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ANNEX

Lead in drinking water: exposure, mitigation and societal cost

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ANNEX I. OVERVIEW OF LEAD SOURCES AND HEALTH EFFECTS

1. Lead in the environment

Annex I.1 provides background information on lead's properties, uses and sources, and gives illustrative levels of lead in environmental media such as air, soils, indoor dust, and human diets.l.

Lead: Properties, uses and environmental fate

Lead is a heavy metal with high density and a low-melting point (327°C). It is soft; flexible; and a good insulator of sound, vibrations and radiation. It is also corrosion-resistant. However, when lead-containing materials, such as storage tanks, pipes, brass fittings[1] and solder used in plumbing, come in contact with acids such as acetic acid, nitric acid, or, simply acidic and soft water, then lead dissolves.

Lead occurs naturally in the environment associated with lead-bearing ore deposits. Lead is relatively immobile in soils with high organic matter content and metal oxides and in most aquifers but may be dissolved in acidic groundwater (Deutch and Siegel 1997). Natural emissions include lead released from volcanoes, seawater sprays, forest fires, and wind-borne soil particles in remote areas (Nriagu 1989).

Lead's ubiquitous occurrence and extensive environmental contamination have resulted from anthropogenic sources due to historic or ongoing mining, smelting, coal and solid waste burning and widespread use since 4000 BC (e.g. Henderson 2000). The cumulative production of lead rose from 20 million MT 2500 years before present to 300 million MT today, with the production over the last 60 years accounting for over 60% of the entire estimated lead production over the previous five millennia (Mao et al 2008). Interestingly, currently recycling accounts for 75% of all lead produced (International Lead Association 2014). Uses in products with no recycling potential included lead containing cookware and utensils; toys; paints; cosmetics; and leaded gasoline. Most of these uses have now been completely phased out but have contributed to the current anthropogenic burden of lead in soils and the aquatic environment, known as lead legacy. Notable recyclable applications of lead include its use in: storage lead-acid batteries; roofing, cable covers and construction, in general; radiation shields; cans; fishing weights and ammunition; and, following lead pipe replacement, pipes and solder used in plumbing (International Lead Association 2014).

Anthropogenic lead is found in two forms: inorganic, as in industry emissions, paints and drinking water from corroded lead plumbing; and organic, as the tetraethyl lead added in gasoline and released to air through car and aviation emissions. Both forms of lead can either cling on particulate material and soil particles or simply be in soluble form. In either case, lead is retained in the soil but could also be dispersed via wind, precipitation, soil erosion, and runoff in large distances away from its emission point to be deposited in remote environments, such as upland freshwater or ocean sediments (a detailed review of the lead cycle in the environment can be found in Mushak 2011).

Lead contamination of air, soils, and indoor dust

Present air lead levels are 10^4 higher today than in the pre-historic era, as shown by estimates of natural lead air levels (Nriagu 1979) and present-time measurements (US EPA 2016; EEA 2015). Phasing out lead from gasoline in the 1970s-1990s resulted in decreases by 99% of lead emissions from cars on a global scale (e.g. EEA 2015; US EPA 2016).

In the UK, lead emissions, prior to 1999 arose primarily from the combustion of leaded petrol (Bailey et al 2016). The lead content of petrol was reduced from around 0.34 g/l to 0.143 g/l in 1986. From 1987, sales of unleaded petrol increased, particularly as a result of the increased use of cars fitted with three-way catalysts. Leaded petrol was then phased out from general sale at the end of 1999. These changes have caused a significant decline in total lead emissions across the UK between 1990 and 2000. The UK-wide emissions of Pb are now dominated by combustion sources (mainly of solid fuels, biomass and lubricants in industrial and domestic sectors), and from metal production processes at foundries and iron and steel works (Bailey et al 2016).

As Annex I.1-Table 1a shows, current lead air concentrations are lower than 0.25 µg/g in EU Member States (EEA 2015); in some cases as in Glasgow, with post-2007 lead levels being below 0.007 µg/m3 (UK National Air Quality Information Archive n.d.), lead concentrations in air are similar to background lead air concentrations such as those recorded in the Arctic in the period before banning lead from gasoline (Pacyna and Ottar 1985). Street dust lead levels have not been reported as systematically as air lead concentrations before and after phasing out lead from gasoline but the illustrative data shown in Annex I-Table1a suggest that they may still be a considerable source of exposure to lead especially in urban areas.

Lead-related industries such as mining, smelting, refineries, and battery recycling factories can substantially contribute to human exposure to lead in their close and wider vicinity (Annex I.1-Table 1b). A particular problem is that the

[1]Brass is an alloy of copper and zinc and often contains lead impurities. Solder is an alloy of tin with lead, antimony, or silver.

presence of this type of industry increases dramatically indoor air lead (e.g. Ordonez et al 2003) and contaminates the soils (e.g. urban gardens and farmland) of the surrounding areas (e.g. Hartwell et al 1983).

Soil lead concentrations have not varied widely over time because in contrast to atmospheric lead from cars and the industry, lead accumulates in the soil. For example, Hilts (2003) reported that regulation of lead emission from lead-emitting industry (e.g. primary smelters) has not always translated into lower levels of lead in soils. The illustrative data for soil lead concentrations presented in Annex I-Table 1b and 1c show that lead in soil can reach levels several orders of magnitude higher than the guideline values for residential, commercial and agricultural land in EU, which are 450, 450 and 750 mg/kg (CLEA 2002), respectively. The high levels in Derbyshire, UK reflect the long-term mining activity and associated accumulation of lead in the soils (e.g. McGrath and Loveland 1992).

Lead-based paint can be a substantial source of residential exposure to lead indoor air dust (Annex I-Table 1d). Indoor dust lead levels increase with the age of the house as a result of the extensive use of lead-based paint before banning in the 1960s in the UK and in1978 in the USA. Lead-based paint is still used widely in developing countries and for painting outdoor constructions, e.g. bridges, worldwide (Weinberg and Clark 2012). A major health risk for young children, however, is the original coats of lead-based paints remaining in the inside and outside walls of houses built before the banning of lead-based paints (WHO 2010). Old paint is more vulnerable to weathering and peeling off and thus tends to contribute to exposures of children to lead during playtime, in the form of flaking paint or simply dust. The illustrative data in Annex I-Table 1d show that even after the banning of lead in gasoline and paint, the indoor environments remained contaminated with lead concentrations as high as 308 µg/g (on average) in Edinburgh (Laxen et al 1987); 1300 µg/g (50th percentile) in Montreal (Levallois et al 2014); and 1043 µg/g (on average) in various US cities (Clark et al 2004). However, these levels are lower than those measured in the soils of areas with heavy urban traffic or near housing or constructions built before the removal of lead from paint (Annex I-Table 1c).

Lead in food is a major source of exposure to lead. Unlike other sources of exposure such as contaminated soil and indoor dust from phased-uses of lead in gasoline and paint, which affect mostly young children, lead in food affects adults and children alike (Mushak 2011). Lead contamination of food increases with lead levels in air and soil; storage and cooking in lead-containing kitchenware; storage in lead-seamed cans; and lead levels in the water used for cooking. Levels in human diets have clearly declined since phasing out lead solder from cans in the 1980s (Bolger et al 1991). EFSA (2010) found that in European diets lead can be higher in products associated with root vegetables and cereals than in other types of food. Levels of lead in food vary considerably by country and year and are not shown here. Lead has also contaminated the food chain: it is higher in waterfowl and top predators, e.g. eagles (e.g. Fisher et al 2006).

Data specific to Scotland are presented in Annex II

Annex I.1-Table 1a: Lead concentrations in air and street-dust pre- and post-banning lead from car fuel.			
Source of lead	Receptor	Typical concentrations	References
Estimated prehistoric levels	Air	2.6 × 10 $^{-4}$µg / m ^{3}	Nriagu 1979
Pre-banning use from gasoline (data from 1970s – 1980s)	Air	Background (Arctic): Up to 9 x 10-3 µg/m3	Pacyna and Ottar 1985
		Urban areas: 0.1 $^{-3}$ µg/m^3	Thomas 1988
		Rural areas: 0.04–0.17 µg/m^3	Strehlow and Barltrop 1987; Thomas 1999
	Outdoor dust	UK –urban average: 970 µg/g	Day et al. 1975 cited in Mushak 2011
		UK – rural average: 85 µg/g	
		The Netherlands –average: 5000 µg/g	Rameau 1973 cited in Mushak 2011
		New Zealand (urban): 1160 µg/g	Fergusson and Schroeder 1985
		USA cities: 300-18,000 µg/g	Nriagu 1978 cited in Mushak 2011
Post- banning lead from gasoline	Air	1995-Europe:<0.02 to >1 µg/m^3	EEA n.d.
		1995-Scotland (Glasgow): 0.051 µg/m^3	UK National Air Quality Information Archive n.d.
		1994-USA: 0.033 µg/m^3	US EPA n.d. cited in Mushak
		2004-Australia: 0.016 µg/m^3	Cohen et al 2005
		2004-Scotland (Glasgow):0.014 µg/m^3	UK National Air Quality Information Archive n.d.
		2004-USA: 0.27 µg/m^3	US EPA n.d. cited in Mushak
		Post-2007: European Union average (except Italy)<0.25 µg/m^3	EEA 2015
		Post-2007-Scotland (Glasgow): <0.007	UK National Air Quality Information Archive n.d.
		Post-2007-USA: 0.02-0.1 µg/m^3	US EPA n.d.
	Outdoor dust	Oslo, Norway: 180 µg/m3	de Miguel et al. (1997)
		USA (urban averages): 100-588	Sutherland et al 2003 and Gillies et al 1999 cited in Mushak 2011
		Madrid, Spain: 1927	de Miguel et al. (1997)
		Honk Kong: 1061-1209	Ho et al 2003 cited in Mushak 2011

Annex I-Table1b: Illustrative lead concentrations in air and indoor dust in the vicinity of lead-related industries.			
Source	Receptor	Concentrations	References
Mines/ Smelters/ battery plants	Air	Near (<2.5 mi): 3.8-10.3 µg/m3	US EPA 1977 and Yankel et al 1977 cited in Mushak
		Far: <5 µg/m3	US EPA 1977 and Yankel et al 1977 cited in Mushak
	Household dust near a smelter	220-1322 µg/g	Ordonez et al 2003
	Soils	USA (yard soils): 295-821 ppm	Hartwell et al 1983; Succop et al 1998 and Bornscheim et al 1991 cited in Mushak 2011; Murgueytio et al 1998
		Derbyshire, UK: Pre-1975: 420-13969 ppm 1992: 3 – 16338 ppm	Barltrop 1975 cited in Mushak McGrath and Loveland 1992
		Arnhem, The Netherlands: 240 ppm	Diemel et al 1981

Annex I.1-Table 1c. Illustrative lead concentrations in urban soils.		
Source	Concentrations in soil	References (cited in Mushak 2011)
Urban emissions	USA: 7 – 13,240 ppm	Clark et al 2004; Chirinje et al 2004; Angle and McIntyre 1982; Rabinowitz and Bellinger 1988
	UK: up to 14,100 ppm	Culbard et al 1988
	The Netherlands: 43-336	Brunekreef et al 1983
	Toronto, Canada: 48-54	O'Heany et al 1988
Lead-based paint*	USA (old housing) Near: 2349-2529 ppm Far: 209-447 ppm	Terhaar and Aranow 1974
	New Zeland (near old housing): 21-1890 ppm	Bates et al 1995
	USA (Remediated bridge) Under: 8127 ppm Far: 197 ppm	Landrigan et al 1982
	USA (houses built before 1978): up to: 200 to more than 5000 ppm	NSLAH 2001 cited in Mushak and Mushak
	USA (houses built before 1978): 200-400	

*Measurements taken near the walls or window-frames of old housing and urban constructions.

Annex I.1-Table 1d. Illustrative lead concentrations in indoor air due to lead-based paint		
Source	Concentration in indoor dust	References
Lead-based paint	Edinburgh (average): 308 µg/g Range: 43-13600 µg/g increasing with age of housing	Laxen et al 1987
	USA cities (average): 1043 µg/g	Clark et al 2004
	Ottawa (average): 233 µg/g	Rasmussen et al 2001
	Montreal (paint-chips / 50th percentile): 1300 µg/g	Levallois et al 2014

ANNEX I. OVERVIEW OF LEAD SOURCES AND HEALTH EFFECTS

I.2. Lead exposure

Lead intake and uptake

Lead exposure depends on lead intake and uptake (Mashuk 2011). Daily lead intake is typically indexed as the product of lead concentration in some medium and the mass (diet) or volume (air or water) of a lead-containing medium taken in daily. Lead intake data can be applied in lead source apportionment and risk assessment to help develop source-specific lead mitigation policies.

Lead uptake refers to the absorption of lead in the body as the percentage of lead retained in the body or as absorption in the blood, bone, dentine (teeth) or other tissues. Approximately 30–40% of inhaled lead reaches the bloodstream (Philip and Gerson 1994). Absorption of ingested lead depends on the nutritional status of an individual; the age of the individual exposed; and the type and lead content of material ingested (i.e. food, water, dust and paint), with water lead being absorbed more easily than food lead (Moore et al 1985; Lacey et al 1985). In general, lead absorption is increased when dietary intakes of phosphorus, calcium, zinc and iron are low (Rabinowitz et al 1980; Blake and Mann 1983). For children already suffering from anaemia or iron deficiency, absorption of ingested lead may be enhanced (Mahaffey 1981). It has been suggested that dietary intake of calcium, zinc, and iron may modify the absorption of lead (Osman et al 1998; Wright 2003). Ballew and Bowman (2001) argued that improvement of children's diets should accompany efforts to eliminate their exposure to lead.

Adults have been reported to absorb an average of 10 to 15% of ingested lead but this can reach up to 22% (Dore 2015). Infants, young children and pregnant women can absorb up to 50% more than the amount reported for adults (Philip and Gerson 1994; Markowitz 2000). Infants and children are more vulnerable than adults to the same level of lead intake because of a higher absorption rate, which can be explained by their higher metabolic rate and rapid body growth (JECFA 2011; WHO 2011). Lead also crosses the placental barrier beginning at 12 weeks gestation, with concentrations of lead in umbilical cord blood being 80–100% of the maternal blood lead level (Philip and Gerson 1994; Gulson et al 1998).

In infants and young children at intakes of lead lower than 4 µg/kg of body weight/day lead is not retained while at intakes greater than 5 µg/kg of body weight/day net retention of lead averages 32% of intake (e.g. Ryu et al

1983). Accordingly, the WHO (2011) had recommended a provisional tolerable weekly intake of 25 µg of lead per kg of body weight (equivalent to 3.5 µg/kg body weight/day in infants) from all sources but, as of today, there has not been agreed a safe threshold for lead intake (JFWEC 2011; CDC 2012; ASDTR 2017).

It takes approximately 4 to 6 weeks for lead to be transported from blood to soft tissues (Philip and Gerson 1994). The half-life of lead in blood and the various soft tissues (i.e. the time required for lead to loose half of its amount) is about 30-40 days for adults but may be variably longer for children (Papanikolaou et al 2005). For this reason, blood lead tests in adults a month or longer after an incident of lead exposure has ceased may underestimate overall lead exposure. The half-life of lead in the skeletal pool is 17-27 years; as a result of this, a 80-95% of the total body burden of lead in adults is found in the skeleton as compared with approximately 75% in children (O'Flaherty 1995). It has also been shown that lead crosses the placental barrier beginning at 12 weeks gestation and continuing throughout development up to birth, with concentrations of lead in umbilical cord blood being 80–100% of the maternal blood lead level (Philip and Gerson 1994; Gulson et al 1998).

Inorganic lead is not metabolised in the body. Unabsorbed dietary lead is eliminated in the faeces; absorbed lead but not retained in the tissues and skeleton is excreted unchanged in urine (Mashuk 2011). Eventually, under conditions of chronic exposure a steady-state distribution of retained and recently taken in lead exists between blood, soft tissues and the skeletal pool (Papanikolaou et al 2005). However, several conditions can increase blood lead levels through bone lead mobilisation, such as pregnancy, lactation, chemotherapy, post-menopausal osteoporosis, and potentially hyperthyroidism (Gulson et al 1998; Klein et al 1998; Tothill et al 1989)

Annex I.2-Table 1 compares levels of daily lead intake in 1-2-year old children from various sources and studies during the past 50 years. As of today, food is one of the major sources of lead intake (EFSA 2010; WHO 2011) but before banning of lead in gasoline, air was a more important source of exposure. Lead intake from water declined in line with reductions in the lead standard in drinking water: lead intake pre-1995 varied from 3 to 100 µg/day in tap water (US EPA 2006) whereas intakes post-1995 could exceed 21 µg / day in foodstuff including tap water (EFSA 2010).

However, tap water is not the only source of lead exposure for infants and 1-2 year old children, as even after banning lead from gasoline, there is potential for considerable lead intake from paint, toys dust, soil, and foodstuff. A recent report by EFSA (2010) showed that cereals, leafy vegetables and drinking water, were the major dietary sources of lead exposure in the general European population, with cereals and tap water contributing by 9 and 4%, respectively, to total lead exposure.

Annex I.2-Table 1. Illustrative changes of lead daily intake levels in children 1 to 2 years old.				
Environmental medium	Lead concentration	Mass or Volume /day	Daily lead intake	References
Air	0.2 µg/m³	2.5 m³	0.5 µg	WHO 2011
Air 1970-1973:	Before banning lead from car fuel	6.8 m³	7.3 µg	US EPA 2008 cited in Mashuk 2011
Air 1994	After banning lead from car fuel	6.8 m³	0.34 µg	US EPA 2008 cited in Mashuk 2011
Tap water	25 µg/l	1 L	25 µg	Mashuk 2011
Tap water	10 µg/l	0.76 L	7.6 µg	WHO 2011
Tap water	20 µg/l	2 L	40 µg	Galal-Gorchev 1991
Tap water 1993	Before applying a lead standard at 15 µg/l	0.31L	3-100 µg	US EPA 2006
Tap water 2001-2003	After applying a lead standard at 15 µg/l	0.31L	0-20 µg	US EPA 2006
Water used for powdered drinks Early 1980s		441g	2.1 µg	US EPA 1986 and Pennington 1983 cited in Mashuk 2011
Food	0.005 ppm	2000g	10 µg	Mashuk et al 2011
Food Early 1980s			25 µg	US EPA 1986 and Pennington 1983 cited in Mashuk 2011
Food Early 1995-2003			2.1 µg	US EPA 1994
Food UNEP/GEMS programme in 1980-1988 UK (1 –year old (assuming 7kg b.w)			11.9 µg	Galal-Gorchev 1991
Food 2003-2009 (including water) (assuming 7kg b.w)			7.7-21.7 µg	EFSA 2010
Paint flakes	0.5%	0.001g	5 µg	Mashuk 2011
Paint dust	2.5%	0.001g	25 µg	Mashuk 2011
Toys	Limit (mg/kg): 2 (dry); 0.5 (liquid); 23 (scraped off)		Should be≤ 3.5 µg	Council Directive 2009/48/EC as amended
Soil	500ppm	0.01g	5 µg 0.18 -0.8	Mashuk 2011 EFSA 2010

Total lead intake

Non-dietary lead exposure has been found to be negligible in Europe (EFSA 2010; WHO 2011), but some children in old housing or contaminated areas from past industrial activity are at a higher risk because lead in indoor dust and garden soil can reach up to tens of mg/g under certain circumstances (see Annex I.1). In general, since the banning of lead in petrol, lead in paint and plumbing remaining in old housing has been the major source of exposure in children (WHO 2011). Given that dust and dirt intake by ingestion peak around 2 years of age (Van Wijnen et al 1990), old lead-containing materials are sources of lead exposure in the indoor environment. However, bottle fed infants are unlikely to be exposed to non-dietary sources of lead but more likely to be exposed to lead in drinking water (e.g. Ryu 1983; Lacey et al 1985).

Few studies have examined the relationship between total lead intake and lead exposure. Studies conducted in Scotland showed that for infants the relationship between blood lead levels and lead intake is curvilinear (Sherlock et al 1982; Sherlock and Quinn 1986); these studies were carried out in a specific context of circumstances in Scotland and therefore are described in Part II.

