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Lead in drinking water: public health, mitigation and economic perspectives

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Contents

Executive Summary

Lead pipes and plumbing components such as lead-solder and brass fittings can contaminate drinking water on its route from the water mains to the premises and pose a public health risk. Exposure to lead in tap water is entirely preventable but challenging to achieve. To contribute to a better understanding of these challenges, this report reviews evidence on the public health, mitigation and economic perspectives of lead in drinking water in Scotland and internationally.

Key Findings

- The review of the national and international literature on lead in drinking water showed that there is sufficient and robust scientific evidence on (i) the contribution of water lead on individual lead exposure; (ii) the adverse health effects and social outcomes of lead exposure in childhood; (iii) the shortcomings and cost of lead mitigation practices; and (iv) the public health and monetary benefits of lead-free practices.
- The predominant source of lead in drinking water is lead pipes and plumbing.
- No safe level or threshold for lead exposure has been agreed by experts. The World Health Organisation has identified lead as a chemical of major public health concern.
- Low exposure to lead (i.e. blood lead concentrations below 10 μg/dl) in children has been associated with intellectual impairment in childhood, and cognitive deficit, loss of individual potential and low income in adulthood.
- The World Health Organisation has warned that there may be a risk for bottle-fed infants through intake of drinking water with a lead concentration of 10 μg/L.
- In Scotland:
    - o Failures of the water lead standard (i.e. 10 μg/L) are predominantly associated with the presence of lead plumbing components and lead supply pipes, which run within the boundary of a property and are homeowners' responsibility to replace.
    - o Failures of the lead standard also arise in supply zones where communication pipes, which connect properties to the mains in the street and are Scottish Water's responsibility to replace, remain made of lead.
    - o Optimised orthophosphate dosing (i.e. the dose required to achieve compliance with the water lead standard of 10 μg/L) has been shown to effectively reduce lead leaching from lead pipes and brass fittings within premises. However, it is not a lead-free strategy.
- Total lead pipe replacement (i.e. replacement of lead pipes in utility's and homeowners' side) can be a lead-free strategy. Despite the availability of state-funded lead pipe replacement grants, homeowners' cooperation has generally been poor because of the disruption and inconvenience involved and the cost incurred in case of means-tested grants.
- Since the 1970s, lead-free policies (e.g. gradually phasing lead out of petrol) and the tightening of the standard for lead in drinking water (i.e. from 100 μg/L to 10 μg/L) co-occurred. This makes it difficult to separate the benefits of water lead mitigation to the proportion of the population consuming lead-contaminated water from the benefits of phasing lead out of petrol to the general population and the environment.
- The greatest economic benefits to the society of removing all sources of residential lead (including lead pipes and plumbing) arise by avoiding the health and social costs of low lead exposure in the affected proportion of the population. These costs refer to provision of medical treatment and special education; combating lead-linked crime; and loss of life-time earnings and contribution to general productivity due to poorer individual potential.

1.0 Introduction

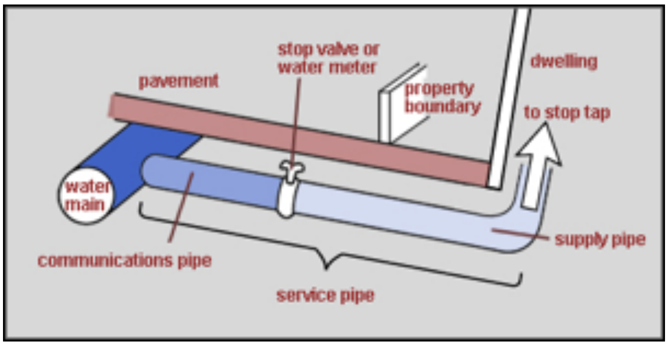
Lead (Pb) is a cumulative neurotoxin when ingested by humans, adversely affecting the mental and physical health of children and causing elevated blood pressure, hypertension, and other cardiovascular conditions in adults (Lidsky and Schneider 2003). Lead may occur naturally in very small quantities but has become a public health issue because of its widespread use (e.g. in heavy industry, gasoline, paint, batteries and plumbing) and associated contamination of a range of environments and consumer products (e.g. food, air, soils, indoor dust and drinking water). Exposure to lead in tap water is entirely preventable; therefore, the challenge for Scotland and internationally is to provide lead-free drinking water. With that in mind, this report reviews national and international evidence on the public health and economic aspects of lead in drinking water.

In Scotland, drinking water is of the highest standard. Public raw water sources are lead-free and the water leaving the public water treatment plants is also lead-free. However, lead in drinking water is a public health concern because of plumbosolvency, i.e. lead leaching from lead-containing pipework and plumbing components such as lead-solder for jointing copper pipes, brass fittings and faucets. In some areas of Scotland, waters are naturally soft and acidic, thus having a great propensity to dissolve lead from the lead water pipes, plumbing and storage tanks through which it may pass (Richards and Moore 1984). In these areas, water from acidic raw sources may be a problem for premises still containing lead pipes and plumbing served by either public or private water supplies in urban or rural areas.

Specific lead-control practices have been in place during the past 50 years in Scotland to reduce lead in drinking water, such as:

- Replacement of lead water mains and communication pipes (Figure 1).
- Orthophosphate dosing into the public water distribution system to reduce lead leaching.
- Provision of information and advice to property owners to replace supply pipes.
- Means-tested, discretionary grants to homeowners for the replacement of their lead pipes, i.e. supply pipes and premise plumbing (Figure 1).

The current standard of 10 µg/L for lead in drinking water has been set by the European Drinking Water Directive-DWD (98/83/EU). The Drinking Water Quality Regulator (DWQR) for Scotland reported that in 2015 this standard was met in 99% of samples from public supply zones managed by Scottish Water (DWQR 2016a) and in up to 92% of private water supplies (DWQR 2016b), which serve approximately 3.5% of the population in Scotland and are their owner's responsibility. Failures were predominantly associated with the presence of lead supply pipes and premise plumbing, which are homeowners' responsibility to replace (Figure 1).



Type of piping	Responsibility
Water mains	Scottish Water
Communication pipe	Scottish Water
Supply pipe	Homeowner
Dwelling plumbing	Homeowner

1.1 Outline of the report

The review starts with a description and evaluation of the literature search strategy (Sections 2 and 3). The report consists of four parts and a stand-alone Annex that contains the full list of articles reviewed. A stand-alone Executive Summary has been also provided.

The report is structured as follows:

- Part I (Section 4 and Annex I) gives an overview of the environmental lead cycle, pathways and types of exposure; factors influencing intake and uptake (absorption) of lead; the adverse health and social outcomes of lead exposure; and the legislation controlling lead emissions and exposure levels in the body. Part I provides the background of this report.
- Part II (Section 5 and Annex II) reviews all available evidence on the potential sources of lead in Scotland and associated lead exposure and health effects in the Scottish population. This part will help to assess the implications of lead levels in drinking water in Scotland.
- Part III reviews evidence on factors influencing lead in drinking water and associated exposure:
    - o Section 6 discusses the advantages and disadvantages of options for the mitigation of lead in drinking water; Annex III details the processes involved in lead leaching.

- o Section 7 evaluates indicators of exposure to lead in drinking water; Annex IV details research on the relationship between water lead and commonly used biomarkers of lead exposure.
- o Section 8 assesses evidence on declines of lead exposure due to lead –free policies (e.g. phasing out of lead petrol) and water-lead regulations.
- o Section 9 compares different regulatory approaches to sampling for lead in drinking water.
- o Section 10 reviews evidence on the cost of different options of mitigation of lead in drinking water in the context of other costs such as cost of illness due to low exposure to lead.

2.0 Literature search strategy

This project reviewed literature related to non-occupational exposure to lead. All available lines of evidence (e.g. medical, regulatory, engineering, environmental and socio-economic) were accounted for. Data from proxy organisms (e.g. rodents, non-human primates) and in vitro studies were not considered. Only evidence published in English language was used. Emphasis was on low (potentially long-term) exposure to lead. The period of interest was the past 50 years and captured publications on public health effects of lead in drinking water until May 2017.

Review of the medical literature focused on the recent, post-2000 findings on the health effects of low lead exposure. The review of lead in drinking water mitigation practices and policies investigated pre- and post-2000 evidence as well. It must be clarified that this report uses the literature-based meaning for **lead exposure, i.e. the actual absorption of lead in the body, which can then be retained and measured as body lead concentration (e.g. in blood, bones, soft tissues), regardless of the source or perceived risk of lead exposure.**

Computerised searches were performed using web-based search engines such as Science Direct (SD), Web of Science (WoS) and Google Scholar (GS). The reason for using three different search engines was to take advantage of the different benefits arising from the use of each one of them. GS enabled the detection of published peer-reviewed and grey literature (e.g. reports from government organisations, water companies or health and regulatory agencies) on the basis of full document searches including results drawn from references; however, the results (in terms of numbers and content) were not 100% reproducible. WoS enabled a detection of peer-reviewed articles tagged for their high scientific impact and close relevance of their title and keywords with the search terms. In addition to the advantages referring to the WoS search engine, SD allowed for reproducibility of search findings from a wider range of peer-reviewed articles, books and conference papers and the targeted search of whole document, i.e. de-emphasising results from references.

The following words-phrases were used as search terms or keywords: Pb, lead, public health, tap water*, drinking OR drink* water*, exposure, Scotland, lead pipe, phosphate OR orthophosphate. Other terms used for further refinement of the results included: plumbosolvent*, blood lead, monitor*, frequenc*, grant*, enforce*, pipe replacement, flush*, cost*, benefit*, social, biomarker*.

Evidence was extracted from peer-reviewed literature and reports from internationally recognised organisations including the World Health Organisation (WHO); the Health Protection Agency (HPA); the Scientific Committee on Health and Environmental Risks (SCHER) of the European Commission; the Centre for Disease Control and Prevention (CDC) in the USA; the Agency for Toxic Substances and Disease Registry (ATSDR) in the USA; Environmental Protection Agencies (EPA); the Water Research Foundation (WRC); the Chartered Institution of Water and Environmental Management (CIWEM); the UK Water Institute of Research (UKWIR); the American Water Works Association (AWWA); the European Food Standards Agency (EFSA); and other organisations. Citations in all relevant articles were also read to develop an understanding on how the relationship between public health, lead in drinking water and lead mitigation are interpreted by the researchers themselves and to identify evidence that was not captured by the search engine.

3.0 Evaluation of the literature search strategy

The combined searches for Pb or lead and any of the terms such as drink* water*, tap water*, or public health delivered a great number of "relevant" articles (Table 1). However, a limited number referred to lead exposure and lead pipe or plumbosolvency in Scotland (Table 1). The following observations were made:

- There was little overlap in the articles captured from the three different search engines.
- The search term "lead" was unhelpful in that it captured articles about leadership; titles containing the verb lead and documents using the verb lead and the word leadership. This explained the great number of articles on Lead+ drink* water* +public health + Scotland with SD and GS. Half of the articles for Scotland were irrelevant to lead in drinking water.

Keywords	SD	WoS	GS
Lead	6,595,270	3,467,577	579,000
Lead + tap water*	153,017	3,188	299,000
Pb	921,944	251,243	582,000
Pb + drink* water*	33,236	3,894	30,000
Pb + tap water*	38,274	1,018	92,900
Pb + public health	55,099	31,817	1,210,000
Pb + drink* water*+ public health	7,421	4,961	16,700
Pb + tap water *+ public health	4,096	845	17,600
Lead + drink* water* +public health + Scotland	2,663	23	19,900
Pb + drink* water* +public health + Scotland	491	12	10,400
Pb + tap water* + public health + Scotland	302	5	3,670
Pb + Scotland	16,193	744	136,000
Pb + Scotland +public health	1,794	182	21,500
Pb + tap water *+ public health+ lead exposure	1,270	111	16,200
Pb + tap water *+ public health+ lead exposure + lead pipe	297	29	14,100
Pb + tap water *+ public health+ lead exposure +orthophosphate	65	4	16,300
Pb + tap water *+ public health+ lead exposure +plumbosolvency	16	2	263
Pb + tap water*+ public health+ lead expos* +plumbosol*+cost			108

Table 1. Number of citations retrieved by keyword/key-phrase and search engine. SD: Science Direct; WoS: Web of Science; GS: Google Scholar.

- The search term Pb delivered articles which had the initials PB in a great number of authors' names and dealt with research on the substance polybutylene (PB). This also explained the great number of articles for Pb and search terms such as drinking or tap water.
- The word cost was mentioned in many articles in the context of the cost of total lead pipe replacement but without specific economic appraisal.

Overall, more than 200 peer reviewed articles alongside additional book reviews and work by regulatory and policy organisations such as EPAs, WRC, CIWEM, UKWIR and other independent reviews to governments were read and used for this report.

PART I - BACKGROUND

4.0 Lead: Lead cycle, health effects and legislation

4.1 Lead in the environment

Lead is naturally found in very small amounts in air, soil and water in areas with lead-bearing ore deposits such as galena, and in association with zinc-, copper, and silver ores (Keim and Markl 2015). 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 (Hernberg 2000). Anthropogenic lead may contaminate locally surface waters and groundwater through lead-emitting point-sources of lead, such as mines and smelters, refineries, recycling and storage plants as well as sewage outfalls and harbours (IARC 2006). **The predominant and direct source of lead in drinking water is lead-containing piping and plumbing (WHO 2011).**

Knowledge of the environmental lead cycle has helped to: (i) understand sources of human lead exposure; (ii) develop source-specific lead policies to control and, where possible, phase out lead from these sources; (iii) set health based thresholds for total lead intake or lead exposure, such as the limit of 5 µg/dl of lead in blood set in the USA by the Centers for Disease Control and Prevention (Brown and Margollis 2012; CDC 2012); and (iv) lay down regulations for achievable limits for the concentration of lead in a range of environmental media, such as in drinking water at 10 µg/L (WHO 2011) and air at 0.5 µg/m^3 (WHO 2001).

The key parts of the lead cycle are detailed in Annex I.1. Anthropogenic lead can be found in:

- Ambient air, as the main transport pathway from anthropogenic sources of organic and inorganic lead (i.e. car and aviation emissions, smelting and other industrial emissions, plumbing, paint) to soils, surface and groundwater waterbodies, indoor air, dust and ecosystems.

- Soils, as the major sources of inorganic lead in surface and groundwater waterbodies via runoff; in crops (food); and, directly or indirectly, in outdoor or indoor dust.

- Paint, as the major source of inorganic lead in indoor dust and residential (garden) soil.

- Plumbing, as the major source of inorganic lead in drinking water and sewage effluents.

- Industry emissions, soil, dust and paint dust as the major sources of non- dietary lead intake.

- Cereals, vegetables and drinking water as the main sources of dietary lead intake; it must be clarified that this refers to lead-contaminated drinking water from lead-containing pipes and plumbing. In Scotland, there are no issues with lead in drinking water for the largest proportion of households served by public water supply (see Section 5.1.4).

- Smoking tobacco and second hand smoke, as everyday sources of lead exposure.

- Traditional remedies, cosmetics (not marketed in the EU) and pottery as sources of lead for specific ethnic groups.

Lead emissions to the environment contribute to the "background" or "remote" levels of lead in the environment, which should not be confused with natural levels (Mushak 2011). Background lead levels must be examined in the context of lead uses and regulations over human history. Environmental, anthropogenic lead is the major source of human exposure to lead.

4.2 Exposure to lead - Types of lead exposure

Lead can enter the human body in three ways. Firstly, by inhalation of lead particles generated by activities such as smelting and informal recycling; flaked off leaded paint, indoors or outdoors; and leaded gasoline emissions, currently almost exclusively from aviation (Markowitz 2000; Zahran et al 2017). Secondly, by ingestion of mainly inorganic lead-contaminated dust; water from leaded plumbing; or food due to use of lead-glazed or lead-soldered containers or lead-containing diet (EFSA 2010; Markowitz 2000). Thirdly, by direct skin contact with organic (i.e. tetraethyl) lead in leaded petrol (CDC 1978).

The main routes of exposure to lead are occupational and residential-environmental. Occupational exposure to lead mainly affects adults; in the UK, it may occur in a variety of workplaces including steel welding and spray coating, battery manufacturing or plumbing (Public Health England 2016). Residential exposure to inorganic lead affects children and adults alike and occurs primarily through peeled off lead paint and lead-contaminated food and drinking water, although exposure may also occur through soil, dust and air (WHO 2011).

Lead exposure may refer to the following:

- Acute exposure: This has been associated with blood lead levels in the range of 100-200 μg/dl in adults and 80-100 μg/dl in children (Papanikolaou et al 2005; WHO 2011). Acute exposure is more often associated with occupational exposures, which are not examined in this report.

- Chronic high exposure: This has been associated with blood lead levels of 40-60 μg/dl for one to two years of exposure and blood lead levels of 50-80 μg/L for longer term periods (Papanikolaou et al 2005). Lead poisoning may refer to acute or chronic exposure and requires specific medical treatment, e.g. chelation (WHO 2011).

- Elevated versus low lead exposure: These can also be chronic but usually refer to lead exposure above or below the level requiring medical, nutritional or educational intervention, respectively. As a concept, elevated and low lead exposures must always be used in the context of the regulations referring to blood lead levels and the period in history. For example, for Public Health England (n.d.) and the WHO (2011) elevated lead exposure refers to blood lead levels above 10 μg/L; for the CDC (2012) in the USA elevated lead exposure refers to blood lead levels above 5 μg/dl; the Global Burden of Disease studies (e.g. GBD 2015) consider as low exposure the blood lead levels at 2 μg/dl due to background sources of environmental lead. Pre-industrial levels of lead exposure have been estimated to be approximately 0.016 μg/dl of lead in blood (Flegal and Smith 1992).

- **Safe lead exposure: this is a non-existent concept. No safe level or threshold for lead exposure has been agreed by experts or health agencies. Instead, it has been emphasised that blood lead levels should be as low as possible given the current background levels of environmental lead (ATSDR 2017; Brown and Margolis 2012; CDC 2012, , GBD 2015; WHO 2011). The background lead levels should not be perceived as natural because of the widespread lead contamination through atmospheric pathways of transport and cumulative deposition (see Annex I.1).**

4.3 Mechanisms of lead toxicity - Dose response relationships

Mechanisms of lead toxicity are summarised in Annex I.3. Once taken in, lead enters the bloodstream and accumulates in bones, teeth, hair and nails and interferes with the function of vital organs (especially the liver, kidneys and brain); in pregnant women it crosses the placental barrier and affects the unborn infant (Mushak 2011:Chapter 8; WHO 2011). Lead mainly targets the central nervous system by interfering with the function of neurotransmitters, thus disrupting learning, memory, and sensory and motor skills, i.e. it causes idiopathic intellectual disability (Lidsky and Schneider 2003), hereafter reported as intellectual impairment[1].

The health effects of lead exposure levels are well studied and well known (Figure 2). These effects 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 intellectual impairment (e.g. decreased IQ scores, speech and language disorders) (ATSDR 2017). Endogenous release of lead into the bloodstream from the bones, where it was stored during past exposure, is a significant source of exposure to lead in prenatal (through trans-placental exposure) and adult life (e.g. Bellinger 2017). It must be noted that many affected children and adults may remain asymptomatic or misdiagnosed for a long time (Kalra et al 2000).

Annex I.4 presents epidemiological studies from the UK, USA, Germany, France, and Canada that explored dose (blood lead)-response (health effect) relationships. The studies on lead dose-response relationships show that **evidence on the adverse effects of lead at ever lower levels of exposure is growing.** The thresholds at which certain clinical and cognitive symptoms or social outcomes appear in specific age groups occur have informed policy on how levels of exposure below these thresholds can be achieved through control of environmental emissions and concentrations in drinking water and other media to protect public health. Table 2 shows the most recent evidence (i.e. by May 2017) for certain effects and symptoms observed at or above specified blood lead levels.

