developed in Europe for use as a final cleaning stage to remove heavy metals (i.e., mercury), organic pollutants (i.e., dioxins and furans), and acid gases (i.e., SO₂, HCl). Three filter designs have been used: cross flow, counter current, and counter-cross flow. The cross-flow design has been applied to utility boilers and waste combustors, the counter current design to waste combustors, and the counter-cross flow design to utility boilers. Most of the information available on carbon filter beds pertains to the cross-flow design.

In the cross-flow filters, the flue gas flows horizontally through the filter bed, while the adsorbent migrates through the filter from top to bottom. The bed is approximately 1 meter (m) thick and is divided into three layers. The first layer removes PM, heavy metals (including mercury), organic compounds, and SO₂. Removal of HCl occurs primarily in the second layer. The third layer acts as a safety layer. The three layers are separated by perforated metal sheets. On the outlet side, there is a slotted sheet designed to prevent particles from being carried out of the filter with the flue gas. Additionally, an impact separator is located downstream of the slotted sheet, functioning as a safety barrier against particle emissions.

Fresh carbon is conveyed to and distributed within the bed by a screw conveyor on the top of the bed. Discharge cylinders at the bottom of each layer allow extraction of carbon from each layer. Pressure drop is the primary parameter for determining the rate of carbon removal from the bed. The designed pressure drop across the bed is 150 to 190mm (6 to 7.5 in.) of water. The pressure drop across the whole system including fans and ducting is approximately 305 mm (12 in.) of water. Because of dust collection in and compaction of each layer, approximately 100 to 130 mm (4 to 5 in.) of material is periodically sloughed from each layer. Because of greater vibration of particles and, thus, greater settling of the particles within the first layer, carbon is sloughed from this layer on the shortest time interval, typically once every 6 to 8 hours. The second layer is sloughed once per day, and the third layer is sloughed once every 2 weeks.
Based on these removal rates and bed thicknesses, the mass of carbon in the filter is fully replaced at an average rate of once per year. For municipal waste combustors (MWCs), this equates to approximately 2 kilograms (kg) carbon/Mg (4 pounds [lb] carbon/ton) of municipal solid waste (MSW) burned.

Spent carbon can be disposed of by combustion if the unit is equipped with a wet scrubbing system. The combustion process destroys the organic compounds captured in the carbon, and the wet scrubber collects the heavy metals and acid gases. Another disposal option more applicable to U.S. sources equipped with dry or semidry flue gas cleaning systems is to dispose of the carbon in a hazardous waste landfill.

In Europe, carbon filter beds have been installed on waste incinerators and utility boilers. Carbon filter beds are also technically feasible applications to sources such as medical waste incinerators (MWIs) or smelters. According to EPA’s 1997 “Mercury Report to Congress,” carbon filter beds have not been put into commercial practice in the U.S. for any of these source types.

12.9.2 Wet Scrubbing

Wet scrubbing (WS) systems can be used to control acid gases, metals, PM, and dioxins and furans. Depending on the intended purpose of the WS system, different designs are available and include:

- Single-stage scrubbers designed primarily for control of acid gases
- Two-stage scrubbers designed for control of acid gases and metals
- Three-stage systems consisting of a two-stage scrubber followed by a high-efficiency wet particulate control system designed for improved control of fine particulates, metals, and dioxins and furans
- Single-stage scrubbers that are installed following other APCD’s and are designed for increased pollutant control (often referred to as polishing scrubbers)

Soluble species of mercury compounds, such as HgCl and mercuric oxide (HgO), can be effectively captured (greater than 90 percent) in the wet scrubber. If there is significant elemental mercury in the flue gas, however, collection efficiencies will be limited. The captured mercury is precipitated out during wastewater treatment through the use of any of several
additives (i.e., sodium sulfide, trimercapto-s-triazine [TMT-15] and dithiocarbamate). Performance data available for three-stage systems on MWIs indicate mercury removal efficiencies of over 90 percent.

WS systems can be applied on MWCs, MWIs, and boilers downstream of other acid gas and PM controls. A factor determining effectiveness of this control is the amount of water soluble mercury in the flue gas stream. Two-stage and three-stage wet scrubbers form the basis of the final emission standards for existing MWIs (standards were promulgated August 15, 1997). These scrubbers appear to be highly effective at removing mercury from MWI flue gas. The high chlorine content in medical waste may result in high concentrations of HgCl, which is efficiently collected by a wet scrubber in this type of application.

