Chapter 7
Heavy Metal Contamination from Construction Materials

Ayodeji Ojo Oteyola and Folasade Adesola Ola-Oladimeji

Abstract Construction or building materials can pose a major public health challenge because they can act as pollutant emission sources. Building materials lead to the contamination of heavy metal. Despite several discoveries as regards the adverse health effects of heavy metals, there is an increase in heavy metal exposure especially in underdeveloped and developing countries. Lead pipes are used within buildings. Lead that dissolves into the drinking water from lead pipes is a threat to human health. The peeling and cracking of lead-based paint is also a major health concern as it has been reported that it affects young children's brain and sense organs. In adults, lead accumulation can lead to high blood pressure, reproduction dysfunction and sometimes death. This chapter focuses on the use of lead and other heavy metals in buildings and their toxicity to humans. The assessment of the risk associated with lead in drinking water is also discussed. In addition, lead pipe replacement is also deliberated. The chapter also reflects on other heavy metals which include cadmium, chromium and mercury with respect to their uses, toxicity and health effects on indoor pollution. Biological monitoring of cadmium, chromium and mercury are also explained.

Keywords Building materials · Cadmium · Chromium · Lead · Mercury · Toxicity

7.1 Introduction

Building materials can act as a source of pollutant emission and thereby affect the quality of indoor air (US EPA 1994). The number of compounds emitted from building materials varies. Exposure to these compounds can be hazardous to human health. There have been several struggles and efforts to limit the exposure in the building design industry. The idea is that if in a building design, a building material

A. O. Oteyola (✉)
Departamento de Morphology, Institute of Biological Sciences, Federal University of Minas Gerais, Av. Antônio Carlos, Belo Horizonte, Minas Gerais, Brazil

F. A. Ola-Oladimeji
Department of Zoology and Environmental Biology, Ekiti State University, Ado-Ekiti, Nigeria

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is known to be a source of pollution and needs more ventilation to neutralize its effects, it will be more advisable to use an alternative material that has less emission rate (CIBSE 1996).

Heavy metals pose a high risk of adverse effects on human health, some metals are carcinogenic, neurotoxic and immunotoxic. Metals are usually found in the environment and as a result of the extensive use in the environment, human exposure is unavoidable. Understanding the mechanisms of heavy metal toxicity has been challenging as a result of the association between metals and living systems. Research has shown that metals are compromised in cardio-respiratory related illnesses that are linked to the pollution of the air (Burnett et al. 2000; Vincent et al. 2001; Claiborne et al. 2002; Sorensen et al. 2005). Osornio-Vargas et al. (2003) reported that transition metals are linked to oxidative stress and respiratory dysfunction.

Some metals are indispensable and vital for life as a result of their role in the provision of required enzymes but excessive concentration of these enzymes can be harmful either by choking important functional groups or altering the conformation of protein molecules (Collins and Stotzky 1989). Metals have a profound effect on physiological and biochemical processes which include photosynthesis and respiration inhibition (Vangronsveld and Clijsters 1994). Transition elements are used in making paints and this poses a serious threat to human health as a result of deviation from manufacturer's standard, unsuitable storage, carelessness in handling and transportation. Lead based paint is still being used in a lot of homes today. The peeling and damage of lead-based paint is hazardous and it can amount to threat to human health especially in children. Cadmium, mercury and some heavy metals are naturally occurring but human exposure occurs as a result of anthropogenic activities.

This chapter focuses on the heavy metals which include lead, cadmium, chromium and mercury as it relates to building materials with emphasis on their structure, uses and its toxicological effects. Awareness about environmental pollutants and their adverse effects on human health cannot be over-emphasized as the aim is to protect people from avoidable exposures.

7.2 Lead

Lead metals play an important role in transporting water and its usage can be traced back as far as the Roman times. Roofing materials can also be produced from lead metals. Lead metals decay slowly which justify its high acceptability in usage. However, lead has been known to be toxic; the first reported effect of lead on human health was the fall of Roman Empire which was influenced by lead poisoning (Dutrizac et al. 1982; Hodge 1981; Nriagu 1983). In the home today, health problems from lead can be from lead pipes which are used in connecting homes to the water source; it can also be from lead roofing and guttering; it can also be from lead-based house paint (Wani et al. 2015). Lead absorption in the body accumulates in the blood, bones, liver, kidney, brain and skin. Krzywy et al. (2010) revealed that lead

Table 7.1 Lead pipes occurrence in Europe (Adapted after: Van den Hoven et al. 1999)

Country	Percentage of Pb communication pipe
Belgium	19
Denmark	0
France	39
Germany	3
Greece	<1
Ireland	50
Italy	2
Luxembourg	7
Netherlands	6
UK	40

causes reproductive dysfunction, hepatic dysfunction and also affects the immune and gastrointestinal systems (Krzywy et al. 2010).

7.2.1 Lead Piping and Water Contamination

Lead pipes are used to convey water from the water main source to the apartment buildings. Lead pipes are buried inside the soil thereby making it difficult to establish their presence or absence. In the 1800s, the beginning of urbanization and mass housing brought about the use of lead connection pipes. Despite the caution that was expressed about the use of lead pipes in the nineteen century (Troesken 2006), the use of lead pipes continued till the 1980s in both Europe and North America. The occurrence of lead pipes in different countries in Europe is shown in Table 7.1. The length of the pipe varies from a few meters to hundreds of meters depending on the distance between the dwelling and the water source. The risk is associated with the length of the pipe, the longer the lead pipe, the greater the risk to human well-being.

Hayes and Skubala (2009) reported that about 25% of dwellings in Europe are likely to be supplied with drinking water through lead pipes. About 2–3% of dwellings in the US and Canada use lead pipes (IWA 2010). The corrosion of the pipe and the contact of the water with the lead pipe determine the amount of lead that will be present in the drinking water. Lead concentration in drinking water can vary from <1 to hundred μg/l.

7.2.2 Lead Toxicity

Lead is stored in the liver and also can be found in the kidneys. Its distribution can also be in the brain, adrenals, prostate, ovary, spleen, fat tissues, heart and spinal

cord (Seervarisai et al. 2015). Lead is harmful to human health; reports from clinical researches have confirmed this claim (Lanphear et al. 2000; Menke et al. 2006; Jusko et al. 2008; Khalil et al. 2009). Lead affects many biochemical processes such as calcium inhibition and reaction with proteins. Upon absorption, lead substitutes with calcium and this leads to interaction with biological processes and thereby causing abnormalities. As a result of its reaction with protein, Pb causes changes in enzyme structure and causes dysfunction in the binding site. The primary mechanism that leads to lead toxicity is the oxidative stress that is caused by lead.

