APPENDIX C

Overview of Airborne Metals Regulations, Exposure Limits, Health Effects, and Contemporary Research

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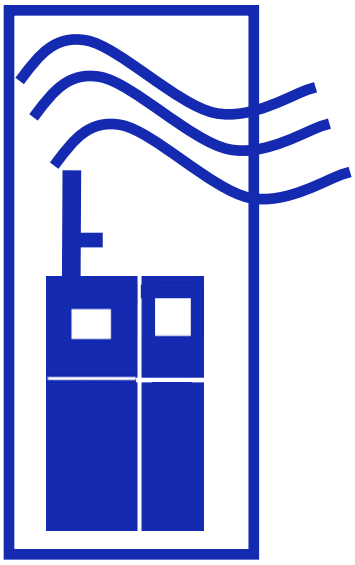
December 3, 2010



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Health Effects Summary

One of the consequences of the current state of industrialization and an increasing demand for modern conveniences and improved quality of life has been an increased exposure to air pollutants from industrial activities, traffic, and energy production. Regulatory bodies such as federal, state, and local environmental protection agencies are responsible for assuring the public that the air is safe to breathe. These agencies are required to set standards, levels, and/or goals that will protect public health with an adequate margin of safety. These standards are established not only to protect healthy individuals, but also to protect sensitive population subgroups, such as children, asthmatics, the elderly, and individuals with emphysema, chronic obstructive pulmonary disease, or other conditions that render the group particularly vulnerable to air pollution. Although there is only one metal National Ambient Air Quality Standard (NAAQS) for lead, there are numerous other workplace and community-based screening levels, exposure limits, and reference concentrations for airborne metals that can be used as guidelines to set acceptable and appropriate levels of exposure and concern.

Assessing risk for metals in ambient air is difficult for a variety of reasons. Because organisms have always been exposed to metals, unlike synthetic organic substances, organisms have developed various means of responding to metals. There are major differences between the persistence of metals or inorganic metal compounds in the body and the persistence of organic compounds. Metals are neither created nor destroyed by biological and chemical processes, but may be biotransformed from one chemical species to another. That is, the metal ion thought to be responsible for the toxicity of a metal may persist in the body regardless of how the metal is metabolized. Some metals are considered essential for normal metabolic function, which is one of the primary factors that differentiate risk assessment for metals and metal compounds from that of synthetic organic chemicals.

Exposure to metals in the air is capable of causing a myriad of human health effects, ranging from cardiovascular and pulmonary inflammation to cancer and damage of vital organs. Contemporary research into air pollution is revealing that the metals components of particulate matter (PM) are contributing significantly to adverse health effects, even at the low concentrations found in ambient air. The EPA set health-based standards for fine particulates in 1997, but the standards do not take into account new research on the composition of the particulate matter or the toxicity of its components. The toxicity of particulate matter, in particular the fine (1 to 2.5 microns [µm]) and ultrafine particles (0.1 to 1 µm), has been proven to cause severe mortality and morbidity in humans over the past 25 years; however, in the past decade, emerging research is providing evidence that the metallic particles may be more dangerous than other PM components. In fact, current evidence is showing that mass concentration of PM alone may not be the best indices for associating health effects with exposure to PM.

The aerodynamic size and associated composition of particles determine their behavior in the mammalian respiratory system. Furthermore, particle size is one of the most important parameters in determining the atmospheric lifetime of particles, which may be a key consideration in assessing inhalation exposures, as well as exposures related to exposure pathways involving deposition onto soil or water. Metals emitted by combustion processes (e.g., the burning of fossil fuels or wastes) generally occur in small particles or the fine fraction, which is often characterized by particles less than 2.5 µm in diameter ($PM_{2.5}$). In contrast, the larger sized, course mode particles result from mechanical disruption, such as crushing, grinding, evaporation of sprays, or suspensions of dust from construction and agricultural operations. Accordingly, metals in course mode particles (i.e., those larger than approximately 1–3 µm) are primarily those of crustal origin, such as aluminum, zinc, and iron.

Generally, the evaluation of most studies shows that the smaller the size and greater the solubility of the PM, the higher the toxicity through mechanisms of oxidative stress and inflammation. A study of $PM_{2.5}$ in 2010 showed that metals were the important source for cellular oxidant generation and subsequent health effects. Health effects are stronger for fine and ultrafine particles for a variety of reasons:

- The studies of the size distribution of metals show that most of the toxic metals accumulate in the smallest particles ($PM_{2.5}$ or less).
- This size fraction can penetrate deeper into the airways of the respiratory tract and predominantly deposits in the alveolar region of the lungs, where the adsorption efficiency for trace elements varies from 60–80%.
- A fine metallic particle in contact with lung tissue/cells involves the release of metal ions into the biological system.
- Ultrafine particles are known to have increased solubility, as compared to larger size particles of the same composition because of the increased surface-to-volume ratio for smaller particle sizes.
- Fine and ultrafine particulate matter have the longest residence time in the atmosphere (~100 days), which allows for a large geographic distribution.
- Recent studies have shown that the metals component in fine and ultrafine PM is particularly toxic and are the primary contributors to negative human health.
- Furthermore, these particles also play a significant role in global climate change and can be transported over long distances by prevailing winds.

These consequences require us to give priority to the chemical characterization of the fine and ultrafine fraction of airborne particles to understand their possible implication to health effects.

In conclusion, for the effective management of air quality, great importance must be attached to the identification of both the sources and characterization of suspended PM. Source apportionment provides an estimate on the PM contribution of various sources to the levels at the receptor; it is also a key component necessary for developing and achieving desired air-quality objectives. The results of source apportionment can be used to evaluate emissions reduction on the PM levels and to devise more efficient emission reduction strategies. Therefore, estimating the airborne PM mass concentration, as well as individual chemical/metal speciation, is critical not only for comparing with recommended values, but also to identify the major sources that affect a particular area. This knowledge will also help regulators both foresee and prevent threats and risks before they become problems.

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1.1 Air Pollution History

Air pollution is not a modern concept; history clearly demonstrates that air pollution has been present for many centuries. Soot found on ceilings of prehistoric caves provides evidence of the high levels of pollution associated with inadequate ventilation of open fires. The forging of metals appears to be a key turning point in the creation of significant air pollution levels outside the home. Core samples of glaciers in Greenland indicate increases in pollution associated with Greek, Roman, and Chinese metal production. The United States (U.S.) Environmental Protection Agency (EPA) states that "an air pollutant is any substance in the air that can cause harm to humans or the environment. Pollutants may be natural or man-made and may take the form of solid particles, liquid droplets or gases." Currently, about four percent of deaths in the United States can be attributed to air pollution, according to the Environmental Science Engineering Program at the Harvard School of Public Health (Schwartz 2000).

In the past century, characterized by the industrial revolution, there are several key events that triggered the increase in air pollution monitoring and regulation. Several key air pollution event s occurred between the 1930's and early 1950's that prompted the development of clean air legislation both nationally and internationally. One initial event occurred in the Neuse Valley of Belgium in December 1930. A thermal inversion trapped fog over a 15-mile-long stretch of high-walled Meuse Valley that contained many farms, villages, steel mills, and chemical plants. At the end of the first day, many residents complained of nausea, shortness of breath, stinging eyes, and burning throats. After 3 days, 60 people had died and a thousand more were ill. The illness and deaths were caused by over thirty different chemical pollutants trapped beneath the dense fog clouds. Death rates were subsequently made ten times above normal (Anderson 2000).

The next event occurred in 1948 in Donora, Pennsylvania, an event also known as the "Donora Smog of 1948." Between October 26, and October 31, 1948 an air inversion trapped industrial effluent (air pollution) from the American Steel and Wire plant and Donora Zinc Works. Within three days, 20 people died; after the inversion lifted, another 50 died. Another 6,000 residents became sick from the fog and smoke combination; hundreds more finished the rest of their lives with damaged lungs and hearts (Pennsylvania DEP 2010).

Another key event was "The Great Smog of '52," a severe air pollution event that affected London, England in December 1952. A period of cold weather, combined with an anticyclone and windless conditions, collected airborne pollutants mostly from the use of coal to form a thick layer of smog over the city. It lasted from Friday to Tuesday, 9 December, 1952, and then quickly dispersed after a change in the weather. Although it caused major disruption due to the effect on visibility, and even penetrated indoor areas, it was not thought to be a significant event at the time, with London having experienced many smog events in the past. In the following weeks however, medical reports estimated that 4,000 had died prematurely and 100,000 more were made ill because of the smog's effects on the human respiratory tract. More recent research suggests that the number of fatalities was considerably higher at around 12,000 (Davis et al. and Bates 2002). It is considered the worst air pollution event in the history of the United Kingdom, and the most significant in terms of its impact on environmental research, government regulation, and public awareness of the relationship between air quality and health. It led to several changes in practices and regulations, including the U.K.'s Clean Air Act 1956.

An overview of U.S. regulations regarding metals and their presence in industrial emissions and ambient air is presented below. Information presented here was procured primarily from the Clean Air Act as written in the United States Code (USC n.d.), Title 42, Chapter 85, the EPA's "History of the Clean Air Act" (EPA 2008a) and "The Plain English Guide to the Clean Air Act" web pages (EPA 2008b).

1.2 Early Clean Air Act Legislation (1963 – 1967)

The Clean Air Act (CAA), similar to other environmental legislation, has continuously evolved. The federal government's first major efforts in regulating air emissions began in 1955 with the Air Pollution Control Act. This Act provided funds for federal research in air pollution. These efforts were enhanced over the next 15 years through a series of enactments, including the CAA. The CAA of 1963 was the first U.S. attempt to **control** air pollution and for the first time recognized pollution hazards from mobile source (cars, trucks, etc) emissions as well as stationary (industry, fireplaces, etc.) sources. The 1963 CAA also authorized research into techniques to minimize air pollution.

The CAA was amended in 1965 to establish motor vehicle emission standards and to promote research into the problem of transboundary pollution into Canada and Mexico. Amendments to the CAA in 1967, called the Air Quality Act (AQA), divided the nation into Air Quality Control Regions for monitoring and enforcement proceedings were initiated in areas subject to interstate air pollution transport. As part of these proceedings, the federal government for the first time conducted extensive ambient monitoring studies and stationary source inspections. The AQA also authorized expanded studies of air pollutant emission inventories, ambient monitoring techniques, and control techniques.

1.3 1970 and 1977 Clean Air Act (CAA) and Amendments

The Clean Air Act of 1970 (1970 CAA) resulted in a major shift in the federal government's role in air pollution control. It authorized the development of Federal and State regulations to limit emissions for both stationary and mobile sources. It created four different programs for controlling and preventing air pollution:

- The National Ambient Air Quality Standard (NAAQS),
- State Implementation Plans (SIP),
- New Source Performance Standards (NSPS),
- And National Emissions Standard for Hazardous Air Pollutants (NESHAPs).

These amendments occurred around the same time as the National Environmental Policy Act (NEPA), which established the EPA in May of 1971. The EPA was established to implement the requirements of the 1970 CAA.

The CAA lists four overarching goals or purposes for the legislation:

1. To protect and enhance the quality of the Nation's air resources so as to promote the public health and welfare and the productive capacity of its population;
2. To initiate and accelerate a national research and development program to achieve the prevention and control of air pollution;

3. To provide technical and financial assistance to State and local governments in connection with the development and execution of their air pollution prevention and control programs; and
4. To encourage and assist the development and operation of regional air pollution prevention and control programs.

The CAA requires regulation of emissions of hazardous air pollutants (HAPs) from a published list of industrial sources referred to as "source categories." HAPs, also known as toxic air pollutants or air toxics, are those pollutants that cause or may cause cancer or other serious health effects, such as reproductive effects or birth defects, or adverse environmental and ecological effects. This initial CAA recognized two types of stationary sources that generate routine emissions of HAPs:

- "Major" sources are defined as sources that emit 10 tons per year of any of the listed toxic air pollutants, or 25 tons per year of a mixture of air toxics. These sources may release air toxics from equipment leaks, when materials are transferred from one location to another, or during discharge through emission stacks or vents.
- "Area" sources consist of smaller-size facilities that release lesser quantities of toxic pollutants into the air. Area sources are defined as sources that emit less than 10 tons per year of a single air toxic, or less than 25 tons per year of a combination of air toxics. Though emissions from individual area sources are often relatively small, collectively their emissions can be of concern - particularly where large numbers of sources are located in heavily populated areas.

As required under the Act, both mobile and stationary source categories must meet control technology requirements for these HAPs. Development of regulations (also known as rules or standards) is required for all industries that emit one or more of the pollutants in significant quantities.

Amendments to the 1970 CAA occurred in 1977. These amendments authorized provisions related to the Prevention of Significant Deterioration and to areas which are non-attainment with respect to the NAAQS.

1.3.1 National Ambient Air Quality Standards

The 1970 CAAA required EPA to set NAAQS for wide-spread pollutants from numerous and diverse sources considered harmful to public health and the environment. The Clean Air Act established two types of national air quality standards. Primary standards set limits to protect public health, including the health of "sensitive" populations such as asthmatics, children, and the elderly. Secondary standards set limits to protect public welfare, including protection against visibility impairment, damage to animals, crops, vegetation, and buildings. The CAA requires periodic review of the science upon which the standards are based and the standards themselves.

EPA has set NAAQS for six principal pollutants, which are called "criteria" pollutants. They are carbon monoxide (CO), lead (Pb), nitrogen dioxide (NO_2), ozone (O_3), particulate matter (PM), and sulfur dioxide (SO_2). On November 12, 2008 EPA substantially strengthened the NAAQS for lead, which is thus far the only metal regulated through the NAAQS. EPA revised the level of the primary (health-based) standard from 1.5 micrograms per cubic meter ($\mu g/m^3$) to 0.15

$\mu g/m^3$, measured as total suspended particles (TSP) and revised the secondary (welfare-based) standard to be identical in all respects to the primary standard.

1.3.2 State Implementation Plans

As written in Section 107 of the 1970 CAAA, "Each State shall have the primary responsibility for assuring air quality within the entire geographic area comprising such State by submitting an implementation plan for such State which will specify the manner in which national primary and secondary ambient air quality standards will be achieved and maintained within each air quality control region in such State." The State Implementation Plan (SIP) is a plan for each State which identifies how that State will attain and/or maintain the primary and secondary NAAQS set forth in the CAA and which includes federally-enforceable requirements. Each State is required to have a SIP which contains control measures and strategies which demonstrate how each area will attain and maintain the NAAQS.

1.3.3 New Source Performance Standards

New Source Performance Standards (NSPS) are pollution control standards issued by the EPA. The term is used in the CAAA of 1970 to refer to air pollution emission standards and in the Clean Water Act (CWA) referring to standards for discharges of industrial wastewater to surface waters. NSPS dictate the level of pollution that a new stationary source may produce. An NSPS has been established for a number of individual industrial or source categories, such as landfills, boilers, petroleum refineries, and turbines.

An example describing the need for NSPS comes from the implementation of the CAAA in Ohio. Between the dates of 1970 and 1977, a rule in the CAA required a reduction in the measured SO_2 emitted by coal fired power plants into the air. Ohio decreased the SO_2 emitted by such plants by increasing the height of the smokestacks on the plants. The result was that the SO_2 was carried in the wind out of the state and there was a reduction in the locally measured SO_2. These kinds of exploits in the Clean Air Act were solved in the 1977 revision of the Clean Air Act, when the NSPS were introduced. NSPS measures the concentration and amount of pollution put into the air, thus making a taller smoke stack useless under the new standard.

1.3.4 National Emission Standards for Hazardous Air Pollutants

NESHAPS are stationary source standards for HAPs, which are those pollutants that are known or suspected to cause cancer or other serious health effects, such as reproductive effects or birth defects, or adverse environmental effects. NESHAPs are found in 40 CFR Part 61 (resulting from the CAAA of 1970) and 40 CFR Part 63 (resulting from the CAAA of 1990, see Section 1.4.1). Part 61 NESHAPs regulate only 7 hazardous air pollutants:

- Asbestos
- Beryllium
- Mercury
- Vinyl chloride
- Benzene
- Arsenic
- Radon/radionuclides

1.4 1990 Clean Air Act (CAA) Amendments

Another set of major amendments to the Clean Air Act occurred in 1990 (1990 CAAA). The 1990 CAAA substantially increased the authority and responsibility of the federal government. New regulatory programs were authorized for control of acid deposition (acid rain) and for the issuance of stationary source operating permits. The NESHAPs were incorporated into a greatly expanded program for controlling toxic air pollutants. The provisions for attainment and maintenance of NAAQS were substantially modified and expanded. Other revisions included provisions regarding stratospheric ozone protection, increased enforcement authority, and expanded research programs.

The 1990 CAA amendments required the EPA to regulate hazardous air pollutants through three inter-related programs: Maximum Achievable Control Technology (MACT) standards also known as NESHAPs, the Urban Air Toxics Strategy, and residual risk standards. The MACT rules establish performance-based standards for industrial sources to measure and control HAPs. Under the Urban Air Toxics strategy, smaller industrial sources of HAPs are regulated with MACT standards. Finally, EPA must review MACT standards for a source category at least every 8 years following promulgation and determine if there is any residual risk that requires the standards to be strengthened in order to protect public health. This ongoing process is comprehensively evaluated through National Air Toxics Assessments (NATA). Thus far, EPA has completed three assessments that characterize the nationwide chronic cancer risk estimates and noncancer hazards from inhaling air toxics. The latest NATA in 2002 was made available to the public in June of 2009 (EPA, EPA Technology Transfer Network (TTN) 2010).

1.4.1 Maximum Achievable Control Technology (MACT)

The NESHAPs promulgated after the 1990 CAAA are found in 40 CFR Part 63. These standards require application of technology- and performance-based emissions standards referred to as MACT. Consequently, these post-1990 NESHAPs are also referred to as MACT standards. MACT standards are designed to reduce HAP emissions to a maximum achievable degree, taking into consideration the cost of reductions and other factors. After the EPA adopts a MACT standard at the federal level, the Regulatory and Compliance Support Unit proposes the same standard for adoption at the state level by the Air Quality Control Division on a semi-annual basis.

When developing a MACT standard for a particular source category, the EPA looks at the current level of emissions achieved by best-performing similar sources through clean processes, control devices, work practices, or other methods. These emissions levels set a baseline, often referred to as the "MACT floor" for the new standard. 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. The EPA can establish a more stringent standard when it makes economic, environmental, and public health sense to do so.

The MACT floor differs for existing sources and new sources.

