



Recycling used lead-acid batteries: health considerations



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1. Introduction

Around 85% of the total global consumption of lead is for the production of leadacid batteries (ILA, 2017) Approximately 85% of the total global consumption of lead is for the production of lead-acid batteries (ILA, 2017). This represents a fast-growing market, especially in Asia (Future Market Insights, 2014). The main uses of these batteries are in motorized vehicles, for storage of energy generated by photovoltaic cells and wind turbines, and for back-up power supplies (for both the consumer market and for critical systems such as telecommunications and hospitals). In developing countries where power supplies are unreliable, lead-acid batteries are used domestically for lighting and electrical appliances (UNEP, 2004). The growth in the use of renewable energy sources and the concomitant need for storage batteries, as well as the increasing demand for motor vehicles as countries undergo economic development, mean that the demand for lead-acid batteries will continue to increase. This is reflected in the increased global demand for refined lead metal, which was estimated at 10.83 million tonnes in 2016 (International Metals Study Groups, 2016). The demand is being met by increases in both primary lead production from mines and recycling. Indeed, currently over half of the global production of lead is from lead recycling (ILA, 2015).

The manufacturing and recycling of lead-acid batteries is practised worldwide in both regulated industries and unregulated, informal establishments (UNEP, 2003). Lead recycling is an important source of environmental contamination and human exposure in many countries (UNEP, 2010; van der Kuijp et al., 2013). This is because it is frequently carried out without the necessary processes and technologies to control lead emissions and, in many developing countries, is a poorly regulated industry (UNEP, 2010; Manhart et al., 2016). The unregulated, informal recycling of used lead-acid batteries presents particular problems as it is mainly carried out by small family businesses, often in domestic backyards, and sometimes in secret (UNEP, 2004; Belay et al., 2015; AGENDA, 2016). Even established, industrial-scale recycling facilities can, however, cause significant environmental contamination and human exposure to lead in countries without adequate standards or when regulatory controls are inadequately enforced (California Environmental Protection Agency, 2015).

Recycling used lead-acid batteries is of public health concern because this industry is associated with a high level of occupational exposure and environmental emissions. Furthermore, there is no known safe level of exposure to lead, and the health impacts of lead exposure are significant. Based on 2016 data, it is estimated that lead exposure accounted for 495 550 deaths and 9.3 million disability-adjusted life years (DALYs) lost due to long term impacts on health, with the highest burden in low- and middle-income countries (IHME, 2016). Young children and women of childbearing age are particularly vulnerable to exposure to, and the toxic effects of, lead.

1.1. Purpose and scope of the document

This document aims to inform the health sector about this issue so it can recognize the recycling of lead-acid batteries as a source of lead exposure and can advocate for this practice to be better controlled and regulated. It also aims to inform policy-makers of the burden of disease associated with lead exposure as a stimulus to introducing and enforcing control measures.

The document outlines the process of recycling used lead-acid batteries and describes how lead exposure can occur. Three case studies illustrate the impact that uncontrolled battery recycling can have on a community. The document then discusses the adverse health impacts resulting from exposure to lead. An overview is given on methods for assessing lead exposure through measurement of blood lead concentrations and environmental sampling. This document does not aim to provide technical details of environmental sampling methods but refers the reader to other sources. Finally, brief information is provided on control measures to prevent the release of lead during recycling.

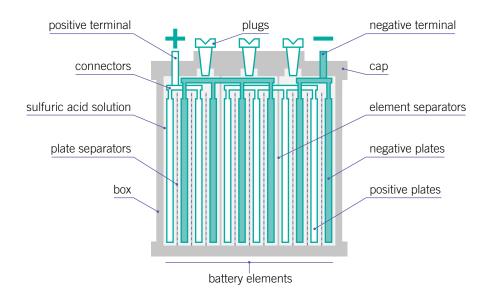
2. How lead exposure occurs during recycling and disposal

The main pathways of exposure to lead from recycling used lead-acid batteries arise from environmental emissions. These occur at various stages in the recycling process, as described below. Lead particles and fumes emitted into the air can be inhaled and are also deposited onto soil, water bodies and other surfaces, including in gardens and homes. Waste materials from lead processing can, if not treated and correctly disposed of, contaminate land and water bodies. Used acid with high concentrations of lead is often dumped on land or released into waterways. Lead can enter the food chain through crops growing on contaminated land, from direct deposition onto crops, through food animals foraging in contaminated areas and consuming lead particles, and from fish and shellfish living in lead-contaminated water (UNEP, 2003; UNEP, 2010).

2.1. Components of a lead-acid battery

A lead-acid battery is made up of the following components, enclosed within a plastic or ebonite box or casing (see Figure 1) (UNEP, 2003). There are positive and negative terminals made of lead, which provide the connection points to external devices. There are sets of positive and negative plates kept apart by plate separators - porous sheets of PVC or polyethylene plastic, glass microfibre, or phenolic resins that allow the free movement of the ions in the electrolyte solution. The positive plates are grids made of lead or lead alloy coated with porous metallic lead paste, and the negative plates are lead grids coated with lead dioxide paste. A series of negative and positive plates plus separators makes up a battery element, and the battery elements are separated by plates of the same material as the battery box. The elements are bathed in a sulfuric acid electrolyte solution, which can be topped up via the plugs. In sealed batteries the electrolyte is either a gel or is soaked onto glass microfibre separators.

Figure 1. Components and structure of a lead-acid battery (UNEP, 2003)



The average amount of lead in automotive batteries can range from 2 to 13 kg, depending on the size of the vehicle (CEC, 2016).

2.2. Steps in the recycling process

Almost all parts of a lead-acid battery can be recycled. The main steps in the recycling process are as follows:

- collection and transportation of the batteries to a recycling facility
- separation of the component parts of the batteries
- smelting and refining of the lead components
- washing then shredding or melting of the plastic components
- purification and treatment of the sulfuric acid electrolyte
- treatment and disposal of waste

The batteries are mechanically or manually broken up to separate out the acid and component parts. The lead components are conveyed to the furnace for smelting. After smelting the slag is removed and the molten unrefined lead may be poured into moulds and cooled or it may immediately be directed to a holding kettle (cast-iron pot) to keep it molten prior to refining. The aim of the refining process is to produce lead of high purity or to produce alloys (requiring the addition of specific trace elements to the refining kettle) that can be used to make a new lead battery. The molten lead is then cast into moulds and allowed to cool (OSHA, 2002).

Recyclable plastic components are washed then shredded or ground and melted. The molten plastic is extruded into pellets, which can then be used

Almost all parts of a lead-acid battery can be recycled

in the manufacture of other plastic goods, including new battery casings (ILA, 2015). In many smaller recycling facilities in low- and middle-income countries, the plastic battery cases are often not recycled and may be dumped or burned.

The electrolyte may be recovered for re-use or neutralized with alkali and treated to remove lead and other contaminants before being released into the sewage system. Alternatively the solution may be purified and sodium sulfate extracted for use in making detergents and other products (UNEP, 2003; ILA, 2015).

Automated, enclosed processes with pollution control devices are necessary to reduce lead emissions

At each of these stages, lead fumes and dust are released into the air (see section 2.3), contaminating both the workplace and the wider environment. The use of automated, enclosed processes with pollution control devices can reduce these emissions.

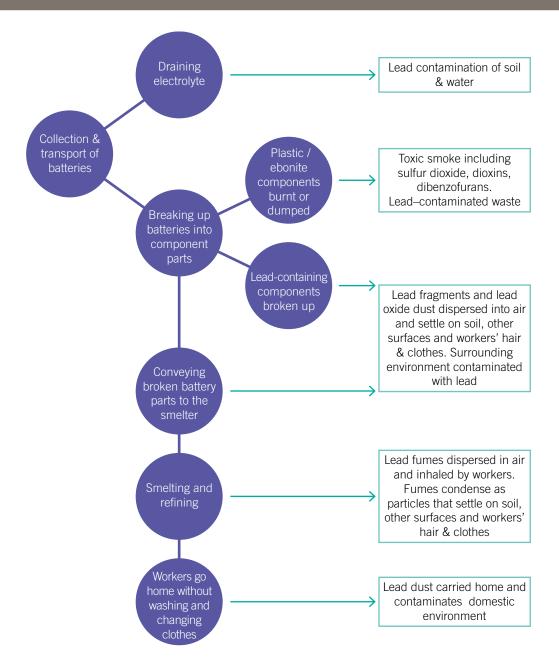
In a typical automated enclosed process, the lead batteries are broken up in a hammermill or shredder and the pieces are fed into tanks filled with water. Here gravity is used to separate the components: the lead and heavy materials sink to the bottom and the plastics rise to the top. The plastic materials are skimmed away and the liquid, including the sulfuric acid electrolyte is drawn off. The metallic components are channelled to closed furnaces for smelting and refining and then piped into casting moulds (OSHA, 2002; UNEP, 2003). Waste from recycling is collected, treated and disposed of at a designated waste disposal site (UNEP, 2003).

In a manual process the batteries are drained then broken up with electric saws, machetes or axes. The components are separated by hand into piles. The lead components are carried to the furnace or taken on an open conveyer belt. The furnace may, in the worst case, be no more than an open pot on a fire (UNEP, 2003; UNEP, 2004; Manhart & Schleicher, 2015). The molten lead is then poured into casting moulds.

2.3. Lead release and exposure during recycling

At the collection and transportation stage, the sulfuric acid electrolyte solution is sometimes drained out to reduce the weight of the batteries or because a higher price is offered for drained batteries (Manhart & Schleicher, 2015). If not done at this stage then the electrolyte may be drained out at the recycling site (in some enclosed processes the batteries are not drained prior to crushing). In addition, electrolyte may leak out of damaged batteries during storage and transportation (UNEP, 2003). If adequate precautions are not taken to avoid skin contact, the acid will cause corrosive injury. The electrolyte contains dissolved lead and, if the electrolyte leaks out or is poured onto the ground rather than into collection tanks, the lead becomes incorporated into soil particles, which subsequently become a source of lead dust (UNEP, 2003). Pouring the electrolyte into ponds or streams will contaminate water that may be used for drinking, fishing and cooking.

Figure 2. Schematic illustrating points at which lead is released during battery recycling



Manually breaking up the batteries releases lead particles and lead oxide dust, which are a source of lead exposure to the worker (Suplido & Ong, 2000; UNEP, 2003). The dust and particles also settle in the surrounding soil and may be blown to more distant areas, contaminating the wider environment and becoming a source of exposure to the community (UNEP, 2003; Haefliger et al., 2009). Hammermills and shredders may release lead mist, which can dry and release lead dust if disturbed. Dust settled on vibrating equipment can become re-suspended in air and inhaled (OSHA, 2002).

Manual recycling methods release large amounts of lead into the environment

During the separation process, water used in automated systems for separating lead from other components becomes heavily contaminated with lead compounds. If this leaks or is not treated before disposal it will contaminate the ground or soil. As this water evaporates it leaves a residue of fine lead dust that may then be dispersed by wind (UNEP, 2003).

When lead components are moved around the recycling site, e.g. on open conveyor belts or in wheelbarrows, and when they are shovelled into the furnace, lead fragments and dust are released.

The temperatures used for refining lead can be up to 1000 °C, which generates large amounts of lead fume. If the furnace is not under negative pressure or if the plant has inadequate ventilation and/or emission controls, the fumes will be inhaled by workers (UNEP, 2003). Lead fumes are particularly hazardous as the small particle size enables the lead to be inhaled into the lower respiratory tract and absorbed (ATSDR, 2007). The fumes will eventually settle as lead particles on surrounding surfaces and the soil, creating lead dust, which can also be inhaled. Fugitive lead emissions from these sources can be substantial and are more difficult to control. Sometimes ash from the smelting process is manually sifted to retrieve metal particles, dispersing lead-contaminated dust into the air (Paddock, 2016)

Washing oneself and changing clothes before returning home protects family members from lead exposure Fume, lead particles and dust released at various stages in the recycling process will also settle on the skin, hair and clothes of workers. If workers do not wash and change clothes before returning home this lead becomes a source of take-home exposure to household members and even, potentially, to the wider community (Daniell et al., 2015). Lead poisoning in the spouses and children of lead workers, caused by transfer of lead from the workplace to home, has repeatedly been documented (Baker et al., 1977; Chisolm, 1978).