12.9.3 Selenium Filters

Selenium filters have been developed to reduce elemental mercury emissions. The filters operate based on the affinity between mercury and metallic selenium. The mercury-laden flue gas passes through the filter, which is constructed of ceramic grains impregnated with metallic selenium. The gas pathway through the filter is tortuous, which increases the contact between the mercury and the selenium, forming mercury selenite (HgSe).

Selenium filters are effective on flue gas streams with inlet mercury concentrations of up to 9 milligrams (mg)/scm (3,900 gr/million scf). At very low mercury concentrations, the removal efficiency decreases because of reduced mercury-selenium molecular collisions. At higher mercury concentrations, the lifetime of the filter is short.

Because the removal of mercury in the filter is based on the formation of HgSe, the selenium in the filter is eventually exhausted. The selenium filter is designed to convert approximately 50 kg of mercury to HgSe per cubic meter of filter material (3 lb/cubic [ft3]). The combination of pressure drop, mercury content in the flue gas and the mechanical construction of the filter determine the ultimate lifetime of the filter. On average, the filter lifetime is 5 years, after which the filter element is replaced. Once the lifetime of the filter mass has expired, the HgSe mass is land-filled (it is not combustible).

While use of this technology has been demonstrated in Sweden for metal smelters and crematories, it has not been demonstrated for a utility boiler. Use of these filters on MWCs and MWIs may also be technically feasible.
12.9.4 Activated Carbon Injection

Activated carbon injection involves the injection of powdered activated carbon into flue gas upstream of an APCD. Activated carbon is a specialized form of carbon produced by pyrolyzing coal or various hard, vegetative materials (i.e., wood) to remove volatile material. The resulting char then undergoes a steam or chemical activation process to produce an activated carbon that contains multiple internal pores and has a very high specific surface area. With this internal pore structure, the activated carbon can adsorb a broad range of trace contaminants, including mercury. After injection into the flue gas and adsorption of mercury and other contaminants, the activated carbon is captured in the PM control device.

The factors affecting the performance of activated carbon injection are the temperature of the flue gas, the amount of activated carbon injected, the concentration and species of mercury in the flue gas, the extent of contact between the carbon and mercury, and the type of carbon used. Flue gas temperature, as noted above, is important because mercury is a vapor at temperatures above 150 to 200°C [300 to 400°F]. The flue gas temperature needs to be within, or preferably below, this range for the mercury to adsorb onto the carbon. The combustion device and the corresponding composition of the flue gas will affect this temperature range. In an MWC, where there is a substantial amount of HCl resulting in the formation of HgCl$_2$, temperatures within and below the noted range have proven to be effective when injecting carbon. Test data from an MWC retrofitted with activated carbon injection indicate mercury removals greater than 95 percent. Pilot testing on a coal-fired utility boiler indicated that a temperature under 90 to 120°C (200 to 250°F) was necessary for effective mercury removal.

With activated carbon injection, efficient distribution of the carbon in the flue gas is also important. The amount of carbon needed to achieve a specific level of mercury removal will vary depending on the fuel being burned, the amount of carbon inherent to the system and the type of PM control device. At a given carbon feed rate, a fabric filter provides more mercury control than an electrostatic precipitator (ESP) because of the additional mercury adsorption that occurs on the bags of the fabric filter (due to the increased gas contact time). Mercury is predominately removed upstream of an ESP-equipped facility where a nominal residence time of one second or less is available, limiting the capture. In addition, mercury is not effectively collected across the ESP, further requiring substantially higher carbon feed rates than the fabric filter-equipped facilities.

Activated carbon injection is applicable to MWCs, MWIs and utility boilers. There are considerable data describing the effectiveness of activated carbon injection for controlling mercury emissions from MWIs and MWCs. These data have been previously published by U.S.
Activated carbon injection for utility boilers has been recently studied by U.S. EPA resulting in a few EPA publications in the last seven years. Activated carbon injection may also be technically feasible for smelters. No information, however, is available on the use of activated carbon injection for smelters.