- For existing sources, the MACT floor must equal the average current emissions limitations achieved by the best-performing 12 percent of sources in the source category, if there are 30 or more existing sources. If there are fewer than 30 existing sources, the MACT floor must equal the average current emissions limitation achieved by the best-performing five sources in the category.

- For new sources, the MACT floor must equal the current level of emissions control achieved by the best-controlled similar source.

Wherever feasible, the EPA writes the final MACT standard as an emissions limit, i.e. a percent reduction in emissions or a concentration limit that regulated sources must achieve. Emissions limits provide flexibility for industries to determine the most effective ways to comply with the standards.

The NESHAPs are delegated to the states, but both EPA and the states implement and enforce these standards. Under the 1990 amendments, the state is required to develop regulations for all sources that emit significant quantities of one or more of the pollutants. In addition, the 1990 CAAA expanded the regulated HAPs from 7 to 191. Four chemicals, methyl ethyl ketone, caprolactam, glycol ethers, and hydrogen sulfide, have since been removed from the HAPs list. To date, EPA has compiled a list of 187 HAPs which include the following metals and metal compounds:

Antimony Compounds
Arsenic Compounds (inorganic including arsine)
Beryllium Compounds
Cadmium Compounds
Chromium Compounds
Cobalt Compounds
Lead Compounds
Manganese Compounds
Mercury Compounds
Nickel Compounds
Selenium Compounds

For all listings which contain the word "compounds" these listings are defined as including any unique chemical substance that contains the named chemical (i.e., antimony, arsenic, etc.) as part of that chemical's infrastructure.

HAPs in urban areas are of special concern because of the large number of people and the variety of pollution sources e.g., cars, trucks, large factories, gasoline stations, and dry cleaners (EPA, Technology Transfer Network (TTN) 2003). Eight of the above metals and their compounds (arsenic, beryllium, cadmium, chromium, lead, manganese, mercury, and nickel) are included in EPA's list of 33 HAPs, i.e. Urban Air Toxics, identified as posing the greatest potential environmental health threat in urban areas. Table C-1 presents federal agency, regional EPA, and selected state health limits.

Table C-1. Community Metals Concentrations of Concern (µg/m^3)

Metal	Typical U.S. National Ambient Air Concentrations[c]			EPA				ATSDR MRL			State Regulations			International
				IRIS		Region IX RSL					Oregon RBC	California REL		
	Rural	Urban	Industrial[d]	RfC	(10^{-6} cancer)	Cancer TR	Noncancer HI	Acute	Inter-mediate	Chronic	Residential	Acute	Chronic	European Union AQS
Antimony[a]	<0.001	0.032	**0.55**	0.2		-	0.21							
Arsenic[a]	**0.002**	**0.02**	**7.6**	0.03[e]	0.0002	0.00057	0.016				0.00057	0.2	0.015	0.006
Beryllium[a]	0.0001	**0.002**	**0.01**	0.02	0.0004	0.001	0.021				0.021		0.007	
Cadmium[a]	0.001	**0.008**	**0.6**	0.01	0.0006	0.0014	0.01	0.03		0.01	0.0014			0.005
Chromium[a]	0.002	0.02	**0.4**				0.01		0.1[h] (5[i])				0.2	
Chromium VI[a]	0.0001	**0.0016**	**0.0153**	0.008[f] (0.1[g])	0.00008	0.000011			0.005[f] (0.3[g])	0.005[f]	0.000029			
Cobalt[a]	0.0001	**0.0005**	**0.61**			0.00027	0.0063			0.1				
Copper	0.01	0.29	0.87									100		
Iron	0.3	1.6	7.0											
Lead [a,b]	0.02	**0.04**	**0.76**			0.03					(>Pv)			0.5
Manganese[a]	0.001	0.02	**0.3**	0.05			0.052			0.3	0.052		0.09	
Mercury[a]	0.0001	0.014	**0.041**	0.3			0.31			0.2	0.31	0.6	0.03	
Nickel[a]	0.006	**0.02**	**0.17**	0.05[e]	0.004[j] (0.002)[k]	0.01	0.052		0.2	0.09	0.0094	6	0.05	0.02
Selenium[a]	0.0001	0.015	0.03				21							
Silver	0.0005	0.004	0.037									5	20	
Vanadium	0.0008	**0.065**	**0.5**			0.1[l]	0.0073	0.8		0.1		30		
Zinc	0.006	0.103	5.0											

[a]Metals designated as Hazardous Air Pollutants by the EPA.

[b] National Ambient Air Quality Standard for lead is 0.15 µm/m^3 as a rolling three month average.

[c]Typical Urban Ambient Air Concentrations procured from the EPA, the ATSDR, the Hazardous Substances Database (HSDB), and/or the World Health Organization (WHO). The majority of the values are based on PM10 measurements taken in the 1980's and 1990's, but may include TSP or PM2.5 and/or more recent (post-2000) measurements. These values are not absolute; they are intended to represent typical concentrations found in urban environments.

[d] Industrial values are concentrations found in ambient air in the vicinity of factories and industry that emit metals; some values may be measurements collected near a particular factory rather than an average of all industrial vicinity ambient air concentrations.

[e] These values are calculated by California EPA

[f] Value for aerosols and mists

[g] Value for particulates

[h] Soluble particulate matter

[i] Insoluble particulate matter

[j] Value for Nickel refinery dust

[k] Value for Nickel subsulfide

[l] Value for metallic V, vanadium pentoxide value is 0.00029

Bold ambient air values exceed one or more of the regulatory health limits for that metal.

EPA = Environmental Protection Agency

IRIS = Integrated Risk Information System

10^{-6} Cancer = Dose at which risk of cancer is one in one million people (Based on the Inhalation Unit Risk)

RfC = Reference Concentration (noncancer)

RSL = Regional Screening Level

Cancer TR = Cancer target risk (1 in one miliion)

Noncancer HI = Noncancer hazard index

ATSDR = Association of Toxic Substances and Disease Registry

MRL = Minimal Risk Levels

RBC = Risk-based Concentration for residential receptors

REL = Reference Exposure Level

AQS = Air Quality Standard (Averaging period = 1 year)

>Pv = The air concentration reported for the RBC exceeds the vapor pressure of the pure chemical. It can be assumed that this constituent cannot create an unacceptable risk by this pathway.

1.4.2 Urban Air Toxics

In addition to MACT rules for major sources, 1990 Clean Air Act Amendments required the EPA to develop an Air Toxics Strategy that identifies 33 HAP of particular concern to urban areas, eight of which are metals and metal compounds (see Section 1.4.1). As required by the amendments, the EPA must identify and regulate the area source categories that represent 90% of the 33 HAP emissions. A total of 70 area source categories that contribute these HAP emissions had been identified and, as of June 2007, rules have been promulgated for 28 of them. Under the Urban Air Toxics Strategy, area sources in the following metal-specific categories were regulated with MACT rules:

Hazardous Waste Incineration
Medical Waste Incinerators
Municipal Waste Combustors
Other Solid Waste Incineration
Chromic Acid Anodizing
Decorative Chromium Electroplating
Hard Chromium Electroplating
Portland Cement Manufacturing
Secondary Aluminum Production
Secondary Lead Smelting
Mercury Cell Chlor-Alkali Plants
Primary Nonferrous Metal Production
Primary Copper Smelting
Secondary Copper Smelting
Chemical Manufacturing: Chromium Compounds
Lead Acid Battery Manufacturing

1.4.3 Residual Risk

Under the 1990 CAA Amendments, the EPA was required to submit a report to Congress regarding public health risks remaining following the implementation of the MACT standards, known as residual risks, and recommended legislation to reduce residual risks. This report, the Residual Risk Report to Congress, was submitted in March of 1999 (EPA, Office of Air Quality Planning and Standards 1999). The EPA is required to conduct risk assessments on each source category subject to MACT standards and determine if additional MACT standards called residual risk standards, i.e., lowered health limits "to protect the public health with an ample margin of safety or to prevent an adverse environmental effect," are necessary to reduce residual risk. As part of the residual risk requirements, EPA must review MACT standards for a source category at least every eight years following promulgation. To assess residual risk of HAPs, the EPA uses the following steps:

- Assessment of the public's exposure level
- Assessment of type and severity of adverse effects
- Dose-Response Assessment
- Overall Risk Characterization

Thus far, none of the residual risk standards involve sources that monitor metals or metal compounds; however, petroleum refineries and primary aluminum smelters are two source categories regulated for metals that are in the next group to undergo residual risk standard promulgation (National Lime Association v US EPA 2000).

1.5 Hazardous Waste Combustor Rule

The hazardous waste combustor rule was promulgated on October 12, 2005 and established national emission standards for HAPs for sources that burn hazardous waste, such as commercial and onsite incinerators, cement kilns, lightweight aggregate kilns, boilers, and hydrochloric acid production furnaces. The October 2005 rule limits emissions of:

- Dioxins and furans,
- Mercury,
- Semivolatile metals (cadmium and lead),
- Low volatile metals (arsenic, beryllium, and chromium),
- Particulate matter, as a surrogate for non-mercury metal, HAPs, including
    - Antimony
    - Manganese
    - Selenium
    - Nickel
    - Cobalt
- Hydrogen chloride and chlorine gas, and
- Organic HAPs

EPA estimated that hazardous waste combustors annually emit approximately 12,650 tons of HAPs (metals, total chlorine, organics, and dioxins/furans) and PM. Depending on the total number of facilities that comply, the total reduction of HAP and PM for existing sources was estimated to be between approximately 2,260 and 3,380 tons per year. EPA found that this rule will also protect human health and the environment by reducing PM in conjunction with the air toxics reductions (EPA 2009).

1.6 OSHA/NIOSH Worker Exposure Limits

The Occupational Safety and Health Act of 1970 was the first comprehensive industrial safety legislation passed at the federal level. The act was passed, in part, due to the rise in the number of work-related fatalities in the 1960s, and particularly the Farmington, West Virginia, mine disaster of 1968, in which 78 miners were killed. The Occupational Safety and Health Act was distinguished by its emphasis on the prevention of, rather than compensation for, industrial accidents and illnesses. The legislation provided for the establishment of the Occupational Safety and Health Administration (OSHA) and the National Institute of Occupational Safety and Health (NIOSH). Among the key provisions of the act were the development of mandatory safety and health standards, the enforcement of these standards, and standardized record-keeping and reporting procedures for businesses.

OSHA and NIOSH developed exposure limits designed to protect a worker through both acute and chronic exposure scenarios. The NIOSH recommended exposure limits (RELs) indicates a time-weighted average (TWA) concentration for up to a 10-hour workday during a 40-hour workweek. A short-term exposure limit (STEL) is a 15-minute TWA exposure that should not be exceeded at any time during a workday. A ceiling REL should not be exceeded at any time. TWA concentrations for OSHA permissible exposure limits (PELs) must not be exceeded during

any 8-hour work shift of a 40-hour workweek. An additional screening value used to protect workers is the Immediately Dangerous to Life and Health (IDLH) concentration. IDLH exposure conditions are defined as "conditions that pose an immediate threat to life or health, or conditions that pose an immediate threat of severe exposure to contaminants, such as radioactive materials, which are likely to have adverse cumulative or delayed effects on health." The IDLH is considered a maximum concentration above which only a highly reliable breathing apparatus providing maximum worker protection should be permitted (NIOSH 2007).

Occupational exposure limits for HAP metals and those metals of concern to human health are listed in Table C-2. Definitions of the various exposure limits presented in Tables C-1 and C-2 are provided in Table C-3.

1.7 Consent Decrees and Surrogates

A consent decree is a judicial decree expressing a voluntary agreement between parties to a suit, especially an agreement by a defendant to cease activities alleged by the government to be illegal in return for an end to the charges. Many EPA standards and rules are promulgated as a result of consent decrees. For example, the Not-To-Exceed (NTE) standard promulgated by the United States Environmental Protection Agency (EPA) ensures that heavy-duty engine emissions are controlled over the full range of speed and load combinations commonly experienced in use. NTE standards were created by the EPA as a result of a consent decree between the EPA and several major diesel engine manufacturers. As part of the resulting consent decree settlement with the EPA, these manufacturers were assessed heavy fines and were subjected to new emissions standards which included NTE (EPA, Office of Transportation and Air Quality 2005).

For many source categories, the EPA requires measurement and control of particulate matter (PM) as a surrogate for metal HAP. Reasons for use of PM as a surrogate for metal HAP include:

1. Metal HAP emitted from combustion sources are incorporated in the fly-ash PM and therefore the same techniques used to measure and control PM are effective for metal HAP.
2. Since different fuels generally all emit PM but vary in the type and amount of metal HAP they emit, using PM as a standard eliminates the need for many different standards based on fuel changes.
3. Using PM as a surrogate eliminates the need for performance testing of numerous standards for individual metals and therefore reduces costs.

U.S. courts allow EPA to use PM as a surrogate for metals if a “Three-Part Test” consisting of the following is passed:

1. If HAP metals are “invariably present” in stack emissions.
2. If PM control technology “indiscriminately captures HAP metals along with other particulates.”
3. If PM control technology is the only method facilities use to reduce HAP emissions.

PM measurement methods are not discussed here but may be found on the EPA’s Emission Measurement Center’s web site (EPA, Technology Transfer Network 2010).

Table C-2. Occupational/Industrial Limits for Metals of Concern (µg/m^3)

Metal	Carcinogen?	IDLH	NIOSH REL (10-hr TWA)	OSHA PEL (8-hr TWA)
Antimony [a]	No	50,000	500	500
Arsenic [a,b]	Yes	500	2[b]	10
Beryllium [a,c]	Yes	400	0.5	2
Bismuth [d]	No	N.D.	5	5
Cadmium [a]	Yes	900	N.E.	0.005
Chromium [a]	No	250,000	0.5	1
Chromium III [a]	No	2,500	0.5	0.5
Chromium VI [a]	Yes		0.001	0.005
Cobalt [a]	No	20,000	0.05	0.1
Copper [e]	No	100,000	1	1
Lead [a,f]	No	100,000	50	50
Manganese [a,g]	No	500,000	1000	5,000
Mercury [a,h]	No	10,000	0.1	100
Nickel [a,i]	Yes	10,000	15	1000
Selenium [a,j]	No	100	200	200
Silver	No	10,000	10	10
Vanadium [k]	No	35,000	50	50

IDLH = Immediately Detrimental to Life and Health

NIOSH = National Institute of Occupational Safety and Health

REL = Recommended Exposure Limit

OSHA = Occupational Safety and Health Administration

PEL = Permissible Exposure Limit

[a] Metals designated as Hazardous Air Pollutants by the EPA.

[b] NIOSH REL for arsenic is a 15-minute ceiling

[c] OSHA PEL for beryllium has a 30-minute ceiling of 5 µg/m^3

[d] REL and PEL for bismuth is a respiratory limit, the total REL is 10 µg/m^3 and total PEL is

[e] Additional REL of 0.1 and PEL of 0.1 for copper fume

[f] NIOSH REL for lead is an 8-hour TWA standard

[g] NIOSH short term exposure limit (STEL) for manganese is 3,000 µg/m^3 and the PEL is a

[h] NIOSH REL for mercury for skin is 50 µg/m3 and the REL is a ceiling

[i] Nickel as $Ni(CO)_4$ has an IDLH of 14,000 µg/m^3 and an REL and PEL of 7 µg/m^3

[j] Selenium as SeF_6 has an IDLH of 2000 µg/m^3 and an REL and PEL of 400 µg/m^3

[k] NIOSH REL for vanadium is a 15-minute limit

Table C-3. Definition of Risk Levels

Regulatory or Risk Level	Acronym	Agency	Definition
Residential			
Reference Concentration	RfC	EPA - IRIS	A RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from a no adverse effect level (NOAEL), low adverse effect level (LOAEL), or benchmark concentration, with uncertainty factors generally applied to reflect limitations of the data used. Generally used in EPA's noncancer health assessments.
10^{-6} Cancer Risk	10^{-6} Cancer	EPA - IRIS	This dose concentration is based on Inhalation Unit Risk (IUR), which is the upper-bound excess lifetime cancer risk estimated to result from continuous exposure to an agent at a concentration of 1 µg/m^3 in air. The interpretation of inhalation unit risk would be as follows: if unit risk = 2 × 10-6 per µg/m^3, 2 excess cancer cases (upper bound estimate) are expected to develop per 1,000,000 people if exposed daily for a lifetime to 1 µg of the chemical per m^3 of air.
Regional Screening Level	RSL	EPA (Region IX)	The residential RSLs (for air) consider human exposure to individual contaminants in air. Generic screening levels are calculated for multiple exposure pathways and for chemicals with both carcinogenic and noncarcinogenic effects. Cancer target risk (TR) is based on one in a million cancer risk and the noncancerous hazard index (HI) is based on a Hazard Quotient (HQ) of 1.
Minimal Risk Level	MRL	ATSDR	An MRL is an estimate of the daily human exposure to a hazardous substance that is likely to be without appreciable risk of adverse noncancer health effects over a specified duration of exposure. MRLs are derived when reliable and sufficient data are available to identify target organs of effect, or the most sensitive health effects for acute (1-14 days), intermediate (15-364 days), and chronic (365 days or longer) exposure durations and for the oral and inhalation routes of exposure (only noncancer effects considered). These substance specific estimates, which are intended to serve as screening levels, are used by ATSDR health assessors and other responders to identify contaminants and potential health effects that may be of concern at hazardous waste sites. It is important to note that MRLs are not intended to define clean up or action levels for ATSDR or other Agencies.
Risk-Based Concentration	RBC	Oregon DEQ	The RBCs are listed separately for each possible pathway (air inhalation, groundwater ingestion, soil ingestion, etc) to human exposure. If any constituent concentrations exceed the minimum RBC for a given exposure pathway that exposure pathway must be explored and eliminated from concern.
Reference Exposure Level	REL	CalEPA/OEHHA*	The concentration level at or below which no adverse health effects are anticipated for a specified exposure duration is termed the reference exposure level (REL). RELs are based on the most sensitive, relevant, adverse health effect reported in the medical and toxicological literature. RELs are designed to protect the most sensitive individuals in the population by the inclusion of margins of safety. Since margins of safety are incorporated to address data gaps and uncertainties, exceeding the REL does not automatically indicate an adverse health impact. The acute REL is calculated for a one-hour exposure period; the chronic REL is intended to be protective for individuals exposed continuously over an entire lifetime. From a practical standpoint, chronic exposure for humans is considered to be greater than 12% of a lifetime of 70 years, thus, human exposures of greater than 8 years are considered chronic exposures and are not adjusted either in their calculation or application.
Air Quality Standard	AQS	European Union	The European Union has developed an extensive body of legislation which establishes health based standards and objectives for a number of pollutants in air. The standards apply over differing periods of time because the observed health impacts associated with the various pollutants occur over different exposure times. Member States should undertake assessments of air pollution levels using measurements and modelling and other empirical techniques. Where levels are elevated, the Member States should prepare an air quality plan or program to ensure compliance with the limit value before the date when the limit value formally enters into force.
Occupational			
Immediately Dangerous to Life and Health	IDLH	NIOSH/OSHA	The IDLH is considered a maximum concentration above which only a highly reliable breathing apparatus providing maximum worker protection should be permitted. In determining IDLH values, NIOSH considered the ability of a worker to escape without loss of life or irreversible health effects along with certain transient effects, such as severe eye or respiratory irritation, disorientation, and incoordination, which could prevent escape. As a safety margin, IDLH values are based on effects that might occur as a consequence of a 30-minute exposure. However, the 30-minute period was NOT meant to imply that workers should stay in the work environment any longer than necessary; in fact, EVERY EFFORT SHOULD BE MADE TO EXIT IMMEDIATELY!
Recommended Exposure Limit	REL	NIOSH	The REL is a level that NIOSH believes would be protective of worker safety and health over a working lifetime if used in combination with engineering and work practice controls, exposure and medical monitoring, posting and labeling of hazards, worker training and personal protective equipment. For NIOSH RELs, "TWA" indicates a time-weighted average concentration for up to a 10-hour workday during a 40-hour workweek.
Permissible Exposure Limit	PEL	OSHA	The PEL is a legal limit in the United States for exposure of an employee to a chemical substance or physical agent. TWA concentrations for OSHA PELs must not be exceeded during any 8-hour workshift of a 40-hour workweek
Short-Term Exposure Limit	STEL	NIOSH/OSHA	The STEL is a 15-minute TWA exposure that should not be exceeded at any time during a workday
Ceiling	C	NIOSH/OSHA	A ceiling REL or PEL is designated by "C" preceding the value; unless noted otherwise, the ceiling value should not be exceeded at any time.