Ryu et al (1983) studied blood lead levels in bottle-fed infants consuming daily formula with lead intake of 61 µg / day from 3.7-6.5 months of age and found blood lead levels above 10 µg/dl by 196 days of age. Another group of infants, exposed to 16 µg Pb/day through their diet did not develop elevated blood lead levels above 10 µg/dl. On this basis, Rye et al. (1983) concluded that a lead intake of 16 µg/day, or else 3-4 µg/kg of body weight/day, was not associated with elevations in blood lead level above 10 µg/dL.

I.3 Mechanisms of lead toxicity

Lead intake and uptake

Lead toxicity is notoriously difficult to diagnose, and creates a wide range of symptoms which are easily overlooked (Kalra et al 2000). The mechanisms of lead toxicity have been studied extensively in relation to nervous system but less well in other systems. This section deals with known mechanisms and effects in order to indicate which functions and parts of the body are targeted by lead once it is taken in and absorbed; thresholds at which the effects can be diagnosed are dealt with in the next section on *Dose-response relationships.*

It widely accepted that any level of lead exposure has the capacity to enter the blood-brain barrier and affect the central nervous system (CNS) of children (Lidsky and Schneider 2003).

Lead interferes with nerve signalling and synapse formation because this process is highly regulated by movements of charged ions, such as calcium, across cell membranes. At picomolar concentrations lead outcompetes calcium from entering cells, halts release of neurotransmitters from the cell, and thus disrupts nerve signalling (Lidsky and Schneider 2003; Needleman 2004). In addition, lead inhibits the absorption of iron and zinc, which are also essential to proper brain and nerve development (Lidsky and Schneider 2003).

Lead effects on the nervous system can be diagnosed by a range of symptoms such as brain damage (encephalopathy); hearing impairment; peripheral neuropathy, e.g. the characteristic "wrist drop" and "foot drop"; and in children as learning disabilities, decreased IQ scores, speech and language disorders, and problems with motor and sensory skills (ATSDR 2017a). However, many affected children and adults may remain asymptomatic or misdiagnosed for a long time.

Lead also has the ability to interact with proteins including those with sulfhydryl, amine, phosphate, and carboxyl groups (ATSDR 2017a). Lead's high affinity for sulfhydryl groups makes it particularly toxic to multiple enzyme systems including haeme (blood protein) biosynthesis, as lead inhibits three important enzymes participating in the process, i.e. delta aminolevulinic acid dehydratase, delta aminolevulinic acid synthase, and ferrochelatase (Piomelli 2002). Lead has a particular affinity with foetal haemoglobin, which is the main oxygen transport protein in the human foetus during the second and third trimesters of gestation and persists in the infant until 6-months of age (Ong and Lee 1980).

The effects of lead on blood synthesis can be practically diagnosed by symptoms such as anaemia, iron deficiency, high levels of delta aminolevulinic acid dehydratase in urine and increased protoporphyrine level in blood or urine (Mashuk 2011 and literature cited there in).

Lead exposure also affects blood pressure and hypertension, which appear to be related to lead effects on vascular reactivity, oxidative stress responses, and the renin–angiotensin– aldosterone system (RAAS) (Mashuk 2011 and literature cited there in). Hypertension is a risk factor for ischaemic heart disease and strokes, and preterm death.

Lead also inhibits the proximal tubular lining cells and causes renal insuf□ciency (Papanikolaou et al 2005). Reversible abnormalities caused by lead exposure include aminoaciduria, glycosuria, and phosphaturia with hypophosphatemia, and increased sodium and decreased uric acid excretion (. Chronic lead kidney disease (nephropathy) includes progressive interstitial □brosis, a reduction in the glomerular □ltration rate, and abnormally high quantities of urea and kreatinin; these effects are irreversible. Acute nephropathy is most frequently reported in children while chronic nephropathy is mainly reported in adult (ATSDR 2017a).

Reproductive and developmental toxicity of lead is related to effects on the foetus and on DNA, which may lead to genotoxicity and carcinogenicity. Several studies have shown that transplacental lead exposure entails both exogenous and endogenous lead exposure for the foetus; endogenous exposure arising from bone calcium mobilization during pregnancy, depending on maternal diet especially in the final trimester when foetal skeletal mineral demands for calcium are maximal (Gulson et al 2004). It has been shown that foetus vulnerability can occur even when mother's exposure had ceased many years before pregnancy (Bellinger 2005). Lead causes disruption in cell migration during critical times of the brain and other parts of the nervous system development (Jomes 2009). As for genotoxicity, a number of recent studies have indicated that lead is genotoxic, although the exact mechanism has not been identified because lead-induced changes in DNA strands are difficult to link to cancer and teratogenesis (Mashuk et al 2011).

Lead has also been found to interfere with the cell-mediated immune system in complex ways that make for adverse responses identified at increasingly lower exposures (Mashuk 2011). Effects are diagnosed as disruption of the the regular function of the immune system rather than as histochemical findings. Mashuk (2011) has also reviewed the effects of lead on the hormonal system: the general consensus is that blood lead may not be a suitable biomarker because results from a variety of studies were inconsistent. Finally, effects of lead on liver have been infrequently studied to provide sufficient conclusions on lead toxicity on liver function.

I.4 Lead: health effects and social outcomes

Dose-response relationships: acute/high/elevated exposures

The most common symptoms of acute exposure (blood lead concentrations > 80 µg/dl) are dullness, restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal cramps, kidney damage, loss of memory, and brain damage (encephalopathy). Chronic exposure (blood lead concentrations > 80 µg/dl results in a range of symptoms from muscle weakness, gastrointestinal cramps, intelligence disability, disturbances in mood, and peripheral neuropathy to tiredness, sleeplessness, irritability, headaches, and joint pain and encephalopathy.

At blood lead levels below 25 µg/dl, lead has also been historically associated with instantaneous abortion, premature delivery, stillbirth, infant mortality, low birth weight, and compromised mental and physical development of infants (Troesken 2006; Mahaffey 1985).

Dose-response relationships at low lead exposure: cross-sectional studies

The most important clinical symptoms of low (below 10 µg/dl) lead exposure include decreased intelligence quotient-IQ scores, and impairment in hearing, growth and blood function (Bellinger and Needleman 2003; Troesken 2006; Needleman and Gee 2013; Brown and Margollis 2012).

Impaired kidney function has been observed in adolescents at blood lead levels as low as 1.5 µg/dL (Fadrowski et al 2010). In addition, decreased height and delayed breast development in pubescent females has been associated with blood lead concentrations of 3 µg/dl (Selevan et al 2003). Also, declines in cognitive function have been linked to blood lead concentrations as low as 3.0 µg/dL (e.g. Schwartz 1994; Bellinger and Needleman 2003), although these studies have not accounted for parental IQ or income, family medical history and preterm birth. Canfield et al (2003) found significant non-linear relationship between blood lead levels below 10 µg/dl in 24 to 30 month old children and cognitive scores at the age of five after adjusting for child's sex, birth weight, and iron status and the mother's IQ, years of education, ethnicity (white or non-white), tobacco use during pregnancy, yearly household income and environmental circumstances. Jusko et al (2008) demonstrated the significance of associations between blood levels below 5 µg/dl in toddlers and cognitive declines in school age after adjusting for preterm weight.

An international pooled analysis (International Pooled Lead Study) of data from seven cohort studies including data from countries such as USA, Australia, Yugoslavia and Mexico) reported an inverse and supra-linear relationship between blood lead concentrations and IQ scores in children, i.e. every increase in up to 1 µg/dl is associated with loss of 1-IQ point (1%) at the population levels (Budtz-Jørgensen et al 2012). The calculations showed only a limited variation between studies in the steepness of the dose (blood lead)-response (IQ) functions. The authors showed that their results were quite robust to modelling assumptions with the best □tting models yielding lower con□dence limits of about 0.1–1.0 µg/dL for the blood lead change leading to a loss of one IQ point at blood lead ranges below 10 µg.dl (Budtz-Jørgensen et al 2012).

Dose-response relationships at low lead exposure: prospective/longitudinal studies

The alterations induced by lead exposure in infancy and early childhood have been associated with effects in later childhood such as lower intelligence quotient scores (IQ), learning disabilities, hyperactivity, attention deficit disorders, hearing/speech impediments, seizures, behavioural impairments/aggression; and even crime or a lower income in adolescence or early adulthood (Needleman et al 1990; Lanphear et al 2005; Lidsky and Schneider 2003; Faust and Brown, 1987; Dietrich et al. 1990; Bellinger et al. 1992; Needleman 2004; Reyess 2015; Bellinger 2017; Reuben 2017).

One of the earliest studies on this kind of associations was conducted by Needleman et al (1979; 1990). They first examined the relationship between low lead exposure and cognitive function. This study demonstrated deficits in intelligence scores, speech, language processing, attention and school performance in second grade children, who were asymptomatic and had blood lead levels below 7 µg/dl but had dentine lead levels above 10 ppm (Needleman et al 1979). Follow up studies showed that those symptoms persisted into early adulthood (Needleman et al 1990) showing for the first time that lead exposure, even in children who remain asymptomatic, may have an important and enduring effect on the quality of life of these children and that lead burden and behavioural deficit are strong predictors of poor school outcome.

These findings have been since supported by a range of long-term (prospective/longitudinal) studies accounting for individuals born in the 1970s and followed up to later childhood and to adulthood to characterize the changes in health, cognitive function and behaviour that are associated with early lead exposure[2]. For example, Lanphear et al (2000) found that an increase in blood lead levels by 1 µg/dl can cause a 0.7 and 1.0 point decline in mean arithmetic and reading scores, respectively. Canfield et al (2003) and later Lanphear et al (2005) showed that an increase in blood

[2] the studies considered in this report also accounted for social and other factors influencing health

lead levels from 1 to 10 µg/dl during lifetime can reduce an individual's IQ by 3.8 to 8 points (3.8 -8%). Interestingly, Lanphear et al (2005) found that the sharpest IQ declines occur when blood lead levels were less than 10 µg/l. Also, children with blood lead concentrations greater than 2 µg/dl in early childhood were at a 4.1-fold increased risk of Attention Deficit Disorder-ADHD in later chidlhood (Braun 2006). Wright et al (2008) found that adjusted rates of total arrest and/or arrests for offenses involving violence were greater for each 5 µg/dl increase in prenatal and postnatal blood lead levels.

Further evidence that the 10 µg/l should not be viewed as a threshold for lead was given on the basis of the Avon Longitudinal Study of Parents and Children (ALSPAC) by Charndramouli et al (2009) in the UK. They studied blood lead levels at 30 months of age and then at of 7-8 years of age. They found that blood lead levels of 5–10 µg/dl were associated with a reduction in scores for reading and writing and blood lead levels above 10 µg/dl were also associated with increased scores for antisocial behaviour and hyperactivity. Mazumdar et al (2011) also found that cognitive function was lower in 10-year old children known to have been exposed to blood lead levels exclusively below 10 µg/dL when at the age of 2 years.

Several researchers also related blood lead levels with the need for special education. Lyngbye et al (1990) showed that, even at low levels of lead exposure, the need for special education increases with the exposure level. Schwartz (1994) reported that 20% of children with blood lead levels above 25 µg/dl may need special education. Nevin et al (2006) suggested that the need for such service could start below this concentration when blood lead levels exceed 10 µg/dl.

Several studies have also shown evidence of a link between low lead exposure in childhood and criminal or delinquent behaviour. Nevin et al (2006) demonstated a relationship between prenatal / preschool lead exposure in France and delinquent behaviour later in life. Similar findings, after adjusting for confounding factors, have been supported by the longitudinal Cincinnati Lead Study of a cohort of 195 urban, inner-city adolescents recruited between 1979 and 1985, of whom 92% were African-American and 53% male (Dietrich et al 2001)[3]. After adjustment for covariates and interactions and removal of non-influential covariates, Needleman et al (2003) found that adjudicated delinquents were four times more likely to have bone lead concentrations above 2.5µg/dl than controls (OR=4.0, 95% CL: 1.4-11.1).

More recently, Reuben et al (2017) presented the results of the Dunedin Multidisciplinary Health and Development Study, which observed participants in a population-representative of the 1972-1973 birth cohort from New Zealand from age 11 years through the age 38 years. Mean blood lead levels at 11 years of age was 10.99±4.63. After adjusting for maternal IQ, childhood IQ, and childhood socioeconomic status, each rise of blood lead by 5 µg/dL in childhood was associated with decreases in adulthood in the following: IQ scores, perceptual reasoning, working memory and socioeconomic status. Reuben et al (2017) emphasised that socioeconomic status in adulthood has been partially affected by lead exposure in childhood; they concluded that other factors, not accounted, had a more important bearing on income.

I.5. Regulations to control lead

I.5.1 Regulation of lead in drinking water

I.5.1.1 The standard for lead in drinking water

Regulation of lead in drinking water has focused on promulgation of a standard maximum tolerable value for lead in drinking water measured at the tap or a minimum concentration that is practically achievable (Lambrinidou et al 2010; WHO 2011). A health-based standard for lead in drinking water was first addressed in the WHO Guidelines for drinking water in 1984 and set to be at 50 µg/l on the basis of toxicity studies and previous recommendations by the 1972 Joint FAO/WHO Expert Committee on Food Additives on a total lead intake at 3 mg/day (FAO/WHO 1972). This lead standard was revisited by WHO in early 1993 to reflect the findings of adverse health effects in infants and reductions in total lead exposure due to phasing out sources such as lead in gasoline and paint. Since then the guideline for a lead standard in drinking water is at 10 µg/l.

Health-based thresholds for lead in water are equal or near to zero. For example, the US EPA maximum goal is equal to zero but the state of California has developed its own Public Health Goal (PHG) for lead in water at 2 µg/L. Health Canada (1992 cited in Dore 2015) has developed a health-based guideline of 10 µg/L for lead for drinking water. The CDC (2010 cite din Dore 2015) advises children and pregnant women to not consume water that contains more than 15 µg/L lead.

Lead limits are usually derived from an estimation of lead concentrations considered at the time economically and technologically feasible to achieve, and as such, are not entirely health-based (Lambrinidou et al 2010; WHO 2011). For example, the previous limits of lead in drinking water in Scotland and the EU were based on such practical implications.

[3]Delinquency was accounted for on the basis of parents reports

I.5.1.2 Lead in drinking water in the European Union

Presently, the Drinking water Directive-DWD (98/83/EU) is the main regulatory tool for controlling lead concentrations at the tap water. In general, DWD applies specific technical and monitoring requirements for water supplies to ensure that the water is "wholesome and clean" (Art. 4). DWD has set standard limit values (referred to as "parametric values") for more than 40 parameters including microbiological and chemical drinking water contaminants; physiochemical parameters such as pH; organoleptic parameters such as taste; and radiation parameters. For all these parameters including lead, DWD requires that:

- The measures taken by EU-MS to implement DWD in no circumstances have the effect of allowing, directly or indirectly, either any deterioration or any increase in the pollution of waters used for the production of drinking water (Art 4(2));
- If, despite the measures taken to meet the obligations to ensure "wholesome and safe water", drinking water does not meet the parametric values laid down in DWD, EU-MS shall ensure that remedial action is taken as soon as possible water quality and shall give priority to enforcement action (Art. 8(2)).
- The parametric value shall be complied with at the point of use, i.e. at the tap water, when water is supplied from a distribution network for human consumption or for food-consumption (Art.6(1)).
- EU-MS shall be deemed to have fulfilled their obligations under Article 4(2) and 6(1) and 8(2) where it can be established that non-compliance with the parametric values is due to the domestic distribution system or the maintenance thereof except in premise and establishments where water is supplied to the public, such as schools, hospitals and restaurants (Art. 6(2)). EU-MS shall nevertheless ensure that appropriate measures are taken to reduce or eliminate the risk of non-compliance with parametric values such as advising property owners of any possible remedial action they could take and/or any other measures, such as the appropriate treatment techniques to change the nature or properties of the water before it is supplied. (Art.6(3)).
- Samples should be taken so that they are representative of the quality of the consumed throughout the year (Art. 7 (1)[4]. EU guidelines for harmonised monitoring may be drawn up in accordance with Council Decision 1999/468/EC.

For lead, DWD requires that:

- The limit value applies to a sample of water intended for human consumption obtained by an adequate sampling method[5] at the tap and taken so as to be representative of a weekly average value ingested by consumers. (…) EU-MS must take account of the occurrence of peak levels that may cause adverse effects on human health (Annex I;Part B; Note 3).
- When implementing the measures to achieve compliance with the limit value EU-MS must progressively give priority where lead concentrations in water intended for human consumption are highest (Annex I;Part B; Note 4).

I.5.1.3 Sampling protocols in Canada

In Canada, the Federal government has set a guideline value for lead in drinking water at 10 µg/l since 1992 (Health Canada 1992 cited in Dore), in accordance with the WHO health-based guideline. Since 2009, however, Health Canada (2009 cited in Dore) has set two options for monitoring lead to control corrosion and assess compliance with corrosion control measures and exposure to lead in drinking water. Option 1 is a two-tier approach for assessing corrosion control on a distribution system-wide scale. The first tier involves sampling after a 6-hour stagnation period. If up to 10% of the sampled sites have lead concentrations above 15 µg/l then a range of remedial actions are recommended (Annex I.6-Box 1). The second tier is taken when more than 10% of the sampled sites have lead concentrations above 15 µg/l; it intends to establish a stagnation profile by sampling four consecutive IL samples after a 6-hour stagnation period.

Annex I.6-Box 1. Remedial actions recommended for Option 1:Tier 1 and Option 2 of the Canadian Federal guidelines for lead in drinking water.

1. Initiation of a public education programme for encouraging consumers to flush water after prolonged stagnation, replace lead service pipes, fittings and fixtures, or treat water at point-of-use to remove lead.
2. Additional sampling within the sites with lead levels above 15 µg/l.
3. Implementation by provinces and municipalities that own water supplies of corrosion control measures, such as pH and alkalinity correction, addition of corrosion inhibitors and communication pipe replacement.
4. Encouraging homeowners to clean debris from aerators and screens , which may retain particulate lead

Source: Health Canada 2009 cited in Dore 2015

[4] Art. 7(2) refers to

[5] To be added following the outcome of the study currently being carried out.

Option 2 is applied when a 6-hour stagnation period is impractical and is intended to evaluate lead corrosion at properties connected to the mains with lead communication or service pipes (Health Canada 2009). Option 2 involves sampling after 30 minutes of stagnation (30MS) of four consecutive IL samples. If the average of four samples exceeds the standard of 10 µg/l in more than 10% of the sites monitored, the same corrective action as in Option 1, tier 1 is applied (Annex I.6-Box 1).

I.5.1.4 Sampling protocols in the USA

In the USA, lead in drinking water is regulated under the Safe Drinking Water Act (SDWA 1986). In 1991, the US Environment Protection Agency (US EPA) promulgated the Lead Copper Rule (LCR), which set an action level of 15 µg/l in 1 L of first draw water sample taken after a 6-hour stagnation period. LCR also established a non-enforceable maximum contaminant level goal of zero lead in drinking water, as the level at which no adverse health effects are likely to occur according to research on blood lead levels carried out and reviewed by the Centres of Disease and Prevention (CDC). However, it has been recognised that the goal of lead-free drinking water in the USA is infeasible because domestic supplies (e.g. private wells) are outwith the control of public drinking water supplies (Brown and Margolis 2012). LCR, in line with DWD in EU and the Canadian guidelines, requires sampling at tap water at similar frequencies compared with those set in Canada (Part III. Section 9). If more than 10% of the samples collected from a water utility serving <50,000 people exceeds the lead action level, the utility must identify and install optimal corrosion control treatment. Utilities serving more than 50,000 people are required to have optimal lead corrosion control (Part III-Section 6.1) regardless of lead levels.

LCR also requires that all utilities exceeding the LCR action level educate the public on suitable remedial action. When exceedance of the action level occur in supply zones with lead pipes and optimised corrosion control treatment, replacement of the lead pipes owned by the utility is mandatory as in Scotland. When a homeowner does not agree to replacement of privately owned lead pipes, then the utility must notify the residents at least 45 days in advance that they might experience a temporary increase in lead levels and must collect a sample within 72-hours after completion of the partial replacement for lead analysis and notify the home owners of the results. Water from systems that serve <25 individuals and private drinking water wells is not regulated under the SDWA; as of 2012, this corresponds to approximately 40-45 million people in the USA (US DHHS 2012).