The effectiveness of activated carbon injection in controlling mercury emissions from MWCs has been demonstrated by the U.S. EPA. The application of activated carbon injection to utility flue gas, however, cannot be directly scaled from the application at MWCs due to differences in the amount and composition of flue gas at utility plants and MWCs. At utility plants, small concentrations of mercury are contained in a large volume of flue gas, and large amounts of activated carbon are needed to provide adequate contact between the carbon particles and mercury. The differences in flue gas characteristics at MWCs and utility plants must be carefully examined before considering any technology transfer assumptions.

The level of mercury control achieved in utility flue gas may depend upon flue gas characteristics such as volume, temperature, flyash, chloride, and mercury content. These properties are distinctly different from those in MWC flue gas.

Typical MWC flue gas is hotter than utility flue gas after leaving an air preheater. The air preheater cools the utility flue gas by transferring heat to the incoming combustion air. Moreover, the mercury concentration of the two gas streams differs significantly. Mercury concentrations in MWC flue gas streams may be up to several orders of magnitude greater than those seen in utility flue gas streams. Likewise, the chloride content of MWC flue gas may be from 1.4 to 400 times greater than the content seen in utility flue gas. Finally, with regard to the volume of flue gas, a utility boiler may have flow rates up to 30 times that of an MWC.

12.9.5 Mercury Controls for Coal-Fired Utility Boilers

Mercury is volatilized and converted to elemental mercury (Hg$^0$) in the high temperature regions of combustion devices. As the flue gas cools, Hg$^0$ is oxidized to oxidized mercury (Hg$^{++}$). The rate of oxidization is dependent on the temperature, flue gas composition and properties, and amount of flyash and any entrained sorbents. In coal-fired combustors, where the concentrations of hydrogen chloride (HCl) are low, and where equilibrium conditions are not achieved, Hg$^0$ may be oxidized to mercuric oxide (HgO), mercuric sulfate (HgSO$_4$), mercuric chloride (HgCl$_2$), or some other mercury compound. The oxidization of Hg$^0$ to HgCl$_2$ and to other ionic forms of mercury is abetted by catalytic reactions on the surface of flyash or sorbents and by other compounds that may be present in the flue gas. Applications of nitrogen oxides (NO$_X$) control technologies such as selective catalytic reduction (SCR) can assist in oxidation of Hg$^0$. 

12-28
Hg$^0$, HgCl$_2$, and HgO are primarily in the vapor phase at flue gas cleaning temperatures. Therefore, each of these forms of mercury can potentially be adsorbed onto porous solids such as flyash, powdered activated carbon, and other sorbents for subsequent collection in a PM control device. These mercury forms may also be captured in carbon bed filters or other reactors containing appropriate sorbents.

Mercury removal with wet scrubbers also appears to be possible. HgCl$_2$ is water-soluble and reacts readily with alkali metal oxides in an acid-base reaction; therefore, conventional acid gas scrubbers used for SO$_2$ control can also effectively capture HgCl$_2$. However, Hg$^0$ is insoluble in water and must be adsorbed onto a sorbent or converted to a soluble form of mercury that can be collected by wet scrubbing. HgO has low solubility and probably has to be collected by methods similar to those used for Hg$^0$. Therefore, the form of mercury that is most easily removed is HgCl$_2$ and this form of mercury is most readily formed when burning coals that are higher in chlorine content, such as Eastern bituminous coals. Furthermore, the equipment on the boiler also plays an important role in determining mercury speciation. For this reason coal type, coal chlorine content, and the boiler equipment all play a significant role in determining the ease with which mercury can be removed from coal combustion flue gas streams.

Approximately 77 percent of the coal-fired utility boilers currently operating in the United States are equipped with only an ESP or a fabric filter (FF). Gaseous mercury (both Hg$^0$ and Hg$^{++}$) can potentially be adsorbed on flyash and be collected in a downstream ESP or FF. The modern ESPs and FFs that are now used on most coal-fired units achieve very high capture efficiencies for total PM. As a consequence, these PM control devices are also effective in capturing PM-bound mercury in the boiler flue gases.