For industrial sources that use PM as a surrogate for metals, emissions of individual metals are not estimated at all, and the concentration of metals emitted from the source is therefore unknown. Metals are present in PM in trace quantities, i.e., metals are typically measured in the micrograms per dry standard cubic meter (µg/dscm) and PM is measured in mg/dscm. The concentration of metals in PM can therefore change considerably, e.g., by a factor of 2 or more, without impacting the overall bulk concentration of PM. Concentrations of metals in PM can change with different fuels and therefore do not remain in constant proportion to the PM. Clearly, using PM as a surrogate for metals allows for substantial uncertainty in actual metal emissions. The EPA accepts the substitution of PM for metals because it is simpler and cheaper than creating standards and conducting performance tests for individual metals, and because the EPA assumes that the same techniques used to measure and control PM are effective for metal HAPs. With current scientific knowledge pointing towards metal components of PM as a significant contributor to adverse human health effects, the speciation of PM is important in protecting human health and the environment. In addition, technology currently exists that is capable of cost-effectively assessing the metals components of PM (see Guide for Developing a Multi-Metals Fence Line Monitoring Plan for Fugitive Emissions Using X-Ray Based Monitors, Cooper Environmental Services, 2010).

1.8 State Guidelines and Goals

State Implementation Plans (SIP), as created in the 1970 CAAA, requires states to monitor and control air pollutants. Air quality guidelines are available both as Regional EPA standards and state-by-state guidelines for both chronic and acute health effects, as well as for residential and occupational/industrial receptors. The SIP consists of narrative, rules, technical documentation, and agreements that an individual state will use to clean up polluted areas. SIPs also include special control strategies for nonattainment areas, i.e. areas that are not meeting the NAAQS. These control strategies often include items such as vehicle inspection and maintenance, lower gasoline vapor pressures, gas pump vapor recovery, and other reasonably available control technologies (RACT). Finally, SIPs include preconstruction permit requirements for projects that may result in emission increases (EPA, State Implementation Plan 2010). Examples of state exposure limits are presented in Table C-1.

1.9 Summary of Non-US Standards and Limits

Air pollution does not recognize state or international boundaries; international air standards have been developed for countries across all continents. The U.S. EPA, NOAA, NPS, tribal, state, and local agencies developed the AIRNow web site (www.AIRnow.gov) to provide the public with easy access to national air quality information. The Web site offers daily air quality index (AQI) forecasts, as well as real-time AQI conditions for over 300 cities across the US, and provides links to more detailed state and local air quality web sites. In addition, the AIRNow web site includes links to international air quality web sites from Australia and Asia to Europe and South America. An additional source of compiled international guidelines, International Toxicity Estimates for Risk (ITER), is available through the U.S. National Library of Medicine's Toxicology Data Network (ITER 2009). This database includes risk information for over 600 chemicals from authoritative groups worldwide.

The United Kingdom government established nationwide air quality measurement networks in the late 1970s. Metals in the air had been considered a problem with records describing diseases caused by breathing emissions from metal smelters from as far back as 1750 (Brown

2008). In 1996, the European Union (EU) published the Air Quality Framework Directive followed by a series of Daughter Directives (DD) that limited the concentrations of a range of metallic, inorganic, and organic pollutants in ambient air across EU member states. The first DD sets a limit value for the concentration of lead in the PM_{10} fraction of particulate matter, whilst the fourth DD sets target values for the concentrations of nickel, arsenic, and cadmium in the PM_{10} fraction of particulate matter, and the concentration of total gaseous mercury (TGM) in ambient air. The concentration of particulate phase mercury is not explicitly covered by the 4th DD. The DDs limit the allowable concentrations at individual monitoring sites.

The EU has developed occupational exposure limits similar to those found in the US. Frequently, international air quality objectives are often based, in part, on US standards and research. Air quality standards were found to be divided into two subsections – those standards developed to protect populations as a whole and standards developed to protect humans in the workplace. A comparison of air quality standards for occupational and residential scenarios between the United States, Australia, European Union, and Denmark showed that international limits were either similar or less stringent than those implemented in the United States (TOXNET 2010).

1.10 Expected Future Regulations

New knowledge concerning health effects of metals and their compounds is continuously uncovered. As new discoveries are made, available standards may be raised or lowered in keeping with current knowledge. A summary of contemporary research is included in Chapter 3 of this document.

The CISWI and Boiler MACT Rules

On July 8, 2007, the Court of Appeals for the District of Columbia vacated both the Commercial and Industrial Solid Waste Incinerators MACT rule (CISWI MACT) and the Industrial, Commercial and Institutional Boilers and Process Heaters MACT rule (Boiler MACT). The Court determined that the EPA improperly excluded commercial incinerators that recapture energy from the CISWI source category. Instead, the EPA classified these sources under the less stringent Boiler MACT Rule. The Court ruling requires EPA to rewrite the CISWI MACT to incorporate all commercial incinerators, including many of those formerly listed under the Boiler MACT. As a result, a large group of sources will no longer be regulated under the Boiler MACT and the Court has thus also required the EPA to rewrite the Boiler MACT rule. In the interim, the EPA has stated that it will apply MACT rules to boilers on a case-by-case basis. In addition, several states have their own Boiler MACT rules that will also apply.

Additional MACT Rules for Area Sources

Under the Urban Air Toxics Strategy, 28 rules have already been promulgated and additional rules for area sources “are under development or will be developed in the future” according to the EPA. The following list contains area source categories that are scheduled to be regulated in the future and may involve control and measurement of metal HAPs:

Sewage Sludge Incinerator Units
Pressed and Blown Glass Manufacturing
Secondary Nonferrous Metals
Stainless and Non-stainless Steel Manufacturing (EAFs)
Steel Foundries

Iron Foundries
Fabricated Metal Products, Electrical and Electronic Equipment - Finishing Op.
Fabricated Metal Products, Fabricated Metal Products, nec
Fabricated Metal Products, Fabricated Plate Work (Boiler Shops)
Fabricated Metal Products, Fabricated Structural Metal Manufacturing
Fabricated Metal Products, Heating Equipment, Except Electric
Fabricated Metal Products, Industrial Machinery and Equipment - Finishing Operations
Fabricated Metal Products, Iron and Steel Forging
Fabricated Metal Products, Primary Metal Products Manufacturing
Fabricated Metal Products, Valves and Pipe Fittings
Plating and Polishing
Ferroalloys Production: Ferromanganese and Silicomanganese
Industrial Inorganic Chemical Manufacturing
Inorganic Pigment Manufacturing
Misc. Organic Chemical Manufacturing (MON)
Brick and Structural Clay Products
Copper Foundries
Industrial Boilers Institutional/Commercial Boilers
Nonferrous Foundries

2.0 Overview of Airborne Metals Health Effects and Exposure Limits

2.1 Metals Overview

A metal is a chemical element that is a good conductor of both electricity and heat and that readily lose electrons to form cations and ionic bonds with non-metals. Metals occupy the bulk of the periodic table, while non-metallic elements can only be found on the right-hand-side of the Periodic Table of the Elements (Figure 1, below). A diagonal line, drawn from boron (B) to astatine (At), separates the metals from the nonmetals. Most elements on this line are metalloids, sometimes called semiconductors. This is because these elements exhibit electrical properties common to both conductors and insulators. Elements to the lower left of this division line are called metals, while elements to the upper right of the division line are called non-metals. Metals on the Periodic Table are further divided into alkali, alkaline earth, transitional, and post-transitional metals, as well as lathanoids and actinoids.

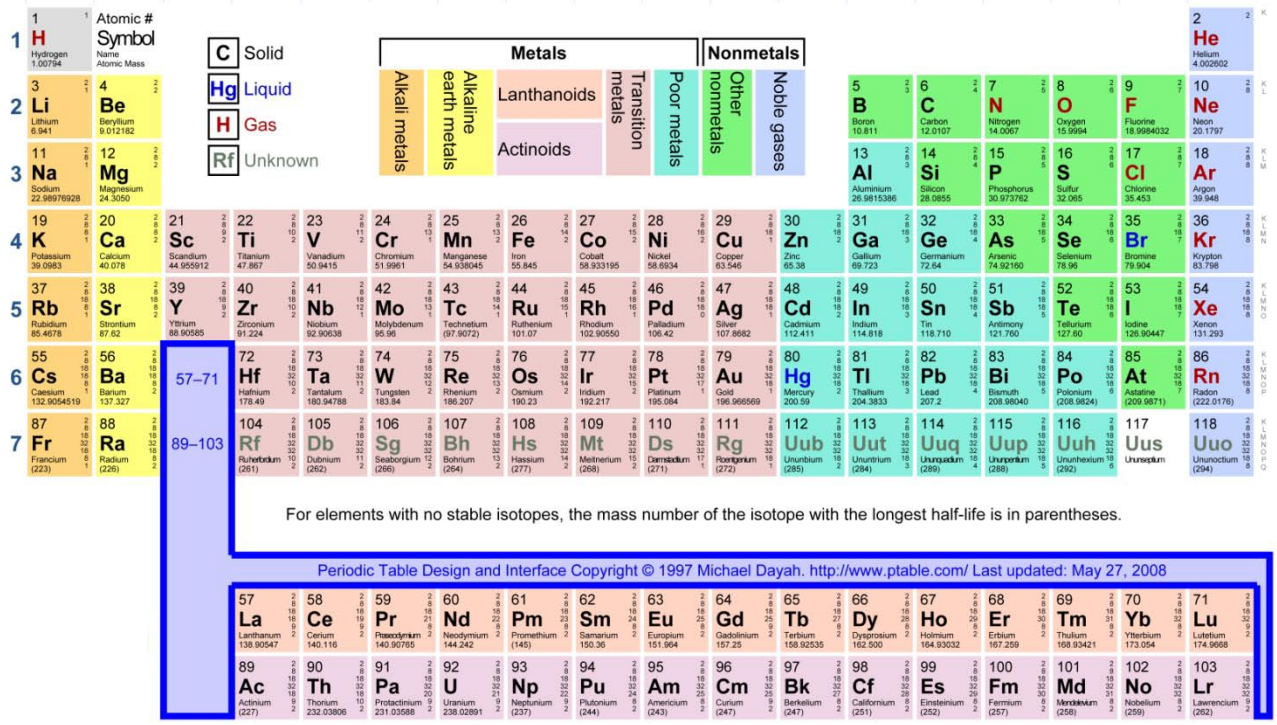

Figure C-1: Periodic Table Showing Metal, Metalloid, and Non-Metal Elements

Metals are found naturally in the environment at low-levels and some are essential nutrients for humans; however, certain types of metals and metals at high concentrations can have detrimental effects on human health and the environment. Because they cannot be degraded or destroyed, metals are persistent in all parts of the environment. Human activity affects the natural geological and biological redistribution of metals through pollution of the air, water, and soil. The primary anthropogenic sources of metals are point sources, such as mines, foundries, smelters, and coal-burning power plants, as well as diffuse sources, such as combustion by-products and vehicle emissions. Humans also affect the natural geological and biological

redistribution of metals by altering the chemical form of metals released to the environment. Such alterations often affect a metal's toxicity by allowing it to bioaccumulate in plants and animals, bioconcentrate in the food chain, or attack specific organs of the body. Monitoring and control of metal compounds in industrial emissions are thus critical for reducing exposure pathways and protecting human health and the environment.

Assessing risk for metals in ambient air is difficult for a variety of reasons. Because organisms have always been exposed to metals, unlike synthetic organic substances, organisms have developed various means of responding to metals. There are major differences between the persistence of metals or inorganic metal compounds in the body and the persistence of organic compounds. Metals are neither created nor destroyed by biological and chemical processes, but may be biotransformed from one chemical species to another. That is, the metal ion thought to be responsible for the toxicity of a metal may persist in the body regardless of how the metal is metabolized. Some metals are considered essential for normal metabolic function, which is one of the primary factors that differentiate risk assessment for metals and metal compounds from that of synthetic organic chemicals (Janssen 2001). Trace elements can be divided into three groups:

- Those known to be essential.
- Those that have beneficial metabolic effects but have not been shown to be essential.
- Those that occur widely in living organisms but seem to be only incidental contaminants, and are not known to be beneficial.

Several elements (e.g. sodium, potassium, magnesium, and calcium) occur in large concentrations in organisms. A second set of metals, termed trace metals, occurs at much lower concentrations (normally < 0.01%) in organisms. Some metals, such as iron, manganese, zinc, copper, cobalt, and molybdenum, have been identified as essential for all living organisms, while the essentiality of other metals, such as nickel, vanadium, iodine, chromium, and selenium, has only been established for a limited number of species.

Table C-4 classifies the metals addressed in this framework by their known essentiality to organisms.

Table C-4. Classification of Metals Based on Characteristics of Health Effects

Nutritionally Essential Metals	Metals with Possible Beneficial Effects	Metals with No Known Beneficial Effects
Cobalt Chromium III Copper Iron Manganese Molybdenum Selenium Zinc	Boron Nickel Silicon Vanadium Iodine	Aluminum Antimony Arsenic Barium Beryllium Cadmium Lead Mercury Silver Strontium Thallium

For those metals considered essential, toxicity can occur when the optimal concentration is exceeded; therefore, all metals are potentially toxic at a high enough dose. Figure 1 below depicts the dependence of biologic function on the tissue concentration of an essential trace element as modified by dietary intake.

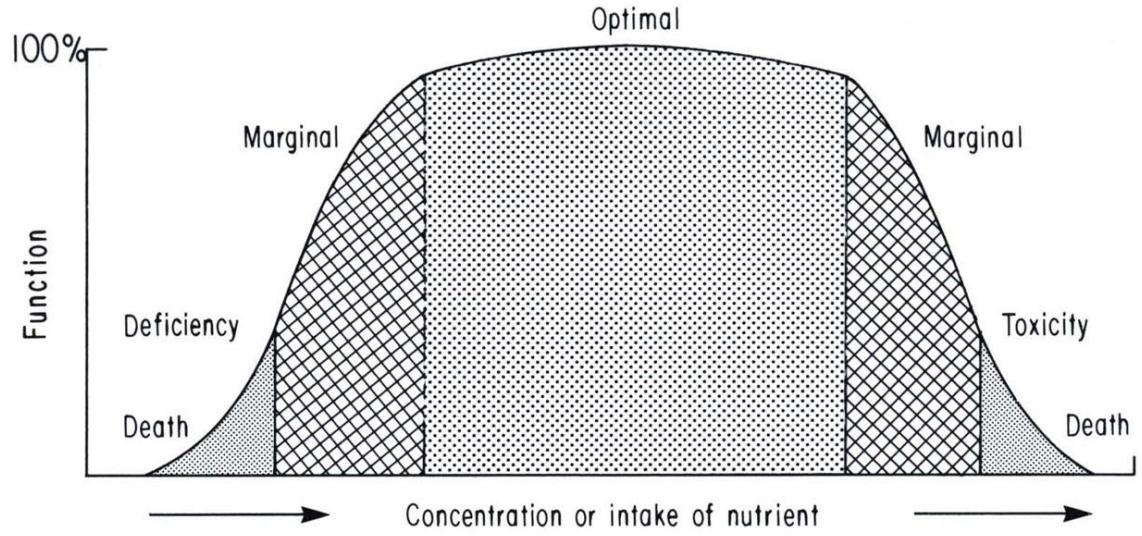

Figure C-2. Dependence of Biologic Function on the Tissue Concentration of Essential Trace Elements

Metals have been associated with a wide range of environmental and health effects including respiratory and pulmonary disorders (Prieditis 2001), neurotoxicity, and cancer (Monn 1999). High concentrations of metals in the environment, especially near industrial facilities, are thus a cause for concern. For example, in 2001, the Missouri Department of Health and Human

Services found that 28% of the town's children ages 6 and under had lead poisoning due to emissions from Doe Run's Herculaneum lead smelter (Missouri DHSS 2002). Within ¼ mile of the smelter, lead poisoning occurred in 56% of children in this age group. Negative health effects from lead poisoning include learning disabilities, behavioral problems, and, at very high levels, seizures, coma, and even death (US DHHS 2007). In addition to their potential health effects, metals are especially hazardous because they will not biodegrade; once released into the environment, they will always be available for re-introduction into the air, water and food chain. Exposure pathways include: breathing contaminated air, eating contaminated food products, drinking contaminated water, ingesting contaminated soil, and touching contaminated soil, dust, or water.