I.5.2 Regulations to control environmental lead emissions and use of lead-containing materials

The main legislations currently controlling lead in the environment, products in the UK and Scotland include:

- Legislations referring to emissions or concentration of lead in water, soil, air
  - Council Directive 76/464/EEC on pollution caused by certain dangerous substances discharged into the aquatic environment of the Community
  - The Water Framework Directive (2000/60/EC).
  - The EU Directive 2010/75/EU on industrial emissions (integrated pollution prevention and control) which aims to prevent or minimise pollution of water, air and soil. The directive targets certain industrial, agricultural, and waste treatment installations.
  - The European Pollutant Release and Transfer Register Regulation (E-PRTR) (166/2006/EC), which requires the estimation and reporting of emissions of a number of heavy metals released from certain industrial facilities
  - The OSPAR convention which protects the marine environment of the north-east Atlantic

- Legislations referring exclusively to air emissions:
  - The EU Directive 98/70/EC, which banned lead form petrol through a combination of fiscal and regulatory measures.
  - The 1979 UNECE Convention on Long-Range Transboundary Air Pollution (LRTAP).
  - The 1989 Basel Convention on the Control of Transboundary Movements of Hazardous Wastes and Their Disposal, which inter alia intends to minimize the amount and toxicity of wastes generated.
  - The 1998 Aarhus Protocol on Heavy Metals (HMs), which aims to control emissions of HMs caused by anthropogenic activities subject to long-range transboundary atmospheric transport over the base year 1990.
  - The EU Directive 2001/80/EC on the limitation of emissions of certain pollutants into the air from large combustion plants (LCP Directive), which aims to limit heavy metal emissions via dust control and absorption of heavy metals.
  - The EU Directive 2004/107/EC relating to heavy metals and polycyclic aromatic hydrocarbons in ambient air, which sets limit values for the further control of air pollutants in ambient air.
  - The EU Directive on Ambient Air Quality and Cleaner

Air for Europe (2008/50/EC)

- The Air Quality Strategy for England, Scotland, Wales and Northern Ireland (n.d.), in which lead is one of the eight key air pollutants targeted for action.

- Legislations referring to the use and trade of leaded consumer products
  - Legislations referring to lead in products such as toys (the Toy Safety Directive 2009/48/EC); paint (Directive 89/677/EEC); animal food and feed (Council Directive 96/23/EC); and foodstuff in general (e.g. Commission Regulation (EC) No 1881/2006)
  - COUNCIL DIRECTIVE 76/769/EEC) on the approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations
  - The REACH Regulation (Registration, Evaluation, Authorization, and Restriction of Chemicals) is a regulation of the European Union as of December 2006, which restricts the use of substances including lead where risks to human health and the environment cannot be managed and encourages the substitution of the most hazardous substances with less dangerous alternatives. It includes restrictions on lead used in paint, jewellery and other consumer products such as toys. REACH is among the strictest laws to date and affects industries worldwide.
  - The EU Restriction of Hazardous Substances Directive 2002/95/EC (RoHS), often referred to as the "lead free directive", which restricts the use of six hazardous substances, including lead, found in electrical and electronic products including household appliances and IT and consumer equipment. In 2011 it was expanded to include a much wider range of products and covers all electronic equipment, cables and spare parts. The Directive requires a maximum lead concentration by weight in solder to be restricted to 0.1%, encouraging the use of alternatives like bismuth.
  - The Waste Electrical and Electronic Equipment Directive 2012/19/EU (WEEE) that is focused on promoting the collection and recycling of electrical goods to solve the toxic e-waste problem.

I.5.3 Regulation of occupational exposure to lead

In the UK occupational exposure to lead is regulated under the Council Directive 98/24/EC. Health Protection England designated 25 µg/dl as the action level for blood lead levels in women of child-bearing age and 50 µg/dl for general lead employees (Health and Safety Executive 2017). It also required workers to be removed from lead exposure when blood lead levels are greater than or equal to 30 µg/dL for women and 60 µg/dl for general employees. Likewise, the U.S. Occupational Safety and Health Administration (OSHA) Lead Standards require workers to be removed from lead exposure when blood lead levels are greater than or equal to 50 µg/dL, and allow workers to return to work when their blood lead level is less than 40 µg/dL (OSHA n.d.).

I.5.4 Regulation of residential exposure

In 1977 the Member States of the European Economic Community (EEC) agreed to carry out a screening programme for blood lead levels to determine levels of lead exposure and assess the need for further lead mitigation measures (EEC 77/31). Lack of consensus among Member States on a specific health-based blood lead standard value of concern or for action precluded the formulation of legislation on mandatory and regular blood lead screening in the EU (Farmer 2012). However, certain reference levels were agreed: if these were exceeded it would be for the Member States themselves to decide on what measures to take and it would not be for the European Commission to express an opinion (Farmer 2012). The 'reference levels' in the Directive EEC 77/31 were set on the basis of biochemical evidence on the effects of lead at blood lead concentrations above 20 µg/dl (e.g. Zielhuis 1974). The results were assessed on the basis of the following frequency distribution of blood lead concentrations in the population (conversion: SI to traditional units: 1 µmol/l = 20.7 µg/dl):

- No more than 50% of the population should be above a blood lead level of 20 µg/dl
- No more than 10% of the population should be above a blood lead level of 30 µg/dl
- No more than 2% of the population should be above a blood lead level of 35 µg/dl

In addition, follow-up investigations were to take place if any individual was over 35 µg/dl. For both adults and children, this limit was subsequently lowered to 25 µg/dl in the UK jurisdictions (Quinn 1985).

Blood lead screening in the general population is not mandatory in the UK. Blood lead screening is mandatory in several States in the USA.

For comparison, and on the basis of up-to-date evidence, the current action level for blood lead concentration in children set by the CDC (2012) in the USA is at 5 µg/dl. This level of lead exposure is approximately 1250 times greater than the blood lead levels in pre-industrial, native Americans estimated from human remains (Flegal and Smith 1992). The level for intervention established by the CDC accounts for evidence referring to adverse health effects in children

aged 1-5 years old who are in the top 2.5% in terms of blood lead levels when compared to children exposed to more lead than most children (CDC 2012).

Prior to the level of intervention for lead exposure the CDC had established a blood lead level of concern, which was used by paediatricians and other health professionals in the USA and by WHO (2011) to establish priorities and help diagnose a child with lead exposure and investigate sources of exposures. The lead exposure (blood lead) level of concern has been revised downwards over time in view of new evidence for adverse health effects at ever lower levels of lead exposure (Figure 2).

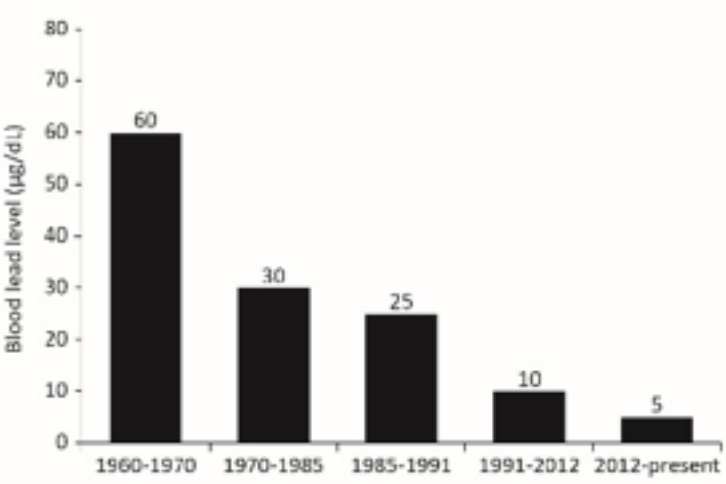


Figure 2. Blood lead level of concern by CDC over time. Source: CDC 2012 cited in ATSDR 2017b

The WHO has recommended the level of 10 µg/dl as a public health goal, however, emphasising that lead exposure, especially in children, must be further reduced or eliminated (WHO 2011). Since 2006 there has been an increase in evidence-based calls to reduce the blood lead level of concern below 5 µg/dl (see section Annex I.5 for a review of this evidence). For example:

- In the USA, Gilbert and Weiss (2006) argued that the level of concern and subsequent action should be revised down to 2 µg/dl as the most effective and practicable way to (i) eliminate the neuro-behavioural consequences of lead exposure in current children and future adults; and (ii) encourage simple procedures at home to lower sources of exposure to lead.
- In the UK, Chandramouli et al (2009) on the basis of a study of 592 children at 30 months of age and at 7-8 years old at Avon, England recommended that the threshold for clinical concern should be reduced from 10 to 5 µg/dl.
- In Australia, Taylor et al (2014) recommended that the Australian authorities adopt a blood lead intervention level of no more than 5 µg/dl, with a national goal for all children under 5 years of age to have a blood lead level of below 1 µg/dl by 2020.
- In Germany, the reference value for children was lowered to 3.5 µg/dl aged 3-14 years (Schultz et al 2011), i.e. it is lower than that recommended by WHO (2011) and that set by the CDC in the USA.
- In France, Etchevers et al (2015) recommended that the reference value for elevated blood lead levels in children should be reduced from 10 to 4.4 µg/dl.
- In 2015, the National Institute of Occupational Safety and Health (NIOSH) in the USA designated 5 µg/dL as an elevated blood lead level in adults (ATSDR 2017b).
- The U.S. Department of Health and Human Services recommends that blood lead levels among all adults be reduced to less than 10 µg/dL (ATSDR 2017b).

In the UK, Health Protection Agency (n.d.) has published a Lead Action Card, which outlines the key actions in response to elevated blood lead levels, i.e. above 10 µg/dl. These actions can be triggered once a notification of potentially elevated lead exposure comes from one of the following organisations: a water company; the Drinking Water Inspectorate (DWI); the Environmental Health Department; a GP or paediatrician; or other relevant organisations.

Also in the UK, the National Screening Committee, which advises Ministers and the NHS in all four UK countries about all aspects of screening policy, has recommended against blood lead screening in asymptomatic children aged 1 to 5 years on the basis of the following reasons(Spiby 2013; UK National Screening Committee 2014).:

(i) A decline in the number of people affected by lead poisoning;

(ii) Very few children being affected by elevated (>10 µg/dl) in the UK;

(iii) The current test being not reliable enough

(iv) A lack of proven treatments for lead poisoning, especially for children only slightly affected, available treatments possibly being harmful in these children.

The benefits of regulations mandating blood lead screening have included:

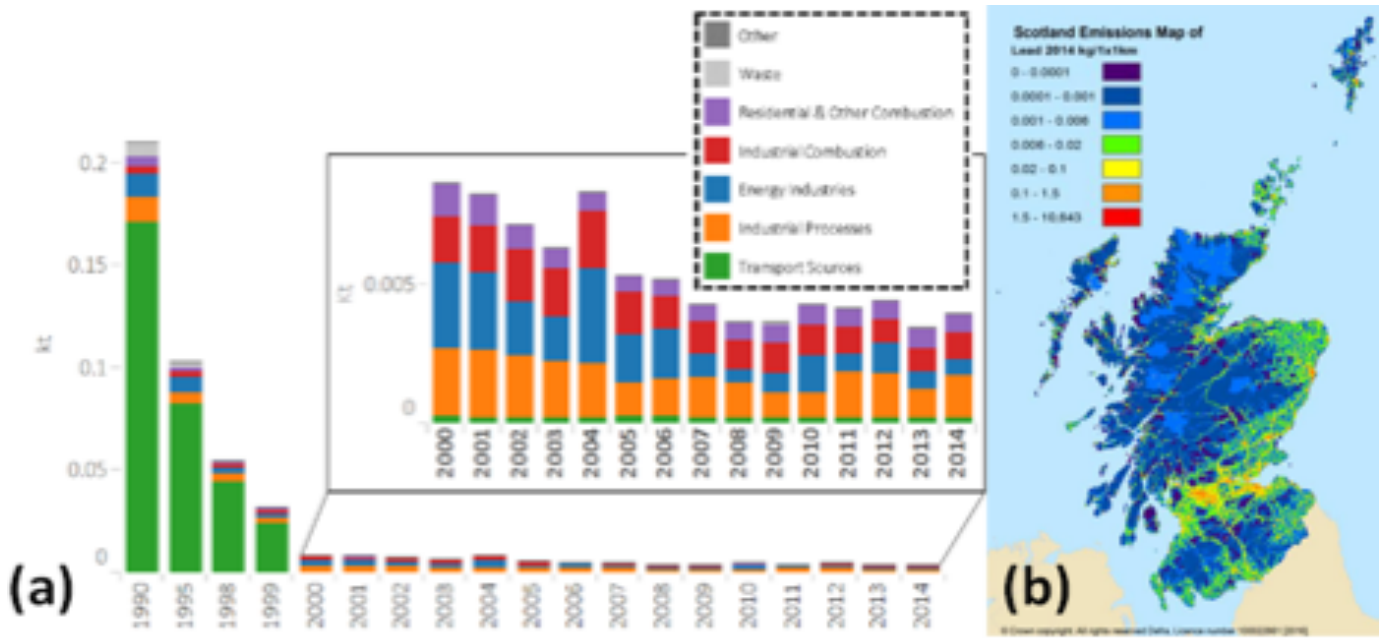
- Effective detection of children exposed to low levels of lead in blood (i.e. between 5 and 10 µg/dl) to enable, in a timely fashion, investigation and removal of invisible sources of lead in a child's environment (e.g. Schultz et al 2011; CDC 2012; Taylor et al 2014 ATSDR 2017b).
- Implementation of educational and nutritional interventions to reduce the risk and the effects of low lead exposure on cognitive development and the absorption of nutrients (e.g. US DHHS 2015).

ANNEX II Lead in Scotland

II.1 Non-waterborne sources of lead exposure in Scotland

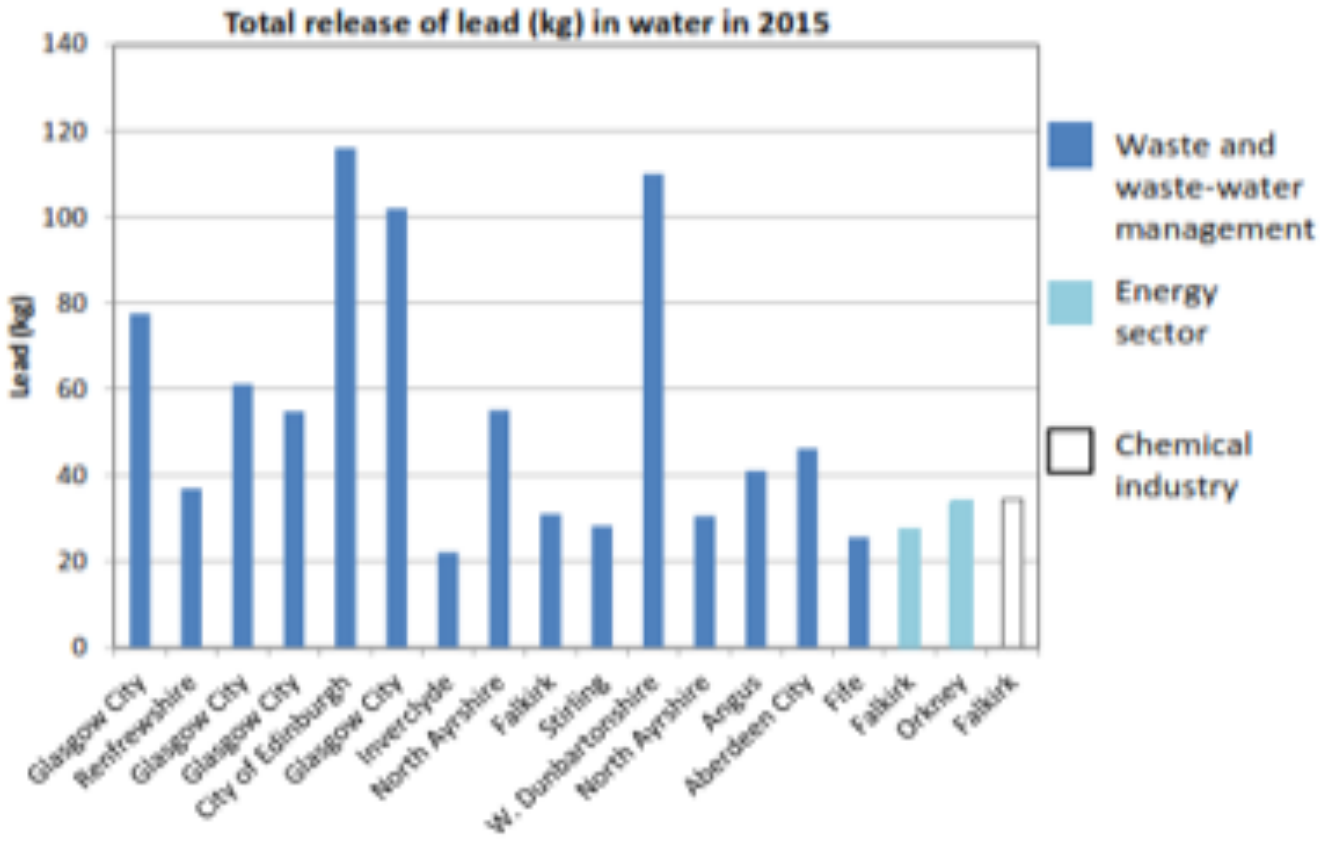
Industrial lead

According to the report on Air Quality Pollutants Inventories for England, Scotland Wales and Northern Ireland: 1990-2010 (MacCarthy et al 2012), emissions of lead were estimated at 3.9 t in 2014 in Scotland and accounted for 6% of the UK total. Emissions have declined by 98% since 1990. sources due to the phase-out of leaded petrol in 1999 (see Annex I.1). The most significant sources of emissions in 2014 were combustion of coal in power generation and the industrial sector; residential combustion (e.g. biomass); and the use of lubricants in transport (Annex II-Figure 1a and b); as a result of this, lead emissions are greater in more densely populated areas and at heavy industry sites (Annex II-Figure 1b). The peak of lead emissions in 2004 was due to an increase in reported emissions from coal combustion in power generation (Annex II-Figure 1a).



Annex II-Figure 1. Lead emissions to air in Scotland (a) Trend and sources of lead emissions to air from 1990 to 2014 (b) Spatial distribution of lead emissions to air in 2014. Source: Bailey et al 2016.

The Scottich Pollution Release Inventory (SPRI) collects data on pollutants released to air, land and water as well as waste transfer in Scotland since approximately 2003. The data for lead release to water in 2015 from different sectors such as the waste and waste-water management, the energy sector and the chemical industry, showed that the threshold for reporting lead emissions to water set by SEPA (i.e. 20 kg/ year) has been exceeded in many areas throughout Scotland (SPRI-SEPA 2015) (Annex II-Figure 2)



Annex II-Figure 2. Lead release to water in 2015 from different sectors throughout Scotland (SPRI-SEPA 2015).

Catchment lead

In Scotland, there has been widespread use of lead since the beginning of the industrial era. Farmer et al (1999) studied lead in lake sediments to understand the effect of lead emissions to the atmosphere on catchment soils and depositional environments and found that lake lead mainly comes from smelting and coal combustion in the 19th century and the electricity generation industry, which peaked after World War II. Lake depositional studies by Cloy et al (2005) showed that lead from vehicular sources made a considerable contribution during the second half of the 20th century through to its ban from gasoline in 2000. Farmer et al (2005) concluded that lead deposition quadrupled from late 19th century till 1960 compared with pre-industrial levels due to smelting and coal combustion, before declining steadily to reach one fifth of the deposition rates in the 19th century.

In Glasgow, isotopic composition studies showed that soils are influenced by a variety of sources, reflecting values for source end-member extremes of imported Australian lead ore, which was used in the manufacture of alkyl lead compounds formerly added to petrol, and indigenous lead ores, used in coal combustion(Farmer et al 2011).