Research from the World Health Organization has shown that lead affects children's central nervous system and development (WHO 2016). Children less than six (6) years old are very susceptible to the harmful effect of lead because of the brain growth and development (Wani et al. 2015; WHO 2016). Lead can impair memory, lower intelligent quotient (IQ) in children. Reports have shown that lead has profound effects on speech, hearing, vomiting and intestinal pain. At high concentration as shown in Table 7.2, lead poisoning can cause anemia, paralysis, convulsion or death (Giel-Pietraszuk et al. 2012; Rehman et al. 2018). During pregnancy, maternal lead exposure can be transferred to the developing embryo via placenta (Rehman et al. 2018; Omeljaniuk et al. 2018). In addition, during pregnancy, when the calcium intake of the mother is low, the lead that is stored in the mother's bone can be transferred via the blood to the fetus. This confirms that maternal exposure to lead before or during pregnancy can have a devastating effect on the developing embryo as a consequence of the stored lead in the bone (CDC 2010; Bellinger 2013). As a result

Table 7.2 Level of blood lead and degree of lead poisoning and the symptoms in children and adult (Adapted after: Giel-Pietraszuk et al. 2012)

Blood lead level (μg/L)	Degrees of lead poisoning	Synonyms	
		Children	Adults
<10	Low	Growth disorders, lower IQ, memory disabilities, hearing disorder, speech disorder	Movement from the maternal bloodstream to the fetal through the placenta
10–40	Mild	Muscle pain, fatigue, irritability, impaired hemoglobin synthesis, apathy	Increased in blood pressure
40–70	Moderate	Weight loss, constipation, vomiting, headache, fatigue, muscular weakness	Chronic hypertension, fertility abnormalities, reduced mental abilities
70–100	Serious	Anemia, nephropathy, paralysis	Reduction in sex drive, constipation, headaches, nephropathy, insomnia
>100	Acute poisoning	Convulsion, death	Anemia, death (>150 μg/L)

of the toxicity of lead in children, WHO has recommended blood lead concentration of 5 μg/dL or less for children (CDC 2010; Wani et al. 2015; WHO 2016).

Lead also has harmful effects in adults. It can lead to dysfunction in the nervous system (fatigue, drowsiness), circulatory system (increased blood pressure), hormones (fertility abnormalities, decreased libido), cardiovascular system and it can also lead to death (Wani et al. 2015). Individuals with high concentration of lead in their blood (500–870 μg/L) often encounter sinus node dysfunction and also atrioventricular block (Zawadzki et al. 2006). In adults, lead concentration in the blood has been linked to blood pressure. Nevertheless, its effect on blood pressure depends on dosage and exposure time (Wani et al. 2015). Navas-Acien and colleagues confirmed that exposure to lead is linked to hypertension (Navas-Acien et al. 2007). Research has also revealed the relationship between blood lead concentration and cancer especially brain, lung tumor (Steenland et al. 2019).

7.2.3 Replacement of Lead Pipes

There are some speculations that lead pipes have been withdrawn but of a truth, very few have been removed and many are still been used especially in places like Africa. Some water corporations also claim that lead pipes has been removed but this has been less effective because some homes still use lead pipes within their dwellings (DWI 2010). Efforts have been made to remove all lead pipes but internal lead piping has been the challenge as a result of the stress and the cost involved. Considering the various harmful effects of lead on children and adults, the idea is to have a lead pipe-free home. To achieve this, laws should be made that the home-owners should remove all the lead pipes before sale or letting it out. In the meantime, Hayes and Croft suggested that the application of point-of-use filters can be used to remove lead from water (Hayes and Croft 2012). Leaded solder was allowed in the 1980s. Many homes still have a leaded solder and the removal is not realistic, unless under special conditions. This suggests that water supply systems require a strategy to control corrosion so as to minimize these lethal effects. In addition, lead-free brass with about 8% lead has been allowed. However, in the 1980s, brass fittings containing >8% lead were used and many homes still have this internal pipework system. To have effective control, all countries need to take after the US by enforcing the use of brass with <0.25% of lead and ban the use of leaded solder.

7.3 Cadmium

7.3.1 Cadmium History and Application

Cadmium (Cd) was first discovered in Germany by Friedrich Stromeyer in 1817. Cd is readily found in little concentration mostly alongside sulphide of zinc, lead and copper. Associated use of Cd as building materials include pigments, stabilizers of PVC and alloys, protective agents of steel from corrosion, Ni–Cd batteries (WHO 1992; IARC 1993; Hayat et al. 2019). Cd compounds are used as coloring agents in borosilicate glass. Cd is a rare element; its estimation is around 0.1–0.2 ppm which signifies that it is the 67th element in order of abundance (Brewers et al. 1987).

7.3.2 Chemical Forms and Properties of Cd

Cadmium (Cd) is a member of the group XII in the periodic table of chemical elements. Cd atomic mass is as a result of eight stable isotopes. The colour of Cd is silvery-white. Cd properties are synthetically related to zinc and mercury. In the s orbital it has double electrons and a complete d orbital. Cd is resistant to corrosion hence, the main reason it serves as a protective agent. It is insoluble in water; it forms cadmium oxide when burned in air.

7.3.3 Cadmium Toxicity

The US environmental protection agency identifies cadmium as one of the 126 priority pollutants. Cadmium is absorbed in high quantities from air, food and water contaminations which confirms the role that building materials play in its absorption. Cadmium has the potential to accumulate throughout a lifetime which makes it more harmful. Cadmium salts accumulate and lead to the toxicity of many organs which includes the kidney, lungs, brain, testes, and the heart. Cadmium can cause anemia, kidney failure, anosmia, non-hypertrophic emphysema. People that make cadmium products like batteries, plastic, soldering or welding building materials can be exposed to cadmium.

Cadmium inhalation linked with respiratory diseases which include lung cancer. Mona and colleagues revealed an increase in cadmium level of smoker's urine (Mona et al. 2018). In addition, it was reported that the percentage of bone pain of smokers increased (95%) non-smokers (37.7%). This suggests that exposure to cadmium results in an ototoxic effect which can in turn lead to bone tissue loss. The epidemic of Itai-Itai disease was as a result of cadmium exposure (Hagino and Yoshioka 1960).

Cadmium exposure even at low doses can result in abnormalities in male and female reproduction and thereby affect pregnancy and offspring outcomes (Kumar

and Sharma 2019). Exposure to cadmium also causes irregularities in the menstrual cycle, alterations in reproductive hormones, pregnancy loss, premature birth and reduction in birth weight (Lokhande et al. 2004). Cadmium is also known to be an endocrine disruptor because it interferes with many hormonal signaling pathways, one of such is that it can bind estrogen receptor α; it can also interfere with signaling transduction through estrogen and mitogen-activated protein kinase (MAPK) signaling pathways (Söderholm et al. 2020).