It is currently believed that mercury is primarily adsorbed onto the unburned carbon in flyash. Approximately 80 percent of the coal ash in pulverized coal (PC) fired boilers is entrained with the flue gas as flyash. PC-fired boilers with low-NO$_x$ burners have higher levels of carbon in the flyash with a correspondingly higher potential for mercury adsorption. Cyclone and stoker boilers tend to have high levels of carbon in the flyash but have lower flue gas concentrations of flyash than PC-fired boilers. Flyash concentrations in fluidized-bed combustors tend to be higher than those in PC-fired boilers. Also, the carbon content of fluidized-bed combustor flyash is generally higher than that of PC-boiler flyash.

Gas-phase mercury in units equipped with an ESP can be adsorbed on the entrained flyash upstream of the ESP. The gas-phase mercury in units equipped with a FF can be adsorbed by entrained flyash or it can be adsorbed as the flue gas passes through the filter cake on the surface of the FF. The degree to which gaseous mercury adsorbs on the filter cake typically depends on
the speciation of gaseous mercury in the flue gas; in general, gaseous Hg$^{++}$ is easier to adsorb than gaseous Hg$^{0}$. The very intimate contact between the gas and collected PM (which can act as a sorbent for the gas-phase mercury) that occurs in a FF significantly enhances the gas-phase mercury collection efficiency of the FF over what is possible with an ESP. For both bituminous and subbituminous coals, mercury collection in boilers equipped only with FFs is much higher than for boilers equipped only with ESPs. This effect also contributes to much more efficient collection of mercury when activated carbon is injected for additional mercury control upstream of a FF as opposed to injection upstream of an ESP.

Plants which employ only post-combustion PM controls display average Hg emission reductions ranging from 0 percent to 89 percent. The highest levels of control were observed for units with FFs. Decreasing levels of control were shown for units with ESPs, particulate scrubbers, and mechanical collectors. The average mercury reduction for two PC-fired units equipped with a FF baghouse and burning bituminous coal averaged 90 percent, while two similarly equipped units burning subbituminous coals displayed an average mercury reduction of 72 percent. The average capture of Hg for PC-fired plants equipped with an ESP was 35 percent for bituminous coal, 3 percent for subbituminous coal, and near zero for lignite.

Currently, there are two main approaches being considered for controlling power plant mercury emissions:

1) Reducing mercury emissions using technologies primarily designed to remove SO$_2$, NO$_X$, and particulate emissions (often called co-benefit reductions), and

2) Reducing mercury emissions using technologies specifically designed to reduce mercury.

While many approaches are being considered, the most common technology discussed to remove mercury from coal plants is powdered activated carbon injection (ACI). ACI systems have been widely deployed in other industries, mainly in waste-to-energy plants (municipal solid waste (MSW) plants). In those applications, they have achieved mercury removal rates in excess of 90 percent. However, ACI systems are only now being widely tested on U.S. coal plants and these plants have several characteristics that will tend to make mercury removal more difficult. For example, coal plants are typically much bigger with more flue gas to treat. They also have much lower concentrations of mercury and chlorine in the untreated gas, and it is questionable whether similar removal levels will be achievable for all coals. Sulfur and trace elements in U.S. coals may also pose problems that will have to be resolved. For example, efforts to remove mercury could create corrosive conditions that would damage other parts of the plants. Presently, the
U.S. Department of Energy’s Office of Fossil Energy has established programs that are actively exploring these issues.

The capacity of powdered activated carbon to adsorb mercury is large enough that it should not be limiting except at temperatures of about 350 °F (177 °C) or more, which is greater than the gas temperature at the exit of most air preheaters. So, with the possible exception of lignite coals, cooling usually has little or no beneficial effect on mercury absorption by activated carbon. However, the ability of flyash and unburned carbon in the flyash to absorb mercury is far less than that of activated carbon and may be enhanced by cooling. Therefore, although spray cooling may enhance mercury adsorption by flyash and downstream capture in the ESP or FF, it is not expected to enhance mercury capture by activated carbon except in the case where lignite coals are burned. Over 90 percent of the coals burned in the United States are bituminous, subbituminous, or blends of bituminous and subbituminous, which are not likely to use spray cooling with activated carbon injection.