Rothwell et al (2007) showed that erosion during storm events was the key mechanism for lead input from blanket peat soils, where lead from atmospheric emissions had accumulated in the past, to stream sediments. DOC loss from catchment soils in stormflow may play a major role in the release of dissolved lead from peatland (Graham et al 2006). Rose et al (2012) showed that, although lead deposition declined due to the phase out of lead from gasoline, trends of lead fluxes from catchments to lakes continue to increase to the most recent decades due to

increased soil erosion and lead leaching from DOC, which is associated with prolonged summer drought followed by high intensity rain events. They warned that this lead flux has the potential to counteract the benefits of reductions in deposition resulting from policy implementation over recent decades and may elevate exposure of aquatic biota to lead (Rose et al 2012).

More recently, there were incidents of lead poisoning in livestock. Since 2015, approximately 460 animals in Scotland have been affected by lead poisoning, with 38 dying as a result (FSS 2017). This was caused by the presence, adjacent or within pasture land, of lead deposits from sources including old machinery, rubbish, vehicle batteries, bonfire ash, electric fencing or lead-based paint. Products from the affected livestock can be above the safe limits laid down in food law, making them illegal for use in the food chain.

Lead in the urban environment

Thornton et al (1990) found elevated lead concentrations in household dust and garden soils, including playground soils, in Edinburgh and Glasgow, suggesting that in urban areas young children may be exposed during playtime to multiple sources of exposure to lead in addition to water lead; see Annex I for illustrative data and comparative tables.

Sugden et al (1993) and Farmer et al (1994) observed that overlaps in isotopic lead composition of a range of environmental media may hinder the quantitative apportionment of source and route in general population surveys of human exposure to lead. In general, atmospheric particulates and street dust had an isotopic composition more similar to leaded petrol (Australian lead ore) than to British lead ore deposits and coal, whereas house dust, paint and tap water from lead pipes had a composition more similar to British lead ore deposits and coal (Sugden et al 1993).

More recently, Dean et al (2017) found that bioaccessibility of lead (i.e. the potential of lead to interact with or be retained in the body) in airborne suspended particles smaller than 10 µm is minimal in Edinburgh.

Exposure to lead in food

Two types of studies have provided information for dietary lead exposure in Scotland: the duplicate diet studies and the surveys by the Food Standards Agency (FSA 2009 cited in EFSA 2010), which have informed the European study on Lead in Food (EFSA 2010). Additional sources of information refer to: increased health risks from regular consumption of lead-shot wild-game, especially bird, meat in Scotland (FSS 2012); and low lead levels (below the limit for lead for this type of foodstuff) in shellfish in studies carried out in 2004 and 2005 (McIntosh 2005). The Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) advised that efforts should continue to reduce exposure to lead from all sources since it is not possible to identify a threshold for the toxic effects of lead.

II.2 Blood Lead Surveys

EEC Blood Lead Surveys

The EEC Blood Lead Surveys carried out in 1979-1981 (EEC 77/31) examined how consumption of plumbosolvent water translated into lead exposure. The Blood Lead Surveys in the UK were organised by the Department of the Environment (DOE 1982 cited in Quinn 1985)[6]. Random surveys were carried in populations influenced by elevated air lead emissions, leadworks and other determining factors for lead exposure (i.e. smoking, alcohol drinking, residence, age and gender) in England; in Scotland surveys targeted two areas of Scotland (i.e. Glasgow and Ayr) influenced by plumbosolvent water. In 1979 there were 39 surveys in which nearly 5000 blood samples were collected from: 2000 adults in major cities in England; 2000 children exposed to leadworks in England; 300 adults and 500 children living near major roads in England; and 150 mothers and their infants exposed to plumbosolvent water in Glasgow (Quinn 1985). In 1981, there were 35 surveys in which about 3500 (different) people took part in England. In Scotland, there were additional surveys in Glasgow, and in Ayr, because of the high water lead concentrations in the area[7] (Quinn 1985).

A major weakness of the EEC Blood Lead Surveys was the sampling design, i.e. stratified sampling without control population. Sampling targeted specific population groups, thus making it difficult to draw conclusions on the average population blood lead levels in Scotland; understand what percentage of the population had been exposed to high levels of lead in the 1970s and before in their childhood; or disentangle the effects of lead in drinking water from lead in other sources of exposure (see also Pfadenhauer et al 2016). More importantly, the multitude of sources of lead exposure for each group remained unexplored, thereby precluding an apportionment of the contribution of different sources and factors on total blood lead levels. Nevertheless, the Blood Lead Surveys provided a very large amount of benchmark data on blood lead distributions in the groups of people studied for Scotland and the UK in general.

Overall, the EEC Blood Lead Surveys showed that blood lead levels were higher in individuals exposed to plumbosolvent water in Scotland (i.e. Glasgow and Air) than in individuals

[6] It was not possible to find hard or digital copies of these publications.

[7] The exact number of individuals is not reported by Quinn 1985.

living in large urban centres in England (e.g. London and Manchester) or having habits such as smoking and heavy alcohol drinking[8] and regardless of social class[9], age of dwelling[10], or ethnicity (Annex II-Table 1). Blood lead levels in individuals exposed to lead in drinking water were comparable with blood lead levels in leadworkers (Table 1). Quinn (1985) concluded the review of the factors influencing blood lead levels in the UK as follows: "The EEC surveys and many other studies have clearly shown that lead in drinking water, where it occurs, is the most serious environmental factor associated with elevated blood lead concentrations."

Annex II-Table 1. Blood lead levels in relation to a range of exposure factors and sources (Quinn 1985). Explanation in text.

Factor/Source			Blood lead concentrations (Geometric mean) µg/dl	
			1979	1981
England	Smoking	Non-smokers	Women: 10.8 Men: 14.2	Women: 9.4 Men: 12.9
		Heavy smokers	Women: 12.7 Men: 15.9	Women: 11.0 Men: 14
	Age of dwelling	Built pre-1945	Children: 15.9 Women: 11.8 Men: 14.9	Children: 12.6 Women: 10.2 Men: 13.6
		Built post-1945	Children: 14.1 Women: 10.4 Men: 13.9	Children: 11.1 Women: 8.7 Men: 12.2
	Urban	Inner	Adults: $12.1^{L} - 17.2^{M}$	
		Outer	Adults: $10.4^{L} - 16.7^{M}$	
	Leadworkers		Adults: 14.6-20.7	Adults: 10.7-16.5
	Around leadworks		Children: 12.9-19.3	Children: 9.5-15.7
	Traffic of major roads	Near	Adults: 9.1-14.8 Children: 8.9-11.4	
		Same authority	Adults: 9.7-16.7	
Scotland	Plumbosolvent water	Glasgow	(Before treatment to raise pH) Adults (Random surveys): 18 3-month old infants*: 16 Mothers: 17	(After treatment to raise pH) Adults (Random surveys): 15 3-month old infants*: 11
		Ay		(Before treatment to raise pH) Mothers: 21

* Bottle-fed infants; L: London; M: Manchester

The Duplicate Diet Studies

The duplicate diet studies[11] in Scotland were co-ordinated by the DOE and the Ministry of Agriculture, Fisheries and Food (MAFF) and were carried out by the Department of Medicine of the University of Glasgow and the Water Department of the Strathclyde Regional Council in 1979-1980. The aim of the studies was to investigate the relationship between dietary lead and blood lead levels in infants and mothers in Glasgow (Lacey et al 1985; Sherlock and Quinn 1986) and Ayr (Sherlock et al 1982).

The studies were part of the EEC Blood Lead Surveys and a UK-wide study examining lead the diet of parts of the population that were more vulnerable to lead toxicity because of age (e.g. infants) or because of a higher risk of exposure to environmental lead, such as in areas with high air or water lead levels (DOE 1982 cited in Quinn 1985). Glasgow and Ayr, which were previously shown to have the highest levels of lead in drinking water in the UK (DOE 1983 cited in Quinn 1985), were therefore considered to pose an increased risk of high total lead intake and high lead exposure.

[8] i.e. men drinking beer, wine or spirits at a rate higher by 12% than non-drinking men (Quinn 1985).

[9] Social Class based on Occupation (Classification of Occupations. Office of Population Censuses and Surveys; London, HMSO, 1980. Cited in Quinn 1985).

[10] The older the age of a building the higher the possibility that it contains leaded paint and plumbing.

[11] The duplicate diet technique is based on the duplicate diet week: each time during the week that a mother gave her infant something to eat or drink, including non-milk items, she made double the amount; half was put in a container for later analyses and the infant fed with the remaining half (Sherlock and Quinn 1986).

The target date for these studies was 13 weeks after the infant's birth. The studies included a limited number of individuals and applied a stratified sampling strategy to identify lead intake and its effect on blood lead levels by focusing on households with plumbosolvent water. For example, in Glasgow, only 29% of the households taking part in the study had tap water lead levels below 50 µg/l, the remainder of households having water lead levels in the range of 100 µg/l to above 500 µg/l (Richards and Moore 1984). Also, only 131 mothers and their babies participated in the dietary lead tests. Likewise, In Ayr, only 31 mothers having children less than five years old and 11 infants were examined; only three of the households had water lead below 50 µg/l while in the remainder of households the water contained more than 110 µg/l of lead (Richards and Moore 1984). Given that one third of households in Scotland was estimated to have water lead levels above 50 µg/l at that time (DOE 1983), representatives from the two third of households, which were presumably served by water containing less than 50 µg/l, have not been included in the Duplicate Diet Studies.

The duplicate diet studies showed that:

- Baby food lead and maternal cord blood lead during pregnancy had a statistically detectable effect on an infant's blood lead levels but this effect was of negligible proportion compared with lead contained in the water used for the preparation of baby food (Lacey et al 1985).
- Blood lead was associated with food lead intake from formula prepared with tap water with a cube root, i.e. "curvilinear", relationship (Sherlock and Quinn 1986): e.g. blood lead (µg/dl)= 3.9 (± 2.6 µg/10ml) +2.43 (± 0.3 µg/dl) 3√(lead intake) (µg/week),
- Water lead was a stronger determinant than diet lead of blood lead in bottle-fed infants, especially when baby food had been prepared using water from the hot tap (Sherlock and Quinn 1986). 90% of the diet of bottle-fed infants is actually tap water, since formula was typically prepared by adding 8 parts of water to 1 part of powder.
- Blood lead in infants was associated with water lead with a cube root relationship (Sherlock and Quinn 1986): e.g. blood lead (µg/dl)= 5.5 +3.3 3√(water lead) (µg/l), $R2=0.23$, $p<0.01$
- Concentrations of lead in water well below the (then) standard for lead in drinking water of 50 µg/l can have proportionally very large effects on blood lead concentrations (Sherlock et al 1982; Lacey et al 1985; Sherlock and Quinn 1986).

The results of the duplicate studies are unique in showing the effect of lead exposure in infants. However, it remains uncertain to what extent drinking water and other types of food in other areas and age groups have contributed to total lead intake in Scotland because of the (unrepresentatively) limited number of individuals used.

The Edinburgh Lead Study

The Edinburgh Lead study was carried out in 1983 and examined the sources of exposure to environmental lead and the relationship between lead exposure and cognitive function in 855 boys and girls aged 6-9 years from 18 schools within a defined area of central Edinburgh (Fulton et al 1987; Laxen et al 1987 Raab et al 1990).

A significant relationship between blood lead in these children and water lead levels was demonstrated by Laxen et al (1987); this is further described in Annex IV in the context of evidence on the relationship between blood lead and drinking water.

Fulton et al (1987) examined a subsample of 501 of these children with a geometric mean of blood lead levels of 10.4 µg/dl (range: 3.3-34 µg/dl) to identify the relationship between blood lead and cognitive ability and educational attainment from the British Ability Scales (BAS). They found a significant negative relationship between log blood lead and BAS, after adjusting for 33 possible confounding factors (*inter alia:* parents' social class, health and mental health, and smoking habits; household ownership; school year; and child's birthweight and length of gestation). There was a dose-response relation between blood-lead and test scores, with no evidence of a threshold or a safe level (Fulton et al 1987). The authors did not preclude that this association could be the result of some confounding variable for which they did not control or of reverse causation, i.e. children who have low scores on cognitive tests would behave in way that renders them more likely to play outdoors in lead-contaminated places and not to wash hands. However they concluded that "reverse causation would be of less importance for water, which is an important source of lead in Edinburgh, than for other sources." (Fulton et al 1987).

The 501 children tested by Fulton et al (1987) were also analysed by Raab et al (1990) to identify the relationship of blood lead levels with reaction time and inspection time). Statistical analyses showed that inspection time in terms of attention ability was influenced by blood lead levels in these children (Raab et al 1990). However, the authors stressed that studies on the relationship between blood lead and cognitive metrics should better be based on long-term prospective (longitudinal) studies, where blood lead is measured in infancy, and at regular interval thereafter to minimise the effects of confounding factors and enable the detection of small effects (Raab et al 1990).

The Dundee study in neonates and mothers

This study was conducted by Zarembski et al (1983) in 1980 in an area of Scotland where water was not (considered as) plumbosolvent. Therefore, water was not discussed as a potential source of blood lead levels in this study. The study examined blood lead levels in 1665 mothers post-partum and in their infants at birth; this constitutes 70.9% of the total births in Dundee district during the period of study. Small numbers of individuals from other population groups (e.g. leadworkers, alcoholics, medical ward patients) were also tested. Blood lead levels in mothers ranged between 1.5 and 21.15 (arithmetic mean: 6.4 µg/dl; geometric mean: 5.96 µg/dl). In infants these ranged between 0.56 and 22.3 µg/dl (arithmetic mean: 4.4 µg/dl; geometric mean: 4.07 µg/dl). These values were considerably lower compared with (i) the blood lead levels in alcoholics and leadworkers tested in the Dundee study, e.g. blood lead in five alcoholic women in Dundee ranged between 16.6 and 22.3 µg/dl (Zarembski et al 1983); (ii) the blood lead levels elsewhere in Scotland and the UK (e.g. see EEC Blood Lead Surveys); and other areas influenced by moderate air lead emissions at that time (Zarembski et al 1983).

Studies comparing past and present sources of lead exposure in the Scottish population

- Delves and Campbell (1992) measured blood or teeth lead isotope ratios by ICP-MS to identify specific sources of childhood lead poisoning and to indicate the relative importance of environmental sources, such as drinking water and lead from petrol. Populations in the UK with low lead uptake usually have ^{206}Pb:^{207}Pb ratios in body tissues within the range 1.13 ± 0.01. Significant deviations from this range were observed in parts of Scotland (source ratio -1.18), because of increased uptake from lead in drinking water, and in inner London (source ratio -1.07) due to increased exposure to petrol lead. In Scotland, water lead contributed approximately 60% to body lead of some individuals with high concentrations of lead in blood or in teeth.

- Farmer et al (1994) studied the isotope ratio values for lead in the teeth of modern children in Edinburgh. They found that the ratios were intermediate between the observed ratios for lead in petrol and tap water and comparable with the ratios reported for food in the UK (Farmer et al 1994). It was stressed that quantitative source apportionment on the basis of one lead isotopic ratio, e.g. ^{206}Pb:^{207} Pb values may be hindered by the great similarity between old paint and tap water (Farmer et al 1994).

- Farmer et al (2006) compared the lead isotopic composition of various sections of teeth between 19th century skulls preserved in museum collections in Scotland and from individuals of known age residing in Scotland in the 1990's and observed striking shifts in the main sources of lead exposure. Lead exposure in the 19th century was mainly due to direct sources, such as local lead smelting and coal combustion, or indirect sources, via e.g. lead- contaminated food or drinking and cooking water contaminated by lead pipes of local origin (Farmer et al 2006). Lead exposure in the 20th century reflected the significant influence of imported Australian lead used as the tetraethyl lead additive in petrol in the UK from the 1930s until the end of the 20th century (Farmer et al 2006).

- Laxen et al (1987) showed as part of the Edinburgh Lead Study that combined residential exposure to water lead and indoor dust lead due to sanding or sloughing off of old leaded paint can explain approximately 32% of the variance of log blood lead in children. They also estimated that water lead can be a more important source of exposure than dust in a limited range of areas in Scotland with, what they characterised as, "moderate" water lead levels at the then action level for lead at 100 µg/l (Laxen et al 1987).

- Moffat (1989) demonstrated that in a rural, former mining area in the Southern Uplands of Scotland, water lead was the single most important determinant of blood lead levels in both adults and children. Water lead explained approximately 13% of the variation in blood lead levels. Other environmental sources of lead examined by Moffat (1989) were airborne dust, household dust, garden soil dust, kitchen surface, hand lead, and homegrown vegetables.

Individual research studies relating blood lead to health effects

Several studies have been conducted pre-1990 to explore the relationship of lead exposure and adverse effects. It should be taken into account that these studies were carried out using techniques with a much higher lead detection limit in blood than today and when researchers had a much poorer understanding of the adverse health effects of lead exposure. In some cases, the specific area of study is not reported. To illustrate:

- Beatie et al 1972 found that members of four families in the highlands had blood lead levels in the range of 23.9-163 µg/dl had symptoms like anaemia, abdominal pain, joint pain, and grout. Asymptomatic member of these families had blood lead levels in the range of 7.9-67.5

µg/dl. Lead in drinking water was found to be the source of lead exposure. Three months after lead pipe and storage tank replacement no decline in blood lead levels was observed; however, there was a significant drop in erythrocyte protoporphyrine levels. The interpretation of these findings was that blood lead is not a reliable and responsive indicator of lead exposure.

- Beatie et al 1975 compared blood lead levels between 77 mentally retarded children aged 2-6 years old who lived in houses with high water lead levels in the first year of life and 77 non-retarded matched controls. They found that the retarded group had significantly higher blood lead levels.
- Campbell et al (1977) measured serum urea and blood lead levels in the residents of old houses in Scotland with water lead levels above 100 µg/l and found that the group that had serum urea above the normal limit also had blood lead levels above 40 µg/dl.
- Beevers et al (1976) found that male hypertensives in the West of Scotland had significantly higher blood lead levels than normo-tensives and observed a positive correlation between blood lead and tap water levels They interpreted this as an explanation for the high prevalence of cardiovascular disease in the area.
- Moore et al (1977) observed that higher blood lead levels in retarded children was associated with high water lead levels in the maternal home during pregnancy.
- Beevers et al (1980) studied blood lead levels in hypertensives and normotensives in Renfrew, where water lead was commonly high due to soft water (hardness=5ppm). They found a significant association between blood lead and blood pressure and concluded that sub-clinical lead exposure may be a factor in the development of hypertension.
- A prospective study by Moore et al (1989)[12] in children aged 2 years old and whose mothers lived in the West of Scotland during pregnancy and had high blood lead levels, concluded that lead exposure was not a significant predictor of psychological function.

Annex III FACTORS INFLUENCING LEAD LEVELS IN DRINKING WATER

III.1 Materials

III.1.1 Lead in the distribution system

Lead may enter domestic and public water plumbing at a number of sites including:

- The water mains
- The service pipes, i.e. the communication and supply pipes.
- The premise plumbing, i.e. pipes, solder and other plumbing components used for conveying water to tap and are within homes, schools and other buildings.

The contribution of these materials on lead in drinking water has been evaluated through field studies, experiments and a review of research evidence by AWWA (2008) and is presented in Annex III-Table 1. A wider discussion on the ways materials can influence lead in drinking water is given below.

Annex III-Table 1. Sources of lead in the distribution system (AWWA 2008).

Source	Degree of contribution to lead in tap water
Lead service pipes	Up to 50-75%
Premise piping	Up to 20-35%
Water meters	Small
Faucets and immediate connective piping	Up to 1-3%
Multiple sources - sinks) and factors due to corrosion, scour, rapid Pb solubilisation and mixing /	Uncertain
Flushed samples representing the water mains lead (due to uptake of lead in service lines and premise plumbing)	Up to 3-15%
Partially replaced lead service pipes through lead migration from service pipes into premise plumbing	More research needed

The water main is an unlikely source of lead in drinking water today. The materials used for water mains are usually: cast or ductile iron; plastic, usually medium density polyethylene (MDPE) but also polyvinylchloride (PVC); asbestos; or cement (Mushak 2011). Leaching of lead from these materials does not normally occur (Hayes 2010). Utilities ceased to use lead since the 1940s in the UK

[12] Only abstract available – no values reported.

including Scotland (Potter 1997), and have fully replaced any lead-containing distribution mains over time. Potter (1997) also reports that in England and Wales lead was used until the 1960s to seal mains repairs and joint water mains to communication pipes; the risk associated with this use is thought to be inconsequential.

