Is there still a problem with lead in drinking water in the European Union?
C. R. Hayes and N. D. Skubala

ABSTRACT

The presence of lead in drinking water poses a range of risks to human health, including the retardation of some aspects of child development, the inducement of abortion, and other clinical disorders. The extent of these risks has not been quantified at the European Union (EU) scale. A number of sampling methods are in use across the EU, some of which are inadequate for determining the concentrations of lead in drinking water at consumers’ taps. In consequence, non-compliance with the EU standards for lead in drinking water has been under-estimated. Emerging data indicates significant non-compliance with these standards in some countries, particularly with the 10 mgl⁻¹ standard that will become a legal requirement in 2013; the current interim standard of 25 mgl⁻¹ is also exceeded in some locations. An initial estimate is that 25% of domestic dwellings in the EU have a lead pipe, either as a connection to the water main, or as part of the internal plumbing, or both, potentially putting 120 million people at risk from lead in drinking water within the EU. These issues are relevant to the implementation of the Protocol on Water and Health and to drinking water safety planning.

Key words | drinking water, lead, ortho-phosphate, public health, sampling

INTRODUCTION

Before the mid-1970s there were not considered to be any problems with lead in drinking water in the United Kingdom (UK), with the possible exception of Glasgow. At that time, water suppliers would normally only contemplate corrective action if 300 mgl⁻¹ was regularly exceeded at a property, on the basis of the upper limit recommended by the World Health Organisation (WHO 1970). Indeed, it was uncommon for lead in drinking water to be routinely analysed, particularly in hard water areas. Some 30 years later, around 95% of the UK’s public water supplies are being dosed with ortho-phosphate (a corrosion inhibitor) to minimise plumbosolvency. This is a remarkable transformation and case studies such as Hayes et al. (2006, 2008) demonstrate that ortho-phosphate dosing can be very successful, once optimised.

In England and Wales, plumbosolvency control is required in order to achieve at least 98% compliance (Drinking Water Inspectorate 2000, 2001) with the EU standard for lead of 10 mgl⁻¹ (European Commission 1998) that will become a legal requirement in 2013. Recent reports (Drinking Water Inspectorate 2008a,b,c,d,e,f,g) have indicated 98.3% compliance with the lead standard of 10 mgl⁻¹ in England and Wales for 2007, based on over 23,000 random daytime samples, providing clear evidence of the success of the treatment-based approach to plumbosolvency control.

It has become apparent that most other EU countries are reluctant to dose ortho-phosphate in their water supplies because of philosophical or environmental concerns (COST Action 637 2007). Measures to counter lead in drinking water have been limited to a few cities, with the phased removal of lead connection pipes by the water suppliers concerned, without removing internal lead pipework. For example, in Brussels the water company has
been replacing around 5,000 lead connection pipes per annum with the aim of replacing all of its lead connection pipes by 2013; the water company is not replacing the lead pipes inside houses (R. Goossens, personal communication 2008). Where the total removal of lead pipes has been attempted, as experienced in The Hague, cooperation of householders has been poor because of the disruption and inconvenience involved (Van Dongen et al. 2007). In this city, 23% of random daytime (RDT) samples were found to exceed 10 µg l⁻¹ after a major lead pipe removal campaign that was backed by government funding.

In most of the EU, little corrective action to reduce lead in drinking water has been taken due in part to the absence of relevant data on lead at consumers’ taps (COST Action 637 2007, 2008). It must therefore be pertinent to ask: is there still a problem with lead in drinking water in the European Union?

This paper outlines the basis for health concerns, summarises a recent review of sampling methods in use in the EU, and presents emerging data on the extent of non-compliance with EU standards.