2.3.1. Informal lead recycling

Non-regulated, informal ("backyard" or "cottage") recycling practices occur in many countries and have resulted in lead exposure and poisoning, with young children being particularly at risk (Matte, 1991; Suplido & Ong, 2000; Haefliger et al., 2009; van der Kuijp et al., 2013; Daniell et al., 2015). This practice is sometimes carried out in urban areas with high population densities, meaning that a recycling operation has the potential to affect a large number of people (Haefliger et al., 2009). There are few (if any) pollution controls. Lead-containing waste products, such as electrolyte solution and slag from the smelting process are often simply dumped, although slag may also be sold on for further smelting. The work may be carried out by small family groups around the home. Children often assist with dismantling the batteries and washing components (van der Kuijp et al., 2013). Because the recycling process is done with little knowledge of the toxicity of lead, and is conducted under poor conditions of safety, health and environmental controls, informal recycling is particularly likely to result

in environmental contamination and human exposure (UNEP, 2004; van der Kuijp et al., 2013; Daniell et al., 2015).

Soil contaminated with lead compounds can spread throughout the community and be tracked into homes. If recycling activities take place around the home, then airborne lead can enter the home and accumulate on the floor, on beds and on other furniture (Haefliger et al., 2009). Settled dust can be re-suspended in the air and inhaled as people walk through or brush up the dust. Young children, who spend large amounts of time on the ground and who frequently put their hands and other objects in their mouths, are at particularly high risk of lead exposure in these settings.

Informal recycling is particularly likely to cause environmental contamination and human exposure to lead

If the plastic components are inadequately washed before re-use for other products, then these products will be contaminated with lead (Manhart & Schleicher 2015). Battery casings may be used around the home as a construction material or as containers, again introducing the possibility of lead contamination (Daniell et al., 2015).

There have been reports in Cameroon and other countries of lead scrap from informal recycling being mixed with scrap aluminium to make cooking pots (CREPD, 2015). Lead can leach out into food being prepared or stored in these pots (Weidenhamer et al., 2014; Weidenhamer et al., 2017).

2.4. Other chemicals released during recycling

While the focus of this document is on the release of lead, there are a number of other hazardous chemicals that can be released during recycling. In addition to the lead terminals and plates, batteries contain various plastics or hard rubber (ebonite) and the sulfuric acid electrolyte solution. The lead components may contain other elements such as arsenic, antimony, barium and cadmium (UNEP, 2003). These substances may form part of the waste and emissions generated at various stages of the recycling

process. The rubber and plastic components may be burned rather than recycled, producing toxic gases, including sulphur dioxide, chlorine, dioxins and dibenzofurans (UNEP, 2003).

2.5. Studies of lead exposure from recycling lead-acid batteries

Recycling lead-acid batteries can result in significant occupational exposure to lead. Were et al. (2012) investigated a lead-acid battery recycling plant in Kenya and found elevated concentrations of lead in the air and elevated blood lead concentrations in workers. The study identified a number of weaknesses in work practices and control measures that resulted in excessive exposure

to lead. Examples included inadequate engineering controls (including poor ventilation systems) and inadequate personal hygiene measures (including lack of respiratory protection and failure to use washing facilities). Among workers involved in recycling lead-acid batteries there is often poor general awareness of the hazards of lead (CREPD, 2015).

A review of published literature on exposures from formal-sector lead-acid battery manufacturing and recycling plants in developing countries found that seriously elevated blood and airborne lead concentrations were common (Gottesfeld & Pokhrel, 2011). In workers involved in battery recycling the arithmetic mean blood lead concentration was 64 μ g/dL, with a range of 37.7 to 112.5 μ g/dL. This contrasted with data from developed countries where few workers had blood lead concentrations above 50 μ g/dL and most were below 25 μ g/dL (though even these concentrations are likely to result in adverse health effects – see section 3.2). The review also found that high airborne lead concentrations were reported in recycling facilities, with a mean value of 367 μ g/m³. This is 7 times higher than the permissible exposure limit of 50 μ g/m³ as an 8-hour time-weighted average (TWA) adopted in the USA (OSHA, 1978).

Communities living near recycling facilities are at risk of exposure to lead, and extensive contamination of soil surrounding many formal sector recycling plants has been reported (Levallois et al., 1991; Wang et al., 1992; Zhang et al., 2016). In their review, Gottesfeld & Pokhrel (2011) summarized 11 studies in seven countries on lead exposure of children residing near lead battery manufacturing and recycling facilities and reported an average blood lead concentration of 29 μ g/dL, with values up to 71 μ g/dL. Recently a large recycling plant in the USA was closed down after it failed to meet emission controls and waste management standards. This plant was found to have contaminated the surrounding area with lead to a distance of 1.7 miles (California Environmental Protection Agency, 2015).

The case studies given below illustrate how environmental contamination caused by the recycling of used lead-acid batteries can result in severe lead poisoning in a community, which may continue even after recycling operations have stopped. The first case describes exposure to lead via reclamation of lead and lead compounds from discarded batteries, and the health consequences. The other two case studies show that closing down or moving battery recycling operations are not sufficient measures on their own to prevent human lead exposure.

2.5.1. Senegal

Between November 2007 and March 2008, 18 children died from an aggressive central nervous system disease of unexplained origin in a neighbourhood of Dakar in Senegal (Haefliger et al., 2009). One of the possibilities considered was lead intoxication, as the mothers of some of the children were engaged

in the recycling of used lead-acid batteries. Informal lead recycling in the region had been taking place since 1995 and various lead compounds had accumulated in the sandy soil over time. Around October 2007, some local residents realised that the accumulated lead in the soil could be sieved and sold. They therefore started to collect lead-enriched soil in sacks, which they brought into the community, sometimes even inside their homes.

An investigative mission was sent to work with the local health authorities in investigating the deaths. For cultural reasons it was not possible to conduct autopsies and post-mortem testing on the children who had died and, therefore, the mission team focused their investigation on the siblings and mothers of the children. Another group of children and adults, who were living in the same community but apparently unrelated to the deceased children, were also investigated to evaluate the extent of lead intoxication in the area. In total 81 individuals were examined and tested and all were found to be poisoned, often severely, with lead. In the children blood lead concentrations ranging from 39.8 μ g/dL to 613.9 μ g/dL were found (levels above 45 μ g/dL indicate potentially serious poisoning).

Environmental investigations found that homes and soil were heavily contaminated with lead. Lead concentrations in outdoor soil were up to 302 000 mg/kg and indoor concentrations were as high as 14 000 mg/kg. For comparison, the US EPA standard for soil in a children's play area is 400 mg/kg and for other residential areas it is 1200 mg/kg (US EPA, 2001). The exposure pathway was most likely via inhalation and/or ingestion of the contaminated soil and dust in suspension as young children were playing on contaminated ground. This indicated that other inhabitants of the affected area (about 940 people, of whom 460 were children and adolescents) might also be poisoned with lead. While the causes of death of the 18 children could not be confirmed, circumstantial evidence, including heavy environmental contamination and the high blood lead concentrations in siblings suggest that most, if not all, of the children died because of encephalopathy as a result of severe lead poisoning. To prevent further exposures the homes were cleaned and contaminated soil was removed and replaced with cleaner soil. A public awareness campaign was also carried out to encourage a change in recycling practices.

2.5.2. Dominican Republic

In Haina, Dominican Republic, a lead screening survey was carried out in $116 \text{ children living close to a lead-acid battery recycling smelter (Kaul & Mukerjee, 1999). Very high blood lead concentrations were found with a mean value of <math>71 \mu \text{g/dL}$. Shortly afterwards the government closed down the recycling plant.

Six months later a follow-up survey was conducted in 146 lead-poisoned children in the same community (Kaul et al., 1999). This found that although the blood

lead concentrations had reduced significantly, with a mean of 32 µg/dL (range 6 to 130 µg/dL), they were nevertheless still high. Only 9% of the children had blood lead concentrations below 10 µg/dL and 28% of the children had blood lead concentrations above 40 µg/dL. For comparison, a survey was also carried out in 63 children living in a nearby community, which had similar demographic characteristics but no smelter. Here the mean blood lead concentration was 14 μ g/dL (range 20 to 99 μ g/dL) and 42% of children had levels <10 μ g/dL.

An environmental assessment found that, although the smelter had shut down, metallic scrap and mixed residual soil and solid materials were still scattered around. Some clean-up activities had begun during the time of visit; however, an assortment of waste materials remained at the site and continued to be a hazard to the neighbourhood. The authors concluded that although closing the battery recycling facility significantly lowered blood lead concentrations of children, the children were still exposed to lead through their environment (Kaul et al., 1999).

In 2008 and 2009 some remediation activities were carried out to remove contaminated soil and educational sessions were given to local children to help minimize their exposure to lead dust and materials (Blacksmith Institute, 2009).

2.5.3. Viet Nam

Dong Mai village in northern Viet Nam has been a centre for recycling lead-acid batteries since the 1980s. A study

> and efforts were subsequently made to move household recycling operations to an industrial zone 1 km outside the village. The zoning

> > was formalised by 2010, though some home-based recycling continued in a few households. A study was subsequently carried out over a one-year period from December 2011 to assess lead exposure in children and environmental levels of lead (Daniell et al., 2015). All 109 children tested were found to have elevated blood lead concentrations, ranging from 12 to >65 µg/dL, with 28% having concentrations >45 µg/dL. The blood samples were measured using the LeadCare analyser, and a sample of the higher results were rechecked using laboratory analysis of venous samples. This generally gave lower



values, but of 24 retested samples 80% were still above 45 μ g/dL. Higher blood lead concentrations were associated with home recycling or parents currently involved in recycling in the industrial zone.

Environmental investigations showed legacy soil contamination in areas where recycling had previously been carried out, with a mean lead concentration of 2500 mg/kg. In other areas soil levels were lower though still high, at a mean of 1000 mg/kg. By contrast, soil lead concentrations at the school were very low with a mean of 34 mg/kg.

Surface lead contamination was tested in 11 homes and a mean value of $95 \,\mu\text{g/cm}^2$ was found, considerably higher than the US EPA standard for dust on household floors of $0.043 \,\mu\text{g/cm}^2$ (US EPA, 2001). Higher concentrations were found in homes with active recycling compared with those where recycling was no longer carried out. In these latter homes the highest concentrations were found around washing areas and in some kitchen and living areas. This suggested take-home lead contamination, with washing done at home rather than at the workplace. While surface contamination with lead was lower at the school, though still high at $41 \,\mu\text{g/cm}^2$, four sleeping mats were found to have lead contamination at a mean of $221 \,\mu\text{g/cm}^2$. This suggested that children had brought lead to school on their shoes and clothing.

3. Main routes of lead exposure and health effects

The health impacts of lead exposure, including low-level exposure, have been extensively reviewed and are summarized here (ATSDR, 2007; JECFA, 2011; NTP, 2012; Health Canada, 2013). Young children, pregnant women and women of childbearing age are particularly vulnerable to the toxic effects of lead.

3.1. Routes of exposure to lead

The main routes of exposure and absorption of lead are inhalation, ingestion and, to a much lesser extent, dermal contact (ATSDR, 2007). Inhalation of fumes and dust is a major route of exposure for people working with lead. Young children are particularly likely to be exposed through contaminated soil and air-borne household dust because they spend a lot of time in one place, tend to play on the ground, and have frequent hand-to-mouth activity (WHO, 2010a). Children with pica, a compulsion to eat non-food substances, may persistently eat lead-contaminated soil (Mielke & Reagan, 1998). Lead exposure may also occur from consumption of contaminated food and water.

The absorption of lead from the gastrointestinal tract is affected by dietary factors, age and nutritional status (JECFA, 2011). Infants and young children absorb proportionately more lead than adults, typically absorbing around 50% of ingested lead compared to around 10% in adults (WHO, 2010a). The absorption of lead is also greater in people with dietary deficiencies of iron or calcium, which may be widespread in economically deprived communities. Once absorbed, lead is distributed to most organs of the body, including the central nervous system, liver and kidneys, but the largest proportion (up to 90% in adults) is stored in bone (Barry, 1975).

Lead accumulates in bone over life up to age 50 to 60 years, followed by a decline due to age-related changes in diet, hormonal concentrations and metabolism (Mushak, 1993). There is an equilibrium between the amount of

Inhalation and ingestion are the main routes of lead exposure

lead in blood and bone, and some lead is gradually released back into blood over time (Rabinowitz, 1991). Lead in bone does not cause toxic effects but it becomes a potential source of toxicity when metabolic changes cause more rapid release. This can occur during pregnancy, lactation, the menopause and following bone fracture (Silbergeld et al., 1988; Markowitz & Weinberger, 1990; Mushak, 1993; Gulson, 2003). If the blood lead is reduced, e.g. following chelation therapy, some lead will be released from bone to restore the equilibrium.

