2

Comprehensive Comparison of Trace Metal Concentrations in Inhaled Air Samples

Mehmet Yaman
Firat University, Science Faculty,
Department of Chemistry, Elazig
Turkey

1. Introduction

Because metals cannot be degraded or destroyed, the assessment of the health risks of metals via ambient air and dietary intake is an issue of special interest. Trace metals in air phase can be classified as metals or metalloids including the semi-metallic elements: boron, arsenic, selenium, and tellurium. Both natural and anthropogenic processes and sources emit metals and their compounds into the air. Anthropogenics; the processing of minerals, incineration of metallic objects, motor vehicle combustion of fuel containing metal additives, and the wearing out of motor vehicle tyres and brake pads result in the emission of metals associated with particulate matter. Metals occur naturally in soil and in rocks rich with minerals; thus weathering of the rocks, mining activities or even wind-blown dust can release these metals into air as particulate matter. Trace metals are part of a large group of air pollutants called air toxics, which upon inhalation or ingestion can be responsible for a range of health effects such as cancer, neurotoxicity, immunotoxicity, cardiotoxicity, reproductive toxicity, teratogenesis and genotoxicity (1-4).

When inhaled, very small particles containing metals or their compounds deposit beyond the bronchial regions of the lungs into the alveoli region. Epidemiological studies have established relationships between inhaled suspended particulate matter and morbidity/ mortality in populations (6-7). Studies in occupational or community settings have established the health effects of exposure to trace metals, such as lead, cadmium, nickel and their compounds (8-9). The accumulation of metals in human body can have middle and long-term health risks and can adversely affect the physiological functions (1-4). Metals can enter the human body mainly through inhalation and ingestion, with the diet being the main route of human exposure for non-occupationally exposed individuals. To evaluate and reduce the health and environmental effects of toxic metals in inhaled ambient air and food matrices, it is vitally important to know their chemical compositions and the way they vary in time and in space. Therefore, there are continuing efforts to determine particularly toxic metals such as Pb, Cd and Ni in air phases and food samples (10-14). In considering lead and cadmium in ambient air samples, this importance increases because the absorption rates of those metals by inhalation are significantly higher (up to 50-60%) than those by ingestion (between 3% and 10%) (10). The localized release of some heavy metals from inhaled particulate matter has been hypothesized to be responsible for the lung tissue damage.

In spite of all these facts, there are fewer studies available on Pb and Cd determinations in air samples compared to other food matrices due to, probably, the excessively lower concentrations of those metals in aerial matrix than the sensitivities of analysis methods (7, 9-12). In order to overcome those difficulties except using analytical techniques with high sensitivity such as electrothermal atomic absorption spectrometry (ETAAS), and inductively coupled plasma-mass spectrometry (ICP-MS), there are increased attentions to the usage of biomonitoring plants and plant parts such as leaves and shoots as biomonitoring (15-18).

2. Legislation

The emissions of three heavy metals, lead, mercury and cadmium, are being regulated in Europe under the Convention on Long-range Transboundary Air Pollution (19). This convention is the first international, legally binding instrument to deal with problems of air pollution on a broad regional basis. It covers 42 countries in Europe and North America and the European Union. Since entering into force in 1983, the Convention has been extended by several protocols dealing with specific pollutants. The Aarhus Protocol (20), the Protocol on Heavy Metals in June 1998, in Aarhus, Denmark (UN/ECE, 2000), targets three particularly harmful metals: cadmium, lead and mercury, to set a framework for national legislation that will lead to the substantial decrease in the emissions of the three metals in Europe and North America. The protocol seeks to cut emissions of heavy metals from industrial sources, combustion processes and waste incineration. The Protocol will enter into force when ratified by sixteen signatory countries; as of 3 July 2000, only six countries had ratified the Protocol (21).

Air toxics are not regulated under the National Environment Protection Measure (NEPM) for Ambient Air Quality, which addresses criteria pollutants in ambient air (22). However, a program initiated by the Commonwealth Government, the Living Cities-Air Toxics Program (ATP) was aimed at addressing urban air quality issues by supporting the development of national approaches to the management of 'air toxics'. For the purpose of the Living Cities initiative, air toxics are defined as: "…gaseous, aerosol or particulate *pollutants (other than the six criteria pollutants) which are present in the air in low concentrations with characteristics such as toxicity or persistence so as to be a hazard to human, plant or animal life*…" (23). The terms 'air toxics' and 'hazardous air pollutants' (HAPs) are used interchangeably (24). The Technical Advisory Group (TAG) for the ATP included the metals cadmium, chromium (VI), mercury, nickel and their compounds in the list of 28 priority air toxics identified in the ATP. Further, lead is the most routinely monitored heavy metal in ambient air in some countries as a result of its presence in motor vehicle fuel (25).

3. Speciation and toxicity

Compared to gaseous compounds, the assessment of metal and metalloid compounds in inhaled ambient air is complicated by the fact that different species with considerably differing toxicity and/or carcinogenic potency may be encountered. In order to fully evaluate the health effects, it is important to know which species do occur in the environment or at least which compounds form the main constituents. In ambient air, metals, metalloids and their compounds are mainly encountered as part of particulate matter. They may be present in the non soluble, non stoichiometric mixture phase such as

spinels or as soluble ionic compounds such as salts. In respect to their effects on the environment and on human health, gaseous forms such as organometallic compounds can be characterized by other parameters, such as water solubility (extended to solubility in biological fluids), particle size distribution, morphology and specific surface area, and chemical heterogeneity of the particles, or the concentration of metals and metalloids in the particles ultimately contacting target tissues in the human body (26). For example, a metal compound is encapsulated in another aerosol or surface enrichment of volatile species.

All parameters mentioned will influence the bioavailability and possible effects. In addition, metal and metalloid containing substances can undergo various chemical and physical transformations in the atmosphere on their way from the source to a possible receptor. For example, As (III) compounds may be oxidized to As (V). Unfortunately, analytical methods normally only identify the elements which are present in atmospheric particles, species specific analysis being extremely difficult in the concentration range occurring in ambient air (typically several ng/m^3. In addition, the state of oxidation may change during sampling. Consequently, information on the concentration of different species in ambient air is very limited at present. Further, the limited knowledge available on the occurrence of species is outlined.

Trace elements are found naturally in the environment and human exposure derives from a variety of sources, including air, drinking water, and food. Concentrations of trace elements in the air are generally low. Levels of As in the air range from approximately 1 to 2,000 ng/m^3, levels of Cd generally range from 1 to 40 ng/m^3 but can reach up to 100 ng/m^3 near emission sources, and levels of Ni in cities and rural areas range from 7 to 12 ng/m^3 (27-54). Workers in the smelting and refining industries and those employed in the production of batteries, coatings, and plastics can be exposed to much higher levels of airborne Cd and Ni (55).

3.1 Trace metal uses and their health effects

3.1.1 Arsenic

There are three major groups of arsenic (As) compounds: inorganic arsenic compounds, organic arsenic compounds, arsine gas and substituted arsines.

Elemental arsenic is utilized in alloys in order to increase their hardness and heat resistance. It is also used in the manufacture of certain types of glass, as a component of electrical devices and as a doping agent in germanium and silicon solid-state products. The uses and source of arsenic compounds are summarized in Table 1.

Organic arsenic compounds in marine organisms occur in concentrations corresponding to a concentration of arsenic in the range 1 to 100 mg/kg in marine organisms such as shrimp and fish. Such arsenic is mainly made up of arsenobetaine and arsenocholine, organic arsenic compounds of low toxicity (56). The substituted arsines are trivalent organic arsenical compounds which, depending on the number of alkyl or phenyl groups that they have attached to the arsenic nucleus, are known as mono-, di- or tri-substituted arsines. Dichloroethylarsine ($C_2H_5AsCl_2$), or ethyldichloroarsine, is a colourless liquid with an irritant odour. This compound was developed as a potential chemical warfare agent. Dichloro(2-chlorovinyl-) arsine ($ClCH:CHAsCl_2$), or chlorovinyldichloroarsine (lewisite), is

an olive-green liquid with a germanium-like odour. It was developed as a potential warfare agent but never used according to our knowledge. Dimethyl-arsine $(CH_3)_2AsH$, or cacodyl hydride and trimethylarsine $(CH_3)_3As$), or trimethylarsenic, are both colourless liquids. These two compounds can be produced after metabolic transformation of arsenic compounds by bacteria and fungi.

Compound	Uses/Source
Arsenic trichloride ($AsCl_3$)	ceramics industry, manufacturing of chlorine-containing arsenicals
Arsenic trioxide (As_2O_3), or white arsenic	purification of synthesis gas, as a primary material for all arsenic compounds, preservative for hides and wood, a textile mordant, a reagent in mineral flotation, a decolourizing and refining agent in glass manufacture
Calcium arsenite ($Ca(As_2H_2O_4)$, Calcium arsenate ($Ca_3(AsO_4)_2$)	insecticides
Cacodylic acid ($(CH_3)_2AsOOH$)	herbicide and a defoliant
cupric acetoarsenite (usually considered $Cu(COOCH_3)_2$ $3Cu(AsO_2)_2$)	Insecticides, for painting ships and submarines
Sodium arsenite ($NaAsO_2$)	herbicide, a corrosion inhibitor, as a drying agent in the textile industry
Arsenic trisulphide	a component of infrared-transmitting glass, a dehairing agent in the tanning industry, the manufacturing of pyrotechnics and semiconductors
Arsenic acid (H_3AsO_4 ·½H_2O)	the manufacturing of arsenates, glass making, wood-treating processes
Arsenic pentoxide (As_2O_5)	Herbicide, a wood preservative, in the manufacture of coloured glass
Arsanilic acid ($NH_2C_6H_4AsO(OH)_2$)	as a grasshopper bait, as an additive in animal feeds
Arsine gas	in organic syntheses, in the processing of solid-state electronic components, inadvertently in industrial processes when nascent hydrogen is formed and arsenic is present

Table 1. Arsenic compounds and their uses/source.

3.1.1.1 Toxicity

Although it is possible that very small amounts of certain arsenic compounds may have beneficial effects, as indicated by some animal studies, arsenic compounds, particularly the inorganic ones, are otherwise regarded as very potent poisons. Acute toxicity varies widely among compounds, depending on their valency state and solubility in biological media. The soluble trivalent compounds are the most toxic. Uptake of inorganic arsenic compounds from the gastrointestinal tract is almost complete, but uptake may be delayed for less soluble forms such as arsenic trioxide in particle form. Uptake after inhalation is also almost complete, since even less soluble material deposited on the respiratory mucosa, will be transferred to the gastrointestinal tract and subsequently taken up. The health effects of arsenic were summarized in Table 2.