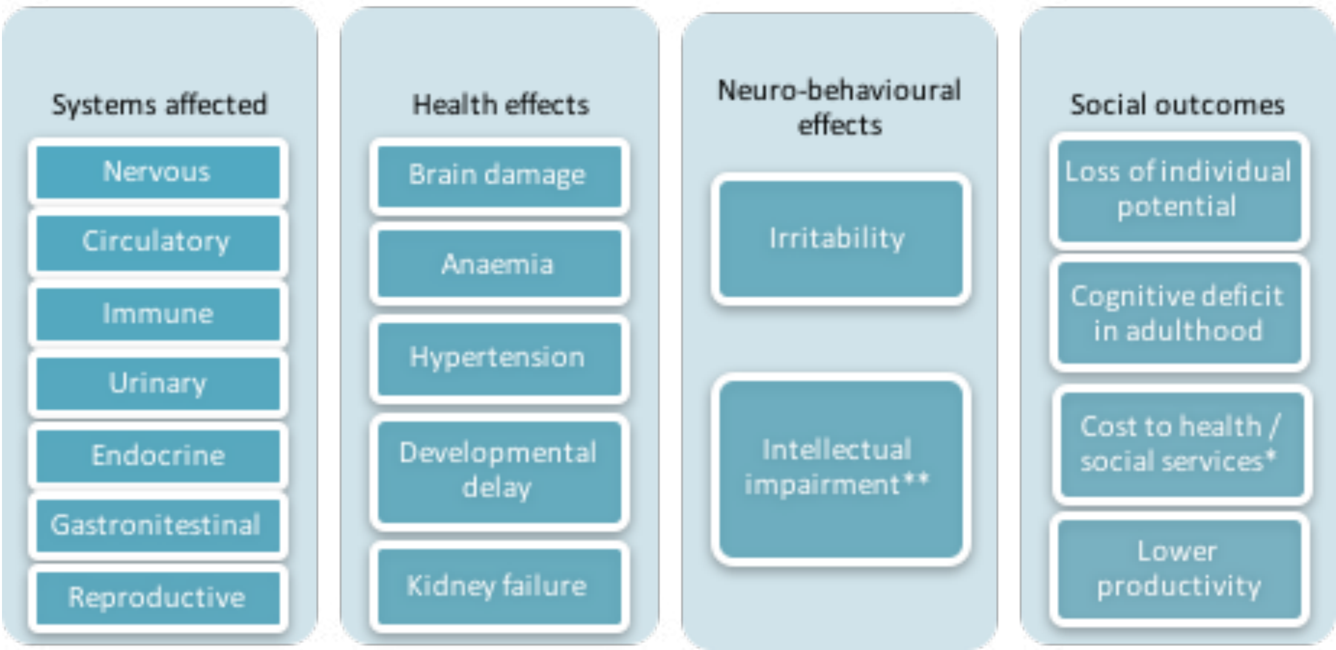


Figure 2 Adverse health effects of lead exposure in children and adults who were exposed as children (ATSDR 2017; Bellinger 2017; Bellinger and Needleman 2003; Brown and Margollis 2012; CDC 2012; Eid and Zawia 2016; Gilbert and Weiss 2006; Lidsky and Schneider 2003; Needleman and Gee 2013; Papanikolaou et al 2005; Reuben et al 2017; Taylor et al 2014; Troesken 2006). Health effects of lead exposure are detailed in Annex I.4. *Cost is discussed in Section 10. **See footnote 1.

[1] Idiopathic developmental intellectual disability (intellectual impairment) includes the following symptoms arising in the developmental period <18 years: language delay; fine motor delay; cognitive delay (e.g. poor memory and logical reasoning); social delay; behavioural disturbances (e.g. hyperactivity and aggression in infants and toddlers); neurologic and physical abnormalities (e.g. visual impairment and hearing deficit).

Table 2. Blood lead-health relationship in children and adults. Main sources as in Figure 2.		
Blood lead levels (µg/dl)	Effects on children's health	Effects on Adults' health
0.016*	Unknown	Unknown
2**	Uncertain	Uncertain
2-10	Intellectual impairment	Maybe asymptomatic if not exposed in childhood. Cognitive deficit, lower productivity and income related to low exposure in childhood.
10-20	Impairment of blood function	Hypertension
20-30	Decreased nerve conduction	Impairment of blood function
30-40	Decreased vitamin D metabolism	Decreased hearing; high systolic blood pressure
40-50	Decreased haemoglobin synthesis	Decreased nerve conduction; infertility (men); kidney failure
>50	Colic, anaemia, kidney failure, brain disorders (encephalopathy)	Decreased haemoglobin synthesis; anaemia, brain disorders
>100	Death	Death

*pre-industrial Native Americans (Flegal and Smith 1992) **Background lead exposure (GBD 2015).

4.4 Lead intake and uptake

A detailed review of the factors influencing intake and uptake and levels of intake from the literature are provided in Annex I.2. To summarise, lead exposure depends on lead intake and uptake which depend on age; body weight; nutritional status; and consumption rate of lead-containing media such as drinking water, food and air. Children, malnourished individuals and pregnant women are at higher risk of absorbing the lead once taken in (Annex I.2; see also Mushak 2011: Chapter 7).

4.5 Lead intake from lead-contaminated water

The current standard for lead in drinking water in Europe, Canada and Australia (10 µg Pb /l) is consistent with the World Health Organisation (WHO) provisional guideline value for maximum lead in drinking water (WHO 2011). The value of 10 µg Pb /l was based on the decision of the Joint FAO/WHO Expert Committee on Food Additives (JECFA 1999) for a provisional tolerable weekly intake (PTWI) of 25 µg/kg per body weight. This translates into a provisional tolerable daily total lead intake of 1.9 µg/kg of body weight in 1–4 year old children and a daily total lead intake of 3.0 µg/kg body weight/ day in adults (JECFA 1999).

Lead intake via lead-contaminated water can be calculated as the product of lead concentration in water and the volume of water consumed daily. To illustrate a worst case scenario, i.e. when lead concentrations in water equal 10 µg/L, and assuming an uptake of 50% of lead from drinking water, tap water lead intake can range from 7.5 µg/day or 1.5 µg/kg of body weight/day for an infant (assuming consumption of 0.75 L drinking water per day and a weight of 5 kg for a three-month old infant) to 10-16 µg/day or 1-1.7 µg/kg of body weight/day for 1-4 year of children (assuming a daily water consumption of 1-1.6 L[2] and a body weight in the range of 10-17 kg[3]) (JECFA 2011; WHO 2011).

JECFA (2011) have since withdrawn the PTWI on the basis of more recent evidence that the previously established PTWI of 25 µg/kg of body weight is associated with a decrease of at least 3 intelligence quotient (IQ) points in children and an increase in systolic blood pressure of approximately 3 mmHg (0.4 kPa) in adults. These changes are important when viewed as a shift in the distribution of IQ or blood pressure within a population (JECFA 2011). WHO recognised this development but acknowledged that achieving lead levels of less than 10 µg/L in drinking water may be very difficult, both economically and technically; therefore, WHO (2011) designated the current guideline for the lead maximum value of 10 µg/L in drinking water as "provisional".

In premises with lead-bearing plumbing, which is the predominant source of lead in drinking water, estimates of water lead intake have a wide margin of error as it is not known to what extent the general public flushes the water system before use, and whether the variability in lead corrosion chemistry causes exposure to higher undetected level of lead in frequent intervals (EFSA 2010) (see also

[2] From drinking water and food (EFSA 2010)

[3] Evidence form Royal College of Pediatrics and Health-RCPCH (n.d.)

Annex III for factors influencing lead chemistry in the distribution system). Arguably, levels of daily water lead intake such as 1.5 μg/ kg of body weight for infants or 1.7 μg/kg of body weight for 1-4 year old children are only slightly lower than the daily total lead intake of 1.9 μg/ kg of body weight/day estimated by JECFA (2011) to be of concern for neurodevelopmental effects in infants and children; see also Sections 4.4 and 4.5. It follows that where additional sources of lead in a child's environment occur, water lead at 10 μg/L may be a surplus exposure. On the basis of these considerations, WHO (2011) has warned that there may be a risk for bottle-fed infants through intake of drinking water with a lead concentration of 10 μg/L.

4.6 Global Burden of Disease

The Global Burden of Disease (GBD) study was launched in 1991 by the World Bank - WHO (1993) to provide comprehensive assessments at regular intervals of the state of health in the world. Collected and analysed by a consortium of more than 2,300 researchers in more than 130 countries, GBD data capture premature death and disability from more than 300 environmental, nutritional, physiological and social determinants of health in 195 countries from 1990 to the present. The GBD studies have defined health impact due to lead exposure regardless of source or the route of exposure (i.e. inhalation of lead-contaminated air or ingestion of lead-contaminated water, food or paint dust) in terms of blood lead levels in μg/dL and/or bone lead levels in μg/g of bone (e.g. GBD 2015).

The global burden of disease is the sum of disability-adjusted life-years (DALYs). DALYs are calculated as the Years of Life Lost (YLL) due to premature mortality in the population and the Years Lost due to Disability (YLD) for people living with a health condition or its consequences (Murray et al 2013). The DALYs can be thought of as a measurement of the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability (WHO 2017). The calculation of DALYs excludes all non-explicitly health-related characteristics such as ethnicity, socioeconomic status or occupation.

Lead exposure has been one of the leading risk factors of the global burden of disease during the past 25 years (Ezzati et al 2002; GBD 2015; Murray et al 2013; WHO 2009). In these studies, the number of deaths and DALYs due to a specific disease attributed to lead exposure per age group, gender, country and year are estimated using the minimum and maximum levels of blood or bone lead level observed and the theoretical minimum level of lead exposure as of 2 μg/dL of lead in blood. This corresponds to background environmental lead levels, as current sources of lead prevent the feasibility of zero exposure (GBD 2015). A two-stage process has been commonly used: (i) identification of the relation between bone or blood lead levels and systolic blood pressure; and (ii) identification of the relation between change in blood pressure and disease outcomes.

GBD studies are reviewed in Section 5.4 in the context of the burden of disease due to lead exposure in Scotland.

It is worth noting that lead exposure was the single contributing risk factor for intellectual impairment in Europe and on a global level (GBD 2015; IHME-GBD Compare 2017). In response to the results of the GBD studies, the WHO has identified lead as one of ten chemicals of major public health concern, requiring action by all countries to protect the health of workers, children and women of reproductive age (WHO 2017).

4.7 Legislations to control lead - Regulatory time-lags

The regulation of lead in drinking water began in earnest in the late 1970s in Europe and elsewhere in the developed world. Specific national and international legislations refer to thresholds in emissions or concentrations of lead in environmental media such as drinking water, air, food, petrol and other consumer products and values of action or concern for actual lead exposure (Annex I.6). In terms of reducing lead at source in order to reduce exposure, all pieces of legislation are relevant to lead-free policies. A timeline of the major regulations in the wider context of scientific understanding of the adverse effects of lead exposure through time is given in Box 1.

Warnings about lead poisoning have existed in relation to pipe manufacturing and the distribution of plumbosolvent water through lead pipes since antiquity and throughout the 19th century. For example, the 1st Century Greco-Roman physician Dioscourides observed that "Lead makes the mind give way"; in 1844 the Scottish toxicologist R. Christison had warned about lead poisoning due to the distribution of acidic water from Loch Katrine through lead-containing pipes in Glasgow (Box 1). However, these and other warnings about the use of lead in food, drinks and petrol went largely unheeded due to the economic implications of phasing out lead from its widespread uses (Hayes 2010; Mushak 2011; Needleman and Gee 2013). Crucially, the advent of engineering solutions and new technologies (e.g. catalytic converters, water treatment for lead removal, lead recycling) and the potential to use alternative materials to lead such as plastics in water distribution systems and titanium oxide in paint, contributed to taking action to phase out or phase down lead (Needleman and Gee 2013).

Until the late 1960s, this regulatory time-lag was also supported by scientific research sponsored by the lead-related industry, including the car and mining industries, which concluded that blood lead levels were "normal" (Needlemann and Gee 2013 and literature cited therein). Such findings influenced guidelines set by the WHO and public perceptions of lead toxicity. Following an inquiry in the USA about the importance of the subclinical health effects of low lead exposure in 1966 (see Box 1), independent scientists sponsored by public funds demonstrated that the lead body burdens arising from all manmade sources of lead were not "normal", as the lead industry claimed (Needlemann and Gee 2013 and literature cited therein).

Box 1A. Timeline of lead use, toxicity warnings and major regulations until 1970.	
Until 1970	Extensive use of lead in water pipes, storage tanks, paint, pottery, and as a sweetener; these uses are now banned.
1st c. AD	Greco-Roman physician Dioscourides observes: "Lead makes the mind give way".
1723	Law bans the use of leaden "worms" in the rum distilling process (USA).
1768	Devonshire physician Sir G. Baker showed that the colic epidemic was caused by the leaden keys used in pressing cider apples; Baker was condemned by the clergy, mill owners and fellow physicians (UK).
1844	R. Christison, Professor of Materia Medica at the University of Edinburgh warns that lead pipes would cause lead poisoning in Glasgow because of the acidic moorland waters of Loch Katrine; his warnings were ignored.
1867	Potteries Regulations (UK) put legal controls on the use of lead in pottery manufacturing 30 years after warnings about lead poisoning "accidents" among workers in the industry.
1878	The Kingdom of Wuerttemberg in Germany bans the use of lead water pipes; other German kingdoms followed.
1922	Tetraethyl lead first added to petrol to improve fuel performance (USA).
By 1925	Extensive evidence shows that children are poisoned by lead in residential paint (Europe, USA, Australia).
1958	The WHO sets a limit for lead in drinking water at 100 µg/L
1962	The WHO sets a limit for lead in drinking water at 50 µg/L
1966	Senator E. Muskie, Chairman of the Senate Subcommitte on Air and Water Pollution organises an inquiry about the importance of subclinical health effects of low lead exposure; this marked a paradigm shift in lead policy.
Sources: Needlemann and Gee 2013; Potter 1997; World Bank-WHO 1993.	

Box 1B. Timeline of lead use, toxicity warnings and major regulations after 1970.	
By 1970	Almost all gasoline used contains lead (All countries).
	Lead in new pipework and plumbing has been superseded by other materials (Developed countries mainly).
1971	Lead-Based Paint Poisoning Prevention Act (USA).
	The WHO resets the limit of 100 µg/L in drinking water.
	UK and Germany begin to reduce permitted levels of lead in petrol
1975	The US EPA adopts the limit of 50 µg/L for lead in drinking water under the Safe Drinking Water Act.
1976-95	Developed countries phase out lead in gasoline.
1977	The EEC Directive 77/31 requires Blood Lead Surveys (1979-1981) and blood lead ≤ 20 µg/L in 50% of population.
1977-78	CDC (USA) starts a lead poisoning screening programme; blood lead action level is at 30 µg/dl.
1978	Ban on residential lead paint (USA) but not replaced in old housing.
1979	US EPA recommends that the geometric mean of blood lead concentration should not exceed 15 µg/dL.
	Guidelines are set for following up any child with blood lead levels > 30 µg/dL after EEC 77/31 Surveys (Netherlands).
1980	Potable Water Directive (80/778/EC) sets a standard of 50 µg/L for lead in "running" water.
1983	Guidelines (UK) for following up any child with blood lead > 30 µg/dl after EEC 77/31 Surveys.
1984	WHO sets a limit for lead in drinking water at 50 µg/L.
1986	Safe Drinking Water Act (USA) sets an action level of lead at 15 µg/L in public supplies and bans lead in plumbing.
	Water supply bylaws ban the use of lead solder from hot and cold water systems (Scotland).
1989	EU Directive 89/677/EEC bans white lead paint.
1991	Lead Copper Rule (USA) sets a maximum contaminant level goal of zero lead in drinking water.
	The CDC (USA) adopts an action level for blood lead concentration at 10 µg/dl (children) and 25 µg/dl (adults).
1992	Regulations are set for controls on injurious substances including lead (UK).
	Guidelines for Canadian Drinking Water Quality state a Maximum Allowable Concentration for lead at 10 µg/L.
1993	WHO sets a health-based guideline maximum value for lead in drinking water at 10 µg/L (still in place).
1995	Ban on lead solder in food cans (USA)
1998	EU Directive 98/83/EU sets an interim standard for lead in drinking water of 25 µg/L from 2003 to 2013 and a standard of 10 µg/L after 2013.
1999	Unleaded petrol accounts for 80% of total sales on a global basis.
2000	EU bans leaded gasoline.
2001	Regulations are set on mandatory communication pipe replacement in the case of lead standard exceedances (Scotland).
2007	REACH/ Lead-free Directive (EU).
2011	WHO warns that the standard of lead in drinking water at 10 µg/L may be too high for bottle-fed infants
2012	Blood lead concentration of 5 µg/dL are set as level of concern for total lead exposure by CDC.
2017	Growing evidence shows that adults exposed to low blood lead (<10 µg/dL) when children have cognitive deficit.
Sources: Bellinger 2017; Brown and Margolis 2012; Eid and Zawia 2015; Hayes 2010; Landrigan 2002; Needlemann and Gee 2013; Quinn and Sherlock 1990; Potter 1997; Public Water Supply (Scotland) Regulations 2014; World Bank-WHO 1993; WHO 2011; Reuben et al 2017.	

PART II - LEAD IN SCOTLAND

5.0 Overview of lead control in Scotland

The use, release and environmental levels of lead are strictly regulated in Scotland to reduce risk to human health and the environment under European and international legislations (Annex I.6). Specific regulations apply for the control of lead levels in tap water (see Annex I.6.1) and its sampling, to ensure that the water lead levels observed are below 10 µg/L and are representative of a weekly average value ingested by consumers. A random daytime sampling approach (see Section 9) is used for the monitoring of lead in drinking water throughout the UK and in Scotland. In addition to water lead control, source-specific legislations are in place to control:

- Industrial releases of lead to air, land and surface waters and groundwater;
- The presence of lead in a wide range of products from toys to electronic waste; and
- The concentration of lead in foodstuff and animal feed (Annex I.6.2).

Of all these legislations, the most relevant to the regulation of lead in drinking water are the:

- Drinking Water Directive –DWD (98/83/EU);
- Water Framework Directive –WFD (2000/60/EC);
- EU Restriction of Hazardous Substances Directive-RoHS (2002/95/EC);
- Registration, Evaluation, Authorization, and Restriction of Chemicals -REACH legislation

These pieces of legislation have already influenced the uses of lead in Scotland. Lead in the blood of workers in the lead industry is also strictly regulated in Scotland (Annex I.6.3). There are no regulations for mandatory blood lead screening in the general population apart from guidance for an action blood lead level at 10 µg/dl (Public Health England n.d.); see also Annex I.6.4.

5.1 Lead in water in Scotland

5.1.1 Lead in raw water resources

In Scotland, groundwater and water from the uplands is normally lead-free or has very low lead levels, i.e. well below the limit of 10 µg/L set in the Water Framework Directive (MacDonald et al. 2005). Public water supplies use lead-free raw sources.

Naturally-enhanced levels of lead in groundwater have been observed in lead mining sites such as Tyndrum and Comrie, and Leadhills in the Southern Uplands (MacDonald et al. 2005). SEPA[4] also found that Glengonnar Water at Leadhills failed the limit for lead in 2010 and 2011 (Chandler et al 2012). Surface watercourses between Leadhills and the River Clyde at Abington had elevated lead levels, with concentrations increasing downstream to a peak of 83 µg/L of dissolved lead (but 174 µg/L total) below the Glendorch smelter mill (Chandler et al 2012). However, it was uncertain where the lead originated because high levels of dissolved lead were observed at sites near point sources, i.e. Leadhills sewage works (17 µg/L); in two local springs (48 and 83 µg/L); and in the local soils (up to 94 g/kg), the floodplain and the alluvium (both over 100 g/kg) (Chandler et al 2012). No public supply sources occur in this area.

Evidence on lead emissions and lead that has accumulated in catchment soils and fluvial deposits in Scotland from historical and recent industrial activity and car emissions is presented in Annex II.1. This evidence suggests that manmade lead is more widespread in remote catchments of Scotland than previously thought. The implications for private water supplies remain unexplored.

5.1.2 Lead in bottled water

A UK-wide study by Smedley (2010) demonstrated that bottled water compositions were mostly similar in their major-ion characteristics to raw groundwaters from the equivalent aquifers in Britain. However, concentrations of several trace elements including lead were appreciably lower, in some cases by one or two orders of magnitude. The most likely mechanism for the reduction is use of aeration, settling and filtration to remove unstable constituents before bottling. The comparatively low concentrations of lead in bottled water were likely to be due to co-precipitation with/adsorption to precipitated metal oxides, although choice of resilient pipework (e.g. stainless steel) in bottling plants may have also been a factor (Smedley 2010).