**HEALTH PERSPECTIVES ON LEAD IN DRINKING WATER**

The WHO’s guideline value (WHO 2004) for lead in drinking water is 10 µg l⁻¹ based on a tolerable weekly lead intake of 25 µg kg⁻¹ body weight with 50% of this intake allocated to drinking water. This guideline value has been adopted by EU Directive 98/83/EC (European Commission 1998) to become a legal requirement from December 2013. The Directive also implemented an interim standard for lead in drinking water of 25 µg l⁻¹ from December 2003, up until the time the tighter standard of 10 µg l⁻¹ applies.

Lead is known to be toxic yet it has been widely used as a plumbing material up until the early 1980s, owing to its resistance to aggressive ground conditions and malleable characteristics, but also most likely in the mistaken belief that the internal corrosion films arising from lead oxidation and their subsequent stabilisation would form a protective layer that would prevent further metal releases into water, and owing to ignorance of health effects (Troesken 2006). Lead contamination of drinking water can occur if it is dislodged or dissolved from pipes, fittings and fixtures (Walker & Oliphant 1982), leached from PVC pipes with lead stabilisers (Packham 1971), leached from sub-quality brasses (Grosvenor et al. 2005) or released from galvanic corrosion involving lead-soldered joints (Walker & Oliphant 1982; Maas et al. 2007). However, on the basis of experience in the UK, the main source of lead in drinking water is lead piping (Hayes et al. 2006, 2008).

Lead in drinking water is considered the most bioavailable source of lead (Moore et al. 1985); this is because soluble lead is more readily absorbed in the intestine than lead from dietary sources (Heard et al. 1985). Furthermore, lead intake is increased from water absorbed into food during cooking (Moore et al. 1979; Smart et al. 1981). Daily intake is highly variable: it is dependent on the length of time the water has stagnated in the pipe, the degree of flushing water through the tap before consumption and by the presence of fluoridation and disinfection agents (WHO 1996; Maas et al. 2007). Infants and foetuses are the groups most susceptible to adverse health effects as they absorb 4 to 5 times more lead than adults and the biological half-life is thought to be considerably longer (WHO 2004).

Studies in Scotland, Wales, Germany and the US have found that high lead concentrations in tap water correlate with elevated body lead burden (Moore et al. 1979; Thomas et al. 1979; Lacey et al. 1985; Sherlock & Quinn 1986; Quinn & Sherlock 1990; Potula et al. 1999; Fertmann et al. 2003). The relationship between water lead and blood lead is illustrated in Table 1, which clearly shows that blood lead concentrations of 20 and 25 µg dl⁻¹ can increase in occurrence, with higher average concentrations of lead in water over the range 20 to 100 µg l⁻¹. Other studies further illustrate this relationship: Fertmann et al. (2003) correlated blood lead concentrations of 1 to 6 µg dl⁻¹ with water lead

<table>
<thead>
<tr>
<th>Mean water lead concentration (µg l⁻¹)</th>
<th>Percentage of children with blood lead concentrations above 20 µg dl⁻¹</th>
<th>Percentage of children with blood lead concentrations above 25 µg dl⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>100</td>
<td>33</td>
<td>12</td>
</tr>
<tr>
<td>50</td>
<td>18</td>
<td>5</td>
</tr>
<tr>
<td>30</td>
<td>10</td>
<td>2.3</td>
</tr>
<tr>
<td>20</td>
<td>8</td>
<td>1.7</td>
</tr>
</tbody>
</table>
concentrations below 5 μg l⁻¹, whereas Beattie et al. (1972) associated blood concentrations of 28.4 ± 13.0 μg l⁻¹ with water lead concentrations ranging from 310 to 1,850 μg l⁻¹ and associated blood lead concentrations of 18.7 ± 7.3 μg l⁻¹ with water lead concentrations ranging from 13 to 252 μg l⁻¹.

Troesken (2006) concluded that the relationship between water lead and blood lead is curvilinear such that the lower water lead concentrations have a proportionally higher influence on blood lead concentrations. The action level for blood lead in the United States, used in the prevention of lead poisoning in children, is 10 μg l⁻¹ (CDC 1991), although it has recently been suggested (Gilbert & Weiss 2006) that it should be lowered to 2 μg l⁻¹ as this lower level can be measured accurately and would encourage further action to reduce childhood lead exposure. The WHO’s guideline value (WHO 2004) for lead in drinking water of 10 μg l⁻¹ is consistent with achieving a blood lead limit of 10 μg l⁻¹.