Cadmium exposure has an epigenetic effect and can also alter gene expression. Exposure to cadmium can result in DNA methylation and histone modifications. Heavy metals can utilize their toxicity through the miRNA. Endogenous miRNA alterations have been associated with impaired signaling pathways which subsequently result in different kinds of cancer and disorders (Wallace et al. 2020). Takiguchi and colleagues (2003) in their study reported that short time exposure (24 h–1 week) results in hypomethylation, whereas long exposure (8–10 weeks) leads to hypermethylation (Takiguchi et al. 2003).

Cadmium has been classified as a cancer causing agent (WHO 1992; IARC 1993). Cadmium toxicity results in oxidative stress that further promotes the spread of tumor through its effect on cell cycle (Rani et al. 2014). Cadmium inhibits DNA repair mechanisms and this can lead to accumulation of damaged DNA hence the initiation of cancer (Giaginis et al. 2006). A study conducted from 2006 to 2014 as reported by Nawrot and colleagues showed that lung cancer is linked to the exposure to cadmium. In this study, it was revealed that cadmium is a risk factor for lung cancer (Nawrot et al. 2015). Furthermore, scientific data also suggests that cadmium exposure can be associated with prostate cancer (Julin et al. 2012), urinary bladder cancer (Feki-Tounsi and Hamza-Chaffai 2014), cancer of the kidney (Song et al. 2015) and pancreas cancer (Chen et al. 2015).

Studies in animal models have shown that antioxidants (vitamin C and E) can protect from toxicity induced by cadmium (Ognjanovic et al. 2003; Beytut et al. 2003). Membrane separation and reverse osmosis are sometimes used to get rid of heavy metals in liquid but it is very expensive and requires large quantities of chemicals.

7.3.4 Biomonitoring of Cadmium

Biomonitoring studies involving cadmium level helps physicians and public health officials to ascertain exposure level of cadmium in individuals and groups of population. Cadmium level in urine and blood help in biological monitoring of cadmium. Blood cadmium usually reveals current exposure and it ranges from 0.2 to 0.8 pg/l. Although, smokers are known to have higher concentrations ranging from 1.4 to 4.5 pg/l as reported by Elinder et al. (1994). Urinary excretion of cadmium for a day is a biological marker of lifespan exposure, whereas cadmium concentration in blood reveals current exposure (Staessen et al. 1996). Data from biomonitoring studies can help researchers to conduct future research on exposure and health effects of cadmium.

7.4 Chromium

7.4.1 Chromium History and Application

Chromium is commonly used together with other metals. It was discovered by Louis Vauquelin in 1797. Louis Vauquelin discovered chromium while he was making an experiment with a material called 'Siberian red lead', which is also known as 'mineral crocoite ($PbCrO_4$)'. Vauquelin produced chromium oxide (CrO_3) by putting together crocoite and hydrochloric acid (HCl) Application of chromium as building materials include dyes and paints, stainless steel, chrome plating and metallurgy.

Chromium is being added to metals to make stainless steel. Its corrosion resistant nature extends the lifespan of products making it an important ingredient in building materials. About 80% of mined chromium is used for metallurgical processes, in which many of the0se are used in making stainless steel. About 15% is used in the manufacturing of chromium chemicals and about 5% is used in refractory applications. The corrosion resistant nature and the attractive nature form the major factor for its selection. Potassium dichromate ($K_2Cr_2O_7$) is used in chemicals for leather tanning and also used for wood preservation (Johnson et al. 2006). Copper chrome arsenic (CCA) is used in the preservation of woods and it contains chromium trioxide, arsenic pentoxide and copper (II) oxide. It is made available in form of paste or water-based concentration and CCA has been used for preservation of wood so as to prevent decay caused by fungi and wood-boring insects (Cocker et al. 2006). Lead Chromate ($PbCrO_4$) has also been known to be used as yellow pigments in paints.

7.4.2 Chemical Form and Properties of Chromium

Chromium is used in manufacturing stainless steel and pressure-treated wood. Data from exposure to chromium from occupation shows that about half a million workers are exposed to chromium in the US and unspecified millions globally (Zhitkovich 2002; OSHA 2006). Exposure to chromium occurs through air emissions (cigarette smoke, industrial, automobile smoke) and water contamination (industrial release, wastewater leaching) (Welling et al. 2015). Cr(0) usually occurs in alloys with metals such as iron in its metallic form. Through welding, Cr(0)is converted to chromium (III) and chromium (VI). Chromium (III) is an indispensable micronutrient that is required in metabolism of glucose (Schwarz and Mertz 1959), but in the present-day, the report is now being debated (Ali et al. 2011; Xia et al. 2016; Vincent 2017, 2019).

Employees are exposed to chromium in more than 80 different industries. In construction companies, tanning, electroplating, metal finishing, welding, wood preservatives and pigments production, workers are exposed to chromium (IARC 1990).

7.4.3 Chromium Toxicity

Regardless of the mode of chromium absorption, the adverse health effects caused by chromium usually depend on the oxidation state, chemical species bioavailability and solubility. After absorption, chromium compounds can undergo alkylation and oxidation state alteration.

Chromium (III) has low membrane permeability, and this can lead to change in the membrane morphology, disruption in cell functions and also can induce DNA damage (Fang et al. 2014). Transferrin, a β-globulin, helps to protect against chromium toxicity that is obtained from food consumption by inhibiting the aggregation of chromium (III) and mediating chromium (III) transmembrane transportation. The transferrin (*Tf*) gene is distinct having about 38 recognized variants, studies linking the recognized variants of transferrin to Cr (III) transport is required to elucidate the effects of the variant (Edward et al. 2019).

Chromium (VI) exposure may affect many systems in the body which include skin, irritation in respiration, kidney failure, hepatic (liver damage and increase in liver enzymes), genetic (genotoxicity, mutagenicity and carcinogenicity) and reproductive effects (birth weight, anovulation, irregular menstrual cycle) (IARC 1990; Teklay 2016; Wang et al. 2017). However, the gravity of these health effects depends on the extent of the exposure to chromium (VI).

Chromium (VI) compounds have been identified as the primary source of lung cancer (IARC 1990). Chromium (IV) has been considered to be carcinogenic (IARC 1990). Low concentration of Chromium (VI) causes DNA breakage. The established limit by the Occupational Safety and Health Administration (OSHA 2006) was 5 $\mu g\ m^{-3}$, recent risk assessment has revealed that of 1000 employees cancer deaths, about 10–50 were exposed to this limit (Beyersmann and Hartwig 2008). Deposits of chromium (VI) are shown in different studies to cause tissue damage which eventually leads to lung cancer. Accumulation of Cr (VI) in tissues leads to DNA impairment, tumor initiation which eventually causes carcinogenesis (Ishikawa et al. 1994; Proctor et al. 2014; Browning and Wise 2017).