2.2 Air Exposure Pathways

The major pathways for human intakes of metals in which air serves as the primary medium of contact are inhalation and dermal. Exposure assessment depends on ambient and anthropogenic concentrations and multiple routes of exposure. People are exposed to toxic air pollutants in many ways that can pose health risks, such as by:

- Breathing contaminated air.
- Eating contaminated food products, such as fish from contaminated waters; meat, milk, or eggs from animals that fed on contaminated plants; and fruits and vegetables grown in contaminated soil on which air toxics have been deposited.
- Drinking water contaminated by toxic air pollutants.
- Ingesting contaminated soil. Young children are especially vulnerable because they often ingest soil from their hands or from objects they place in their mouths.
- Touching (making skin contact with) contaminated soil, dust, or water (for example, during recreational use of contaminated water bodies).

Other indirect pathways in which air serves as a medium include:

- Deposition of metals to surface dusts and intake from ingestion, inhalation, or dermal contact;
- Deposition to surface water and sediment and intake from ingestion and dermal contact; and
- Uptake of deposited metals into aquatic and/or terrestrial biota, entrance into the human food chain, and intake from ingestion.

Although, in most instances in which airborne metals have resulted in environmental contamination, ingestion of surface dust tends to be the dominant contributor to human health risk, this may not always be the case. Bioavailability of inhaled metals can be much higher than for other routes of intake. This can result in relatively high internal doses from inhalation even when inhalation intakes are similar to intakes from other routes. An example of this is the large contribution made by cigarette smoking to the body burden of cadmium (Newman 2004). Infants and children can be particularly vulnerable to airborne metal particulates because differences in airway geometry and airstream velocities tend to result in higher deposition fractions of inhaled particulates in infants and children than in adults at similar exposure levels. In addition, research shows that particle pollution may significantly reduce lung function growth in children (C. EPA 2004).

Human exposures to airborne metals are usually to metal-bearing particulates, which necessitates measurements of particle sizes in the breathing zone of receptors of concern to achieve accurate estimates of deposition rates in the respiratory tract. Exceptions to this are exposures to mercury vapor or other gaseous forms of metals, such as arsine gas. In most applications, monitoring of metals for use in environmental risk assessment consists of measuring total metal (i.e., unspeciated) captured in the PM_{10} fraction (the fraction of particles that are less than or equal to 10 micrometers [µm] in diameter) of samples collected in stationary samplers.

The fate of inhaled particulates deposited in the respiratory tract is substantially affected by particle size, below a diameter of 10 µm (James 1994). For example, a substantial fraction of the inhaled particles larger than 1 µm can be expected to be deposited in the upper respiratory tract and subsequently transferred by mucociliary transport to the gastrointestinal tract, where fractional absorption is likely much different from that of particles absorbed from the respiratory tract. Measurement of unspeciated metals introduces uncertainties into risk estimates for inhaled metals because the physiological solubility of a metal affects the mechanisms, rate, and extent of absorption of metals from the respiratory tract (Newman 2004). Lack of information about the particle sizes, chemical form, and solubility of the airborne metals in the breathing zone of receptors of concern can have important implications for accurate modeling of rates of absorption, internal dose of inhaled metal particulates, and risk (Khoury 2003).

An important risk-assessment concept in evaluating levels of airborne metals is the background concentration. Background concentrations are a function of regional geology and local soil and sediment conditions. It is the regional concentration in a medium that has not been increased by a local source of contamination; furthermore, it is the concentration of a metal in a medium (e.g. soil, air, water) as it existed before being affected by human activity. Occasionally, background metal concentrations can exceed environmental quality criteria at some sites (Langmiur 2004).

2.3 **Designated HAP Metals**

The following subsections summarize potential health effects, sources, and exposure pathways for 11 different metals as described in EPA's Health Effects Notebook for Hazardous Air Pollutants (EPA, Health Effects Notebook for HAPs 2010) and also for an additional six metals of concern that are not designated as HAPs. The information provided herein is intended as a general overview and not an exhaustive reference for health effects of metals. Metals information and data presented in the following subsections comes from current air research data as well as the Technology Transfer Network (TTN) Air Toxics Web Site (ATW) (EPA, Technology Transfer Network 2010), including data from the Agency for Toxic Substances and Disease Registry (ATSDR 2010), integrated risk information system (IRIS 2010), and the EPA. Other sources of health effect information include the American Conference of Industrial Hygienists (ACGIH 2010), the hazardous substances database (HSDB 2010), and the California Office of Environmental Health Hazard Assessment (OEHHA 2008). A selection of applicable regulatory human health limits for industrial and residential populations are available in Tables C-1 and C-2; the standards are defined in Table C-3. Table C-1 also provides as typical air concentrations for metals in rural, urban, and source-oriented scenarios.

2.3.1 Antimony (Sb)

General: Antimony is a silvery-white metal that is found in the earth's crust. Antimony ores are mined and then either changed into antimony metal or combined with oxygen to form antimony oxide. Antimony may be used in grid metal for lead acid storage batteries, solder, sheet and pipe, bearing metals, castings, type metal, and fire retardants for plastics, textiles, rubber, adhesives, pigments, and paper.

Exposure: Antimony is found naturally in the environment at very low levels, e.g., food contains low amounts of antimony. The most likely exposure pathway to higher than background levels of antimony is inhalation. People can be exposed through breathing air or dust near factories that convert antimony ores into metal, ingesting or touching contaminated soil near hazardous waste sites or antimony-processing sites.

Ambient Air Concentrations: The EPA's TTN ATW site lists ambient air concentrations of Antimony ranging from less than 1 ng/m^3 to about 170 ng/m^3; it may be present at levels up to 1,000 ng/m^3 near factories that convert antimony ores into metal or make antimony oxide.

Short-term Health Effects: Skin and eye effects can occur from inhalation and gastrointestinal effects from ingestion. In animals, high acute exposure has resulted in respiratory effects, such as significant decrease in ventilatory function, congestion, edema, and hemorrhage, as well as effects on the cardiovascular system, and liver.

Long-term Health Effects: Chronic inhalation can result in respiratory effects, e.g., inflammation of the lungs, chronic bronchitis, and chronic emphysema. Specific respiratory effects include antimony pneumoconiosis (inflammation of the lung), alteration in pulmonary function, chronic bronchitis, chronic emphysema, inactive tuberculosis, pleural adhesions, and irritation. Cardiovascular effects have also been reported.

Cancer Risk: Animal studies have linked antimony inhalation exposure with lung tumors, but no conclusive link between cancer and antimony has been found for humans. EPA has not classified antimony for carcinogenicity.

2.3.2 Arsenic (As)

General: Aside from occurring naturally in the environment, arsenic can be released in larger quantities through volcanic activity, erosion of rocks, forest fires, and human activity. The wood preserving industry uses about 90% of the industrial arsenic in the U.S. Arsenic is also found in paints, dyes, metals, drugs, soaps and semi-conductors. Animal feeding operations and certain fertilizers and pesticides can release high amounts of arsenic to the environment as can industry practices such as copper or lead smelting, mining, and coal burning. Arsenic is also used in veterinary medicine. Inorganic arsenic solutions were used to treat diseases such as syphilis and psoriasis up until the 1940s. Arsine, a short-lived, extremely toxic gas, is used in the microelectronics industry and in semiconductor manufacture.

Exposure: Inorganic arsenic is found in low levels throughout the environment. The most common exposure pathway for inorganic arsenic is through food ingestion with lower amounts coming from drinking water and air. Inhalation may occur near metal smelters and by burning wood treated with an arsenic wood preservative. Exposure to arsine occurs through inhalation.

Ambient Air Concentrations: The average concentration of arsenic compounds in the air measured at 13 cities across the U.S. was 2 ng/m3 (Chen and Lippmann 2009). The ToxGuide for arsenic lists environmental levels in air ranging from 1 to 3 ng/m^3 in remote locations and 20-100 ng/m^3 in urban areas *(ATSDR., ToxGuide for Arsenic 2007)*. Workers in metal smelters and nearby residents may be exposed to above-average inorganic arsenic levels.

Short-term Health Effects: Arsenic is odorless and tasteless. Gastrointestinal effects (nausea, diarrhea, abdominal pain) and central and peripheral nervous system disorders can occur from acute inorganic arsenic inhalation and ingestion. Acute oral exposure to inorganic arsenic can result in death. Arsine is extremely toxic and can result in headaches, vomiting, and abdominal pains occurring within a few hours of exposure. Acute exposure to high levels of arsine can also result in death. Lower level exposure can cause decreased production of red and white blood cells, abnormal heart rhythm, damage to blood vessels, and a sensation of "pins and needles" in hands and feet.

Long-term Health Effects: Inhalation of inorganic arsenic can result in skin and mucous membrane irritation. Gastrointestinal effects, anemia, peripheral neuropathy, skin lesions, hyperpigmentation, and liver or kidney damage can occur with long-term oral exposure. Long-term low level exposure can cause darkening of the skin and the appearance of small corns or warts on the palms, soles, and torso. Inhalation of inorganic arsenic is strongly associated with lung cancer and oral exposure has been linked to a form of skin cancer and also to bladder, liver, and lung cancer. Women who work in, or live near, metal smelters may have higher than normal spontaneous abortion rates and their children may exhibit lower than normal birth weights.

Cancer Risk: Human inhalation studies have reported inorganic arsenic exposure to be strongly associated with lung cancer. Inorganic arsenic is classified by the EPA as a Group A human carcinogen and can cause cancer of the skin, lungs, liver, and bladder.

2.3.3 Beryllium (Be)

General: Pure beryllium is a hard gray metal that does not occur naturally but does occur as a chemical component of certain kinds of rocks, coal and oil, soil, and volcanic dust. It is also present in a variety of compounds, such as beryllium fluoride, beryllium chloride, beryllium sulfate, beryllium oxide, and beryllium phosphate. Beryllium is used in electrical components, tools, and structural components for aircraft, missiles, satellites, some metal-fabrication, televisions, calculators, personal computers, and other consumer products.

Exposure: The greatest exposure to Beryllium occurs in or near facilities where it is mined, processed, or converted into alloys and chemicals. Individuals may also be exposed through inhalation of beryllium dust or fumes from burning coal, burning fuel oil, or smoking tobacco. Beryllium can also be ingested from fruits, vegetables, and water, as well as soil via hand-to-mouth.

Ambient Air Concentrations: The average concentration of Beryllium measured in the air in the United States during the 1980s was 0.03 ng/m^3. Ambient concentrations measured in 50 cities between 1977 and 1981 were 0.1-0.4 ng/m^3.

Short-term Health Effects: Short-term exposure to high levels of beryllium via inhalation may cause inflammation of the lungs or acute pneumonitis (reddening and swelling of the lungs). Symptoms may be reversible after exposure ceases. Acute animal tests have demonstrated

beryllium compounds to vary in acute toxicity, ranging from high to extreme acute toxicity from oral exposure.

Long-term Health Effects: Long-term exposure to beryllium can result in chronic beryllium disease (berylliosis), in which granulomatous lesions (noncancerous) develop in the lung. Symptoms of chronic beryllium disease include irritation of the mucous membranes, reduced lung capacity, shortness of breath, fatigue, anorexia, dyspnea, malaise, and weight loss. Chronic inhalation exposure has caused immunological effects in humans and animals. Additional chronic effects include chronic pneumonitis, conjunctivitis, and skin allergies.

Cancer Risk: Beryllium is suspected to cause cancer; human studies have shown a causal relationship between beryllium exposure and an increased risk of lung cancer, and animal studies have linked inhalation of beryllium to lung cancer. Beryllium has been classified by the EPA as Group B1, probable human carcinogen.

2.3.4 Cadmium (Cd)

General: Cadmium is a soft silver-white metal that is usually found in combination with other elements. Cadmium is most commonly a byproduct from the smelting of zinc, lead, or copper ores. Cadmium is also used in manufacturing (pigments and batteries), metal-plating, and in the plastics industry.

Exposure: Inhalation and ingestion of contaminated food are the two major exposure pathways. Cadmium is emitted into the air from burning fossil fuels, from incineration of municipal waste materials, and from zinc, lead, and copper smelters. Smoking cigarettes is another source of airborne cadmium; smokers have about twice the amount of cadmium in their bodies as do nonsmokers. Cadmium can occur in food as a result of the application of phosphate fertilizers or sewage sludge to farm fields.

Ambient Air Concentrations: Ambient air cadmium concentrations have generally been estimated to range from 0.1 to 5 ng/m^3 in rural areas, from 2 to 15 ng/m^3 in urban areas, and from 15 to 150 ng/m^3 in industrialized areas (ICdA 2009). Cadmium has been measured in air as high as 600 ng/m^3.

Short-term Health Effects: The short-term effects of cadmium inhalation include lung effects such as bronchial and pulmonary irritation. A single acute exposure to high levels of cadmium can result in long-lasting impairment of lung. Animal tests have shown high acute toxicity for cadmium.

Long-term Health Effects: Long-term effects of cadmium inhalation and ingestion can result in cadmium build-up in the kidneys and can have effects on the liver, lung, bone, immune system, blood, and nervous system. Animal studies have demonstrated fetal malformations and other developmental effects as a result of cadmium exposure, although no conclusive evidence exists in humans. *Itai-itai disease* is caused by cadmium poisoning due to mining in Toyama Prefecture Japan. The cadmium poisoning caused softening of the bones (brittle bones) and kidney failure.

Cancer Risk: Cadmium exposure has been tentatively linked to an increased risk of lung cancer. Cadmium is classified by the EPA as a Group B1, probable human carcinogen.

2.3.5 Chromium (Cr)

General: The metal, chromium, is a steel-gray solid with a high melting point. Chromium is used to make steel and other alloys, and its compounds either in the form of chromium (III) or chromium (VI), are used in chrome plating, manufacturing dyes and pigments, preserving leather and wood, and treating water in cooling towers. It is used in small amounts in drilling mud, textiles, and toner for printers/copying machines. Chromium (III) is an essential element in humans.

Exposure: Chromium occurs naturally in the environment, predominantly either as chromium (III) or chromium (VI). Chromium (VI) is more commonly produced by industrial processes. Exposure to high levels of chromium occurs mainly by inhalation of airborne chromium from ferrochrome production, ore refining, chemical and refractory processing, cement-producing plants, automobile brake lining and catalytic converters for automobiles, leather tanneries, and chrome pigments. Chromium exposure also occurs through food and drinking water. Touching products that contain chromium, e.g., wood treated with copper dichromate or leather tanned with chromic sulfate is also an exposure pathway.

Ambient Air Concentrations: Average daily intake from air is estimated to be less than 200 to 400 nanograms; average air concentration is 3 ng/m^3 measured at 13 cities across the U.S. (AIRS sites) (Chen and Lippmann 2009). People who live in the vicinity of chromium waste disposal sites or chromium manufacturing and processing plants have a greater probability of elevated chromium exposure than the general population. The maximum chromium level measured in ambient air near a chromate manufacturing plant in Corpus, Christi, Texas was 5,500 ng/m^3 with an annual average concentration in ambient air of 400 ng/m^3. Chromium VI concentrations range from 0.013 ng/m^3 to 15.3 ng/m^3 (HSDB 2010).

Short-term Health Effects: The short-term effects of chromium (VI) inhalation include shortness of breath, coughing, wheezing, and other effects on the respiratory tract. Chromium (VI) is much more toxic that chromium (III) and both inhalation and ingestion may also cause gastrointestinal effects including abdominal pain, vomiting, and hemorrhage.

Long-term Health Effects: Long-term effects of chromium (VI) inhalation exposure include effects on the respiratory tract including perforations and ulcerations of the septum, bronchitis, decreased pulmonary function, and pneumonia. Asthma, nasal itching, and soreness have been reported; chromium exposure may also produce effects on the liver, kidney, gastrointestinal and immune systems, and possibly the blood. Chromium (VI) exposure may result in complications during pregnancy and childbirth.

Cancer Risk: Inhaled chromium (VI) is clearly linked to an increased risk of lung cancer; animal studies have shown chromium (VI) to cause lung tumors. Chromium (VI) classified by the EPA as a Group A, known human carcinogen by inhalation route of exposure.

2.3.6 Cobalt (Co)

General: Cobalt is a steel-gray, shiny, hard metal that is insoluble in water and usually occurs in the environment in association with other metals such as copper, nickel, manganese, and arsenic. Cobalt is used in superalloys, alloys that maintain their strength at high temperatures approaching their melting points, and pigment manufacture. Cobalt is an essential element in humans and is used as a treatment for anemia, because it stimulates red blood cell production.

Exposure: Cobalt is found naturally throughout the environment. The general population may be exposed to cobalt in the air, drinking water, and food. Higher-than-normal exposure levels for cobalt can occur in the air and water near industrial areas, particularly near hard metal industrial sites.

Ambient Air Concentrations: Average concentration in ambient air in the U.S. is approximately 0.4 ng/m^3; however, in one industrial area, levels of 610 ng/m^3 were measured.

Short-term Health Effects: The short-term effects of cobalt inhalation include respiratory effects such as a significant decrease in ventilatory function, congestion, edema, and hemorrhage of the lung. Acute animal tests in rats have shown cobalt to have extreme toxicity from inhalation exposure.

Long-term Health Effects: Long-term effects of cobalt inhalation include respiratory irritation, wheezing, asthma, pneumonia, and fibrosis. Cardiac effects, congestion of the liver, kidneys, and conjunctiva, and immunological effects that include cobalt sensitization are also potential effects from chronic exposure. Animal studies have reported respiratory, cardiovascular, and CNS effects, decreased body weight, necrosis of the thymus, and effects on the blood, liver, and kidneys from inhalation exposure to cobalt.

Cancer Risk: No conclusive link between cancer and cobalt has been determined. EPA has not classified cobalt for carcinogenicity, as limited data is available on carcinogenic effects of cobalt.

2.3.7 Lead (Pb)

General: Lead is a naturally occurring, bluish-gray metal that is found in small quantities in the earth's crust. Pure lead is insoluble in water; however, the lead compounds vary in solubility. The primary use of lead is in manufacturing batteries. Lead is also used in the production of metal products, including sheet lead, solder, and pipes, and in ceramic glazes, paint, ammunition, cable covering, and other products. Tetraethyl lead was used in gasoline to increase the octane rating until lead additives were phased out and eventually banned from use in gasoline in the U.S. by the EPA by 1996.

Exposure: The largest source of lead in the atmosphere has been from leaded gasoline combustion; however, air lead levels have decreased considerably with the phase-down of lead in gasoline. Exposure to lead can occur by inhalation of airborne lead from combustion of solid waste, coal, and oils, emissions from iron and steel production and lead smelters, and tobacco smoke. Ingestion of lead in food and soil are also common pathways. The hand-to-mouth pathway is common in children (i.e., they commonly put hands, toys, and other items that may come in contact with lead-contaminated soil and dust in their mouths), therefore this group is especially at risk to lead exposure. Lead in paint products is major pathway for exposure in children as a result. Drinking water and food sources are also pathways of due to the presence of lead in old pipes, solder, and fixtures as well as its environmental persistence and potential for bioaccumulation.