Based on published literature, control technologies using powdered activated carbon injection into the flue gas appear to hold promise for reducing mercury emissions from utility boilers. Full-scale and pilot-scale tests indicate that these technologies may be able to provide significant mercury removal from the flue gas of coal-fired utility boilers.

Other technologies have shown promise for control of mercury, one being a coal treatment technology known as K-Fuel. K-Fuel is a beneficiated coal that is derived from western coal. The resulting fuel is lower in ash, higher in Btu value, and produces lower pollutant emissions than untreated western subbituminous coals. K-Fuel uses a pre-combustion process that improves the quality of the coal—including removing the mercury, moisture, ash, sulfur, and some of the fuel NOX precursors—before the coal is burned at the power plant. Because these constituents are removed prior to combustion, the need for post-combustion controls may be reduced. K-Fuel has been tested on utility boilers, and a commercial production plant is under construction.
References


Appendix: Summarized MACTs for Petroleum Refineries and Synthetic Organic Chemical Manufacturing Industry

The purpose of this section is to highlight some of the control technologies that are required by the MACTs for two selected industrial categories: petroleum refineries and synthetic organic chemical manufacturing industry. Sometimes, the MACTs may require the use of a specific control device (i.e., flare); but, the majority of the time, the MACT will not specify the control device mandated. Instead the MACT may require a certain reduction of HAPs (i.e., 98% reduction of HAPs or an outlet concentration no greater than 20 ppmv). Sometimes the MACT required control technology is unique to the source. For example, storage vessels may be required to have internal floating roofs with a double seal system and utilize specified deck fittings.

A.1: National Emission Standards for Hazardous Air Pollutants from Petroleum Refineries, 40 CFR 63, Subpart CC

Applicability: All process vents, storage vessels, wastewater streams and treatment operations, equipment leaks, gasoline loading racks, and marine vessel loading racks that are located at major sources and that handle one or more HAPs listed in the subpart.

Standard: Process Vents (Group 1)

- Reduce emissions using a flare that operates with no visible emissions and meets 40 CFR 63.11(b); or

- Reduce emissions using a control device by 98 percent by weight or to an outlet concentration of 20 ppmv, whichever is less stringent.

Standard: Storage Vessels (Group 1)

- The provisions for Group 1 storage vessels are essentially the same as 40CFR60, Subpart Kb.

Standard: Wastewater (Group 1): Comply with the provisions of 40CFR61, Subpart FF, for tanks, surface impoundments, containers, individual drain systems, oil-water separators, treatment processes, and closed vent systems and control devices. The basic requirements of these provisions are as follows:
- **Tanks:** Install, operate, and maintain a fixed-roof and closed-vent system that routes all organic vapors vented from the tank to a control device.

- **Surface Impoundments:** Install, operate, and maintain on each surface impoundment a cover and closed-vent system that routes all organic vapors vented from the impoundment to a control device.

- **Containers:** Install, operate, and maintain a cover on each container used to handle, transfer or store waste; transfer waste into the container using a submerged fill pipe; use a closed-vent system and route vapors to a control device if waste treatment is performed in the container.

- **Individual Drain Systems:** Install, operate, and maintain on each drain system opening a cover and closed-vent system that routes all organic vapors vented from the drain system to a control device; or equip each drain with water-seal controls or a tightly-sealed cap or plug; equip each junction box with a cover and use water seal controls or a closed-vent system on any vent pipe; and cover or enclose each sewer line.

- **Oil-Water Separators:** Install, operate, and maintain a fixed-roof and closed-vent system that routes all organic vapors vented from the oil-water separator to a control device.

- **Treatment Processes:** Install, operate, and maintain a treatment process that either reduces benzene in the waste stream to less than 10 ppmw, or removes 99% or more of the benzene on a mass basis, or destroys the benzene by incineration in a combustion unit that achieves a destruction efficiency of 99% or greater; seal all openings in the treatment system and keep them closed at all times waste is being treated.

- **Closed-Vent Systems and Control Devices:** The closed-vent system must operate with no detectable emissions. If the control device is a combustion unit, it must provide a minimum residence time of 0.5 seconds at a minimum temperature of 1,400°F, or reduce organic emissions by 95 weight percent or greater, or achieve an outlet concentration of 20 ppmv. If the control device is a vapor recovery system, it must control organic compounds with an efficiency of 95 weight percent or greater and benzene emissions with an efficiency of 98 weight percent or greater.
**Standard**: Equipment Leaks

- Existing sources shall comply with 40 CFR 60, Subpart VV.
- New sources shall comply with 40 CFR 63, Subpart H.