In vitro studies showed that chromium (VI) can form a reaction with histone and then be transported to the nucleus (Levina et al. 2006). The reaction and transportation suggests a mechanism of DNA damage. Cr (VI) exposure induces histone modifications which include reduced acetylation and increased biotinylation and this mechanism has been suggested as a mechanism of tumor initiation and progression (Xia et al. 2014).

Cr (VI) compounds are soluble penetrating the cell through a negatively charged ion carrier (Buttner and Beyersmann 1985). On getting to the cell, reduction reaction takes place by various chemical and enzymatic reductants and it eventually forms Cr (III). There is formation of reactive chromium species Cr (II), Cr (IV), Cr (V) during this reduction reaction. This can induce reactive oxidative stress (ROS) (Borthiry et al. 2007).

7.4.4 Biomonitoring of Chromium

The literature has provided enough reports as regards the biomonitoring of chromium level in urine in exposed populations. For instance in population of people working with materials linked to chromium (for instance, welder) (Mutti et al. 1984; Minoia and Cavalieri 1988; Brand et al. 2010). Little has been done regarding investigating residential populations that are potentially exposed to environmental chromium, the reason being the presence of high levels of chromium in urine of both exposed and controlled populations.

In a population of people handling timber treated with copper chrome arsenic (CCA), exposure was confirmed (Garrod et al. 1999). Dermal exposure was the primary route of exposure and exposure through inhalation was observed as relatively low. Chromium concentration in urine of workers that were exposed to wood preservatives is significantly higher than the non-exposed people.

Human biomonitoring results and concentration of metal air of 36 welders welding solid steel were revealed by Mutti et al. (1984). It was revealed in this study that water soluble Cr (IV) concentrations in the air ranged from 0.010 to 0.152 mg m^{-3} with a median of 0.065 mg m^{-3}, while total Cr concentrations in the air ranged from 0.012–0.224 mg m^{-3} with a median of 0.094 mg m^{-3}. The mean urinary chromium was 33.3 μg/g. Gube and colleagues revealed that after exposure, the mean chromium concentration in the urine is 1.61 μg/l (Gube et al. 2013).

7.5 Mercury

7.5.1 Mercury History and Application

Mercury is represented by the symbol "Hg". "Hg" was coined from Latin word "*hydrargyrum*". Mercury is liquid at ambient temperature. The use of mercury as building materials is related to its use in fluorescent lamps. It can also be utilized in technological applications such as thermometers, barometers, and gas meters. Culturally, it can be used as preservatives in home paints and also as antiseptics. The technological and cultural uses of mercury make it an indoor air pollutant in residential places.

Properties such as low melting and boiling points, biological cycle's participation makes mercury (Hg) to be considered as global pollutants. Mercury is released through natural and anthropogenic activities but studies have revealed that it is the anthropogenic emission that contaminates the environment. Estimates revealed that total release of mercury from both sources from past activities is 6000–11000 tons per year (Swartzendruber 2012). It is believed that emission through anthropogenic activities constitute about 80% of the yearly discharge to the environment. Following the industrial revolution, the mercury level of the surface water has tripled. Mercury concentrations are now about 10–12 times higher in marine animals today

as compared to the preindustrial times (prior to 1800) (Braune et al. 2005; Lamborg et al. 2014). Mercury compounds are mostly used in paint production. The content of mercury in a commercial paint is usually about 0.05% and this may cause air pollution.

7.5.2 Chemical Form and Properties of Mercury

Mercury is an odorless and silvery metallic liquid and it is insoluble in water. It is the 80th member on the periodic table. Mercury is ubiquitous and it is usually in three forms; (1) elemental (2) inorganic and (3) organic mercury. Reports have shown that all the forms of mercury have toxic effects on mammals (Clarkson et al. 2003; WHO 2007). Elemental (metallic) Hgwhich is liquid at room temperature has a high vapour pressure and it is released to the environment in the form of mercury vapour. The methylation of the inorganic form of mercury leads to the formation of the methylmercury. In the environment today, mammalian species are exposed to all the three forms of mercury; elemental (Hg), inorganic [Hg(I)], [Hg(II)] and organic mercuric compounds (WHO 2007).

Human exposure to elemental mercury is from artisanal gold mining. Inhalation of vapourized elemental mercury is usually absorbed into the blood and crosses the blood-brain barrier. Estimation shows that 10–15 million miners (mostly in developing countries) are affected by chronic mercury intoxication (Ha et al. 2017).

7.5.3 Mercurial Toxicity

Generally, mercury(II) chloride or mercuric chloride ($HgCl_2$) and methylmercury (MeHg) play a crucial role in inducing many biological processes ranging from increased lipid peroxidation to formation of ROS, depletion of glutathione (GSH) (Berntsen et al. 2003; Crespo-Lopez et al. 2009). Additionally, exposure to $HgCl_2$ and MeHg alters cell signaling (Chen et al. 2006), reduced integrity of cell membrane (Polunas et al. 2011), impaired DNA repair mechanism (Christie et al. 1985; Gadhia et al. 2012; Pieper et al. 2014; Ryu et al. 2014), immunomodulatory impacts (Gallagher et al. 2011) and also altered the DNA methylation (Farina et al. 2011) eventually resulting in adverse health effects. Many literature reviews discuss various mechanisms by which mercury exposure results in neurotoxicity and cell damage (Clarkson 2002; Mergler et al. 2007; Crespo-Lopez et al. 2009; Farina et al. 2011; Antunes et al. 2016).

In vitro studies have revealed the relationship between MeHg intoxication and neuronal deaths. Exposure to MeHg results in neuritic degeneration (Fujimura et al. 2009), oxidative damage (Yin et al. 2007) and this disruption of biological processes leads to neuronal deaths. It has also been well-documented that organic mercury has the ability to bind with thiol residues which eventually lead to dysfunction in protein

containing thiol groups. Mercury exposure can also inhibit the activities of enzymes that correct oxidative stress in cells. MeHg and $HgCl_2$ inhibit the action of thioredoxin (Trx), glutathione reductase (GR) thioredoxin reductase (TrxR), superoxide dismutase (SOD), while MeHg inhibit the action of glutaredoxin (Grx), glutathione peroxidase (Gpx), neuronal nitric oxide synthase (nNOS) (Carvalho et al. 2008; Farina et al. 2009; Carvalho et al. 2011; Robitaille et al. 2016).