Ambient Air Concentrations: Nationwide, average concentrations of lead in the air have dropped nearly 94 percent between 1980 and 2007. Average lead concentration measured in 13 cities across the U.S. (AIRS sites) is 6 ng/m^3 (Chen and Lippmann 2009). The average concentration of lead in air samples in 2002 is less than 50 ng/m^3 (ATSDR., ToxGuide for Lead 2007). Urban

concentrations of lead average 1,100 ng/m^3 of lead, non-urban concentrations average 210 ng/m^3, and remote areas average 20 ng/m^3 (HSDB 2010).

Short-term Health Effects: Lead is very toxic in low doses and can cause death in children with high lead blood levels. Short-term exposure to lead can also lead to brain damage, kidney damage, and gastrointestinal distress.

Long-term Health Effects: Long-term effects of lead exposure can lead to problems with the blood, CNS, blood pressure, kidneys, and Vitamin D metabolism. Neurological symptoms have been reported in workers, and slowed nerve conduction in peripheral nerves in adults. Chronic lead exposure can cause loss of IQ, slowed cognitive development, reduced growth, hearing loss, and other developmental effects in children. Additional effects of lead exposure can include reproductive effects e.g., decreased sperm count, spontaneous abortions, low birth weight, slowed postnatal neurobehavioral development.

Cancer Risk: EPA considers lead to be a Group B2, probable human carcinogen; human studies are inconclusive regarding lead exposure and increased cancer risk.

2.3.8 Manganese (Mn)

General: Manganese is a silver-colored metal that forms compounds in the environment with chemicals such as oxygen, sulfur, and chlorine. Manganese is used in the production of steel, carbon steel, stainless steel, and high-temperature steel, cast iron, superalloys, dry-cell batteries, matches, and fireworks. Manganese chloride is used in the chlorination of organic compounds, in animal feed, and in dry-cell batteries. Manganese sulfate is used as a fertilizer, livestock nutritional supplement, in glazes and varnishes, and in ceramics, and potassium permanganate is used for water purification. Manganese is an essential nutritional element in humans.

Exposure: Manganese is a naturally occurring element found in low levels in water air, soil, and food. Elevated levels of manganese can occur in the air near iron and steel production plants, power plants, and coke ovens. The most common route of exposure to high doses of manganese is through inhalation of contaminated air.

Ambient Air Concentrations: Average air levels are approximately 20 ng/m^3 as reported in the ATSDR Toxicological Profile. Average manganese concentration measured in 13 cities across the U.S. (AIRS sites) is 6 ng/m^3 (Chen and Lippmann 2009).

Short-term Health Effects: No short-term effects for acute manganese exposure have been reported in humans; however, contemporary researchers are evaluating manganese health effects. Some tests in animals have shown effects on the lungs via inhalation. Manganese is considered to have moderate acute toxicity based on the short-term tests in rats.

Long-term Health Effects: Long-term exposure to manganese leads primarily to effects on the CNS, including slowed visual reaction time, hand steadiness, and eye-hand coordination. Inhalation exposure specifically can cause respiratory effects. Chronic exposure to high levels can also result in a syndrome called manganism, which typically begins with feelings of weakness and lethargy, tremors, a mask-like face, psychological disturbances, and can involve impotence and loss of libido.

Cancer Risk: EPA has classified manganese as a Group D, not classifiable as to carcinogenicity in humans.

2.3.9 Mercury (Hg)

General: There are three forms of mercury, all of which are toxic to humans: elemental mercury, inorganic mercury, and organic mercury. Elemental mercury is used in thermometers, barometers, other pressure-sensing devices, batteries, lamps, industrial processes, refining, lubrication oils, and dental amalgams. Most uses of inorganic mercury have been discontinued (e.g., use of mercury in paint, laxatives, skin-lightening creams, and soaps) in the U.S.; however, mercuric chloride is still used as a disinfectant and pesticide. Organic mercury such as methyl mercury is formed in the environment from the methylation of the inorganic mercury ion.

Exposure: Inhalation in occupational settings is a major exposure pathway for elemental mercury. Ingestion of methyl mercury through consumption of contaminated foods such as fish is also a major route to exposure; mercury is very persistent in the environment and has great bioaccumulation potential. Exposure to inorganic mercury can occur though the use of old paint (made with mercury before the ban).

Ambient Air Concentrations: Elemental mercury vapor (Hg^0) is present globally in ambient air at concentrations on the order of 1.5 -2.0 ng/m^3 (NCDENR 2009). The EPA reports that the average atmospheric concentration of total mercury ranges from 2 to 10 ng/m^3. Total mercury in the air is normally found to be less than 0.1 ng/m^3 in regions unaffected by local sources and at or above 41 ng/m^3 in more industrialized urban environments (HSDB 2010).

Short-term Health Effects: For high levels of elemental mercury, short-term exposure can result in CNS effects such as tremors, mood changes, and slowed sensory and motor nerve function. Short-term exposure to inorganic mercury can cause nausea, vomiting, and severe abdominal pain via the oral ingestion route. Methyl mercury acute exposure can cause blindness, deafness, and impaired level of consciousness in humans

Long-term Health Effects: Long-term exposure to elemental mercury can cause effects on the CNS including erethism (increased excitability), irritability, excessive shyness, and tremors. For inorganic mercury, long-term exposure can lead to kidney damage; alterations in testicular tissue, increased resorption rates, and abnormalities of development have been reported in animals as well. Long-term exposure to methyl mercury can cause CNS effects with symptoms such as paresthesia (a sensation of pricking on the skin), blurred vision, malaise, speech difficulties, and constriction of the visual field. Ingestion of methyl mercury can lead to developmental effects in infants such as mental retardation, ataxia, constriction of the visual field, blindness, and cerebral palsy.

Cancer Risk: Elemental mercury has not been conclusively linked to cancer, so is classified as Group D, not a human carcinogen. The EPA has classified inorganic and methyl mercury as a Group C, possible human carcinogen.

2.3.10 Nickel (Ni)

General: Nickel is used in alloys, electroplating, batteries, coins, industrial plumbing, spark plugs, machinery parts, stainless-steel, nickel-chrome resistance wires, and catalysts. Nickel carbonyl is used in nickel refining, but its use is severely limited.

Exposure: Nickel is found naturally throughout the environment and occurs in small amounts in food, water, soil, and air. The major exposure pathway for nickel is ingestion of food. Exposure can also occur though touching everyday items such as nickel-containing jewelry, stainless steel cooking and eating utensils, as well as smoking tobacco. Inhalation of nickel is a pathway for those in the vicinity of nickel production, processing, and use, and releases from oil and coal combustion, nickel metal refining, sewage sludge incineration, manufacturing facilities, etc. Exposure to nickel carbonyl is very rare.

Ambient Air Concentrations: Typical average levels of airborne nickel are: 0.01 - 3 ng/m^3 in remote areas; 3 - 30 ng/m^3 in urban areas having no metallurgical industry; 70 - 770 ng/m^3 in nickel processing areas (HSDB 2010). Average nickel concentration measured in 13 cities across the U.S. (AIRS sites) is 3 ng/m^3 (Chen and Lippmann 2009).

Short-term Health Effects: One case of short-term exposure to a high level of nickel via inhalation showed damage to the lungs and kidneys. Effects of ingestion through drinking water can include gastrointestinal distress (e.g., nausea, vomiting, and diarrhea) and neurological effects. Nickel carbonyl exposure can cause pulmonary fibrosis and renal edema. Current research is showing that Nickel at ambient levels is capable of causing acute changes in heart rate and other health effects, partially due to its ability to create reactive oxygen species (ROS) (Zelikoff 2002). Reduced mortality in humans is associated with longer-term Nickel concentrations extending from 19 ng/m^3 to a national average of 1.9 ng/m^3 (Lippmann et al. 2006).

Long-term Health Effects: Long-term exposure via skin contact can result in nickel dermatitis, consisting of itching of the fingers, hands, and forearms. Inhalation exposure can have respiratory effects and has been linked to an increased risk of lung and nasal cancers.

Cancer Risk: Nickel inhalation in animals has been linked to lung tumors; human studies have reported an increase risk of lung and nasal cancers among nickel refinery workers exposed to nickel refinery dust. Nickel refinery dust and nickel subsulfide are classified by the EPA as Group A human carcinogens, and nickel carbonyl is classified as a Group B2 probable human carcinogen.

2.3.11 Selenium (Se)

General: Selenium is used in the electronics industry, the glass industry, pharmaceutical preparation, antidandruff shampoos, fungicides, pesticide formulations, and in pigments used in plastics, paints, enamels, inks, and rubber. It is also a nutritional feed additive for poultry and livestock.

Exposure: Selenium is found naturally in the environment and is a nutritionally essential element. A major pathway for selenium exposure is ingestion of food. Air and drinking water also have low levels of selenium. Exposure to high levels of selenium can occur via inhalation in the vicinity of metal industries, selenium-recovery processes, painting, and special trades.

Ambient Air Concentrations: An average ambient air selenium concentration is estimated to be below 10 ng/m^3. Concentration ranges of selenium associated with particulate matter in urban atmospheres were 0.2 to 30 ng/m^3 in the U.S. (HSDB 2010).

Short-term Health Effects: Acute exposure of humans via inhalation to selenium compounds, such as selenium dioxide, hydrogen selenide, results primarily in respiratory effects. Short-term exposure to elemental selenium dust via inhalation can cause irritation of the mucous membranes in the nose and throat, nosebleeds, dyspnea, bronchial spasms, bronchitis, and chemical pneumonia. Acute animal tests have shown hydrogen selenide to have extreme toxicity via oral exposure.

Long-term Health Effects: Long-term exposure to high selenium levels via ingestion can result in discoloration of the skin, pathological deformation and loss of nails, loss of hair, excessive tooth decay and discoloration, garlic odor in breath and urine, lack of mental alertness, and listlessness.

Cancer Risk: EPA has classified elemental selenium as Group D, not classifiable as to human carcinogenicity. Ingestion of selenium sulfide may result in an increase of liver and lung tumors; therefore, EPA has classified selenium sulfide as Group B2, a probable human carcinogen.

2.4 **Non-Designated HAP Metals**

2.4.1 Copper (Cu)

General: Copper is used to make wire, plumbing pipes, and sheet metal. U.S. pennies made before 1982 are made of copper, while those made after 1982 are only coated with copper. Copper is also combined with other metals to make brass and bronze pipes and faucets. Copper compounds are commonly used in agriculture to treat plant diseases like mildew, for water treatment and, as preservatives for wood, leather, and fabrics. Copper is also used in contraception as intrauterine devices.

Exposure: Copper is a metal that occurs naturally throughout the environment, in rocks, soil, water, and air. Copper is an essential element in plants and animals (including humans). Plants and animals absorb some copper from eating, drinking, and breathing. In general the soluble ionized salts of copper are much more toxic than the insoluble or slightly dissociated compounds. Sources of exposure are from fume, from copper ore smelting and related metallurgic operations, from welding, and from dusts of copper metal and salts.

Ambient Air Concentrations: Atmospheric levels of copper in the United States have been reported to vary from 10-570 ng/m^3, the highest values being found in urban areas (HSDB 2010).[26] Average copper concentration measured in 13 cities across the U.S. (AIRS sites) is 3 ng/m^3 (Chen and Lippmann 2009).

Short-term Health Effects: Inhalation of dust, fumes, and mists of copper salts can result in irritation of nasal mucous membranes, eye irritation, upper respiratory tract irritation; metallic taste, nausea, and metal fume fever. Acute copper poisoning can cause liver injury, methemoglobinemia, and hemolytic anemia. Effects of single exposure following suicidal or accidental oral exposure have been reported as metallic taste, epigastric pain, headache, nausea, dizziness, vomiting and diarrhea, tachycardia, respiratory difficulty, hemolytic anemia, massive gastrointestinal bleeding, liver and kidney failure, and death.

Long-term Health Effects: Mammals have efficient mechanisms to regulate copper stores such that they are generally protected from excess dietary copper levels; however, at high enough

levels, chronic overexposure to copper can damage the liver and kidneys. Chronic exposure may also result in an anemia. Vineyard sprayer's lung disease, a lung and liver disease, occurs in individuals exposed to copper sulfate spray for 2 to 15 years. Wilson's disease is inherited, genetic disorders in which copper builds up in the liver; symptoms include liver toxicity (jaundice, swelling, pain) usually do not appear until adolescence.

Cancer Risk: Although some studies of workers exposed to copper have shown increased cancer risks, they were also exposed to other potentially carcinogenic chemicals. Copper is currently categorized as Group D, not classifiable as to carcinogenicity in humans.

2.4.2 Iron (Fe)

General: Iron is the second most abundant metal and the fourth most abundant element in the earth's crust, comprising 5.1% (by weight) of the earth's crust. Hydrous iron(II) oxides are generally red-brown gels and are the major constituents of soil. Iron is an essential element that is required by all forms of life. Iron is a natural constituent of all foods of plant or animal origin, and occurs in foods as iron oxides, inorganic and organic salts, and organic complexes, such as hemoglobin. Iron is the most widely used of all the metals, accounting for 95% of worldwide metal production. Its low cost and high strength make it indispensable in engineering applications such as the construction of machinery and machine tools, automobiles, the hulls of large ships, and structural components for buildings. Since pure iron is quite soft, it is most commonly used in the form of steel.

Exposure: The production and use of iron compounds as catalysts, pigments, drugs, as well as their use in agriculture, nutrition, metallurgy, and leather tanning may result in their release to the environment through various waste streams. The mining and processing of iron ores also may result in the release of iron compounds to the environment. The iron and steel industries are also likely sources of emissions of iron compounds to the environment. Occupational exposure to iron compounds may occur through inhalation and dermal contact with these compounds at workplaces where iron compounds are produced or used.

Ambient Air Concentrations: The average vanadium concentration measured in 13 cities across the U.S. (AIRS sites) is 108 ng/m^3 (Chen and Lippmann 2009). An average iron concentration in urban air of 1.6 µg/m^3 has been reported; other sources provide a range of atmospheric iron concentration s of 0.9 to 1.2 µg/m^3, as particular ferric oxide. In a study monitoring air in nonproduction departments of 147 pulp and paper mills in 11 countries mean concentration s for iron, iron oxide, and iron oxide fumes in maintenance, construction, cleaning areas were reported to be 914, 260, and 260 µg/m^3, respectively.

Short-term Health Effects: Toxicity occurring with acute iron overdose results from a combination of the corrosive effects on the gastrointestinal mucosa and the metabolic and hemodynamic effects caused by the presence of excessive elemental iron. Inhalation of ferric salts as dusts & mists is irritating to the respiratory tract. Ferric salts are regarded as skin irritants. Early symptoms of acute iron toxicity include diarrhea, sometimes containing blood; fever; nausea, severe; stomach pain or cramping, sharp; vomiting, severe, sometimes containing blood. Late symptoms of acute iron toxicity include bluish-colored lips, fingernails, palms of hands; drowsiness; pale, clammy skin; seizures; unusual tiredness or weakness; weak and fast heartbeat. Pulmonary siderosis results from inhalation of iron dust or fumes.

Long-term Health Effects: The corrosive effect of iron results in stomach and intestinal erosions and ulceration (i.e., hemorrhagic gastritis and enteritis with blood loss); however, there is a lack

of correlation between the severity of intestinal damage and death. Large chronic doses of iron may so interfere with assimilation of phosphorus as to cause severe rickets in infants.

Cancer Risk: Free iron is a pro-oxidant and can induce oxidative stress and DNA damage. The carcinogenicity of iron has been demonstrated in animal models, and epidemiologic studies have shown associations with several human cancers.

2.4.3 Silver (Ag)

General: The principal use of silver is as a precious metal and its halide salts, especially silver nitrate, are also widely used in photography. The major outlets are photography, the electrical and electronic industries and for domestic uses as cutlery, jewelry and mirrors. Silver is also employed in the electrical industry: printed circuits are made using silver paints, and computer keyboards use silver electrical contacts. Other applications are as a catalyst in oxidation reactions, in dentistry, and in high-capacity zinc long-life batteries. Silver has also been used in lozenges and chewing gum to help people stop smoking. Silver is often found as a by-product during the retrieval of copper, lead, zinc, and gold ores.

Exposure: Silver is a naturally occurring element. It is found in the environment combined with other elements such as sulfide, chloride, and nitrate.

Ambient Air Concentrations: Ambient air concentrations of silver are in the low ng/m^3 range. Atmospheric concentration of silver varied little from non-industrial to industrial cities with values ranging from 0.04 to 10.5 ng/m^3. Chadron, Nebraska (1973), which has a population of 6,000 in a sparsely inhabited region, and San Francisco (1970) had the same average ambient air concentration of silver in 0.15 ng/m^3 (HSDB 2010).

Short-term Health Effects: Exposure to high concentrations of vapors may cause dizziness, breathing difficulty, headaches or respiratory irritation. Extremely high concentrations may cause drowsiness, staggering, confusion, unconsciousness, coma or death. Acute symptoms of overexposure also include decreased blood pressure, diarrhea, stomach irritation and decreased respiration. Exposure to high levels of silver in the air has resulted in breathing problems, lung and throat irritation, and stomach pains. Skin contact with silver can cause mild allergic reactions such as rash, swelling, and inflammation in some people.

Long-term Health Effects: Chronic exposure to silver compounds includes fatty degeneration of the liver and kidneys and changes in blood cells. Long-term inhalation of soluble silver compounds or colloidal silver may cause argyria and/or argyrosis (a permanent bluish-gray discoloration of the skin or eyes). Soluble silver compounds are also capable of accumulating in small amounts in the brain and muscles.

Cancer Risk: Silver in any form is not thought to be carcinogenic.

2.4.4 Thallium (Tl)

General: According to the EPA, man-made sources of thallium pollution include gaseous emission of cement factories, coal burning power plants, and metal sewers. The main source of elevated thallium concentrations in water is the leaching of thallium from ore processing operations. The major source of thallium for practical purposes is the trace amount that is found in copper, lead, zinc, and other heavy-metal-sulfide ores. Approximately 60–70% of thallium production is used in the electronics industry, and the rest is used in the pharmaceutical industry

and in glass manufacturing. It is also used in infrared detectors. Thallium is highly toxic and was used in rat poisons and insecticides. Its use has been cut back or eliminated in many countries because of its nonselective toxicity.