Lead readily crosses the placenta exposing the fetus. The lead concentrations in maternal and fetal blood are similar (Graziano, 1990; WHO, 1995).

Lead is present in breast milk from external sources of exposure or remobilized from skeletal stores, though the concentrations in breast milk are low (Ettinger et al., 2004; Ettinger et al., 2014).

3.2. Toxic effects of lead

Lead has no apparent physiological function. It has an affinity for sulfhydryl groups and other organic ligands in proteins and can mimic other biologically essential metals, such zinc, iron and, in particular, calcium (Health Canada, 2013). This enables lead to disrupt enzyme systems dependent on these ions, accounting for many of its toxic effects (Lidsky & Schneider, 2003; Garza et al., 2006).

The toxic effects of lead are wide-ranging and affect almost all body systems. Acute lead poisoning from a single exposure is relatively rare and chronic poisoning is more common; however, the clinical features of poisoning are similar in both cases. The presenting signs and symptoms are very variable in both adults and children and may include gastrointestinal, haematological and neurological effects. Young children are particularly vulnerable to the neurological toxicity of lead and this is the main reason that lead is of public health concern. Lead also has toxic effects on the reproductive, endocrine and cardiovascular systems. Important toxic effects are summarized below by body system.

The duration of illness in lead poisoning may be long and periodic, requiring the monitoring of blood lead concentrations and repeated courses of antidotal chelation therapy.

3.2.1. Gastrointestinal effects

Gastrointestinal effects are common in lead toxicity and may be the reason that an exposed person first seeks medical attention. The effects include loss of appetite with weight loss, constipation, abdominal pain or discomfort, nausea, vomiting and a metallic taste in the mouth. Diarrhoea occurs occasionally (Winship, 1989). Lead colic (intense, painful, intermittent abdominal cramps)

The toxic effects of lead are wide-ranging and affect almost all body systems

is associated with severe constipation and vomiting, and can be mistaken for other conditions such as appendicitis, peptic ulcer, pancreatitis or intestinal obstruction (Janin et al., 1985). Gastrointestinal bleeding has occasionally been reported (McNutt et al., 2001; Frith et al., 2005).

Patients with poor dental hygiene may have a 'lead line' (Burton or blue line) along the gums (ten Bruggenkate et al., 1975). This line is composed of dark granules of lead sulfide precipitated by the action of hydrogen sulfide (from bacterial degradation of organic matter) on lead. There may also be grey spots on the buccal mucosa and on the tongue (ten Bruggenkate et al., 1975).

3.2.2. Neurological effects

Lead exerts toxic effects in all parts of the nervous system. Lead poisoning can cause life-threatening encephalopathy (disruption of brain function), particularly in young children. Encephalopathy is less commonly seen in adults (ATSDR, 2007). Initial signs include sporadic vomiting, loss of appetite, behavioural changes with aggression, irritability and agitation, headache, clumsiness and intermittent lethargy. This may progress to persistent vomiting, ataxia, convulsions, severe cerebral oedema, raised intracranial pressure, coma and death.

Lead encephalopathy is a life-threatening condition and children can be left with mental retardation, seizure disorders, blindness and hemiparesis (weakness of the entire left or right side of the body) (Perlstein & Attala, 1966; Chisolm & Barltrop, 1979; Al Khayat et al., 1997). Such severe impacts are now relatively uncommon in developed countries but can still be seen in places where there are high levels of exposure and limited or no access to diagnosis and treatment (Haefliger et al., 2009; Greig et al., 2014). Analysis of a large case series of lead-poisoned children in Nigeria found that concurrent infection with malaria increased susceptibility to the neurotoxic effects of lead (Greig et al., 2014).

Chronic lead toxicity may also cause more subtle changes in neurological function in children and adults. There is a large literature on the neurodevelopmental toxicity of lead in children (Lidsky & Schneider, 2003; Bellinger, 2004a; Koller et al., 2004; Needleman, 2004; NTP, 2012). The effects include reduced cognition and behaviour scores, changes in attention (including attention deficit hyperactivity disorder), impaired visual-motor and reasoning skills, and impaired social behaviour and reading ability. Some of these effects have been found to persist into later childhood and adulthood (Needleman et al., 1990; Fergusson & Horwood, 1993; White et al., 1993; Tong et al., 1996; Fergusson et al., 1997; Tong, 1998; Tong et al., 1998; Stokes et al., 1998). Delinquent behaviour has also been associated with lead exposure (Needleman et al., 1990; Needleman et al., 1996; Dietrich et al., 2001; Wright et al., 2008). Poor scores for social/emotional functioning have been reported in preschool children (Mendelsohn et al., 1998).

Even low levels of exposure, and blood lead concentrations below 5 μ g/dL, can be associated with neurological damage in children (NTP, 2012). Indeed, studies to date suggest that there may be no threshold blood lead concentration for neurotoxic effects in children and that the association between blood lead concentration and IQ may be non-linear (JECFA, 2011). A pooled analysis by Lanphear et al. (2005) found a steeper decline in IQ in children with maximal blood lead concentrations below 7.5 μ g/dL compared with those having maximal blood lead concentrations above 7.5 μ g/dL.

Studies suggest there may be no safe threshold of lead exposure

In adults, case reports and small studies describe a higher incidence of malaise, forgetfulness, headache, fatigue, lethargy, irritability, dizziness and weakness in occupationally exposed adults (ATSDR, 2007). Lead exposure may also be associated with a greater risk of neuropsychiatric and neurobehavioural problems (Valciukas et al., 1978; Williamson & Teo, 1986; Stollery et al., 1991; Chia et al., 1997; Bleecker et al., 2005; Chen et al., 2005; Schwartz et al., 2005).

Lead can cause both motor and sensory neuropathy. In individuals with severe, chronic lead toxicity wrist drop and foot drop (inability to extend the wrist or foot) may be seen. These effects are more commonly observed in adults than children with lead toxicity (ATSDR, 2007). Motor weakness usually resolves once the individual is removed from exposure but this may not be the case with sensory neuropathies (Rubens et al., 2001).

Poor postural stability has been reported in children with mildly elevated blood lead concentrations (Bhattacharya et al., 1990) and in lead-exposed workers (Chia et al., 1996; Ratzon et al., 2000; Iwata et al., 2005).

Lead may also cause visual impairment and reduced hearing (Cavalleri et al., 1982; Otto & Fox, 1993; Rothenberg et al., 2002). Hearing impairment in children may occur even with blood lead concentrations below 10 μ g/dL (NTP, 2012).

3.2.3. Cardiovascular

Lead exposure is associated with an increased risk of hypertension in adults and pregnant women, even at levels of exposure below $10 \,\mu\text{g/dL}$ (NTP, 2012). Significant, though modest, associations have been found between lead concentrations in blood and bone and blood pressure (Cheng, 2001; Nawrot et al., 2002; ATSDR, 2007). The association is stronger with bone lead, suggesting that the increase in blood pressure is related to the long-term effects of lead exposure earlier in life (Cheng, 2001; Gerr et al., 2002).

3.2.4. Renal

Lead can cause damage to the renal tubules with impairment of renal function; however, acute renal damage is usually reversible (Green et al., 1976; Chisolm

& Barltrop, 1979); Wedeen, 1988; Loghman-Adham, 1997). Chronic lead exposure may cause a progressive nephropathy (Loghman-Adham, 1997). The onset of lead-induced renal impairment is subtle and patients may remain asymptomatic until there is significant renal dysfunction (Loghman-Adham, 1997). Even low levels of exposure to lead can be associated with abnormalities in renal function (NTP, 2012).

3.2.5. Endocrine

Environmental lead exposure has been associated with delays in sexual maturity in girls (Selevan et al., 2003; Wu et al., 2003; NTP, 2012). Lead exposure has also been associated with delays in growth and reduced growth (e.g. smaller stature, smaller head circumference) in children (NTP, 2012).

3.2.6. Reproductive system and pregnancy

Impotence and decreased libido are occasionally reported in lead-poisoned patients (Cullen et al., 1983). Reduced fertility has been found in couples during periods when the blood lead concentration in the male is elevated (JECFA, 2011). Possible causes include reduced sperm motility, decreased sperm count and reduced semen volume (NTP, 2012).

Lead has long been known to be harmful in pregnancy and has been used as an abortifacient (Bastrup-Madsen, 1950). Maternal lead exposure, even at low levels, may be associated with reduced fetal growth, lower birth weight, preterm birth and spontaneous abortion (NTP, 2012; Health Canada, 2013). Lead exposure is a risk factor for hypertension in pregnancy (gestational hypertension) and high levels of exposure may be a risk factor for pre-eclampsia, which can be life-threatening for both the mother and baby (Troesken, 2006; CDC, 2010).

3.2.7. Haematological

High levels of exposure to lead reduce the synthesis of haem, which is necessary for the production of red blood cells, resulting in anaemia (ATSDR, 2007). Coarse basophilic stippling of red blood cells may be seen, though this is not found in all patients with lead poisoning. Interference with haem synthesis also has other negative impacts, for example haem is needed for the formation of cytochrome c, which is essential for cellular respiration, and this may contribute to the neurotoxicity of lead (ATSDR, 2007).

3.3. Toxic effects in relation to blood lead concentrations

The most widely used method for assessing exposure to lead is the measurement of lead in whole blood (see section 6.1). There is, however, considerable interindividual variation in the blood lead concentration at which specific signs of poisoning manifest. Some individuals may apparently be clinically well at blood lead concentrations that are associated with encephalopathy in others

(Bellinger, 2004a). This also applies to subclinical effects such as effects on IQ, meaning that children with the same blood lead concentration do not necessarily have the same risk of impaired neurodevelopment (Bellinger, 2004a). Table 1 summarizes information on toxic effects that have been reported at specific blood lead concentrations and illustrates some of the variability in response.



Table 1. Association of subclinical and clinical effects with blood lead concentrations

Blood lead concentration	Health effect	Reference	
	Children:	- NTP, 2012	
<5 μg/dL	 Decreased IQ, cognitive performance and academic achievement increased incidence of problem behaviours and diagnosis of attention deficit hyperactivity disorder Reduced fetal growth (based on maternal blood lead concentration) 		
	All ages:		
	 Impaired renal function, Reduced synthesis of delta-aminolevulinic acid dehydratase (ALAD), contributing to anaemia 		
	Children:		
	Delayed puberty		
	Adults:		
<10 μg/dL	 Hypertension Increased cardiovascular-related mortality (based on limited evidence) Spontaneous abortion (based on maternal blood lead concentration) (based on limited evidence) Preterm birth (based on maternal blood lead concentration) (based on limited evidence) 		
>20 µg/dL	Children:	Schwartz et	
	Anaemia	al., 1990	
>30 µg/dL	Children:		
	Reduced nerve conduction velocity		
	Children:	ATSDR, 2007	
	Decreased haemoglobin synthesis		
>40 µg/dL	Adults:		
	Peripheral neuropathyNeurobehavioural effectsAbdominal colic		
. FO/dl	Adults:		
>50 μg/dL	Decreased haemoglobin synthesis		
>50 µg/dL (= lowest concentration in	Children:	Crair at al. 2014	
children with malaria)	Severe neurological features	Greig et al., 2014	
- 60 ug/dl	Children:		
>60 μg/dL	Abdominal colic	NAS, 1972 quoted in ATSDR, 2007	
>60 µg/dL	Children:		
(= lowest concentration; mean 178 μg/ dL)	Features of acute poisoning but no encephalopathy		
>90 µg/dL	Children:		
(= lowest concentration, mean 330 μg/ dL)	vest concentration, mean 330 μg/ • Encephalopathy		
>105 µg/dL	Children:		
(= lowest concentration in children without malaria)	Severe neurological features	Greig et al., 2014	
		NAS, 1972	
≥150 µg/dL	Children:	quoted in	
		ATSDR, 2007	
>216 µg/dL (= lowest concentration, range 216-460 µg/dL)	Death	Thurtle et al., 2014	

4. The public health impact of lead exposure

At a population level the main impacts of lead exposure arise from its effects on neurocognitive development in children and on cardiovascular disease in adulthood.

