CHAPTER 4

Air Quality Health Impacts and Risks

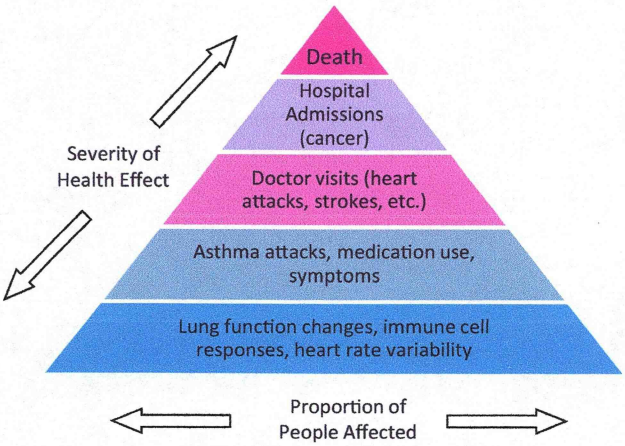
This chapter serves as a primer on understanding potential air pollutant health impacts and health risks.

4.1 Pollutant Health Impacts Overview

Each of the pollutants targeted in this report can be categorized as either a criteria pollutant or a hazardous air pollutant (HAP). HAPs are also referred to as air toxics or as both criteria pollutants and HAPs (e.g., Lead, Pb, is regulated as a criteria pollutant but Pb-based compounds are on the EPA's HAPs list). Each of these pollutants has health effects that range from mild to severe chronic and acute health effects, as well as premature death. Figure 4-1 provides an overview of the population proportions associated with the severity of health effects—in general, the more severe the effect, the smaller the proportion of the population affected. The figure describes different degrees of health effects, and it should be understood that different pollutants will have different health impacts and levels of severity. The following sections describe the potential health effects of each pollutant.

There are six (6) criteria pollutants. A discussion of concerns over their public health impacts follow:

- **Carbon monoxide (CO)** is a colorless and odorless gas that can cause various physiological damages by displacing oxygen in the bloodstream. At high concentrations, CO has known health effects including dizziness, unconsciousness, and death. At lower concentrations more typical of ambient settings in the United States, individuals with cardiovascular disease are at risk of myocardial infarctions (heart attacks) or other exacerbations.
- **Lead (Pb)** is a soft, malleable metal in the "heavy metal" category. Pb is a concern for its ability to cause a range of neurological damage from all exposure pathways (inhalation, ingestion, and dermal contact).
- **Nitrogen dioxide (NO_2)** is one of the nitrogen oxides (NO_x) that is part a family of gases, mainly represented by NO and NO_2, that can contribute to respiratory disease exacerbations. In addition to its direct health impacts, NO_x is well known as a precursor to ozone (O_3) formation. Furthermore, NO_x also contributes to the formation of nitrate aerosols that can have respiratory and cardiovascular health effects.
- **Ozone (O_3)** is a pollutant that generally is not directly emitted from most sources. Within the troposphere, it is formed through a complex interaction (chemical reaction) mainly involving NO_x, and volatile organic compounds (VOCs) in the presence of sunlight. O_3 can contribute to respiratory health effects through inflammation of airways and decrements in lung function, with evidence of increased respiratory symptoms among sensitive individuals such as asthmatics and those with chronic obstructive pulmonary disease, as well as evidence of increased



Source: Adapted from Environmental Health & Engineering, Inc. (EH&E) (2011). "Emissions of Hazardous Air Pollutants from Coal-Fired Power Plants." EH&E Report 17505. Prepared for the American Lung Association, Washington, D.C., March 7.

Figure 4-1. Severity of health effects versus proportion of people affected.

hospitalizations and premature deaths. Because of the formation of O_3 from directly emitted pollutants (from many different sources) within a relatively large area, O_3 is characterized as a regional issue even though it is a local air quality concern.

- **Particulate matter (PM)** is tiny solid, liquid, or mixed solid and liquid particles suspended in the air. These are of concern since ambient concentrations of PM have been shown to be correlated with serious respiratory and cardiovascular illnesses and premature mortality. PM sizes (aerodynamic diameters) range from greater than 100 μm to the ultrafine range of below 0.1 μm. The smaller the size, the deeper they are able to penetrate into the respiratory system, possibly even resulting in blockages of the gas–blood interfaces within the lungs. Figure 4-2 provides an overview of the portions of the respiratory system affected by the different PM size ranges. While the discrete PM size ranges shown generally correspond to different degrees of respiratory penetration, it should be understood that different size ranges can be deposited