Lead is not an essential trace element in any organism and has no known biological function (Quinn & Sherlock 1990). Its toxicity was recognised as early as 200 BC (Needleman 2004) with both acute and chronic effects arising from lead exposure from various sources (such as air, water and paint). Adverse health effects include: interference with haem biosynthesis, interference with calcium and vitamin D metabolism, gastrointestinal irritation, dullness, restlessness, irritability, poor attention span, headaches, muscle tremor, abdominal cramps, kidney damage, hallucination, loss of memory, encephalopathy, hearing impairment, gonad dysfunction and violent behaviour (Assennato et al. 1987; Bryce-Smith & Ward 1987; Schwartz & Otto 1991; WHO 1996; Stretesky & Lynch 2001; Needleman 2004). The relationship between blood lead burden and adverse health effects is illustrated in Table 2 from a range of case studies and related literature sources.

Perhaps the greatest health concern associated with lead is reduced IQ in infants (Laxen et al. 1978; Tong et al. 1996; Canfield et al. 2003a,b; Chiodo et al. 2007; Bellinger 2008). Tong et al. (1996) found a reduction in the mean full scale IQ of 3 points in children aged 11 to 13 years for an increase in lifetime average blood lead concentration from 10 to 20 μg l⁻¹. Pocock et al. (1994) reviewed 26 epidemiological studies from 1979 and found that a doubling of blood lead from 10 to 20 μg l⁻¹ was associated with a mean deficit in full scale IQ of around 1 to 2 points. Canfield et al. (2003a) found that blood lead was inversely and significantly associated with IQ; the relationship found was non-linear with a decline in IQ of 7.4 points when average lifetime blood lead increased from 1 to 10 μg l⁻¹ with a further decline of 4.6 points with each increase of 10 μg l⁻¹ in the lifetime average blood lead concentration. Chiodo et al. (2007) determined a relationship (r = −0.26, p > 0.001) between blood lead and neurobehavioural outcomes in 7-year-old children (N = 506) over the blood lead range 1 to 20 μg l⁻¹. Bellinger et al. (1992) found a decrease in full scale IQ over a blood lead range of 0 to 25 μg l⁻¹ in infants aged 24 months.

**Table 2** | Blood lead burden and reported adverse health effects

<table>
<thead>
<tr>
<th>Blood lead burden (μg l⁻¹)</th>
<th>Reported adverse health effect</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal levels &gt; 15 μg l⁻¹</td>
<td>Associated with reduced birth weight</td>
<td>Dietrich et al. (1987)</td>
</tr>
<tr>
<td>12 to 120 μg l⁻¹</td>
<td>Vitamin D metabolism interference</td>
<td>Mahaffey et al. (1982)</td>
</tr>
<tr>
<td>Less than 30 μg l⁻¹</td>
<td>Learning difficulties</td>
<td>WHO (2005)</td>
</tr>
<tr>
<td>Increase from 10 to 20 μg l⁻¹</td>
<td>Reduction in IQ by 3 points</td>
<td>Tong et al. (1996)</td>
</tr>
<tr>
<td>Increase from 10 to 20 μg l⁻¹</td>
<td>Reduction in IQ by 1 to 2 points</td>
<td>Pocock et al. (1994)</td>
</tr>
<tr>
<td>Increase from 1 to 10 μg l⁻¹</td>
<td>Reduction in IQ by 7.4 points</td>
<td>Canfield et al. (2003a)</td>
</tr>
<tr>
<td>20 μg l⁻¹</td>
<td>Increased hearing threshold compared with 4 μg l⁻¹</td>
<td>Schwartz &amp; Otto (1987)</td>
</tr>
<tr>
<td>Greater than 37 μg l⁻¹</td>
<td>Hypertension</td>
<td>Pocock et al. (1994)</td>
</tr>
<tr>
<td>40 to 50 μg l⁻¹</td>
<td>Decreased sperm counts</td>
<td>Assennato et al. (1987)</td>
</tr>
<tr>
<td>40 to 120 μg l⁻¹</td>
<td>A range of neurological and behavioural effects</td>
<td>Agency for Toxic Substances &amp; Disease Registry (2007)</td>
</tr>
</tbody>
</table>
Epidemiological studies undertaken in Glasgow (UK), a city with historically high water lead concentrations due to highly plumbosolvent waters and the extensive presence of lead plumbing, found evidence of elevated blood lead levels (Moore et al. 1979; Lacey et al. 1985). Studies in Glasgow also suggested development delays and reduced birth weight (Jones 1989); development deficiencies among children (Beattie et al. 1972); ischaemic heart disease, renal damage, gout and hypertension (Moore 1977) related to high lead concentrations in the tap water.