In children the greater risk of reduced cognitive ability, IQ, attention and visual-motor and reasoning skills, as well as impacts on social behaviour all contribute to an increased public health and economic burden. While the estimated IQ decrease in children from lead poisoning is small (6.9 points over the blood concentration range 2.4 to 30 μ g/dL), the impact at the population level can be important (Bellinger, 2004b; Lanphear et al., 2005). It is estimated that a mean IQ reduction of 3 points from 100 to 97 would increase the number of individuals with an IQ below 100 by 8% and there would be a 57% increase in individuals with an IQ below 70 (commonly considered the cut-off for identifying individuals with intellectual disability). There would also be a 40% reduction in potentially high-achieving individuals with an IQ score greater than 130 (Bellinger, 2004b; JECFA, 2011).

In the case of blood pressure, a review by Healey et al. (2010) estimated that for the Canadian population a change in blood lead concentrations in adults from 1 μ g/dL to 4 μ g/dL would be associated with an estimated increase in mean systolic blood pressure of approximately 0.8 mmHg (0.11 kPa) among Caucasian males and 1.4 mmHg (0.19 kPa) in susceptible subpopulations. While the impact at the individual level is small, increases in blood pressure are associated with age-specific increased mortality rates for both ischaemic heart disease and stroke (Lewington et al., 2002; Fewtrell et al., 2003).

Based on 2015 data, lead exposure is estimated to account for 12.4% of the global burden of idiopathic intellectual disability, 2.5% of the global burden of ischaemic heart disease and 2.4% of the global burden of stroke (IHME, 2016).

5. Economic impact of lead exposure in countries

The economic costs attributable to the effects of lead on intellectual development in children are estimated to be 1.2% of global GDP (Attina & Trasande, 2013)

The economic impact of lead exposure is made up of direct and indirect costs. Direct costs include those associated with screening and the medical care of acute and chronic lead poisoning, as well as the provision of special education, and managing juvenile delinquency and other criminal behaviours. Indirect costs reflect the economic burden on society from a variety of factors including reduced intelligence and the consequent reduction in economic productivity and tax revenue.

Trasande & Liu (2011) estimated that the total annual cost of lead poisoning in the USA due to lost economic productivity was US\$ 50.9 billion in 2008. There was an estimated US\$ 5.9 million in medical care costs. In France, Pichery et al. (2011) estimated that lost lifetime earnings as a result of lead exposure in children amounted to €53.9 billion (US\$ 69.8 billion at 2008 values). In Europe, Bartlett & Trasande (2014) estimated economic costs attributable to lead exposure to be around US\$ 57 billion, based on estimated IQ losses and impact on economic productivity.

The estimated economic costs attributable to the neurodevelopmental impacts of childhood lead exposure amounted to 1.2% of global gross domestic product (GDP) in 2011 (Attina & Trasande, 2013). Expressed in terms of loss to regional GDP, the estimated cost in Africa was 4.03%, in Latin America and the Caribbean 2.04%, and in Asia 1.88% (Attina & Trasande, 2013).

6. Assessment of lead exposure

Exposure assessment is one component of the risk assessment process that culminates in the characterization of the health risks posed by a chemical to a population (WHO, 2010b). An investigation of lead exposure from recycling should involve identification or confirmation of the source(s) of exposure, determination of the routes and the media by which exposure is occurring, and assessment of the severity and associated health impacts of exposure. Confirmation of exposure and determination of severity and the need for treatment involves measurement of the blood lead concentration, together with a medical examination for signs and symptoms of lead poisoning. The source(s) of exposure can be identified by taking an exposure history and by carrying out environmental investigations.

6.1. Blood lead measurements

Lead can be measured in a range of human tissues and fluids, including hair, teeth, bone, blood and urine; however, measuring the concentration of lead in whole blood is the most accepted tool for screening, diagnostic and management purposes (WHO, 2011a). This is because there is a large body of information linking blood lead concentrations with clinical effects and treatment outcomes. Moreover, validated analytical methods and reliable blood quality-control and reference materials are available (Barbosa et al., 2005; CLSI, 2013).

Analytical techniques include point-of-care devices and laboratory-based methods, and information about the use of these methods can be found in the WHO publication *Brief guide to analytical methods for measuring lead in blood* (WHO, 2011a). The choice of method is determined by the available resources and the specific needs of the study in terms of limit of detection, number of samples to be analysed and turnaround time.

Point-of-care testing for lead involves the use of a portable analytical device that can be taken and used near the site of exposure or patient care. These devices can analyse very small samples taken from a capillary by finger-prick The blood lead concentration is the most accepted tool for screening, diagnosis and management of lead exposure

or from a vein. They have a limited operating range and are most suitable for screening purposes: an elevated blood lead concentration should be confirmed by a laboratory method (WHO, 2011a).

The advantages of a point-of-care device are that: 1) it does not require skilled laboratory personnel for its operation; 2) it can be used at locations where transport of blood samples to an appropriate reference laboratory is difficult; and 3) the result can be provided within a few minutes. It is extremely important that steps are taken to avoid lead contamination. If the device is to be used in the field then it should be set up in a clean room with adequate precautions to prevent ingress of dust.

Laboratory-based methods for measuring blood lead concentrations have greater accuracy and a lower detection limit, potentially as low as 1-2 $\mu g/dL$ with some methods. Examples include atomic absorption spectrometric (AAS) methods, such as graphite furnace atomic absorption spectrometry (GFAAS), and inductively coupled plasma mass spectrometry (ICP-MS) (WHO, 2011a). An advantage of GFAAS and ICP-MS is that they can measure very small samples, in the range of 10–50 μL , which is useful when testing young children. The more advanced laboratory-based methods are, however, more expensive to buy and run and require specific laboratory expertise.

For all analytical methods some basic principles should be observed. Blood sampling should be performed by a trained health-care worker (WHO, 2010c). Universal biosafety precautions should be observed to avoid transmission of infection. With both venous and capillary sampling there is a risk of lead contamination from the skin. Sample collection should take place in a clean, lead-free environment and the puncture site should be thoroughly cleansed beforehand (CDC, 2013). Venous blood should be collected into tubes containing anticoagulant, preferably EDTA (CLSI, 2013). All sampling equipment should be of good quality and certified free from trace metal contamination. If blood samples need to be transported they should be kept cool, either with cool-packs in an insulated container, or refrigerated until analysis (CLSI, 2013). For point-of-care analysers, note should be taken of any specific temperature requirements for sample storage. Care should be taken to avoid lead contamination of samples during storage, transportation and analysis.

When choosing a laboratory, it is important to ensure that it has an adequate quality management system in place. Ideally the laboratory should be accredited by a recognized body and should participate in a proficiency-testing scheme for blood lead analysis. An example of such a scheme is the Lead and Multi-element Proficiency Program (LAMP)¹, which is provided by the US Centers for Disease Control and Prevention free-of-charge to laboratories around the

¹ https://www.cdc.gov/labstandards/lamp.html

world. Participation in LAMP does not provide accreditation but it is a means for monitoring laboratory performance.

No safe level of lead exposure has been identified; therefore, to determine the blood lead concentration that indicates excessive exposure the value can be compared to a reference value for the population as a whole. This is usually the geometric mean blood lead concentration found in the highest 2.5% or 5% of the population, i.e. the 97.5th or 95th percentile respectively. In the USA, for example, for children under six years the reference value is currently 5 μ g/dL, which is the 97.5th percentile blood lead concentration (CDC, 2012). This same concentration is the 98th percentile value for children under 7 years in France (Haut Conseil de la santé publique, 2014). Germany has adopted reference values of 3.5 μ g/dL for children aged 3-14 years, 7 μ g/dL for women and 9 μ g/dL for men (Wilhelm, 2010).

6.2. Taking an exposure history

As part of an investigation into an individual's exposure to lead, a thorough environmental and occupational history should be taken. This should include questions about the following potential sources of exposure:

- individual's occupation, work practices and those of co-habitants;
- participation in, or proximity to, lead recycling activities or manufacturing with recycled lead (e.g. fishing weights);
- hobbies that might involve lead exposure;
- use of traditional medicines:
- diet and the possible consumption of foods cultivated on contaminated land or collected from contaminated water bodies; and
- source of drinking-water, e.g. piped water supply, wells, river etc.



Examples of approaches to history-taking are provided by the US Agency for Toxic Substances and Disease Registry (ATSDR, 2015) and WHO (WHO, 2010a).

6.3. Environmental assessment

Lead battery recycling is associated with significant environmental contamination and therefore investigations of off-site contamination are warranted in the vicinity of these facilities. Potential environmental pathways of exposure include soil and dust, air, water and food, and all of these media can be analysed for lead. In most cases there are guideline or reference values or regulatory standards to compare analytical results against, which will give an indication of the importance of that pathway to exposure.

6.3.1. Soil and dust

Soil and dust samples can be collected and sent to a laboratory for analysis, or concentrations can be measured *in situ* using an X-ray fluorescence (XRF) device that reports results almost immediately. A strategy should be devised that ensures collection of a representative set of samples. Account should be taken of possible dispersion of lead-contaminated soil from a recycling site to a wider area by wind or floodwaters. A recent publication by the US Centers for Disease Control and Prevention provides guidance on planning and conducting an environmental survey of lead contamination (Hodge et al., 2015). Guidance on soil sampling is also provided by Demetriades & Birke (2015). Documenting a geographical positioning system (GPS) reading at each sample location greatly assists in mapping areas of high and low contamination.

Standards for lead in soil exist in a number of countries, and at least one country, the USA, also has standards for lead in household dust. A global review of standards for lead in residential soil found values ranging from 50 to 400 mg/kg (Jennings, 2013). The standard for non-residential soils may be higher. In the USA, for example, the federal standard for residential soil in areas where children play is 400 mg/kg (US EPA, 2001). The USA standard for lead in household floor dust is 0.043 µg/cm² (US EPA, 2001).



As described above, various stages in the recycling process can result in the release of lead fumes and particles in the air. Studies have shown that there is high airborne lead exposure in lead-acid battery recycling facilities (Gottesfeld & Pokhrel, 2011; Were et al., 2012). Airborne lead concentrations have been shown to correlate with blood lead concentrations in workers (Were et al., 2012). Airborne lead eventually settles and contaminates surrounding surfaces.

In an occupational setting there are two ways of measuring airborne lead levels: area air sampling and personal sampling. Area air sampling involves placing a pump in



the work area being tested. The pump runs for a specific period of time (usually corresponding to the normal working day) at a specific flow rate. Air sampling can provide a general overview of the air quality and helps to determine whether further (personal) sampling is needed (US EPA, 1993).

A personal air sampling pump is worn by an individual to assess their exposure to lead over a period of time, e.g. a working day (US EPA, 1993). This method monitors air concentrations in a worker's breathing zone to measure representative employee exposures. The equipment comprises a battery-operated pump and sample medium, which the individual can wear, for example, hung from their belt. A tube is attached to the pump and the other end of the tube is clipped at the collar area close to the person's breathing zone. Samples are then taken to a laboratory for analysis of total lead.

Examples of workplace exposure limits for airborne lead are given below:

- Australia: 0.15 mg/m³ as an 8-hour time-weighted average (Safe Work Australia, 2013)
- European Union: 0.15 mg/m³ as an 8-hour time-weighted average (EC, 1998)
- Mexico: 0.05 mg/m³ as an 8-hour time-weighted average, 40 hours per week (CEC, 2016)
- USA: 0.05 mg/m³ as an 8-hour time-weighted average (OSHA, 1978).

A number of countries have ambient air quality standards for lead concentrations in the outdoor environment. The guideline value set by WHO for the annual mean concentration is $0.5 \,\mu\text{g/m}^3$ (WHO, 2000) but it should be noted that this is not a health-based standard. A detailed discussion of methods for monitoring pollutants, including lead, in ambient air can be found in the WHO publication *Monitoring ambient air quality for health impact assessment* (EURO, 1999). Examples of national standards are:

- Australia: ambient air quality standard: 0.5 μg/m³ averaged over one year (NEPC, 2016)
- China: ambient air quality standard: $0.5 \,\mu\text{g/m}^3$ averaged over one year, with a seasonal limit of $1 \,\mu\text{g/m}^3$ (MoEP, 2012)
- EU limit value: 0.5 μg/m³ averaged over one year (EC, 2008)
- US National Air Ambient Quality Standard: 0.15 μg/m³, 3-month average concentration (US EPA, 2016).