5.1.3 Tap water lead levels before liming and orthophosphate dosing

Although the raw water sources used for public water supplies contain no lead, the chemistry of raw water sources and the extensive use of lead in the water distribution materials and plumbing components since the 19th century resulted in high levels of lead in drinking water in certain areas of Scotland. In the era of industrialisation and urban growth, remote, upland water sources had to be used to reduce the risk of waterborne disease outbreaks in urban centres (UK Department of the Environment - DOE 1983). The problems of water acidity were compounded by the extensive use of lead in the public network and the domestic plumbing systems to cover, cost-effectively, the increasing urban population demands (Moore 1985). In addition to this, until 1967, there was widespread use of lead pipes to link cast-iron mains and domestic pipes (Moore 1985) and of lead-lined water storage tanks in urban and rural areas (DOE 1983; Potter 1997).

The plumbosolvency of public water in Scotland was officially demonstrated during the UK-wide survey of lead in drinking water which was organised by DOE in 1975-1976 (DOE 1983; Richards and Moore 1984). This survey showed that 21% of households in Scotland had random daytime (RDT) (see Section 9) water lead levels equal or above 100 µg/L versus 2.6% and 2.3% for England and Wales, respectively. In addition, lead levels in the range of 50– 100 µg/ L were found in 13.4% of households in Scotland versus only 5.2% and 6.5% for England and Wales, respectively. Overall, over one-third of households in Scotland had random daytime (RDT) (see Section 9) water lead levels above 50 µg/L (DOE 1983). The highest levels were observed in houses with lead and copper pipes (18% of households) and houses with lead pipes only (28% of households) (DOE 1983). In the West of Scotland, the pH of the raw water used for the public network was very low

[4] Lead data from surface waters (collected to meet the requirements of WFD) are not readily available on SEPA's website.

(Addis and Moore 1977). For example, the pH of water at source and without treatment for pH correction was 6.3 in Glasgow in 1976 and 4.5-5.5 in Ayr in 1980/1981 (Moore et al 1985).

5.1.4 Tap water lead levels after liming and orthophosphate dosing

Surveys conducted in 1993 in Glasgow after orthophosphate dosing and liming (i.e. the water was treated to achieve pH=8-9 and dosed with 2 mg/L of phosphate as reported by Richards and Moore, 1984), showed that an estimated 83% of mothers lived in households with tap water lead concentrations below 10 μg/L (Watt et al 1996; 2000).

After the adoption of the DWD and until 2013 the majority of public supply zones complied with the standard value of 25μg/L (DWQR 2016a), with compliance in more than 99% of samples collected with the random daytime (RDT) sampling protocol (see Section 9). The standard value for lead reduced from 25 to 10μg/L in 2013. In 2015, 99% of 1499 RDT samples from randomly selected consumer taps complied with the standard of 10 μg/L (DWQR 2016a). The 1% of samples that failed the lead standard was due to 15 failures, which occurred singly, in separate supply zones[5] (DWQR 2016a: p. 13): eight occurred in zones with orthophosphate treatment in place, although three were not fully optimised (optimisation refers to the dose required to achieve compliance with the water lead standard of 10 μg/L; see also Section 6.1); and seven zones were considered to be of low risk , i.e zones considered as not requiring orthophosphate dosing to address plumbosolvency. In four cases, customer-side pipes were acknowledged to be of lead. In two cases the cause was undetermined.

5.1.5 Lead in piping and premise plumbing

The use of lead in premise plumbing was phased out during the 1950s-60s (Scottish Government 2014). Potter (1997), in a review to inform the members of the UK parliament on the problem of lead in drinking water, mentioned that homes built after the late 1960s are unlikely to have lead pipes and that no new lead-containing water storage tanks have been installed since the 1970s. By that time, lead use in plumbing was discouraged and locally-made water byelaws had been introduced to ban the use of lead plumbing; the ban was consolidated in byelaws made by the Regional and Islands Councils during 1987 (Ramsay 2003; Scottish Government 2014).

Peters et al (1999) examined the corrosion products, obtained from lead service pipes carrying the public drinking water supply to the Glasgow area. Lead carbonate or basic lead carbonate formed in the presence of pH-adjusted water. A variable proportion (up to or similar to 30% w/w) of a phosphate species (e.g. lead hydroxyapatite, Pb-5(PO4)(3)OH) formed in areas where the water supply had been treated with orthophosphate and pH adjustment for up to eight years; see also Annex III.2.1 for evidence on lead scale solubility.

The Scottish New Homes Lead Survey (SNHLS) funded by the Scottish Executive Health Department showed that lead solder and brass fittings had been used for jointing the domestic copper pipework in new housing long after 1987 (Ramsay 2003). This use was characterised as "illegal" by Ramsay (2003). It was estimated that the proportion of new houses identified as being affected by the "illegal" use of leaded solder could refer to more than 15% of new homes built between 1987 and 2000 and that 31.4% of houses built in 2000 had water lead levels equal or above 5 μg/L (Ramsay 2003). The highest water lead levels were observed in samples taken after overnight stagnation (263.9 μg/l) followed by random daytime (see Section 9) samples (93.3 μg/l); the lowest water lead levels were observed in fully flushed samples (2.4 μg/l) (Table 3). The study also concluded that random daytime sample testing underestimated the number of affected houses by more than 50% (Ramsay 2003).

Table 3. Results from the Scottish New Homes Lead Survey (SNHLS). Source: Ramsay 2003

Lead Level (μg/l)	Overnight		Random		Stagnation		Flushed	
	No.	%	No.	%	No.	%	No.	%
<5	34	56.7	49	81.7	53	89.8	59	100
5<10	8	13.3	4	6.7	4	6.8	0	0
10<25	9	15.0	3	5.0	2	3.4	0	0
25<50	2	3.3	3	5.0	0	0	0	0
50<100	4	6.7	1	1.7	0	0	0	0
100<200	1	1.7	0	0	0	0	0	0
200<300	2	3.3	0	0	0	0	0	0
TOTAL	60	100	60	100	59	100	59	100

Sampling

Overnight: Sampling following overnight stagnation of the cold water system

Random: Sampling irrespective of when the tap had been used last

Flushed: Sampling after the cold tap was left running for a period of minutes, to account for background lead levels in the mains

Stagnation: Sampling following 30 minutes of stagnation of the cold water system

[5] DWQR served Scottish Water (SW) with a Consideration of Enforcement letter in July 2015; subsequently, measures were put in place by Scottish Water (DWQR 2016a).

5.1.6 Enabling regulations and practices for the mitigation of lead in drinking water

Under the Housing (Scotland) Act 2006, local authorities can use the wholesome water supply criterion of the standard for lead in drinking water to indicate houses that do not meet this criterion. Scottish Water holds information on areas likely to have lead pipes[6] and whether it has treated the supply to an area with orthophosphate to reduce plumbosolvency; this information can be made available to local authorities (Scottish Government 2009). Tools for dealing with houses in which tap water may potentially be a source of lead exposure due to lead supply piping and premise plumbing are available to local authorities through the Water (Scotland) Act 1980 and the Housing (Scotland) Act 2006. These tools include discretionary grants to replace lead plumbing in houses served by Scottish Water or private water supplies.

As of 2014, Scottish Water's (SW) business plan has allowed for £4.4 million for the anticipated removal of 6,500 lead communications pipes, which is around 9% of the remaining lead communications pipes in the network (SW 2014). Further, 93 supply zones covering 1.5 million customers are treated with carefully controlled orthophosphate dosing (SW 2014) to enable the formation of a lead phosphate passivating layer between the lead pipe and the water. The contribution of this phosphate to waste water phosphorus is not as big as previously thought (pers.com. Rachel Philip SW); phosphate is removed in waste water treatment plants by means of chemical precipitation to reduce phosphorus discharges to the environment.

5.2 Non-waterborne lead in Scotland

Evidence on the range of sources of lead exposure in Scotland is provided in Annex II.1. Strict regulations have resulted in remarkably lower lead levels in air presently than in the past. How historic and present day manmade lead emissions into air and water translate to contamination of the food chain and human lead exposure remains unexplored. In any case, the implication is that considerable amounts of lead have been stored (sequestered) in Scottish soils.

5.3 Lead exposure in Scotland

The most important evidence on elevated lead exposure in relation to plumbosolvent water in Scotland comes from the Blood Lead Surveys conducted in 1979-1981 under the European Economic Community (EEC) Directive (EEC 77/312), known as the Blood Lead Screening Directive (Annex I.6.4). This Directive laid down thresholds for lead exposure in terms of the prevalence (percentage) of specific blood lead reference levels in the general population. For example, no more than 2% (98th percentile) of the population including infants should have a blood lead level above 35 µg/dl. The reference levels set in the Blood Screening Directive were exceeded in Glasgow and Ayr; this finding was attributed to high drinking water lead levels in the public water distribution system (Quinn 1985). The findings of the Blood Lead Surveys in Scotland are described Annex II.2 alongside findings from all other surveys of lead exposure in Scotland during the past 50 years.

After implementing water treatment measures to reduce lead corrosion, the Scottish Health Department funded a random maternal blood lead survey in 1993 in Glasgow (see also Section 5.1); the households tested contained random daytime (see Section 9) water lead levels within a range of 0 µg/L to above 50 µg/L (Watt et al 1996; 2000). The blood lead tests showed that "only" 3.6% of the 1726 mothers tested had blood lead levels above 10 µg/dl (Watt et al 2000); no evidence was gathered on the prevalence of elevated lead exposure in infants or in areas with a lower range of water lead values in Scotland. The mean maternal blood lead concentration was 3.7 µg/dl in the population at large, compared with 3.3 µg/dl in households with negligible or absent tap water lead (Watt et al 2000).

The findings from the maternity blood lead surveys in Glasgow were considered to represent a safe level of lead exposure in adults and, in the context of the then evidence (e.g. WHO 1995 cited in Watt et al 2000), a considerable decline in blood lead levels due to lead mitigation in drinking water (Watt et al 2000). However, Watt et al (2000) concluded that *"while the long-term aim should be to eliminate all lead service[7] pipework in the water supply, resources should be targeted in the short term to reduce lead exposure in high risk groups, including pregnant women and bottle-fed infants. This may mean developing priority lead pipe replacement programmes for high risk population groups, to run in parallel with programmes based on more traditional property-based factors."*

A review of the available evidence in Scotland (Annex II.2) showed that many local studies have been carried out in areas with plumbosolvent water; these examined blood lead levels or sources of lead exposure in relation to local sources in the past and present or identified health effects of lead exposure. However, extensive (in terms of thousands of participants), cross-sectional, prospective or epidemiological studies on lead exposure with sufficient numbers of "control" data have never been carried out in relation to plumbosolvent water or other sources in Scotland.

[6] Only 'customer-side' and only where studies have been done (pers. com Bill Byers).

[7] Service pipes consist of communication (utility's side) and supply (homeowner's side) pipes; see also figure one.

5.4 Burden of disease due to lead exposure in Scotland

This section is based on Country Profiles, which provide an overview of findings from the Global Burden of Disease (GBD) studies, which are based on over 80,000 different data sources used by researchers to produce the most scientifically rigorous estimates possible. Estimates from the GBD study may differ from national statistics due to differences in data sources and methodology. These profiles can be freely downloaded and distributed (IHME-Country Profiles 2017).

The contribution of lead exposure to the average number of deaths and total disability-adjusted life-years (DALYs; see Section 4.6) in the total population of Scotland gradually decreased from 1990 to 2015 (IHME-GBD Compare 2017). A comparison of trends in the number of deaths, DALYS, and burden of disease between Scotland and the global average showed striking differences (see also Figure 3):

- In Scotland, number of deaths attributed to lead exposure reduced from 884 (range: 428-1361) in 1990 to 169 (range: 164-696) in 2015 (IHME-GBD Compare 2017). By contrast, the GBD study for 2013 (GBD study 2015) estimated that on a global level the average number of deaths due to lead exposure increased by 27.6% in 2013 as compared with 1990[8].

- In Scotland, DALYs due to lead exposure declined from 14,715 (range: 6782-23341) in 1990 to 4,992 (range: 1685-9084) in 2015 (IHME-GBD Compare 2017). Yet the GBD studies for 2013 (GBD study 2015) estimated that the average DALYs due to lead exposure increased by 8.5% in 2013 as compared with 1990, mainly due to population growth and ageing[9].

- In Scotland, the burden of intellectual impairment on the total population due to lead exposure was lower than on a global level from 1990 to 2015 (Figure 3a). In the total population, these levels ranged between 2.9% (in 2010) and 8.2% (in 1990) in Scotland and between 12.4% (in 2015) and 21.5% (in 1995) on a global level (Figure 3a). According to IHME-GBD Compare 2017, lead exposure among children younger than 5-years old in Scotland has accounted for 34-50% of the burden of intellectual impairment before 2000 and for approximately 23% of this burden since 2005 (Figure 3a).

- In Scotland, DALYs due to ischaemic heart disease, strokes and chronic kidney disease in the total population due to lead exposure were slightly greater than on a global level from 1990 to 2000 but slightly lower in Scotland than on a global level since 2005 (Figure 3b). However, both in Scotland and on a global level, the burden of these diseases attributed to lead exposure was always greater among adults older than 50 years of age than among younger individuals.

To sum up:

✓ The number of deaths and DALYs attributed to lead exposure in Scotland were three to four times lower in 2015 compared with 1990.

✓ Ischaemic heart disease is the top cause of death and DALYs in Scotland; it contributes to 9.16% of total DALYs in Scotland and it exclusively refers to adults (IHME-GBD Compare 2017).

✓ The burden of intellectual impairment attributed to lead exposure in Scotland is three to five times greater on children than on the total population and contributes to 0.13% of total DALYs in the country (IHME-GBD Compare 2017).

[8] Age-standardisation showed decreases in the number of deaths (by 3.3% on average) in 2013 compared with 1990 indicating a shift of the effects of lead exposure on life expectancy towards older age (GBD study 2015).

[9] Age-standardisation showed decreases in the DALYs (by 10.9% on average) due to lead exposure indicating a shift of the effects of lead exposure on

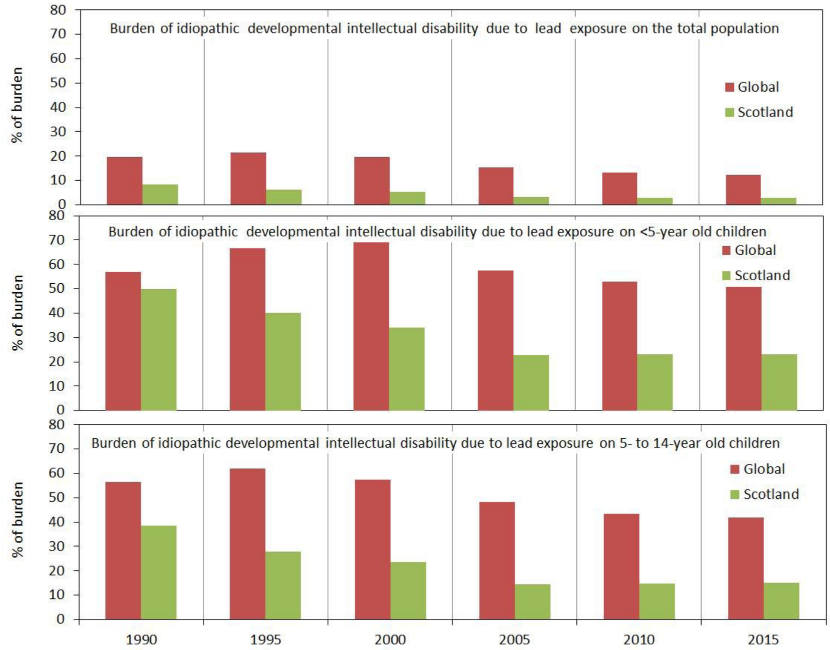


Figure 3a. Average burden of intellectual impairment attributed to lead exposure on a global level and in Scotland. Source: IHME-GBD Compare 2017.

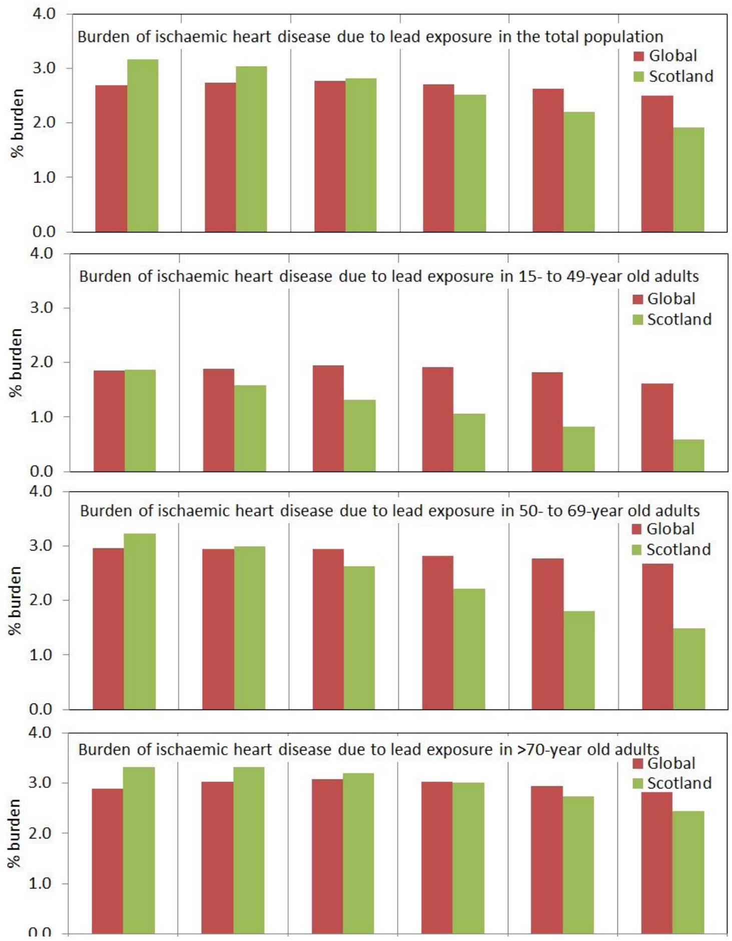


Figure 3b. Average burden of ischaemic heart disease attributed to lead exposure on a global level and in Scotland. Source: IHME-GBD Compare 2017.

Part III EVIDENCE REVIEW ON THE PUBLIC HEALTH ASPECTS OF LEAD IN DRINKING WATER

6.0 Factors influencing and reducing the presence of lead in drinking water

In Scotland, lead in drinking water arises from contact with lead-bearing pipes and plumbing materials in the drinking water route from the mains to the consumer tap within properties[10]. Lead concentrations at abstraction points of decentralised, small water supplies would depend on site geology and proximity to lead-contaminated land and groundwater sites, mainly due to smelting, mining, landfills and sewage discharge (Bower and Hayes 2016). Experiments have also shown that possibly all types of water have the potential to cause lead corrosion when in contact with lead-bearing plumbing and cause exceedances of the current lead limit of 10µg/L (Hayes 2010 p. 29).

The factors influencing the amount of lead that can leach from lead piping and plumbing components in the tap water have been studied extensively, (e.g. see reviews by: AWWA 1990; 2008; Bower and Hayes 2016; Cardew 2009; Croll 2000; de Mora et al 1987; Hayes 2010; Mushak 2011; Schock et al 1996). The following factors have been implicated in causing or increasing lead in drinking water after the treatment plant (Figure 4):

- Type of materials comprising the water distribution system (see details in Annex III.1.1).
- Presence of copper-lead (galvanic) and PVC-lead or brass connections (details in Annex III.1.2).
- Water chemistry (Annex III.2).
- Other factors, including the type of temperature; stagnation time, i.e. the period of time water sits stagnant in the distribution system; age of the system; and quality of workmanship (Annex III.2 and Annex III.3).

