# APPENDIX C

# AIR QUALITY BACKGROUND INFORMATION

# CRITERIA AIR POLLUTANTS

This appendix presents a brief description of the criteria air pollutants ozone, carbon monoxide, nitrogen dioxide, sulfur dioxide, and particulate matter, including information on adverse health effects and formation processes.

## Ozone

Ozone ($O_3$) is a photochemical oxidant and the primary component of smog. $O_3$ is not directly emitted into the air but is formed through complex chemical reactions between precursor emissions of organic compounds and oxides of nitrogen in the presence of sunlight. Both organic compounds and oxides of nitrogen are emitted by mobile (transportation) and stationary (industrial) sources. $O_3$ located in the upper atmosphere (stratosphere) acts in a beneficial manner by shielding Earth from harmful ultraviolet radiation emitted by the sun. However, $O_3$ located in the lower atmosphere (troposphere) is a major health and environmental concern. Because sunlight and heat serve as catalysts for reactions between $O_3$ precursors, peak $O_3$ concentrations typically occur during summer in the northern hemisphere (EPA 2002). In general, $O_3$ concentrations over or near urban and rural areas reflect an interplay of emissions of ozone precursors, transport meteorology, and atmospheric chemistry (Godish 1991).

The adverse health effects associated with exposure to $O_3$ primarily pertain to respiratory systems. Scientific evidence indicates that ambient levels of $O_3$ affect not only sensitive receptors, such as asthmatics and children, but also healthy adults. Exposure to ambient levels of $O_3$ ranging from 0.10 to 0.40 part per million (ppm) for 1–2 hours has been found to significantly alter lung functions by increasing respiratory rates and pulmonary resistance, decreasing tidal volumes, and impairing respiratory mechanics. Ambient levels of $O_3$ above 0.12 ppm are linked to symptomatic responses such as throat dryness, chest tightness, shortness of breath, headache, and nausea. In addition, evidence also exists relating $O_3$ exposure to an increase in the permeability of respiratory epithelia, leading to an increase in responsiveness of the respiratory system to bronchoconstrictive challenges, and the interference or inhibition of the immune system's ability to defend against infection (Godish 1991).

## Carbon Monoxide

Carbon monoxide (CO) is a colorless, odorless, and poisonous gas produced by incomplete burning of carbon in fuels, principally from mobile sources of pollution. In fact, 77% of the nationwide CO emissions are from mobile sources. The remaining 23% consists of CO emissions from wood-burning stoves, incinerators, and industrial sources. Peak CO levels are

localized near areas with high concentrations of mobile sources and occur typically during winter months when calm air conditions (i.e., not windy) are common.

CO enters the bloodstream through the lungs by combining with hemoglobin, which normally supplies oxygen to the cells. However, CO combines with hemoglobin much more readily than oxygen does, resulting in a drastic reduction in the amount of oxygen available to the cells. Adverse health effects associated with exposure to CO concentrations include dizziness, headaches, slow reflexes, and fatigue. CO exposure is especially harmful to individuals who suffer from cardiovascular and respiratory diseases (EPA 2002).

**Nitrogen Dioxide**

Nitrogen dioxide ($NO_2$) is a brownish, highly reactive gas present in all urban environments. The major anthropogenic (human-made) sources of $NO_2$ are combustion devices, such as boilers, gas turbines, and mobile and stationary reciprocating internal combustion engines. Combustion devices primarily emit nitric oxide (NO), which reacts with oxygen in the atmosphere to form $NO_2$ (EPA 2002). The combined emissions of NO and $NO_2$ are referred to as oxides of nitrogen ($NO_X$), which are reported as equivalent $NO_2$. Because $NO_2$ is formed and depleted by reactions associated with photochemical smog ($O_3$), the $NO_2$ concentration in a particular geographical area may not be representative of the local $NO_X$ emission sources.

Inhalation is the most common route of exposure to $NO_2$. Because $NO_2$ has relatively low solubility in water, the principal site of toxicity is in the lower respiratory tract. The severity of the adverse health effects depend primarily on the concentration inhaled rather than the duration of exposure. An individual may experience a variety of acute symptoms, including cough, difficulty breathing, vomiting, headache, and eye irritation during or shortly after exposure. After a period of approximately 4–12 hours, an exposed individual may experience chemical pneumonitis or pulmonary edema with breathing abnormalities, cough, hemoptysis, cyanosis, chest pain, and rapid heartbeat. Severe, symptomatic $NO_2$ intoxication after acute exposure has on occasion been linked with prolonged respiratory impairment with such symptoms as chronic bronchitis and decreased lung functions.

**Sulfur Dioxide**

Sulfur dioxide ($SO_2$) is produced by such stationary sources as coal and oil combustion, steel mills, refineries, pulp and paper mills, and nonferrous smelters. The major adverse health effects associated with $SO_2$ exposure pertain to the upper respiratory tract. $SO_2$ is a respiratory irritant with bronchoconstriction occurring with inhalation of $SO_2$ at 5 ppm or more. On contact with the moist mucous membranes, $SO_2$ produces sulfurous acid, which is a direct irritant. Concentration rather than duration of the exposure is an important determinant of

respiratory effects. Exposure to high concentrations of $SO_2$ may result in edema of the lungs or glottis and respiratory paralysis.

**Particulate Matter**

Respirable particulate matter 10 micrometers or less in diameter is referred to as $PM_{10}$. $PM_{10}$ consists of particulates directly emitted into the air, such as fugitive dust, soot, and smoke from mobile and stationary sources, construction operations, fires and natural windblown dust, and particulates formed in the atmosphere by condensation and/or transformation of sulfur dioxide and reactive organic gases (EPA 2002).

The adverse health effects associated with $PM_{10}$ depend on the specific composition of the particulate matter. For example, health effects may be associated with metals, polycyclic aromatic hydrocarbons, and other toxic substances adsorbed onto fine particulates, which is referred to as the piggybacking effect, or with fine dust particles of silica or asbestos. Generally, adverse health effects associated with $PM_{10}$ may result from both short-term and long-term exposure to elevated $PM_{10}$ concentrations and may include breathing and respiratory symptoms, aggravation of existing respiratory and cardiovascular diseases, alterations in the body's immune system, carcinogenesis, and premature death (EPA 2002).

**References**

Godish, T. 1991. *Air Quality*. Second edition. Lewis Publishers. Chelsea, MI.

U.S. Environmental Protection Agency (EPA). 2002. Available <www.epa.gov/ttn/chief/ap42/index.html>. Accessed 2002.