Metal-Route of exposure	Health effects	Diagnosis/medical monitoring
Inorganic and organic As-inhalation, ingestion, skin	- Acute exposure: nausea, diarrhea, Gastro intestinal (GI) bleeding, cardiovascular effects, shock, and death. Liver, kidney damage and seizures have been reported. - Chronic exposure: hyperpigmentation of skin, warts, corns, heart disease, neuropathy, liver damage, peripheral vascular disease (gangrene of lower limbs), and increased risk of skin, liver, lung and bladder cancer. Arsenic in drinking water can also cause diabetes and hypertension. - Anaemia, leucopenia and granulocytopenia - The soluble trivalent compounds are the most toxic - The fatal dose of ingested arsenic trioxide has been reported to range from 70 to 180 mg. Death may occur within 24 hours, but the usual course runs from 3 to 7 days. - Cancer of the respiratory tract has been reported in excess frequency among workers engaged in the production of insecticides containing lead arsenate and calcium arsenate, in vine-growers spraying insecticides containing inorganic copper and arsenic compounds, and in smelter workers exposed to inorganic compounds of arsenic and a number of other metals. The latency time between onset of exposure and the appearance of cancer is long, usually between 15 and 30 years. - Arsine is one of the most powerful haemolytic agents found in industry. Inhalation of 250 ppm of arsine gas is instantly lethal. Exposure from 25 to 50 ppm for 30 minutes is lethal, and 10 ppm may be lethal after longer exposures.	Urinary arsenic level is the most reliable indicator of recent exposure to arsenic. Arsenic in hair and fingernails can indicate exposure to high levels in the past 6–12 months.
Cd-inhalation, ingestion	- Cell proliferation, differentiation, apoptosis, and other cellular activities, numerous molecular lesions caused carcinogenesis. - Cadmium targets the lung, liver, kidney, and testes. - In acute intoxication: nephrotoxicity, immunotoxicity, osteotoxicity, and tumors after prolonged exposures. - Prostate, lung, testicular, renal, and skeletal cancers. - In vivo: Generate $O_2^{\bullet -}$, H_2O_2, and $^{\bullet}OH$ accompanied by activation of redox-sensitive transcription factors. - After inhalation above 1 mg Cd/m^3 in air for 8 hours; chemical pneumonitis, and in severe cases pulmonary oedema.	Cadmium levels in blood are mainly an indication of the last few months exposure, but can be used to assess body burden a few years after exposure has ceased. The individual critical concentrations of

Metal-Route of exposure	Health effects	Diagnosis/medical monitoring
Cd-inhalation, ingestion	- After ingestion of drinks exceeding 15 mg Cd/l; nausea, vomiting, abdominal pains and sometimes diarrhoea. - Prolonged exposure in air at concentrations exceeding 0.1 mg Cd/m^3 ; Pulmonary emphysema - Exposure for more than 20 years to concentrations of about 0.02 mg Cd/m^3 , certain pulmonary effects. - Exceeding 200 µg Cd/g (this is critical concentration) wet weight of renal cortex; tubular dysfunction with decreased reabsorption of proteins from the urine, tubular proteinuria with increased excretion of low-molecular-weight proteins. - The average cadmium concentration in workroom air (8 hours per day) should not exceed 0.01 mg Cd/m^3.	cadmium in urine and/or in blood are 50 nmol/l whole blood or 3 nmol/mmol creatinine.
Lead-Inhalation, ingestion, skin	- Hematologic: decreased heme synthesis enzymes, anemia. - Cardiovascular: elevated blood pressure. - Cognitive, neurobehavioral, and psychological effects. - Gastrointestinal: colic or abdominal cramps. - Peripheral neuropathy; encephalopathy (at high levels). - Reduced fertility. - Immune system: alterations in T cell, reduced IgG serum levels. - Children: lethargy, loss of appetite, anemia, colic, neurological impairment, and impaired metabolism of Vit D. - Exposure in uterus and during childhood can result in impaired neurological development, IQ deficits, and growth retardation.	Lead in whole blood is a reliable test. Erythrocyte protoporphyrin (EP) test can also be used but it is not sensitive to detect high levels of lead in children.
Mercury-Inhalation, ingestion	- All forms of mercury are toxic to the central nervous system (CNS). - Exposure to high levels can damage brain, kidneys, and developing fetus. (methyl mercury is the most toxic form). - Toxicity to brain results in irritability, tremors, visual changes, and memory problems. - Mercury salts can cause abdominal cramps, diarrhea, and kidney damage.	Acute exposure is best measured by mercury in blood and chronic exposure by mercury in urine

Metal-Route of exposure	Health effects	Diagnosis/medical monitoring
Nickel-Inhalation, ingestion	- The National Maximum Workplace Concentration Committee (NMWCC) of the Netherlands proposed that urine nickel concentration 40 µg/g creatinine, or serum nickel concentration 5 µg/l (both measured in samples obtained at the end of a working week or a work shift) be considered warning limits for further investigation of workers exposed to nickel metal or soluble nickel compounds. - Exposures are classified as "mild" if the initial 8-h specimen of urine has a nickel concentration less than 100 µg/l, "moderate" if the nickel concentration is 100 to 500 µg/l, and "severe" if the nickel concentration exceeds 500 µg/l. - Chronic exposure of workers to inhalation of low atmospheric concentrations of nickel carbonyl (0.007 to 0.52 mg/m^3) can cause neurological symptoms such as insomnia, headache, dizziness, memory loss, and other manifestations including chest tightness, excessive sweating, alopecia.	

Table 2. Reported metal toxicity and diagnosis/medical monitoring.

Occupational exposure to inorganic arsenic compounds through inhalation, ingestion or skin contact with subsequent absorption may occur in industry. Acute effects at the point of entry may occur if exposure is excessive. Dermatitis may occur as an acute symptom but is more often the result of toxicity from long-term exposure, sometimes subsequent to sensitization.

In occupational exposure to mainly airborne arsenic, skin lesions may result from local irritation. Two types of dermatological disorders may occur:

1. an eczematous type with erythema (redness), swelling and papules or vesicles
2. a follicular type with erythema and follicular swelling or follicular pustules.

Dermatitis is primarily localized on the most heavily exposed areas, such as the face, back of the neck, forearms, wrists and hands. Patch tests have demonstrated that the dermatitis is due to arsenic, not to impurities present in the crude arsenic trioxide. Chronic dermal lesions may follow this type of initial reaction, depending on the concentration and duration of exposure. These chronic lesions may occur after many years of occupational or environmental exposure. Hyperkeratosis, warts and melanosis of the skin are the conspicuous signs (57).

3.1.1.2 Carcinogenic effects

Inorganic arsenic compounds are classified by the International Agency for Research on Cancer (IARC) as lung and skin carcinogens (58). There is also some evidence to suggest that

persons exposed to inorganic arsenic compounds suffer a higher incidence of angiosarcoma of the liver and possibly of stomach cancer. A synergistic action of tobacco smoking has been demonstrated for lung cancer. Long-term exposure to inorganic arsenic via drinking water has been associated with an increased incidence of skin cancer. This increase has been shown to be related to concentration in drinking water.

3.1.1.3 Organic arsenic compounds

Organic arsenicals used as pesticides or as drugs may also give rise to toxicity, although such adverse effects are incompletely documented in humans. Toxic effects on the nervous system have been reported in experimental animals following feeding with high doses of arsanilic acid, which is commonly used as a feed additive in poultry and swine.

The organic arsenic compounds that occur in foodstuffs of marine origin, such as shrimp, crab and fish, are made up of arsinocholine and arsinobetaine. It is well known that the amounts of organic arsenic that are present in fish and shellfish can be consumed without ill effects because these compounds are quickly excreted, mainly via urine.

Many cases of acute arsine poisoning have been recorded, and there is a high fatality rate. Arsine is one of the most powerful haemolytic agents found in industry. Its haemolytic activity is due to its ability to cause a fall in erythrocyte-reduced glutathione content. Signs and symptoms of arsine poisoning include haemolysis, which develops after a latent period that is dependent on the intensity of exposure. Inhalation of 250 ppm of arsine gas is instantly lethal. Exposure from 25 to 50 ppm for 30 minutes is lethal, and 10 ppm may be lethal after longer exposures. The signs and symptoms of poisoning are those characteristic of an acute and massive haemolysis. After acute and severe exposure, a peripheral neuropathy may develop and can still be present several months after poisoning. Little is known about repeated or chronic exposure to arsine, but since the arsine gas is metabolized to inorganic arsenic in the body, it can be assumed that there is a risk for symptoms similar to those in long-term exposure to inorganic arsenic compounds (59).

3.1.2 Cadmium

Cadmium (Cd) has many chemical and physical similarities to zinc and occurs together with zinc in nature. In minerals and ores, cadmium and zinc generally have a ratio of 1:100 to 1:1,000. Cadmium is highly resistant to corrosion and has been widely used for electroplating of other metals, mainly steel and iron. Screws, screw nuts, locks and various parts for aircraft and motor vehicles are frequently treated with cadmium in order to withstand corrosion. Nowadays, however, only 8% of all refined cadmium is used for platings and coatings. It was established that Cd of used in developed and industrialized countries is about 3% in certain alloys, 8% for platings and coatings, 30% for pigments and stabilizers in plastics, 55% for rechargeable, small portable cadmium-containing batteries used in mobile telephones and similars.

The most important Cd compound is cadmium stearate, which is used as a heat stabilizer in polyvinyl chloride (PVC) plastics. Cadmium sulphide and cadmium sulphoselenide are used as yellow and red pigments in plastics and colours. Cadmium sulphide is also used in photo- and solar cells. Cadmium chloride acts as a fungicide, an ingredient in elecroplating baths, a colourant for pyrotechnics, an additive to tinning solution and a mordant in dyeing and printing textiles. It is also used in the production of certain

photographic films and in the manufacture of special mirrors and coatings for electronic vacuum tubes. Cadmium oxide is an elecroplating agent, a starting material for PVC heat stabilizers and a component of silver alloys, phosphors, semiconductors and glass and ceramic glazes (60).

As a result, cadmium can represent an environmental hazard, and many countries have introduced legislative actions aimed towards decreasing the use and subsequent environmental spread of cadmium.

3.1.2.1 Toxicity

The health effects of cadmium were summarized in Table 2. Metallothioneins play a role in the homeostasis of essential metals such as copper, detoxification of toxic metals such as cadmium, and protection against oxidative stress. Gastrointestinal absorption of ingested cadmium is about 2 to 6% under normal conditions. Individuals with low body iron stores, reflected by low concentrations of serum ferritin, may have considerably higher absorption of cadmium, up to 20% of a given dose of cadmium. Significant amounts of cadmium may also be absorbed via the lung from the inhalation of tobacco smoke or from occupational exposure to atmospheric cadmium dust. Pulmonary absorption of inhaled respirable cadmium dust is estimated at 20 to 50%. After absorption via the gastrointestinal tract or the lung, cadmium is transported to the liver, where production of a cadmium-binding low-molecular-weight protein, metallothionein, is initiated (61).