In vivo studies revealed that methylmercury is neurotoxic and teratogenic (Stern et al. 2004; Karagas et al. 2012). Toxicogenomics and proteomics studies of tissues from whole organisms revealed that exposure to organic and inorganic mercury can change gene expression and proteins that are involved in oxidants and antioxidants defenses. Altered antioxidants enzymes include glutaredoxin (Grx), glutathione peroxidase (GPx), glutathione reductase (GR), superoxide dismutase (SOD), thioredoxin (Trx), thioredoxin reductase (TrxR), nitric oxide synthase (NOS), peroxiredoxin. Metallothioneins (MTs), genes and proteins involved in metabolism were altered in unicellular alga *Chlamydomonas Reinhardtii* (Beauvais-Flickr et al. 2017), yeast (Jin et al. 2008), macrophytes (Dranguet et al. 2017), *Caenorhabditis Elegans* (Ruda Galvyte et al. 2013), Zebra (Ho et al. 2013), pregnant mice (Robinson et al. 2010; Godefroy et al. 2012) and human (Pinheiro et al. 2008; Grotto et al. 2010).

7.5.4 Biomonitoring of Mercury

Excretion of mercury occurs within a week after exposure. It can be found in urine and faeces at low levels after some months (Goldfrank et al. 1994). Mercury levels in blood that are higher than 35 μg/dl and mercury levels in urine that are higher than 150 μg/dl are considered toxic to humans (NPIS 1996). Mercury vapour in air should not exceed 0.1 mg/m^3. A report showed that 200 mg/l of mercury in blood and 50 mg/g in hair are the established limits and a higher concentration is considered toxicity (Harada et al. 1997).

For biological monitoring of mercury exposure, the common biological samples to examine mercury exposure due to occupation are urine and blood. Hair is best known to examine environmental exposure to methylmercury (Satoh 2000). The mean urinary concentration of mercury in the US population is 0.72 μg/l (95% CI, 0.6–0.8), the mean blood concentration is 0.34 μg/l (95% CI, 0.3–0.4) (CDC 2003). Brune and colleagues (1993) reported that in Europe, the blood concentration is higher. Also, exhaled air could be a possible biomarker of exposure to elemental mercury vapour as a result of its excretion through the lungs. The assessment of methylmercury is done through the blood and scalp hair.

7.6 Remedial Actions

Reduction in metal emissions from industries and waste incinerators will help to reduce environmental and human exposure. Older fluorescent bulbs should be changed to the new energy-efficient bulbs, because the older ones contain mercury. In schools, especially in chemistry laboratories, mercury may be present or in health clinics generally where thermometers and thermostats are often used, it should be handled by experts and also with care. Planting of trees and other vegetation should be encouraged in urban areas so as to improve the quality of life. Trees help in particle uptake and hence can be helpful in lessening the toxicity of heavy metals.

7.7 Recommendations

7.7.1 New Buildings

1. Avoid the use of lead piping
2. Avoid connection of new pipeline to an old lead connection pipe
3. Avoid the use of soldiers containing lead
4. Should in case brass fittings are used, ensure that the lead content is less than 0.25%
5. Avoid inhaling dust and smokes (cigarette or automobile)
6. Parents and guardians should monitor infants and toddler and prevent them from ingesting dust from soil through hand-mouth activities.

7.7.2 Old Building

1. In the presence of lead pipes or brass containing high contents of lead, in the morning before taking a drinking water from the pipe, flush the pipework for at least 2 min.
2. Lead pipes or fittings that contain high lead contents should be removed and treated as hazardous waste.

References

Ali A, Ma Y, Reynolds J et al (2011) Chromium effects on glucose tolerance and insulin sensitivity in persons at risk for diabetes mellitus. Endocr Pract: Official J Am Coll Endocrinol Am Assoc Clin Endocrinol 17(1):16–25. https://doi.org/10.4158/EP10131.OR

Antunes SA, Appel Hort M, Culbreth M et al (2016) Methylmercury and brain development: a review of recent literature. J Trace Elem Med Biol 38:99–107

Beauvais-Flickr, Slaveykova VI, Cosio C (2017) Cellular toxicity pathways of inorganic and methyl mercury in the green microalga *Chlamydomonas Reinhardtii*. Sci Rep 7(1):8034

Bellinger DC (2013) Prenatal exposures to environmental chemicals and children's neurodevelopment: an update. Saf Health Work 4:1–11

Berntsen MH, Aatland A, Handy RD (2003) Chronic dietary mercury exposure causes oxidative stress, brain lesions, and altered behaviour in Atlantic salmon (*Salmo Salar*) parr. Aquat Toxicol 65(1):55–72

Brewers JAM, Barry PJ, MacGregor DJ (1987) Distribution and cycling of cadmium in the environment. In: Nriagu JO, Sprague JB (eds) Cadmium in the aquatic environment. Wiley, New York

Beyersmann D, Hartwig A (2008) Carcinogenic metal compounds: recent insight into molecular and cellular mechanisms. Arch Toxicol 82(8):493–512. https://doi.org/10.1007/s00204-008-0313-y

Beytoote YA, Kamiloglu NN, Aksakal M (2003) Role of dietary vitamin E in cadmium-induced oxidative damage in rabbit's blood, liver and kidneys. Int J Vitam Nutr Res 73:351–355

Borthiry GR, Antholine WE, Kalyanaraman B et al (2007) Reduction of hexavalent chromium by human cytochrome b5: generation of hydroxyl radical and superoxide. Free Radic Biol Med 42:738–755

Brand P, Gube M, Gerards K et al (2010) Internal exposure, effect monitoring, and lung function in welders after acute short-term exposure to welding fumes from different welding processes. J Occup Environ Med 52:887–892

Braune BM, Outridge PM, Fisk AT et al (2005) Persistent organic pollutants and mercury in marine biota of the Canadian Arctic: an overview of spatial and temporal trends. Sci Total Environ 351–352:4–56

Browning CL, Wise JP (2017) Prolonged exposure to particulate chromate inhibits RAD51 nuclear import mediator proteins. Toxicol Appl Pharmacol 331:101–107. https://doi.org/10.1016/j.taap.2017.05.030

Brune D, Aitio A, Nordberg G et al (1993) Normal concentration of chromium in serum and urine—a TRACY project. Scand J Work Environ Health 1:39–44

Burnett RT, Brook J, Dann T et al (2000) Association between particulate and gas-phase components of urban air pollution and daily mortality in eight Canadian cities. Inhal Toxicol 12(Suppl 4):15–39

Buttner B, Beyersmann D (1985) Modification of the erythrocyte anion carrier by chromate. Xenobiotica 15:735–741

Carvalho CM, Chew EH, Hashemy SI et al (2008) Inhibition of the human thioredoxin system. A molecular mechanism of mercury toxicity. J Biol Chem 283(18):11913–11923

Carvalho CM, Lu J, Zhang X et al (2011) Effects of selenite and chelating agents on mammalian thioredoxin reductase inhibited by mercury: implications for treatment of mercury poisoning. FASEB J 25(1):370–381

CDC (2003) Centers for Disease Control and Prevention. Second national report on human exposure to environmental chemicals, 4 March 2003. Available online at: http://www.cdc.gov/exposurereport. Accessed 30 Dec 2020