6.3.3. Food and water

Foods that could become contaminated with lead include fruit and vegetables (especially leafy vegetables) grown on land close to recycling activities, foraging animals such as goats, sheep, pigs and poultry, and fish and shellfish caught from waters contaminated with recycling waste.

A common source of water contamination in piped water supplies is the use of lead materials in the water distribution system; however, this is outside the scope of this document. Lead-acid battery recycling can contaminate surface waters that are used for drinking, cooking and bathing. Dissolved lead can percolate through soil into groundwater (UNEP, 2004).

If the exposure history suggests that consumption of contaminated food and/ or water is a source of exposure then these can be analysed. The Codex Alimentarius provides a list of maximum permissible levels of lead in selected foods (FAO, 2016). Most countries have a drinking-water standard for lead and the WHO guideline value for drinking-water is $10 \mu g/L$ (WHO, 2011b).

7. Control measures

At every stage in the recycling process there are measures that can be taken to prevent or reduce the release of lead. A detailed description of control measures is beyond the scope of this document but brief information is given here. Further information can be found in technical guidelines and a training manual published by the Basel Convention Secretariat and in technical guidelines published by the Commission for Environmental Cooperation (CEC). These provide practical advice and guidance to national authorities on the environmentally sound management of used lead-acid batteries (UNEP, 2003; UNEP, 2004; CEC, 2016). The US Occupational Safety and Health Administration provides information about engineering and other controls in its e-learning tool on secondary lead smelting (OSHA, 2002).

7.1. Battery collection, storage and transportation

Measures that should be taken at battery collection and storage sites include the following (CEC, 2016; UNEP, 2003). Batteries should not be drained at the collection point. They should be stored securely and sheltered from the weather. The storage location should be well ventilated and the ground should be coated with acid-resistant concrete or other resistant material. Leaking batteries should be placed in acid-resistant containers. The number of stored batteries should be controlled. There should be prominent hazard warnings. Battery collectors should not sell their batteries on to unlicensed lead smelters.

Used lead-acid batteries should be transported as hazardous waste. The batteries should be kept upright and separated by cardboard or other non-conducting material and then placed in sealed containers or otherwise secured, e.g. on pallets covered with shrink wrap, to prevent them moving about.

7.2. Battery recycling

To minimize lead exposure and environmental contamination, lead battery recycling should only be conducted at adequately equipped and regulated facilities that have the requisite engineering controls, trained staff, provision of protective equipment, and environmental and occupational monitoring.

Lead battery recycling should only be conducted at adequately equipped and regulated facilities

The most important and effective control measures are engineering and emission controls. However, work practice controls are also needed to protect the health of workers and reduce emissions to the outside (OSHA, 2002). Engineering controls include the use of fully automated and enclosed operations for the dismantling and separation of lead-acid batteries and the smelting and refining of lead. Fugitive emissions from the various stages of the recycling process can be reduced by the use of negative pressure enclosures, i.e. a sealed area where adequate ventilation is in place to create a negative pressure that is exhausted through an emission control device and/or a high-efficiency particulate air (HEPA) filter to trap particles and dust. The use of hooding and exhaust ventilation over open areas of operation, e.g. battery saws and crushers, furnace feed conveyors and furnace charging points, will trap dusts and fumes. Keeping molten lead at lower temperatures will reduce the amount of fume. Dust and particle emissions should be trapped in a filter baghouse, by using a wet electrostatic precipitator or by other similar technologies (CEC, 2016). These traps should be regularly cleaned and the contents fed into the smelter to recover the lead. There should be an effluent treatment station to treat all water used in the recycling process and for cleaning (CEC, 2016). Rainwater run-off from roofs and other surfaces, which are likely to be contaminated with lead, should also be collected and treated (UNEP, 2003).

Other techniques to reduce dispersion of dust include keeping all open operations wet, ensuring that batteries and slag are safely stored under cover and kept away from water, and ensuring that the whole operating area is kept clean using wet methods and HEPA vacuuming. There should be regular environmental monitoring to ensure that control measures are effective.

In addition to providing adequate ventilation, workers' exposure can further be reduced by the following measures (OSHA, 2002; UNEP, 2003; Kosnett et al., 2007; CEC, 2016):

- training on the hazards of lead and measures to prevent exposure;
- providing, and enforcing the use of, personal protective equipment (see below);
- prohibition of smoking, eating or drinking in the workplace;
- providing a segregated eating area well away from recycling operations;
- providing a clean air room, maintained at positive pressure and with filtered air, for the removal of respirators;
- providing, and enforcing the use of, facilities for workers to change into clean work clothing before starting work and to wash and change clothes at the end of the working day;
- the implementation of a policy for regular blood lead testing, with a specified medical removal level to remove an over-exposed worker from working around lead, together with provisions for alternative employment or compensation.

Medical removal levels vary from country to country and are usually lower for women than for men. There is also a voluntary industry initiative among battery companies in a number of countries to maintain workers' blood lead concentrations below 30 μ g/dL (EUROBAT, 2013).

Ensuring that workers wash and change before leaving the facility is important for protecting household members from take-home exposure to lead.

7.2.1. Personal protective equipment

The required personal protective equipment will vary according to the specific tasks being carried out and the concomitant risk of exposure to lead and other hazards. Equipment may include: full-body coveralls, apron, gloves, hard hats, shoes or disposable shoe covers, respirators, face shields or vented goggles (OSHA, 2002; CEC, 2016). It is important that this equipment is regularly cleaned and maintained in good repair. It should be noted that respiratory protection is not a substitute for adequate ventilation and pollution controls in the workplace but can be used along with these other measures.

7.3. Informal recycling

It is clear from the above description of control measures that it is not possible for informal recycling to be carried out in an occupationally and environmentally sound way. Preventing informal recycling presents a number of challenges and it is important to take a holistic approach. Recycling is often carried out in a covert way, e.g. at night or in constantly changing locations. Therefore, identifying operations in order to close them down may be difficult. In addition, for people with limited employment opportunities, recycling may provide a critical source of family income with consequent repercussions if it is stopped and no alternative is provided.

The Basel Convention Secretariat devotes a chapter of its training manual to the problem of controlling informal recycling and suggests a number of approaches (UNEP, 2004). These include determining the likely scale of operations through a market analysis of battery imports and sales, and identifying the points of interaction between the informal and formal sectors, as well as collecting information about recycling practices to determine where interventions can be targeted.

The life-cycle of a lead-acid battery involves manufacturers, retailers, scrap dealers, secondary smelters and consumers. Each can play a part in preventing the supply of used lead-acid batteries to the informal recycling sector. Some suggested approaches include:

 encouraging the collection of used batteries by licensed retailers when replacement batteries are being bought, for example by requiring a returnable Manufacturers, retailers, scrap dealers, secondary smelters and consumers can each play a part in reducing informal lead-acid battery recycling

deposit or refund scheme that would price lead batteries at a level closer to the actual intrinsic value of the lead and more than the informal sector would be willing to pay;

- requiring manufacturers to take back used lead-acid batteries in order to sell them to licensed recyclers (extended producer responsibility);
- informing consumers about the value of recycling lead-acid batteries and the dangers of dumping or supplying batteries to unlicensed smelters;
- informing the public at large about the health and environmental hazards of lead;
- encouraging the technological development of lead-acid batteries to make them last longer;
- prohibiting the sale of used lead-acid batteries to unlicensed smelters;
- creating a role for the informal sector e.g. by developing the necessary infrastructure that encourages scavengers to take batteries to licensed smelters; and
- sharing experiences between countries on approaches to controlling informal recycling (UNEP, 2004).

7.4. The problem of legacy pollution

Lead is persistent and highly immobile in the environment and surface lead concentrations are unlikely to migrate to subsurface soils even after a prolonged period (Kabala & Singh, 2001). Sites where recycling activity has taken place will, therefore, continue to pose an exposure risk to local populations. Consideration should therefore be given to methods of mitigating exposure, for example through environmental remediation.

7.5. Policy measures

Enforcement of the control measures described above requires a national policy to be put in place for the sound management of used lead-acid batteries that encompasses standards for collection, recycling, emissions, and occupational safety (UNEP, 2003; UNEP, 2004). Relevant regulatory measures include landuse planning laws concerning the location of secondary smelters (e.g. distance from residential areas), environmental standards governing emissions and discharges, and occupational standards for workplace and worker monitoring (UNEP, 2004; van der Kuijp et al., 2013).

For these measures to be successful there is also a need for adequate technical capacities, such as trained inspectors and laboratory facilities for the measurement of lead in biological and environmental samples, as well as appropriate enforcement measures. It is also important that the health sector, particularly at primary care level, is able to recognise possible lead poisoning and to initiate the necessary diagnostic and treatment interventions.

8. Conclusions and way forward

As lead-acid batteries are the fastest growing segment of lead usage, there will be a continuing demand for lead. Economics dictate that lead batteries will be recycled to augment supplies that come from mining sources (UNEP, 2013). Lead can be recycled indefinitely, although there are some losses at each stage, the amount depending on the efficiency of the recycling process. Informal recycling is particularly inefficient in terms of lead losses; moreover the purity of the final product is poor. Lead mining uses large amounts of energy and environmental resources, particularly water. It causes significant environmental degradation and loss of habitat as well as generating large amounts of contaminated waste tailings. The environmental impact of recycling is potentially much less, for example, recycling lead saves approximately 55–65% of the energy used in mining and processing (UNEP, 2013). Provided it is carried out using appropriate technologies and to an adequate environmental standard, recycling can have less impact on the environment and on human health than mining.

As this document indicates, recycling lead-acid batteries must be carried out with care to minimize environmental contamination and protect the health of workers and communities. While much of the responsibility for ensuring the sound management of used lead-acid batteries lies with the environment sector, the health sector must also play its part. This includes ensuring that health-care practitioners have training on, and resources for, the diagnosis and management of lead poisoning, educating local communities on the health hazards of lead, and taking action to inform the responsible authorities when lead poisoning associated with recycling is discovered. Furthermore, health ministries should aim to ensure the availability of laboratory capacity for blood lead testing and should work with industry to reduce employee exposures.

9. References

AGENDA (2016) Used lead acid battery (ULAB) recycling in Tanzania: survey report. Dar es Salaam: AGENDA for Environment and Responsible Development; 2016 (http://www.econet.international/index.php?id=3, accessed 3 January 2017).

Al Khayat A, Menon NS, Alidina MR (1997) Acute lead encephalopathy in early infancy – clinical presentation and outcome. Annals Tropical Paediatrics. 1997; 17(1):39-44.

ATSDR (2007) Toxicological profile for lead. Atlanta (GA): Agency for Toxic Substances and Disease Registry; 2007 (https://www.atsdr.cdc.gov/ToxProfiles/tp13.pdf, accessed 3 January 2017).

ATSDR (2015) Case studies in environmental medicine: Taking an exposure history. Atlanta (GA): Agency for Toxic Substances and Disease Registry; 2015 (https://www.atsdr.cdc.gov/csem/exphistory/docs/exposure_history.pdf, accessed 3 January 2017).

Attina TM, Trasande L (2013). Economic costs of childhood lead exposure in low- and middle-income countries. Environmental Health Perspectives. 2013; 121:1097–1102 (http://dx.doi.org/10.1289/ehp.1206424, accessed 3 January 2017).

Baker EL, Folland DS, Taylor TA, Frank M, Peterson W, Lovejoy G, Cox D, Housworth J, Landrigan PJ (1977). Lead poisoning in children of lead workers: home contamination with industrial dust. New England Journal of Medicine. 1997; 296(5):260–261 (http://www.nejm.org/doi/pdf/10.1056/NEJM197702032960507, accessed 3 January 2017).

Barbosa F, Tanus-Santos JE, Gerlach RF, Parsons PJ (2005). A critical review of biomarkers used for monitoring human exposure to lead: advantages, limitations, and future needs. Environmental Health Perspectives. 2005; 113(12):1669–1674 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1314903/, accessed 3 January 2017).

Barry PSI (1975). Comparison of concentrations of lead in human tissues. British Journal of Industrial Medicine. 1975; 32:119–139 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1008038/pdf/brjindmed00086-0027.pdf, accessed 3 January 2017).