About 80 to 90% of the total amount of cadmium in the body is considered to be bound to metallothionein. This prevents the free cadmium ions from exerting their toxic effects. It is likely that small amounts of metallothionein-bound cadmium are constantly leaving the liver and being transported to the kidney via the blood. The metallothionein with the cadmium bound to it is filtered through the glomeruli into the primary urine. Like other low-molecular-weight proteins and amino acids, the metallothionein-cadmium complex is subsequently reabsorbed from the primary urine into the proximal tubular cells, where digestive enzymes degrade the engulfed proteins into smaller peptides and amino acids. Free cadmium ions in the cells result from degradation of metallothionein and initiate a new synthesis of metallothionein, binding the cadmium, and thus protecting the cell from the highly toxic free cadmium ions. Kidney dysfunction is considered to occur when the metallothionein-producing capacity of the tubular cells is exceeded. The kidney and liver have the highest concentrations of cadmium, together containing about 50% of the body burden of cadmium. The cadmium concentration in the kidney cortex, before cadmium-induced kidney damage occurs, is generally about 15 times the concentration in liver. Elimination of cadmium is very slow. As a result of this, cadmium accumulates in the body, the concentrations increasing with age and length of exposure (62). Based on organ concentration at different ages the biological half-life of cadmium in humans has been estimated in the range of 7 to 30 years.

3.1.2.2 Acute toxicity

Inhalation of cadmium compounds at concentrations above 1 mg Cd/m^3 in air for 8 hours, or at higher concentrations for shorter periods, may lead to chemical pneumonitis, and in severe cases pulmonary oedema. Symptoms generally occur within 1 to 8 hours after exposure. They are influenza-like and similar to those in metal fume fever. The more severe symptoms of chemical pneumonitis and pulmonary oedema may have a latency period up

to 24 hours. Death may occur after 4 to 7 days. Exposure to cadmium in the air at concentrations exceeding 5 mg Cd/m3 is most likely to occur where cadmium alloys are smelted, welded or soldered. Ingestion of drinks contaminated with cadmium at concentrations exceeding 15 mg Cd/l gives rise to symptoms of food poisoning. Symptoms are nausea, vomiting, abdominal pains and sometimes diarrhoea. Sources of food contamination may be pots and pans with cadmium-containing glazing and cadmium solderings used in vending machines for hot and cold drinks. In animals parenteral administration of cadmium at doses exceeding 2 mg Cd/kg body weight causes necrosis of the testis. No such effect has been reported in humans.

3.1.2.3 Chronic toxicity

Chronic cadmium poisoning has been reported after prolonged occupational exposure to cadmium oxide fumes, cadmium oxide dust and cadmium stearates. Changes associated with chronic cadmium poisoning may be local, in which case they involve the respiratory tract, or they may be systemic, resulting from absorption of cadmium. Systemic changes include kidney damage with proteinuria and anemia. Lung disease in the form of emphysema is the main symptom at heavy exposure to cadmium in air, whereas kidney dysfunction and damage are the most prominent findings after long-term exposure to lower levels of cadmium in workroom air or via cadmium-contaminated food. Mild hypochromic anemia is frequently found among workers exposed to high levels of cadmium. This may be due to both increased destruction of red blood cells and to iron deficiency. Yellow discolouration of the necks of teeth and loss of sense of smell (anosmia) may also be seen in cases of exposure to very high cadmium concentrations.

Pulmonary emphysema is considered a possible effect of prolonged exposure to cadmium in air at concentrations exceeding 0.1 mg Cd/m^3. It has been reported that exposure to concentrations of about 0.02 mg Cd/m^3 for more than 20 years can cause certain pulmonary effects. Cadmium-induced pulmonary emphysema can reduce working capacity and may be the cause of invalidity and life shortening. With long-term low-level cadmium exposure the kidney is the critical organ (i.e., the organ first affected). Cadmium accumulates in renal cortex. Concentrations exceeding 200 μg Cd/g wet weight have previously been estimated to cause tubular dysfunction with decreased reabsorption of proteins from the urine (63). This causes tubular proteinuria with increased excretion of low-molecular-weight proteins such as α,α-1-microglobulin (protein HC), β-2-microglobulin and retinol binding protein (RTB). Recent research suggests, however, that tubular damage may occur at lower levels of cadmium in kidney cortex. As the kidney dysfunction progresses, amino acids, glucose and minerals, such as calcium and phosphorus, are also lost into the urine. Increased excretion of calcium and phosphorous may disturb bone metabolism, and kidney stones are frequently reported by cadmium workers. After long-term medium-to-high levels of exposure to cadmium, the kidney's glomeruli may also be affected, leading to a decreased glomerular filtration rate. In severe cases uraemia may develop. Excessive cadmium exposure has occurred in the general population through ingestion of contaminated rice and other foodstuffs, and possibly drinking water. The itai-itai disease, a painful type of osteomalacia, with multiple fractures appearing together with kidney dysfunction, has occurred in Japan in areas with high cadmium exposure. Though the pathogenesis of itai-itai disease is still under dispute, it is generally accepted that cadmium is a necessary aetiological factor. It

should be stressed that cadmium-induced kidney damage is irreversible and may grow worse even after exposure has ceased.

3.1.2.4 Carcinogenic effects

Cd competes with Zn for binding sites and can therefore interfere with some of Zinc's essential functions. Thus, it may inhibit enzyme reactions and utilization of nutrients. Cd can generate free radical tissue damage because it may be a catalyst to oxidation reactions. Furthermore, excessive Cd exposure can cause renal damage, reproduction problems, cardiovascular diseases and hypertension. There are several sources of human exposure to Cd, including employment in primary metal industries, production of certain batteries, some electroplating processes and consumption of tobacco products. Consequently, it was reported by International Agency Research on Cancer (IARC) that through inhalation cadmium could cause lung cancer in humans and animals (64). As a result, the World Health Organization (WHO) (65) established provisional tolerable weekly intakes (PTWIs) of Cd of 0.007 microgram/kg body weight, for all human groups.

There is strong evidence of dose-response relationships and an increased mortality from lung cancer in several epidemiological studies on cadmium-exposed workers. The interpretation is complicated by concurrent exposures to other metals which are known or suspected carcinogens. Continuing observations of cadmium-exposed workers have, however, failed to yield evidence of increased mortality from prostatic cancer, as initially suspected. The IARC in 1993 (64) assessed the risk of cancer from exposure to cadmium and concluded that it should be regarded as a human carcinogen. Since then additional epidemiological evidence has come forth with somewhat contradictory results, and the possible carcinogenicity of cadmium thus remains unclear. It is nevertheless clear that cadmium possesses strong carcinogenic properties in animal experiments.

3.1.2.5 Limitations

The kidney cortex is the critical organ with long-term cadmium exposure via air or food. The critical concentration is estimated at about 200 μg Cd/g wet weight, but may be lower, as stated above. In order to keep the kidney cortex concentration below this level even after lifelong exposure, the average cadmium concentration in workroom air (8 hours per day) should not exceed 0.01 mg Cd/m^3.

To ensure that excessive accumulation of cadmium in the kidney does not occur, cadmium levels in blood and in urine should be checked regularly. Cadmium levels in blood are mainly an indication of the last few months exposure, but can be used to assess body burden a few years after exposure has ceased. A value of 100 nmol Cd/l whole blood is an approximate critical level if exposure is regular for long periods. Cadmium values in urine can be used to estimate the cadmium body burden, providing kidney damage has not occurred. It has been estimated by the WHO that 10 nmol/mmol creatinine is the concentration below which kidney dysfunction should not occur. Recent research has, however, shown that kidney dysfunction may occur already at around 5 nmol/mmol creatinine. Since the mentioned blood and urinary levels are at levels at which action of cadmium on kidney has been observed, it is recommended that control measures should be applied whenever the individual concentrations of cadmium in urine and/or in blood exceed 50 nmol/l whole blood or 3 nmol/mmol creatinine respectively.

3.1.3 Chromium

In addition to chromic acid, the ferrous chromite ($FeOCr_2O_3$) ore contains variable quantities of other substances. Only ores or concentrates containing more than 40% chromic oxide (Cr_2O_3) are used commercially, and countries having the most suitable deposits are the Russian Federation, South Africa, Zimbabwe, Turkey, the Philippines and India. The prime consumers of chromites are the United States, the Russian Federation, Germany, Japan, France and the United Kingdom.

The most significant usage of pure chromium is for electroplating of a wide range of equipment, such as automobile parts and electric equipment. Chromium is used extensively for alloying with iron and nickel to form stainless steel, and with nickel, titanium, niobium, cobalt, copper and other metals to form special-purpose alloys.

Chromium forms a number of compounds in various oxidation states. Those of II (chromous), III (chromic) and VI (chromate) states are most important; the II state is basic, the III state is amphoteric and the VI state is acidic. Commercial applications mainly concern compounds in the VI state, with some interest in III state chromium compounds.

The chromous state (Cr^{II}) is unstable and is readily oxidized to the chromic state (Cr^{III}). This instability limits the use of chromous compounds. The most important compounds containing chromium in the Cr^{VI} state are dichromate compounds and chromium trioxide.

Compounds containing Cr^{VI} are used in many industrial operations: the manufacture of important inorganic pigments such as lead chromes, molybdate-oranges, zinc chromate and chromium-oxide green; wood preservation; corrosion inhibition; and coloured glasses and glazes. Basic chromic sulphates are widely used for tanning. The dyeing of textiles, the preparation of many important catalysts containing chromic oxide and the production of light-sensitive dichromated colloids for use in lithography are also well-known industrial uses of chromium-containing chemicals.

Chromic acid is used not only for "decorative" chromium plating but also for "hard" chromium plating, where it is deposited in much thicker layers to give an extremely hard surface with a low coefficient of friction.

Because of the strong oxidizing action of chromates in acid solution, there are many industrial applications particularly involving organic materials, such as the oxidation of trinitrotoluene (TNT) to give phloroglucinol and the oxidation of picoline to give nicotine acid (66).

3.1.3.1 Toxicity

Compounds with Cr^{III} oxidation states are considerably less hazardous than are Cr^{VI} compounds. Compounds of Cr^{III} are poorly absorbed from the digestive system. These Cr^{III} compounds may also combine with proteins in the superficial layers of the skin to form stable complexes. Compounds of Cr^{III} do not cause chrome ulcerations and do not generally initiate allergic dermatitis without prior sensitization by Cr^{VI} compounds.

In the Cr^{VI} oxidation state, chromium compounds are readily absorbed after ingestion as well as during inhalation. The uptake through intact skin is less well elucidated. The irritant and

corrosive effects caused by Cr^{VI} occur readily after uptake through mucous membranes, where they are readily absorbed. Work-related exposure to Cr^{VI} compounds may induce skin and mucous membrane irritation or corrosion, allergic skin reactions or skin ulcerations.

The untoward effects of chromium compounds generally occur among workers in workplaces where Cr^{VI} is encountered, in particular during manufacture or use (67). The effects frequently involve the skin or respiratory system. Typical industrial hazards are inhalation of the dust or fumes arising during the manufacture of dichromate from chromite ore and the manufacture of lead and zinc chromates, inhalation of chromic acid mists during electroplating or surface treatment of metals, and skin contact with Cr^{VI} compounds in manufacture or use. Exposure to Cr^{VI}-containing fumes may also occur during welding of stainless steels.

Numerous sources of exposure to Cr^{VI} can be listed as contact with cement, plaster, leather, graphic work, work in match factories, work in tanneries and various sources of metal work. Workers employed in wet sandpapering of car bodies have also been reported with allergy. Affected subjects react positively to patch testing with 0.5% dichromate.

