Scientific Committee on Health and Environmental Risks

SCHER

Lead Standard in Drinking Water

The SCHER adopted this opinion at its 11th plenary of 11 January 2011
About the Scientific Committees

Three independent non-food Scientific Committees provide the Commission with the scientific advice it needs when preparing policy and proposals relating to consumer safety, public health and the environment. The Committees also draw the Commission’s attention to the new or emerging problems which may pose an actual or potential threat. They are: the Scientific Committee on Consumer Safety (SCCS), the Scientific Committee on Health and Environmental Risks (SCHER) and the Scientific Committee on Emerging and Newly Identified Health Risks (SCENIHR) and are made up of external experts.

In addition, the Commission relies upon the work of the European Food Safety Authority (EFSA), the European Medicines Evaluation Agency (EMEA), the European Centre for Disease prevention and Control (ECDC) and the European Chemicals Agency (ECHA).

SCHER

Opinions on risks related to pollutants in the environmental media and other biological and physical factors or changing physical conditions which may have a negative impact on health and the environment, for example in relation to air quality, waters, waste and soils, as well as on life cycle environmental assessment. It shall also address health and safety issues related to the toxicity and eco-toxicity of biocides.

It may also address questions relating to examination of the toxicity and eco-toxicity of chemical, biochemical and biological compounds whose use may have harmful consequences for human health and the environment. In addition, the Committee will address questions relating to methodological aspect of the assessment of health and environmental risks of chemicals, including mixtures of chemicals, as necessary for providing sound and consistent advice in its own areas of competence as well as in order to contribute to the relevant issues in close cooperation with other European agencies.

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All Declarations of working group members are available at the following webpage: http://ec.europa.eu/health/scientific_committees/environmental_risks/members_wg/index_en.htm

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Opinion to be cited as:

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1. BACKGROUND
Lead is a cumulative poison that can severely affect the central nervous system. In adults, about 10% of ingested lead is absorbed but in children this figure can be 4-5 times higher. It has been shown to cause neurological deficits in children, affecting the IQ by 3 to 4 points for each 10 µg Pb/dL increase in the blood levels of children.

In 1993, WHO proposed an amended guideline value for lead in drinking water of 10 µg Pb/L, based upon an overall assessment of studies of human populations. In view of the revision of the 1980 Drinking Water Directive in 1998, the European Commission’s Directorate-General for the Environment (then DG XI) requested the advice of the Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) on the application of this value and on the timeframe in which the necessary changes should take place in order to minimize the potential health risks resulting from water with concentrations of lead at the 1980 directive’s maximum acceptable concentration (50 µg/L).

In 1994, CSTEE considered that human exposure to lead should be minimized on health grounds. It was agreed that, in conformity with the precautionary principle, the maximum level of lead in drinking water should be ultimately reduced to 10 µg/L as recommended by the World Health Organization (WHO). From an ecotoxicity point of view, a concentration of lead in water around 10 µg/L and perhaps even 1 µg /L would be a better objective than the present guideline of 50 µg/L.

In 1998, the revised drinking water directive, adopted by the Council in 1998, a maximum concentration of lead of 10 µg/L was laid down, effective 25 December 2013.

In 2008, WHO confirmed the guideline value for lead in drinking water (10 µg/L), fully taking into account that the use of lead-containing additives in petrol is decreasing, that lead concentrations in air are declining, and that lead intake from drinking water constitutes a greater proportion of the total intake.

2. TERMS OF REFERENCE
In its letter of 18 March 2010, the Institut Européen pour la gestion raisonnée de l’environnement (IEGRE) questioned the rationale for this 10 µg/L limit and asked the Commission to raise the limit concentration of lead in drinking water to “maybe 15 or 20 µg/L”. The reason behind this request is that the significant reduction of the sources of lead other than water makes possible an increase of lead in the drinking water while maintaining the same total intake.

Furthermore, IEGRE questions WHO’s method used for deriving the lead standard of 10 µg/L and IEGRE states that since lead is considered to be a parameter without threshold, there is no need to decrease the provisional tolerable weekly intake (PTWI).

IEGRE requested this matter to be considered, taking into account:

- the application of a standard of 10 µg Pb/L effective 25 December 2013 on;
- the toxicological and epidemiological evidence concerning lead;
- the confirmation of the lead guidelines for drinking water by WHO in 2003, referring clearly to the decrease in the use of lead in car fuels and in lead-containing solder in the food industry;
- sensitive subgroups of the population such as pregnant women, infants and children.