Exposure: Thallium and its compounds are extremely toxic, and should be handled with great care. Contact with skin is dangerous, and adequate ventilation should be provided when melting this metal. Thallium compounds have a high aqueous solubility and are readily absorbed through the skin. Exposure to them should not exceed 0.1 mg per m^2 of skin in an 8-hour time-weighted average (40-hour work week).

Ambient Air Concentrations: Data on thallium concentrations in ambient air are limited. One study reported a mean value of 0.22 ng/m^3 for North America. A study of six large cities in the United States reported a range of <0.04 to 0.1 ng/m^3. The thallium concentration near a coal-burning power plant was estimated to be 700 ng/m^3 by the EPA in 1988.

Short-term Health Effects: The human body absorbs thallium very effectively, especially through the skin, the breathing organs and the digestive tract. Thallium poisoning, mainly caused by accidental uptake of rat poison, causes stomachaches and nervous system damage, with consequences such as trembling, paralyses and behavioral changes will remain possible death. With unborn children thallium poisoning can cause congenital disorders. Studies in people who ingested large amounts of thallium over a short time have reported vomiting, diarrhea, temporary hair loss, and effects on the nervous system, lungs, heart, liver, and kidneys. It has caused death.

Long-term Health Effects: Due to accumulation of thallium in the bodies of humans, chronic effects consist, such as tiredness, headaches, depression, lack of appetite, leg pains, hair loss, and sight disturbances. Further effects that can be related to thallium poisoning are nerve pains and joint pains. These are consequences of thallium uptake through food. A study on workers exposed on the job over several years reported nervous system effects, such as numbness of fingers and toes, from breathing thallium. It is not known if breathing or ingesting thallium affects human reproduction. Studies showed that rats that ingested thallium for several weeks had some adverse reproductive effects. Animal data suggest that the male reproductive system may be susceptible to damage by low levels of thallium.

Cancer Risk: Thallium is a suspected human carcinogen; however, the EPA has not classified thallium as to its human carcinogenicity.

2.4.5 Vanadium (V)

General: Vanadium is a compound that occurs in nature as a white-to-gray metal, and is often found as crystals. Pure vanadium has no smell. It usually combines with other elements such as oxygen, sodium, sulfur, or chloride. Vanadium and vanadium compounds can be found in the earth's crust and in rocks, some iron ores, and crude petroleum deposits. Vanadium is mostly combined with other metals to make special metal mixtures called alloys. Vanadium in the form of vanadium oxide is a component in special kinds of steel that is used for automobile parts, springs, and ball bearings. Most of the vanadium used in the United States is used to make steel. Vanadium oxide is a yellow-orange powder, dark-gray flakes, or yellow crystals. Vanadium is also mixed with iron to make important parts for aircraft engines. Small amounts of vanadium are used in making rubber, plastics, ceramics, and other chemicals.

Exposure: Vanadium exposure can occur through eating foods containing vanadium, higher levels are found in seafood; breathing air near an industry that burns fuel oil or coal; these industries release vanadium oxide into the air; working in industries that process it or make products containing it; breathing contaminated air or drinking contaminated water near waste sites or landfills containing vanadium. Vanadium is not readily absorbed by the body from the stomach, gut, or contact with the skin.

Ambient Air Concentrations: The average vanadium concentration measured in 13 cities across the U.S. (AIRS sites) is 3 ng/m^3 (Chen and Lippmann 2009). Small amounts of vanadium compounds are found in air where there is no known anthropogenic source; such concentrations are found to be in the range from 0.02 to 2.0 ng/m^3. Urban sites may have vanadium levels as high as 164 ng/m^3, while industrialized urban centers with a high consumption of residual fuel oil may have maximum levels up to 1,300 ng/m^3 (range of 450-1,300 ng/m^3). Occupational exposure to vanadium may involve exposure to levels in the order of milligrams per cubic meter (mg/m^3) of vanadium, or roughly 106 times typical background levels (HSDB 2010).

Short-term Health Effects: Breathing high levels of vanadium may cause lung irritation, chest pain, coughing, and other effects. Vanadium is considered a metal of concern, particularly due to its ability to produce reactive oxygen species (Chen and Lippmann 2009). High level acute exposures may result in CNS effects including paralysis, respiratory depression, convulsions, and death. Since vanadium is poorly absorbed from the gastrointestinal tract, inhalation exposures potentially pose the greatest risk. Vanadium intoxication (i.e., rhinorrhea, sneezing, lacrimation, and sore throat) has been reported in workers exposed to concentrations of V_2O_5 during the workshift ranging from 10 to 33 mg/m^3. Concentrations of V_2O_5 exceeding 56 mg V/m^3 have resulted in local respiratory effects; other workers exposed intermittently to 56 mg V/m^3 showed no evidence of intoxication.

Long-term Health Effects: Workers exposed to a range of vanadium pentoxide dust levels for as little as 1 day or as long as ≥6 years show mild respiratory distress, such as cough, wheezing, chest pain, runny nose, or sore throat. One study of chronically-exposed workers showed increased neutrophils in the nasal mucosa.

Cancer Risk: The International Agency for Research on Cancer (IARC) has classified vanadium pentoxide as possibly carcinogenic to humans based on evidence of lung cancer in exposed mice. The US Department of Health and Human Services (DHHS), IARC, and EPA have not classified vanadium as to its human carcinogenicity.

2.4.6 Zinc (Zn)

General: Zinc is one of the most common elements in the earth's crust. It is found in air, soil, and water, and is present in all foods. Pure zinc is a bluish-white shiny metal. Zinc has many commercial uses, such as coatings to prevent rust, in dry cell batteries, and mixed with other metals to make alloys like brass, and bronze. A zinc and copper alloy is used to make pennies in the United States. Common zinc compounds found at hazardous waste sites include zinc chloride, zinc oxide, zinc sulfate, and zinc sulfide. Zinc compounds are widely used in industry to make paint, rubber, dyes, wood preservatives, and ointments. Many alloys contain zinc, including brass, an alloy of zinc and copper. Zinc is the fourth most common metal in use, trailing only iron, aluminum, and copper with an annual production of about 10 megatons. The

production for sulfidic zinc ores produces large amounts of sulfur dioxide and cadmium vapor. Smelter slag and other residues of process also contain significant amounts of metals.

Exposure: Zinc is an essential trace element, necessary for plants, animals, and microorganisms. Zinc is found in nearly 100 specific enzymes (other sources say 300). It is typically the second most abundant transition metal in organisms after iron and it is the only metal which appears in all enzyme classes. Some zinc is released into the environment by natural processes, but most comes from human activities like mining, steel production, coal burning, and burning of waste. It attaches to soil, sediments, and dust particles in the air. Rain and snow remove zinc dust particles from the air. Depending on the type of soil, some zinc compounds can move into the groundwater and into lakes, streams, and rivers. Most of the zinc in soil stays bound to soil particles and does not dissolve in water. It builds up in fish and other organisms, but it does not build up in plants. Ingesting small amounts present in your food and water. Humans are exposed to zinc through drinking contaminated water or a beverage that has been stored in metal containers or flows through pipes that have been coated with zinc to resist rust; eating too many dietary supplements that contain zinc; and working in any of the following jobs: construction, painting, automobile mechanics, mining, smelting, and welding; manufacture of brass, bronze, or other zinc-containing alloys; manufacture of galvanized metals; and manufacture of machine parts, rubber, paint, linoleum, oilcloths, batteries, some kind of glass, ceramics, and dyes.

Ambient Air Concentrations: The average vanadium concentration measured in 13 cities across the U.S. (AIRS sites) is 15 ng/m^3 (Chen and Lippmann 2009). A mean zinc concentration of 103 ng/m^3 was reported in the aerosol (0.01 to 1.0 µm particle size) collected in Detroit, Michigan in August 1996. The annual mean atmospheric concentration of zinc was 5 µg/m^3 near a lead smelter in Kellogg, Idaho, and the 24-hour values ranged from 0.27 to 15.7 µg/m^3.

Short-term Health Effects: Zinc is an essential element in our diet. Too little zinc can cause problems, but too much zinc is also harmful. Harmful effects generally begin at levels 10-15 times higher than the amount needed for good health. Large doses taken by mouth even for a short time can cause stomach cramps, nausea, and vomiting. Inhaling large amounts of zinc (as dusts or fumes) can cause a specific short-term disease called metal fume fever. Acute exposure to zinc oxide can result in coughing, substernal pain, upper respiratory tract irritation, rales, chills, fever, nausea, and vomiting. Zinc chloride fume is an irritant of the eyes, skin, mucous membranes, and lungs in humans. The signs and symptoms of acute exposure to zinc chloride fume include conjunctivitis, irritation of the nose and throat, hoarseness, cough, dyspnea, wheezing, rales, rhonchi, chest tightness and/or pain, nausea, vomiting, epigastric pain, listlessness, lightheadedness, and a metallic taste in the mouth.

Long-term Health Effects: Excessive concentrations of zinc taken on a long-term basis can cause anemia and decrease the levels of good cholesterol. Chronic exposure to zinc oxide by skin contact may result in papular-pustular skin eruptions in the axilla, inner thigh, inner arm, scrotum and pubic areas. Excessive absorption of zinc suppresses copper and iron absorption. The U.S. Food and Drug Administration (FDA) has stated that zinc damages nerve receptors in the nose, which can cause anosmia (loss of sense of smell).

Cancer Risk: Epidemiologic studies of zinc refinery workers found no correlation between industrial zinc exposures and lung or other types of cancer. Based on incomplete information from human and animal studies, the EPA has determined that zinc is not classifiable as to its human carcinogenicity.

3.0 Summary of Contemporary Research on Airborne Metals Health Effects

3.1 Ambient Air Health Effects

Numerous epidemiological events led to the development of the United States NAAQS for particulate matter. Initially, PM was evaluated as total suspended particulates (TSP); however, this included larger, noninhalable particles. By 1987, the importance of the inhalable fraction less than 10 μm became the focus of new ambient air standards. More stringent PM NAAQS led to not only a reduction of overall ambient air PM, but also saved lives (Laden 2006). Research was clearly beginning to show that particulate pollution was associated with respiratory system episodes, whereas no connection was apparent for other air components such as ozone and sulfur dioxide (Braun-Fahrlander 1992). Clearly, epidemiological data demonstrates a significant increase in pneumonia, chronic obstructive pulmonary disease, and other respiratory-inflammation-induced deaths associated with high air pollution days (Schwartz and Dockery 1992). However, according to Morton Lippmann, of New York University's (NYU) School of Environmental Medicine, sharpening the focus of the PM NAAQS by particle-size characteristics has gone as far as is reasonable to go. He states that to serve the public needs, the EPA and the scientific community need to generate data to provide a sufficient basis for chemical component-specific PM NAAQS that will target the PM components and/or sources that are most directly responsible for the adverse effects associated with PM mass concentrations (Lippmann 2010).

Exposure to metals in the air is capable of causing a myriad of human health effects, ranging from cardiovascular and pulmonary inflammation to cancer and damage of vital organs (Utsunomiya 2004). Contemporary research into air pollution is revealing that the metals components of particulate matter (PM) are contributing significantly to adverse health effects, even at the low levels found in ambient air (Pope et al. 1995). The EPA set health-based standards for fine particulates in 1997, but the standards do not take into account new research on the composition of the particulate matter or the toxicity of its components (Konkel 2009). The toxicity of particulate matter, in particular the fine and ultrafine particles (those particles smaller than 2.5 μm, has been proven to cause severe mortality and morbidity in humans over the past 25 years; however, in the past decade, emerging research is providing evidence that the metallic particles may be more dangerous than other PM components (Konkel 2009). In addition, current evidence is showing that mass concentration of PM alone may not be the best indices for associating health effects with exposure to PM (Costa and Dreher 1997)(Carter 1997).

The most recently published U.S. Census presents that approximately 80% of the U.S. population lives in urban areas (U.S. Bureau of the Census, 2000). Accordingly, a majority of the U.S. population is exposed to typical ambient metals concentrations found in urban environments. Furthermore, a significant segment of this population also lives in the vicinity of metals sources, such as waste incinerators, metal processors, metal fabrication, welding, etc., where they may be exposed to airborne metals greatly in excess of the typical ambient concentrations. Recent monitoring data in East St. Louis, Illinois depicts levels of metal HAPs in the general community and near schools that not only exceed residential regulatory limits, but reach levels above guidelines set for exposure in an occupational setting (arsenic, measured at approximately 2,340 ng/m^3; occupational short-term exposure limit is 2,000 ng/m^3)(Pettersen 2010). Levels such as these would prompt wearing respiratory personal protective equipment in

an industrial setting, yet these levels were found near not only unprotected members of the general public, but children, who are more sensitive to elevated airborne metals.

3.1.1 Metals in Particulate Matter

As mentioned in Section 2.0, trace metals are released to the atmosphere by the combustion of fossil fuels and wood, high temperature industrial activities and waste incinerations. Natural emissions are mainly from volcanism, wind erosion, as well as from forests fires and the oceans (Nordberg 2007). Specifically, the combustion of fossil fuels constitutes the principal anthropogenic source for beryllium, cobalt, mercury, molybdenum, nickel, antimony, selenium, tin and vanadium. Fossil fuel combustion also contributes to anthropogenic release of arsenic, chromium, copper, manganese and zinc. In addition, a large percentage of arsenic, cadmium, copper, nickel and zinc are emitted from industrial metallurgical processes. Exhaust emissions from gasoline formerly contained variable quantities of Lead, Copper, Zinc, Nickel and Cadmium. Zinc emission is also associated with tire rubber abrasion (Councell 2004).

Several independent groups of investigators have shown that the sizes of the airborne particles determine the potential to elicit inflammatory injury, oxidative damage, and other biological effects (Costa and Dreher, 1997; Lippmann, 2006; Ghio et al. 2002; Sangani et al, 2010; Utsonomiya et al., 2004). The particle size distribution of an aerosol will also determine the deposited fraction of inhaled particles in the various regions of the respiratory tract (Oller 2010). PM is a complex mixture of extremely small particles and liquid droplets and is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, and soil or dust particles. The EPA is concerned about particles that are 10 μm in diameter or smaller, because those are the particles that generally pass through the throat and nose and enter the lungs. Once inhaled, these particles can affect the heart and lungs, travel throughout the body, deposit in organs, penetrate cell membranes, and cause serious health effects (Adachi and Buseck 2010). EPA groups particle pollution into two categories:

- "Inhalable coarse particles," such as those found near roadways and dusty industries, are larger than 2.5 μm and smaller than 10 μm in diameter.
- "Fine particles," such as those found in smoke and haze, are 2.5 μm in diameter and smaller. These particles can be directly emitted from sources such as forest fires, or they can form when gases emitted from power plants, industries and automobiles react in the air.

These particles can be further defined with reference to relevant health effects for various regions, as described by Nieboer et al. (2005):

- The Inhalable aerosol fraction" is the fraction of total airborne particles that enters the body through the nose and/or mouth during breathing. This fraction corresponding to particles with aerodynamic diameter (d_{ae}) > 100 μm is relevant to health effects throughout the respiratory tract such as rhinitis, nasal, bronchial effects, and lung cancer. This fraction is also relevant for systemic effects.
- The ‘‘thoracic aerosol fraction" is a subfraction of the inhalable fraction [d_{ae} < 30 μm] composed of particles that can penetrate into the tracheo-alveolar region of the lung and is important for asthma, bronchitis, and lung cancer.

- The ‘‘respirable aerosol fraction” (or alveolar fraction) is the subfraction of the inhaled particles [d_{ae} < 10 μm] that penetrates into the alveolar region of the lung (i.e., includes the respiratory bronchioles, the alveolar ducts and sacs) and is pertinent to the development of such chronic diseases as pneumoconiosis and emphysema.

3.1.2 Characteristics of Fine Particulate Matter

The World Health Organization states that 2.4 million people die each year from causes directly attributable to air pollution (WHO 2002), particularly to fine particles (K. M. Ravindra 2001). According to current human health research, we now know that free radicals similar to those in cigarettes are also found in airborne fine particles and potentially can cause many of the same life-threatening conditions (Dollemore 2008).

In a study of trace metals in PM performed in the Detroit urban atmosphere, Utsonomiya et al. postulates that if toxic trace elements are homogenously dispersed as impurities in insoluble larger-size particles, risks to human health and the environment are less than if they occur as major constituents in individual, trace-metal, nanoscale particles (2004). Generally, the evaluation of most studies shows that the smaller the size and solubility of the PM, the higher the toxicity through mechanisms of oxidative stress and inflammation (Valavandis 2008). A study of $PM_{2.5}$ in 2010 showed that metals were the important source for cellular oxidant generation and subsequent health effects (Maciejczyk 2010). Health effects are stronger for fine (1 to 2.5 μm) and ultrafine (0.1 to 1 μm) particles for a variety of reasons:

1. The studies of the size distribution of metals show that most of the toxic metals accumulate in the smallest particles ($PM_{2.5}$ or less) (K. M. Ravindra 2008).
2. This size fraction can penetrate deeper into the airways of the respiratory tract and predominantly deposits in the alveolar region of the lungs, where the adsorption efficiency for trace elements varies from 60–80% (Pope and Dockery 2006).
3. A metallic particle in contact with lung tissue/cells involves the release of metal ions into the biological system (Midander 2007).
4. Fine and ultrafine particulate matter have the longest residence time in the atmosphere (~100 days), which allows for a large geographic distribution (Utsunomiya 2004).
5. Ultrafine particles are known to have increased solubility, as compared to larger size particles of the same composition because of the increased surface-to-volume ratio for smaller particle sizes (Navrotsky 2001).
6. Recent studies have shown that the metals component in fine and ultrafine PM is particularly toxic and are the primary contributors to negative human health (Magari et al. 2002).
7. Furthermore, these particles also play a significant role in global climate change and can be transported over long distances by prevailing winds (WHO. 2007).

These consequences demand to give priority to the chemical characterization of the fine and ultrafine fraction of airborne particles to understand their possible implication to health effects (K. M. Ravindra 2008). The size of PM with associated ability to penetrate the nasal, throat, and pulmonary boundaries can be seen below in Figure 3 (Particulate Danger 2008).