It has been shown that Cr^{VI} penetrates the skin through the sweat glands and is reduced to Cr^{III} in the corium. It is shown that the Cr^{III} then reacts with protein to form the antigen-antibody complex. This explains the localization of lesions around sweat glands and why very small amounts of dichromate can cause sensitization. The chronic character of the dermatitis may be due to the fact that the antigen-antibody complex is removed more slowly than would be the case if the reaction occurred in the epidermis.

Inhalation of dust or mist containing Cr^{VI} is irritating to mucous membranes. At high concentrations of such dust, sneezing, rhinorrhoea, lesions of the nasal septum and redness of the throat are documented effects. Sensitization has also been reported, resulting in typical asthmatic attacks, which may recur on subsequent exposure. At exposure for several days to chromic acid mist at concentrations of about 20 to 30 mg/m^3, cough, headache, dyspnoea and substernal pain have also been reported after exposure. The occurrence of bronchospasm in a person working with chromates should suggest chemical irritation of the lungs.

In previous years, when the exposure levels to Cr^{VI} compounds could be high, ulcerations of the nasal septum were frequently seen among exposed workers. This untoward effect results from deposition of Cr^{VI}-containing particulates or mist droplets on the nasal septum, resulting in ulceration of the cartilaginous portion followed, in many cases, by perforation at the site of ulceration. Frequent nose-picking may enhance the formation of perforation.

Necrosis of the kidneys has also been reported, starting with tubular necrosis, leaving the glomeruli undamaged. Diffuse necrosis of the liver and subsequent loss of architecture has also been reported. Soon after the turn of the century there were a number of reports on human ingestion of Cr^{VI} compounds resulting in major gastro-intestinal bleeding from ulcerations of the intestinal mucosa. Sometimes such bleedings resulted in cardiovascular shock as a possible complication. If the patient survived, tubular necrosis of the kidneys or liver necrosis could occur.

Increased incidence of lung cancer among workers in manufacture and use of Cr^{VI} compounds has been reported in a great number of studies from France, Germany, Italy,

Japan, Norway, the United States and the United Kingdom (68). Chromates of zinc and calcium appear to be among the most potent carcinogenic chromates, as well as among the most potent human carcinogens. Elevated incidence of lung cancer has also been reported among subjects exposed to lead chromates, and to fumes of chromium trioxides. Heavy exposures to Cr^{VI} compounds have resulted in very high incidence of lung cancer in exposed workers 15 or more years after first exposure, as reported in both cohort studies and case reports.

Thus, it is well established that an increase in the incidence of lung cancer of workers employed in the manufacture of zinc chromate and the manufacture of mono- and dichromates from chromite ore is a long-term effect of work-related heavy exposure to Cr^{VI} compounds. Some of the cohort studies have reported measurements of exposure levels among the exposed cohorts. Also, a small number of studies have indicated that exposure to fumes generated from welding on Cr-alloyed steel may result in elevated incidence of lung cancer among these welders.

There is no firmly established "safe" level of exposure. However, most of the reports on association between Cr^{VI} exposure and cancer of the respiratory organs and exposure levels report on air levels exceeding 50 mg Cr^{VI}/m^3 air (69).

Water-soluble, acid soluble and water insoluble chromium is found in the lung tissues of chromate workers in varying amounts.

Although it has not been firmly established, some studies have indicated that exposure to chromates may result in increased risk of cancer in the nasal sinuses and the alimentary tract. The studies that indicate excess cancer of the alimentary tract are case reports from the 1930s or cohort studies that reflect exposure at high levels than generally encountered today.

3.1.4 Iron

In addition to ferroalloys, the most important industrial iron compounds are the oxides and the carbonate, which constitute the principal ores from which the metal is obtained. Of lesser industrial importance are cyanides, nitrides, nitrates, phosphides, phosphates and iron carbonyl.

3.1.4.1 Toxicity

Industrial dangers are present during the mining, transportation and preparation of the ores, during the production and use of the metal and alloys in iron and steel works and in foundries, and during the manufacture and use of certain compounds. Inhalation of iron dust or fumes occurs in iron-ore mining; arc welding; metal grinding, polishing and working; and in boiler scaling. If inhaled, iron is a local irritant to the lung and gastrointestinal tract. Reports indicate that long-term exposure to a mixture of iron and other metallic dusts may impair pulmonary function. Inhaling dust containing silica or iron oxide can lead to pneumoconiosis, but there are no definite conclusions as to the role of iron oxide particles in the development of lung cancer in humans. Based on animal experiments, it is suspected that iron oxide dust may serve as a "co-carcinogenic" substance, thus enhancing the development of cancer when combined simultaneously with exposure to carcinogenic substances (70).

Mortality studies of haematite (Fe_2O_3) miners (containing up to 66% iron) have shown an increased risk of lung cancer, generally among smokers, in several mining areas such as Cumberland, Lorraine, Kiruna and Krivoi Rog. In experimental studies, ferric oxide has not been found to be carcinogenic; however, the experiments were not carried out with haematite (71). The presence of radon in the atmosphere of haematite mines has been suggested to be an important carcinogenic factor. Epidemiological studies of iron and steel foundry workers have typically noted risks of lung cancer elevated by 1.5- to 2.5-fold. The International Agency for Research on Cancer (IARC) classifies iron and steel founding as a carcinogenic process for humans. The specific chemical agents involved (e.g., polynuclear aromatic hydrocarbons, silica, metal fumes) have not been identified. An increased incidence of lung cancer has also been reported, but less significantly, among metal grinders. The conclusions for lung cancer among welders are controversial. The dangerous properties of the remaining iron compounds are usually due to the radical with which the iron is associated. Thus ferric arsenate ($FeAsO_4$) and ferric arsenite ($FeAsO_3 \cdot Fe_2O_3$) possess the poisonous properties of arsenical compounds. Iron carbonyl ($FeCO_5$) is one of the more dangerous of the metal carbonyls, having both toxic and flammable properties (71).

Ferrosilicon production can result in both aerosols and dusts of ferrosilicon. Animal studies indicate that ferrosilicon dust can cause thickening of the alveolar walls with the occasional disappearance of the alveolar structure. The raw materials used in alloy production may also contain free silica, although in relatively low concentrations. There is some disagreement as to whether classical silicosis may be a potential hazard in ferrosilicon production. There is no doubt, however, that chronic pulmonary disease, whatever its classification, can result from excessive exposure to the dust or aerosols encountered in ferrosilicon plants.

3.1.5 Lead

About 40% of lead is used as a metal, 25% in alloys and 35% in chemical compounds. Because populations in, at least, 100 countries are still exposed to air pollution with lead in spite of banning the usage of lead in gasoline in many countries, usage of lead and lead compounds will be detailed. Due to its malleability, low melting point, and ability to form compounds, Pb has been used in hundreds of products such as pipes, solder, brass fixtures, crystal, paint, cable, ceramics, and batteries (72). Metallic lead is used in the form of sheeting or pipes where pliability and resistance to corrosion are required, such as in chemical plants and the building industry; it is used also for cable sheathing, as an ingredient in solder and as a filler in the automobile industry. It is a valuable shielding material for ionizing radiations. It is used for metallizing to provide protective coatings, in the manufacture of storage batteries and as a heat treatment bath in wire drawing. Lead is present in a variety of alloys and its compounds are prepared and used in large quantities in many industries. Lead oxides are used in the plates of electric batteries and accumulators (PbO and Pb_3O_4), as compounding agents in rubber manufacture (PbO), as paint ingredients (Pb_3O_4) and as constituents of glazes, enamels and glass.

Lead salts form the basis of many paints and pigments; lead carbonate and lead sulphate are used as white pigments and the lead chromates provide chrome yellow, chrome orange, chrome red and chrome green. Lead arsenate is an insecticide, lead sulphate is used in

rubber compounding, lead acetate has important uses in the chemical industry, lead naphthenate is an extensively used dryer and tetraethyllead is an antiknock additive for gasoline, where still permitted by law.

Other metals such as antimony, arsenic, tin and bismuth may be added to lead to improve its mechanical or chemical properties, and lead itself may be added to alloys such as brass, bronze and steel to obtain certain desirable characteristics. The very large numbers of organic and inorganic lead compounds are encountered in industry.

3.1.5.1 Toxicity

The prime hazard of lead is its toxicity. For a long time, it is known that lead is toxic for brain, kidney and reproductive system and can also cause impairment in intellectual functioning, infertility, miscarriage and hypertension. Several studies have shown that lead exposures in school-aged children can significantly reduce IQ and has been associated with aggressive behavior, delinquency and attention disorders (73). The health effects of lead were summarized in Table 2.

Clinical lead poisoning has always been one of the most important occupational diseases. Industrial consumption of lead is increasing and traditional consumers are being supplemented by new users such as the plastics industry. Hazardous exposure to lead, therefore, occurs in many occupations. In lead mining, a considerable proportion of lead absorption occurs through the alimentary tract and consequently the extent of the hazard in this industry depends, to some extent, on the solubility of ores being worked. The lead sulphide (PbS) in galena is insoluble and absorption from the lung is limited; however, in the stomach, some lead sulphide may be converted to slightly soluble lead chloride which may then be absorbed in moderate quantities. In lead smelting, the main hazards are the lead dust produced during crushing and dry grinding operations, and lead fumes and lead oxide encountered in sintering, blast-furnace reduction and refining.

Lead sheet and pipe are used principally for the construction of equipment for storing and handling sulphuric acid. The use of lead for water and town gas pipes is limited nowadays. The hazards of working with lead increase with temperature. If lead is worked at temperatures below 500 °C, as in soldering, the risk of fume exposure is far less than in lead welding, where higher flame temperatures are used and the danger is higher. The spray coating of metals with molten lead is dangerous since it gives rise to dust and fumes at high temperatures (74).

The demolition of steel structures such as bridges and ships that have been painted with lead-based paints frequently gives rise to cases of lead poisoning. When metallic lead is heated to 550 °C, lead vapour will be evolved and will become oxidized. This is a condition that is liable to be present in metal refining, the melting of bronze and brass, the spraying of metallic lead, lead burning, chemical plant plumbing, ship breaking and the burning, cutting and welding of steel structures coated with paints containing lead tetroxide.

3.1.5.2 Absorption

The degree of absorption depends on the proportion of the dust accounted for by particles less than 5 microns in size and the exposed worker's respiratory minute volume. Since the most important route of lead absorption is by the lungs, the particle size of industrial lead

dust is of considerable significance and this depends on the nature of the operation giving rise to the dust. Fine dust of respirable particle size is produced by processes such as the pulverizing and blending of lead colours, the abrasive working of lead-based fillers in automobile bodies and the dry rubbing-down of lead paint. The exhaust gases of gasoline engines yield lead chloride and lead bromide particles of 1 micron diameter. The larger particles, however, may be ingested and be absorbed via the stomach. A more informative picture of the hazard associated with a sample of lead dust might be given by including a size distribution as well as a total lead determination.

In the human body, inorganic lead is not metabolized but is directly absorbed, distributed and excreted. The rate at which lead is absorbed depends on its chemical and physical form and on the physiological characteristics of the exposed person such as nutritional status and age. Inhaled lead deposited in the lower respiratory tract is completely absorbed. The amount of lead absorbed from the gastrointestinal tract of adults is typically 10 to 15% of the ingested quantity; for pregnant women and children, the amount absorbed can increase to as much as 50%. The quantity absorbed increases significantly under fasting conditions and with iron or calcium deficiency (75).