DG ENV sought SCHER’s opinion on IEGRE’s request, asking in particular whether, following the reduction of the use of lead in car fuels and in the food processing industry, relaxing the standard from 10 µg/L to 15 or 20 µg/L will not cause a potential risk for human health.
3. OPINION

3.1. Sources of lead
Lead is an environmental contaminant that occurs naturally and may be widely distributed by anthropogenic activities. Lead ions are the predominant form in the environment. The restrictions on lead compounds as additives to gasoline and measures to regulate lead in paint, gasoline, food cans and pipes during the past 30 years have resulted in a pronounced reduction in lead exposure and a significant decrease in lead blood levels in the general population.

Human exposure to lead can occur via food, water, air, soil and dust (SCHER, 2009). Food, including drinking water, is the major source of exposure to lead for the majority of population (EFSA, 2010). Some consumer products may represent an additional exposure source (SCHER, 2010). The use of lead in pipes for water circulation remains an important source of lead in some areas. The formerly widespread use of lead-based paint and soil and/or dust ingestion are additional sources of potential exposure (especially indoors), specifically for infants (VRAR, 2008).

3.2. Health effects of lead

Toxicokinetics. The absorption of lead after ingestion is dependent on age and physiological status. In adults, only 5 to 10% of orally ingested lead is absorbed by the gastrointestinal tract (Alexander et al., 1973; Rabinowitz et al., 1973). In contrast, children absorb up to 70% of an oral lead dose. Absorbed lead is transferred from the blood to tissues and is accumulated in bones. Placental transfer of lead occurs in humans as early as the 12th week of gestation and continues throughout development. The body burden of lead increases from birth to adulthood.

Toxic effects of lead exposures. Detailed reviews of the effects of lead in human populations after occupational and environmental exposures and in experimental animals are available (ATSDR, 2007; EFSA, 2010; IPCS, 2000; VRAR, 2008).

In primates, significant behavioural and cognitive effects have been observed following postnatal exposure resulting in blood lead levels ranging from 11 to 33 µg/dL (WHO, 2008). Lead is not a teratogen in experimental animals. Renal tumours have been induced in experimental animals exposed to high concentrations of lead compounds in the diet, and IARC has classified inorganic lead compounds as Group 2B (possible human carcinogen). However, there is evidence from studies in humans that adverse toxic effects other than cancer may occur at low doses of lead. Due to the non-genotoxic mechanism of lead carcinogenicity to the kidney, exposure guideline intakes, derived to protect from the neurotoxic effects of lead, are also expected to be protective regarding potential carcinogenic effects (WHO, 2006; VRAR, 2008; SCHER, 2009).

In humans, specifically in children, the hematopoetic system, the central nervous system and the kidney are the most sensitive target organs for lead toxicity. Infants and children up to 6 years of age and pregnant women are most susceptible to the adverse health effects of lead due to the higher bioavailability and a higher sensitivity of the developing central nervous system to lead-induced effects.

Inhibition of delta-aminolaevulinic dehydratase in children is observed at blood lead concentrations ≥ 5 µg/dL. However, adverse effects on the hematopoietic system are not associated the inhibition at this level (WHO, 2008). Lead also interferes with calcium metabolism, both directly and by interfering with vitamin D metabolism at blood lead levels > 11 µg/dL in children. There is electrophysiological evidence of effects on the nervous system in children with blood lead levels well below 30 µg/dL. Encephalopathy with seizures may occur in children at blood lead concentrations much below those that induce similar effects in adults. Furthermore, decreased nerve conduction velocity and
cognitive deficits are observed at lower lead blood concentrations in children than in adults (ATSDR, 2007).

**Epidemiological evidence.** Neurobehavioral outcomes were associated (EFSA, 2010) with lead blood concentrations below 10 µg/dL (Wang et al., 2008). The WHO International Program on Chemical Safety (IPCS) concluded that there is an effect of lead exposure at low levels on the intelligence quotient (IQ) and estimates the effect to be about 1 to 3 points of IQ lost for each 2.4-10 µg/dL lead in blood. Studies show that school performance may also be adversely affected by early childhood exposure to lead (Kordas et al., 2006; Miranda et al., 2007).

Newer studies and a recent pooled analysis confirmed the relationship between blood lead levels and IQ in children and conclude that a threshold level cannot be identified (Lanphear et al., 2005). Therefore, EFSA determined the 95th percentile lower confidence limit of the benchmark dose (BMD) of 1% extra risk (BMDL01) of 1.2 µg lead /dL as a reference point for the risk characterization of lead when assessing the risk of intellectual deficits in children measured by the Full Scale IQ score (EFSA, 2010). The BMDL01 of 1.2 µg lead/dL for neurodevelopmental effects corresponds to a dietary lead exposure in infants and children of 0.5 µg lead/kg bw per day.