CDC (2010) Centers for Disease Control and Prevention. Guidelines for the identification and management of lead exposure in pregnant and lactating women. Atlanta. https://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf. Accessed 28 Dec 2020

Chen C, Xun P, Nishio M et al (2015) Cadmium exposure and risk of pancreatic cancer: a meta-analysis of prospective cohort studies and case-control studies among individuals without occupational exposure history. Environ Sci Pollut Res 22:17465–17474

Chen YW, Huang CF, Tsai KS et al (2006) The role of phosphoinositide 3-kinase/Akt signaling in low-dose mercury-induced mouse pancreatic beta-cell dysfunction *in vitro* and *in vivo*. Diabetes 55(6):1614–1624

Christie NC, Sugiyama O, Cattabeni M et al (1985) Differences in the effects of Hg(II) on DNA repair induced in Chinese Hamster ovary by ultraviolet or X-rays. Mol Pharmacol 24:173–178

CIBSE (Chartered Institute of Building Services Engineers) (1996) Editorial. Building Serv J 18(11):7

Claiborne CS, Larson T, Sheppard L (2002) Testing the metals hypothesis in Spokane, Washington. Environ Health Perspect 110(Suppl 4):547–552
Clarkson TW (2002) The three modern faces of mercury. Environ Health Perspect 110(Suppl. 1):11e23
Clarkson TW, Magos L, Myers GJ (2003) The toxicology of mercury–current exposures and clinical manifestations. N Engl J Med 349(18):1731–1737
Cocker J, Morton J, Warren N et al (2006) Biomonitoring for chromium and arsenic in timber treatment plant workers exposed to CCA wood preservatives. Ann Occup Hyg 50(5):517–525
Collins YE, Stotzky G (1989) Factors affecting the toxicity of heavy metals to microbes. In: Beveridge TJ, Doyle RJ (eds) Metal ions and bacteria. Wiley, Toronto, pp 31–90
Crespo-Lopez ME, Macedo GL, Pereira SI et al (2009). Mercury and human genotoxicity: critical considerations and possible molecular mechanisms. Pharmacol Res 60(4):212–220
Dranguet P, Cosio C, Le Faucheur S et al (2017) Transcriptomic approach for assessment of the impact on microalga and macrophyte of *in-situ* exposure in river sites contaminated by chlor-alkali plant effluents. Water Res 121:86–94
Drinking Water Inspectorate (DWI) (2010) Guidance on the water supply (water quality) Regulations 2000 (as amended) in England, September 2010
Dutrizac J, O'Reilly J, Macdonald R (1982) Roman lead plumbing: did it really contribute to the decline and fall of the empire? CIM Bull M75:111–115
Edwards KC, Kim H, Vincent JB (2019) Release of trivalent chromium from serum transferrin is sufficiently rapid to be physiologically relevant. J Inorg Biochem 202:110901. https://doi.org/10.1016/j.jinorgbio.2019.110901
Elinder C-G, Friberg L, Kjellstrom T et al (1994) Biological monitoring of metals. Chemical Safety Monographs, IPCS. World Health Organization, Geneva
Fang Z, Zhao M, Zhen H et al (2014) Genotoxicity of tri- and hexavalent chromium compounds *in vivo* and their modes of action on DNA damage in vitro. PLoS ONE 9(8):e103194. https://doi.org/10.1371/journal.pone.0103194
Farina M, Campos F, Vendrell I et al (2009) Probucol increases glutathione peroxidase-1 activity and displays long-lasting protection against methylmercury toxicity in cerebellar granule cells. Toxicol Sci 112(2):416–426
Farina M, Aschner M, Rocha JB (2011) Oxidative stress in MeHg-induced neurotoxicity. Toxicol Appl Pharmacol 256(3):405–417
Feki-Tounsi M, Hamza-Chaffai A (2014) Cadmium as a possible cause of bladder cancer: a review of accumulated evidence. Environ Sci Pollut Res 18:10561–10573
Fujimura M, Usuki F, Sawada M (2009) Methylmercury exposure downregulates the expression of *Racl* and leads to neuritic degeneration and ultimately apoptosis in cerebrocortical neurons. Neurotoxicology (Little Rock) 30(1):16–22
Gadhia SR, Calabro AR, Barile FA (2012) Trace metals alter DNA repair and histone modification pathways concurrently in mouse embryonic stem cells. Toxicol Lett 212(2):169–179
Gallagher CM, Smith DM, Meliker JR (2011) Total blood mercury and serum measles antibodies in US children, NHANES 2003–2004. Sci Total Environ 410–411:65–71
Garrod ANI, Martinez M, Pearson J et al (1999) Exposure to preservatives used in the industrial pretreatment of timber. Ann Occup Hyg 43:543–555
Giaginis C, Gatzidou E, Theocharis S (2006) DNA repair systems as targets of cadmium toxicity. Toxicol Appl Pharmacol 213:282–290
Giel-Pietraszuk M, Hybza K, Chełchowska M et al (2012) Mechanisms of lead toxicity. Adv Cell Biol 39:17–248
Godefroy D, Gosselin RD, Yasutake A et al (2012) The chemokine CCL2 protects against methylmercury neurotoxicity. Toxicol Sci 125(1):209–218
Goldfrank FL, Flomenbaum NE, Lewin NA et al (1994) Toxicologic emergencies. Appleton and Lange, Norwalk, CT
Grotto D, Valentini J, Fillion M et al (2010) Mercury exposure and oxidative stress in communities of the Brazilian Amazon. Sci Total Environ 408(4):806–811

Gube M, Brand P, Schettgen T et al (2013) Experimental exposure of healthy subjects with emissions from a gas metal arc welding process–part II: biomonitoring of chromium and nickel. Int Arch Occup Environ Health 86:31–37

Ha E, Basu N, Bose-O'Reilly S et al (2017) Current progress on understanding the impact of mercury on human health. Environ Res 152:419–433

Hagino N, Yoshioka Y (1960) A study of the etiology of Itai-Itai disease. J Jpn Orthop Assoc 35:812–815

Harada M, Nakachi S, Cheu T et al (1997) Mercury pollution in Tanzania: relation between scalp hair, mercury level and health. Institute of Molecular Embryology and Genetics, Kumamoto, Japan, p 14

Hayat MT, Nauman M, Nazir N et al (2019) Environmental hazards of cadmium: past, present, and future. Cadmium toxicity and tolerance in plants. Academic Press, Cambridge, MA, USA, pp 163–183

Hayes CR, Skubala ND (2009) Is there still a problem with lead in drinking water in the European Union? J Water Health 4:569–580

Hayes CR, Croft TN (2012) An investigation into the representativeness of random daytime sampling for lead in drinking water, using computational modelling. J Water Supply Res Technol-Aqua 61:142–152. https://doi.org/10.2166/aqua.2012.092