Once in the blood, lead is distributed primarily among three compartments—blood, soft tissue (kidney, bone marrow, liver, and brain), and mineralizing tissue (bones and teeth). Mineralizing tissue contains about 95% of the total body burden of lead in adults.

The lead in mineralizing tissues accumulates in subcompartments that differ in the rate at which lead is resorbed. In bone, there is both a labile component, which readily exchanges lead with the blood, and an inert pool. The lead in the inert pool poses a special risk because it is a potential endogenous source of lead. When the body is under physiological stress such as pregnancy, lactation or chronic disease, this normally inert lead can be mobilized, increasing the lead level in blood. Because of these mobile lead stores, significant drops in a person's blood lead level can take several months or sometimes years, even after complete removal from the source of lead exposure.

Of the lead in the blood, 99% is associated with erythrocytes; the remaining 1% is in the plasma, where it is available for transport to the tissues. The blood lead not retained is either excreted by the kidneys or through biliary clearance into the gastrointestinal tract. In single-exposure studies with adults, lead has a half-life, in blood, of approximately 25 days; in soft tissue, about 40 days; and in the non-labile portion of bone, more than 25 years. Consequently, after a single exposure a person's blood lead level may begin to return to normal; the total body burden, however, may still be elevated (76).

For lead poisoning to develop, major acute exposures to lead need not occur. The body accumulates this metal over a lifetime and releases it slowly, so even small doses, over time, can cause lead poisoning. It is the total body burden of lead that is related to the risk of adverse effects.

3.1.5.3 Physiological effects

Whether lead enters the body through inhalation or ingestion, the biologic effects are the same; there is interference with normal cell function and with a number of physiological processes.

Neurological effects: The most sensitive target of lead poisoning is the nervous system. In children, neurological deficits have been documented at exposure levels once thought to cause no harmful effects. In addition to the lack of a precise threshold, childhood lead toxicity may have permanent effects. Some studies showed that damage to the central nervous system (CNS) that occurred as a result of lead exposure at age 2 resulted in continued deficits in neurological development, such as lower IQ scores and cognitive deficits, at age 5 (77).

Adults also experience CNS effects at relatively low blood lead levels, manifested by subtle behavioural changes, fatigue and impaired concentration. Peripheral nervous system damage, primarily motor, is seen mainly in adults. Lead neuropathy is believed to be a motor neuron, anterior horn cell disease with peripheral dying-back of the axons. Frank wrist drop occurs only as a late sign of lead intoxication.

Lead inhibits the body's ability to make hemoglobin by interfering with several enzymatic steps in the heme pathway. A decrease in the activity of ferrochelatase enzyme results in an increase of the substrate, erythrocyte protoporphyrin (EP), in the red blood cells. Recent data indicate that the EP level, which has been used to screen for lead toxicity in the past, is not sufficiently sensitive at lower levels of blood lead and is therefore not as useful a screening test for lead poisoning as previously thought.

Lead can induce two types of anemia. Acute high-level lead poisoning has been associated with hemolytic anemia. In chronic lead poisoning, lead induces anemia by both interfering with erythropoiesis and by diminishing red blood cell survival. It should be emphasized, however, that anemia is not an early manifestation of lead poisoning and is evident only when the blood lead level is significantly elevated for prolonged periods.

A strong inverse correlation exists between blood lead levels and levels of vitamin D. Because the vitamin D-endocrine system is responsible in large part for the maintenance of extra- and intra-cellular calcium homeostasis, it is likely that lead impairs cell growth and maturation and tooth and bone development.

A direct effect on the kidney of long-term lead exposure is nephropathy. There is also evidence of an association between lead exposure and hypertension, an effect that may be mediated through renal mechanisms. Maternal lead stores readily cross the placenta, placing the foetus at risk. Increasing evidence indicates that lead not only affects the viability of the foetus, but development as well. Developmental consequences of prenatal exposure to low levels of lead include reduced birth weight and premature birth. Lead is an animal teratogen; however, most studies in humans have failed to show a relationship between lead levels and congenital malformations.

Inorganic lead and inorganic lead compounds have been classified as Group 2B, possible human carcinogens, by the International Agency for Research on Cancer (IARC) (78).

3.1.5.4 Organic lead intoxication

The absorption of a sufficient quantity of tetraethyllead, whether briefly at a high rate or for prolonged periods at a lower rate, induces acute intoxication of the CNS. The milder manifestations are those of insomnia, lassitude and nervous excitation which reveal itself

in lurid dreams and dream-like waking states of anxiety, in association with tremor, hyper-reflexia, spasmodic muscular contractions, bradycardia, vascular hypotension and hypothermia. The more severe responses include recurrent (sometimes nearly continuous) episodes of complete disorientation with hallucinations, facial contortions and intense general somatic muscular activity with resistance to physical restraint. Such episodes may be converted abruptly into maniacal or violent convulsive seizures which may terminate in coma and death.

3.1.5.5 Legislation

Clinical lead poisoning has historically been one of the most important occupational diseases, and it remains a major risk today. The considerable body of scientific knowledge concerning the toxic effects of lead has been enriched since the 1980s by significant new knowledge regarding the more subtle subclinical effects. Similarly, in a number of countries it was felt necessary to redraft or modernize work protective measures enacted over the last half-century and more (25).

Some regulation, such as the Occupational Safety and Health Administration (OSHA) lead standard, specifies the permissible exposure limit (PEL) of lead in the workplace, the frequency and extent of medical monitoring, and other responsibilities of the employer. As of this writing, if blood monitoring reveals a blood lead level greater than 40 μg/dL, the worker must be notified in writing and provided with medical examination. If a worker's blood lead level reaches 60 μg/dL (or averages 50 μg/dL or more), the employer is obligated to remove the employee from excessive exposure, with maintenance of seniority and pay, until the employee's blood lead level falls below 40 μg/dL (79).

3.1.6 Nickel

Since nickel, copper and iron occur as distinct minerals in the sulphide ores, mechanical methods of concentration, such as flotation and magnetic separation, are applied after the ore has been crushed and ground. The nickel concentrate is converted to nickel sulphide matte by roasting or sintering. The matte is refined by electrowinning or by the Mind process. In the Mind process, the matte is ground, calcined and treated with carbon monoxide at 50 °C to form gaseous nickel carbonyl ($Ni(CO)_4$), which is then decomposed at 200 to 250 °C to deposit pure nickel powder. Worldwide production of nickel is approximately 1.2 million ton/year (80).

More than 3,000 nickel alloys and compounds are commercially produced. Stainless steel and other Ni-Cr-Fe alloys are widely used for corrosion-resistant equipment, architectural applications and cooking utensils. Monel metal and other Ni-Cu alloys are used in coinage, food-processing machinery and dairy equipment. Ni-Al alloys are used for magnets and catalyst production. Ni-Cr alloys are used for heating elements, gas turbines and jet engines. Alloys of nickel with precious metals are used in jewellery. Nickel metal, its compounds and alloys have many other uses, including electroplating, magnetic tapes and computer components, arc-welding rods, surgical and dental prostheses, nickel-cadmium batteries, paint pigments (e.g., yellow nickel titanate), moulds for ceramic and glass containers, and catalysts for hydrogenation reactions, organic syntheses and the final methanation step of coal gasification. Occupational exposures to nickel also occur in

recycling operations, since nickel-bearing materials, especially from the steel industry, are commonly melted, refined and used to prepare alloys similar in composition to those that entered the recycling process.

3.1.6.1 Toxicity

Human health hazards from occupational exposures to nickel compounds generally fall into three major categories: allergy, rhinitis, sinusitis and respiratory diseases, and cancers of the nasal cavities, lungs and other organs. The health effects of nickel were summarized in Table 2.

Nickel and nickel compounds are among the most common causes of allergic contact dermatitis. This problem is not limited to persons with occupational exposure to nickel compounds; dermal sensitization occurs in the general population from exposures to nickel-containing coins, jewellery, watch cases and clothing fasteners. In nickel-exposed persons, nickel dermatitis usually begins as a papular erythema of the hands. The skin gradually becomes eczematous, and, in the chronic stage, lichenification frequently develops. Nickel sensitization sometimes causes conjunctivitis, eosinophilic pneumonitis, and local or systemic reactions to nickel-containing implants (e.g., intraosseous pins, dental inlays, cardiac valve prostheses and pacemaker wires). Ingestion of nickel-contaminated tap water or nickel-rich foods can exacerbate hand eczema in nickel-sensitive persons.

Workers in nickel refineries and nickel electroplating shops, who are heavily exposed to inhalation of nickel dusts or aerosols of soluble nickel compounds, may develop chronic diseases of the upper respiratory tract. Chronic diseases of the lower respiratory tract including bronchitis, pulmonary fibrosis have also been reported, but such conditions are infrequent (81).

Epidemiological studies of nickel-refinery workers in Canada, Wales, Germany, Norway and Russia have documented increased mortality rates from cancers of the lung and nasal cavities. Certain groups of nickel-refinery workers have also been reported to have increased incidences of other malignant tumours, including carcinomas of the larynx, kidney, prostate or stomach, and sarcomas of soft tissues, but the statistical significance of these observations is questionable. The increased risks of cancers of the lungs and nasal cavities have occurred primarily among workers in refinery operations that entail high nickel exposures, including roasting, smelting and electrolysis. Although these cancer risks have generally been associated with exposures to insoluble nickel compounds, such as nickel subsulphide and nickel oxide, exposures to soluble nickel compounds have been implicated in electrolysis workers.

Epidemiological studies of cancer risks among workers in nickel-using industries have generally been negative, but recent evidence suggests slightly increased lung cancer risks among welders, grinders, electroplaters and battery makers. Such workers are often exposed to dusts and fumes that contain mixtures of carcinogenic metals (e.g., nickel and chromium, or nickel and cadmium). Based on an evaluation of epidemiological studies, the International Agency for Research on Cancer (IARC) concluded in 1990: "There is sufficient evidence in humans for the carcinogenicity of nickel sulphate and of the combinations of nickel sulphides and oxides encountered in the nickel refining industry. There is inadequate evidence in humans for the carcinogenicity of nickel and nickel alloys". Nickel compounds

have been classified as carcinogenic to humans (Group 1), and metallic nickel as possibly carcinogenic to humans (Group 2B) (82).

3.1.6.2 Biological monitoring

Analyses of nickel concentrations in urine and serum samples may reflect the recent exposures of workers to metallic nickel and soluble nickel compounds, but these assays do not furnish reliable measures of the total body nickel burden. The uses and limitations of biological monitoring of nickel-exposed workers have been summarized. A technical report on analysis of nickel in body fluids was issued in 1994 by the Commission on Toxicology of the International Union of Pure and Applied Chemistry (IUPAC) (83). The National Maximum Workplace Concentration Committee (NMWCC) of the Netherlands proposed that urine nickel concentration 40 μg/g creatinine, or serum nickel concentration 5 μg/l (both measured in samples obtained at the end of a working week or a work shift) be considered warning limits for further investigation of workers exposed to nickel metal or soluble nickel compounds. If a biological monitoring programme is implemented, it should augment an environmental monitoring programme, so that biological data are not used as a surrogate for exposure estimates (83).