Corrective water treatments of Glasgow’s water supplies were applied in 1979 (lime to raise pH) and again in the mid-1980s (additional dosing of orthophosphate). Table 3 summarises the reductions in water lead and blood lead that were achieved, based on the study by Moore et al. (1998). In another study by Watt et al. (2000), the geometric mean blood lead concentration in 2000 had fallen to 3.7 µg dl⁻¹ compared with 11.9 µg dl⁻¹ 12 years previously.

Blood lead level reductions were also observed in Edinburgh (UK) after drinking water treatment, with a 64% reduction in blood lead levels in 8 years between the original and follow-up studies undertaken in 1983–1985 and 1992–1993, respectively (Macintyre et al. 1998).

The historic account by Troesken (2006), which focuses on the 19th and early 20th centuries, provides numerous case examples of lead poisoning from drinking water. It also makes reference to the use of lead ‘plaster’ tablets to induce abortion, quoting doses of 0.004 grains of lead per day. This abortifacient dose is equivalent to a daily intake of lead making reference to the use of lead ‘plaster’ tablets to induce abortion, quoting doses of 0.004 grains of lead per day. This abortifacient dose is equivalent to a daily intake of lead.

Table 3 | Reductions in water lead and blood lead in Glasgow (from Moore et al. 1998)

<table>
<thead>
<tr>
<th>Water lead</th>
<th>Mean blood lead in mothers from Glasgow</th>
</tr>
</thead>
<tbody>
<tr>
<td>83% of RDT samples &gt; 50 µg l⁻¹ in 1977</td>
<td>14.6 µg dl⁻¹ in 1977</td>
</tr>
<tr>
<td>13% of RDT samples &gt; 50 µg l⁻¹ in 1980</td>
<td>8.1 µg dl⁻¹ in 1980</td>
</tr>
<tr>
<td>2% of RDT samples &gt; 50 µg l⁻¹ in 1993</td>
<td>3.5 µg dl⁻¹ in 1992–93</td>
</tr>
</tbody>
</table>

This suggests the potential for higher prenatal mortality rates within a population if the drinking water is sufficiently plumbosolvent.

**SAMPLING ISSUES**

Within the European Union (EU), the Member States have failed to agree a harmonised monitoring method for lead at consumers’ taps. A preliminary assessment (COST Action 637 2008) of the sampling methods used in the EU to determine compliance for lead with Directive 98/83/EC (European Commission 1998) indicates (Table 4) that a wide array of sampling methods are being used, some of which are inappropriate for monitoring human exposure from the pipes and fittings that constitute the connections to the water supply and the domestic supply systems.

Random daytime (RDT) first-draw sampling from consumers’ taps is regarded as an appropriate method that is also logistically feasible (Van den Hoven et al. 1999) for assessing exposure to lead in drinking water in a water supply zone (e.g. a city or town), if sufficient samples are taken (Jackson 2000). However, it must be realised that the minimum audit sampling requirements of Directive 98/83/EC (European Commission 1998) are very low and will not provide a reliable assessment of population exposures. For example, a zone supplying 50,000 people only requires four audit samples to be taken per annum.