Ho NY, Yang L, Legradi J et al (2013) Gene responses in the central nervous system of zebrafish embryos exposed to the neurotoxicant methyl mercury. Environ Sci Technol 47(7):3316–3325

Hodge A (1981) Vitruvius, lead pipes and lead poisoning. Am J Archaeol 85:486–491

International Agency for Research on Cancer (IARC) (1990) Chromium, nickel and welding. Monographs Evaluat Carcinogenic Risks Humans 49:1–648. https://publications.iarc.fr/67

International Agency for Research on Cancer (IARC) (1993) Monographs on the evaluation of the carcinogenic risks to humans. Beryllium, cadmium, mercury and exposures in the glass manufacturing industry. IARC Scientific Publications, Lyon, France, pp 119–238

International Water Association (2010) In: Hayes CR (ed) Best practice guide on the control of lead in drinking water. IWA Publishing, London

Ishikawa Y, Nakagawa K, Satoh Y et al (1994) "Hot spots" of chromium accumulation at bifurcations of chromate workers bronchi. Cancer Res 54(9):2342–2346. PubMed: 8162579. https://cancerres.aacrjournals.org/content/canres/54/9/2342.full.pdf. Accessed 30 Dec 2020

Jin YH, Dunlap PE, McBride SJ et al (2008) Global transcriptome and deletome profiles of yeast exposed to transition metals. PLoS Genet 4(4):e1000053

Johnson J, Schewel L, Graedel TE (2006) The contemporary anthropogenic chromium cycle. Environ Sci Technol 40:7060–7069

Julin B, Wolk A, Johansson E et al (2012) Dietary cadmium exposure and prostate cancer incidence: a population-based prospective cohort study. Br J Cancer 107:895–900

Jusko TA, Henderson CR Jr, Lanphear BP et al (2008) Blood lead concentrations <10 microg/dL and child intelligence at 6 years of age. Environ Health Perspect 116(2):243–248

Karagas MR, Choi AL, Oken E et al (2012) Evidence on the human health effects of low level methylmercury exposure. Environ Health Perspect 120(6):799–806

Khalil N, Wilson J, Talbott E et al (2009) Association of blood lead concentrations with mortality in older women: a prospective cohort study. Environ Health 8:15

Krzywy I, Krzywy E, Pastuszak-Gabinowska MA et al (2010) Lead—is there something to be afraid of? Ann Acad Med Stetin 56:118–128

Kumar S, Sharma A (2019) Cadmium toxicity: effects on human reproduction and fertility. Rev Environ Health 34:327–338

Lamborg CH, Hammerschmidt CR, Bowman KL et al (2014) A global ocean inventory of anthropogenic mercury based on water column measurements. Nature 512(7512):65–68

Lanphear BP, Dietrich K, Auinger P et al (2000) Cognitive deficits associated with blood lead concentrations <10 microg/dL in US children and adolescents. Public Health Rep 115(6):521–529

Levina A, Harris HH, Lay PA (2006) Binding of chromium (VI) to histones: implications for chromium (VI)-induced genotoxicity. J Biol Inorg Chem 11(2):225–234. https://doi.org/10.1007/s00775-005-0068-3
Lokhande B, Patil PS, Uplane MD (2004) Studies on cadmium oxide sprayed thin films deposited through non-aqueous medium. Mater Chem Phys 84:238–242
Menke A, Muntner P, Batuman V et al (2006) Blood lead below 0.48 μmol/l (10 μg/dl) and mortality among US adults. Circulation 114:1388–1394
Mergler D, Anderson HA, Chan LH et al (2007) Methylmercury exposure and health effects in humans: a worldwide concern. Ambio 36(1):3–11
Minoia C, Cavalieri A (1988) Chromium in urine, serum and red blood cells in the biological monitoring of workers exposed to different chromium valence states. Sci Total Environ 71:323–327
Mona T, Heba MA, Eman S et al (2018) Impact of occupational cadmium exposure on bone in sewage workers. Int J Occup Environ Health 24:101–108
Mutti A, Pedroni C, Arfini G et al (1984) Biological monitoring of occupational exposure to different chromium compounds at various valencystates. Int J Environ Anal Chem 17:35–41
Navas-Acien A, Guallar E, Silbergeld EK et al (2007) Lead exposure and cardiovascular disease—a systematic review. Environ Health Perspect 115:472–482
Nawrot TS, Martens DS, Hara A et al (2015) Association of total cancer and lung cancer with environmental exposure to cadmium: the meta-analytical evidence. Cancer Causes Control 26:1281–1288
NPIS (L) (1996) Monograph: mercury—organic and inorganic. National poison information Service (London) Medical Toxicology Unit, Guy's and St Thomas' Hospital, London
Nriagu J (1983) Saturnine gout among Roman aristocrats. Did lead poisoning contribute to the fall of the empire? N Engl J Med 308:660–663
Occupational Safety and Health Administration (OSHA) (2006) Department of labor. Occupational exposure to hexavalent chromium. Final rule. Fed Reg 71:10099–10385
Ognjanovic BI, Pavlovic SZ, Maletic SD et al (2003) Protective influence of vitamin E on antioxidant defense system in the blood of rats treated with cadmium. Physiol Res 52:563–570
Omeljaniuk W, Socha K, Soroczynska J et al (2018) Cadmium and lead in women who miscarried. Clin Lab 64:59–67
Osornio-Vargas A, Bonner JC, Alfaro-Moreno E et al (2003) Proinflammatory and cytotoxic effects of Mexico City air pollution particulate matter *in vitro* are dependent on particle size and composition. Environ Health Perspect 111(10):1289–1293
Pieper I, Waha CA, Bornhorst J et al (2014) Mechanisms of Hg species induced toxicity in cultured human astrocytes: genotoxicity and DNA-damage response. Metall 6(3):662–671
Pinheiro MC, Macchi BM, Vieira JL et al (2008) Mercury exposure and antioxidant defenses in women: a comparative study in the Amazon. Environ Res 107(1):53–59
Polunas M, Halladay A, Tjalkens RB et al (2011) Role of oxidative stress and the mitochondrial permeability transition in methylmercury cytotoxicity. Neurotoxicology (Little Rock) 32(5):526–534
Proctor DM, Suh M, Campleman SL et al (2014) Assessment of the mode of action for hexavalent chromium-induced lung cancer following inhalation exposures. Toxicology 325:160–179. https://doi.org/10.1016/j.tox.2014.08.009
Rani A, Kumar A, Lal A et al (2014) Cellular mechanisms of cadmium-induced toxicity: a review. Int Environ Health Res 24:378–399
Rehman K, Fatima F, Waheed I (2018) Prevalence of exposure of heavy metals and their impact on health consequences. J Cell Biochem 119:157–184
Robinson JF, Griffith WC, Yu X et al (2010) Methylmercury induced toxicogenomic response in C57 and SWV mouse embryos undergoing neural tube closure. Reprod Toxicol 30(2):284–291
Robitaille S, Mailloux RJ, Chan HM (2016) Methylmercury alters glutathione homeostasis by inhibiting glutaredoxin 1 and enhancing glutathione biosynthesis in cultured human astrocytoma cells. Toxicol Lett 256:1–10