In general, these factors are interrelated. It has been suggested that these factors must be concurrently controlled to reduce effectively lead leaching from lead pipes and plumbing (Brown et al 2015; US EPA 2016). Common tap water lead mitigation practices include:

1. Controlling the chemistry of water entering the supply line and premises to reduce lead solubility (Section 6.1)

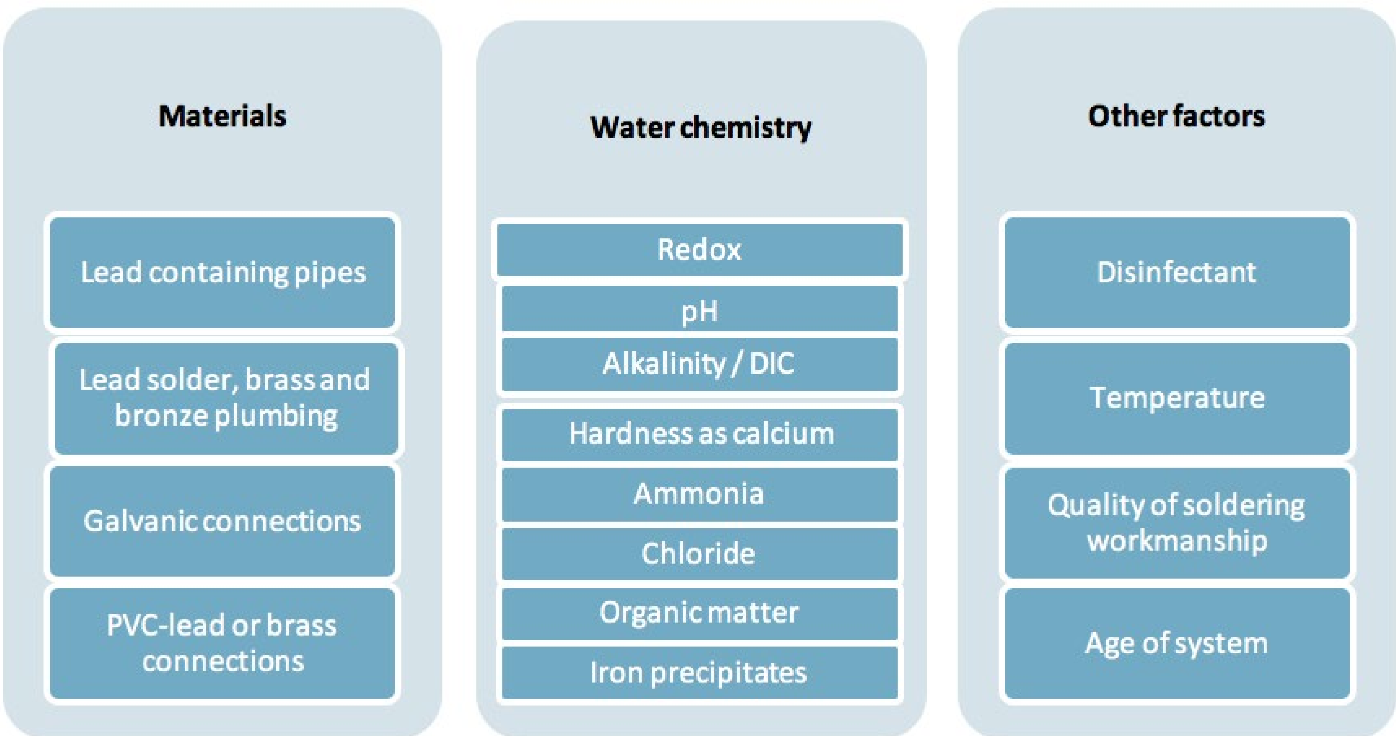


Figure 4. Summary of factors influencing lead leaching (plumbosolvency). The mechanisms whereby these factors influence lead solubility are detailed in Annex III.

[10] In other countries, where public raw sources may contain lead, lead is removed in water treatment plants of centralised, large (public) water supplies; in these cases, lead concentrations in the water leaving the plant can be up to 2-3 µg/L (Mushak 2011) but typically below 0.5 µg/L (Trueman et al 2016).

2. Replacement or renovation of lead pipes to eliminating any lead sources in the service lines and premise plumbing (Section 6.2)

3. Flushing to remove any detached scale or suspended particulate lead during typical use (Section 6.3)

4. Educational interventions (Section 6.4).

5. Installing an additional treatment barrier at the tap such as a point of use (POU) or at a point of entry (POE). The use of whole-house filters has been suggested as a partly-mitigating POE method as well as a particulate lead sampling approach to quantify semi-random release of particulate lead in systems with partial-lead pipe replacement (Partial-LPR) (e.g. St Clair et al 2016). POU and POE barriers are briefly examined in Section 6.5 for lead in small supplies.

The evidence presented in Annex III is not site or country specific, even though the studies have been conducted in specific country contexts. Where geography as a factor influences lead in drinking water, this is explicitly mentioned in the description of findings. Information on the conditions of the experiments has been reported, where possible or relevant, to inform transferability of the results of these studies in the Scottish context.

6.1 Chemical control of lead leaching

Water utilities have historically managed the release of lead from lead-containing pipe materials by controlling water pH and alkalinity to reduce the solubility of lead scales that coat plumbing materials (Brown et al 2015). More recently, the use of corrosion inhibitors, such as orthophosphate, zinc-orthophosphate, polyphosphate or a blend of phosphate types, has been promoted as a cost-effective approach to reducing lead concentrations compared with total pipe replacement (e.g. Brown et al 2015; UKWIR 2012; US EPA 2016).

Chemical control builds on a thorough evaluation of combinations of treatment conditions as regards pH, DIC, free chlorine, and phosphate or other corrosion inhibitors in both public and individual/small/private water systems.

Several reviews (e.g. AWWA 2008; Brown et al 2015; CIWEM 2016) and the evidence presented in Annex III.2 show that before a chemical control approach is applied, the benefits of lead control must be carefully weighed against potentially negative impacts on:

- Iron discolouration problems
- Microbial and algal growth from the treatment plant to households.
- Scales and the conditions maintaining the low solubility of already formed scales.
- Wastewater facilities and associated discharges to the environment (e.g. Zn and phosphate).

In this context, the following options can be considered for the chemical control of lead in drinking water (Brown et al 2013; 2015; Hayes 2010; US EPA 2016):

1. Maintenance of oxidized conditions with high free chlorine residuals (typically >1 mg/L as $Cl2$ to form and maintain insoluble lead (IV) scale); see also Annex III.2.2i.

2. The control of pH and alkalinity (DIC); background evidence is detailed in Annex III.2.2ii.

3. The use of orthophosphate within appropriate pH ranges; see also Section 6.1.1 and Annex III.3.4i.

4. The use of zinc-orthophosphates; see also Annex III.3.4ii.

5. The use of a blend of polyphosphates and orthophosphate; however, It has to be noted that polyphosphate alone is not normally considered as a lead corrosion inhibitor (Cantor et al 2000; US EPA 2016); see also Annex III.3.4ii.

6. The use of silicates; see also Annex III.3.4iii.

6.1.1 Orthophosphate dosing

It has been argued that orthophosphate dosing is the most successful of all the chemical strategies for the mitigation of lead in drinking water (AWWA 2008; Brown et al 2015; Hayes 2010; Hayes et al 2016; US EPA 2016). Optimised (see below *Optimisation*) orthophosphate dosing has helped keep lead levels below the set standard value for lead in drinking water in many countries but with considerable variability in resulting water lead levels by company, supply zone and circumstances (AWWA 2008; Hayes 2010; Hayes and Skubala 2009; UKWIR 2012; US EPA 2016). **Hayes (2010) reported that lead in drinking water is not a problem in the UK because of the extensive orthophosphate dosing. Orthophosphate dosing has been shown to be effective in the great majority of cases (UKWIR 2008 cited in Comber et al 2011). A recent British study found that, in addition to reducing lead levels from supply pipes overall, orthophosphate dosing is also effective in reducing lead leaching from brass fittings within premises (UKWIR 2014; 2016).**

However, orthophosphate can impact galvanic corrosion, encourage microbial growth, and increase the phosphorus content of wastewater discharges and mains water leakages

(Brown et al 2015: p.17; Gooddy et al 2017); see also Section 6.1.3 and Annex III.3.4i.

There are a number of caveats with respect to the effectiveness of orthophosphate dosing once pH, DIC, disinfectant and water usage have been accounted for. These include:

- The presence on aluminium. Aluminium can interfere with orthophosphate and form aluminium phosphate precipitates, thus reducing the amount of orthophosphate available for lead control (US EPA 2016). These precipitates may also form scales on the interior of piping systems, which may reduce the effective diameter of the pipes, resulting in loss of hydraulic capacity, increases in system head loss and operational costs (AWWA 2005). Changes in flow and water quality may dislodge aluminium phosphate precipitates causing increase of lead in tap water (Schock 2007).

- The effect of temperature on phosphate. Schock and Lytle (2011) showed that orthophosphate reacts more quickly at higher temperatures; therefore reduction in lead levels may take longer in colder months (or colder areas) than in warmer months. AWWA (2008) also showed that this is a practical consideration in England. See also Annex III.3.1.

- The presence of lead oxides. A recent study by Schock et al 2014 found that lead oxide scales are associated with low lead levels in tap water - as low as or lower than those found when orthophosphate treatment is used. In water supply zones that are compliant with the lead standard without phosphate dosing but contain lead at some parts of the water distribution system (outwith or within premises), switching from free chlorine to chloramine increases lead oxide solubility and thus may trigger lead non-compliances and high exposures to children and adults that may not be detected by regulatory sampling (Edwards et al 2009; Swtzer et al 2006). See also Annex III.2.2i.

Boyd et al (2010) studied the interaction between disinfectant, type of lead scales in the distribution system and orthophosphate effectiveness in achieving compliance with the water lead standard in Seattle and Washington, DC. They concluded that a distinction has to be made as follows:

- For lead plumbing materials that do not have extensive accumulation of surface scales containing lead dioxide, <u>changes in disinfectant are not likely to significantly impact lead leaching.</u>

- For lead plumbing materials that are passivated and likely to have developed scales that are rich in lead dioxide, <u>switch to chloramines is likely to cause a notable increase of lead leaching</u> when conversion to chloramines is implemented.

On the basis of their results, Boyd et al (2010) suggested that in systems with free-chlorine residual (i.e. high redox) tests should be performed to determine the nature of scales lining the service pipes and changes of redox potential associated with chlorine/chloramine conversion in the presence of orthophosphate.

6.1.1i Optimisation

The term optimisation may refer to <u>orthophosphate optimisation</u> to achieve compliance with the lead standard (as in the UK context, e.g. Hayes et al 2008). Or, it may be a more general term referring to <u>any</u> lead control treatment that minimizes the lead and copper concentrations at users' taps while insuring that the treatment helps to achieve drinking water quality objectives, supports ongoing water and wastewater system operations, and the ecology of receiving waters, as in the USA context (Brown et al 2015).

Preference for the one or the other chemical lead corrosion control option may be related to raw water, length of lead pipes, environmental reasons or reasons related to consumers' perception of the impact of treatment on organoleptic parameters such as taste and odour. For example, in the Netherlands, since the 1980s, the most common practice to reduce plumbosolvency was pH correction (Hayes 2010) and was found to be an effective solution to reduce lead below the standard value of 10 μg/L (Douglas et al 2007a cited in Hayes 2010). In the USA, all the methods have been examined and are in use depending on a utility's budget, the chemistry of raw water sources and the ecological status of receiving water bodies (McNeill and Edwards 2004; US EPA 2016).

Optimisation is not straightforward; it depends on the specific circumstances applying to a water supply zone, e.g. length of lead pipes, chemistry of raw water source, temperature and consumption patterns, which will influence lead concentrations in stagnation and random daytime (see Section 9) samples (AWWA 2008). Optimisation also requires the correct pH to be maintained, natural organic constituents of the water to be minimised and the distribution network to be kept free from iron discolouration problems (CIWEM 2011). Brown et al (2013) developed a decision-tree outlining re-evaluation of an existing chemical lead corrosion strategy to help regulatory agencies (i.e. US EPA) prevent exceedances of the lead standard in drinking water and the utilities to conduct a self-assessment (Figure 5).

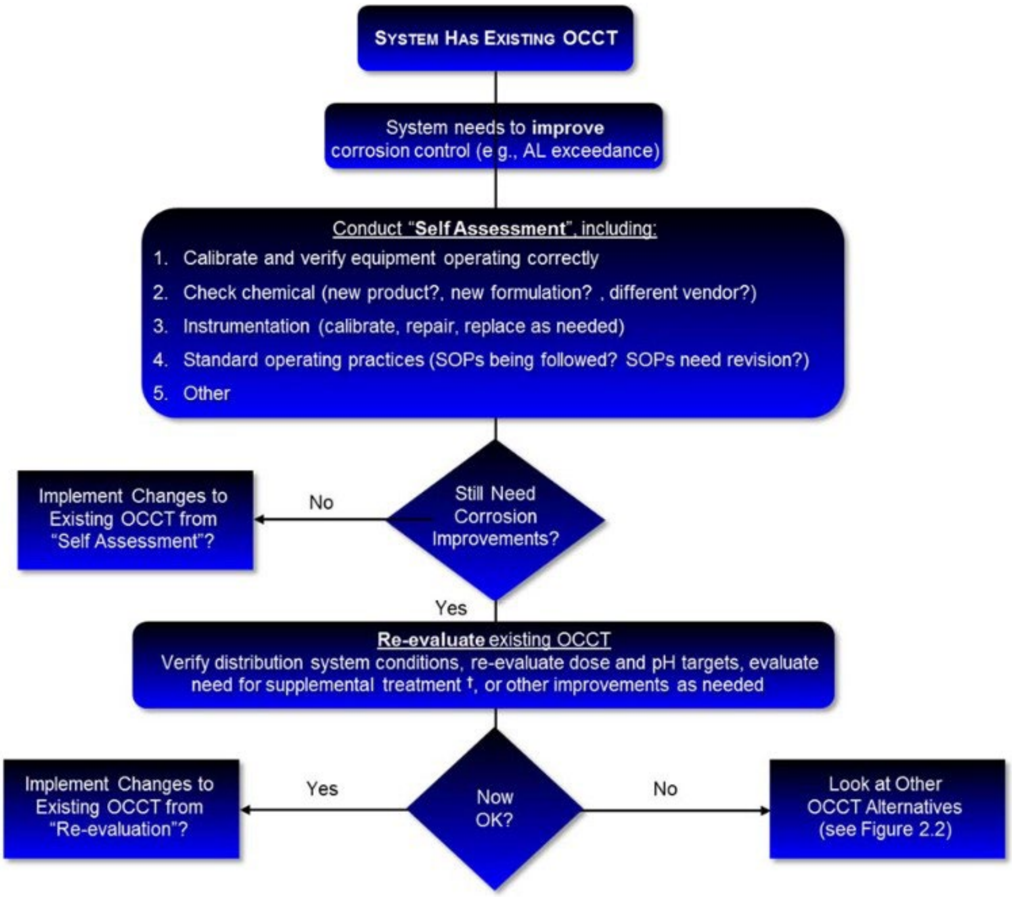


Figure 5. A decision-tree outlining re-evaluation of an existing chemical lead corrosion control strategy. AL = Action Level, OCCT = Optimized Corrosion Control Treatment †For example, removal or disinfection byproducts to improve maintenance of free chlorine residuals. Source: Brown et al 2013.

6.1.1ii Additional considerations for the use of orthophosphate

The 2015-2021 business report by Scottish Water (SW) mentioned that water leakages are considered as a high priority issue for improvement among consumers in Scotland (SW 2014). Mains water leakage (MWL) potentially leads to a direct input of phosphorus into the environment (Gooddy et al 2017). Based on orthophosphate dosing data reported by Comber et al. (2011) for the period between 2000 and 2006 (which are based on mean values for 160 UK water resource zones) and assuming equal or higher orthophosphate dosing values for the period from 2006 to 2013 when the lead standard was tightened), Gooddy et al (2017) estimated that as of 2011 phosphorus loads from MWL were in the range of 24% of sewage treatment effluent and 16% of agricultural emissions in the Thames catchment.

The phosphorus in MWL coming from orthophosphate dosing is highly bioavailable. Phosphate dosing typically achieves concentrations of phosphorus in drinking water supplies that are some 30 times higher than current U.K. standards for phosphorus in rivers; therefore, the leaking of drinking water could represent a signi□cant source of phosphorus contamination in groundwater and surface waters (Gooddy et al 2015 and literature cited therein). In the US, water utilities must weigh the use of phosphate as a corrosion inhibitor against the risk of violating regulations on phosphorus mitigation in watercourses under the Clean Water Act (US EPA 2016).

Smith and Russel (2013) reported that around 90% of the UK population is receiving phosphate dosed water and approximately 25% of this population is served by lead pipes; therefore, it was deduced that approximately 60% of the UK population is receiving and paying for phosphate dosed water without direct benefit from it. However, it is believed that it would cost more for everyone to do programmes of something else that would reduce lead levels (pers. com. Bill Byers, DWQR).

Smith and Russel (2013) also questioned the use of orthophosphate in the context of sustainability and demand for phosphorus in large quantities in the production of agricultural fertilisers and animal feeds and in key industries (e.g. food production, pharmaceuticals, cosmetics, and high-tech electronics). Phosphorus is indispensable for plant, human and animal life and plays an essential role in soil fertility and world food security (e.g. Scholz et al 2013). Its major source, i.e. phosphate rock, is a non-renewable resource; from an economic perspective, it is a low-cost commodity under normal circumstances (Scholz et al 2013). Regardless of agreeing with the potential of peak phosphorus in the short- or the long-term (Heckenmüller et al 2014),volatility in global markets could lead to dramatic increases in the price of phosphorus fertiliser, for example by 800% in 2008 caused by interruption of phosphorus mining due to an earthquake in China (Cordell and White 2011).

With increasing population size and demand for more and better food across the world as well as biofuel, more phosphorus will be required for fertilisers and animal feed, which may influence the availability and cost for other phosphorus applications e.g. orthophosphate dosing (Heckenmüller et al 2014 and literature cited therein; Scholz et al 2013). In addition to demand, the concentration of the known phosphate mines in only a few countries (such as Morrocco, USA, China and Russia) may suggest the possibility of additional issues for obtaining phosphate related to access, transport feasibility and geopolitical stability (Heckenmüller et al 2014; Scholz et al 2013; WRc 2013). It is also important to note that although phosphorus is a non- renewable resource, lead is: lead recycling including lead pipe recycling, is an effective way of producing lead today (Annex I.1).

6.1.2 Comparison of chemical control options

The advantages and disadvantages of the options identified here for the chemical control of plumbosolvency in public water supplies with respect to practical issues and biogeochemical consequences in the distribution system and the environment are summarised in Table 4 on the basis of the review of the evidence presented in Section 6.1 and Annex III.2 and III.3. The cost of these chemical control strategies, wherever known, is discussed in Section 10 in comparison with other mitigation practices for lead in drinking water and in the context of the health and social costs associated with lead exposure in the population.

Table 4a. Advantages of the different chemical lead corrosion control options for public water supply systems. See also Annexes III.2 and III. 3.

Maintaining redox with free chlorine	pH and DIC control	Orthophosphate	Zn-orthophosphate	Poly-orthophosphate Blend	Silicates
• Keeps Pb at low levels by maintaining insoluble Pb-dioxide scales. • Results in lower Pb levels compared with chloramine in systems without phosphate dosing. • Effective when (Zn or poly-) orthophosphate cannot be used for environmental reasons • Linked to slower Pb release from systems using unplasticised PVC • It results in equal or lower levels of dissolved and particulate Pb compared with chloramine, in phosphate dosed systems, when Pb-dioxide scales are already formed. References: Boyd et al. 2008, 2010; Brown et al 2013; Lytle and Schock 2005; UKWIR 2016; Vasquez et al 2006; Wang et al 2012. For a full list of references see Annex III2.2i.	• Keeps Pb at low levels due to the formation of insoluble Pb-carbonate scales. • Effective when orthophosphate cannot be used for environmental reasons References AWWA 1990; Brown et al 2015; De Mora et al 1987; US EPA 2016. For a full list of references see Annex III2.2ii.	• Keeps Pb at low levels due to the formation of insoluble Pb-phosphate scales. • Has optimal effectiveness within a range pH that is lower than that needed for pH/DIC adjustment. • Effective in terms of achieving a very high percentage of compliance with regulations in a wider range of water chemistry conditions when compared with all other methods (in the optimal pH range). • Suitable for cupro-solvency control • Not affected by changes in disinfectant, in the absence of Pb(IV) containing scales, it is • Stabilises scales. • Inhibits the formation of Pb(IV) scales (which are more soluble and sensitive to redo changes). References: AWWA 1990; Boyd et al 2010; 2008; Hayes et al 2006; 2008; Hayes 2010; Comber et al 2011; Schock et al 1996; UKWIR 2012; 2016. For a full list of references see Annex III3.4i.	• Keeps Pb at low levels due to the formation of insoluble Pb-phosphate scales. • Reduces Pb leaching from brass faucets. • Provides better corrosion protection for cement at low alkalinity/hardness/ pH conditions. References: US EPA 2016. For a full list of references see Annex III3.4ii.	• Reduces iron and manganese oxidation at high pH, thus minimising the risk of discolouration, staining and scaling. • Helps form passivating film depending on Ca concentration and P speciation. References: Cantor et al 2000. For a full list of references see Annex III3.4ii.	• Raise pH thus reducing Pb dissolution. • Reduce Pb and Cu levels in first draw/ first litre tap water samples. • Sequester iron and manganese, thus reducing black or red water problems. References: Schock et al 1996; US EPA 2016. For a full list of references see Annex III3.4iii.