3.1.7 Mercury

3.1.7.1 Inorganic mercury

Mercury combines readily with sulphur and halogens at ordinary temperatures and forms amalgams with all metals except iron, nickel, cadmium, aluminium, cobalt and platinum. It reacts exothermically with alkaline metals, is attacked by nitric acid but not by hydrochloric acid and, when hot, will combine with sulphuric acid. Inorganic mercury is found in nature in the form of the sulphide (HgS) as cinnabar ore, which has an average mercury content of 0.1 to 4%. Mercury ore is extracted by underground mining, and mercury metal is separated from the ore by roasting in a rotary kiln or shaft furnace, or by reduction with iron or calcium oxide. The vapour is carried off in the combustion gases and is condensed in vertical tubes.

The most important uses of metallic mercury and its inorganic compounds have included the treatment of gold and silver ores; the manufacture of amalgams; the manufacture and repair of measurement or laboratory apparatus; the manufacture of incandescent electric bulbs, mercury vapour tubes, radio valves, x-ray tubes, switches, batteries, rectifiers, etc.; as a catalyst for the production of chlorine and alkali and the production of acetic acid and acetaldehyde from acetylene; chemical, physical and biological laboratory research; gold, silver, bronze and tin plating; tanning and currying; feltmaking; taxidermy; textile manufacture; photography and photogravure; mercury-based paints and pigments; and the manufacture of artificial silk (84). Some of these uses have been discontinued because of the toxic effects that the mercury exposure exerted upon workers.

3.1.7.2 Organic mercury compounds

Organic compounds of mercury may be considered as the organic compounds in which the mercury is chemically linked directly to a carbon atom. Carbon-mercury bonds have a wide range of stability; in general, the carbon-to-mercury bond in aliphatic compounds is more

stable than that in aromatic compounds. It was estimated that more than 400 phenyl mercurials and at least that number of alkyl mercury compounds have been synthesized. The three most important groups in common usage are the alkyls, the aromatic hydrocarbons or aryls and the alkoxyalkyls. Examples of aryl mercury compounds are phenylmercuric acetate (PMA), nitrate, oleate, propionate and benzoate. Most available information is about PMA.

In medical practice, organic mercury compounds are used as antiseptics, germicides, diuretics and contraceptives. In the field of pesticides, they serve as algicides, fungicides, herbicides and slimacides, and as preservatives in paints, waxes and pastes; they are used for mildew suppression, in antifouling paints, in latex paints and in the fungus-proofing of fabrics, paper, cork, rubber and wood for use in humid climates. In the chemical industry, they act as catalysts in a number of reactions and the mercury alkyls are used as alkylating agents in organic syntheses.

3.1.7.3 Toxicity

Vapour inhalation is the main route for the entry of metallic mercury into the body. Around 80% of inhaled mercury vapour is absorbed in the lung (alveoli). Digestive absorption of metallic mercury is negligible (lower than 0.01% of the administered dose). The main routes of entry of inorganic mercury compounds (mercury salts) are the lungs (atomization of mercury salts) and the gastrointestinal tract. In the latter case, absorption is often the result of accidental or voluntary ingestion. It is estimated that 2 to 10% of ingested mercury salts are absorbed through the intestinal tract. The health effects of mercury were summarized in Table 2.

Skin absorption of metallic mercury and certain of its compounds is possible, although the rate of absorption is low. After entry into the body, metallic mercury continues to exist for a short time in metallic form, which explains its penetration of the blood-brain barrier. In blood and tissues metallic mercury is rapidly oxidized to Hg^{2+} mercury ion, which fixes to proteins. In the blood, inorganic mercury is also distributed between plasma and red blood cells. The kidney and brain are the sites of deposition following exposure to metallic mercury vapours, and the kidney following exposure to inorganic mercury salts (85).

3.1.7.4 Acute poisoning

The symptoms of acute poisoning include pulmonary irritation (chemical pneumonia), perhaps leading to acute pulmonary oedema. Renal involvement is also possible. Acute poisoning is more often the result of accidental or voluntary ingestion of a mercury salt. This leads to severe inflammation of the gastrointestinal tract followed rapidly by renal insufficiency due to necrosis of the proximal convoluted tubules.

3.1.7.5 Chronic exposure

Chronic mercury poisoning usually starts insidiously, which makes the early detection of incipient poisoning difficult. The main target organ is the nervous system. Initially, suitable tests can be used to detect psychomotor and neuro-muscular changes and slight tremor. Slight renal involvement (proteinuria, albuminuria, enzymuria) may be detectable earlier than neurological involvement. If excessive exposure is not corrected, neurological

and other manifestations (e.g., tremor, sweating, dermatography) become more pronounced, associated with changes in behaviour and personality disorders and, perhaps, digestive disorders (stomatitis, diarrhoea) and a deterioration in general status (anorexia, weight loss). Once this stage has been reached, termination of exposure may not lead to total recovery.

In chronic mercury poisoning, digestive and nervous symptoms predominate and, although the former are of earlier onset, the latter are more obvious; other significant but less intense symptoms may be present. The duration of the period of mercury absorption preceding the appearance of clinical symptoms depends on the level of absorption and individual factors. The main early signs include slight digestive disorders, in particular, loss of appetite; intermittent tremor, sometimes in specific muscle groups; and neurotic disorders varying in intensity. The course of intoxication may vary considerably from case to case. If exposure is terminated immediately upon the appearance of the first symptoms, full recovery usually occurs; however, if exposure is not terminated and the intoxication becomes firmly established, no more than an alleviation of symptoms can be expected in the majority of cases.

There have been studies over the years on the relationships between renal function and urinary mercury levels. The effects of low-level exposures are still not well documented or understood. At higher levels (above 50 μg/g (micrograms per gram) abnormal renal function (as evidenced by N-acetyl-B-D-glucosaminidase (NAG), which is a sensitive indicator of damage to the kidneys) have been observed. The NAG levels were correlated with both the urinary mercury levels and the results of neurological and behavioural testing (86).

Chronic poisoning is accompanied by mild anemia sometimes preceded by polycythaemia resulting from bone marrow irritation. Lymphocytosis and eosinophilia have also been observed.

Absorption of phenylmercuric acetate (PMA) may occur through inhalation of aerosols containing PMA, through skin absorption or by ingestion. The solubility of the mercurial and the particle size of the aerosols are determining factors for the extent of absorption. PMA is more efficiently absorbed by ingestion than are inorganic mercuric salts. Phenylmercury is transported mainly in blood and distributed in the blood cells (90%), accumulates in the liver and is there decomposed into inorganic mercury. Some phenylmercury is excreted in the bile. The main portion absorbed in the body is distributed in the tissues as inorganic mercury and accumulated in the kidney. On chronic exposure, mercury distribution and excretion follow the pattern seen on exposure to inorganic mercury.

Occupational exposure to phenylmercury compounds occurs in the manufacture and handling of products treated with fungicides containing phenylmercury compounds. Acute inhalation of large amounts may cause lung damage. Exposure of the skin to a concentrated solution of phenylmercury compounds may cause chemical burns with blistering. Ingestion of large amounts of phenylmercury may cause renal and liver damage. Chronic poisoning gives rise to renal damage due to accumulation of inorganic mercury in the renal tubules.

Available clinical data do not permit extensive conclusions about dose-response relationships. They suggest, however, that phenylmercury compounds are less toxic than inorganic mercury compounds or long-term exposure. There is some evidence of mild adverse effects on the blood.

Alkyl mercury compounds. From a practical point of view, the short-chained alkyl mercury compounds, like methylmercury and ethylmercury, are the most important, although some exotic mercury compounds, generally used in laboratory research, have led to spectacular rapid deaths from acute poisoning. These compounds have been extensively used in seed treatment where they have been responsible for a number of fatalities. Methylmercuric chloride forms white crystals with a characteristic odour, while ethylmercury chloride; (chloroethylmercury) forms white flakes. Volatile methylmercury compounds, like methylmercury chloride, are absorbed to about 80% upon inhalation of vapour. More than 95% of short-chained alkyl mercury compounds are absorbed by ingestion, although the absorption of methylmercury compounds by the skin can be efficient, depending on their solubility and concentration and the condition of the skin (87).

Methylmercury is transported in the red blood cells (95%), and a small fraction is bound to plasma proteins. The distribution to the different tissues of the body is rather slow and it takes about four days before equilibrium is obtained. Methylmercury is concentrated in the central nervous system and especially in grey matter. About 10% of the body burden of mercury is found in the brain. The highest concentration is found in the occipital cortex and the cerebellum. In pregnant women methylmercury is transferred in the placenta to the foetus and especially accumulated in the fetal brain.

3.1.7.6 Toxicity of organic mercury

Poisoning by alkyl mercury may occur on inhalation of vapour and dust containing alkyl mercury and in the manufacture of the mercurial or in handling the final material. Skin contact with concentrated solutions results in chemical burns and blistering. In small agricultural operations there is a risk of exchange between treated seed and products intended for food, followed by involuntary intake of large amounts of alkyl mercury. On acute exposure the signs and symptoms of poisoning have an insidious onset and appear with a latency period which may vary from one to several weeks.

On chronic exposure the onset is more insidious, but the symptoms and signs are essentially the same, due to the accumulation of mercury in the central nervous system, causing neuron damage in the sensory cortex, such as visual cortex, auditory cortex and the pre- and post-central areas. The signs are characterized by sensory disturbances with paresthaesia in the distal extremities, in the tongue and around the lips. With more severe intoxications ataxia, concentric constrictions of the visual fields, impairment of hearing and extrapyramidal symptoms may appear. In severe cases chronic seizures occur.

The period in life most sensitive to methylmercury poisoning is the time in utero; the foetus seems to be between 2 and 5 times more sensitive than the adult. Exposure in utero results in cerebral palsy, partly due to inhibition of the migration of neurons from central parts to the peripheral cortical areas. In less severe cases retardation in the psychomotor development has been observed.

The most common alkoxyalkyl compounds used are methoxyethyl mercury salts (e.g., methoxyethylmercury acetate), which have replaced the short-chain alkyl compounds in seed treatment in many industrial countries, in which the alkyl compounds have been banned due to their hazardousness. The available information is very limited. Alkoxyalkyl compounds are absorbed by inhalation and by ingestion more efficiently than inorganic

mercury salts (88). The distribution and excretion patterns of absorbed mercury follow those of inorganic mercury salts. Excretion occurs through the intestinal tract and the kidney. To what extent unchanged alkoxyalkyl mercury is excreted in humans is unknown. Exposure to alkoxyalkyl mercury compounds can occur in the manufacture of the compound and in handling the final product(s) treated with the mercurial. Methoxyethyl mercury acetate is a vesicant when applied in concentrated solutions to the skin. Inhalation of methoxyethyl mercury salt dust may cause lung damage, and chronic poisoning due to long-term exposure may give rise to renal damage.