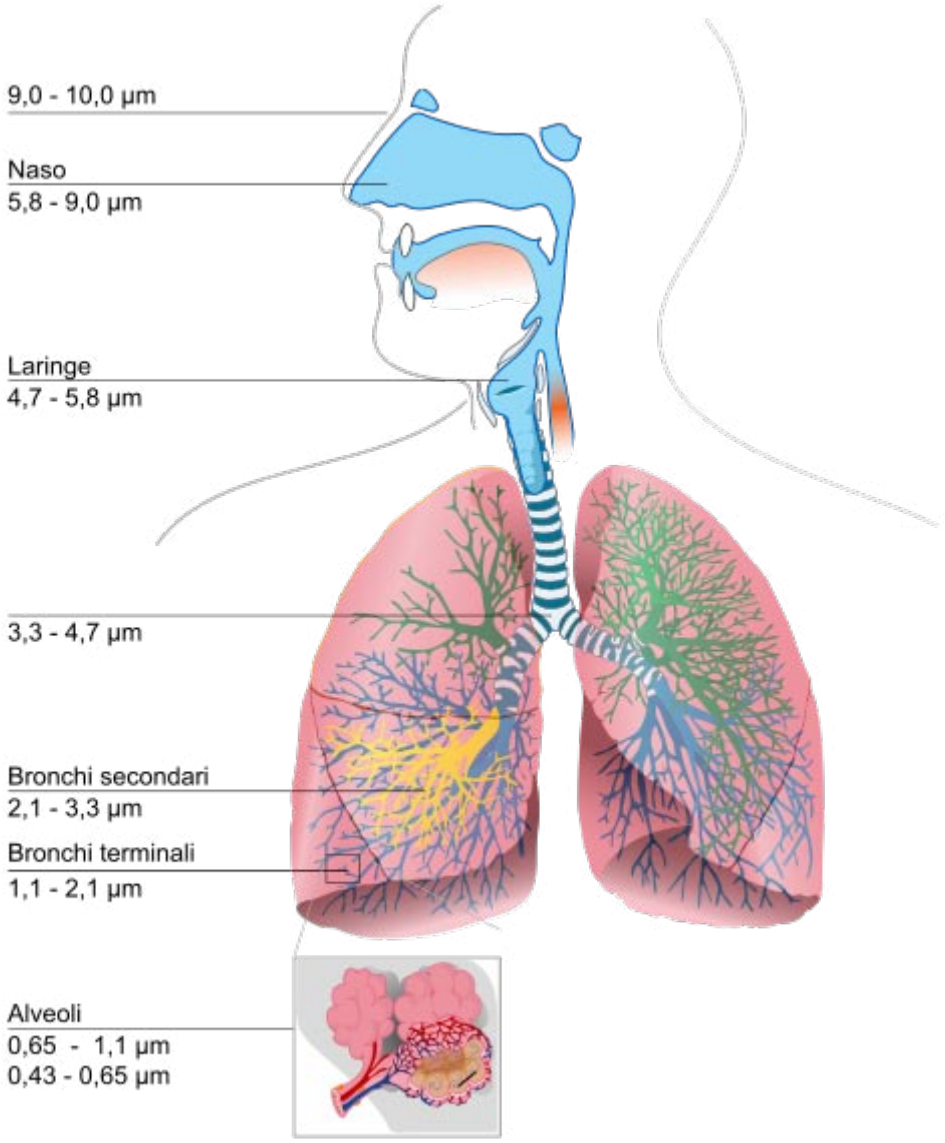

Figure C-3. Particulate size with associated depth of lung deposition

3.1.3 Bioavailable Transition Metals

In-vitro and *in-vivo* studies have identified generation of ROS and increased oxidative stress as a primary biological process that may contribute to produce such a variety of diverse health effects (Hou et al. 2010). Ionic forms of metals will be most bioavailable and therefore most likely to affect cells and organs beyond their deposition sites in the lung airways (Lippmann 2010). Bioavailability has been defined as the fraction of a compound in a matrix that is released from that matrix and absorbed into the body's blood plasma (Spear et al. 1998). An influential study by Daniel Costa and Kevin Dreher in 1997 evaluated the relative importance of bioavailable metal to that of the mass dose of PM. Their results indicated that the lung dose of bioavailable transition metal, not instilled PM mass, was the primary determinant of the acute inflammatory response for both the combustion source and ambient PM samples (Costa and Dreher 1997). Transition metals, such as iron, vanadium, nickel, chromium, copper, and zinc,

have been particularly cited a most likely to be toxic on the basis of their ability to support electron exchange (Ghio 1996) and catalyze and generate ROS in biological tissues (Chen and Lippmann 2009). ROS, such as hydroxyl radicals (OH·), are thought to be involved in various forms of lung injury and are considered to be both genotoxic and carcinogenic (Knaapen et al. 2004).

A study in the Utah Valley looked at the effect of metal, in particular transition metal, removal on the toxicity of airborne particulate matter (Molinelli et al. 2002). This study was prompted by human health data discovered following the closure of an open-hearth steel mill over a year in1987 (Pope 1989); respiratory disease and related hospital admissions were reduced significantly and increased upon reopening (Dye et al. 2001). In a subsequent study of PM effects, metals were removed from an aqueous extract of PM collected in the Utah Valley during the operation of the steel mill. The treated (i.e. metals removed) and untreated (i.e. including original transition metal content) PM samples were exposed to human airway epithelial cells and to rats *in vivo*. Cells that were incubated with the untreated extract (the extract containing metals) showed a significant concentration-dependent increase in the inflammatory mediator interleukin-8 (IL-8) when compared to the control cells. It was also found that significant increases in lactate dehydrogenase (LDH) and total protein occurred in the rats exposed to the untreated extract effects. Generally speaking, the extracts that were stripped of their transition metal content lost their potency to elicit inflammatory responses in the lung. Taken together, this research supports a role for transition metal involvement in PM-associated increases in morbidity and mortality (NHEERL 2010).

A similar effect was found in Redcar, England when a steel plant was closed temporarily. The total metal content of PM_{10} collected before and during the closure period were similar, but on reopening of the steel plant there was a significant 3-fold increase ($p < 0.05$) compared with the closure and pre-closure samples. Of metals analyzed, iron was most abundant in the total and acid extract, while zinc was the most prevalent metal in the water-soluble fraction. Conclusions of the study found that PM_{10}-induced inflammation in the rat lung was related to the concentration of metals in the PM_{10} samples tested, and activity was found in both the soluble and insoluble fractions of the particulate pollutant (Hutchison et al. 2005).

Recent work at NYU's Department of Environmental Medicine looked at daily exposure of concentrated ambient particulate matter (CAPs) to ApoE mice. After exposure, significant increases in heart rate and heart rate variability were found on 14 of the 103 days. A back trajectory analysis of the weather patterns on all 14 of these days demonstrated a wind from the northwest. In addition, trace metal analyses found significant increases in nickel, chromium, and iron, even though the CAPs concentration was markedly reduced on those days. It was then discovered that the air mass passed by the largest nickel refinery plant in North America, located in Sudbury, Ontario, thus suggesting a source for the observed changes in heart rate and heart rate variability. The CAPs inhalation studies at NYU suggest that CAPs overall, and nickel in particular, yields evidence that current levels of ambient air concentrations produce health effects of interest in terms of public health (Lippmann et al. 2006).

Metals in the water-soluble fraction of air pollution particles decrease whole-blood coagulation time. A 1999 study of pulmonary toxicity of PM found that the pulmonary response and cell injury following exposure to this urban dust is related to soluble material, probably metal ions, rather than to the number or composition of the insoluble particles (Adamson 1999). These metals can potentially contribute to procoagulative effects observed following human exposures to air pollution particles (Sangani et al. 2010). Transition metals are also being shown to have involvement in the immunotoxicity of inhaled ambient PM. Iron and Nickel reduced the

clearance of bacteria from the lungs of infected rats, demonstrating that inhaled ambient metals can worsen the outcome of pulmonary infection (Zelikoff et al. 2002).

Additional data not only extrapolates animal toxicity testing to humans, but also provides evidence linking transition metals directly to human health effects. Groups of particular interest include the elderly, pregnant women, those with heart and lung disorders, and young children. A study performed by researchers at the Columbia University's Center for Children's Environmental Health followed more than 700 children between birth and two years of age living in northern Manhattan and the south Bronx. New York City, NY has the highest average ambient air concentration of Nickel at 19 ng/m^3 (versus the national average of 1.9 ng/m^3; (Lippmann et al. 2006)). Researchers found that children exposed to nickel and vanadium in the air were more likely to wheeze. Michelle Bell, a Yale University environmental health scientist, looked at respiratory and cardiovascular hospital admissions of patients 65 years or older and found that counties with higher nickel, vanadium, and elemental carbon were found to have higher risk of hospitalizations associated with short-term particulate exposure (Konkel 2009).

Evidence points towards genotoxicity of inhaled transition metals as well. Mitochondria are the major intracellular source and primary target of ROS, which are generated under normal conditions as by-products of aerobic metabolism in animal and human cells. Compared with nuclear DNA, mitochondrial DNA lacks protective histones and has diminished DNA repair capacity, and is therefore particularly susceptible to ROS-induced damage. Cells challenged with ROS have been shown to synthesize more copies of their mitochondrial DNA and increase their mitochondrial abundance to compensate for damage and meet the increased respiratory demand required for ROS clearance. At the same time, ROS are also generated from the increased mitochondria in these cells and thus cause additional oxidative damage to mitochondria and other intracellular constituents including DNA, RNA, proteins, and lipids. In a study by Hou et al., increased mitochondrial DNA in circulating blood leukocytes was positively correlated with blood markers of oxidative stress. Mitochondrial damage and dysfunction, as reflected in increased mitochondrial DNA copies, may thus represent a biological effect along the path linking PM inhalation to its health effects (Hou et al. 2010).

Table C-5 presents a chronological summary of studies that implicate metals as significant contributors to human health effects.

3.1.4 Vanadium and Nickel producing ROS

Current research is providing strong evidence that nickel and vanadium are accounting for a large portion of adverse human health effects. A majority of *in vitro* and animal model investigations support the postulation that transition metals, which are present in such substances as residual oil fly ash (ROFA), participate in Fenton-like chemical reactions to produce ROS (Ghio et al. 2002). In the Fenton reaction, iron reacts with naturally present hydrogen peroxide to form free radicals, such as hydroxyl radicals (OH·). Research shows that special attention must be taken with Ni and other transition metal concentrations, because of their high potential bioavailability. Nickel is important, in particular, mainly due to the high solubility of the chemical forms of Ni in the finest particles (Fernandez-Espinosa 2004).

Table C-5. Chronological Summary of Studies Implicating Metals

Date	Type	Corresponding Author	Relevant Key Conclusions
1971	R	EPA	NAAQS for total suspended particles (TSP) include larger, noninhalable particles
1987	R	EPA	Regulatory focus on combination of fine (<2.5 μm) and coarse (<10 μm) particulate matter (PM), all of which can be inhaled into the respiratory tract.
1989	E, H	Pope A.C. III	"Closure of an open-hearth steel mill in the Utah Valley over the winter of 1987 was associated with reductions in respiratory disease and hospital admissions in valley residents; effects were a result of reduction in PM levels."
1992	H, E	Braun-Fahrlander et al.	"Particulate pollution (TSP) was associated with incidence and duration of respiratory symptom episodes. No such associations were observed with $S0_2$ or ozone."
1992	H, E	Schwartz and Dockery	"A 100 μg/m^3 increase in TSP was associated with an increase in mortality due to COPD, pneumonia, and cardiovascular disease equal to 19, 11, and 10%, respectively. On high pollution days, COPD, pneumonia, and dead-on-arrival deaths were disproportionately increased."
1995	R	Pope, Bates, and Raizenne	"...Respirable particulate air pollution is likely an important contributing factor to respiratory disease."
			"Observed (PM) health effects include increased respiratory symptoms, decreased lung function, increased hospitalizations and other health care visits for respiratory and cardio-vascular disease, increased respiratory morbidity as measured by absenteeism from work or school or other restrictions in activity, and increased cardiopulmonary disease mortality. "
			"These (PM) health effects are observed at levels common to many U.S. cities including levels below current U.S. National Ambient Air Quality Standards for particulate air pollution."
1996	T	Ghio et al.	"Transition metals have the capacity to support electron exchange and catalyze free radical production."
1997	T	Carter et al.	"Free radicals can induce alterations in host pulmonary defense, inflammation, and lung injury."
1997	T, A	Costa and Dreher	"..The lung dose of bioavailable transition metal, not instilled PM mass, was the primary determinant of the acute inflammatory response for both the combustion source and ambient PM samples."
			"...Soluble metals from PM mediate the array of PM-associated injuries to the cardiopulmonary system of the healthy and at-risk compromised host."
1999	T, A	Adamson et al.	"This (study) suggests that the pulmonary response and cell injury following exposure to this urban dust is related to soluble material, probably metal ions, rather than to the number or composition of the insoluble particles."
2001	E, H, T	California Air Resources Board	"Groundbreaking long-term studies of children's health conducted in California have demonstrated that particle pollution may significantly reduce lung function growth in children."
2001	R	Ravindra et al.	"Data on daily mortality show that, on a global scale, 4% to 8% of premature deaths may occur due to the exposure of suspended particulate matter (SPM) and especially of fine particles ($PM_{2.5}$) in the ambient and indoor environment."
2002	R	California Air Resources Board	"Premature deaths linked to particulate matter or "PM" are now at levels comparable to deaths from traffic accidents and second-hand smoke. "
2002	H, T	Magari et al.	"The metals component in fine and ultrafine PM is particularly toxic and are the primary contributors to negative human health."
			"…Results of this study suggests an association between exposure to airborne metals (vanadium, nickel, chromium, lead, copper, and manganese) and significant alterations in cardiac autonomic function."
2002	R	Ghio et al.	"…Transition metals present in ROFA (especially vanadium) participate in Fenton-like chemical reactions to produce reactive oxygen species (ROS)."
2002	T, A	Zelikoff et al.	"…Both iron and nickel (inhaled) reduced pulmonary bacterial clearance in previously infected rats."
2002	T, A	Molinelli et al.	"…Ambient air particles in the Utah Valley induce their health effects in part by delivering transition metals to the airway epithelium in a catalytically-active form."
			"Results (*in vivo* and *in vitro*) support a role for transition metal involvement in PM-associated increases in morbidity and mortality."
2004	T, H	Fernandez-Espinosa and Ternero-Rodriguez	"Physical speciation of lead showed that the major risk for health was from fine particles less than 2.7 μm, because particles between 2.7 and 0.6 μm were the size fractions of the TSP with the major mass abundance of lead (mainly particles between 2.7 and 1.3 μm) and because particles less than 0.6 μm were the fraction of the airborne particles most abundant in the urban air."
			"Chemical speciation results showed that special attention must be taken with Ni and Cd concentrations because of their high potential bioavailability, mainly a result of the high solubility of the chemical forms of Ni in the finest particles."
2004	T	Utsonomiya et al.	"If toxic trace elements are homogenously dispersed as impurities in insoluble larger-size particles, risks to human health and the environment are less than if they occur as major constituents in individual, trace-metal, nanoscale particles "
			"Fine and ultrafine particulate matter have the longest residence time in the atmosphere (~100 days), which allows for a large geographic distribution"
			"Ultrafine particles…have increased solubility as compared to larger size particles of the same composition because of the increased surface-to-volume ratio for smaller particles."
2005	R, E	Thurston et al.	"…treating all particles that contribute to mass concentration (of PM) equally in the regulatory process may lead to inefficient protection of public health. A potentially more effective approach would be to address the individual types of particles independently, focusing control efforts on the most toxic categories."
2006	E, H, T	Karthikeyan et al.	"Daily Respiratory Uptake (DRU) values were significantly higher for several metals, including Zn, Cu, and Fe, during bushfires. ...Measurements showed that the particulate samples collected during bush fires generate more toxic hydroxyl radicals (OH·) than those in the background air, due to the presence of more soluble iron ions."
			"As a result (of increased metals from bushfires), people seeking treatment for respiratory-tract infections went up 25% and asthma cases went up 29%."
2006	T, A	Lippmann et al.	"Nickel appears to be the component (of fine particulate matter) most likely to cause acute cardiac responses."
2006	T, A	Pope and Dockery	"The fine PM less than 2.5 μm fraction predominantly deposits in the alveolar region of the lungs, where the adsorption efficiency for trace elements varies from 60–80%."

Table C-5. Chronological Summary of Studies Implicating Metals

Date	Type	Corresponding Author	Relevant Key Conclusions
2007	E	WHO	"Fine particles also play a significant role in global climate change and can be transported over long distances by prevailing winds."
2008	O,T	Ravindra et al.	"The studies of size distribution of metals show that most of the toxic metals accumulate in the smallest particles ($PM_{2.5}$ or less)."
			"Variations in natural/anthropogenic fractions of $PM_{2.5}$ were…found to be a function of meteorological conditions as well as of long-range transport of air masses from industrialized regions…"
2008	R	Dollemore	"Epidemiological studies suggest that more than 50,000 Americans die each year from cardiopulmonary disease linked to breathing fine particle air pollution."
			"According to current human health research, we now know that free radicals similar to those in cigarettes are also found in airborne fine particles and potentially can cause many of the same life-threatening conditions."
2008	T	Valavandidis et al.	The smaller the size of the PM, the higher the toxicity through mechanisms of oxidative stress and inflammation
			Recent data emphasize the importance of water-soluble constituents, such as transition metals..known to be adsorbed onto the surface cavities of the PM and can be released in the lung alveoli and deposited in the lung parenchyma.
			Toxicological studies in vivo and in vitro showed that PM have the ability to generate continuously high reactive ROS, which contribute to the genotoxic and cytotoxic mechanisms of PM.
2009	MA	Konkel	"(There is)…evidence linking nickel, vanadium, and elemental carbon in the air to wheeze and cough in inner city children."
			"Total levels of particulates were not significantly associated with wheeze or cough, suggesting that individual ingredients - not fine particles as a whole - may be harmful."
2009	R	Chen and Lippmann	"Residual Oil Fly Ash (ROFA), which is…notably high in vanadium and nickel, were more toxic than other source-related mixtures that have been tested in laboratory animals in vivo or in cell in vitro."
			"…Concentrations of nickel and vanadium in ambient air PM were associated with significant differences in mortality rates, while other measured PM components were not."
			"Ambient air fine particulate matter toxicity may vary depending on its source and chemical composition."
			"...Reduction of metals in PM associated with a year-long closure of a steel mill was associated with improved health conditions in the local population. The role of metals was further confirmed by later studies using human clinical as well as animal toxicology studies."
2010	T,H	Maciejczyk et al.	"Metals in PM2.5 (especially nickel) were the important source for cellular oxidant generation and may be responsible for subsequent health effects associate with particle air pollution."
			"Three metals (nickel, barium, and manganese)… appear to be much more influential on lung cell responses than black carbon and sulfate ions that are present at much higher mass concentrations."
2010	R	EPA	"...Recent studies have shown that extracts stripped of transition metals lose their potency and that solutions of pure transition metals in the ratios and concentrations found in the particles can induce similar effects in animals as the complete extracts. This conformed to a theory of particle toxicity emphasizing the ability of metals on fine particulates to induce toxic oxygen radical species and subsequent inflammatory response in the lung."
			"While particle size does matter, particle composition seems also to be extremely important in the pulmonary inflammatory response to particulate air pollution."
2010	T	Sangani et al.	"Metals in the water-soluble fraction of air pollution particles decrease whole-blood coagulation time."
2010	MA	Lippmann	"...The EPA and the scientific community need to generate data to provide an adequate basis for chemical component-specific PM NAAQS that will target the PM components and/or sources that are most directly responsible for the adverse effects associated with PM mass concentrations."
			"...The lack of daily data on concentrations makes it all but impossible to accurately study acute responses to peaks in exposure."