Table 4b. Disadvantage of the different chemical lead corrosion control strategies. See also See also Annexes III.2 and III. 3.

Maintenance of redox with free chlorine	pH and DIC control	Orthophosphate	Zn-orthophosphate	Blend of Poly-orthophosphate	Silicates
• Not normally considered a corrosion control strategy. • Optimal pH to reducing Pb corrosion may be sub-optimal for chlorine disinfection and formation of disinfection by products (DBP). • Health impacts due to DBP challenges. • Not compatible with switch to chloramines if Pb-dioxide scales have been formed. References: Brown et al 2015; Boyd et al 2008; Edwards and Dudi 2004; Lytle and Schock 2005; Schock et al 1996; US EPA 2016. For a full list of references see Annex III2.2i.	• Optimal pH to reducing Pb corrosion may be sub-optimal for chlorine disinfection. • Raising pH and DIC to reduce Pb corrosion may cause calcium carbonate precipitation. • Increasing the pH enhances iron and manganese oxidation leading to red of black water. • Larger doses of pH adjustment chemicals (lime, caustic soda, soda ash) than for achieving optimal pH for orthophosphate. • Costly. • Variable and sensitive to temperature and water chemistry fluctuations. References: AWWA 1990; Brown et al 2015; Schock et al 1996. For a full list of references see Annex III2.2ii.	• Can cause blue water in galvanic systems. • Sensitive to pH (stagnation) and temperature fluctuations. • Compromised by change in redox by chloramines, depending on the presence of formerly formed Pb-dioxide scales. • Requires minimisation of natural organic matter. • Requires the network to be free from iron discolouration problems. • Effectiveness relies on the composition of scales before dosing. • Requires extensive treatment at sewage water treatment plants to prevent pollution. • May be released to the environment through water mains leakage. • Costly. • Non-renewable resource. References: AWWA 1990; 2005; 2008; Gooddy et al 2015; 2017; Switzer et al 2006; WRC 1992 cited in Potter 1997. For a full list of references see Annex III3.4i.	• It has a lower effectiveness than orthophosphate -It is linked to high turbidity. • It does not provide additional copper control compared with orthophosphate. References: AWWA 1990. For a full list of references see Annex III3.4ii.	• May increase lead leaching; scale formation is different from that with orthophosphate dosing. References: Cantor et al., 2000. For a full list of references see Annex III3.4ii.	• It requires a pre-existing film to allow binding of silicates. • It requires large doses to be effective, thus it may be very costly. • Mechanism of action remains unclear. References: US EPA 2016. For a full list of references see Annex III3.4iii.

6.2 Lead pipe replacement options and renovation techniques

6.2.1 Lead-free approaches

Total lead pipe replacement (Total-LPR) removes the source of exposure to lead in drinking water by replacing all communication and supply pipes in the utility's and the homeowners' side. Total-LPR has been characterised as the most sustainable mitigation practice for lead in drinking water for the UK (Gooddy et al 2017; Hayes 2010; Potter 1997) and the USA (e.g. AWWA 2008; Brown et al 2015).

Atenstaedt (2016) suggested that water companies in England and Wales should fund the cost of total pipe replacement on the grounds that local authorities in the UK are already too financially stretched to award sufficient grants and the cost of replacing lead pipes and premise plumbing is too high for many families. Smith and Russel (2013) mentioned discussions taking place on the transfer of ownership of the supply pipes to the water companies in the UK (England and Wales), similar to the recent transfer of private sewers and lateral drains; however, this may be a slow process. The US EPA Scientific Advisory Board (SAB) (2011), on the effectiveness of Total-LPR, recommended that the priority strategy for reducing lead in drinking water should be Total-LPR, and that managers of water utilities should strive to fully remove lead pipes by budgeting for feasible annual removal goals.

Renovation (or rehabilitation) of existing lead pipes has also been considered as a lead-free approach. It involves installing a plastic lining or epoxy coating from the mains or communication pipes to the consumers' tap which acts as a barrier between drinking water and leaded pipes and premise plumbing (Boyd et al 2000; Kirmeyer 2000).

6.2.2 Partial lead pipe replacement (Partial-LPR)

Partial lead pipe replacement (Partial-LPR) refers to removing the lead pipes only in the utility's side, whereas lead pipes in the homeowner's side remain in place constituting a source of exposure to lead in drinking water (AWWA 2008; Hayes 2010; Schock et al 2014). The effectiveness of Partial-LPR depends on homeowners' willingness or ability to pay for the LPR in their side and within their premises and on the type of distribution materials (Annex III.1).

Effectiveness issues with Partial-LRP due to lead supply pipes and lead-bearing premise plumbing can be illustrated in a range of cases. For example, in the Hague, following Partial-LPR, 23% of random daytime (RDT) (see Section 9) samples were found to exceed 10 µg/L due to lead in premise piping and plumbing such as faucets and brass fittings (Van Dongen et al 2008 cited in Hayes 2010). Likewise, in the UK grant uptake for LPR by homeowners has been historically low: only around 1.5% of households replaced their lead pipes every year in the early 1990s (WRC 1992 cited in Potter 1997). With this rate of replacement and assuming a rate of 6,000 properties being demolished every year, half of which having lead communication and supply pipes, it was estimated that 38% of homes would still have lead supply pipes by the year 2015 in the UK (CRC 1995 cited in Potter 1997). The House of Lords Select Committee (1996 cited in Potter 1997) commented on this: "At the present rate of voluntary replacement it would take another fifty to sixty years to remove all domestic lead piping in this country".

Galvanic action can also play an important role in lead release. Partial-LPR has largely contributed to high tap lead levels due to galvanic corrosion (see Annex III.1.2).

6.2.3 Feasibility versus effectiveness of lead pipe replacement and pipe rehabilitation

Over time Total-LPR will ultimately result in lead free drinking water. However, for most public systems Total-LPR can become prohibitively expensive (Section 10). Total-LPR is also complicated by logistical and legal constraints, i.e. the disruption of traffic and ground conditions, the need to shut water off for days to make repairs, and the private ownership of premise piping and plumbing. For example, where government-backed through grants total removal of lead pipes has been attempted, cooperation of householders has been poor despite the availability of grants, because of the disruption and inconvenience involved and the cost incurred to homeowners in the case of means-tested grants (Hayes 2010; Potter 1997).

Irrespective of the cost of replacement (Section 10) and the rate of uptake of grants by homeowners, it has been observed that the rate of Total-LPR is faster in countries where orthophosphate dosing is not the preferred alternative because of environmental or country-specific reasons (COST Action 637 2010). This may explain the higher prevalence of lead communication and supply pipes in countries and areas where utilities have prioritised orthophosphate dosing over other mitigation practices such as in the UK, as noted by Hayes (2010).

Extensive surveys comparing the effectiveness of Total-LPR versus orthophosphate dosing to reduce lead in drinking water have only been carried out in the USA, such as the study by AWWA (2008) comparing water lead levels from 11 different utilities in the USA and the UK in the context of LPR and orthophosphate dosing. AWWA (2008) accounted for field observations, optimisation procedures and perceptions of effectiveness from the utilities for a prolonged period of time, the shortest period referring to data from Thames Water (2002-2004). AWWA (2008) concluded that:

- The major contributor to lead concentrations at tap water was lead service lines
- The major contributor to non-compliances with the lead standard[11] in standing, first-litre samples (which represent the water that has been exposed to the faucet and premise system only) were the premise piping and the faucet water.
- Partial-LPR alone did not improve compliance with the standard for lead in drinking water; besides elevated lead levels were observed at tap after partial replacement.
- Total-LPR reduced the overall mass of lead measured at the tap and improved compliance at individual residential sites in the long-term but high levels were observed after total replacement for a period that was utility - and supply zone - specific.
- In systems with Partial-LPR and optimised corrosion treatment (e.g. orthophosphate), corrosion control treatment reduced the mass of lead measured at the tap in random daytime, overnight stagnation and fully flushed samples.
- Utilities with optimised lead corrosion control treatment and Partial-LPR still experienced lead levels at or above the standard for lead in drinking water. The recommendation put forward for these utilities was to re-evaluate their chemical control strategies.

[11] In the USA the regulations dictate that supplies must comply with the Lead-Copper Rule, according to which 90% of samples should be below the action level of 15 µg/l.

Brown et al (2015) have suggested that Total-LRP is a sustainable solution only for small, rural, domestic water supplies. However, this may not always be the case. For example, it has been hypothesised that lead from service lines and other sources in the distribution system (small or large) can migrate over the years and be deposited on premise plumbing between the lead sources and the customer's tap (AWWA 2008; Brown et al 2015). The Madison, WIS case, where lead levels remained high in some households following Total-LPR provides an example of this potential source of lead (Schock et al 2014; see Annex III.1.2 this report). Although further research is needed to elucidate the mechanisms of this so-called lead "seeding", it practically means that even if all of the original lead sources are replaced (brass faucets, soldered copper piping, and lead service lines), the lead levels can still be high due to lead migrating from the lead scale deposits on the remaining plumbing. Brown et al (2015) recommended that in these cases, all the plumbing, not just the original lead-containing plumbing, needs to be replaced, thereby adding to the cost and complexity of this approach to lead control, even in small systems.

A surprising advantage of Total-LPR has been revealed by Smith and Russel (2013). It was argued that the lead pipes removed from the ground, can then be recycled to partially offset the cost of lead pipe replacement; see also Annex I.1. As for the gains of recycling the communication pipes, they may be much lower than those for Total-LPR depending on the length of communication pipes (see Annex III.1).

Lead rehabilitation (e.g. lining) has been previously tested (Boyd et al 2000; Kirmeyer 2000; UKWIR 2012), but has yet to be widely applied. Smith and Russel (2013) and UKWIR (2012) have reported a lower cost for this technology than for any of the lead pipe replacement techniques due to fewer excavations (only two) needed to complete the lining than to carry out the replacement. The implication of this is that the rehabilitation within a supply zone can be completed in a much shorter period than that needed for open trench replacement, with much less traffic disruptions and risk of damage to other buried utilities and regardless of type of soil and surface conditions (Hayes 2010; Kirmeyer 2000).

However, extensive studies have also shown the shortcomings of lead pipe rehabilitation techniques. These include reductions in pipe diameter, restrictions of use in pipes with tight loops and bends (as with the slip lining technology) and extended interruption in water service due to the time (up to 24 hours) required for the materials to cure in place (as in the case of the epoxy lining technique (Kirmeyer 2000). Hayes (2010) reflecting the opinion the network of experts of the Cost Action 637 programme also reports the unknown degree of success in achieving lead- free drinking water as an additional disadvantage. Gooddy et al (2017) argued that the timescales involved in widespread lining or replacement may ultimately make these actions an unlikely solution to mains water leakages of phosphorus in anything but the long-term.

A summary of the advantage and disadvantages of total and partial-LPR in the context of public exposure to lead is presented in Table 5 on the basis of the evidence presented in Annex III and the discussion provided in this Section.

Table 5. Advantages and disadvantages of options for eliminating leaded materials in the water distribution system. References as in Section 6.2 and Annex III.1.

	Total-LPR	Partial-LPR	Epoxy lining
Advantages	• Removes the major source of exposure to lead in drinking water. • Lead-free approach. • Some practices of replacement are less costly than others. • Potential for recycling, if lead pipes removed from the ground.	• Cost-effective when used in combination with optimised corrosion control treatment. • Removes a great part of the major source to lead exposure, i.e. lead service pipes.	• Removes the major source of exposure to lead in drinking water. • Less traffic disruption. • Low risk of damage of other buried utilities. • It is suitable for all soils and surface site conditions.
Disadvantages	• Prohibitive cost of some techniques. • Requires concurrent removal of plumbing components such as faucets, soldered copper piping, brass fittings, which is logistically, practically, and economically difficult. • Assumes engagements from homeowners. • Very slow grant uptake, where grants are in place. • (In some cases) May require replacement of all piping and premise plumbing, even non-lead components, to eliminate the potential effect of lead "seeding". • Disruptive approach in terms of traffic, ground conditions risk of damage of property.	• Not 100% effective • Not a Pb-free approach. • Does not remove the major cause of non-compliances, which is premise plumbing and piping. • Requires chemical lead corrosion control. See also disadvantages of corrosion control strategies in Table 4b. • May cause galvanic action across dissimilar metals. • May cause continuing issue of transport of particulate lead into renewed pipework.	• Long-term timescales of application on all systems. • May reduce pipe diameter • Uncertain or little evidence effectiveness due to small-scale application.

6.3 Flushing

The effect of flushing on lead concentrations in tap water is explicitly discussed in Annex III.3.3. Tap water after flushing until tap water is colder is expected to be lead-free because it represents the water that is in contact with the lead-free water mains after it sits stagnant, e.g. overnight. In public networks where partial-LPR is common or in the case of small, domestic supplies, consumers have been given advise to flush tap water for 2-3 minutes as an effective way of minimising exposure to lead (NSW HEALTH 2014; SW n.d.).

The research reviewed in Annex III.3.3 has shown that, in many cases, flushed samples may contain high or higher lead levels than non-flushed samples in systems with Partial-LPR due to particulate lead release compared with minimisation of dissolved lead (e.g. Triantafyllidou et al 2007); see Annex III.3.2 for a detailed review. Many studies reviewed in Annex III.3.3 have also shown that in systems with Partial-LPR, flushing as a lead reduction strategy should account for:

- Optimal flushing, i.e. the duration of flushing at the optimal flow needed to reduce sustainably exposure to particulate lead. The duration of the remedial effect of flushing varies from one day to three months (e.g. Brown and Cornwell 2015).
- The persistence of the remedial effect after optimal flushing, which will determine the frequency of repeating optimal flushing.
- Lead-containing premise plumbing components, which have been shown to cause variation in the remedial effectiveness of flushing (e.g. Brown and Cornwell 2015).

6.4 Educational interventions

Stand-alone educational interventions do not appear to be effective in lowering blood lead levels. For example, a German randomised control trial examined the effect of instructing women with low and elevated blood levels to minimise exposure to lead by flushing lead-contaminated tap water or by excluding exposure to lead in tap water by using bottled water. "Minimizers" could lower their blood lead levels by about 21% of the initial value; "excluders" by about 37%. Interestingly, the majority of these women judged neither minimizing nor excluding tap water as practicable health preventive behaviour pattern in the long run. Most importantly, despite the robust design of the study, the effect of minimising or excluding the use of lead-contaminated tap water on blood lead levels was not significant (Fertmann et al 2004; Pfadenauer et al 2016).

Two American randomised control studies examined the effectiveness of educational interventions targeting mothers' behaviour in order to reduce blood lead levels in their children. Both studies focused on population of Afro-American and Hispanic origin in socio-economically deprived areas. One study examined the effect of peer health educators (Jordan et al 2003): the educational curriculum included information on sources of lead (e.g. water, dust, paint, soil, and risks from home repairs), health consequences of lead exposure, and lead exposure reduction strategies, including safe use of water and nutritional guidelines. The other study examined the effect of individually tailored health education targeted to pregnant women (Dugbatey et al 2005)[12]: the interventions, tailored for each woman on the basis of responses to a survey and environmental measurements, included case management with hands-on instruction on property maintenance, cleaning and nutritional guidelines. Both studies showed that the specific educational interventions directed at mothers were no more effective than standard educational interventions (i.e. providing information) in lowering children's blood lead levels.

The lack of effectiveness of educational interventions is consistent with the lack of effectiveness of interventions to reduce exposure to lead in paint and soils (Yeoh et al 2012). The ineffectiveness of interventions to reduce lead in drinking water, such as flushing, may be related to the issues discussed in Section 6.3. However, education, and flushing or use of bottle water, has been considered critical as a co-intervention alongside chemical mitigation and lead pipe replacement practices (Pfadenauer et al 2016); see also Section 6.1 and 6.2.

6.5 Small water supplies

A detailed account of the chemical lead corrosion control strategies and best practices referring to small supplies has been provided by Bower and Hayes (2016). These include point of use (POU) and point of entry (POE) approaches. It is generally accepted that POU barriers can reduce lead exposure at the tap but require careful monitoring of their performance and their effectiveness depends on water chemistry such as pH (Bower and Hayes 2010 and literature cited therein). Removal at POU can be achieved by raising pH, with uncertain success. A GAC filter can be used to remove the lead ions. Removal at source or POE could include the use of chemical filters or adsorptive filters (i.e. GAC and granular ferric oxide/hydroxide); ion exchange; and reverse osmosis.

There is no extensive evidence on the statistics of the risk of lead exceedances or the current factors influencing the lead chemistry in small supply systems relying on domestic treatment. Preliminary studies in private water supply systems showed that there is widespread lead contamination due to lead plumbing and release of particulate lead in the system, which may remain unaccounted for by regulatory sampling. For example, in the USA a random subset of samples selected to quantify particulate lead indicated that, on average, 47% of lead in the first draw samples was in the particulate form, although the occurrence was highly variable (Pieper et al 2015). Flushing the tap reduced lead below 15 µg/L for most systems, i.e. below the standard for lead in drinking water in the USA; however, some systems experienced an increase, perhaps attributable to particulate lead or lead-bearing components upstream of the faucet e.g., valves, pumps (Pieper et al 2015).

In a study of domestic supplies in New South Wales, Harvey et al (2016) demonstrated that commercially available plumbing products posed an appreciable source of exposure to lead in drinking water through exceedances of 10 µg/L, which were prolonged over time. Flushing the tap water for 2-3 minutes was also found to be ineffective in reducing water lead levels in some domestic supplies and unsustainable in terms of water availability (Harvey et al 2016). For example, under circumstances of water scarcity, such as in domestic, rural supplies in New South Wales, Australia, it may be impractical and unsustainable to flush the system for a prolonged period of time on a regular basis (Harvey et al 2016).

7.0 Indicators of lead exposure in drinking water

7.1 Selecting the appropriate biomarkers

Lead biomarkers are reviewed in Annex IV.1. Commonly used biomarkers include samples from: blood, plasma, bones, teeth, urine and faeces. Selecting the appropriate biomarker depends on (i) the purpose of monitoring lead exposure, e.g. identification of health effect or exposure levels to detect individuals above levels or concern or action; (ii) the timescales of effects in question, e.g. past versus recent exposures; and (iii) the context of exposure, e.g. low and chronic vs high and short-term (Bergdahl and Skerfving 2008).

Bergdahl and Skerfving (2008) suggested suitable biomarkers in terms of reliability of results to detect exposure to lead for three different purposes: (i) epidemiological studies of the relation between lead exposure and health; (ii) studies on organic (tetraethyl) lead exposure, past or current; and (iii) studies to identify exposure above levels of concern. Bergdahl and Skerfving (2008) suggested that:

- Blood is the most suitable biomarker for epidemiological

[12] The women were predominantly poor and of African-American, Hispanic, Asian, and Caucasian backgrounds. The interventions, tailored for each woman on the basis of responses to a survey and environmental measurements, included case management with hands-on instruction on cleaning techniques, property maintenance, hygiene, and nutrition to reduce exposure of newborns to lead. It was hypothesized that the probability of lead poisoning (blood lead levels greater than 10 µg/dL) would be reduced among mothers who received the interventions compared with those who received only printed educational material.

studies on reversible but adverse health effects of short-term, low lead exposure, such as impairment of blood function, and decreased haemoglobin synthesis and absorption of minerals in the gut.

- Bone or teeth are suitable to detect past exposures to lead.
- Faeces and blood are suitable to detect current (gastrointestinal) exposure.
- Urine is the best indicator to detect past or current exposure to organic lead as in petrol.
- Combined use of blood and urine or plasma/serum is suitable for studies on acute (occupational) exposure.
- Blood is the best indicator to detect exposure above levels of concern may be blood.