Most exposure to organic mercury compounds involves mixed exposure to mercury vapour and the organic compound, as the organic mercury compounds decompose and release mercury vapour. All technical measures pertaining to exposure to mercury vapour should be applied for exposure to organic mercury compounds. Thus, contamination of clothes and/or parts of the body should be avoided, as it may be a dangerous source of mercury vapour close to the breathing zone. Special protective work clothes should be used and changed after the workshift. Spray painting with paint containing mercurials requires respiratory protective equipment and adequate ventilation. The short-chained alkyl mercury compounds should be eliminated and replaced whenever possible. If handling cannot be avoided, an enclosed system should be used, combined with adequate ventilation, to limit exposure to a minimum.

Great care must be exercised in preventing the contamination of water sources with mercury effluent since the mercury can be incorporated into the food chain, leading to disasters such as that which occurred in Minamata, Japan.

3.1.8 Metal carbonyls

Metal carbonyls have the general formula $Me_x(CO)_y$, and are formed by combination of the metal (Me) with carbon monoxide (CO). Physical properties of some metal carbonyls are listed in Table 3 (89). Most are solids at ordinary temperatures, but nickel carbonyl, iron pentacarbonyl and ruthenium pentacarbonyl are liquids, and cobalt hydrocarbonyl is a gas. Since iron pentacarbonyl and cobalt hydrocarbonyl also have high vapour pressures and potential for inadvertant formation, they warrant serious consideration as possible occupational toxicants. Most metal carbonyls react vigorously with oxygen and oxidizing substances, and some ignite spontaneously. Upon exposure to air and light, nickel carbonyl decomposes to carbon monoxide and particulate nickel metal, cobalt hydrocarbonyl decomposes to cobalt octacarbonyl and hydrogen, and iron pentacarbonyl decomposes to iron nonacarbonyl and carbon monoxide (90).

Metal carbonyls are used in isolating certain metals (e.g., nickel) from complex ores, for producing carbon steel, and for metallizing by vapour deposition. They are also used as catalysts in organic reactions (e.g., cobalt hydrocarbonyl or nickel carbonyl in olefin oxidation; cobalt octacarbonyl for the synthesis of aldehydes; nickel carbonyl for the synthesis of acrylic esters). Iron pentacarbonyl is used as a catalyst for various organic reactions, and is decomposed to make finely powdered, ultra pure iron (so-called carbonyl iron), which is used in the computer and electronics industries. Methycyclopentadienyl manganese tricarbonyl (MMT) ($CH_3C_5H_4Mn(CO)_3$) is an antiknock additive to gasoline.

Metal carbonyl	Mol. Wt.	Sp. Gr. (20°C)	M.P. (°C)	B.P. (°C)	V.P. (25°C) mm Hg
$Ni(CO)_4$	170.75	1.31	–19	43	390
$CoH(CO)_4$	171.99	–	–26	–	high
$Co_2(CO)_8$	341.95	1.87	51	52*	1.5
$Co_4(CO)_{12}$	571.86	–	60*	–	very low
$Cr(CO)_6$	220.06	1.77	110*	151	0.4
$Fe_2(CO)_9$	363.79	2.08	80*	–	–
$Fe(CO)_5$	195.90	1.46	–25	103	30.5
$Fe(CO)_4$	167.89	2.00	approx. 140*	–	–
$Mo(CO)_6$	264.00	1.96	150*	156	0.2
$Ru(CO)_5$	241.12	–	–22	–	–
$W(CO)_6$	351.91	2.65	approx. 150*	175	0.1

*Decomposition starts at temperature shown.

Table 3. Physical properties of some metal carbonyls.

3.1.8.1 Toxicity

The toxicity of a given metal carbonyl depends on the toxicity of carbon monoxide and of the metal from which it is derived, as well as the volatility and instability of the carbonyl itself. The principal route of exposure is inhalation, but skin absorption can occur with the liquid carbonyls. The relative acute toxicity (LD_{50} for the rat) of nickel carbonyl, cobalt hydrocarbonyl and iron pentacarbonyl may be expressed by the ratio 1:0.52:0.33. Inhalation exposures of experimental animals to these substances induce acute interstitial pneumonitis, with pulmonary oedema and capillary damage, as well as injury to the brain, liver and kidneys (91).

Iron pentacarbonyl can be formed inadvertently when carbon monoxide, or a gas mixture containing carbon monoxide, is stored under pressure in steel cylinders or fed through steel pipes, when illuminating gas is produced by petroleum reforming, or when gas welding is carried out. Presence of carbon monoxide in emission discharges from blast furnaces, electric arc furnaces and cupola furnaces during steel-making can also lead to the formation of iron pentacarbonyl.

3.1.8.2 Nickel carbonyl

Nickel carbonyl ($Ni(CO)_4$) is mainly used as an intermediate in the Mind process for nickel refining, but it is also used for vapour-plating in the metallurgical and electronics industries and as a catalyst for synthesis of acrylic monomers in the plastics industry. Inadvertent formation of nickel carbonyl can occur in industrial processes that use nickel catalysts, such as coal gasification, petroleum refining and hydrogenation reactions, or during incineration of nickel-coated papers that are used for pressure-sensitive business forms (92).

3.1.8.3 Toxicity

Acute, accidental exposure of workers to inhalation of nickel carbonyl usually produces mild, non-specific, immediate symptoms, including nausea, vertigo, headache, dyspnoea and chest pain. These initial symptoms usually disappear within a few hours. After 12 to 36 hours, and occasionally as long as 5 days after exposure, severe pulmonary symptoms develop, with cough, dyspnoea, tachycardia, cyanosis, profound weakness and often gastrointestinal symptoms. Human fatalities have occurred 4 to 13 days after exposure to nickel carbonyl; deaths have resulted from diffuse interstitial pneumonitis, cerebral hemorrhage or cerebral oedema. In addition to pathologic lesions in the lungs and brain, lesions have been found in liver, kidneys, adrenals and spleen. In patients who survive acute nickel carbonyl poisoning, pulmonary insufficiency often causes protracted convalescence. Nickel carbonyl is carcinogenic and teratogenic in rats; the European Union has classified nickel carbonyl as an animal teratogen. Processes that use nickel carbonyl constitute disaster hazards, since fire and explosion can occur when nickel carbonyl is exposed to air, heat, flames or oxidizers. Decomposition of nickel carbonyl is attended by additional toxic hazards from inhalation of its decomposition products, carbon monoxide and finely particulate nickel metal.

Chronic exposure of workers to inhalation of low atmospheric concentrations of nickel carbonyl (0.007 to 0.52 mg/m^3) can cause neurological symptoms such as insomnia, headache, dizziness, memory loss, and other manifestations including chest tightness, excessive sweating, alopecia. Electroencephalographic abnormalities and elevated serum monoamine oxidase activity have been observed in workers with chronic exposures to nickel carbonyl. A synergistic effect of cigarette smoking and nickel carbonyl exposure on the frequency of sister-chromatid exchanges was noted in a cytogenetic evaluation of workers with chronic exposure to nickel carbonyl.

Because of its flammability and tendency to explode, nickel carbonyl should be stored in tightly closed containers in a cool, well-ventilated area, away from heat and oxidizers such as nitric acid and chlorine. Flames and sources of ignition should be prohibited wherever nickel carbonyl is handled, used or stored. Nickel carbonyl should be transported in steel cylinders. Foam, dry chemical, or CO_2 fire extinguishers should be used to extinguish burning nickel carbonyl, rather than a stream of water, which might scatter and spread the fire.

Exposures are classified as “mild” if the initial 8-h specimen of urine has a nickel concentration less than 100 µg/l, “moderate” if the nickel concentration is 100 to 500 µg/l, and “severe” if the nickel concentration exceeds 500 µg/l (93). Sodium diethyldithiocarbamate is the drug of choice for chelation therapy of acute nickel carbonyl poisoning. Ancillary therapeutic measures include bed rest, oxygen therapy, corticosteroids and prophylactic antibiotics. Carbon monoxide poisoning may occur simultaneously and requires treatment.

4. Comparison of metal concentrations in air samples

In literature, the most published articles on metal concentrations in air phases is related with lead, nickel, cadmium and arsenic levels in airborne aerosol samples. Liang et al. (1990) examined six metal levels in air taken from laboratory and clean room (32) Gucer et al.

(1992) determined six metal concentrations in ambient air taken from Malatya city depending on month of year and distance from center of city (28). Jaradat and momani (1999) analyzed soil, plant and air samples taken from both sides of the major highway connecting for Cu, Pb, Cd and Zn (31). Fernandez et al. (2000) determined five metal concentrations in four fractionation of particulate matters in air (94). Fuchtjohann et al. (2000) used GFAAS and ICP-MS to determine soluble ($NiCl_2$ and partly $NiCO_3$) and insoluble (NiO) nickel compounds in ambient air dusts (airborne particulate matters) taken from locations close to two metallurgical plants (95). They found maximum concentrations in total nickel at both sampling sites as 40 ng/m^3 and 160 ng/m^3 whereas the mean values were 9 ng/m^3 and 28 ng/m^3 (95). Bolt et al. (2000) determined Ni levels in airborne dusts collected from a metal factory processing nickel and nickel alloys (96). They found Ni concentrations in range of 10.000-4.920.000 ng/m^3. Hadad et al. (2003) found Pb, Cr and Fe concentrations in Tehran air samples as 1.040.000, 56.000 and 2.720.000 ng/m^3, respectively (30). Wada et al. (2001) reported mean Pb, Ni and Mn concentrations in airborne samples taken from Nagasaki as 8, 4 and 12 ng/m^3 (37). Bhat and Pillai (1997) determined mean 0.42 ng Be /m3 in air sample taken from the vicinity of Be metal plant at New Bombay (97). Gurjar and Mohan (2003) reported Cd, Cr and Ni concentrations in atmospheric environment at fourten cities of India in ranges of 1-21, 16-207 and 23-257 ng/m^3, respectively (34). Pekney and Davidson (2005) determined 28 metal concentrations in ambient particulate matter by ICP-MS (33). Vijayanand et al. (2008) assessed seven trace metals in the ambient air of Coimbatore city in India (29). They found Ni, Pb and Cr concentrations in range of BDL-310, 210-620 and 5-880 ng/m^3, respectively (29). Limbeck et al. (2009) found Ni, Pb and Cr concentrations in ranges of 6-10, 9-11 and 4-6 ng/m^3, respectively (27). Canepari et al. (2009) compared XRF, and ICP-OES results for multielement concentrations in ambient air suspended particulate matter (98). Chen and Lippmann (2009) reviewed effect of metal concentrations in ambient air particulate matter on human health (35). They reported that Ni, Pb and Cr levels in PM2.5 for 13 city in USA were in ranges of 1-2, 2-14 and 1-3 ng/m^3, respectively. Morishita et al. (2006) attempted to determine source of pollution by using concentrations of trace metals including Pb, Zn, Cd and Fe in PM2.5 (99). Odabasi et al. (2002) determined 11 trace metals in ambient air samples taken from Izmir city center and compared their results with other values reported in literature (36). Newhook et al. (2003) reviewed trace metal concentrations in ambient air (PM10) samples near copper smelter and refineries, and zinc plants in Canada (40). Mohanraj et al. (2004) determined six trace metals in airborne samples from India (38). They found 2.147 ng Pb /m3 in particulate matter taken from industrial location. Pierre et al. (2002) attempted to determine relationship between blood lead concentration of workers employed in crystal industry and ambient air lead (100). Vanhoof et al. (2003) determined Pb, Cu and Zn concentrations in ambient air from nonferrous metal industry (39). Fang et al. reviewed 7 metal concentrations in particulate matter (PM2.5 and PM10) taken fro Asia countries between 2000-2004 (101). Krzemińska-Flowers et al. (2006) determined 15 trace metals in urban air particulate matter by ICP-MS (102). They compared the results taking into consideration summer and winter season. They found that winter-Ag, As and Hg concentrations in both PM3 and PM10 taken from all three locations having different pollution were higher than in summer season whereas other trace metals changed depending on season and locations. These results reveal the difficulties in comparison of trace metal concentrations in air samples due to many factors affected the values. The detailed concentrations and information about those studies were given in Table 4.