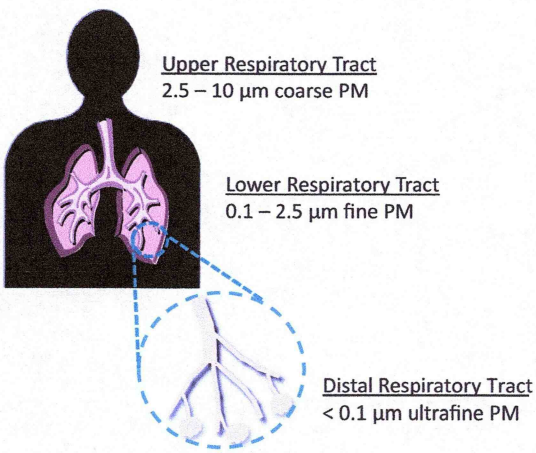


Figure 4-2. PM penetration into the human respiratory system.

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throughout the respiratory system. PM with a size range of 10 μm or less are referred to as PM_{10} and those with a size range of 2.5 μm or less are referred to as $PM_{2.5}$. NAAQS concentrations are currently only specified for these two size ranges. In addition to these regulated size ranges, PM in the ultrafine range (less than 0.1 μm in diameter) is thought to contribute to health effects. Ultrafine particles are of particular concern at airports because of relatively higher concentrations (higher than background) found near aircraft operations. Other PM types and components include nitrates, sulfates, and black carbon (BC). Also known as elemental carbon (EC), BC is composed of pure carbon clusters and is differentiated from organic carbon (OC), which is composed of organic compounds. BC is a significant contributor to the health effects caused by $PM_{2.5}$ and ultrafines. Nitrates and sulfates can penetrate deep in the respiratory system and can also react with other chemicals to form harmful compounds (e.g., acids).

- **Sulfur dioxide (SO_2)** is a sulfur oxide (SO_x). So_x refers to a family of gases mainly represented by SO_2 that can act as irritants to the respiratory system and can contribute to asthma attacks and other health outcomes. As with NO_x, concerns about SO_x often relate to its ability to form sulfate aerosols in the atmosphere, with the corresponding health effects seen for fine particulate matter.

HAPs are generally defined as those pollutants that are known or suspected of being able to cause serious health effects such as cancer, birth defects, etc. The EPA maintains a list of close to 200 HAPs comprised of VOCs, aldehydes, polycyclic aromatic hydrocarbons (PAHs), dioxins, furans, metals, acids, etc. A discussion of the formation and concerns over these pollutants follows:

- **Volatile organic compounds (VOCs)** are comprised of a large group of carbon-based compounds with relatively high vapor pressures. The EPA further defines these as chemicals that participate in atmospheric photochemical reactions. They are emitted through evaporation from certain operations (e.g., painting, dry cleaning, etc.) and through incomplete combustion of fossil fuels. Indoor concentrations of VOCs are usually higher than outdoor concentrations—up to 10 times higher. Health effects depend on the specific species as well as exposure duration, but some short-term effects may include headaches, nausea, sore throat/eyes/nose, etc. Long-term effects may include cancer. Examples of VOCs include benzene, toluene, xylene, 1,3-butadiene, etc.
- **Aldehydes and ketones** are subsets of VOCs. Sometimes they are treated separately, which is in part due to the different methods required to measure these compounds. Both groups of compounds are made up of a double-bonded carbon-oxygen core (C=O). An aldehyde has at least one hydrogen bonded to the carbon atom while a ketone has two hydrocarbon groups attached to the carbon atom. Aldehydes are used in production of commercial applications including the production of alcohols, resins, detergents, perfumes, etc. Ketones have industrial uses as solvents, polymer precursors, and pharmaceuticals, etc. As VOCs, both groups have relatively high vapor pressures, and their health effects are similar: irritation of the eyes and air passages under short-term exposure and lower concentrations. Long-term exposures and/or high concentrations can cause depressions of the central nervous system and cancer. Examples of aldehydes are formaldehyde, acrolein, and acetaldehyde. Examples of ketones are acetone and acetophenone. Methyl ethyl ketone (MEK) is also a ketone but not a HAP—EPA removed this from their official list.
- **Polycyclic aromatic hydrocarbons (PAHs)** are comprised of a group of compounds that generally have more than two benzene rings (a ring of six carbon atoms). They tend to stick to solid particles (e.g., soot) and are formed from incomplete combustion processes such as those from coal burning, automobile gasoline combustion, forest fires, coke and coal tar processing, etc. Animal testing has indicated that it is reasonable to expect PAHs to cause birth defects and cancer. Examples of PAHs include anthracene, benzo-a-pyrene, naphthalene, chrysene, etc. Of these, only naphthalene is currently listed on the EPA HAPs list.