Bartlett ES, Trasande L (2014). Economic impacts of environmentally attributable childhood health outcomes in the European Union. European Journal of Public Health. 2014; 24(1):21-26 (http://eurpub.oxfordjournals.org/content/24/1/21. long, accessed 3 January 2017).

Bastrup-Madsen P (1950). Dimercaprol in acute lead poisoning. Lancet. 1950; 2(5):171-172 (http://www.sciencedirect.com/science/article/pii/S0140673650911494, accessed 3 January 2017).

Belay M, Belay A, Genet Z (2015). Safety practices and awareness of lead acid battery recyclers in Addis Ababa, Ethiopia. Addis Ababa: Pesticide Action Nexus Association; 2015 (http://www.econet.international/index.php?id=3, accessed 3 January 2017).

Bellinger DC (2004a). Lead. Pediatrics. 2004; 113(4, Supplement 3):1016-1022 (http://pediatrics.aappublications.org/content/113/Supplement_3/1016, accessed 3 January 2017).

Bellinger DC (2004b). What is an adverse effect? A possible resolution of clinical and epidemiological perspectives on neurobehavioral toxicity. Environmental Research. 2004; 95:394-405.

Bhattacharya A, Shukla R, Bornschein RL, Dietrich KN & Keith R (1990). Lead effects on postural balance of children. Environmental Health Perspectives. 1990; 89:35-42 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1567808/pdf/envhper00422-0036.pdf, accessed 3 January 2017).

Blacksmith Institute (2009). The problem. In: Pollutant: Lead [website]. New York: Blacksmith Institute; 2009 (http://www.blacksmithinstitute.org/haina. html, accessed 3 January 2017).

Bleecker ML, Ford DP, Lindgren KN, Hoese VM, Walsh KS, Vaughan CG (2005). Differential effects of lead exposure on components of verbal memory. Occupational & Environmental Medicine. 2005; 62(3):181-187 (http://oem.bmj.com/content/62/3/181.long, accessed 3 January 2017).

California Environmental Protection Agency (2015). DTSC announces order to close Exide facility and steps to protect community with enhanced cleanup. Department of Toxic Substances Control News Release, March 12 2015. Available at (https://www.dtsc.ca.gov/HazardousWaste/Projects/upload/News-Release-T-06-15.pdf, accessed 3 January 2017).

Cavalleri A, Trimarchi F, Gelmi C, Baruffini A, Minoia C, Biscaldi G, Gallo G (1982). Effects of lead on the visual system of occupationally exposed subjects. Scandinavian Journal of Work Environment & Health. 1982; 8(Suppl 1):148-151.

CDC (2010). Guidelines for the identification and management of lead exposure in pregnant and lactating women. Atlanta (GA): US Centers for Disease Control and Prevention; 2010 (http://www.cdc.gov/nceh/lead/publications/leadandpregnancy2010.pdf, accessed 3 January 2017).

CDC (2012). Low level lead exposure harms children: A renewed call for primary prevention. Atlanta (GA): US Centers for Disease Control and Prevention;

2012 (http://www.cdc.gov/nceh/lead/acclpp/final_document_030712.pdf, accessed 3 January 2017).

CDC (2013). Guidelines for measuring lead in blood using point of care instruments. Atlanta (GA): US Centers for Disease Control and Prevention; 2013 (http://www.cdc.gov/nceh/lead/ACCLPP/20131024_POCguidelines_final. pdf, accessed 3 January 2017).

CEC (2016). Environmentally sound management of spent lead-acid batteries in North America: Technical guidelines. Montreal: Commission for Environmental Cooperation; 2016 (in English, French & Spanish) (http://www3.cec.org/islandora/en/item/11665-environmentally-sound-management-spent-lead-acid-batteries-in-north-america, accessed 25 January 2017)

Chen SS, Chen TJ, Lin CH, Tseng YT, Lai SL (2005). Neurobehavioral changes in Taiwanese lead-exposed workers. Journal of Occupational & Environmental Medicine. 2005; 47:902-908.

Cheng Y, Schwartz J, Sparrow D, Aro A, Weiss ST, Hu H (2001). Bone lead and blood lead levels in relation to baseline blood pressure and the prospective development of hypertension: the Normative Aging Study. American Journal of Epidemiology. 2001; 153(2):164-171 (http://aje.oxfordjournals.org/content/153/2/164.full.pdf+html, accessed 3 January 2017).

Chia SE, Chia HP, Ong CN, Jeyaratnam J (1996). Cumulative concentrations of blood lead and postural stability. Occupational & Environmental Medicine. 1996; 53:264-268 (http://oem.bmj.com/content/53/4/264.full.pdf+html, accessed 3 January 2017).

Chia SE, Chia HP, Ong CN, Jeyaratnam J (1997). Cumulative blood lead levels and neurobehavioral test performance. Neurotoxicology. 1997; 18(3):793-803.

Chisolm JJ Jr, Barltrop D (1979). Recognition and management of children with increased lead absorption. Archives of Diseases of Childhood. 1979; 54(4):249-262 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1545299/pdf/archdisch00794-0005.pdf, accessed 3 January 2017).

Chisolm JJ Jr (1978). Fouling one's own nest. Pediatrics. 1978; 62:614–617.

CLSI (2013). Measurement procedures for the determination of lead concentrations in blood and urine; approved guidelines - 2nd edition. CLSI document C40-A2. Wayne (PA): Clinical and Laboratory Standards Institute; 2013.

CREPD (2015). Baseline study about facts in waste lead-acid battery recycling in Cameroon. Yaoundé: Centre de Recherche et d'Education pour le Développement; 2015 (http://www.econet.international/index.php?id=3, accessed 3 January 2017).

Cullen MR, Robins JM, Eskenazi B (1983). Adult inorganic lead intoxication: Presentation of 31 new cases and a review of recent advances in the literature. Medicine. 1983; 62:221-247.

Daniell WE, Tung LV, Wallace RM, Havens DJ, Karr CJ, Diep NB, Croteau GA, Beaudet NJ, Bao ND (2015). Childhood lead exposure from battery recycling

in Vietnam. BioMed Research International. 2015; Article ID 193715 (http://dx.doi.org/10.1155/2015/193715, accessed 3 January 2017).

Demetriades A, Birke M (2015). Urban geochemical mapping manual: Sampling, Sample preparation, Laboratory analysis, Quality control check, Statistical processing and map plotting. Brussels: EuroGeoSurveys; 2015 (http://www.eurogeosurveys.org/wp-content/uploads/2015/10/Urban_Geochemical_Mapping_Manual.pdf, accessed 3 January 2017).

Dietrich KN, Ris MD, Succop PA, Berger OG, Bornschein RL (2001). Early exposure to lead and juvenile delinquency. Neurotoxicology & Teratology. 2001; 23(6):511-518 (http://www.sciencedirect.com/science/article/pii/S0892036201001842, accessed 3 January 2017).

EC (1998). Council Directive 98/24/EC of 7 April 1998 on the protection of the health and safety of workers from the risks related to chemical agents at work (fourteenth individual Directive within the meaning of Article 16(1) of Directive 89/391/EEC). Official Journal 5 May 1998; L 131, 41:11-23 (http://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:31998L0024&from=EN, accessed 3 January 2017).

EC (2008). Directive 2008/50/EC of the European Parliament and of the Council of 21 May 2008 on ambient air quality and cleaner air for Europe. Official Journal 11 June 2008, L 152, 51:1-44 (http://eur-lex.europa.eu/LexUriServ/LexUriServ. do?uri=OJ:L:2008:152:0001:0044:en:PDF, accessed 3 January 2017).

Ettinger AS, Roy A, Amarasiriwardena CJ, Smith D, Lupoli N, Mercado-García A, Lamadrid-Figueroa H, Tellez-Rojo MM, Hu H, Hernández-Avila M (2014). Maternal blood, plasma, and breast milk lead: lactational transfer and contribution to infant exposure. Environmental Health Perspectives. 2014; 122:87–92 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3888576/, accessed 3 January 2017).

Ettinger AS, Tellez-Rojo MM, Amarasiriwardena C, Gonzalez-Cossio T, Peterson KE, Aro A, Hu H & Hernandez-Avila M (2004). Levels of lead in breast milk and their relation to maternal blood and bone lead levels at one month postpartum. Environmental Health Perspectives. 2004; 112:926-931 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1242024/, accessed 3 January 2017).

EURO (1999). Monitoring ambient air quality for health impact assessment. Copenhagen: WHO Regional Office for Europe; 1999 (http://www.euro.who.int/data/assets/pdf_file/0010/119674/E67902.pdf, accessed 3 January 2017).

EUROBAT (2013). Battery Associations from North America and Europe, the Middle East and Africa join forces to strengthen workers' protection. Press release 19 June 2013 (https://eurobat.org/battery-associations-north-america-and-europe-middle-east-and-africa-join-forces-strengthen-workers%E2%80%99, accessed 26 January 2017).

FAO (2016). General standard for contaminants and toxins in food and feed (Codex Stan 193-1995). Rome: Food and Agriculture Organization; 2016 (http://www.fao.org/fao-who-codexalimentarius/sh-proxy/en/?lnk=1&url=https%253A%252F%252Fworkspace.fao.org%252Fsites%252Fc

odex%252FStandards%252FCODEX%2BSTAN%2B193-1995%252FCXS_193e. pdf, accessed 3 January 2017).

Fergusson DM, Horwood LJ (1993). The effects of lead levels on the growth of word recognition in middle childhood. International Journal of Epidemiology. 1993; 22:891-897.

Fergusson DM, Horwood LJ, Lynskey MT (1997). Early dentine lead levels and educational outcomes at 18 years. Journal of Child Psychology & Psychiatry. 1997; 38:471-478.

Fewtrell L, Kaufmann R, Prüss-Ustün A (2003). Lead – Assessing the environmental burden of disease at national and local levels. Environmental Burden of Disease Series, No.2. Geneva: World Health Organization; 2003 (http://www.who.int/quantifying_ehimpacts/publications/en/, accessed 3 January 2017).

Frith D, Yeung K, Thrush S, Hunt BJ, Hubbard JG (2005). Lead poisoning – a differential diagnosis for abdominal pain. Lancet. 2005; 366:2146 (http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(05)67893-2.pdf, accessed 3 January 2017).

Future Market Insights (2014). Lead Acid Battery Market: Global Industry Analysis and Opportunity Assessment 2014 – 2020 [website] (http://www.futuremarketinsights.com/reports/global-lead-acid-battery-market, accessed 3 January 2017).

Garza A, Vega R, Soto E (2006). Cellular mechanisms of lead neurotoxicity. Medical Science Monitor. 2006; 12:RA57-65 (http://www.medscimonit.com/download/index/idArt/447121, accessed 3 January 2017).

Gerr F, Letz R, Stokes L, Chettle D, McNeill F, Kaye W (2002). Association between bone lead concentration and blood pressure among young adults. American Journal of Industrial Medicine. 2002; 42(2):98-106.

Gottesfeld P & Pokhrel, AK (2011). Review: lead exposure in battery manufacturing and recycling in developing countries and among children in nearby communities. Journal of Occupational & Environmental Hygiene. 2011; 8(9):520-532.

Graziano JH, Popovac D, Factor-Litvak P, Shrout P, Kline J, Murphy MJ, Zhao YH, Mehmeti A, Ahmedi X, Rajovic B, Zvicer A, Nenezic DU, Lolacono NJ, Stein Z (1990). Determinants of elevated blood lead during pregnancy in a population surrounding a lead smelter in Kosovo, Yugoslavia. Environmental Health Perspectives. 1990; 89:95-100 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1567790/pdf/envhper00422-0095.pdf, accessed 3 January 2017).

Green VA, Wise GW, Callenbach J (1976). Lead poisoning. Clinical Toxicology. 1976; 9:33-51.

Greig J, Thurtle N, Cooney L et al. (2014). Association of blood lead level with neurological features in 972 children affected by an acute severe lead poisoning outbreak in Zamfara State, Northern Nigeria. PLoS ONE 9(4): e93716. (http://www.plosone.org/article/info%3Adoi%2F10.1371%2Fjournal.pone.0093716, accessed 3 January 2017).

Gulson BL, Mizon KJ, Korsch MJ, Palmer JM, Donnelly JB (2003). Mobilization of lead from human bone tissue during pregnancy and lactation — a summary of long-term research. Science of the Total Environment. 2003; 303:79-104.