Having said that, the major disadvantages of blood lead as a biomarker of low lead exposure is that it is not indicative of the source of exposure to lead, unless isotope signalling is used (Barbosa et al 2005; Bergdahl and Skerfving 2008; Moffat 1989; Ramsay 2003). For this reason, blood is not considered as a useful indicator of residential exposure in the UK; instead blood lead is generally accepted and used as indicator of occupational exposure to lead (Health and Safety Executive 2012). Blood lead has been considered as unreliable for blood lead screening in children and the general population in the UK on the grounds that the much lower levels of exposure expected to occur need greater analytical accuracy and precision than presently available in UK laboratories (UK National Screening Committee 2014); see also Annex I.6.4. Ironically, and illustrative of the variety of perspectives, blood lead monitoring in lead workers has been found to have important shortcomings in that it shows a poor response to changes in exposure at high lead levels (Bergdahl and Skerfving 2008 and literature cited therein).

To sum up, and as explicitly reported in the studies reviewed in Annex IV.1, almost all lead biomarkers lack systematic data on variation within and between individuals. However, the blood lead's major advantage as a lead biomarker is its extensive use in single, cross-sectional and prospective studies in a variety of contexts which are reviewed in Section 7.3 and Annex IV.2.

7.2 Problems in identifying the relationship between tap water and lead exposure

Research and experience has shown that the major problems of linking lead exposure to changes in lead levels in drinking water refer to (Brown and Margolis 2012; Mushak 2011; Needleman and Gee 2013; Quinn and Sherlock 1990; Potter 1997):

- Designing a research sampling programme enabling lead in water to be reliably associated to changes in lead levels in the body. For example, a robust sampling programme should include (i) a sufficient number of participants, running over a long period time and including sufficient "control" data (i.e. blood lead under background exposure to lead); (ii) concurrent monitoring of the blood lead in the population and the levels of lead in drinking water; and (iii) sampling from a range of water lead levels and levels of other environmental sources of lead, and from a range of ages, social circumstances and habits/professions in the population
- Selecting a suitable lead biomarker specific to low levels of exposure and population subgroups and specific to the time elapsed since exposure (past or present); see also Section 7.1
- Perceptions in the general public of lead as an occupational hazard that causes immediate, acute clinical symptoms and not as a cumulative poison that initially causes sub-clinical (not obvious) neurodevelopmental and biochemical effects (see also Annex I.3 and I.4). These perceptions preclude seeking medical advice and allowing the detection of elevated water lead.
- Invisibility of factors influencing lead leaching from plumbing material (e.g. low water pH, galvanic corrosion in the presence of jointed copper-lead pipes, water stagnation time, temperature fluctuations, change in disinfection products; and presence lead solder; see Section 6.0 and Annex III). These factors, under certain circumstances, have the potential to compromise the effectiveness of orthophosphate dosing, thus resulting to lead exposure in the general public (see section 6.1.1).
- Reliance of Total-LPR policies on householders' willingness or ability to pay or take action to modernise lead-bearing plumbing in older premises. This resulting in implementation gaps between goals (such as lead-free water) and reality (lead exposure). An additional issue is that grants for supply pipe replacement are discretionary and depend on the granting organisations' budget. As a result the process may be too slow to enable a swift and robust evaluation of its effect on lead exposure; see also Section 6.2.
- Regulatory sampling's ability to account for the range of fluctuations in lead leaching in the public network and in small (private) water supplies and to capture non-compliances with the standard for lead in drinking water (see Section 9). If a proportion of non-compliances remain undetected, then levels of lead exposure in the population may be severely underestimated.

Robust sampling design is essential to enable any potential association between water lead and the levels of blood lead or any other biomarker. However, available data are often unreliable, even when there is a statutory requirement for their collection. The reasons are illustrated below.

1. **Lack of blood lead level or any other biomarker measurements for all sensitive age groups.** In addition, it has been shown that single studies are insufficient to clarify any of age-related and source type- and issues (e.g. Lacey et al 1985; Pfadenauer et al 2016). For example:

   (i) In the UK there is no formal screening of blood lead levels in children or adults in the general population in relation to residential lead exposure. Any available evidence for children is based cases of clinically diagnosed lead poisoning, usually due to paint ingestion, but the burden of sub-clinical effects on children goes largely unnoticed (British Paediatric Surveillance Unit-BPSU 2013). Besides, even if water was source of lead poisoning, it remains uncertain whether water has always been tested as a source.

   (ii) In the USA, children's blood lead measurement is part of public health policy but screening is targeted to children aged 1 to 6 years, who are at highest risk for exposure to lead paint and lead dust hazards (CDC 2012); relatively little data is available for bottle-fed children aged less than 9 months old, who are most vulnerable to lead exposure through water (e.g. Edwards et al 2009; Shannon et al 1992).

2. **Lack of blood lead data in relation to all relevant sources in an individual's environment.** In some cases, mitigation of water lead levels only slightly reduced blood lead, if high levels of lead have been stored in bone because of endogenous lead release (Gwiazda et al 2005; Rust et al 1999). Fleming et al (1999) developed a kinetic model which suggests that a smelter worker with a compact bone concentration of 100 µg/g can expect a continuous endogenous contribution to blood lead of 16 µg/dL. On the other hand, a pregnant woman with a compact bone lead concentration of 50 µg/g can expect a contribution of 8µg/dL in blood-lead from endogenous lead exposure, without accounting for the increased rate of bone turnover associated with pregnancy (Fleming et al 1999). This practically means that in the absence of current lead exposure, a pregnant woman exposed to lead in the past can still be a source of lead exposure to the foetus. Such findings show that lead in drinking water must be disentangled from other potential sources and pathways of exposure to lead in the residential environment to enable reliable estimates of the water lead-blood lead relationship.

3. **Lack of water lead level measurements in specific residential environments of risk groups such as schools, care homes or small private water supplies in rural areas.** In the UK, and under the general provisions of the DWD, domestic supplies serving fewer than 50 people are not tested for lead or any other contaminants, unless the homeowners or a risk assessment has indicated the need of sampling; private water supplies that serve a public activity such as schools and hospital are tested but at very low annual frequencies (usually up to 2 -3 times a year). Therefore it is impossible to assess the impact of lead exposure in, usually old, rural premises.

4. **Sampling size and frequency for lead in drinking water.** Fluctuations in lead levels in the distribution system may arise as a result of detachment of particulate lead due to changes in flow; temperature variation; galvanic corrosion; change in the disinfectant used; different duration of stagnation and flushing before consumption or sampling; these factors are reviewed in Annex III.2. Pocock (1980) argued that a single regulatory sample cannot provide a reliable estimate of an individual's exposure to water lead. In the UK, at the specified frequencies under the provisions of DWD and with random daytime (RDT) sampling (see Section 9: Table 8), it is difficult to account for lead fluctuations on the supply zone scale (from mains to tap) (Dore 2015); see also Section 9.

5. **Sampling and analytical method for the measurement of lead in drinking water.** Lead in drinking water is not always in dissolved form, as usually assumed by regulations; this may significantly underestimate lead concentrations in tap water (Edwards and Dudi 2004). Particulate lead may adhere to the plastic sampling containers, and be "missed" when aliquots are taken for measurement (Triantafyllidou et al 2007). In the USA, the most recent sampling guidance for schools (US EPA 2006 cited in Trantafyllidou 2011) was to "induce a small (e.g., pencil-sized) steady flow of water from the outlet". These instructions translated to an unrealistically low flow rate of less than 1 L/min, which is likely to miss 90% of lead spikes due to particulate lead release (Edwards 2005 cited in Trantafyllidou 2011).

6. **Individual variations in the levels of water consumption or lead intake remain largely unaccounted by regulations.** Lacey et al (1985) suggested the adoption of a percentile approach to lead exposure. They argued that a percentile, e.g. 10% of the population exceeding a given threshold for blood lead or lead intake or drinking water above the standard for lead in drinking water, has the potential to be a better indicator for individual variation than an arithmetic or geometric mean or a single value from an area. The percentile approach as indicator of the risk of exposure has been adopted in the USA and Canada (Annex I.6.1).

It is also interesting to note that lead-free policies as in the case of lead-free petrol and paint and tightening of the standard for lead in drinking water co-occurred and researchers have always pointed out that it was difficult to separate the effects of each policy on blood lead levels. This caveat is further discussed in Section 8.

7.3 Water lead –blood lead relationships

The relationship between blood lead levels and drinking water lead levels is explicitly reviewed in Annex IV.2. The findings are summarised in Table 6.

Table 6. Link between water lead and blood lead levels. Detailed description of studies is given in Annex IV

Area	Relationship	Reference
Glasgow, Scotland	Blood $lead_{bottle\text{-}fed\ infants}$ (µg/dl)=5.5 + 3.3 $\sqrt[3]{\text{water lead}}$ (µg/L) R^2=0.23, p<0.01	Quinn and Sherlock 1990
Ayr, Scotland	Blood $lead_{mothers}$ (µg/dl)=5.6 + 2.62 $\sqrt[3]{\text{water lead}}$ (µg/L) R^2=0.65, p<0.001	Sherlock et al 1984
Edinburgh, Scotland	Blood $lead_{children}$-µg/dl= $(53.26 +1.03 \text{ (water lead-µg/L)} + 0.0381 \text{ (dust lead-µg/g)})^{0.5}$	Laxen et al 1987
England, UK	Blood lead(µg/dl) = 0.699 + 0.0003 * water lead (µg/dl)	Pocock et al 1983
Vosgian Mountains, France	When Water lead ≤20 µg/L then Blood lead‡$_{Men}$= 15 µg/dl Blood lead‡$_{Women}$=11 µg/dl	Bonnefoy et al 1985
Wales, UK	When Water lead increases by 60 µg/L then Blood lead increases by 5.5 µg/dl	Elwood et al 1984
Ste-Agathe-des Monts, Canada	Blood lead (µg/dl)= 10+ 7 x water lead x water consumption, R^2=0.25	Savard 1992
Rochester, New York, USA	When Water lead increases from 0.5 µg/L to 15 µg/L then Blood $lead_{children}$ increases by 1.6 µg/dl	Lanphear et al 1998
Rochester, New York, USA	When Water lead is >5 µg/L then Blood lead# =8.4 µg/dl and 36.9% of children had Blood lead >10 µg/dl	Lanphear et al 2002
Hamburg, Germany	When Water lead is >5 µg/L then Blood lead# = 31 µg/dl When Water lead is <5 µg/L then Blood lead# = 24 µg/dl	Fertmann et al 2004
Wayne County, North Carolina, USA	Number of records=7,270 before and after change in disinfection from chlorine to chloramine under zinc-orthophosphate dosing¥: Before: Blood lead‡= 4.19 µg/dL After: Blood lead‡= 4.93 µg/dL Blood Lead Levels (BLL) in people residing in housing built before 1926> in 1926–1950> in 1951–1975	Miranda et al 2007

‡Average
#Geometric mean
¥Miranda et al 2007 have not reported the water lead levels

The review revealed that exposure to lead in drinking water due to lead leaching from piping was in many occasions, including in Scotland, associated with significantly greater blood lead levels than exposure to lead from car or industrial emissions. In addition, blood lead levels responded to changes (increases or decreases) in water lead levels in many occasions. The relationship of blood lead to water lead was also found to depend on exposure to other sources of lead via food and other sources in the residential environment and study-specific factors (e.g. number of individuals studied, age, range of water lead levels, age of groups included, and rates of water consumption- water intake, season). It must be also noted that the water lead – blood lead relationship has not always been described in the succinct form of a simple equation; many studies used complex multivariate statistical techniques. For this reason only a part of the evidence reviewed in Annex IV.2 could be included in Table 6.

The key conclusion from this evidence is that the relationship between water lead and blood lead is not linear. This may be explained as an effect of a saturation of lead-binding sites in red blood cells (Bergdahl et al 1998). Scottish studies have shown that it may be curvilinear (Figure 6). This means that large reductions in water lead levels (e.g. from 25 to 10 μg/L) may have a small impact on blood lead levels; conversely, large increases in blood lead levels can occur with very small increases in the range of low water lead levels as demonstrated by the studies of Lanphear et al (1998) and Edwards et al (2009). However, linearity can be assumed when the focus is on predicting risk from consumption of lead-contaminated water (e.g. Pocock et al 1983); see also Table 6. For example, the US EPA's Water Criteria Document (US EPA 1986 cited in Rosen et al 2017), published formulae for calculating blood lead levels in children and adults from water lead levels assuming linearity:

Children: Blood lead levels = 0:16 * daily lead intake from water

Adults: Blood lead levels = 0:06 * daily lead intake of lead from water.

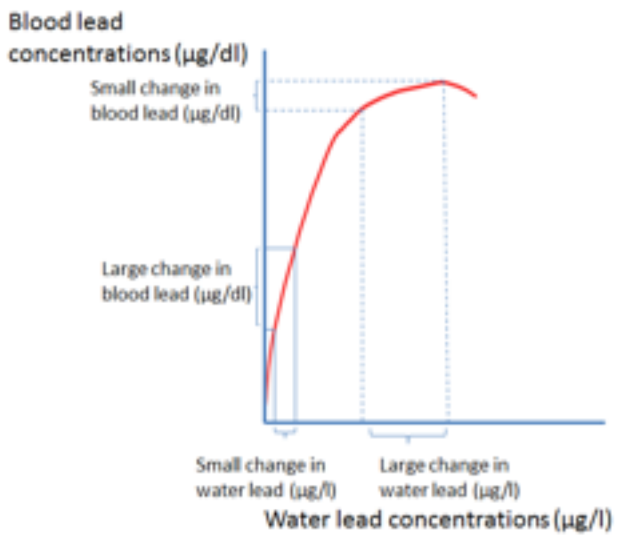


Figure 6. An example of a curvilinear relationship between water lead and blood lead levels as indicated by Scottish studies. Adapted from: Moore et al 1985.

7.4 Key points

- To detect a significant relationship between lead in drinking water and low blood lead levels one has to: (i) design studies with a sufficient number of participants, running over a long period time and including sufficient "control" data (i.e. blood lead under background exposure to lead); (ii) enable concurrent monitoring of the blood lead in the population and the levels of lead in drinking water; and (iii) account for a range of water lead levels and levels of other environmental sources of lead, and a range of ages, social circumstances and habits/ professions in the population.
- Blood lead has more advantages than other biomarkers including ease of analysis, low detection limit, relatively low cost, extensive evidence from cross-Sectional or prospective (longitudinal) studies and use in the regulation of lead exposure of the general public in many countries.
- A strong, non-linear association between lead in water and lead in blood has been documented through scientific research in the past 50 years. Blood lead levels may be disproportionally high for a small percentage of the population and for small changes in water lead levels.

8.0 Declines in lead exposure due to lead regulation

8.1 Blood lead declines due to phasing lead out of petrol, paint and food cans

Phasing lead out of petrol and paint resulted in declines in lead pollution in outdoor and indoor air (Annex I.1), and in a significant fall in blood lead levels in children and the general population worldwide (DOE 1990; Ducoffre et al 1990; Meyer et al 2008; Pirkle et al 1994; Strömberg et al 2008; Thomas et al 1999; WHO-ENHIS 2009). This led Landrigan (2002) to characterise the almost universal phase out of lead in petrol (in the time of his publication) as a success story in the history of lead regulation. Lead control regulations in relation to other sources of exposure to lead, such as paint have also contributed to declines in water lead levels observed in more recent decades (Luo et al 2003; Muntner et al 2005; Stromberg et al 2008,). Also the GBD studies (Section 4.6 and 5.4) indicated remarkable declines in lead exposure since the 1990s.

The declines in blood lead levels were remarkable. For example, in the USA the National Health and Nutrition Examination Survey for 1988–91 (NHANES III) showed that public health efforts to reduce lead in petrol and soldered cans containing food were associated with a 78% decrease in blood lead levels in the US population compared with the 1976–80 period (Pirkle et al 1994). In the 1991–1994 NHANES, the overall prevalence of blood lead levels equal to or above 10 μg/dL was 2.2% but decreased to 0.7% by the 1999–2002 survey (Jones et al 2009). Overall, the geometric mean (GM) decreased significantly ($p<0.05$; two-tailed t-test) from 2.3 $\mu g/dL$ to 1.6 $\mu g/dL$ during the same time period. Despite the declines, social disparities in risk for exposure have persisted over time. For example, the mean blood lead levels for non-Hispanic black children (1.9 *μg*/dL) were significantly higher (36%) than that of white children (1.4 *μg*/dL) during 2007–2008 (Jones et al 2009). It remains uncertain what the contribution of water lead regulations was in bringing about these remarkable declines (Brown and Margollis 2012).

In the UK[13], from the early 1970s to the mid-1980s there appears to have been a long-term downward trend in blood lead of around 4% per year (DOE 1988). A survey conducted in mid-1990s with similar methodology and objectives indicated that blood lead levels had fallen by a factor of 2.6–3.0 in adults and 3.6–5.0 in children since the 1984–87 period (Delves et al 1996), with geometric mean blood lead levels in different age groups being in the range of 2 µg/dl (children) to 4.6 µg/dl (over 65 years of age).

The beneficial effects of a switch to unleaded petrol were also shown by a series of measurements of levels of blood in children living in an urban environment in Sweden: the geometric mean lead level was 5.8 µg/dl in 1978–1982, 3.4 µg/dl in 1989, 2.3 µg/dl in 1993 and below 1.5 µg/dl since 2005 (Strömberg et al 2008). The mean level of lead in children's blood in Germany has fallen by more than 50% since 1995 (WHO-ENHIS 2009). In general, following the phasing out of lead in petrol, the rate of decline of blood lead levels continued with respect to arithmetic or geometric mean values but high blood lead levels persisted in the population. To illustrate, in France there has been a significant fall in the (average) amount of lead in the blood since 1995 but about 10% of children still had levels above 5.0 µg/dl in the early 2000s (La Ruche G et al 2004 cited in WHO-ENHIS 2009). Le Bot et al (2016) found that despite the declines and the lead-free policies for petrol, paint and food cans, lead in drinking water and in dust are still causing elevated blood lead levels, with 78,466 (17,171–139,761) children having blood lead levels over 10 µg/dL due to lead in drinking water.

Where blood lead screening programmes are in place, it has been shown that the tightening of lead regulations has reduced the number of residential acute lead poisoning cases, which are now very rare (Brown and Margollis 2012; Gilbert and Weiss 2006; Tong et al 2000); however, these blood lead surveys have also shown that continuous exposure to low levels of lead is still a public health issue in industrialized countries, especially among ethnic minorities and socioeconomically disadvantaged groups. By contrast, in countries where regulations and policies are missing, such

8.2 Blood lead declines due to tightening of the water lead standard

Many studies have shown the relationship between blood lead levels and lead in drinking water (e.g. Brown and Margollis 2012; Lanphear et al 1998; Oulhote et al 2013; Watt et al 2000); see also Section 7.3 and Annex IV.2 for a more detailed account. However, long-term prospective studies studying the response of blood lead levels to specific regulations for lead in drinking water have not been carried out.

A recent review of the 6466 studies examined the robustness of evidence on effectiveness of lead control policies in drinking water (such as the DWD and the Lead-Copper rule in the USA) (Pfadenauer et al 2016). The study concluded that the effectiveness of lead mitigation in drinking water has not been evaluated using a robust population study design, thereby resulting in likely biased results about decline in blood lead levels, or exposure (Pfadenauer et al 2016).

Pfadenauer et al (2016) examined four Scottish studies, which reported declines in the mean values of blood lead levels (geometric and arithmetic mean) after the implementation of lead corrosion treatment in Glasgow, Ayr and Edinburgh (Sherlock et al 1984; Watt et al 1996; Watt et al 2000); see also Section 7.3 and Annex IV.2[14]. As noted by Pfadenauer et al (2016), no statistical analysis was performed in the data from the Scottish studies. It should be added that the Scottish studies provided no clear or robust indication as to the percentage of the population in Scotland exposed to elevated blood lead levels in the 1980s and 1990s. In this respect, Pfadenauer et al (2016) noted: "one must be careful in generalizing these findings to other environmental[15] interventions."

Pfadenauer et al (2016) also suggested that conducting natural experiment-type study designs with or without a control group (Waters et al 2006 and Craig et al 2012 cited in Pfadenauer) provides a feasible way of evaluating the effectiveness of mitigation practices for lead in drinking water (and other media). As an example of this approach they suggested the design of interrupted time series-ITS (Fretheim et al., 2013 cited in Pfadenauer et al 2016; Waters et al 2006 cited in Pfadenauer et al 2016), whereby multiple observations are made over time interrupted by an intervention. This approach is feasible in countries where routine blood lead screening programmes are in place or

[13] No such studies have been conducted in Scotland, where the focus of surveys was in relation to drinking water lead (see Part II).