Location	Character	Size	Cd	Pb	Ni	Cu	As	Fe	Cr	Ref.
Boston-USA	PM2.5=16.5 μg/m³	PM2.5		240	9	11		62		7
St. Louis-USA	PM2.5=19.2 μg/m³			213	2	30		144		
Knoxville-USA	PM2.5=21.1 μg/m³			109	1	13		117		
Madison-USA	PM2.5=11.3 μg/m³			33	0.5	6		44		
Steubenville-USA	PM2.5=30.5 μg/m³			185	4	12		542		
Topeka-USA	PM2.5=12.2 μg/m³			72	0.6	7		72		
Schafberg	Out of City-Viena	PM10	0.4	8.9	7.0	7.2	0.7	250	3.8	27
Kendlerstrabe	City Suburb-Moderate Traffic		0.5	11	5.7	21	0.9	780	5.5	
Rinnböckstrabe	Iner-City-Density Traffic		0.5	11	9.9	20	1.2	740	5.0	
Malatya-Turkey	City Center		ND	3630-27270		1080-1760		4280-6030		28
Coimbatore-India	Residential and Traffic Area	SPM	BDL	340	120	660		2850	BDL	29
Coimbatore-India	Residential and Traffic Area		BDL	560	150	610		3150	470	
Coimbatore-India	Industrial Area		BDL	280	90	880		1850	630	
Coimbatore-India	Industrial Area		BDL	320	230	510		3650	170	
Coimbatore-India	Industrial Area		BDL	230	82	310		3300	430	
Coimbatore-India	Industrial and Traffic Area		BDL	520	BDL	290		2200	40	
Coimbatore-India	Industrial and Traffic Area		BDL	430	130	460		2950	390	
Coimbatore-India	Residential and Traffic Area		BDL	170	120	730		2200	280	
Coimbatore-India	Industrial and Traffic Area		BDL	320	100	690		4100	460	
Coimbatore-India	Industrial and Traffic Area		BDL	430	120	530		6000	510	
Tehran	Traffic-Industrial Summer	SPM		1.040.000				2720.000	56	30

Location	Character	Size	Cd	Pb	Ni	Cu	As	Fe	Cr	Ref.
	Traffic-Industrial Winter			1.000.000				1740.000	40	
Shiraz	Traffic-Industrial Summer			410				3252.000	15	
	Traffic-Industrial Winter			669				1891.000	15	
Amman-Jordan	Highweigh-Roadside			260-1370		260-600				31
Connecticut-laboratory	Nov. 1988			1.25		1.89		6.65		32
	June 1989			1.24		1.43		16.9		
Connecticut-Clean room	Nov 1988			0.19		0.31		1.54		
	June 1989			0.18		0.41		4.13		
Pitsburgh	Ambient particulate matter	PM2.5		20			20	200		33
Andhra Pradesh – India	Ambient air		14		68				32	34
Bihar			11		208				133	
Chandigarh (UT) - India			5		257				207	
Gujarat-India			16		45				51	
Haryana-India			8		134				90	
Himachal Pradesh-India			8		66				64	
Karnataka-India			1		43				17	
Kerala-India			6		23				18	
Orissa-India			8		78				144	
Punjab-India			4		107				82	
Rajasthan-India			20		63				16	
Tamil Nadu-India			6		28				17	
Uttar Pradesh-India			21		222				71	
Burlington-USA	Air particulate matter	$PM_{2.5}$	-	-	2	2	-	-	2	35
Philadelphia-USA			-	5	6	4	-	-	2	
Atlanta-USA			-	3	-	2	1	1	-	
Detroit-USA			-	6	2	6	2	-	2	
Chicago-USA			-	6	1	4	1	1	1	
ST.Louis-USA			-	14	2	14	2	1	2	
Houston-USA			-	2	2	3	1	1	1	
Minneapolis-USA			-	5	2	3	2	-	2	
Boulder-USA			-	5	1	4	-	-	2	
Phoenix-USA			-	-	3	6	2	3	2	
Seattle-USA			-	4	2	3	1	1	2	
Sacramento-USA			-	-	10	6	2	-	2	

Location	Character	Size	Cd	Pb	Ni	Cu	As	Fe	Cr	Ref.
Riverside-Rubidojx-USA			-	6	2	6	2	-	3	
Izmir, Turkey	City suburb	TSP	8	111	39	154			11	36
Nagasaki City	High Traffic			7.95	3.78					37
India, (6 sampling station)	Urban residential, industrial, highway,		2.8	143.5	31.37	388.6			14.2	38
Location 1	Southern side the road	Rural		29		20				39
Location 2	Eastern side the road	PM10		17		12				
Location 3	Northern side the road	TSP		26		17				
Canada	Copper smelters	PM	1	197			28			40
Canada	Noranda-horne	PM	2	18			33-255			
Canada	Copper refineries	PM	0	34			8			
Canada	Zinc plants	PM	19							
Japon, Sapporo	Urban City	TSP		43.9	3.81	20.9		625	2.61	41
Japon, Tokyo				125	5.63	30.2		677	6.09	
China, Hong Kong	Airborne	TSP		79	-	88		1421	-	42
	Traffic	PM10		98740	8620	35380		860	6850	
		PM2.5		76860	5340	17320		250	2430	
China, Hong Kong	Industry	PM10		100520	9580	63530		790	5750	43
		PM2.5		91620	6000	36780		480	4510	
	Urban	PM10		62750	8270	15330		620	4970	
		PM2.5		60130	6330	9710		190	4190	
China, Shanghai	University	PM2.5		270	-	-		820	-	44
	Urban	PM2.5		280	-	-		900	-	
Vietnam, Ho Chi Minh	Urban	PM2		73	-	3		1222	-	45
		PM2-10		79	-	2		261	-	
		TSP		146	-	-		2904	-	
India,Sakinaka, Mumbai	Traffic Junction	SPM		1060	-	370		165500	-	46
India, Gandhinagar		SPM		820	-	1550		265500	-	
Indonesia, Bukit Tinggi	Tropical Jungle	PM2.5		1.22	-	<0.14		2.6	-	47
		PM2.5-10		<0.3	-	<0.16		14.8	-	
Indonesia, Pontianak	Rural	PM2.5		8.7	-	0.22		4.7	-	
		PM2.5-10		4.2	-	0.56		53	-	
		PM2.5		26	11	-		581	-	
Indonesia	Rural	PM2.5-10		3	2	-		1479	-	48
		TSP		39	18	-		2700	-	
		<2.5		88	54	32		994	360	

Location	Character	Size	Cd	Pb	Ni	Cu	As	Fe	Cr	Ref.
Indonesia	Temple	PM2.5-10		120	73	14		568	147	49
Taiwan	Traffic Junction (daytime)	TSP		180	-	240		1710	-	50
	Traffic Junction (nighttime)	TSP		180	-	230		1660	-	
	Inland Urban	PM10		150	30	-		1730	-	
Taiwan, Kaoshiung	Inland Industrial	PM10		80	90	-		2090	-	51
	Coastal Industrial	PM10		190	40			2140	-	
	Coastal Urban	PM10		340	30	-		1740	-	
Korea, Taejon	Industrial	TSP		269	33.6	54.9		1839	31.8	52
		PM10		195	42.6	32.4		1577	39.3	
Korea, Seoul	Urban	PM2.5		96.4	19.6	27.8		743	13.7	53
		PM10		124	47.8	50.1		2321	18.8	
Annual means-Sites in Europe	Rural	TSP	0.03-0.7		0.1-3.5		0.19-4.2			54
	Urban		0.11-1.2		1.6-13		0.8-3.1			
	Traffic		0.21-2.4		2.4-21		0.05-4.1			
	Industrial		0.2-23.7		2.2-102		1.2-97			

PM: particulate matter; TSP: Total Suspended Particles; SPM: Suspected Particule Matter
ND: Not detection; BDL: Below detection limit

Table 4. Reported metal concentrations and information related with air particules in literature. The metal concenttrations are ng/m^3.

5. Conclusion

Exposure to chemicals is a serious public health problems that affect wildlife, soils, water, and air and can have very harmful human health effects. Exposures to chemicals including metals must be identified promptly, and individuals exposed to them must be evaluated and managed without delay. Major sources of metal emissions into environment can be able to change due to the updating in industrial activities and legislations. Before forbidding tetraethyl Pb in gasoline, automobile emissions were the primary source of Pb emissions while, nowadays, piston engine aircraft and industrial sources seem the two largest sources, depending on developing of countries (25).

As it is seen from Table 4, the reported toxic metal concentrations in air phases are significantly difference even if taking consideration their character such as rural, urban, traffic and industry. It was found that metal concentrations (ng/m^3) were in the ranges of 0.03-21 for Cd, 0.18-1.040.000 for Pb, 0.1-9.580 for Ni, 0.14-63.530 for Cu, 0.7-255 for As, 1-3.252.000 for Fe and 1-6.850 for Cr. Particularly, the observed metal concentrations in air of industry area in China are

extremely higher. As a result, there are a greet need for determinations of metal concentrations in air phases, to obtain reliable and considerable values.

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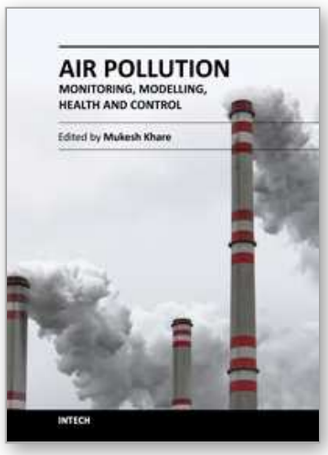

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Air pollution has always been a trans-boundary environmental problem and a matter of global concern for past many years. High concentrations of air pollutants due to numerous anthropogenic activities influence the air quality. There are many books on this subject, but the one in front of you will probably help in filling the gaps existing in the area of air quality monitoring, modelling, exposure, health and control, and can be of great help to graduate students professionals and researchers. The book is divided in two volumes dealing with various monitoring techniques of air pollutants, their predictions and control. It also contains case studies describing the exposure and health implications of air pollutants on living biota in different countries across the globe.

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51000 Rijeka, Croatia
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中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
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