In a detailed evaluation of the available neurobehavioral toxicity of lead in children, the EU Risk Assessment Report (VRAR, 2008) concludes that effects of lead exposure cannot be measured at lead concentrations below 5 µg/dL due to limited precision of the behavioural testing and methods for lead quantisation in blood. Therefore, the RAR proposes an "epistemic" threshold for impacts of lead upon societal cognitive resources of 5 µg/dL noting that effects may occur at blood levels less than 10 µg/dL and a "real" threshold for lead effects cannot be identified. The use of an epistemic threshold of 5 µg/dL is intended to provide a population benchmark for blood levels in children and serves as a target to reduce the probability that individual children may exceed a blood lead level of 10 µg/dL (VRAR, 2008).

### 4. CURRENT GUIDELINES FOR LEAD IN DRINKING WATER

WHO proposed a guideline value of 10 µg/L for lead in drinking water considering an allocation of 50% of the weekly tolerable intake (PTWI) to water (WHO, 2008). The weekly intake (PTWI) was considered more appropriate as peaks in exposure levels and daily-exposure variations are less relevant for lead due to its long half-life (WHO, 2003, 2008). The WHO proposal was integrated in the new EU Drinking Water Directive 98/83/EC (03.11.1998) where the limit of 10 µg/L was set for implementation on 25.12.2013. Based on the WHO guidelines, the USA decided to propose a limit value of 15 µg/L, taking into account the reduction of other sources of lead. EFSA concluded that the PTWI for lead is no more valid due to the absence of a demonstrable threshold for lead-induced effects.

### 5. EXPOSURE TO LEAD

To derive the potential impact of changes in drinking water limits for lead on lead intake and blood levels, SCHER performed an exposure assessment for lead using different levels of lead in drinking water and the dietary intake derived in a recent EFSA opinion (EFSA, 2010). The levels of lead in drinking water used in this exposure assessment started with the future maximum tolerable concentration limit of 10 µg/L and went up to concentrations of 15-30 µg/L. Since exposure to lead is commonly monitored by measuring blood lead levels, both estimated intakes and resulting blood levels were calculated. Biomonitoring data and modelling allows the conversion of the blood concentrations into daily dietary intake. EFSA calculated that a blood lead level of 1.2 µg/dL (the BMDL01 for neurodevelopmental effects) corresponds to a daily intake of 0.5 µg/kg bw (EFSA, 2010).
Dietary lead exposure for average adult consumers in Europe was estimated by EFSA to range from 0.36 to 1.24 µg/kg body weight (bw) per day for average consumers and from 0.73 to 2.43 µg/kg bw per day for high consumers. Cereals, and vegetables were the most important contributors to lead exposure in the general European population. Drinking water was included in the calculation of the intake by EFSA. The mean lead levels in drinking water range from 1.5 - 3 µg/L (EFSA, 2010). When using these concentrations and the contribution of diet to lead intake, drinking water gives only a 4% contribution to the dietary intake.

Compared to adults, children’s exposures to lead are higher because they are anabolic and absorb nutrients more effectively. Therefore, children are considered the most sensitive population, and the exposure scenarios are developed specifically for infants and for the age groups 1-18 years. Data on water consumption for children aged 1-18 years are used consistent with the approach used in the opinion on fluoridation of drinking water (SCHER, 2010).

5.1. Nursing and/or formula-fed infants

Breast- and formula-fed infants are exposed to lead from breast milk or infant formula consumption as well as to lead accumulated during prenatal exposure and released through neonatal bone turnover (Gulson et al., 2003). EFSA (2010) selected an age of three months and a body weight of 6.1 kg with an estimated average daily consumption of about 800 g, and a high consumption of 1,200 g of breast milk and/or infant formula for the exposure assessment of infants below six months of age. EFSA assumed average lead drinking water levels of 2.1 µg/L. Mean lead level in ready-to-drink infant formula was estimated as 2.0 to 4.7 µg/kg resulting in an estimated average exposure in the range of 0.3 to 0.6 µg/kg bw per day and an exposure of 0.4 to 0.9 µg/kg bw per day in high consumers.

Using EFSA’s average (800 g) and high (1200 g) daily formula consumption estimates of a 3 months old child the SCHER has calculated the increased daily exposures and blood lead levels resulting from drinking water that contains 10, 15, 20 and 30 µg Pb/L. (see Table 1).