[14] For comparison with the evidence reported in this Section, the geometric mean of blood lead levels in the 1993 Glasgow study, where the lowest published blood lead levels have been reported, the mean maternal blood lead concentration was 3.7 mg/litre in the population at large, compared with 3.3 mg/litre in households with negligible or absent tap water lead (Watt ret al 2000). These values are similar to values reported for the UK during the same period of time but two to three times higher than the blood lead levels reported elsewhere in a similar period of time or later.

[15] Environmental interventions refer to water treatment interventions, such as raising pH and adding orthophosphate (Pfadenauer et al 2016).

relevant health-epidemiological data are regularly collected.

To sum up, the remarkable declines in lead exposure during the past 50 years have mainly been due to phasing lead out of petrol, paint and food cans. In addition, many studies have shown the relationship between blood lead levels and lead in drinking water. However, robust "before versus after" comparisons of blood lead levels in relation to the tightening of the water lead standard have not been carried out in Scotland and internationally. This makes it difficult to separate the benefits of water lead mitigation to the proportion of the population consuming lead-contaminated water from the benefits of phasing lead out of petrol to the general population and the environment.

9.0 Sampling approaches

9.1 Overview of sampling protocols

Fluctuations of lead in tap water can be caused by changes in pH temperature and water treatment and any other factors that may disturb the structure of the passivating scales formed in the distribution system (see Annex 3). These fluctuations are possible within a supply zone, a neighbourhood, or a single home and may not always be captured by regulatory sampling, even if water is collected under a standard protocol (Schock 1990). Sampling methods must address the fluctuations in lead leaching in order to meet the requirement of representativeness (as in DWD) and to capture maxima values. Renner (2009) identified four monitoring practices which may fail to capture lead fluctuations but allow compliance with the lead standard, thus y hiding serious water lead contamination: (i) failure to pick the worst-case houses; (ii) not allowing water to stagnate long enough inside the plumbing before sampling; (iii) removing the faucet aerator screen before sampling; and (iv) sampling in cooler months.

In the course of one day, flushed water, or water collected after short stagnation times, tends to contain lower lead levels; conversely, first-draw water drawn after stagnation can have comparatively greater lead levels (Annex III.3.2i. However, stagnation time is influenced by consumer behaviour and particularly water use pattern (e.g. Bailey et al 1986). The *water use pattern* refers to parameters such as mean inter-use time, volume of water, and flow rate per household on average in a supply zone.

The effects of stagnation time and water use patterns on compliance with the water lead standard have been addressed in monitoring programmes for water lead in Scotland and internationally. Sampling protocols can be split in two broad categories:

1. Sampling protocols that do not account for stagnation time and inter-use time:

   (i) *fully flushed (FF) samples,* when the pipes are flushed completely before collection; therefore the influence of stagnation time is excluded from assessment.

   (ii) *random daytime (RDT) samples*, when the samples are taken at a random time during the day; therefore the influence of variations in stagnation time is not accounted for.

2. Sampling protocols that account for stagnation time (van der Hoven et al 1999:

   (i) *fixed stagnation time sampling*, whereby after flushing of the tap, the water is allowed to stand for a defined time ranging between 30 minutes (30MS) to 2- or 4-hours.

   (ii) *first draw sampling* after a stagnation time that is long enough to establish equilibrium lead concentration *e.g. overnight (6- to 12-hours). This is also referred as worst case scenario*[16] *and may include* at least 3 plumbing volumes water from the mains in flowing from the tap.

   (iii) *composite proportional sampling,* whereby the first draw of water used for drinking or food preparation at the monitored tap is collected and integrated over a one-week period; therefore this method is representative of the average weekly intake of the consumer. Usually, a small constant proportion (=5%) of every volume of water drawn is collected.

A variety of protocols from RDT to 12-h stagnation time is applied In the EU (Section 9.2). Canada and the USA apply a 6-hour stagnation tiered protocols (Annex I.6.1). However, Ontario, Canada has adopted a 30MS protocol (Dore 2015).

Several studies have evaluated the above mentioned sampling protocols. For example, Jackson (2000) argued that the RDT sampling protocol can be representative only if sufficient samples per supply zone are taken. Bailey et al (1986) found that in the UK the mean inter-use stagnation decreases with the number of individuals in a household and can range between 47 minutes to 18 minutes; the average stagnation time per household was found to be 30 minutes

[16] Tests can also be conducted at the treatment plant: a rig of lead pipes is flushed with water and periodically allowed to stand for 24 hours after which a sample is drawn).

but the values varied considerably. Elsewhere, studies showed that 30MS gave results comparable to composite proportional samples such as in France (Baron 2001) and the Netherlands (Van den Hoven 1986 cited in Dore 2015). Hayes 2010 reported that zonal surveys of consumers' taps based on the 30MS method for lead in drinking water have provided biased assessments, mainly because of the potential for dilution from water standing in non-lead pipework. On a zonal scale RDT and COMP protocols gave similar results (Hayes 2009). Several authors have shown that the 6-hour stagnation (or longer) sampling protocol most accurately reflected saturation levels in lead leaching therefore it is more likely to account for peak exposure to lead in drinking water (Kuch and Wagner 1983; Lilly and Maas 1990; Lytle and Schock 2000); see also Annex III.

In addition to stagnation time, the volume of water potentially in contact with lead pipes and plumbing components is important in selecting sampling protocol. For example, an 8.8 metre length of 12 mm internal diameter copper pipe has a volumetric capacity of 1 litre and it can be readily appreciated that even short lengths will exert a significant dilution effect (Hayes and Skubbala 2009).

Van der Hoven et al (1999) evaluating methods that could be incorporated in the DWD suggested that on average the volume of water drawn from the tap should be 1.2 litres. However, the range of volumes collected after 6-hour stagnation may vary from 1 to more than 12 litres in the case of long service pipes, larger volumes having the potential to account for the influence of lead pipes outwith the premises (Cornwell and Brown 2015). This approach is also known as profile sampling and is useful in identifying the peak lead at a given location which can then be used for evaluating other sampling approaches by comparison to the peak lead occurring in the profile samples, as in the investigation by del Toral et al (2013).

This review did not detect many studies comparing the above mentioned sampling protocols with the results of profile sampling. For example, Lytle and Schock (2000) have advocated obtaining stagnation profiles to predict exposure of consumers to lead and to assess lead corrosion control treatment. By obtaining profile samples, Guidotti et al (2008) showed that peak lead was found in the fourth litre after 6-hour stagnation protocol, demonstrating that the volume of water drawn can determine the level of lead in drinking water. They're research on the effects of stagnation and flushing on lead concentrations has been reviewed in Annex III.

It is also interesting to note that whatever type of water sample is collected, a single sample cannot provide a reliable estimate of the resident's exposure to water lead, as argued by Pocock (1980). To illustrate, during an environmental assessment of a lead-poisoned child in Washington DC in 2004, the DC Department of Health (DOH) concluded that drinking water was not a potential hazard, based on collection of a single flushed water sample which measured lead at a reassuring concentration of 11 µg/L. Freedom of information act (FOIA) requests revealed that in four other flushed samples collected by the local water utility, lead in water ranged between 19-583 µg/L (Edwards 2005 cited in Triantafyllidou 2011).

The advantages and disadvantages of the various sampling protocols are summarised in Table 7.

Table 7. Advantages and disadvantages of the available sampling protocols for compliance monitoring for lead in drinking water. RDT: Random Daytime sampling. FF: Fully Flushed. 30MS: 30 minutes stagnation. COMP: composite proportional sampling. Stagnation refers to sampling after over 2-h stagnation. Source: Literature in Section 9.1.

	RDT	FF	30MS	COMP	Stagnation
Advantages	• Unbiased assessment of zonal compliance • Cost effectiveness • Accounting of consumers' behaviour • Practicality	• If high it can indicate the presence of particulate lead in the system	• More practical than stagnation or COMP samples	• Direct measure of lead ingested • Reliable for zonal compliance	• Accounting for maximum risk of exposure to lead • Reliable for zonal compliance • Useful in detecting location of peak lead within the supply zone (worst case)
Disadvantages	• Not representative of maximum lead concentration in water • Underestimation of COMP lead • Not accounting of stagnation (stagnation profiles) • Not representative of risk from exposure to lead • Variable-not reproducible • Requires higher frequencies than those applied in EU.	• Not compatible with compliance monitoring • Maybe influenced by particulate • Underestimates COMP lead	• Underestimation of COMP lead • Incompatible with evidence form stagnation profiles	• High cost of COMP device • Requires a large number of sampling events across the supply zone • Not reliable for the assessment of lead in individual properties • Not equal to weekly lead intake • Not accounting for risk of maximum exposure to lead	• Less practical than all others • Possibly more costly • Sensitive to property selection

Dore (2015) argued that selecting appropriate tap water frequency is key to detecting all the range of lead variations on a property scale or within a supply zone per year and in minimising the health risks and social costs associated with lead in drinking water. However, different sampling protocols and different numbers of samples per year are also applied for the detection of non-compliances (Table 8). This renders comparisons of exposure to lead in drinking water between different jurisdictions problematic.

Table 8. Comparisons of samples taken to detect non-compliances with the standard of lead in drinking water in EU Member States, Canada and the USA. Source: Dore 2015; DWD 1998; Hayes 2010.

Volume of water distributed (no of people)	EU (no. of samples per year	Canada and USA (sites sampled once per year)	
		(initial monitoring)	(reduced monitoring)**
Less than 2	*	5	5
>2 - ≤200	*	10	5
>200- ≤2000	1	20	10
>2000 - ≤20,000	1 + 1 for each 660 people and part thereof of total population	40	20
>20,000 - ≤200,000	3 + 1 for each 2000 people and part thereof of total population	60	30
>200,000	10 + 1 for each 5000 people and part thereof of total population	100	50

* The frequency will be decided by each Member State.

** Reduced monitoring is applied only when in an annual survey two consecutive 6-monthly surveys comply; in a triennial survey the 90th percentile concentration is less than 5 µg/L in two consecutive 6-monthly surveys; in a survey every nine years the population served is <3300 and the 90th percentile concentration is 5 µg/L in two consecutive 6-monthly surveys and the system is free of lead pipes and leaded brass and solder.

9.2 Sampling protocols in the EU

The "adequate sampling method" mentioned in Annex 1. Part B. Note 3 of DWD refers to the outcome of the study "Developing a new protocol for the monitoring of lead in drinking water"; EUR 19087 EN (Van der Hoven et al 1999), hereafter reported as the EU Report. The EU Report assessed the results of three sampling protocols (RDT, FF and 30MS) against the composite proportional sampling (COMP) method. The COMP sampler is a sampling device, which is attached to the consumer's kitchen tap in order to determine the average lead concentration over a period of one week. Consumers were required to turn on the device only when they are consuming water for dietary purposes. This can capture 5% of water drawn. The EU report mentioned that the COMP method is the only method that captures all the factors influencing average weekly lead intake by consumers (Van der Hoven et al 1999, p. 32). It also mentions that, on the basis of a literature review, that a 6-hour stagnation sampling protocol would show when lead concentration reaches the saturation level.

However, the EU Report proposed to use a random day-time sampling (RDT) or 30 minute stagnation time (30MS) before sampling for compliance monitoring and this has been adopted by the Standing Committee on Drinking Water (Art. 12). RDT and 30MS were deemed to have the potential to satisfy the criteria of representativeness logistic feasibility and cost-effectiveness for assessing exposure to lead in drinking water in a supply zone (e.g. a city or town). The EU Report concluded that RDT was representative (i.e. >80% of the problem properties could be detected); it overestimated the real exposure on average; and had a poor reproducibility, due to an undefined stagnation time. The 30MS sampling method was representative (>70% of problem properties detected) and reproducible.

It must be noted that the DWD requirements for lead control and monitoring have been extensively criticised in the literature. Hayes (2010) argued that the DWD contains vague requirements for lead control and monitoring that have unsurprisingly largely been overlooked or resulted in a failure to agree a harmonised monitoring method. In the same line, a report by the Joint Research Committee of the European Commission argued that the DWD is not clear in the practical protection of the consumer and that the weekly average (mentioned in Annex 1.Part B. Note 3 of DWD) may not be representative for the consumption for one year (Hoekstra et al 2004).

The EU report and its recommendations have been extensively challenged by Dore (2015) and Hoekstra et al (2004) on the grounds of failing to take into account the public health cost associated with maximum exposure to lead. Dore (2015) argued that the statistical analysis used in the EU report was erroneously interpreted by its authors and that in fact it pointed to a combination of the RDT and FF methods as more suitable than the 30MS or RDT protocols separately in terms of cost effectiveness, consumer acceptability, and accuracy. Referring to the EU Report, Dore (2015) criticised the DWD for not spelling out the sampling protocol thus in practise the EU-Member States have to rely on a "faulty report" in terms of monitoring guidelines. Most importantly, Dore (2015) demonstrated that neither the COMP nor any of the FF, RDT and 30MS sampling protocols are reliable measures of the average weekly levels of lead as it is the average weekly intake of lead. In the author's view, "From the health point of view, what matters most is the maximum exposure and not the average exposure" (Dore 2015).

Hoekstra et al (2004) compared the results presented for the three protocols in the EU report, i.e. RDT, 30MS and FF and found that these underestimated the levels estimated by COMP in 44, 56 and 71% of the properties, respectively. This underestimation of COMP was influenced by the size of the supply zone and varied in the range of 25-70%, 25-95% and 55-100% of COMP, respectively. All three methods also had a poor prediction of the real exposure. In only about 20% of the properties all three methods consistently predicted the COMP concentrations. In view of these findings, Hoekstra et al (2004) recommended to EU that a broader and more detailed study is carried out to establish stagnation times in domestic properties in Europe to inform sampling protocols. As for frequency, Hoekstra et al (2009) argued that the minimum frequencies applied in the EU are inadequate for operational control monitoring for the effectiveness of plumbosolvency control and the effect of other treatments on lead concentrations.

Indicative of the lack of agreement in "adequate sampling method" for lead in drinking water and with the recommendations of the EU Report is that whereas the UK and Scotland apply the RDT sampling approach other countries do not. For example, France applies the 30MS protocol (Oulhote et al 2013); Denmark applies a 12-hour stagnation time before sampling to account for a worst case scenario; and Germany applies a 4-hour stagnation time which was found to give 95% protection of the general public from exposure to lead in drinking water (Hoekstra et al 2004; Hayes 2010; Dore 2015).

10.0 Cost of mitigation versus social cost of lead exposure

Any change in lead regulations comes at a cost and any delay in taking the appropriate action to remove lead exposure also has a cost. This Section intends to juxtapose the costs of mitigating lead in drinking water against the cost of health and social interventions needed to treat those exposed to low levels of lead. The reason for the focus on low lead exposure is justified by the very small part of the population being currently exposed to high levels of lead above 20 µg/dl or needing chelation treatment (e.g. WHO-ENHIS 2009).

<u>Cost of pipe replacement or rehabilitation.</u> In general, interventions such as total-LPR have been considered infeasible in the short-term due to their cost for the responsible authorities or homeowners or both (Aetensdedt 2016; AWWA 2012; Brown et al 2015; Goody et al 2017; Hayes 2010; UKWIR 2012). For example, in the USA the AWWA (2012) has estimated the cost of replacing drinking water infrastructure at around $1 trillion over the next 25 years. For a population of 100,000, Total-LPR has been estimated to cost between £12.9M and £51.7M spread over 10 years, with per property cost at £862-£3448.3 per total lead pipe replaced (Hayes 2010). Hayes (2010) has also provided estimates of net present value[17] for Total-LPR at £10.05 – £40.3 M. Epoxy lining has been estimated to cost between £215.5 and £1724.1 per connection, therefore pipe rehabilitation has been estimated to cost two to four times less than pipe replacement.

In the UK, the cost of communication pipe replacement has been estimated to cost approximately £431 to £1724.1 per pipe replaced, depending on ground and surface conditions at each specific site and associated labour costs (Hayes 2010). In the case of premise pipes the estimated cost was in the same range, depending on pipe length, and works at the premise frontages (Hayes 2010). Using data provided by one company as a worked example, UKWIR (2012) estimated that the cost of replacing all <u>communication pipes only</u> would amount to over £390M as net present value taking account of replacement costs and chemical savings. Replacing communication and supply pipes was estimated to add up to over £890 million. Although these calculations were based on a model developed at a supply zone scale for water companies to weigh costs of different lead mitigation options, the size of the population it refers to remains unclear[18].

<u>Chemical treatment for lead corrosion control.</u> The cost of orthophosphate varies from year to year depending on market prices (see Section 6.1.3). As of 2009 its cost in the UK was £750 per tonne. At an optimised dose of 1 mg/l, a unit cost of £3.2 /Ml and an operational cost of £1.3/Ml can be expected, meaning a total unit cost of £4.5/Ml (Hayes 2010). The estimated net present value[19] has been estimated to be £0.564M (Hayes 2010). In the UK, it has been estimated that the market price of phosphorus used to

[17] Based on a 25 year planning horizon and a discount rate of 6% for a city of 100,000 population.
[18] Only the Executive summary of this report was available online.
[19] Assuming £89,206.90 capital cost, 20% capital replacement every 5 years and annual operating costs of £32,724.10.

dose raw water must go up by a factor of 20 before Total-LPR would be financially viable (UKWIR 2012).

Cost of impact of phosphorus contained in mains water leakages: This externality is considered here because water leakages are considered as a high priority issue for improvement among consumers in Scotland (SW 2014). It has to be noted that the sustainable economic level of leakage (S)ELL from the public network is very sensitive to the assumed water cost. For example, a 1% increase in the value of the lost water could lead to the (S)ELL falling by 10% (POST 1995 cited in Gooddy et al 2017). In the UK, the estimation of (S)ELL incorporates estimates for the carbon costs, the interruption to water supplies, the disturbance to vehicle movement and the impact of noise pollution due to leakage, alongside the environmental benefits of reduced water abstraction following reductions in mains water leakage (MWL). For example, at an assumed damage cost of approximately £33 per kg of phosphorus (Pretty et al 2003 cited in Gooddy et al 2017) and the estimate of 1200 tonnes of MWL-P per year (Ascott et al 2016), multiplying these figures gives the total damage costs associated with phosphorus from MWL, which would be approximately £39M in the UK (Gooddy et al 2017).

Clearly, this estimate assumes that all MWL-P remains within the environment and contributes to environmental damage. It remains uncertain whether more accurate calculations of MWL-P will give an output of a higher or lower cost due to the orthophosphate dosing-related emissions to the environment. Gooddy et al (2017) argued that the implication of accounting for MWL-P as an externality would be to lower the (S)ELL and thereby to reduce phosphorus loads discharged to the environment from MWL, assuming that (S)ELL targets were met. However, a proportion of any additional capital or operating costs associated with meeting a lower (S)ELL target would be borne by water customers, which would require approval from the economic regulator in England and Wales and may well meet resistance from water customers (Gooddy et al 2017).

Health and social costs of lead exposure: Several researchers have used data to calculate the social cost of the presence of lead in drinking water. The results of these studies are used here figuratively and not literally, because of the growing evidence on effects of lead at ever lower levels of exposure. Quantifying the health and social costs of lead exposure is quintessential for a lead free residential environment. For example, commenting on policies for the control of lead in paint, Swartz (1994, p. 105) argued: "As long as attention focuses on the costs of lead-paint abatement and ignores the costs of not abating and as long as people add up the costs of removing paint but not the costs of medical care, compensatory education, and school dropouts, substantial action is unlikely."

The following findings illustrate the health and social costs of not mitigating lead from all sources:

- Grosse et al (2002) used data from 1976 onwards and found that, if IQ points increase by 0.185-0.323 in the population for every 1 μg/dl[20] of lead in blood and each IQ point increases worker productivity by 1.8-2.4%, then the overall monetary benefit to society as a result of lead hazard control, would be in the range of \$110-\$318 billion (counting one IQ point in 2000 dollars).

- Landrigan et al (2002) also considered IQ loss as the main consequence of lead exposure, in line with the GBD studies (Section 4.6 and 5.4), and estimated a total annual loss to society from lead exposure in childhood at \$43.4 billion; for comparison the total annual costs of environmentally attributable diseases ranged from \$48.8 to \$64.8 billion.