Zonal surveys of consumers’ taps based on stagnation sampling methods for lead in drinking water are invalidated by the potential for dilution from water standing in non-lead pipework. The normal procedure is to flush the pipework for a few minutes, allow the water to stagnate for a period (e.g. 30 minutes) and then sample the first litre of water that flows from the tap. It is common in the UK for the lead connection pipe to run into a section of copper piping up to the tap used for drawing drinking water. An 8.8 metre length of 12mm 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.

Zonal judgements based on lead pipe test rigs alone are also potentially invalid because the time dependency of the dissolution of lead into the water varies in a water-specific...
manner (Hayes 2007), such that the extent of peak events may either be underestimated or overestimated. Sampling prior to domestic pipework or sampling consumers’ taps after flushing will clearly underestimate the concentrations of lead in drinking water, whereas first-draw sampling after prolonged standing (e.g. overnight) may overestimate. Split-flow composite (COMP) sampling has been used by Van den Hoven et al. (1999) for the direct measurement of weekly average lead concentrations in drinking water (each time the tap is opened to draw drinking water, a small part of the flow is taken for collection in a sample container); while undoubtedly appropriate for the purpose of assessing average lead concentrations over the weekly period of use, it is not feasible logistically for use as a zonal survey tool.

It can be concluded that: (a) the most representative and logistically feasible method for monitoring lead emissions across a zone is RDT sampling, although sufficient samples are needed to gain adequate reproducibility in making zonal judgements; (b) COMP will be the most reliable method for investigating lead emissions at individual dwellings, although there are logistical constraints.

**EMERGING EVIDENCE OF PROBLEMS**

Arising from the preliminary assessment of sampling methods (COST Action 637 2008), the data for lead that has so far emerged, based on at least 100 samples in each case, is summarised in Table 5. The term ‘emerging evidence’ is used as either the data has only recently been obtained or the data has not been previously published in a collated EU-wide format. In Table 5, with the exception of table contents:
of the data from Wales, all data presented was obtained between 2002 and 2008. The data from Wales derived from 1990 to 1995, prior to the commencement of ortho-phosphate dosing to reduce plumbosolvency; it is typical of the UK more generally (prior to ortho-phosphate dosing) based on other case studies (for example: Hayes et al. 2006) and has been included for comparison.

It can be concluded that non-compliance with the lead standard of 10 μg l⁻¹ (which will become a legal requirement in 2013) is significant in some parts of the EU and that plumbosolvency problems are not just confined to the UK (where comprehensive corrective action by water treatment has now been undertaken). Non-compliance with the interim standard of 25 μg l⁻¹ is less pronounced but still conspicuous in some locations; in collating the data shown in Table 5, the authors were not made aware of any measures being taken by the European Commission to secure compliance.

This emerging evidence of non-compliance is not generally evident in the recent synthesis report from the European Commission (2008) for the period 1999 to 2001, although four countries were identified where lead problems existed; however, some countries did not submit their national report, some national reports were incomplete, the methods of sampling used for lead were not identified, and the benchmark for compliance assessment was the earlier standard for lead of 50 μg l⁻¹ from Directive 80/778/EEC.

There is further evidence of problems with lead. Extensive laboratory-based plumbosolvency testing in the UK using the method of Colling et al. (1987) indicates (Hayes 2007) that all types of drinking water in supply are likely to be sufficiently plumbosolvent so as to cause non-compliance with both the EU standards for lead (i.e. 10 and 25 μg l⁻¹) wherever lead pipes are present (if corrosion inhibitors are not dosed). This data is
summarised in Table 6 for the treated water from 158 water treatment works in the UK (obtained over the period 1999 to 2004), prior to any dosing of corrosion inhibitor, at the test temperature of 25°C, for three simplified categories.