Lead, lead-lined or lead alloyed service pipes were commonly used in many countries including Scotland until the 1960s for conveyance of drinking water from the water main to commercial and domestic premises (Potter 1997; Mushak 2011). Their resistance to corrosive soil conditions and their malleable nature helped to endure changing ground conditions (Hayes 2010). Yet, a historian described this use in major cities as "one of the most serious environmental disasters" (Troesken 2006). Following banning, extensive replacement of lead service pipes with iron, copper or plastic pipes has taken place worldwide. However, districts with older premises may still be connected to the mains with lead communication and supply pipes, lead goosenecks and other pure lead components (Hayes 2010). Service pipes have been estimated to account for 50-75% of lead contamination in older homes where they are still in place and depending on pipe length and diameter and water pH, softness and consumption patterns (AWWA 2008). Hayes (2010) reports that the typical service pipe length in an urban/suburban residential estate may be between 5-100 m but, in rural settings, it can reach up to 300 m.

Lead pipes have also been used extensively within premises in Europe and elsewhere in the developed world until banning. Lead piping was gradually superseded by copper, steel or plastic piping from the 1950s in new buildings and during kitchen refurbishments through today (Hayes 2010). However, replacement of lead pipes buried within the walls of older premises is unlikely to have occurred even after banning lead in plumbing.

Solder containing 40-50% lead by weight and brass (and bronze) plumbing components (e.g. strainers, check valves, water meters, couplings, fittings, faucets, drinking fountains) were also used commonly within premises until banning. However, lead solder is still legal for other uses such as central heating systems. There is evidence that plumbers still use lead solder illegally or accidentally in jointing drinking water copper pipes (Ramsay et al 2002). In addition, "lead-free" brass in the UK still contains <3.5% of lead.

The contribution of lead solder to lead in water at a given tap is extremely variable, and depends on the number of joints, their age, workmanship when the joint was created, surface area of the solder exposed to water at each joint and the water chemistry (AWWA 2008). Recent cases of childhood lead poisoning from drinking water in North Carolina and in Maine were tied to lead solder particles that corroded and detached into the water supply (Triantafyllidou et al 2007). Recent problems with persistent lead contamination of tap water (up to 300 µg/L lead) in new buildings at the University of North Carolina at Chapel Hill were attributed to "lead-free" brass/bronze ball valves, installed before drinking water fountains. Locating and removing these ball valves was necessary to eliminate the lead problems at the fountains (Elfland et al 2010).

A recent British study (UKWIR 2014) found that both high- and low- lead brass fittings can cause exceedances of the 10 µg/l limit (and even of the value of 25 µg/l) for lead after a few hours of stagnation in the absence of any other lead leaching pipework or fittings without phosphate dosing; orthophosphate dosing was found to reduce both the time taken to reach a stable lead leaching concentration during stagnation and the final concentration compared with non-phosphate dosed water. The same study found that stagnation, lead content of brass fittings and the age of an installation are the key issues affecting observed leaching (UKWIR 2014).

Prevalence of lead pipes

The prevalence of lead piping remains largely unquantified. Available but relatively old surveys refer to:

- An estimated 25% of domestic dwelling sin the EU have a lead pipe, either as a connection to the water main, or as part of the internal plumbing, or both, potentially putting 120 million people at risk from lead in drinking water within the EU (Hayes and Skubala 2009).
- A collection of "best estimates" of occurrence of lead communication and supply/internal pipes in pre-2000 EU Member States (MS) showing a wide range of lead pipe occurrence from 0% in Denmark and Greece to approximately 40% of service lines and connected premises in the UK and France (van der Hoven et al 1999).
- Rough estimates that 34% of households in England and Wales had at least some lead pipework as part of their drinking water supply in mid-1990s (Water Research Centre-WRC cited in Potter 1997).
- In Ireland there were still approximately 5000 meters of lead mains in use in 2012 (Irish EPA 2013).
- Surveys in Japan showing a total of 667 km lead pipe below roads and 3,248 km of lead pipe in residential areas (Osawa 2002 cited in Triantafyllidou et al 2012).

III.1.2 Interactions between materials

Galvanic corrosion

Galvanic, i.e. electrochemical, reactions are the major mechanism whereby lead in premise pipes, solder, brass fittings, faucets and similar premise plumbing components leaches into water when exposed to water flow through copper pipes (in service or premise pipes) (Triantafyllidou et al 2009). Lead in plumbing materials functions as the anode and copper functions as the cathode. The short-term effect of galvanic corrosion is due to disturbance of the lead dioxide scales that have accumulated on the lead pipe over decades/centuries of use, and/or from creation of metallic lead particles when the lead pipe is cut during partial pipe replacement (Triantafyllidou et al 2009; Abokifa et al 2017).

Generally, increasing the chloride: sulphate mass ratio of the water results in higher lead levels in water when copper pipe - lead solder or copper - lead pipe galvanic couples are present (Cuppett 2017). Several studies have shown that galvanic corrosion may be triggered by conditions that cause higher chloride and lower sulfate in the water due to, e.g. road salt entering the water supply from runoff; coagulant type (chloride-based vs. sulfate-based); desalination; chloride-based anion exchange treatment; brine leak from hypochlorite generation system (Oliphant 1983; Gregory 1985; Reiber 1991; Singley 1994; Lauer 2005, Nguyen et al 2010; Nguyen et al 2010; Triantafyllidou and Edwards 2011; Clark et al 2013; Wang et al 2013; Cuppett 2017). Studies by the Water Research Foundation (Cuppett 2017) also demonstrated that galvanic effects appear to be very sensitive to chlorine/chloramines (or vice versa) transitions; however, the effect was highly transient in their experiments and under steady state conditions, the shock of initial exposure quickly waned.

Recent research has also shown that in some situations, the creation of a galvanic cell (i.e. battery) between the lead pipe and the copper pipe may create serious water lead contamination in both the short-term and longer-term (Triantafyllidou et al 2009; Abokifa et al 2017). This is an important finding in the context of this report: for example, the higher incidence of elevated blood lead levels in children living in homes with partially replaced lead pipe, when compared to homes with full lead pipes, has been attributed to galvanic corrosion (Renner 2010a; Brown et al 2012).

Arnold and Edwards (2012) studied the role of water use patterns, disinfectant type, and orthophosphate on lead release to water from galvanic lead-cupper couples. They demonstrated that if galvanic lead-copper connections were present low flow markedly increased the mass of lead release to water and resultant consumer exposure. Three chemical mechanisms were identified that could reduce lead release at higher flow (Arnold and Edwards 2012): (1) formation of lead(IV), (2) potential reversal of Pb:Cu couples, after which lead is protected, and (3) reduced formation of corrosive microenvironments at lead surfaces in galvanic couples. Interestingly, potential reversal (lead pipe becoming cathodic to copper thus copper pipe is leached and lead pipe or components remain unaffected) occurred only in the presence of free chlorine with continuous flow, and it did not occur with chloramine, with intermittent flow, or if orthophosphate was present (Arnold and Edwards 2012).

Polyvinyl chloride (PVC)-lead or brass connections

Several studies have found that nitrification in PVC premise plumbing is a weak function of pH over the range 6.5–8.5 and is insensitive to phosphate concentrations 5–1000 ppb. Zhang (2008) found that lead pipe enhanced nitrification relative to PVC. Lead materials have been found to convert products of nitrification (nitrite and nitrate) back to ammonia via anodic corrosion reactions (Uchida dn Okuwaki 1998 cited in Zhang 2008). Zinc in galvanized pipe has also been reported to convert nitrite to ammonia under drinking water conditions (Kunzler et al 1983 cited in Zhang 2008). It has been argued that nitrifier (microbial) growth on lead and zinc alloy surfaces would be favoured relative to more inert surfaces such as polyvinyl chloride (PVC), exacerbating nitrification occurrence and undermining efforts to mitigate lead contamination of water supplies from pH adjustment with orthophosphate dosing (Edwards and Dudi 2004; Zhang 2008; Zhang and Edwards 2009).

Partial versus total pipe replacement

Field observations of actual partial lead pipe partial replacement showed elevated lead release following replacement of the lead pipes with metal or plastic pipes due to galvanic corrosion, the levels being higher with metal pipes than with plastic pipes in the utility's side (Welter et al 2013; Schock et al 2014a and literature cited therein). However, it took time—in some cases, four to six months (Welter et al 2013), in other cases more than four years (Schock et al 2014a) —for the accumulated lead to be released and then additional time for re-equilibration with the "new" relatively lead-free water. Other studies conducted in the USA concluded that partial lead pipe replacement may be worse than leaving the service pipes intact due to the potential for elevated particulate lead release (Trueman et al 2016; Abokifa and Biswas 2017).

Total lead pipe replacement in Madison, illustrated that removing the source of lead that resupplies the lead accumulation on manganese- and iron-rich scale can eventually eliminate the significant particulate lead concentration found at the faucets (Schock et al 2014a). In

Marshfield, partial pipe replacement showed that if the lead source remains in the system but the manganese source is diminished through control of dissolved and particulate manganese entering a residence, the erratically high lead concentrations can also be lowered (Schock et al 2014a).

A recent study in Halifax, NS, also implicated iron particulates as an important factor in elevated lead levels following partial lead pipe replacement (Camera et al 2013). Conversely, in Guelph, Ont., where a high-alkalinity groundwater was used (Muylwyk et al, 2011, 2009 and Gilks 2008 cited in Camara et al 2013), total pipe replacement resulted in rapidly, consistently, and permanently removing the major source of exposure to lead in drinking water in first draw and in fully flushed lead levels.

Triantafyllidou (2011) illustrated the effect of lead in service pipes and plumbing components in lead levels in tap water with a thought experiment to put the lead pipe problem into perspective. She calculated that a typical lead supply pipe in the US is about 19 kg. If only 0.1% of this lead pipe is dissolved, degraded, or sloughed off the pipe wall due to corrosion and is released to the water, the released lead mass of 19 grams is sufficient to contaminate every drop of water used by a US family of three for three years over 15 µg/L, which is the US EPA limit for lead in drinking water[13].

III.2 Water chemistry

III.2.1 Forms of lead in drinking water

Lead from the water distribution system can be present in tap water as:

- Dissolved free aqueous ions, inorganic and organic complexes and associations with colloidal matter which result from the corrosion of lead-bearing pipes and plumbing components or from dissolution of suspended or deposited particulate lead (De Rosa and Williams 1992 cited in Triantafyllidou and Edwards 2011 2011).
- Suspended particulate lead, which mainly includes insoluble particles larger than 0.45 µm. Particulate lead can derive from physical degradation or detachment of lead-bearing deposits inside the water distribution system, or by flaking off during turbid water flow (Schock 1990; Triantafyllidou et al 2007).
- Deposited particulate lead, also known as scales or passivating layer, which can be produced by electrochemical precipitation of soluble lead at various points in the distribution system or in premise plumbing (De Rosa and Williams 1992) or chemical precipitation of insoluble lead (Schock and Lytle 2011).

Dissolved lead is not always the predominant form of lead (Triantafyllidou et al 2007). Several studies have shown that suspended particulate lead in tap water is more common than originally thought, as shown e.g. in water from rural areas near Glasgow (De Mora et al 1987) and for the UK in general (De Rosa and Williams 1992 cited in Triantafyllidou and Edwards 2011). The contribution of particulate lead to the total lead concentration has also been observed by others (e.g. McNeill and Edwards 2004; Zhang and Edwards 2009; Deshommes et al. 2010; Triantafyllidou and Edwards 2011). The presence of suspended particulate lead may result in concentrations as high as 1000 µg/l, as shown in numerous instances from around the US (McNeill and Edwards 2004). Deshommes et al. (2010) demonstrated that lead release was associated with iron particles, whereas Schock et al. (2008) found significant amounts of iron in lead-based scales within lead pipes. In view of these findings, Schock et al. (2008) warned that lead released as particulates may result in long-term intermittent high lead levels in tap water that can easily go undetected.

As for deposited particulate lead, three situations have been observed for the fate of scales (Schock and Lytle 2011): (i) chemical precipitation onto a previously deposited scale, including scale deposited onto or co-deposited with scale of a different composition; (ii) break off and then re-deposition at a different site of the distribution system; or (iii) formation of a layer on larger suspended lead-containing particles. In general, many lead-bearing distribution systems conveying water with high carbonate alkalinity have a coating of calcium carbonate scales. If lead leaching occurs, any addition of lead would be compensated by growth of a lead-carbonate coating, which provides a protective, low solubility layer between drinking water and lead in plumbing for as long as water pH is approximately 9.8 (Schock et al 1996). However, when mechanically disrupted, e.g. by high and intermittent flow, the lead-carbonate layer becomes a sudden, episodic source of high lead levels in the tap water (Schock et al. 2008).

When water is very soft and has low alkalinity, lead leaching results in the formation of a lead (IV) oxide coating of lead oxide (PbO_2), which has low solubility under certain circumstances. Maintaining the low solubility requires the water chemistry be sufficiently oxidizing to maintain lead in the +IV oxidation state; the presence of free chlorine as disinfectant in contact with lead pipes provides the oxidising conditions and results in a low dissolved lead concentration (Vasquez et al 2006; Boyd et al. 2008; Wang et al 2012). The solubility of Pb(IV) in the form of lead dioxide (PbO_2) has been found to be very low compared to Pb(II) carbonate and phosphate species (Schock et al 1996; Schock 1990). Experiments by Lytle and Schock (2005) showed the

[13] calculation based on 1135 L/day water usage for the whole family

formation of a layer of lead dioxide in the presence of free chlorine used for disinfection.

Evidence shows that the factors with the potential to hinder the formation of lead (IV) scales include:

- Reduced pH (Lytle and Schock 2005); the effect of pH is discussed in section -pH
- Natural organic matter-NOM (Dryer and Koshyn 2007; Lin and Valentine 2009); the effect of NOMs is discussed in section 3.1.2.2-Organics.
- Chloramines (e.g. Lin and Valentine 2009); the effect of switching to chloramine disinfection on lead leaching with and without orthophosphate dosing is discussed more explicitly in section -Disinfectant.
- The presence of orthophosphate (e.g. Lytle et al 2009); this is further explained in section -Corrosion inhibitors.

III.2.2 Chemical factors influencing lead leaching

(i) Disinfectant-Redox-Flow

An earlier CREW report (Postlewait 2012) has delivered a review of the effects of switching to chloramines for disinfection and recommended that it is not advisable to use chloramines in combination with lead pipes. Section III3.3 here refers strictly to chemical mechanisms of lead leaching related to redox due to type of disinfectant, to help understand the potential of exposure to lead through fluctuations in redox.

The concern about the type of disinfectant in relation to lead leaching and plumbosolvency control is of relatively recent vintage. This is mainly because of the "lead in drinking water crisis" triggered by a change in disinfectant from free chlorine to chloramine in November 2000 in Washington, DC (Edwards and Dudi 2004). This led to rises in water lead levels from below 15 µg/l with free chlorine to above 40 µg/l within six months after the switch to chloramine and rises in children's blood lead levels well above the specified allowable limits (Guidotti 2007; CDC 2010; Edwards 2010; Brown et al 2012; Edwards 2013). Since then, a number of studies have shown that the circumstances under which the commonly used treatment of water with chloramines and corrosion inhibitors is effective in reducing lead leaching are not fully understood (e.g. Lin et al 1997; Edwards and Dudi 2004; Boyd et al 2008; Wang et al 2012; UKWIR 2016; US EPA 2016). There were also laboratory and field findings showing that the type of disinfectant is, under certain circumstances, less crucial than optimising orthophosphate dosing by controlling pH, alkalinity and organics (e.g. Cantor et al 2003; Vasquez et al 2006; Wilczack et al 2010).

However, the current generally agreed understanding of the effect of disinfectants on lead leaching, as reviewed by Boyd et al (2008) and Liu et al (2009), is that

(i) The type of disinfectant exerts a strong influence on redox, which controls lead solubility by determining the dominant form of lead in the distribution system.

(ii) Chloramines can destabilise the deposited scales.

(iii) The effect of disinfectant on lead leaching is interrelated with all the other factors influencing lead leaching, such as pH, DIC, organics, temperature, ammonia, sulphate and the type of materials present in the distribution systems in addition to lead (e.g. copper, PVC).

A summary of this evidence is given below.

Why does the type of disinfectant matter in the context of lead in drinking water and public health

The effect of disinfectant on redox is crucial because redox determines lead solubility. The solubility of lead dioxide film remains low when free chlorine is used for disinfection (Boyd et al. 2008; Wang et al 2012). Chlorine is a very strong oxidizer maintaining a high redox potential, thus allowing for the formation of a stable film of passivating lead dioxide scales with lead (IV), which limits the release of lead and other metals into drinking water (Edwards and Dudi 2004; Vasquez et al 2006; Boyd et al 2008; Switzer et al 2006; Wang et al 2012). A lead precipitation study by Lytle & Schock (2005) demonstrated that lead dioxide scale formed over time when a high redox potential was maintained using free chlorine. The lead dioxide formation rate increased with increasing pH, and lead dioxide either dominated or coexisted with lead(II) mineral forms including lead carbonate, hydrocerussite and/or cerussite.

A recent study by Schock, Cantor, et al 2014 found that communication and supply pipes with lead oxide scales under free chlorine were associated with low lead levels in tap water - as low as or lower than those found when orthophosphate treatment is used. The Pb(IV) scale, however, can be compromised by small changes in the oxidation state in the distribution system. This is sometimes difficult to reliably monitor and control, resulting in intermittent problems exceeding the lead standard (Brown et al 2015).

How do chloramines destabilise the deposited scales

Chloramine alters the chemistry of conveyed water by oxidizing lead in service pipes, solder, and brass to soluble lead(II), which in turn causes unexpected lead leaching from lead communication and supply pipes and premise plumbing (Edwards and Dudi 2004; Renner 2009; Lytle and Schock 2005). Decomposition of chloramines has been found to be proportional to lead(II) release through a reduction of lead oxide (Lin and Valentine 2008). Examples of sudden rises of

lead in drinking water in low risk supply zones because of change of disinfectant have been reported in Washington DC (e.g. Edwards and Dudi 2004); and Greenville, NC (e.g. Renner 2010b).

Chloramines are also known to cause nitrification if too much free ammonia is allowed to remain in the distribution system (Sung et al 2005; Zhang and Edwards 2009; Zhang 2008). The nitrification process has potential to locally lower the pH in alkaline waters and cause lead leaching and dissolution of the scale. In some systems using chloramines, brass in plastic plumbing systems might be more susceptible to lead/copper leaching, and accelerated dezincification, due to lower pH values resulting from nitrification (Zhang et al 2008).

Under laboratory conditions, (DIC =10 mg, copper-lead, T=24°C, pH=7.75-8.1, 3 mgl/L Chlorine target), Lytle et al (2009) showed by means of SEM and XRD analysis that orthophosphate dosing (30 mg/L) inhibited the formation of lead(IV) oxides. The authors argued that this helped explain why many water systems did not observe elevated lead release upon change form free chlorine to monochloramine as their secondary disinfectant. Presumably in such systems, lead(II)-phosphate compounds were the major components in deposits on lead surfaces and, as a result, lead(IV) oxides never developed. Therefore, the deposits were not impacted by redox changes associated with disinfectant changes.

How is disinfectant interrelated with the other factors influencing lead leaching?

Xie and Giammar (2011) conducted experiments on the dissolution of lead dioxide and concluded that dissolution rates decreased in the following order:

no disinfectant>monochloramine>chlorine,

which was consistent with the trend in the redox potential.

Edwards and Dudi (2004) found that chloramines did not significantly increase lead release from new lead pipe without a galvanic connection under mostly stagnant conditions in the presence of orthophosphate. Arnold and Edwards (2012) observed the same behaviour for new lead pipe and orthophosphate with intermittent flow but not continuous flow. Both studies emphasised the negative impact of chloramines on the formation of protective lead dioxide layers and facilitation of potential reversal between lead and copper pipe (Edwards and Dudi 2004; Arnold and Edwards 2012).