- Stefanak et al (2005) estimated that the cost of child lead poisoning on the healthcare system due to screening and treating can cost the system \$969 per child when blood lead levels exceed 20 μg/dl compared with \$29 for children with blood lead below 10 μg/dl. In addition, the total cost for special education for children with blood lead levels above 25 μg/dl were estimated to be approximately \$500,000; the cost for juvenile justice services for children with blood lead levels above 25 μg/dl was estimated to be approximately \$224,000 for each one year cohort of children.

- Zahran et al (2009) argued that a one-time payment for pre-school lead exposure prevention would be more cost –effective than having to pay for the costs associated with neurotoxic damage associated with lead exposure during pre-school years.

- Gould (2009) estimated that the net benefit to society as a result of lead hazard control can be worth \$181-269 billion (for example cost saved from reduced costs for medical treatment, criminal activity, special education). The researcher estimated that for every dollar invested to reduce lead hazards in housing benefits of \$17 to \$220 would accrue.

- Pichery et al (2011) assessed the monetary benefits of lead abatement in terms of avoided national costs in France using data from a 2008 survey on blood-lead levels in children aged one to six years old. Adverse health outcomes of lead exposure were translated into social burden and economic costs based on literature data from

[20] More recently it was found that the IQ-blood relationship is supra-lineal and for every 1 μg/dl of lead in blood a 1% loss of IQ is expected (Budtz-Jørgensen et al 2012).

research for effects above 1.5 μg/dl, 2.4 μg/dl and 10.0 μg/dl. The estimations included direct health benefits (from avoiding blood lead screening programmes, hospitalisation and treatment of children); social benefits (from avoiding loss of life-time earnings, special education costs and crime costs[21] and intangible (*pretium dolloris*) avoided costs. Costs of pollutant exposure control were partially estimated in regard to homes lead-based paint decontamination, investments aiming at reducing industrial lead emissions and removal of all lead drinking water pipes. The authors concluded that reducing only lead exposures above 10 μg/dl of lead in blood has little economic impact due to the small number of children who now exhibit such high exposure levels. They added that prudent public policies would help avoiding future medical interventions, limit the need for special education and increase future productivity, and hence lifetime income for children exposed to lead. The overall annual costs and benefits for 2008 estimated by Pichery et al (2011) are shown in Table 9.

Table 9. Results of analysis of costs and benefits arising from the abatement of lead and the avoidance of public health and social costs arising from lead exposure for 2008 for the cohort of 1-6-year old children in France. Source: Pichery et al 2011.

Blood lead levels (BLL, μg/dl)	No. of children (%) of total population in France	COSTS from abatement		BENEFITS of avoiding costs for (€billion)
		Per child in the cohort (€)	Total (€billion)	Avoided costs
<1.5	2,348,091 (50%)	0	0	0
1.5<=BLL<2.4	1,648,975 (35.1%)	629	2.95	22.72
2.4<=BLL<10	693,783 (14.8%)	629	2.95	10.72
>=10	5,333 (0.1)	185	0.9	0.44

- Attina and Trasande (2013) estimated the economic costs attributable to childhood (cohort: 1-5 year old children) lead exposure in "low- and middle-income countries". Analysis was based on an environmentally attributable fraction model to estimate lead-attributable economic costs; only neurodevelopmental impacts of lead, assessed as loss of IQ points, were accounted for. The researchers estimated a total cost of $977 billion of international dollars in low- and middle income countries, with economic losses equal to: $134.7 billion in Africa (approximately 4% of gross domestic product-GDP); $142.3 billion in Latin America and the Caribbean (approximately 2.04% of GDP); and $699.9 billion in Asia (1.88% of GDP). The sensitivity analysis indicated a total economic loss in the range of $728.6–1162.5 billion in n low- and middle-income countries. These estimates for the economic cost of childhood lead exposure amounted to 1.20% of world GDP in 2011.

- Bartlett and Trasande (2013) undertook a more general study of the economic impact of the environmentally attributable childhood exposure on health outcomes in the EU. Data on exposures, disease prevalence and costs were analysed at a country level, and then costs were aggregated across EU member states to estimate overall economic impacts within the EU. They used a cost-of-illness approach to estimate health care system costs, and environmentally attributable fraction modelling to estimate the proportion of childhood disease due to environmental exposures. They found that the combined environmentally attributable costs of lead exposure, methylmercury exposure, developmental disabilities, asthma and cancer to be $70.9 billion in 2008 (range: $58.9–$90.6 billion). Their study reported data on the economic costs of lead exposure in the UK in 2008, i.e. $ 2.3billion ($2.2-$2.74 billion).

[21] On the basis of French national data, Pichery et al (2011) reported the following lead-linked crime-associated costs for 2008: burglaries=€96M; robberies=€2.3M; Aggravated assaults=€44.3M; rape=€4.8M; murder=€0.9M.

To sum up, the greatest economic benefits of removing all sources of residential lead (lead pipes and plumbing, soil, dust, leaded paint) arise by avoiding the health and social costs of low lead exposure in the general population. Arguably, this evidence shows that the economic benefits (or saved costs) of moving towards an ultimately lead free outdoor and indoor environment are greater than the economic cost of controlling lead concentrations to comply with the specified thresholds in a range of environmental media.

The caveat, however, is the small transferability of this information for the UK and Scotland, given that the proportion of the population with blood lead levels in the range 2-10 µg/dl is unknown due to the absence of a systematic blood lead screening programmes. However, the case study form France (Pichery et al 2011) may hold useful lessons for Scotland because of the similar regulations, living standards and rates of prevalence of lead pipes in the public network in these two countries (see Annex III.1.1-Prevalence of lead pipes).

Table 10 summarises the findings of this comparative analysis of costs relating to lead mitigation and lead exposure from all sources.

Table 10. Comparative analysis of costs relating to lead mitigation, environmental impacts and public health effects from lead exposure from all sources.

A. Cost of mitigation options for lead in drinking water		References
Total-LPR	£862-£3,448.3	Hayes (2010)
Cost per total service pipe replaced	£12.9M -£51.7M	
Cost per 100K people spread over 10 years	£10.05–£40.3M	
Cost net present cost (as of 2010,see text)		
Partial-LPR		Hayes (2010)
Cost per communication pipe replaced	£431 to £1724.1	
Epoxy lining		Hayes (2010)
Cost per connection	£215.5 - £1,724.1	
Orthophosphate dosing of 1 mg/l for a		Hayes (2010)
Annual operational cost for a 100K people	£32,724.1	
Annual net cost (as of 2010, see text)	£0.564M	
B. Environment and social impacts of lead		
Annual environmental impact of phosphorus in water mains leakages in the UK	£39M	Gooddy et al (2017)
Total loss to society from lead exposure in childhood	$43.4 Billion	Landrigan et al (2002
Annual Cost of lead poisoning on the healthcare system per child:		Stefanak et al (2005)
Blood lead levels>20 µg/dl	$969	
Blood lead levels<10 µg/dl	$29	
Annual cost of lead poisoning on special education services for children:		Stefanak et al (2005)
Blood lead levels>25 µg/dl	$500,000	
Cost of lead poisoning on justice services for children:		Stefanak et al (2005)
Blood lead levels>25 µg/dl	$224,000 for each one year cohort of children	
Annual Cost-of-illness due to lead exposure in the UK in 2008	$2.3 billion ($2.02- 2.74 billion)	Bartlett and Trasande 2013
C. Benefits of lead-policies and lead mitigation		References
Net benefit to society from lead hazard control due to savings from reduced costs for medical treatment, criminal activity, special education	$181-269 billion	Gould (2009)
Benefits to productivity for every drop in blood lead levels by 1 µg/dl	$110-$318 Billion	Grosse et al (2002)

11.0 Summary of results

The findings of the literature review on the public health and economic aspects of lead in drinking water in Scotland and internationally are summarised below.

1(i) Lead's widespread use (e.g. in mining, smelting, coal burning, heavy industry, petrol, paint, batteries and plumbing) has resulted in extensive environmental contamination. Despite this, lead is unlikely to be present in source water unless a specific anthropogenic source of contamination exists.

1(ii) Lead exposure refers to the actual absorption of lead in the body (e.g. in blood, bones, soft tissues), regardless of the source or perceived risk of lead exposure; it can be measured as body lead concentration, e.g. blood lead in µg/dl. Once taken in by ingestion or inhalation, lead enters the bloodstream, accumulates in bones, teeth, hair and nails, and interferes with the function of vital organs (especially the liver, kidneys and brain). In pregnant women it crosses the placental barrier and affects the unborn child. Lead mainly targets the central nervous system by interfering with the function of neurotransmitters, thus disrupting learning, memory, and sensory and motor skills, i.e. it causes ID.

1(iii) Lead exposure has been found to be the single contributing risk factor for intellectual impairment in Europe and on a global level. The World Health Organisation has identified lead as a chemical of major public health concern, requiring action by all countries to protect the health of workers, children and women of reproductive age.

1(iv) The current standard for lead in drinking water (10 µg/l) is consistent with the provisional tolerable weekly intake (PTWI) of 25 µg/kg of lead per body weight, agreed as safe by the Joint Expert Committee on Food Additives on the basis of evidence published by 1999. However, in view of more recent evidence, this provisional intake was withdrawn because it was associated with neurodevelopmental effects in infants and children and an increase in systolic blood pressure in adults. In areas where additional sources of lead in a child's environment occur (e.g. lead in residential paint), water lead at 10 µg/l may be a surplus exposure.

2(1) In Scotland, specific lead-control practices have been in place for the past 50 years to reduce lead in drinking water, such as: replacement of lead water mains and communication pipes (i.e. partial lead pipe replacement); orthophosphate dosing to reduce lead leaching; means-tested grant-funding to homeowners to replace their pipes; and provision of information and advice to property owners to replace their lead pipes. The Drinking Water Quality Regulator (DWQR) for Scotland reported that in 2015 the current standard of 10 µg/l for lead in drinking water was met in 99% of samples from public supply zones managed by Scottish Water and in up to 92% of private water supplies[22].

2(ii) Many small-scale studies have examined blood lead levels in areas of Scotland with plumbosolvent water and in relation to local sources of environmental lead in the past and present. However, these studies should be interpreted with caution because they relied on small numbers of participants under specific circumstances that may not be representative for the general public and used no sufficient "control" data (e.g. no data on exposure to lead in non-plumbosolvent supplies).

2(iii) The available evidence for Scotland can be examined in the context of lead control measures:

- Pre-1990, i.e. before water (e.g. orthophosphate dosing) and air lead control measures.
    - o Water was not the only source of lead exposure. At zero water lead levels, blood lead levels in adults and bottle-fed infants were projected to be at approximately 5-6 µg/dl.
- Post-1990, i.e. after water (e.g. orthophosphate dosing) and air lead control measures.
    - o Blood lead in mothers consuming lead-free water was projected at 3.3 µg/dl (i.e. low).
    - o No official data on trends or current levels of exposure to lead are available but research analyses have shown that the number of years lost to disability attributed to lead exposure were three to four times lower in 2015 compared with 1990.

3(i) Lead in drinking water is not a problem in the UK because of the extensive orthophosphate dosing. Yet, it is not a lead-free strategy and does not preclude low exposure to lead in drinking water. Optimisation of the orthophosphate dose is sensitive to factors influencing lead scale solubility in the distribution system, such as:

- Galvanic (lead-copper) connections (under partial

[22] Private water supplies serve approximately 3.5% of the population in Scotland and are their owner's responsibility.

lead pipe replacement, whereby the homeowners' side contains lead piping), which may induce electrochemical reactions disturbing the lead scales that have accumulated on the lead pipes over time.

- Changes in flow, which may trigger resuspension of lead scales.
- Temperature fluctuations, which may alter the reaction rate of orthophosphate with lead.
- Lowering of redox potential, as when switching from chlorine to chloramine disinfection without first adjusting orthophosphate dosing for the associated changes in lead solubility and accounting for the composition of lead-containing scales within the pipes.
- Prolonged stagnation, with water lead fluctuations during the first 72 hours of stagnation.

3(ii). Total lead pipe replacement (Total-LPR) is often prohibitive due to high cost and practical problems (e.g. need for removal of all premise plumbing, disruption of traffic and risk of damage of property). Effectiveness depends on homeowners' willingness or ability to pay or the availability of state grants for the LPR in the homeowners' side. Alternative solutions have been suggested in the literature with the aim to reduce the cost for homeowners, such as: transfer of cost to water utilities; or transfer of ownership of premise pipes to utilities. However, it has been argued that even if all of the original lead sources are replaced (brass faucets, soldered copper piping, and lead pipes) the lead levels can still be high due to lead migrating from the lead scale deposits on the remaining lead-free plumbing before replacement. Other options are either not feasible in the short term (e.g. epoxy lining of lead pipes); or not applicable for long term use (e.g. installing point-of-use or whole-house lead removal filter systems).

3(iii). Partial lead pipe replacement (Partial-LPR) refers to replacing the lead pipes only in the utility's side and controlling lead leaching from homeowners' pipes by orthophosphate dosing and advice on water use patterns. Effectiveness mainly depends on the type of distribution materials, e.g. in the presence of lead-copper or brass (galvanic) connections randomly occurring spikes in lead concentrations above the current water lead standard may occur, even after orthophosphate dosing.

4. The best biomarker for current low level exposure to lead in drinking water is blood lead; bone and teeth lead are the best indicators of past and long-term exposure. For high exposure, clinical symptoms can be used as indicators. The risk of exposure to lead in drinking water can be assessed by the occurrence of lead pipes and plumbing and breaches of the water lead standard.

5. To detect a significant relationship between lead in drinking water and low blood lead levels in the population one has to: (i) design studies with a sufficient number of participants, running over a long period time and including sufficient "control" data (i.e. blood lead under background exposure to lead); (ii) enable concurrent monitoring of the blood lead in the population and the levels of lead in drinking water; and (iii) account for a range of water lead levels and levels of other environmental sources of lead, and a range of ages, social circumstances and habits/professions in the population. The water lead-blood lead relationship is not linear. As a rule of thumb, large reductions in water lead levels may have a small impact on the blood lead levels of an individual; conversely, large increases in blood lead levels can occur with small increases in water lead levels.

6. Phasing lead out of lead in petrol resulted in clear declines in blood lead levels in areas of the world where other sources of exposure to lead were minimal or absent, suggesting that mitigating all sources of lead is essential. However, it remains uncertain whether regulations for the mitigation of lead in drinking water (e.g. partial pipe replacement and orthophosphate dosing) have caused declines in blood lead levels mainly because of the lack of robust "before versus after" comparisons in premises where tap water lead declined due to the regulations.

7(i) The UK and Scotland currently apply the random daytime (RDT) sampling and the frequency provisions of the EU Drinking Water Directive for lead. RDT has been cost-effective for public supplies because sampling is practical and results are representative of the average water consumption behaviour of the consumer and the average stagnation time in a supply zone. However, the maximum exposure to lead within a supply zone is not accounted for, thus potentially increasing the risk of underestimating exposure to lead by more than 40%.

7(ii) Elsewhere, legislation requires the use of a stagnation period prior to sampling to test compliance with the lead standard: 30-minutes in France and in Ontario, Canada; 12-hours in Denmark; 4-hours in Germany; and 6-hours in the USA and some Canadian jurisdictions.

7(iii) Research outwith Scotland showed widespread lead contamination in private water supply systems due to lead plumbing and fluctuations of particulate lead release in the system. It was argued that these fluctuations remain undetected by regulatory sampling (i.e. once a year).

8(i) A comparison of available data on the cost of mitigation of lead in drinking water showed that the net annual operational cost for orthophosphate dose of 1mg/l for a population of 100K, which is approximately £0.56M, has been substantially lower than the net cost of Total-LPR for 100K people spread over 10 years, i.e. £10M-£40M, or £862-£3448 per connection (utility's and homeowner's side). However, these estimates have not taken into account the net annual cost of environmental damage due to orthophosphate in mains water leakages (WML-P). This cost has been estimated to be approximately £39M, assuming that all WML-P contributes to environmental damage.

8(ii) As of international research published post-2002, the monetary benefits of lead-free policies (as savings in health and social costs due to the avoidance of adverse effects of residential and low lead exposure from all sources of lead in the environment) have been estimated to range in the order of billions of dollars or euros per year on a country scale. Arguably, the economic benefits of moving towards an ultimately lead-free outdoor and indoor environment have been found to be greater than the economic cost of controlling lead concentrations to comply with the specified thresholds in a range of environmental media.

12.0 Concluding remarks

This report reviewed the evidence on the public health aspects of mitigation and monitoring practices for lead in drinking water and accounted for all factors influencing one's exposure to lead and the health and social outcomes of this exposure. The report analysed results and observations of research carried out during the past 50 years. The evidence reviewed showed a fast evolution in the scientific understanding of the adverse and irreversible, in many cases, health effects of lead exposure.

The consensus among scientists is that the focus of regulation and preventative policies should be to keep exposure in the range of blood lead levels of 2-10 µg/dl because exposure to greater levels is now rare. Legislation has caught up with this knowledge leading to strict controls on lead uses and emissions and lead-free petrol, paint and food cans. However, lead is still in use in certain sectors of industry because of its unique and irreplaceable properties (e.g. acid lead batteries). Background lead levels in the environment have declined by more than 90% since the 1990s but they are not at zero levels. Large amounts of lead have been stored in the environment (e.g. soil) because of historical every day and industrial uses of lead, thereby potentially contaminating food and outdoor and indoor dust. Old housing may still contain leaded paint and some toys still contain lead. The water distribution systems in many countries still contain lead supply pipes and lead in premise plumbing components in ever decreasing percentages. Chemical lead corrosion control is the most commonly used strategy to mitigate lead in tap water and protect public health from the adverse effects of low lead exposure.

The key findings of the literature review are given below.

- The review of the national and international literature on lead in drinking water showed that there is sufficient and robust scientific evidence on (i) the contribution of water lead on individual lead exposure; (ii) the adverse health effects and social outcomes of lead exposure in childhood; (iii) the shortcomings and cost of lead mitigation practices; and (iv) the public health and monetary benefits of lead-free practices.

- The predominant source of lead in drinking water is lead pipes and plumbing.

- No safe level or threshold for lead exposure has been agreed by experts. The World Health Organisation has identified lead as a chemical of major public health concern.

- Low exposure to lead (i.e. blood lead concentrations below 10 µg/dl) in children has been associated with intellectual impairment in childhood, and cognitive deficit, loss of individual potential and low income in adulthood.

- The World Health Organisation has warned that there may be a risk for bottle-fed infants through intake of drinking water with a lead concentration of 10 µg/L.

- In Scotland:

    - Failures of the water lead standard (i.e. 10 µg/L) are predominantly associated with the presence of lead plumbing components and lead supply pipes, which run within the boundary of a property and are homeowners' responsibility to replace.

    - Failures of the lead standard also arise in supply zones where communication pipes, which connect properties to the mains in the street and are Scottish Water's responsibility to replace, remain made of lead.

  - o Optimised orthophosphate dosing (i.e. the dose required to achieve compliance with the water lead standard of 10 μg/L) has been shown to effectively reduce lead leaching from lead pipes and brass fittings within premises. However, it is not a lead-free strategy.

- Total lead pipe replacement (i.e. replacement of lead pipes in utility's and homeowners' side) can be a lead-free strategy. However, homeowners' cooperation has generally been poor, irrespective of the availability of state-funded lead pipe replacement grants, because of the disruption and inconvenience involved and the cost incurred in case of means-tested grants.

- Since the 1970s, lead-free policies (e.g. gradually phasing lead out of petrol) and the tightening of the standard for lead in drinking water (i.e. from 100 μg/L to 10 μg/L) co-occurred. This makes it difficult to separate the benefits of water lead mitigation to the proportion of the population consuming lead-contaminated water from the benefits of phasing lead out of petrol to the general population and the environment.

- The greatest economic benefits to the society of removing all sources of residential lead (including lead pipes and plumbing) arise by avoiding the health and social costs of low lead exposure in the affected proportion of the population. These costs refer to provision of medical treatment and special education; combating lead-linked crime; and loss of life-time earnings and contribution to general productivity due to poorer individual potential.

This report has provided sufficient evidence and context to analyse and evaluate the public health and economic implications of lead-free drinking water in Scotland.

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