The median 30 minutes contact (30MC) data for each sample tested, when adjusted for temperature, equates to a 30 minutes stagnation (30MS) sample for water in contact with a lead pipe in a water supply (assuming no dilution from water standing in non-lead pipework). If the 30MC test data at 25°C is halved, it equates to the annual average temperature of water supplies in the UK (12 to 13°C) (B. T. Croll, personal communication 1999). This temperature adjustment will be lower for countries where the annual average water supply temperature is higher.

Work on water use patterns (Lacey & Jolley 1986) has suggested that 30 minutes is a reasonable estimate of the average water–pipe contact time in a domestic dwelling, implying that 30MS is a reasonable estimate of the average concentration of lead emitted from a tap (assuming no dilution from water standing in non-lead pipework). On this basis, not only were all 158 test waters found to be capable of exceeding the EU’s lead standard of 10 μg l⁻¹, which is based on a weekly average concentration, but most were also found to be capable of exceeding the interim EU standard of 25 μg l⁻¹.

The lead concentrations after 16 hours stagnation give an indication of the equilibrium concentrations of the test waters when in contact with lead piping. Halving the test data (temperature adjustment) indicates that concentrations as high as 1,675 μg l⁻¹ could be expected from consumers’ taps in the UK after overnight standing (assuming no dilution from water standing in non-lead pipework).

Further evidence of the potential scale of problems with lead in drinking water can be inferred from the pan-European study on lead monitoring reported by Van den Hoven et al. (1999) that involved France, Germany, the Netherlands, Portugal and the UK. Split-flow composite (COMP) sampling was undertaken at 289 houses across 11 water supply zones, of which two were phosphate dosed and one partly phosphate dosed; 69% of the houses had a lead service pipe or internal lead piping. COMP provided a direct measure of the weekly average lead concentration at the houses investigated. The 90th percentile concentration determined in each zone varied from 9.6 to 107.8 μg l⁻¹; 44% of the houses investigated exceeded the EU’s standard of 10 μg l⁻¹.

It is interesting to note here the relationship between the percentage of houses that exceeded the EU’s lead standard of 10 μg l⁻¹ and the percentage of houses supplied by a lead pipe. In the Van den Hoven et al. (1999) study, 64% of the houses with a lead pipe exceeded the standard, based on COMP sampling. This compares very closely to the 67% of houses with a lead pipe that were found by RDT sampling to exceed 10 μg l⁻¹ in a major plumbosolvency control programme (Hayes et al. 2008) in Wales prior to phosphate dosing, further supporting the view (Van den Hoven et al. 1999) that RDT and COMP are equivalent in zonal assessment terms (if the zones are mostly without corrosion inhibitor).

Taken together, these two studies suggest that around 65% of houses that have a lead pipe, either as a connection pipe or as internal piping, can be expected to exceed the EU’s lead standard of 10 μg l⁻¹, unless corrosion inhibitor is dosed (and correctly so), which in the EU is not common outside the UK. This raises the question: how many houses are supplied by a lead pipe?

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Table 6 | Summary of plumbosolvency testing results (from Hayes 2007)

<table>
<thead>
<tr>
<th>Type</th>
<th>Alkalinity</th>
<th>N</th>
<th>Ave pH</th>
<th>pH range</th>
<th>Ave median 30MC Pb (μg l⁻¹)</th>
<th>Range in median 30MC Pb (μg l⁻¹)</th>
<th>Ave 16h stagnation Pb (μg l⁻¹)</th>
<th>Range in 16h stagnation Pb (μg l⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ground</td>
<td>High</td>
<td>47</td>
<td>7.8</td>
<td>7.2–8.3</td>
<td>66</td>
<td>23–167</td>
<td>254</td>
<td>65–860</td>
</tr>
<tr>
<td>Surface</td>
<td>High</td>
<td>10</td>
<td>7.7</td>
<td>7.3–8.3</td>
<td>97</td>
<td>62–151</td>
<td>442</td>
<td>222–750</td>
</tr>
<tr>
<td>Surface</td>
<td>Low</td>
<td>101</td>
<td>7.8</td>
<td>6.7–8.8</td>
<td>172</td>
<td>42–694</td>
<td>855</td>
<td>109–3,350</td>
</tr>
</tbody>
</table>