Woszczynski et al (2013) carried out studies in an experimental lead-copper system (copper pipes, lead solder), where the treated water had a low alkalinity (<5 mg=L as CaCO3), neutral pH at 7.3, and low hardness (<5 mg=L as CaCO3) and a zinc orthophosphate dose of 0.8-mg/l of phosphate. In their case study, the lead(IV) oxides present under free chlorine were more stable and resistant to high velocities, as compared to the lead complexed with ammonia in the chloramine system. They also showed that the total lead concentration (dissolved and particulate) was lower with chlorine than chloramine following 24 h and 30 minutes of stagnation. The results are illustrated in Annex III-Figure 2.

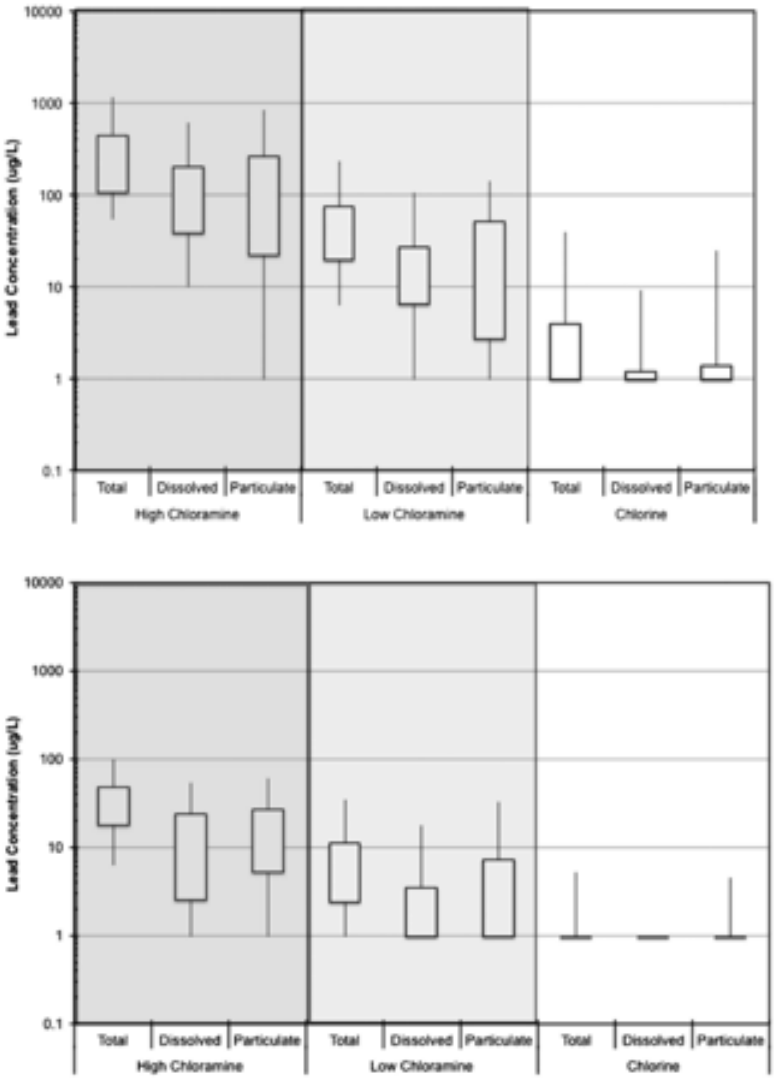


Figure 5. Comparative presentation of the results of a case study to assess lead release in a pipe rig system with 60 pipe loops that had copper loops and lead solder, which provided the only source of lead. The system was disinfected with either chloramines or free chlorine and was treated with zinc orthophosphate (0.8 mg/l of phosphate) for lead corrosion inhibition. Top: samples collected after 24-hour stagnation. Bottom: samples collected after 30 minutes stagnation. Source: Woszczynski et al 2013.

In the absence of phosphate, at an alkalinity range of 67-197 mg/l as CaCO3 and a pH range of 7.7-8.3[14], Vasquez et al (2006) compared laboratory and field data from eight identical pilot distribution systems containing copper and lead to assess the effect of chlorine versus chloramine

[14] This range overlaps with the range achieved via orthophosphate dosing and for this reason this study may be relevant in the context of orthosphosphate dosing.

on lead leaching in lead containing systems in Florida. Treated groundwater, surface water, desalinated water and blended water were used. The experiments based on 6-8-hour stagnation samples showed that chloramine residual produced a lower redox potential in the water and promoted a hydrocerussite-controlling solid phase, which released more total lead (dissolved and particulate) relative to free chlorine. Conversely, free chlorine produced a higher redox potential and a lead dioxide-controlling solid phase, which released less total lead compared to chloramines (Vasquez et al 2006).

Pilot experiments (five experimental treatments with six replicates each) by UKWIR (2016) showed that switching the disinfection of soft water from chlorine to chloramine, without lead corrosion control, significantly increased lead leaching from brass fittings after a 16-hour stagnation period. In orthophosphate treatments, lead leaching was significantly lower in both hard and soft water with chloramine compared with non-phosphate chlorine and chloramine disinfection. It was noted that "The limited experimentation comparing chlorinated and chloraminated treatments, found some statistical differences in leaching characteristics between each form of chlorine residual and types of waters, which could warrant further investigation." (UK WIR 2016).

(ii) pH/alkalinity/DIC

The pH, alkalinity (i.e. the capacity of water to neutralise acid or $2CO_3^{2-} + HCO_3^- + OH^- – H^+$) and DIC (i.e. $CO_2 + H_2CO_3 + CO_3^{2-} + HCO3^-$) are closely related. It is generally known that the pH influences buffer capacity, alkalinity and the effectiveness of orthophosphate dosing and the DIC determines the availability of carbonate that is available for lead to form the protective lead carbonate coating. At pH values below about 5, lead is soluble as Pb(II). At pH values higher than 9 and depending on the alkalinity and DIC, lead is insoluble as lead carbonate. Chemical reactions between water components (e.g. iron, chlorine and chloramine) and the water distribution materials (e.g. lead and copper) and microbiological activity can cause fluctuations in the pH, which can be buffered by the carbonate and bicarbonate ions. Detailed description of the factors that influence water pH can be found in reviews by the American Water Works Association-AWWA (e.g. 1990; 2008) and also in research publications (De Mora et al 1987; Schock 1990; Schock et al 1996; Croll 2000; Schock and Lytle 2011; Brown et al 2015; US EPA 2016).

Lead corrosion control using pH/alkalinity adjustment may be problematic:

- Optimal pH to reducing lead corrosion may be sub-optimal or unsuitable for chlorine disinfection. For example, increasing the pH prior to adding chlorine may reduce disinfection performance and require an increase in chlorine dose or contact time (Schock 1989). Also, increasing pH may not be suitable for all supplies because of the risk of formation of disinfection byproducts, such as THM (Schock et al 1996; Schock and Lytle 2011) and difficulty in maintaining pH and alkalinity from the treatment plant to the tap (Hayes 2010). US EPA (2016) advise that a supply system must develop disinfection/pH profiles and calculate disinfection benchmarks for certain microbial strains to ensure optimum protection of the general public from lead leaching and microbiological contamination.
- Raising pH and DIC to reduce lead corrosion may cause calcium carbonate precipitation; however, this is site-specific and depends on many factors that cannot always be predicted without proper system evaluation (Brown et al 2012). Also, this problem refers mainly to high calcium-related hardness. Calcium carbonate precipitation is known to cause clogging of the distribution system, depending on use (cold or hot water) and pipe diameter and produce cloudy water.
- Increasing the pH has been found to increase the rate of iron and manganese oxidation (Gagnon et al 2004; Lytle et al 2004; Sarin et al 2004).). This causes discolouration of water e.g. black and/or red water, which is one of the major complaints to water suppliers (Rahman and Gagnon 2014).
- (compared with the commoner practice of orthophosphate), larger doses of pH adjustment chemicals (lime, caustic soda, soda ash) are needed to adjust pH to decrease lead solubility without than with orthophosphate (Schock 1989; AWWA 1990). Brown et al (2015) argued that this may not be so in a softening plant.

(iii) Iron, Manganese and Aluminium

Manganese and iron can react with dissolved lead and form deposits on lead service lines and plumbing components within premises. Schock et al (2014) showed that the manganese deposited on pipe scales (up to 10 percent by weight of scale composition) captured dissolved lead and later, when disturbed by higher flow, released it back into the drinking water in Madison, WI.

A well-known consequence of pH increase for lead control is the "black" or "red" water complaints due to oxidation of iron and manganese in the distribution system. Iron and manganese removal at the treatment plant, or possibly the use of sequestering agents (e.g. polyphosphates or silicates), can be used in these cases (US EPA 2016). The problem is that polyphosphate may increase lead leaching (see section-Corrosion Inhibitors). An additional problem is that lead may be sorbed into iron- or manganese precipitates and released as a result of iron and manganese corrosion

of galvanized, ductile iron, cast iron, and other pipes and premise plumbing (Camara et al. 2013, Schock et al. 2008 and 2014). This would be an indirect benefit of controlling for iron and manganese corrosion (Brown et al 2015).

Manganese and iron deposits within lead pipes could function as sites of adsorption of lead and as sources from which lead, iron and manganese are released (Deshomes et al 2010; Schock et al 2014b; Knowles et al 2015). Whereas manganese scale typically develops from a source water constituent, iron scale can develop from either source water composition or from the corrosion of iron pipes (cast-iron water mains and galvanized iron premise piping) (Schock et al 2014b). Experiments in a model distribution system dose with orthophosphate (0.5 and 1 mg/l) showed that orthophosphate can decrease lead levels but it cannot reduce the effect of an upstream iron main on triggering the release of colloidal lead and iron particles from iron-enriched lead scales (Trueman and Gagnon 2016).

Aluminium in water can also reduce the amount of orthophosphate available for lead control and may precipitate on the interior of piping systems, thus reducing the effective diameter of the pipes, with important implications for hydraulic capacity and operational costs (AWWA 2005 cited in Brown et al 2015). If sloughed by changes in flow and water quality, these precipitates may become entrapped in the premise plumbing and/or the faucet screen, potentially increasing lead in tap water (Schock 2007 cited in Brown et al 2015). Water systems using coagulants may have raw water at pH 7 to 8 but then they drop the pH to 6 or lower for optimal coagulation, thereby requiring even more pH adjustment chemicals to get to pH 9 or higher as needed to control lead solubility without orthophosphate (Brown et al 2015).

(iv) Ammonia

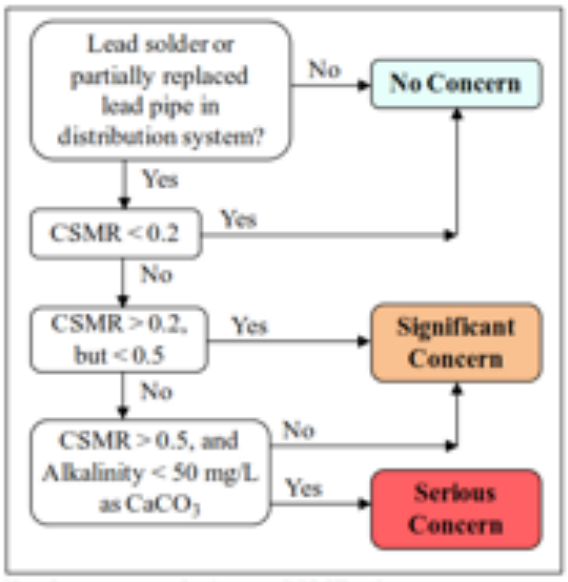
Ammonia increases lead leaching because it can reduce the pH of the water. Ammonia is removed at treatment plants of large supplies but may be present in small water supplies or be elevated when chloramines are used for disinfection in public water systems (Nguyen 2011). The chloramine is assumed to decay first to chlorine and ammonia, and then the ammonia is converted, by biological action, to nitrite and then nitrate (Kirmeyer 2004). In the case of ammonia from chloramine, lead leaching has been attributed to pH declines, which result from the release of H+ during the chloramine autodecomposition (Lin and Valentine 2008). Ammonia from chloramines has been implicated in the corrosion of lead containing brass fittings (e.g. Edwards and Dudi 2004) and polivynil chloride (PVC) (e.g. Zhang and Lin 2015). Lead-containing materials have also been found to convert products of nitrification (nitrite and nitrate) back to ammonia via anodic corrosion reactions (Uchida and Okuwaki 1998 cited in Zhang et al 2008).

Zhang and Lin (2015) showed that in the presence of ammonia from monochloramine, lead release from systems using unplasticised PVC was faster than that in the presence of free chlorine: lead in the tap water in the presence of chloramines were 81, 120 and 92 µg/l, which were 1.9, 2.3 and 2.8 times higher than those observed in chlorinated water or disinfectant-free water. Zhang and Lin (2015) emphasised the importance of the buffer capacity of water in maintaining the correct range of pH values in the presence of chloramine/ammonia.

Ammonia may also be a problem in small water supplies when elevated ammonia may lead to nitrification, which may lower pH and trigger lead corrosion (Douglas et al 2004).

(v) Chloride and sulphate

The chloride:sulphate mass ratio (CSMR) in drinking water is used as an indicator of lead corrosion (Hill and Cantor 2011 cited in Masten et al 2016). Lower chloride:sulphate ratios suggest lower lead dissolution due to the formation of an insoluble sulphate lead deposit; higher chloride:sulphate ratios indicate the formation of a soluble chloride complex due to galvanic (electrochemical) corrosion (Nguyen et al 2010; 2011). The levels of CSMR are very important in controlling lead leaching. In Columbus, Ohio, the 90th percentile lead levels in the water increased by almost 360% after a change in coagulant from aluminium to ferric chloride, which resulted in an increase in the CSMR by up to 170% (Hill & Cantor 2011 cited in Masten et al 2016). An extensive survey of water utilities in the USA showed that 100% of utilities with a CSMR <0.58 met USEPA action limit for lead (15 µg/l) but only 3 6 % of utilities with a CSMR >0.58 met this limit. (Edwardsand Triantafyllidou 2007). Nguyen et al (2010) developed a chart to assess the level of lead concern in relation to CSMR, which was suggested to be at the value of 2 (Annex III-Figure 1).



Annex III-Figure 1. Level of lead concern in relation to CSMR. Source: Nguyen et al 2010.

The recent water crisis in Flint, which caused very high blood lead levels in the children of the area, has been associated inter alia with a failure to recognise the importance of the CSMR rise as an indicator of lead leaching (Hanna-Attisha et al 2016; Masten et al 2016). It is important to note that at some point during the crisis, the alkalinity of the water was less than 5 0 mg/l and the CSMR greatly exceeded 0.2 (Masten et al 2016). Despite this, phosphate dosing was not used as corrosion inhibitor; however, Masten et al (2016) argued that "the CSMR of the treated Flint River water was so high that, even with the addition of phosphate, the water may have been so corrosive that lead levels in the system might have still exceeded the action level."

(vi) Organics

Fulvic and humic acids also have the potential to increase dissolution of lead from lead pipes by up to ten times due to their effect on pH (Hayes and Skubala 2009). Dryer and Koshin (2007) provided evidence that lead release (mobilisation) in the presence of natural organic matter (NOM) may be a slow process evolving throughout many weeks depending the concentration of NOM but not significantly influenced by the type of NOM used in the experiments. Experimental work by Liu et al (2009) suggested a mechanism for this, whereby lead dioxide particles are susceptible to colloidal disaggregation and mobilization in the presence of NOM due to the sorption of NOM and ensuing alteration of the surface properties of lead dioxide particles. The experiments by Liu et al (2009) did not show any effect on scale mobilisation from the presence of chloramine.

III.3 Other factors

III.3.1 Temperature

Concerns on the effect of temperature variations on lead leaching are of relatively recent vintage. At higher temperatures, the rate of electrochemical reactions between lead and copper increases, but the solubility of lead-coating on lead service pipes and premise plumbing may increase or decrease, depending on their composition (Britton and Richards 1981; Schock 1990; Vasquez et al 2006). Temperature effects also depend on the source of lead in the distribution system. For example, plumbing within premises (e.g. internal pipes, lead solder, brass fittings and faucets) is less likely to be influenced by seasonal variation but more likely to be influenced by day to day and house-to-house, within a supply zone variations (Cartier et al 2011; Trueman et al 2016). On the other hand, lead in service lines depends on seasonal variations of temperature, as shown in a Scottish study (Britton and Richards 1981).

Seasonal temperature rise (e.g. summer versus winter) was found to accelerate lead dissolution from lead pipes in many instances. For example, Ngueta et al. 2014 reported marked winter-to-summer changes in water lead levels in the Montreal area, with the geometric means of lead (± SE) being 2.7 ± 2.2 µg/L during winter and 8.1 ± 1.5 µg/L during summer. Trueman et al (2016) showed that between 13 and 19 °C, a 1 °C increase accompanied an average 1.1 µg/l increase in lead release from lead service lines in 5 min flushed samples in a supply zone in Halifax, Canada treated with a blended zinc-ortho/polyphosphate corrosion inhibitor (75% orthophosphate, 25% polyphosphate).

In a similar study in the Montreal area, Cartier et al (2011) found a 5% rise in dissolved lead per 1oC increase in temperature from lead service lines and a lower contribution from premise plumbing. This seasonality dependence of lead concentrations was also captured by a 2000-2004 survey on the effectiveness of plumbosolvency control at London's water company Thames Water, which was evaluated by AWWA (2008). This survey showed that, after the addition and optimisation of orthophosphate dosing, lead concentrations co-varied with temperature, with non-compliances with the standard of 10 µg/l increasing in the summer time (AWWA 2008).

In support to these findings, experiments by Masters et al (2016) in supply zones in Washington, DC and Providence, RI showed that (i) in the presence of natural organic matter, lead dioxide dissolution was 36 times greater (36 versus 1277 ppb) at 20 °C compared to 4 °C due to accelerated reductive dissolution; (ii) dissolved lead release increased by as much as 2-3 times in the summer versus winter; (iii) particulate lead increased 2-6 times in the summer versus winter; and (iv) tap water lead levels increased in summer compared with winter in four out of eight homes studied in Providence, RI (USA).

Overall, many researchers have stressed that these findings show that seasonal temperature variation may affect the reliability of cross-sectional studies (i.e. studies based on data collected from a population, or a representative subset, at a specific point in time) and compliance monitoring protocols (e.g. AWWA 2008; Cartier et al 2011; Trueman et al 2016; Masters et al 2016; Ngueta et al 2014).

III.3.2 Stagnation time-Flow – Flushing

(i) Stagnation

The effect of stagnation time on lead levels in tap water has been studied extensively to inform the design of compliance monitoring protocols and recommendations for domestic, every day tap water, e.g. duration of flushing.

Lead dissolution is an exponential function of stagnation time. Over 60% of lead leaching can occur within 1-hour of stagnation and up to 30% may be found in the first 10-minute sample (Lilly and Maas 1990). Tests carried out by AWWA (1990) showed that maxima in tap water lead levels appear within 16 and 64 hours of water stagnation in the distribution system within premises. LytLe and Schock (2000) found that the sharpest increase in lead dissolution occurs over the first 20–24 h of stagnation but equilibrium concentrations appear at or after 6-hours of stagnation; however, resulting concentrations in tap water are difficult to predict and dependent on water chemistry.

Likewise, Kuch and Wagner (1983), who established stagnation profiles for lead in drinking water, found that despite the increase of lead leaching at lower pH and alkalinity values, lead equilibrium concentration appears at or after the 6-hour mark. Hayes (2009) reported that the amount of lead released in the water is lower in less plumbosolvent waters but reaches equilibrium at 16-hours of stagnation. Edwards and McNeill (2002) compared the behaviour of different types of lead corrosion control treatments and found that lead levels in water fluctuate during a 72-hour stagnation period; the narrower degree of variation in a three-year period was tied in with the use of orthophosphate as lead corrosion inhibitor.