- **Dioxins and furans** are comprised of a family of toxic substances that are similar in chemical structure and more formally referred to as polychlorinated dibenzo-para-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs). In addition to exposures through ingestion of food containing these compounds, exposures through inhalation of emissions from incineration (e.g., of municipal solid waste), copper smelters, cement kilns, coal-fired power plants, etc., are common. Potential health effects include birth defects, suppressed immune system, changes in hormone levels, and cancer. On the EPA HAPs list, these pollutants are listed as 2,3,7,8-Tetracholordibenzo-p-dioxin and dibenzofurans.
- **Metals** make up a small but important portion of the EPA HAPs list. Either in elemental form or as part of a compound, they can typically be emitted as PM from combustion sources including power plants, industrial operations, ore refining, etc. Three of the common metals are mercury (Hg), lead (Pb), and chromium (Cr). Exposure to air emissions of Hg can result in various disorders including tremors, emotional changes, neuromuscular changes, changes in nervous response, reductions in cognitive function, etc. As previously indicated, exposures to Pb can result in neurological damage. Air exposure to Cr (III), the most common form of chromium in the air, can result in damage to the respiratory system. Exposure to Cr (VI) can result in more serious respiratory damage, as well as lung cancer.
- **Acids** make up a small subset of the EPA HAPs list, and hydrochloric acid (HCl) and hydrogen fluoride (HF) are two of the more well-known HAPs. In addition to being used in various industrial activities such as refining ore, metals processing, glass etching, aluminum production, etc., they also can be generated through combustion of coal and other fuels containing chlorine (Cl) and fluorine (F). Acute health effects of these acids are similar in that they are corrosive and can cause serious damage to the respiratory system. Chronic effects for HCl include gastritis, bronchitis, and dermatitis as well as hyperplasia of the nasal mucosa, larynx, and trachea. HF chronic effects include increased bone density and damage to the liver, kidneys, and lungs.

To ensure no misunderstandings regarding these health effects, it should be noted that while the descriptions provide a comprehensive view of the current understanding of health impacts by pollutant type (or category), they do not directly indicate the risks associated with airport air quality impacts. Other details such as emissions and exposure need to be taken into account and are discussed in the next section.

4.2 Health Risk Factors

As defined by the EPA (see http://www.epa.gov/risk_assessment/basicinformation.htm), health risk is "the chance of harmful effects to human health or to ecological systems resulting from exposure to an environmental stressor" where stressors are described as "any physical, chemical, or biological entity that can induce an adverse response." Characterizations of risk are accomplished by conducting both exposure pathway assessments (how the pollutant interacts with the population) and dose-response assessments (how much of the pollutant is required to cause harm). These are general definitions used to describe risk for environmental impacts.

For air quality, health risk can be described as being influenced by three components: emissions, exposure, and toxicity. As indicated in Figure 4-3, each of these components encompasses details regarding the source, pollutants, and the exposed public. The emissions of each pollutant depend on source characteristics. Source characteristics include emission factors (or rates) that are dependent on type of source, equipment age, emissions control, etc. Toxicity is the degree to which a pollutant can harm a human being. Toxicity is characterized differently for criteria air pollutants versus HAPs. For criteria air pollutants, concentration–response relationships are generally constructed from epidemiological literature. These epidemiological studies typically contain concentrations representative of the current range of concentrations in the United States, and the concentration–response functions are applied as continuous functions to quantify

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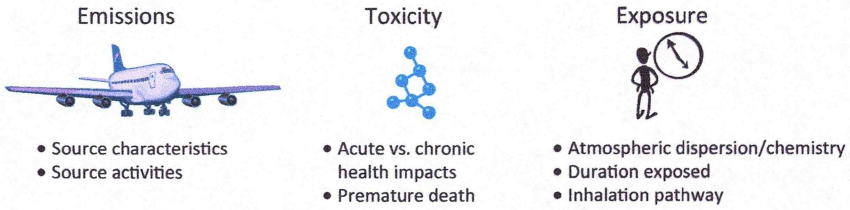

Figure 4-3. Air quality health risk components.

incremental health effects of concentration changes. For HAPs, conventional risk assessment differentiates between carcinogenic effects and non-cancer effects, with the presumption that most carcinogens demonstrate low-dose linearity and that most non-cancer health effects display a putative population threshold. There are an increasing number of counterexamples that contradict this model, but most health risk assessments to date maintain this structure. Within the structure, for non-cancer health effects, inhalation reference concentrations (RfCs) are used. For carcinogenic effects, unit risk factors are used. The EPA defines these terms as follows:

- RfC: An estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure of a chemical to the human population through inhalation (including sensitive subpopulations), that is likely to be without risk of deleterious noncancer effects during a lifetime. (See http://www.epa.gov/ttn/atw/hlthef/hapglossaryrev.html.)
- Unit Risk: A unit risk is an upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 μg/L in water or 1 μg/m^3 in air. (See http://www.epa.gov/iris/help_ques.htm.)