30MC refers to 30 minutes’ contact between water and lead piping during testing.
OCCURRENCE OF LEAD PIPES IN THE EU

The data on the occurrence of lead pipes in the EU is poor; the most recent assessment by Van den Hoven et al. (1999) is summarised in Table 7. The basis of this data is not known, but it is likely to be no more than a collation of best estimates. These estimates suggest that about 25% of houses in the EU have a lead pipe, putting 120 million people at risk in today's 27 member states. There is obvious scope to improve knowledge of the occurrence of lead pipes in the EU.

RDT sampling has been used in the UK to assess compliance with lead at consumers' taps since 1989 and in recent years many UK water companies increased their monitoring frequencies for lead as part of a major plumbosolvency control campaign prompted by the UK Government (for England and Wales, Drinking Water Inspectorate 2000, 2001). As bacteriological sampling frequencies are approximately 10 to 20 times higher than for trace metal 'audit' samples, and as most of the costs of monitoring derive from sampling logistics, the opportunity was taken to take samples for lead analysis at the same locations being sampled for bacteriological parameters. Assuming that no significant operational changes have taken place, it is entirely reasonable to bulk data from several years to increase the size of the datasets. By determining the percentage of samples in which lead was detected analytically (typical limit of detection 1 \( \mu g l^{-1} \) or less) it has been possible to gain an estimate of the percentage of houses with a lead pipe.

This simple approach was used in the plumbosolvency control programme in Wales (Hayes et al. 2008) and enabled a zonal emission model to be calibrated; that good validation of predicted RDT sample results was obtained from actual RDT sample results indicated that this simple methodology was adequate for estimating the percentage of houses with a lead pipe. However, to put this into perspective, over 11,000 results were available across 29 water supply systems, with an average of 383 RDT sample results per scheme.

DISCUSSION

It is undeniable that lead in drinking water poses a health risk. A wide range of studies have found an association between the concentration of lead in drinking water and blood lead concentrations; as an approximation an average concentration of 50 \( \mu g l^{-1} \) lead in drinking water is associated with a blood lead concentration of 20 \( \mu g d l^{-1} \) (Quinn & Sherlock 1990), twice the blood lead action limit of 10 \( \mu g d l^{-1} \) used in the US in the prevention of childhood lead poisoning. Above this action limit, a wide range of health effects have been demonstrated. Even below this action limit, one study (Canfield et al. 2003a) has detected adverse health effects. By clear implication, average lead concentrations in drinking water (at individual dwellings) need to be significantly lower, and the EU standards for lead in drinking water (25 \( \mu g l^{-1} \) in the interim and 10 \( \mu g l^{-1} \) from 2013) are strongly justified.

The question is: to what extent are there health effects arising from lead in drinking water in the EU? The only direct way to answer this question will be to: (a) undertake widespread human health assessment studies linked to properly constructed water lead surveys; and (b) undertake epidemiological assessments of the geographical areas at greatest risk from the occurrence of lead pipes (i.e. the older cities and towns) for the illnesses and disorders most closely linked to lead poisoning, including prenatal mortality rates. It can be noted that blood lead surveillance has been carried out in 46 States in the USA.