The key effect of prolonged stagnation time is that it initiates or enhances galvanic corrosion problems (e.g. Nguyen et al 2010); see also Annex III-1.2-Galvanic corrosion. Triantafyllidou and Edwards (2011) demonstrated that galvanic connections between copper pipe and lead pipe (either new or aged) increased lead release compared to a full length of lead pipe alone, under a so-called "worst case scenario", i.e. 8-hour overnight stagnation. Eight-hour stagnation is considered atypical of normal domestic water consumption patterns and reflects peak lead exposure conditions (AWWA 2008). Levin et al (2008) argued that periods of little or no consumption during weekends and breaks in schools or other public buildings, may result in long stagnation periods of the water inside partially replaced lead piping and therefore in higher lead levels in the tap water.

III.3.3 Flushing

Flushing has been shown to exert the opposite effect to stagnation. A range of studies demonstrated that flushing the night before sampling, even for as little as 10 minutes, can reduce the lead collected the following morning (Murphy 1993; Triantafyllidou et al 2009). However, high lead levels return if flushing is not repeated on subsequent days. Murphy (1993) found that 10 minutes of flushing reduced lead levels by about 50 percent. These flushing studies were conducted at low velocity, not the higher velocity conditions analogous to unidirectional flushing in the distribution system.

Elevated particulate lead levels following Partial-LPR have been associated with inter alia dislodging of particulate lead from the remaining service pipe during construction (AWWA 2008); galvanic corrosion (Nguyen et al 2010; Edwards and Triantafyllidou 2007; Britton and Richards 1981; McFadden et al 2008; AWWA 2008) and mechanical disturbances or sudden flow changes (McFadden et al 2011; AWWA 2008). As explained by Triantafyllidou et al 2007 (and literature cited therein), lead particulate re-mobilisation under high pressure flow may occur when the lead scale on the inside of the pipes is degraded during intermittent fluctuations in water chemistry and flow.

Data reported by Boyd et al. (2004) for simulations of lead pipe replacements in laboratory studies indicated that at continuous low flow conditions, lead levels equilibrated at <10 µg/L after about a week of flushing following simulated lead pipe replacement, and after about 48 hours at a flow of 0.3 gpm. Under intermittent conditions (including stagnation periods) there was no improvement within the first two weeks at either flow rate. These results suggest that even low flushing rates can accelerate the return to stable conditions after partial lead pipe replacement, and that higher flow rates may accelerate the remediation even further.

The evidence provided in Annex III.3.2 supports the contention that particulate lead levels can be effectively abated with both low and high flow flushing in systems with partial-LPR (e.g. Boyd et al 2004; AWWA 2008). However, two caveats should be noted. Firstly, if premise plumbing components are responsible for elevated lead levels in a particular household, then flushing does not specifically target the source of lead causing a variation in the remedial effectiveness of flushing as shown in many studies (AWWA 2008; Clark et al 2014; Brown and Cornwell 2015). Secondly, flushing has a temporary remedial effect on lead levels in drinking water (Murphy 1993, Triantafyllidou et al. 2009, Cantor 2010). In addition to research evidence, this problem has been stressed in reviews of water lead regulations (EUREAU 1994 cited in Potter 1997; Potter 1997) and by water companies (e.g. Wessex Water n.d. advises for flushing after 30 minutes of stagnation). The duration of the remedial effect of flushing varies from one day to three months (Murphy 1993 cited in Brown et al 2015, AWWA 2008; Triantafyllidou et al. 2009, Cantor 2010; Brown and Cornwell 2015). It also varies with duration of flushing and intensity of flow (e.g. Boyd et al 2004; Clark et al 2014).

AWWA (2008) cited information from a survey among state school participants indicating that flushing after partial lead pipe replacement did improve lead levels after the service pipe was disturbed by the replacement activities, though in some cases a short duration of flushing may not have been enough. For example, 15 minutes or less of flushing was

shown to have some positive impact in some cases, but not in others.

It must be also noted that the term flushing also refers to flushing for traditional lead profiling in the system. In this context, utilities collect sequential litres of water that flow from a consumer tap after a stagnation event to understand sources of lead and the factors influencing lead release remaining in the system after Total- or Partial-LPR.

III.3.4 Corrosion inhibitors

(i) Orthophosphate

Prevalence: Orthophosphate dosing is a common practice in the UK, and the USA (AWWA 2008; Hayes 2010; Hayes et al 2016; US EPA 2016) but limited elsewhere (Hayes 2010). Around 95% of the UK's public water supplies are currently being dosed with orthophosphate (Hayes and Skubala 2009). A major benefit of optimised orthophosphate dosing is the reduction of lead levels in distribution systems with partial pipe replacement below the specified limit value (AWWA 2008; Hayes et al 2006; 2008; 2009; UKWIR 2012). An additional benefit of orthophosphate dosing is that it can also reduce copper levels below the specified limit value (Edwards et al 2002; Comber et al 2011) but this is not further examined here.

Mechanism of action – causes of shortcomings: Orthophosphate dosing promotes the formation of a low solubility lead-phosphate passivating film by converting part of the lead carbonate layer to lead phosphate, which is less soluble (Schock et al 1996; Hayes 2010). When orthophosphate is used, lead pipe scales are often dominated by crystalline lead(II) orthophosphate compounds such as hydroxypyromorphite, Pb9(PO4)6, or Pb3(PO4)2 (DeSantis and Schock 2014). Orthophosphate also exerts a stabilising influence on the deposited layer (AWWA 2008). These benefits can be achieved with a phosphorus concentration in the range 0.6–2 mg/L[15]; within which increasing the orthophosphate concentration tends to reduce the solubility of lead (Cantor et al 2002; AWWA 2008). Below 0.6 or above 2 mg /L, additional benefits are minimal (AWWA 2008).A higher orthophosphate dose (e.g. 3 mg/l of phosphorus) has been suggested when organic matter content is high (Hayes 2010).

Schock (1989) illustrated that the orthophosphate dose needed to achieve a particular lead solubility value increases as DIC increases. there are a number of possible combinations of pH, DIC, and orthophosphate that can produce lead solubility below 10 µg/L, but higher doses of orthophosphate under the same pH and DIC conditions produces even lower lead solubility. For example, at pH 7.5 and an orthophosphate dose <0.2 mg/L as phosphate (~<0.06 mg/L as phosphorus), the lead solubility is 100 µg/l when the alkalinity is ~30 mg/L as CaCO3 (DIC ~4 mg/L as C); increasing orthophosphate dose to about 3.3 mg/L as phosphate (~1 mg/L as phosphorus) decreases the lead solubility by about a factor of approximately 14 at 7 µg/l (Schock 1989).

It has been shown that within the optimal pH range and regardless of type of disinfection, dissolved lead concentrations (i.e. optimised dosing) can be largely maintained below standard values (Edwards and McNeill 2002; Boyd et al 2010). This benefit could be lost by raising or lowering the pH outwith the optimal range (e.g. Awwa 1990) and allowing a 3-day stagnation period with very high or very low alkalinity (Edwards and McNeil 2002). It has been also shown that chloramine (III.3.3-Disinfectant), temperature (III.3.1) and galvanic corrosion (III.3.3) may increase lead leaching in phosphate dosed systems. In some cases results achieved with orthophosphate are comparable with pH correction alone (Brown et al 2015). Orthophosphate was found to increase lead corrosion in chlorinated water with continuous flow, as orthophosphate hindered potential reversal of galvanic lead corrosion presumably due to inhibition of Pb(IV) formation, but reduced lead release with chloramine (Arnolds and Edwards 2012).

In experiments with a simulated, phosphate-dosed premise plumbing system (copper pipes, stainless steel taps and "lead free" brass fittings), Ng and Lin (2016) found that lead-free" brass fittings were identified as the source of lead contamination. Orthophosphate was able to suppress total lead levels below 10 µg/l but caused "blue water" problems. Physical disturbances, such as renovation works, could cause short-term spikes in lead release in the presence of orthophosphate (Ng and Lin 2016).

Surveys by water companies in England in treatment plants and on-site showed that it may take up to 36 months of dosing to ensure that very old and scaled pipe internal surfaces are adequately protected with a lead phosphate film but a reduction in lead at customers' tap can be seen within the first six months (AWWA 2008). In the past, 90 per cent of the water companies in the UK surveyed by the Water Research Foundation (WRF) felt that there were benefits to be gained by ceasing orthophosphate dosing for lead control because a rise in the phosphate level of the distributed water "can result in problems with increased algal growth in stored water and an increase in mould growths, particularly in consumers' bathrooms" (WRC 1992 cited in Potter 1997). A similar concern has also been reported on behalf of water utilities in the USA in a review paper by McNeill and Edwards (2002) on phosphate inhibitor use in USA utilities.

[15] An orthophosphate concentration of 3 mg/L as PO4 is roughly equivalent to 1 mg/L as P.

Isotope studies showed that the isotopic signature of the phosphate dosed at the treatment plant exits the water distribution system (via leakages or the tap) with a slightly modified signature, presumably due to microbial uptake of phosphorus or, less likely due to phosphorus release from iron pipe corrosion (Goody et al 2015). As argued by Goody et al (2015) distribution networks are complex biogeochemical reactors that accumulate a large reservoir of phosphate with the potential to subsequently exchange with phosphate in drinking water.

(ii) Other phosphate-based corrosion inhibitors: polyphosphates and zinc-orthophosphate

Polyphosphates are primarily applied to reduce red water discolouration by iron precipitates (AWWA 2008). However, they have the potential to inhibit lead leaching under certain circumstances, e.g. when polyphosphates revert to orthophosphate in the distribution system (Schock and Clement, 1998, p. 21; Cantor et al 2000). Polyphosphate has been found to increase lead leaching in many instances (e.g. Holm and Schock, 1991; Cook 1992; Dodrill and Edwards 1995; Cantor et al 2000; Edwards and McNeil 2002).

In the USA polyphosphate is used in combination with orthophosphate dosing, known as "blended phosphates", to control lead and copper corrosion and bind up iron and manganese to maintain their solubility in water, thereby minimising the risk of discolouration, staining and scaling (Cantor et al 2000; AWWA 2008; Hill and Cantor 2011 cited in Masten et al 2016). Typically, in a mix of blended phosphates, the orthophosphate fraction would range from 0.05 to 0.7 (US EPA 2016). Using polyphosphate or blended ortho-polyphosphates may reduce iron and manganese oxidation under high pH, but may also cause increases in the lead concentrations in tap water (Schock 1999; Cantor et al 2000; Edwards and McNeill, 2002). In addition, scales in systems with blended phosphates do not follow the same trends as orthophosphate and seem to be influenced by calcium concentrations and phosphorus speciation (DeSantis and Schock 2014).

Zinc-orthophosphate has also been found to reduce lead leaching from brass faucets (Lee et al. 1989). Zinc orthophosphate inhibitors are used in the USA and typically have zinc: phosphate weight ratios between 1:1 and 1:10 (US EPA 2016). However, both British (e.g. Colling et al 1987) and US (e.g. AWWA 1990) studies found its use being linked with enhanced turbidity, through zinc acetate precipitation, and a generally lower effectiveness than orthophosphate to inhibit lead corrosion and form lead phosphate film. Recent research found that zinc orthophosphate did not provide additional lead and copper control compared to orthophosphate (Schneider et al 2007); however, the zinc did provide better corrosion protection for cement at low alkalinity/hardness/pH conditions.

(iii) Silicates

Other lead corrosion inhibitors are silicate-based mixtures of soda ash and silicon dioxide and have been used in a few cases in the USA (US EPA 2016). Silicate-based inhibitors have been shown to successfully reduce lead and copper levels in first draw/ first litre tap samples (Schock, Lytle, et al 2005). Nevertheless, in the USA their full-scale use has been limited due to cost and greater dose requirements than orthophosphate; also, their effectiveness in inhibiting lead corrosion remain uncertain and unclear (US EPA 2016). Silicates form a layer on the surface of the pipe; this may act as a barrier that prevents lead dissolution, on the condition that a pre-existing film allows for the binding of the silicate layer (LaRosa-Thompson et al 1997 cited in US EPA 2016). Silicates can also be used to sequester iron and manganese, depending on their concentration in the raw water, and reduce black or red water problems caused by raising pH to reduce lead corrosion (Schock et al 1996; Kvech and Edwards 2001).

ANNEX IV LEAD BIOMARKERS - THE WATER LEAD –BLOOD LEAD RELATIONSHIP

IV.1. Lead biomarkers

Quantifying lead exposure requires the measurement of lead concentration in blood and other tissues, where lead is transported to be retained or excreted. Lead biomarkers have been recently listed and extensively reviewed (Barbosa et al 2005; Bergdhal and Skerfvig 2008; Liu 2013). In brief:

- Blood lead reflects lead's affinity to haemoglobin and foetal haemoglobin. Concentrations indicate a combination of exposures that took place (i) during the last three to five weeks after intake and absorption and (ii) several years or decades ago, due to endogenous lead release from bone. Venus or capillary blood has been the primary biological fluid used for assessment of lead exposure, both for screening and diagnostic purposes, and for biomonitoring purposes in the long term during the past 50 years. As of today, measurements can reach a precision of 5% for detection limits in the order of 1µg/dl; intercallibration among laboratories is widespread worldwide, including the UK (e.g. UK National External Quality Assessment Service). It is considered as a less costly biomarker compared with other biomarkers. Recent

studies (such as those reported in Annex IV) have used plasma-mass spectroscopy.

- Plasma / serum lead makes up less than 1% of the total blood lead. Plasma lead analysis requires sensitive analytical equipment, especially inductively coupled plasma mass spectrometry (ICP-MS) but due to lack of certified reference materials and cost it has not been used as widely as blood for lead tests. Detection limits of 0.001-0.1 mg/dl have been reported.

- Saliva lead has been proposed as a lead biomarker as it is easily collected, with minimal cost. It is not generally accepted as a reliable biomarker of lead exposure because of: uncontrolled variation in salivary flow rates; lack of standard reference materials; absence of reliable reference values for human populations; and the very low levels of lead present in saliva.

- Hair lead can be easily and non-invasively collected, with minimal cost, and it is easily stored and transported to the laboratory for analysis. However, there is no reliable method of distinguishing between lead that has been absorbed into the blood and incorporated into the hair matrix and lead that has been derived from external contamination.

- Urinary lead reflects lead that has diffused from plasma and is excreted through the kidneys. Collection is not invasive and thus it is suitable for long-term biomonitoring, especially for occupational exposures; monitoring patients during chelation-therapy; and, until very recently, clinical evaluation of potential candidates for chelation therapy. It is subject to large biological variation and has been found to correlate more strongly with plasma than blood lead. Urine lead levels are generally lower than blood lead levels; a detection limit of 1 µg/dl is desirable.

- Faecal lead reflects unabsorbed, ingested lead and lead that is eliminated via endogenous faecal routes. It gives an integrated measure of lead exposure as intake from all sources, dietary and environmental, inside and outside the home. It can be used for estimating the overall amount of childhood lead intake but it is generally considered more useful in clinical cases where a very recent large oral intake of lead is suspected. However, the collection of complete faecal samples over multiple days may not be feasible.

- Nail lead is a reflection of long-term exposure, especially in the case of toenails which have a 50% slower growth rate than fingernails, because nails remain isolated from other metabolic activities in the body and thus may provide a longer integration of lead exposure. Nails offer only limited scope in assessing exposure to lead because of large variations in lead levels, even after rigorous washing procedures.

- Bone lead is a reliable indicator of body burden through cumulative lead exposure, which may not be adequately discerned through measurement of blood lead levels alone, and endogenous sources of lead exposure, when bone lead is mobilised into the blood circulation. Differing bone types have differing bone lead mobilization characteristics, with lead in trabecular (spongy) bone being more easily mobilised than lead in cortical (compact) bone; therefore measurements of the trabecular bone reflect a shorter time-span of exposure than the cortical ones. Isotope ratios of bone lead accumulated from past exposures and current sources can help evaluate exposure pathways. Bone lead measurements are based on non-invasive *in vivo* X-ray fluorescence (XRF) methods, which may require planning in advance and a greater budget. There are no problems with detection limits but the sensitivity of this biomarker has been found to be insufficient for individuals exposed to residential, low lead levels.

- Tooth lead reflects cumulative exposure from the prenatal period and especially of exposure during pre- and neo-natal period, when the tooth is formed, until the time of shredding. Teeth have a much slower lead release from teeth than bone; are easier to collect and analyse than other biomarkers; and are suitable for long-term preservation. Teeth are unreliable in indicating exposure period, i.e. pre-, neo-natal or later in early childhood. Concentrations are usually above detection limits.

- Breast milk has rarely been used for biomonitoring of lead exposure but it is useful in evaluating the risk to an infant's health using a non-invasive (for the infant) method. Milk lead levels vary widely around the world in the range of 0.5-126.6 µg/l. This variability may be related to the time of sampling; the time of lactation; the method of sampling; maternal lead burden; type of maternal exposure; maternal habits, such as smoking; and method of analysis.

Plasma/serum lead has also been found to correlate with bone and blood lead levels (e.g. Chuang et al 2001); however it remains uncertain whether this correlation reflects a preferential binding of endogenous lead on plasma/serum lead or an inter-correlation between all involved lead biomarkers. Saliva lead levels have been found to correlate with plasma, especially the fraction not bound to proteins, or blood lead levels in a limited number of studies (Omokhodion and Crockford 1991). Hair lead has been found to be generally associated with blood lead concentrations (Barbosa et al 2005); however, variation in hair lead may be as high as ±100%, rendering any correlations with other biomarkers unreliable. Urinary lead has been found to be clearly associated with blood with a curve-shaped (curvilinear) relationship (Bergdahl et al 1997; Gulson et al 1998); and with plasma lead with a

linear relationship (Bergdahl et al 1997). Faecal lead may be the only biomarker reflecting short-term (i.e. within hours) gastrointestinal exposure; therefore it is not correlated with any of the other biomarkers, which reflect longer-term or cumulative exposure (Bergdahl and Skerfving 2008). Likewise, there is no good correlation between teeth and blood lead, probably because of the different timescales of exposure, e.g. prenatal vs infancy or early childhood (Grandjean et al 1989).

Blood has also been suggested as the most reliable indicator for current lead exposure and as an indicator of the average lifetime exposure because it reflects the balance between current blood lead levels in the blood stream and the lead released to the blood stream via endogenous exposure (e.g. from bones) (Barbosa et al 2005; Bergdhal and Skerfvig 2008; Liu 2013). Further credence to blood as a reliable biomarker of lead exposure has been lent through its extensive use in:

- State funded blood lead screening programmes in the USA (Mahaffey et al 1982; CDC 2012, Brown and Margolis 2012; Reyes et al 2015) and in Europe (e.g. Directive EEC 77/31; WHO-ENHIS 2009; Etschever et al 2015)
- Research investigating changes in blood lead levels in response to lead mitigation policies such as the ban of lead in petrol and paint and the regulation of lead in drinking water (e.g. Thomas et al 1999; Meyer et al 2008; WHO-ENHIS 2009; Brown and Margolis 2012; Oulhote et al 2013; Ngueta et al 2014; Hanna-Attisha 2016; Reuben et al 2017)
- Research investigating the adverse health effects of lead exposure (Annex I.3)
- Research on the relationship between water lead and blood lead (Section 7.3 and Annex IV.2)
- Studies exploring the link between geospatial data on water lead levels in different supply zones of the public network and blood lead levels (e.g. Miranda et al 2007; Hanna-Attisha et al 2016).

It has to be noted that one of the most important and promising biomarker methodologies is the use of isotope fingerprinting. The four natural stable lead isotopes are ^{204}Pb, ^{206}Pb, ^{207}Pb, 208Pb (Rabinowitz 1995). The relative abundances of these isotopes vary between different geologic sources of lead in petrol, gasoline, pipes and solder. Because the isotope ratios (IR) remain constant, it is possible to measure differences in lead IRs from different sources and compare them with the isotope ratios in the body. This information can be used to evaluate potential sources of exposure in human populations. If the isotope composition of lead in various sources of lead exposure is different, the difference will be reflected in the isotope composition of the blood lead, teeth or bone IR, therefore providing an accurate method to link lead found in the body to a potential source (Naeher et al 2003). This method has been also used to identify sources of lead exposure in Scotland (Moffat 1989; Ramsay 2003; Delves and Campbell 1992); see also Annex II.