Type:
O = Original
A = Animal Study
E = Epidemiological
H = Human Study
T = Toxicological
R = Review
MA = Magazine Article

3.2 **Olfactory Risk**

Recent studies have found that certain metals can result in olfactory and neurological injury. The olfactory system forms a direct interface between the nervous system and the external environment (Aschner et al. 2005). The olfactory (nasal) neuron can provide a pathway by which foreign materials can reach the brain (Bondier et al. 2008). The metals with potential to

travel through the olfactory nerve are aluminum, cadmium, cobalt, mercury, manganese, nickel, and zinc.

Two metals that have recently been shown to be particularly toxic through the olfactory nerve transportation pathway are cadmium and manganese. As in inhalation studies, results of investigations into particle size uptake of manganese (Fechter et al. 2002) suggested that particle size may influence the delivery of manganese to the rat olfactory bulb because manganese delivery appeared to be smaller for the larger particles (18 μm) and larger for the fine particles (1.3 μm). The addition of methylcyclopentadienyl manganese tricarbonyl (MMT) to gasoline in some countries led to increased levels of manganese in the atmosphere; furthermore, the nervous system is the major target organ of manganese. Manganese species and solubility also have an influence on the brain distribution of Mn in rats (Normandin et al. 2004) and were found in dopamine-rich areas of the brain (Antonini 2010). Bondier et al. recommend that the olfactory route of entry for cadmium should be taken into account as cadmium is able to penetrate the CNS and cause brain injury (2008). Although both metals are considered dangerous via the olfactory route, manganese is also capable of traveling along secondary and tertiary neurons, whereas cadmium is not (Aschner et al. 2005).

3.3 Industrial Health Effects

Occupational exposure to particles has been related to respiratory tract cancer in humans in the past and present time. Most of the epidemiological evidence for this has been gathered from high occupational exposures in the past (Knaapen et al. 2004).

Foundry work has been associated with various adverse health outcomes, including cardiovascular disease, respiratory disease, and lung cancer, that may depend on ROS-induced damage and genotoxicity. Even in modern foundry facilities that adopt state-of-art measures for exposure reduction, workers are still exposed to substantially higher levels of airborne PM compared to those found outdoors. In a study by Hou et al., the association of blood mitochondrial DNA with exposures to airborne PM and its metal components was investigated for foundry workers exposed to a wide range of PM levels (2010). The results of this study showed that PM exposure is associated with damaged mitochondria, which subsequently intensify oxidative-stress production and effects.

As reported by Oller and Oberdorster, even though the issue of particle size distribution differences between animal and human aerosols has been raised before and is well known to inhalation toxicologists, it has not yet been incorporated into Occupational Exposure Limits (OEL) setting frameworks in the European Union or the United States (2010). When comparing the levels considered to adversely affect human health at a community level versus levels permissible in an occupational setting, it is clear that a large discrepancy exists. Although the occupational scenario considers a shorter duration of exposure (i.e. an 8- to 10-hour work day), occupational exposure levels (OELs) are not necessarily keeping pace with current scientific knowledge (Oller and Oberdoerster. 2010). In addition, extreme caution must be exercised with such metals as lead and beryllium as the U.S. National Research Council has been unable to establish any safe level of exposure (NRC 2008).

The advantage of OELs is that they are produced through a very strict process that provides checks and balances, which makes these limits legally enforceable (Meagher 2002). However, when looking at exposure limits for Nickel, the most relevant medical and toxicological literature lists an acute (1-hour) average of 6 $\mu g/m^3$ (as reported in California EPA's reference exposure level (REL), whereas the 8-hour permissible exposure limit (PEL) is 1,000 $\mu g/m^3$ (equaling 125

$\mu g/m^3$ for 1 hour). Even more recent data, such as research being performed by Morton Lippmann and Chi Chen, suggests that nickel at even the lowest ambient levels has the potential to cause adverse health effects (2009).

3.4 International Air Quality

The World Health Organization (WHO) estimates that urban air pollution contributes to approximately 800,000 premature deaths and 4.6 million lost life-years worldwide each year (HEI 2004). PM pollution is estimated to cause 22,000-52,000 premature deaths per year in the United States and 200,000 premature deaths per year in Europe (Mokdad et al. 2004). Emissions from industrial and mobile sources are not bound by country borders. The concentration, composition, and size of suspended particulate matter at any given site are determined by such factors as: meteorological properties of the atmosphere, topographical influences, emission sources, and particulate parameters such as density, shape, and hygroscopicity (Fang et al. 2010). Data on daily mortality show that, on a global scale, 4% - 8% of premature deaths may occur due to the exposure of suspended PM and especially of fine PM in the ambient and indoor environment. The anthropogenic group of metals, such as zinc, lead, sulfur, vanadium, chromium, copper, nickel, and bromine occur in scattered relative abundance in $PM_{2.5}$ samples (K. M. Ravindra 2008). The concentration pattern seems to be related to the changes in meteorological conditions, such as wind speed, wind direction, or, on a larger scale, long range transports, which can cause large differences in the amount of different anthropogenic elements in $PM_{2.5}$.

Countries without the regulatory infrastructure to properly monitor and reduce air toxics are particularly susceptible to elevated levels of airborne metals in ambient air. In fact, in a report by the Health Effects Institute in Boston, Massachusetts, of the 800,000 premature deaths attributable to urban air pollution worldwide, two-thirds of the deaths occur in developing countries in Asia (HEI 2004). Some of these countries do not have high enough amounts of industrial activity to incite human health risks, such as in some African countries; however, other countries, such as Mexico and China, have levels of air toxics introduced by industrial activity far above what would be considered “safe” in more regulated systems. In Shanghai, China, concentrations of trace metals, such as chromium, manganese, nickel, and zinc, in PM are reported far above the human health protection levels listed by the U.S. EPA (Weisheng et al. 2007). These types of modern environmental health hazards are products of rapid development in the absence of health and environmental safeguards, as well as the unsustainable consumption of natural resources (WHO 1997).

Significant health impacts from metal-containing air pollution have been reported in several developing countries. Bushfires in Singapore created significant increases in the concentration of Zn, Fe, and Cu. As a result, people seeking treatment for respiratory-tract infections went up 25% and asthma cases went up 29% (Karthikeyan et al. 2006). Electron paramagnetic resonance (EPR) measurements showed that the particulate samples collected during the bushfires generated more toxic hydroxyl radicals than those in the background air, most likely due to the presence of more soluble iron (and other transition metal) ions. In Africa, an important source of direct human exposure to high levels of mercury is artisanal gold mining and processing. Exposure to vaporized mercury occurs during burning to separate gold from the gold-mercury amalgam. The release of mercury occurs within the breathing zone of workers who are typically not equipped with personal protective equipment (Nweke and Sanders 2009).

A study of metals in traffic pollution performed in Spain found important relationship between Pb and Cu, which supports current research into PM speciation. Physical speciation of lead showed

that the major risk for health was from fine particles less than 2.7 μm, because particles between 2.7 and 0.6 μm were the size fractions of the total suspended particles with the major mass abundance of lead (mainly particles between 2.7 and 1.3 μm) and because particles less than 0.6 μm were the fraction of airborne particles most abundant in the urban air. Chemical speciation results showed that special attention must be taken with nickel and cadmium concentrations, because of their high potential bioavailability, mainly a result of the high solubility of the chemical forms of Ni in the finest particles (Fernandez-Espinosa 2004).

A study of metal nanoparticles, defined in this study as particles smaller than 50 nanometers, in Mexico City revealed that well over half of the 572 metal nanoparticles contained two or more metals. Of these, iron, lead, or zinc occurred in more than 60%. In addition, many of the metal-bearing nanoparticles were attached to or embedded within host particles, which were mainly organic matter and sulfate, both of which are more soluble than most metal-bearing nanoparticles and will dissolve in the deposited region of the body/lung, leaving the metal nanoparticles behind. The findings of this study implied that different physiological areas and effects would result from the inhalation of either free-floating metal nanoparticles or metal nanoparticles carried by host material (Adachi and Buseck 2010).

3.5 Future Research on Metals in Air Pollution

As described in the previous sections, contemporary researchers in the field of airborne metals' health effects are finding that the metals components of PM are quite toxic and cause various significant health effects from pulmonary inflammation (Costa and Dreher 1997) to increased heart rate variability (Lippmann et al. 2006) to decreased immune response (Zelikoff et al. 2002). These effects are not only seen from chronic exposure, but also from short-term peaks in ambient air concentrations (Chen and Lippmann 2009).

When dealing with a community near a single source of HAP metals, a 24 hour integrated average sample taken ever third or sixth day is not likely to provide an adequate indicator of short-term exposure levels. This is due in part to the fact that when a monitor is close to a source, some of the time the wind will move the emissions in the opposite direction of the monitor, and the metal concentration at the monitor will be close to zero. Thus, to achieve the average, the concentrations at shorter time intervals must be substantially greater than the 24 hour average.

Accurate assessment of human exposures to air pollution is an important part of environmental health effects research; however, most air pollution epidemiological studies rely upon imperfect surrogates of personal exposure, such as information based on available central-site outdoor concentration modeling or modeling data. In a 2008 study modeling population exposures to HAPs, results indicated that the total predicted chronic exposure concentration of outdoor HAPs from all sources are lower than the modeled ambient concentrations by about 20% on average for most gaseous HAPs and by about 60% on average for most particulate HAPs (Ozkaynak et al. 2008). These findings highlight the importance of applying exposure-modeling methods, which incorporate information on time-activity, commuting and exposure factors data, in order to accurately determine human exposure to HAPs.

Researchers are calling for further investigation into the characterization of ambient air metals. Terry Gordon of NYU's Department of Environmental Medicine (2007) calls for:

1) Larger numbers of study subjects
2) More frequent time points for response and exposure assessment

3) More precise (time resolution & component speciation) measurements of exposure to account for the temporal-spatial variability in exposure parameters.

As reported by Morton Lippmann, continuous records of PM components such as coarse mode PM, fine PM, and perhaps ultrafine PM would greatly accelerate the accumulation of knowledge on PM component exposure–response relationships that would provide a sound basis for more targeted air quality standards and pollution control measures. He also states that "the lack of daily data on concentrations makes it all but impossible to accurately study acute responses to peaks in exposure" (Lippmann 2010). Furthermore, legislation concerned only with measuring physical PM mass concentrations fails to address potential health effects linked to chemical variations in ambient aerosols (Moreno 2009). Most importantly, equal treatment of all particles that contribute to mass, irrespective of composition, may be leading to less-optimal control strategies to avoid the adverse human health effects of PM. A more effective approach would be to address the types of particles independently, focusing control efforts on the most toxic categories (Thurston 2005).

4.0 Key Source Indicating Metals for Apportionment

The goal of source apportionment is to determine contributions of various pollution sources to a location of interest (e.g. outdoor, indoor, or personal exposure measurement). Accurate size-resolved chemical characterization of emissions from potential sources in each air shed is an essential component in receptor modeling studies. Lack of this source information is currently one of the major, if not most significant, factors limiting further improvements in the accuracy and precision of quantitative source apportionment. Identifying and quantifying a source's contribution relies on a comparison of the chemical and physical features of the ambient aerosol measured at the receptor with the features of an aerosol emitted from a potential source.

Ambient air fine PM is a chemically nonspecific pollutant, and may originate from, or be derived from, various emission source types; thus, fine PM toxicity may well vary, depending on its source and chemical composition. If the fine PM toxicity could be associated with specific source signatures, then health effects research could be better focused on specific FPM components that come from those sources and specific biological mechanisms could be postulated for further consideration by toxicological studies.

Source composition varies according to industrial and other anthropogenic activities, as well as naturally occurring geologic events. Typical source contributions can be characterized in the following categories (Cooper 1999):

a. Geological – soil dust, agricultural tilling, rock crushing; mostly larger, coarse particles
b. Automotive and truck exhaust – fuels, prior use of leaded gasoline, diesel (indicated by high elemental carbon); mostly fine particles
c. Stationary fossil fuel combustion – residual oil, coal, distillate oil, natural gas; fine and coarse particles
d. Primary emissions from industrial point sources – typically fine particles, varies with industrial activity
e. Residential solid fuels (wood and coal) – high levels of pollutants with current technology; fine and coarse particles
f. Secondary aerosols – sulfate, nitrate, hydrocarbons; fine particles
g. Natural sources – pollen, spores, leaf fragments, biomass emissions; mostly coarse particles
h. Miscellaneous sources (galvanizing, boiler cleaners, construction, etc)
i. Background aerosols – marine, continental, material entering an airshed with the prevalent air mass, not subject to control

A study performed in Japan measured concentrations of elements vanadium, calcium, cadmium, iron, barium, magnesium, manganese, lead, strontium, zinc, cobalt and copper in aerosols with ICP-MS. The results showed that calcium, magnesium, manganese, strontium, cobalt, and iron were mainly associated with coarse particles (>2.1 μm), primarily from natural sources. In contrast, the elements zinc, barium, cadmium, vanadium, lead, and copper dominated in fine aerosol particles (<2.1 μm), implying that the anthropogenic origin is the dominant source. Results of the factor analysis on elements with high crust values (>10) showed that emissions from waste combustion in incinerators, oil combustion (involving waste oil burning and oil combustion in both incinerators and electricity generation plants), as well as coal combustion in electricity generation plants were major contributors of anthropogenic metals in the ambient atmosphere in Kanazawa (Wang et al. 2006).

Table C-6: Examples of key indicating elements with associated sources

Source	Indicating Elements
Crustal/Geologic	Na, Mg, Al, Si, K, Ca, Sc, Ti, Mn, Fe, Ga, Rb, Sr, and Zr
Coal Combustion	Crustal plus fine PM such as Ge, As, Se, Sb, Ba, W, U, Hg, and B
Oil Combustion	V, Ni, and Mo (fine PM)
Petroleum Refinery	La, Ce, Nd, and other elements specific to process
Automotive	Br and Pb (fine PM)
Copper, Nickel, and Lead Smelters	Cu, As, Cd, Pb, In, Sn, Sb
Marine Aerosol	Na, Cl
Vegetative Burning	Organic Carbon, Elemental Carbon, K, Cl, Zn
Iron and Steel Industry	Fe, Co, Cr, Ni, and Mg (fine PM)
Na=sodium, Mg=magnesium, Si=silicon, K=potassium, Ca=calcium, Sc=Scandium, Ti= titanium, Mn=manganese, Fe=iron, Ga=gallium, Rb=rubidium, Sr=strontium, Zr=zirconium, Ge=germanium, As=arsenic, Se=selenium, Sb=antimony, Ba=barium, W=tungsten, U=uranium, Hg=mercury, B=boron, V=vanadium, Ni=nickel, Mo=molybdenum, La=lanthanum, Ce=cerium, Nd=neodymium, Br=bromine, Pb=lead, Cu=copper, Cd=cadmium, In=indium, Sn=tin, Cl=chlorine, Zn=zinc, Co=cobalt, Cr=chromium	

Understanding the sources of ambient particulate matter has become increasingly important. While local sources can be monitored and subjected to local control regulations, particulate matter that has been transported into the region cannot be easily monitored or controlled. The commonly identified source categories include: secondary sulfate/coal burning (sometimes over 50 percent of the mass), secondary organic carbon/mobile sources, crustal sources, biomass burning, nitrate, industrial, smelters and metal processing, and sea salt in coastal regions. Frequently, the smaller sources and the mobile sources appear as combinations of sources. The combined sources are usually those that would naturally affect the receptor concurrently, such as a mobile — road dust or road salt combination. The main tools for apportioning the sources rely on variations in source strength to separate the sources; hence, sources acting together cannot be separated. Consequently, while separating the mobile sources into diesel- and gasoline-based emissions is clearly a goal for several of the studies, the success depends on either additional data or analyses.

Airborne PM pollution is presently regulated by the NAAQS using gravimetric mass as the particle metric to assess air quality. However, an enormous number of different chemical species are associated with the various types of ambient particles, depending upon their source origins (e.g., Cooper and Watson 1980). For example, primary particles emitted from coal combustion are characteristically enriched with arsenic and selenium, whereas residual oil combustion particles are more enriched in nickel and vanadium, and soil particles are especially enriched in the crustal elements (e.g., silicon, aluminum). In addition, secondary components of particles (e.g., sulfates, nitrates, and organic compounds) are formed in the atmosphere from gaseous pollutant emissions. These secondary components can either condense on primary particles or form secondary particles that can then collide and coagulate with primary particles. Individual particles in an urban airshed can contain both primary and secondary components,

and the composition of ambient aerosols have been found to reflect source PM emission characteristics differences over space (e.g., between cities) and time (e.g., across seasons) (e.g., Spengler and Thurston 1983). Because the composition of particle types varies greatly, it is probable that some types of particles are more toxic than others. Thus, treating all particles that contribute to the mass concentration equally in the regulatory process may lead to inefficient protection of public health. A potentially more effective regulatory approach would be to address the individual types of particles independently, focusing control efforts on the most toxic categories.

In conclusion, for the effective management of air quality, great importance must be attached to the identification of the sources of suspended PM. Source apportionment provides an estimate on the PM contribution of various sources to the levels at the receptor; it is also a key component necessary for developing and achieving desired air-quality objectives. Source apportionment methods rely on the principle that if a group of chemical constituents have a common origin, they should show a similar variation. The results of source apportionment can be used to evaluate emissions reduction on the PM levels and to devise more efficient emission reduction strategies. Therefore, estimating the airborne PM mass concentration, as well as individual chemical/metal speciation, is very critical not only for comparing with recommended values, but also to identify the major sources that affect a particular area. This knowledge will also help regulators both foresee and prevent threats and risks before they become problems (Abdul-Wahab 2004).

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