Haefliger P, Mathieu-Nolf M, Lociciro S, Ndiaye C, Coly M, Diouf A, Faye AL, Sow A, Tempowski J, Pronczuk J, Filipe Junior AP, Bertollini R, Neira M (2009). Mass lead intoxication from informal used lead-acid battery recycling in Dakar, Senegal. Environmental Health Perspectives. 2009; 117(10):1535-1540 (https://ehp.niehs.nih.gov/0900696/, accessed 3 January 2017).

Haut Conseil de la santé publique (2014). Détermination de nouveaux objectifs de gestion des expositions au plomb. Synthèse et recommandations. Paris: Haut Conseil de la santé publique; 2014 (http://www.hcsp.fr/explore.cgi/avisrapportsdomaine?clefr=444, accessed 3 January 2017).

Healey N, Jones-Otazo H, Walker M, Knafla A (2010). Toxicological review and recommended toxicological reference values for environmental lead exposure in Canada. Report prepared for Health Canada (http://www.paho.org/hq/index.php?option=com_docman&task=doc_details&gid=21069&Itemid=270&Iang=en, accessed 3 January 2017).

Health Canada (2013). Final human health state of the science report on lead. Ottawa: Health Canada; 2013 (http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/dhhssrl-rpecscepsh/index-eng.php, accessed 3 January 2017).

Hodge J, Nielsen J, Dignam T, Brown MJ (2015). Small area surveillance to estimate prevalence of childhood blood and environmental lead levels. Atlanta (GA): US Centers for Disease Control and Prevention; 2015 (http://www.cdc.gov/nceh/lead/BLL_PrevalenceStudy_TrainingManual_Final_508.pdf, accessed 3 January 2017).

IHME (2016). Global lead exposure. In: GBD Compare [website]. Seattle (WA): Institute for Health Metrics and Evaluation, University of Washington; 2016 (http://vizhub.healthdata.org/gbd-compare, accessed 3 January 2017).

ILA (2015). Lead recycling fact sheet. London: International Lead Association; 2015 (http://www.ila-lead.org/UserFiles/File/ILA9927%20FS_Recycling_V08. pdf, accessed 3 January 2017).

ILA (2017) Lead use statistics. In: Lead facts [website] (http://www.ila-lead.org/lead-facts/lead-uses--statistics, accessed 8 June 2017).

International Metals Study Groups (2016). Metals Despatch. 2016; Issue No 21 (http://www.ilzsg.org/generic/pages/list.aspx?table=document&ff_aa_document_type=N&from=1, accessed 3 January 2017).

Iwata T, Yano E, Karita K, Dakeishi M, Murata K (2005). Critical dose of lead affecting postural balance in workers. American Journal of Industrial Medicine. 2005; 48(5):319-325.

Janin Y, Couinaud C, Stone A, Wise L (1985). The "lead-induced colic" syndrome in lead intoxication. Surgery Annual. 1985; 17:287-307.

JECFA (2011). Safety evaluation of certain food additives and contaminants. WHO Food Additive Series: 64. 73rd Report of the Joint FAO/WHO Expert Committee on Food Additives, pages 381-497. Geneva: World Health Organization; 2011 (http://apps.who.int/iris/bitstream/10665/44515/1/WHO_TRS_960_eng. pdf, accessed 3 January 2017).

Jennings AA (2013). Analysis of worldwide regulatory guidance values for the most commonly regulated elemental surface soil contamination. Journal of Environmental Management. 2013; 118: 72–95

Kabala C, Singh BR (2001). Fractionation and mobility of copper, lead, and zinc in soil profiles in the vicinity of a copper smelter. Journal of Environmental Quality. 2001; 30(2):485-492 (http://karnet.up.wroc.pl/~kabala/jeq-30-2-485. pdf, accessed 26 January 2017).

Kaul B, Mukerjee H (1999). Elevated blood lead and erythrocyte protoporphyrin levels of children near a battery-recycling plant in Haina, Dominican Republic. International Journal of Occupational & Environmental Health. 1999; 5(4):307-312.

Kaul B, Sandhu RS, Depratt C, Reyes F (1999). Follow-up screening of lead-poisoned children near an auto battery recycling plant, Haina, Dominican Republic. Environmental Health Perspectives. 1999; 107(11):917–920 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1566703/, accessed 3 January 2017).

Koller K, Brown T, Spurgeon A, Levy L (2004). Recent developments in low-level lead exposure and intellectual impairment in children. Environmental Health Perspectives. 2004; 112(9):987-994 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1247191/pdf/ehp0112-000987.pdf, accessed 3 January 2017).

Kosnett MJ, Wedeen RP, Rothenburg SJ, Hipkins KL, Materna BL, Schwartz BS, Hu H, Woolf A (2007). Recommendations for medical management of adult lead exposure. Environmental Health Perspectives. 2007; 115(3):463-471 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1849937/pdf/ehp0115-000463.pdf, accessed 26 January 2017).

Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, Canfield RL, Dietrich KN, Bornschein R, Greene T, Rothenberg SJ, Needleman HL, Schnaas L, Wasserman G, Graziano J, Roberts R. (2005). Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environmental Health Perspectives.2005; 113(7):894–899. (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1257652/pdf/ehp0113-000894.pdf, accessed 3 January 2017).

Levallois P, Lavoie M, Goulet L, Nanel AJ, Gingras S (1991). Blood lead levels in children and pregnant women living near a lead-reclamation plant. Canadian Medical Association Journal. 1991; 144(7): 877-885.

Lewington S, Clarke R, Qizilbash N, Peto R, Collins R; Prospective Studies Collaboration (2002). Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet 2002; 360(9349):1903-1913 (http://www.thelancet.com/pdfs/journals/lancet/PIIS0140-6736(02)11911-8.pdf; Erratum

in: Lancet. 2003; 361(9362):1060 http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(03)12816-4/fulltext, accessed 3 January 2017).

Lidsky TI, Schneider JS (2003). Lead neurotoxicity in children: basic mechanisms and clinical correlates. Brain. 2003; 126:5-19 (http://brain.oxfordjournals.org/content/126/1/5.long, accessed 3 January 2017).

Loghman-Adham M (1997). Renal effects of environmental and occupational lead exposure. Environmental Health Perspectives. 1997; 105:928-939 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1470371/pdf/envhper00322-0042.pdf, accessed 3 January 2017).

Manhart A, Schleicher T (2015). The recycling chain for used lead-acid batteries in Ghana. Freiburg: Oeko-Institut; 2015 (http://www.econet.international/index. php?id=3, accessed 3 January 2017).

Manhart A, Smera T, Kuepouo G, Mathai D, Mng'anya S, Schleicher T (2016). The deadly business – findings from the lead recycling Africa project. Freiburg: Oeko-Institut; 2016 (https://www.oeko.de/oekodoc/2549/2016-076-de.pdf, accessed 3 January 2017).

Markowitz ME, Weinberger HL (1990). Immobilization-related lead toxicity in previously lead-poisoned children. Pediatrics 1990; 86:455-457 (http://pediatrics.aappublications.org/content/pediatrics/86/3/455.full.pdf, accessed 3 January 2017).

Matte TD, Figueroa JP, Ostrowski S, Burr G, Jackson-Hunt L, Baker EL (1991). Lead exposure from conventional and cottage lead smelting in Jamaica. Archives of Environmental Contamination & Toxicology. 1991; 21:65–71.

McNutt TK, Chambers-Emerson J, Dethlefsen M, Shah R (2001). Bite the bullet: lead poisoning after ingestion of 206 lead bullets. Veterinary & Human Toxicology. 2001; 43:288-289.

Mendelsohn AL, Dreyer BP, Fierman AH, Rosen CM, Legano LA, Kruger HA, Lim SW, Courtlandt CD (1998). Low-level lead exposure and behavior in early childhood. Pediatrics. 1998; 101:E10 (http://pediatrics.aappublications.org/content/pediatrics/101/3/e10.full.pdf, accessed 3 January 2017).

Mielke HW, Reagan PL (1998). Soil is an important pathway of human lead exposure. Environmental Health Perspectives. 1998; 106(Suppl 1):217-229 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1533263/pdf/envhper00536-0227.pdf, accessed 3 January 2017).

MoEP (2012). Ambient air quality standards GB 3095-2012. Beijing: Ministry of Environmental Protection; 2012 (http://kjs.mep.gov.cn/hjbhbz/bzwb/dqhjbh/dqhjzlbz/201203/W020120410330232398521.pdf, accessed 3 January 2017).

Mushak P (1993). New directions in the toxicokinetics of human lead exposure. Neurotoxicology. 1993; 14:29-42.

NAS (1972). Lead: airborne lead in perspective. Committee on Biologic Effects of Atmospheric Pollutants, Division of Medical Sciences. Washington (DC): National Academy of Sciences; 1972.

Nawrot TS, Thijs L, Den Hond EM, Roels HA, Staessen JA (2002). An epidemiological re-appraisal of the association between blood pressure and blood lead: a meta-analysis. Journal of Human Hypertension. 2002; 16:123-131 (http://www.nature.com/jhh/journal/v16/n2/pdf/1001300a.pdf, accessed 3 January 2017).

Needleman H (2004). Lead poisoning. Annual Review of Medicine. 2004; 55:209-222.

Needleman HL, Riess JA, Tobin MJ, Biesecker G, Greenhouse JB (1996). Bone lead levels and delinquent behavior. JAMA. 1996; 275:363-369.

Needleman HL, Schell A, Bellinger D, Leviton A, Allred EN (1990). The long-term effects of exposure to low doses of lead in childhood. An 11-year follow-up report. New England Journal of Medicine. 1990; 322:83-88 (http://www.nejm.org/doi/full/10.1056/NEJM199001113220203#t=article, accessed 3 January 2017).

NEPC (2016). National Environment Protection (Ambient Air Quality) Measure. Canberra: National Environment Protection Council; 2016 (https://www.legislation.gov.au/Details/F2016C00215, accessed 3 January 2017).

NTP (2012). Health effects of low-level lead. National Toxicology Program Monograph. Bethesda (MD): National Institutes of Health; 2012 (http://ntp. niehs.nih.gov/pubhealth/hat/noms/lead/index.html, accessed 3 January 2017).

OSHA (1978). Lead Standard 29 CFR 1910.1025. Washington (DC): Occupational Safety and Health Administration, US Department of Labor; 1978. (https://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=10030, accessed 3 January 2017).

OSHA (2002) Lead – secondary lead smelter. In: eTools [website]. Washington (DC): Occupational Safety and Health Administration; 2002 (https://www.osha.gov/SLTC/etools/leadsmelter/index.html, accessed 3 January 2017).

Otto DA, Fox DA (1993). Auditory and visual dysfunction following lead exposure. Neurotoxicology. 1993; 14(2-3):191-207.

Paddock RC (2016). The toxic toll of Indonesia's battery recyclers. National Geographic. 31 May 2016 (http://news.nationalgeographic.com/2016/05/indonesia-s-toxic-toll/, accessed 3 January 2017).

Perlstein MA, Attala R (1966). Neurologic sequelae of plumbism in children. Clinical Pediatrics. 1966; 5:292-298.

Pichery C, Bellanger M, Zmirou-Navier D, Glorennec P, Hartemann P, Grandjean P (2011). Childhood lead exposure in France: benefit estimation and partial cost-benefit analysis of lead hazard control. Environmental Health. 2011; 10:44 (https://ehjournal.biomedcentral.com/articles/10.1186/1476-069X-10-44, accessed 3 January 2017).

Rabinowitz MB (1991). Toxicokinetics of bone lead. Environmental Health Perspectives. 1992; 91:33-37 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1519353/pdf/envhper00387-0038.pdf, accessed 3 January 2017).

Ratzon N, Froom P, Leikin E, Kristal-Boneh E, Ribak J (2000). Effect of exposure to lead on postural control in workers. Occupational & Environmental Medicine. 2000; 57:201-203 (http://oem.bmj.com/content/57/3/201.full, accessed 3 January 2017).

Rothenberg SJ, Schnaas L, Salgado-Valladares M, Casanueva E, Geller AM, Hudnell HK, Fox DA (2002). Increased ERG a- and b-wave amplitudes in 7- to 10-year-old children resulting from prenatal lead exposure. Investigative Ophthalmology & Visual Science. 2002; 43:2036-2044 (http://iovs.arvojournals.org/article.aspx?articleid=2123755, accessed 3 January 2017).