IV.2 Studies showing a relationship between blood lead and water lead

Scotland

1. Moore et al. (1977) conducted one of the first surveys to show a strong, curvilinear (cube root) relationship between water lead and blood lead "in different sectors of the Scottish population" (Blood lead=11 .0 +2.36 $\sqrt[3]{}$water lead; R=0.52). They also found that 18% of people drinking first-flush water lead equal to 298 µg/L had blood lead equal to 41 µg/dl. The ramifications of this very high exposure in the general population remain unexplored in Scotland, in terms of current evidence on lead exposure. It also has to be noted that at zero water lead levels lead exposure would be approximately at 11 µg/dl, suggesting either a strong influence of other, non-waterborne, sources of lead at that time in Scotland, and/or, more likely, a wide range of water lead levels.

2. Moore et al (1981) showed that lime dosing in Glasgow city water supplied by Loch Katrine was indirectly associated with lower blood lead levels in 1980 after liming, which resulted in 80% of water samples containing less than 100µg/l, compared with 1977, when more than 50% of random daytime water samples exceeded 100 µg/l. Blood lead samples were obtained from 236 mothers in 1977 and 475 mothers in 1980. Moore et al (1981) observed that 47.5% of the mothers examined in 1980 had blood lead levels in the range of 5.4-9.3 µg/dl compared with 22.3% of the mothers examined in 1977 for the same range of blood lead levels. 22.9% of the mothers in the 1977 survey had blood lead levels above 21.9 µg/dl compared with only 1.4% in the 1980 survey. It remained unexplored whether this decline was also associated with the phasing down of lead in petrol at that time.

3. Quinn and Sherlock 1990 fitted a cube root relationship to link blood lead in 85 wholly bottle-fed 3-month old infants and water (composite kettle) lead levels under the Glasgow duplicate diet study in 1979-1980:

   $\text{Blood lead}_{\text{bottle-fed infants}}$ (µg/dl)=5.5 (±2.6) + 3.3 (0.5) $\sqrt[3]{}$(water lead) (µg/l), R^2=0.23, p<0.01.

   This curvilinear relationship implies that at an average water lead concentration of 10 µg/l, a 5% (95th percentile) of 3-month old bottle fed infants would have

blood lead concentrations above 20 µg/dl; also, a 1% (99th percentile) of bottle fed infants would have blood lead concentrations above 20 µg/dl.

4. Sherlock et al (1984) found a significant blood lead-water lead relationship when they combined data from the Blood Lead Surveys in 1979-1981 in Ayr (i.e. before lead corrosion control) and follow-up studies in 1982-1983 after measures to reduce plumbosolvency (increasing the pH from 5.0 to 8.5, and replacing some lead pipes). After increasing the pH of the water supply, water lead levels significantly dropped, and median blood lead levels also dropped from 21 µg/dL to 13 µg/dL. A cube root equation could be fitted using the same 74 mothers before and after treatment, whereby the point of non-contribution of lead in water (lead-free water) to blood lead was found to be at 5.6 µg/l (Sherlock et al 1984; Moore et al 1985):

   $\text{Blood lead}_{\text{mothers}}$ (µg/dl)=5.6 (±0.73) + 2.62 (±0.13) $\sqrt[3]{}$(water lead) (µg/l), R^2=0.65, p<0.001.

5. Laxen et al (1987) (Edinburgh Lead study-1983) reported that in the central area of Edinburgh water lead concentrations ranged between 0 and 456 µg/l, with a mean at 37 µg/l and a geometric mean of 17 µg/l. It was also reported that in a population of 495 primary 3 and 4 children, 21% were served by cold water samples that contained more than 50 µg/l of lead (i.e. the standard under the 1980 EEC Drinking water Directive). Blood lead levels ranged between 3.3 and 33.6 µg/dl, with an arithmetic mean at 10.7 µg/dl and a geometric mean of 10.1 µg/dl. The following fitted regression explained 32% of the variance on blood lead data and showed that water lead was a significant determinant of lead exposure in areas with high water lead levels:

   $\text{Blood lead}_{\text{children}}$-µg/dl= (53.26 +1.03 (water lead-µg/l) + 0.0381 (dust lead-µg/g))$^{0.5}$

6. Watt et al. (1996, 2000) assessed the relationship between tap water lead and maternal blood lead concentrations in Glasgow, after the water supply was subjected to maximal (liming and orthophosphate dosing) water treatment to reduce plumbosolvency. Tap water lead remained the main correlate of raised maternal blood lead concentrations, accounting for 76% of cases of maternal blood lead concentrations above 10 µg/dL. The mean maternal blood lead concentration was 3.7 mg/l in the population at large, compared with 3.3 mg/litre in households with negligible or absent tap water lead (Watt et al 2000). The authors concluded that although tap water lead and maternal blood lead concentrations had fallen substantially since the early 1980s, tap water lead was still a public health problem in that area, especially for the estimated 13% of infants who were exposed via bottle feeds to tap water lead concentrations exceeding the WHO guideline of 10 µg/l.

7. Ramsay (2003) compared the isotopic signals of water lead in premises with illegally used lead-bearing plumbing components and blood lead in the 45 residents of those premises. In households that contained water with lead above 5 µg/l due to lead solder and had pH in random daytime samples generally above 8.5 (i.e. water was not plumbosolvent), blood lead levels ranged below 20.7 µg/dl. The study showed a significant and positive correlation between water and blood lead levels and the isotopic ratios of illegally used lead plumbing and blood lead.

France

1. Huy et al (1986) compared the blood lead levels in adults from three different areas in France to meet the requirements of the Directive EEC 77/31 for Blood Lead Surveys in the population of the then Member States of the European Economic Community: in eight metropolitan areas (having more than 500,000 inhabitants); another eight "critical" areas surrounding industrial emission sources; and one area (Vosgian Mountains) having high water lead levels. Individuals exposed to higher water lead levels ran seven times greater risk of exceeding the 35 µg/dl (9.3% of population) blood-lead limit relative to urban populations (1.4 % of the population) where the lead comes mostly from vehicle emissions into the atmosphere. Huy et al (1986) concluded that although vehicle exhausts were the subject of much current concern in the early eighties, the problem of lead water pipes appeared to be of far greater importance.

2. A parallel study at the Vosgian Mountains by Bonnefoy et al (1985) showed that for water lead levels up to 20 µg/l, blood lead levels of both men and women remained relatively constant, but if lead in water exceeded 20 µg/l, blood lead levels increased sharply above the population average of 15 µg/dl for men and 11 µg/dl for women.

3. Leroyer et al (2000) showed that blood lead levels doubled for children in Northern France, who reported consuming tap water in homes with lead plumbing, compared with children consuming bottled water. They suggested that visual identification of lead plumbing and the use of questionnaires should always be accompanied by water lead sampling.

4. Tagne-Fotso et al (2016) studied blood lead levels in 2000 inhabitants of northern France in a current and past industrial area, aged between 20 and 59 years, using the quota method with caution. Variation factors were studied separately in men and women using multivariate stepwise linear and logistic regression models. The geometric mean of the blood lead levels was 1.88 µg/dl (95% confidence

interval [CI]: 1.83–1.93). Occupational factors affected blood lead only in men and represented 14% of total explained variance in blood lead. Other significant factors were: tobacco; tap water; other drinks (e.g. wine, coffee, tea); raw vegetables; housing characteristics (built prior to 1948, lead piping in the home); and do-it-yourself or leisure activities (paint stripping or rifle shooting). The authors concluded that although the blood lead levels were similar to the international range in countries with strict lead control, these blood lead levels remain a public health issue in regard to non-threshold toxicity attributed to lead.

5. Etchevers et al (2015) and Oulhote et al (2016) studied 484 French children aged from 6 months to 6 years, and collected data on social, housing and individual characteristics, and lead concentrations in blood and environmental samples (water, soils, and dusts). Using a multivariate generalized additive model to account for the sampling design and the sampling weights, Oulhote et al (2016) found that very low concentrations of lead in dust, soil and water were significant predictors of children's blood lead levels, after adjustment for potential confounding variables. Both studies found that household dust and tap water were the major contributors and blood lead levels (geometric mean: 1.4 µg/l) increased by 5% when lead content in water increased from the 25th to the 95th percentile. Etchevers et al (2015) demonstrated that lead concentrations in tap water above 5 µg/l were also positively correlated with the geometric mean, 75th and 90th percentiles of the blood lead levels in children drinking tap water. Oulhote et al (2016) also observed that (i) the steepest increase in blood lead levels occurred at the lowest levels of lead-contaminated floor dust, which indicated that lead contamination should be kept as low as possible from all sources; and (ii) unless standards for e.g. water and blood lead levels are set at very low levels to prevent the neurocognitive effects associated with blood lead levels in the range of 2-10 µg/l, they will only benefit a small proportion of children who have the highest exposures.

England & Wales

6. Elwood et al (1984) assessed the relative contributions of water lead, dust lead and air lead to blood lead of 192 women in various areas of Wales. The regression model, i.e.

   Log(blood lead)=1.06 + 0.62 $\sqrt[3]{}$(water lead) + 0. 18 Log(air lead) -0 .0 2Log (dust lead))

   indicated that even in areas with relatively low water lead levels for that time period, water was an important source of blood lead, with an increase of water lead from 0 to 60µg/l associated with an increase of 5.5 µg/dl in blood lead (Elwood et al 1984).

7. Pocock et al. (1983) examined the link between water lead levels and blood lead levels in over 900 men in England. The researchers grouped the men into nine groups of first-draw water samples after stagnation. The lowest group contained the men water with lead at 60 µg/l while the other eight groups were split out based on increasingly higher water lead levels with 50 men in each group. The data showed a positive correlation between water lead and blood lead that allowed for developing a significant linear regression model: blood lead (µg/dl) = 0.699 + 0.0003 * water lead (µg/dl). Pocock et al. (1983) also demonstrated significant inverse correlation between water hardness (defined by amount of dissolved calcium and magnesium in water) and water lead levels, thus again highlighting the complexity of the link between drinking water chemistry and water lead at the tap.

Canada

8. A Canadian study conducted at Ste-Agathe-des Monts demonstrated a link between blood lead and water lead as well as the presence of lead service pipes (Savard 1992). On the basis of field investigations and 383 blood lead analyses, blood lead levels above 20 µg/dL were associated with the presence of lead service pipes (Savard 1992). A mathematical model was developed for the 72 citizens in whose household water lead levels were measured (i.e. blood lead= 10+ 7 x water lead x water consumption, R^2=0.25). Lead concentrations in those samples were as high as 4200 µg/l. Water consumption was obtained on the basis of a questionnaire. Plumbosolvency was rapidly identified as the problem (pH as low as 4.8 measured in some houses) and corrective measures were taken by increasing the pH to 8.4. After less than a month, water lead levels were reduced by more than 90%, and the measured blood lead levels were significantly reduced by 24% in less than a year.

9. Ngueta et al (2014) estimated the magnitude of winter-to-summer changes in household water lead levels to predict the impact of these variations on blood lead levels in young children in Montreal in a cross-sectional study (2009-2010); a follow-up (2011) helped characterize exposure to lead in water, dust and paint. Using fully flushed and 30MS sampling protocols and non-linear regression and general linear mixed models they modelled seasonal effects on water lead levels and by means of the Integrated Exposure Uptake Biokinetic (IEUBK) model they predicted the impact of changes in water lead on children's blood lead levels. Marked increases in water lead levels were observed from winter to summer (by 6 µg/l in flushed samples and 10.5 µg/l in 30MS samples in homes with lead service pipes but only by 0.3 µg/l in lead-free homes). The change in the probability of blood lead levels equal or above 5µg/dL due to winter-to-

summer changes in water lead was increased from below 5% (in winter) to about 20% (in summer) in children aged 6-months to 2-years. The likelihood of having blood lead levels equal or above 5µg/dL in young children during warm months was reduced by at least 40% by flushing tap-water. Seasonal changes in water lead may translate into an increase of 1 µg/dL in blood lead.

Germany

10. Englert et al (1994) measured tap water lead and blood lead in school children from two locations in Southern Saxonia, Germany, in an area where lead pipes were used in about 50% of their houses. Water lead levels explained up to 43% of the variation in blood lead levels in logarithmically transformed data, suggesting that drinking water was a greater concern than lead paint and other sources.

11. Fertmann et al (2004) found significant correlation between blood lead and average lead in tap water, with individuals from households in Germany with drinking water lead levels above 5 µg/l having significantly higher blood lead levels (median blood lead: 31 µg/dl) than those that had no detectable lead levels in their drinking water (median blood lead: 24 µg/dl). Minimising exposure to lead in drinking water by flushing tap water or excluding exposure to lead in tap water by using bottled water, resulted in lower blood lead levels by 11 µg/dl (on average). Exclusion resulted in lowering blood lead levels by 37%. The authors concluded that *"lead in tap water stands for an avoidable surplus exposure."*

USA

12. Lanphear et al. (2002) compared the contribution of lead in water and other sources (i.e. house dust, soil and paint) to children's blood lead levels during early childhood. Children from 6 until 24 months of age were monitored in Rochester, New York, an area where drinking water always met the lead standard. House dust was determined as the main source of lead exposure; however, water lead concentrations were also directly associated with blood lead levels ($p<0.001$). Children who lived in housing with water lead concentration greater than 5 µg/L had a (geometric) mean blood lead concentrations of 8.4 µg/dl, which was by 1.0 µg/dL greater than the concentration in children who had home water lead levels below 5 µg/L

13. Miranda et al (2007) tested the potential effect of switching to chloramines for disinfection in water treatment systems (dosed with Zinc-orthophosphate and not) on childhood blood lead levels using data from Wayne County, located in the central Coastal Plain of North Carolina. They constructed a unified geographic information system (GIS) to link blood lead screening data with age of housing, drinking water source, and census data for 7,270 records. The data were analysed using both exploratory methods and more formal multivariate regression techniques. The analyses indicated that the change from chlorine to chloramine disinfection was associated with significantly higher blood lead levels (from 4.19 to 4.9 µg/dl), the impact of which was progressively mitigated in newer housing.

14. Brown et al (2012) evaluated the effect of changes in the water disinfection process, and the presence of lead service pipes on children's blood lead levels in children younger than 6 years of age in Washington, DC. The final study population consisted of 63,854 children with validated addresses. They found that this association was strongest during 2003 when chloramine alone was used for water disinfection. The association persisted after controlling for the age of housing. Partial lead pipe replacement did not result in a decrease in the association between lead service pipes and elevated blood lead levels. For children tested after lead pipes in their houses were replaced, those with partially replaced lead pipes were >3 times as likely to have blood lead levels ≥10 µg/dL versus children who had never been exposed to lead pipes. Also children's blood lead levels declined when chloramine+orthophosphate treatment was applied compared with chloramine alone but were higher than when water was treated with chlorine alone. The authors who are affiliated with the prestigious Centers for Disease Control and Prevention (CDC) concluded that the most effective strategy to reduce blood lead levels remains controlling or eliminating sources of lead in children's environments before they are exposed. They added that the "consequences of changes in water disinfection practices on a range of health issues including exposure to lead should be carefully considered by water utilities before they are adopted."

15. Edwards et al (2009) found that the correlation between lead levels in drinking water and blood lead levels above 10 µg/dl in children in Washington, DC was stronger when only children under 1.3 years of age were included in the study than when children up to 2.5 years of age were included, presumably because of higher intake levels per body weight in younger children. Their study showed that the increase in the 90th percentile water lead levels after the switch to chloramine disinfectant above the limit of 15 µg/l was associated with increases in the percentage of younger children with blood lead levels above 10 µg/dl by four times.

1. Hanna-Attsiha et al (2016) reviewed blood lead levels for children younger than 5 years before (2013) and after (2015) water source change in Greater Flint, Michigan, assessed the percentage of elevated blood lead levels in

both time periods, and identified geo-graphical locations through spatial analysis. They found that the prevalence of elevated blood lead levels in the population increased from 2.4% to 4.9% ($P < .05$) after water source change, and neighbourhoods with the highest water lead levels experienced a 6.6% increase but no significant change was seen outside the city. Geospatial analysis identfied disadvantaged neighbourhoods as having the greatest elevated blood lead level increases and informed response prioritization during the declared public health emergency.

IV.2 Studies showing lack of a relationship between blood and water lead or lead pipes

1. Goldberg (1974) studied blood lead levels in the residents of households with different amount of lead in premise plumbing. He found that water lead levels in households with lead plumbing could be up to 1800 µg/l. Blood lead levels in children and adults ranged between 30-40 µg/dl. No lead toxicity effects were concluded for the individuals studied. No statistical analysis or comparison with a control population group was carried out. This study is very old and obviously very misleading in view of the current evidence. It is just reported here to show the scale of divergence in scientific perspectives between today and then.

2. Rabinowitz et al (1985) examined the association of blood lead levels in 24-month old children in Boston with lead in dust, soil, indoor air, paint and tap water. Statistically significant correlations were found with lead in dust (Spearman's $\rho = 0.4$, $p<0.0001$); lead in soil (Spearman's $\rho = 0.3$, $p < 0.001$); and lead in paint (Spearman's $\rho = 0.2$, $p < 0.01$), but not with lead in water (Spearman's $\rho = 0.14$, $p>0.05$). It is possible that analytical limitations in the quantification of water lead may have confounded the blood-water lead relationship, such as in the case of anodic stripping voltammetry which, many years later, was found to be inaccurate in the measurement of particulate lead (Triantafyllidou et al 2007).

3. Lubin et al (1984 cited in Triantafyllidou 2011) collected water samples in the homes of 50 children in Columbus, Ohio with blood lead levels above 30 µg/dl. They found that lead in water was always below 10 µg/l. The tap water in that study had high pH of 9.6 and high hardness of 101 mg/L. As a result of this, there was no correlation between lead in water and lead in blood, even in the presence of lead pipes at the children's homes.

4. Meyer et al (1998) found no significant association between tap water lead and blood lead in children in a German town, where lead in tap water was extremely low (< 1µg/l).

5. Gasana et al (2006) found no significant relationship between blood lead in children and water lead in Miami Inner City, Florida, where water lead measured in 120 homes was reportedly below 15 µg/l (i.e. below the standard for lead in drinking water in the USA).

6. Costa et al (1997) reported that very high water lead levels in a state school in rural Utah (up to 840 µg/L) were not associated with increases in blood lead levels compared with children exposed to lower water lead levels. One case of elevated blood lead was dismissed as unrelated to water lead. In that study, measurements of blood lead were undertaken for only 40% of students, more than 16 days after notification of the problem and advice to drink bottled water. It is possible that during that time lead blood lead levels dropped, as expected from blood lead half-life.

7. Stokes et al (2004 cited in Triantafyllidou 2011) reported that in 201 households where tap water lead exceeded the value of 300 µg/l of lead in Washington DC, none of the residents were found to have blood lead levels above 10 µg/dl. Another study on the same topic cited the same data, and did not find an association between the high water lead levels and blood lead, concluding that "there appears to have been no identifiable public health impact from the elevation of lead in drinking water in Washington DC, in 2003 and 2004" (Guidotti et al 2007). Both studies (Stokes et al 2004 cited in Triantafyllidou 2011; Guidotti et al 2007) pooled all the blood lead data for Washington, DC together, an approach considered by Edwards et al (2009) to have masked differences between neighbourhoods. Neither study focused on infants, who are most vulnerable to harm from lead in water. In addition, virtually no residents had been consuming tap water for months prior to having their blood lead drawn, rendering the data useless for assessing impacts of lead in water on lead in blood (Edwards et al 2009). The "no-harm" conclusion of Guidotti et al (2007) has since been removed (Errata in Environmental Health Perspectives 2009).

8. Bornschein et al 1985 and Clark et al (1985) investigated sources of lead intake in children including painted surfaces and dust, soil samples in outside playing area, street dirt, and any suspicious items which the children could potentially ingest in urban settings. Blood lead levels were systematically monitored from birth through 5 years of age. Water samples were not collected in this otherwise very thorough and definitive study but water lead data collected by the water utility from the distribution system (before reaching premise lead plumbing) were used; as a result water lead concentrations were lower than 6 µg/L (Clark et al 1985). This led to the conclusion that exposure to lead in drinking water was insignificant.

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