Table 7 | Occurrence of lead pipes in Europe (from Van den Hoven et al. 1999)

<table>
<thead>
<tr>
<th>Country</th>
<th>% Pb communication pipes</th>
<th>% Pb supply pipes or internal Pb plumbing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Belgium</td>
<td>19</td>
<td>15 to 30</td>
</tr>
<tr>
<td>Denmark</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>France</td>
<td>39</td>
<td>38</td>
</tr>
<tr>
<td>Germany</td>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>Greece</td>
<td>&lt;1</td>
<td>0</td>
</tr>
<tr>
<td>Ireland</td>
<td>50</td>
<td>51</td>
</tr>
<tr>
<td>Italy</td>
<td>2 (?)</td>
<td>5 to 10</td>
</tr>
<tr>
<td>Luxembourg</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>Netherlands</td>
<td>6</td>
<td>8</td>
</tr>
<tr>
<td>Portugal</td>
<td>?</td>
<td>32</td>
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There are several major drivers for the health impacts of lead in drinking water to be properly determined in the EU and for the prerequisite sampling issues to be resolved. The World Health Organisation (WHO 2004) has advocated the adoption of drinking water safety planning, a proactive approach for hazard identification, risk assessment and risk management. To ignore the potential problems of lead in drinking water would be entirely inconsistent with this recommended safety planning approach. The European Commission is expected to include ‘risk management strategies’ in the next revision of the drinking water directive (Hoekstra 2008), thereby making risk assessment and management a legal requirement within the EU.

The Protocol on Water and Health sets a number of legal obligations for its 21 Parties (United Nations 2007) in relation to the prevention and control of ‘water-related disease’. Article 2(1) defines ‘water-related disease’ as ‘any significant adverse effects on human health, such as death, disability, illness or disorders, caused directly or indirectly by the condition, or changes in the quantity or quality, of any waters’. The health impact of lead in drinking water clearly falls within this definition and the Protocol requires:

- Adequate supplies of wholesome drinking water—Article 4(2)(a).
- Effective systems for monitoring situations likely to result in water-related disease—Article 4(2)(e).
- Preventive action to avoid incidents of water-related disease, with special consideration for vulnerable people—Articles 5(e) and 5(k).
- Establishing and publishing local targets, that need to be achieved or maintained for a high level of protection against water-related disease—Articles 6(2) and 6(3).
- Participating States shall establish surveillance and early warning systems, contingency plans and response capacities—Article 8(3).
- The development of indicators to show how far action on water-related disease has been successful—Article 12(b).
- Preparation of water-management plans and schemes for improving water supply—Article 14(a).

CONCLUSIONS

Sampling deficiencies have undoubtedly caused an underestimation of the extent of non-compliance with the EU standards for lead in drinking water. This position is incompatible with the Protocol on Water and Health and conflicts with the recommendations of the World Health Organisation for drinking water safety planning.

Random daytime first-draw sampling at consumers’ taps is the most appropriate method for determining the extent of problems with lead in drinking across a water supply zone, although the number of samples taken must be far greater than the minimum sampling frequencies required by the EU drinking water directive. Such increased sampling for lead is feasible logistically, at little extra cost, if linked to bacteriological sampling visits to consumers’ dwellings.

Emerging data indicates that many water supplies in the EU still have a lead problem and that about 65% of dwellings that have a lead pipe are likely to exceed the EU 10 \( \mu \text{g} \ell^{-1} \) standard, the legal requirement from 2013.

Lead pipes are common across the EU, but there are uncertainties about occurrence in many countries as the property owner is usually responsible for domestic plumbing. An initial estimate is that about 25% of dwellings in the EU have a lead pipe, but in some cities the percentage of houses affected will be much higher.

The health effects of lead in drinking water are well proven, although the extent of these effects has not been quantified at the EU scale. An initial estimate is that 120 million people in the EU are at potential risk from lead in drinking water.

Although the total removal of all lead pipes must be the goal, there are practical difficulties as well as financial constraints. The dosing of ortho-phosphate (a corrosion inhibitor), once optimised, has been shown to be very effective, based on experience in the UK, but this approach is being resisted elsewhere in the EU on philosophical and environmental grounds.

Once the health risks from lead in drinking water have been properly evaluated, there can be no doubt that solutions to these difficulties and concerns will be found. Continuing to ignore these risks is no longer an option.
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