Rubens O, Logina I, Kravale I, Eglite M, Donaghy M (2001). Peripheral neuropathy in chronic occupational inorganic lead exposure: a clinical and electrophysiological study. Journal of Neurology, Neurosurgery & Psychiatry. 2001; 71:200-204 (http://jnnp.bmj.com/content/71/2/200.longm, accessed 3 January 2017).

Safe Work Australia (2013). Workplace exposure standards for airborne contaminants. Canberra: Safe Work Australia; 2013 (http://www.safeworkaustralia. gov.au/sites/SWA/about/Publications/Documents/772/Workplace-exposure-standards-airborne-contaminants.pdf, accessed 3 January 2017).

Schwartz BS, Lee BK, Bandeen-Roche K, Stewart W, Bolla K, Links J, Weaver V, Todd A (2005). Occupational lead exposure and longitudinal decline in neurobehavioral test scores. Epidemiology. 2005; 16:106-113.

Schwartz J, Landrigan PJ, Baker Jnr EL, Orenstein WA, von Lindern IH (1990). Lead-induced anemia: dose-response relationships and evidence for a threshold. American Journal of Public Health. 1990; 80:165-168 (http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1404621/pdf/amjph00215-0029.pdf, accessed 3 January 2017).

Selevan SG, Rice DC, Hogan KA, Euling SY, Pfahles-Hutchens A, Bethel J (2003). Blood lead concentration and delayed puberty in girls. New England Journal of Medicine. 2003; 348:1527-3156. (http://www.nejm.org/doi/full/10.1056/NEJMoa020880#t=article, accessed 3 January 2017).

Silbergeld EK, Schwartz J, Mahaffey K (1988). Lead and osteoporosis: mobilization of lead from bone in postmenopausal women. Environmental Research. 1988; 47:79–94.

Stokes L, Letz R, Gerr F, Kolczak M, McNeill FE, Chettle DR, Kaye WE (1998). Neurotoxicity in young adults 20 years after childhood exposure to lead: the Bunker Hill experience. Occupational & Environmental Medicine. 1998; 55:507-516 (http://oem.bmj.com/content/55/8/507.full.pdf+html, accessed 3 January 2017).

Stollery BT, Broadbent DE, Banks HA, Lee WR (1991). Short term prospective study of cognitive functioning in lead workers. British Journal of Industrial Medicine. 1991; 48:739-749 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1035449/pdf/brjindmed00035-0019.pdf, accessed 3 January 2017).

Suplido ML, Ong CN (2000). Lead exposure among small-scale battery recyclers, automobile radiator mechanics, and their children in Manila, the Philippines. Environmental Research Section A. 2000; 82:231-238.

ten Bruggenkate CM, Lopes Cardozo E, Maaskant P, van der Waal I (1975). Lead poisoning with pigmentation of the oral mucosa. Review of the literature and report of a case. Oral Surgery, Oral Medicine & Oral Pathology. 1975; 39(5):747-753.

Thurtle N, Grieg J, Cooney L, Amitai Y, Ariti C, Brown MJ, Kosnett MJ, Moussally K, Sani-Gwarzo N, Akpan H, Shanks L, Dargan PI (2014). Description of 3180 courses of chelation with dimercaptosuccinic acid in children ≤5 years with severe lead poisoning in Zamfara, northern Nigeria: a retrospective analysis of programme data. PLOS Medicine. 2014; 11(10):1-18 (http://www.plosmedicine. org/article/info%3Adoi%2F10.1371%2Fjournal.pmed.1001739, accessed 3 January 2017).

Tong S (1998). Lead exposure and cognitive development: persistence and a dynamic pattern. Journal of Paediatrics & Child Health. 1998; 34:114-118.

Tong S, Baghurst P, McMichael A, Sawyer M, Mudge J (1996). Lifetime exposure to environmental lead and children's intelligence at 11-13 years: the Port Pirie cohort study. British Medical Journal. 1996; 312:1569-1575 (http://www.bmj.com/content/312/7046/1569; accessed 3 January 2017).

Tong S, Baghurst PA, Sawyer MG, Burns J, McMichael AJ (1998). Declining blood lead levels and changes in cognitive function during childhood: the Port Pirie Cohort Study. JAMA. 1998; 280:1915-1919 (http://jamanetwork.com/journals/jama/fullarticle/188249, accessed 3 January 2017).

Trasande L, Liu Y (2011). Reducing the staggering costs of environmental disease in children, estimated at \$76.6 billion in 2008. Health Affairs (Millwood). 2011; 30(5):863–870 (http://content.healthaffairs.org/content/30/5/863.long, accessed 3 January 2017).

Troesken W (2006). Lead exposure and eclampsia in Britain, 1883-1934. Environmental Research. 2006; 101(3):395-400.

UNEP (2003). Technical guidelines for the environmentally sound management of waste lead-acid batteries. Secretariat of the Basel Convention. Basel Convention series/SBC No. 2003/9. Geneva: Basel convention Secretariat; 2003 (http://www.basel.int/Portals/4/Basel%20Convention/docs/pub/techguid/tech-wasteacid.pdf, accessed 3 January 2017).

UNEP (2004). National management plans for used lead acid batteries. Training manual for the preparation of national used lead acid batteries environmentally sound management plans in the context of the implementation of the Basel Convention. Basel Convention Series / SBC No 2004/5. Geneva: Basel convention Secretariat; 2004 (http://archive.basel.int/meetings/sbc/workdoc/tm-ulab/tm_ulab.pdf, accessed 3 January 2017).

UNEP (2010). Final review of scientific information on lead. Nairobi: United Nations Environment Programme; 2010 (http://www.unep.org/chemicalsandwaste/

Portals/9/Lead_Cadmium/docs/Interim_reviews/UNEP_GC26_INF_11_Add_1_Final_UNEP_Lead_review_and_apppendix_Dec_2010.pdf, accessed 3 January 2017).

UNEP (2013). Environmental risks and challenges of anthropogenic metals flows and cycles. Paris: United Nations Environment Programme; 2013 (http://www.unep.org/resourcepanel/publications/environmentalchallengesmetals/tabid/106142/default.aspx, accessed 3 January 2017).

US EPA (1993). Personal Air Sampling and Air Monitoring Requirements under 29 CFR 1910.120. Publication 9360.B-17FS. Washington (DC): United States Environmental Protection Agency; 1993.

US EPA (2001). Lead; identification of dangerous levels of lead; Final Rule. Federal Register. 2001; 66(4):1205-1240 (https://www.gpo.gov/fdsys/pkg/FR-2001-01-05/pdf/01-84.pdf, accessed 3 January 2017).

US EPA (2016). Fact sheet: Decision - National Ambient Air Quality Standards for lead. Washington (DC): United States Environmental Protection Agency; 2016 (https://www.epa.gov/sites/production/files/2016-09/documents/pb_naaqs_nfr_fact_sheet.pdf, accessed 3 January 2017).

Valciukas JA, Lilis R, Eisinger J, Blumberg WE, Fischbein A, Selikoff IJ (1978). Behavioral indicators of lead neurotoxicity: results of a clinical field survey. International Archives of Occupational & Environmental Health. 1978; 41(4):217-236.

van der Kuijp TJ, Huang L, Cherry CR (2013). Health hazards of China's lead-acid battery industry: a review of its market drivers, production processes, and health impacts. Environmental Health. 2013; 12:61 (http://ehjournal.biomedcentral. com/articles/10.1186/1476-069X-12-61, accessed 3 January 2017).

Wang J-D, Jang C-S, Hwang Y-H, Chen Z-S (1992). Lead contamination around a kindergarten near a battery recycling plant. Bulletin of Environmental Contamination & Toxicology. 1992; 49:23-30 (https://www.researchgate.net/publication/21764004_Lead_contamination_around_a_kindergarten_near_a_battery_recycling_plant, accessed 25 January 2017).

Wedeen RP (1988). Bone lead, hypertension, and lead nephropathy. Environmental Health Perspectives. 1988; 78:57-60 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1474623/pdf/envhper00429-0059.pdf, accessed 3 January 2017).

Weidenhamer JD, Kobunski PA, Kuepouo G, Corbin RW, Gottesfeld P (2014). Lead exposure from aluminum cookware in Cameroon. Science of the Total Environment. 2014; 496:339-47.

Weidenhamer JD, Fitzpatrick MP, Biro AM, Kobunski PA, Hudson MR, Corbin RW, Gottesfeld P (2017). Metal exposures from aluminum cookware: an unrecognized public health risk in developing countries. Science of the Total Environment. 2017; 579: 805-813.).

Were FH, Kamau GN, Shiundu PM, Wafula GA, Moturi CM (2012). Air and blood lead levels in lead acid battery recycling and manufacturing plants in Kenya. Journal of Occupational & Environmental Hygiene. 2012; 9(5):340-344.

White RF, Diamond R, Proctor S, Morey C, Hu H (1993). Residual cognitive deficits 50 years after lead poisoning during childhood. British Journal of Industrial Medicine. 1993; 50(7):613-622 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1035497/pdf/brjindmed00007-0037.pdf, accessed 3 January 2017).

WHO (1995). Lead. Environmental Health Criteria 165. Geneva: World Health Organization; 1995 (http://www.inchem.org/documents/ehc/ehc/ehc165.htm, accessed 3 January 2017).

WHO (2000). Air quality guidelines for Europe, 2nd edition. Copenhagen: WHO Regional Office for Europe; 2000 (http://www.euro.who.int/__data/assets/pdf_file/0005/74732/E71922.pdf?ua=1, accessed 3 January 2017).

WHO (2010a) Childhood lead poisoning. Geneva: World Health Organization; 2010 (http://www.who.int/ceh/publications/leadguidance.pdf, accessed 3 January 2017).

WHO (2010b). WHO Human health risk assessment toolkit. Geneva: World Health Organization; 2010 (http://www.who.int/ipcs/methods/harmonization/areas/ra_toolkit/en/, accessed 3 January 2017).

WHO (2010c). WHO Guidelines on drawing blood: best practices in phlebotomy. Geneva: World Health Organization; 2010 (http://www.who.int/injection_safety/sign/drawing_blood_best/en/, accessed 3 January 2017).

WHO (2011a). Brief guide to analytical methods for measuring lead in blood. Geneva: World Health Organization; 2011 (http://www.who.int/ipcs/assessment/public_health/lead_blood.pdf, accessed 3 January 2017).

WHO (2011b). Guidelines for Drinking-water Quality, 4th edition. Geneva: World Health Organization; 2011 (http://www.who.int/water_sanitation_health/publications/dwq-guidelines-4/en/, accessed 3 January 2017).

Wilhelm M, Beinzow B, Angerer J, Schulz C (2010). Reassessment of critical lead effects by the German Human Biomonitoring Commission results in suspension of the human biomonitoring values (HBM I and HBM II) for lead in blood of children and adults. International Journal of Hygiene and Environmental Health. 2010; 213:265-269 (http://www.sciencedirect.com/science/article/pii/S143846391000043X, accessed 3 January 2017).

Williamson AM, Teo RKC (1986). Neurobehavioural effects of occupational exposure to lead. British Journal of Industrial Medicine. 1986; 43(6):374-380 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1007666/pdf/brjindmed00170-0014. pdf, accessed 3 January 2017).

Winship KA (1989). Toxicity of lead: a review. Adverse Drug Reactions & Acute Poisoning Reviews. 1989; 8(3): 117-152.

Wright JP, Dietrich KN, Ris MD, Hornung RW, Wessel SD, Lanphear BP, Ho M, Rae MN (2008). Association of prenatal and childhood blood lead concentrations with criminal arrests in early adulthood. PLoS Medicine. 2008;5(5):e101 (http://journals.plos.org/plosmedicine/article/file?id=10.1371/journal.pmed.0050101&type=printable, accessed 3 January 2017).

Wu T, Buck GM, Mendola P (2003). Blood lead levels and sexual maturation in U.S. girls: the Third National Health and Nutrition Examination Survey, 1988-1994. Environmental Health Perspectives. 2003; 111(5):737-741 (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1241484/pdf/ehp0111-000737.pdf, accessed 3 January 2017).

Zhang F, Liu Y, Zhang H, Ban Y, Wang J, Liu J, Zhong L, Chen X, Zhu B (2016). Investigation and evaluation of children's blood lead levels around a lead battery factory and influencing factors. International Journal of Environmental Research and Public Health. 2016; 13:541. (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4923998/pdf/ijerph-13-00541.pdf, accessed 3 January 2017).

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