Ruda Galvyte M, VanDuyn N, Aarnio V et al (2013) Methylmercury exposure increases lipocalin related (lpr) and decreases activated in blocked unfolded protein response (abu) genes and specific miRNAs in Caenorhabditis Elegans. Toxicol Lett 222(2):189–196

Ryu TH, An KG, Kim JK (2014) Genotoxicity in earthworm after combined treatment of ionising radiation and mercury. Radiat Prot Dosim 159(1–4):111–117

Satoh H (2000) Occupational and environmental toxicology of mercury and its compounds. Ind Health 38:153–164

Schwarz K, Mertz W (1959) Chromium (III) and the glucose tolerance factor. Arch Biochem Biophys 85:292–295. https://doi.org/10.1016/0003-9861(59)90479-5

Seervarisai J, Kao Jarern S, Chanprasertyothin S et al (2015) Environmental lead exposure, catalase gene, and markers of antioxidant and oxidative stress relation to hypertension: an analysis based on the EGAT study. Biomed Res Int 2015: https://doi.org/10.1155/2015/856319

Söderholm M, Borné Y, Hedblad B et al (2020) Blood cadmium concentration and risk of subarachnoid haemorrhage. Environ Res 180:

Song JK, Luo H, Yin XH et al (2015) Association between cadmium exposure and renal cancer risk: a meta-analysis of observational studies. Sci Rep 5:17976

Sorensen M, Schins RPF, Hertel et al (2005) Transition metals in personal samples of PM2.5 and oxidative stress in human volunteers. Cancer Epidemiol Biomark Prevent 14(5):1340–1343

Staessen JA, Buchet JP, Lauwerys RR et al (1996) on behalf of the working Groups (1996) Public health implications of environmental exposure to cadmium and lead: an overview of epidemiological studies in Belgium. J Cardiovasc Risk 3:26–41

Steenland K, Barry V, Anttila A et al (2019) Cancer incidence among workers with blood lead measurements in two countries. Occup Environ Med 76:603–610

Stern AH, Jacobson JL, Ryan L et al (2004) Do recent data from the Seychelles Islands alter the conclusions of the NRC Report on the toxicological effects of methylmercury? Environ Health 3(1):2

Swartzendruber PJD (2012) Mercury in the environment pattern and process. University of California Press, London, England

Takiguchi M, Achanzar WE, Qu W et al (2003) Effects of cadmium on DNA-(Cytosine-5) methyltransferase activity and DNA methylation status during cadmium-induced cellular transformation. Exp Cell Res 286:355–365

Teklay A (2016) Physiological effect of chromium exposure: a review. Int J Food Sci Nutr Dietet S7(001):1–11. https://doi.org/10.19070/2326-3350-SI07001

Troesken W (2006) The great lead water pipe disaster. MIT Press, Cambridge, MA

US EPA (US, Environmental Protection Agency) (1994) Indoor air pollution: an introduction for health professionals. Washington, DC: US Environmental Protection Agency

Van den Hoven TJL, Buijs PJ, Jackson PJ et al (1999) Developing a new protocol for the monitoring of lead in drinking water. EUR 19087. https://op.europa.eu/en/publication-detail/-/publication/318e84bf-1029-46ed-a49a-a5b9156ad1aa. Accessed 31 Dec 2020

Vangronsveld J, Clijsters H (1994) Toxic effect of metals. In: Farago MG (ed) Plant and the chemical elements. VCH, Weinheim, New York, Basel, Cambridge, Tokyo, pp 149–177

Vincent JB (2017) New evidence against chromium as an essential trace element. J Nutr 147:2212–2219. https://doi.org/10.3945/jn.117.255901

Vincent JB (2019) Effects of chromium supplementation on body composition, human and animal health, and insulin and glucose metabolism. Curr Opin Clini Nutr Metabolic Care 22(6):483–489. https://doi.org/10.1097/MCO.0000000000000604

Vincent R, Kumarathasan P, Goegan P et al (2001) Acute cardiovascular effects in rats from exposure to urban ambient particles. Health Effects Inst Res Rep 104:1–64

Wallace DR, Taalab YM, Heinze S et al (2020) Toxic-metal-induced alteration in miRNA expression profile as a proposed mechanism for disease development. Cells 9:901

Wang Y, Su H, Gu Y et al (2017) Carcinogenicity of chromium and chemoprevention: a brief update. Onco Targets Thera 10:4065–4079. https://doi.org/10.2147/OTT.S139262

Wani AL, Ara A, Usmani JA (2015) Lead toxicity: a review. Int Toxicol 8:55–64

Welling R, Beaumont JJ, Petersen SJ et al (2015) Chromium VI and stomach cancer: a meta-analysis of the current epidemiological evidence. Occup Environ Med 72(2):151–159. https://doi.org/10.1136/oemed-2014-102178

WHO (1992) World Health Organization. Environmental health criteria 134: Cadmium; World Health Organization: Geneva, Switzerland

WHO (2007) Preventing disease through a healthy environment: exposure to mercury: a major public health concern. World Health Organization Mercury Flyer

WHO (2016) Expert consultation. Available evidence for the future update of the WHO global Air Quality Guidelines (AQGs) (2016).https://www.euro.who.int/__data/assets/pdf_file/0013/301720/Evidence-future-update-AQGs-mtg-report-Bonn-sept-oct-15.pdf. Accessed 28 Dec 2020

Xia B, Ren X, Zhuang Z et al (2014) Effect of hexavalent chromium on histone biotinylation in human bronchial epithelial cells. Toxicol Lett 228(3):241–247. https://doi.org/10.1016/j.toxlet.2014.05.010

Xia W, Hu J, Zhang B et al (2016) A case-control study of maternal exposure to chromium and infant low birth weight in China. Chemosphere 144:1484–1489. https://doi.org/10.1016/j.chemosphere.2015.10.006

Yin Z, Milatovic D, Aschner JL (2007) Methylmercury induces oxidative injury, alterations in permeability and glutamine transport in cultured astrocytes. Brain Res 1131(1):1–10

Zawadzki M, Poręba R, Gac P (2006) Mechanisms and toxic effects of lead on the cardiovascular system. Med Pr 57:543–549

Zhitkovich A (2002) Chromium: Exposure, toxicity and biomonitoring approaches. In: Wilson SH, Suk WA (eds) Biomarkers of environmentally associated disease: technologies, concepts, and perspectives. CRC Press, New York, pp 269–287