**Standard**: Gasoline Loading Racks

- Comply with 40 CFR 63, Subpart R.

**Standard**: Marine Tank Vessel Loading

- Comply with 40 CFR 63, Subpart Y. In general, this subpart requires that each terminal be equipped with a system that collects vapors displaced during the loading of a marine tank vessel and that prevents vapors collected at one loading berth from being discharged to the atmosphere through another loading berth. The collected vapors must then be reduced by 95% to 98%, depending on the applicable section, before being emitted to the atmosphere.


**Subpart F**: **Applicability provisions** (Sect. 63.100) - the SOCMI plant:

- Must be a major source.

- Produce one of the chemicals listed in Table 1 of Subpart F (Note: Table 1 is a broader list than Table 2 because it includes non-HAPs toxic chemicals).

- Use or produce one of the chemicals in Table 2 of Subpart F (Note: Table 2 is a list of organic HAPs regulated by HON -131 chemicals listed).

- Exemptions (i.e., does not apply to petroleum refineries process units).

**Subpart G**: Control standards for all process units (including storage vessels).

**Subpart H**: Standards for equipment leaks.
Examples of HON control requirements for HON storage vessels are as follows:

**HON applicability for storage vessels:** A "storage vessel" is a tank or other vessel that is used to store liquid organic HAP's and is assigned to a chemical manufacturing process subject to the HON. Group 1 and Group 2 storage vessels are defined in §63.119 of Subpart G. The vessel's design capacity and the vapor pressure of the stored liquid are used to determine whether a storage vessel is Group 1 or Group 2. Group 1 storage vessels at existing sources are storage vessels with capacities of 75 m\(^3\) or greater but less than 151 m\(^3\) storing liquids with a vapor pressure of total organic HAP of 13.1 Kilopascal (kPa) or greater. Storage vessels at existing sources with capacities of 151 m\(^3\) or greater storing liquids with a vapor pressure of total organic HAP of 5.2 kPa or greater are also Group 1 storage vessels. Group 1 storage vessels at new sources are storage vessels with capacities or 38 m\(^3\) or greater but less than 151 m\(^3\), storing liquids with a vapor pressure of total organic HAP of 13.1 kPa or greater. Storage vessels at new sources with capacities of 151 m\(^3\) or greater storing liquids with a vapor pressure of total organic HAP of 0.7 kPa or greater are also Group 1 storage vessels.

**HON Storage Vessel Control Requirements:** Group 1 storage vessels must meet the control requirements in §63.119 of Subpart G unless they are included in an emissions average. Compliance options for Group 1 storage vessels include:

- Reducing emissions of organic HAP's using a fixed-roof tank equipped with an internal floating roof which is operated according to specified work practices (i.e., keeping access hatches closed and bolted), equipped with specified deck fittings, and equipped with specified seal configurations (i.e., a single liquid-mounted seal, a single metallic shoe seal, or double seals).

- Reducing emissions of organic HAP's using an external floating roof tank operated according to specified work practices, equipped with specified deck fittings, and equipped with specified seal configurations (i.e., double seals, with the primary seal to be either a liquid-mounted or a metallic shoe seal).

- Reducing emissions of organic HAP's using an external floating roof tank converted to a fixed-roof tank, equipped with an internal floating roof, which is operated according to specified work practices, equipped with specified deck fittings, and equipped with specified seal configurations (i.e., a single liquid-mounted seal, a single metallic shoe seal, or double seals).
- Reducing emissions of organic HAP's by 95 weight percent using a closed vent system (i.e., vapor collection system) and control device or combination of control devices (or reducing emissions of organic HAP's by 90 weight percent using a closed-vent system and control device if the control device was installed before December 31, 1992).

- Reducing emissions of organic HAP's by routing the emissions to a process or a fuel gas system, if emissions are routed to a process the emissions must meet one of the same ends listed for transfer operation emissions routed to a process or fuel gas system.
References