Exposure encompasses both the pathway leading to the interaction between pollutants and the exposed population (i.e., concentrations experienced by the population) as well as the duration of the interaction. This is partly dependent on how pollutants disperse in the atmosphere and undergo chemical conversions to form other pollutants. It also is dependent on the size and activities of the local population and their locations.

In general, the health impacts from specific sources can be evaluated from either an individual perspective or a population perspective, and this holds for airport emissions as well. In the former case, the influential factors will be those that cause an individual to have greater risk from airport emissions than other individuals. In the latter case, the influential factors will be those that cause the public health burden from airport emissions to be greater. The factors will overlap but will not be identical.

From an individual perspective, proximity to the airport is clearly the dominant factor, although not necessarily in a simple distance-dependent fashion. Multiple studies indicate that being immediately downwind of a primary departure runway significantly increases exposures to multiple combustion pollutants, including ultrafine particulate matter, NO_x, and black carbon. However, some studies indicate the potential for exposure over a fairly broad geographic area, especially related to arrivals—appreciable impacts can be observed more than 1 km from the airport, in a manner that is not strictly distance-dependent. The common influence of wind direction on aircraft movement patterns and plume dynamics creates challenges in interpreting monitoring data, but location relative to prevailing winds is clearly an important factor for individual risk. When spatiotemporal patterns differ across pollutants, which locations are most important from an individual health perspective are more difficult to ascertain, but evidence shows similar patterns across most pollutants with major public health implications. The one major exception is ozone, which has a large public health burden but is generally reduced in close proximity to airports given the significant local contribution of NO_x emissions.

From a population perspective, proximity and prevailing winds clearly influence the population health burden from airport emissions as well, but population density and spatial patterns of at-risk populations also must be considered. For example, pollutants such as fine particulate matter (with significant contributions from secondary formation) may have public health impacts that can span hundreds (or thousands) of kilometers. Thus, even if individual health impacts may be greatest at relatively close proximity to an airport, the public health impacts will be spread over a very large geographic area where the characteristics of the exposed population needs to be taken into account. That is, health impacts will be influenced not only by exposures, but also health status and other factors that make individuals or subpopulations more susceptible to the effects of air pollution. Elderly individuals and young children, as well as those with pre-existing respiratory or cardiovascular disease, are generally considered to be at greatest risk. That main point is that population-based health assessments that take into account the exposed area and population characteristics may show differing results from an individual perspective where distance is the major factor.

Two general approaches can be used to estimate the public health burden associated with either an individual source (such as an airport) or a source category (such as LTO emissions). Epidemiological investigations involve developing new associations between exposures and health outcomes for a defined population, which can be interpreted as causal given supporting evidence from other epidemiological and toxicological studies. There have been numerous epidemiological studies evaluating ambient air pollution and its effects on respiratory and cardiovascular health, and the methods for conducting these studies are well established in the literature. However, epidemiological studies rarely associate air pollution specific to aviation with health outcomes. This is both because the contribution from aviation to ambient air pollution is generally small and because the pollutants associated with aviation are similar to those from traffic and other local combustion sources. There have been a limited number of occupational epidemiological studies of airport workers, which can better capture exposures specific to the airport environment but may not generalize to the public given differences in exposure levels and health status.

Because direct epidemiological studies of air pollution specific to airports are generally impractical, it is far more common to use health risk assessment methods to quantify the health impacts of airport emissions. These methods typically involve bottom-up analyses linking airport emissions inventories with atmospheric fate-and-transport models, yielding estimates of the marginal contribution of airport emissions to ambient air quality across a region. These contributions are then linked with concentration–response functions for mortality and morbidity, derived from the general air pollution epidemiological literature. In other words, air pollution epidemiology provides the association between specific pollutants and health outcomes, and this evidence is assumed to be applicable to airport-related air pollution. For pollutants that do not differ by source, this approach has fewer uncertainties, beyond exposure assessment uncertainties and general concerns about whether the epidemiological evidence can be interpreted as causal. For fine particulate matter, where the composition from aviation may differ from the ambient composition in a manner that influences health effects, there are additional uncertainties. However, the application of ambient air pollution epidemiology to determine contributions from specific source categories is a well-established approach in the health risk assessment literature, and constituent-specific epidemiology could be used when available and based on statistical models appropriate for health risk assessment.