Table 1: Estimated exposure of a 3 months old child to lead (Pb) and blood Pb levels at different concentrations of lead in drinking water

<table>
<thead>
<tr>
<th>Age (mo.)</th>
<th>Weight (kg)</th>
<th>Formula (mL)</th>
<th>net mL of water 90%</th>
<th>Daily intake of lead (µg/kg bw) and expected blood lead levels (µg/dL) at different concentrations of lead in the drinking water (from 10 to 30 µg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>10 µg/L</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Daily intake of Pb</td>
</tr>
<tr>
<td>3</td>
<td>6.1</td>
<td>800</td>
<td>720</td>
<td>1.2</td>
</tr>
<tr>
<td>3</td>
<td>6.1</td>
<td>1200</td>
<td>1080</td>
<td>1.8</td>
</tr>
</tbody>
</table>

Our calculations show that for formula fed infants of 3 months of age, under all scenarios, the daily intake of lead is above the EFSA-accepted level of 0.5 µg/kg bw per day. Those daily intakes would result in blood Pb levels significantly higher than the reference value of 1.2 µg/dL used by EFSA for risk characterization of lead. According to EFSA (2010) an increase in blood lead levels from 2.8 to 8.5 µg/dL results in an IQ loss of about 5 points (average daily consumption); an increase in blood levels from 4.3 to 13.1 µg/dL depresses the IQ by about 7 points (high consumption).
5.2. Children age groups from 1 to 18

For children between 1 and 18 years of age, EFSA performed a detailed dietary exposure assessment for lead. For the age group **1-3 years**, mean dietary lead exposure was estimated to range from 1.1 to 5.5 µg/kg bw; from 0.8 to 4.8 µg/kg for the age group **4-7 years**; and from 0.4 to 3.4 µg/kg bw per day for the age group of **8-14 years**. The **age group 14 to 18** years is included in the adult group with estimated lead dietary exposure ranging from 0.4 to 2.4 µg/kg bw per day (EFSA, 2010). Tables 2a and 2b show calculations on lead exposure at a consumption of one litre of water including water-based beverages. These data show that, without considering the dietary intake from food, any increase in the level of lead in the drinking water will increase the daily intake per body weight of children (in particular for the age group 1-8 years) above the BMDL01 of 0.5µg/kg bw/d resulting in blood Pb levels above the EFSA-determined reference value for risk assessment (1.2 µg/dL).

**Table 2a:** Children’s exposure to lead from drinking water, assuming consumption of one litre of drinking water per day at different concentrations of lead in drinking water

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Body weight range</th>
<th>Daily intake of lead (µg/kg bw) at various levels of lead in drinking water calculated for the upper and lower bounds of the body weight range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Children</td>
<td>1-3</td>
<td>10.9–13.4</td>
<td>0.7–0.9</td>
</tr>
<tr>
<td></td>
<td>4–8</td>
<td>16.7–25.3</td>
<td>0.4–0.6</td>
</tr>
<tr>
<td></td>
<td>Boys</td>
<td>9–13</td>
<td>28.1–45.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>14–18</td>
<td>50.8</td>
</tr>
<tr>
<td>Boys</td>
<td>9–13</td>
<td>28.5–46.1</td>
<td>0.2–0.3</td>
</tr>
<tr>
<td></td>
<td>14–18</td>
<td>50.3–56.6</td>
<td>0.2</td>
</tr>
</tbody>
</table>

**Table 2b:** Expected blood concentrations of lead, assuming consumption of one litre of drinking water per day at different concentrations of lead in drinking water.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Age</th>
<th>Body weight range</th>
<th>Expected blood concentrations (µg/dL) at various levels of lead in drinking water calculated for the upper and lower bounds of the body weight range</th>
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<tbody>
<tr>
<td></td>
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</tr>
<tr>
<td></td>
<td>4–8</td>
<td>16.7–25.3</td>
<td>0.9–1.4</td>
</tr>
<tr>
<td></td>
<td>Boys</td>
<td>9–13</td>
<td>28.1–45.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>14–18</td>
<td>50.8</td>
</tr>
<tr>
<td>Girls</td>
<td>9–13</td>
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<td>28.5–46.1</td>
</tr>
<tr>
<td></td>
<td>14–18</td>
<td>50.3–56.6</td>
<td>0.5</td>
</tr>
</tbody>
</table>
5.3. Pregnant women and foetus

Lead crosses the placenta, and the blood lead concentration of the foetus is similar to that of the mother (Graziano et al., 1990). Because infant formula and other foods for infants contain lead in the water used for their preparation, breastfed infants are exposed to less lead (Gulson et al., 1998).

Prenatal exposure of the growing foetus is of potential concern. Direct transfer from maternal blood to foetus is the main route of exposure. The foetal/maternal cord blood lead concentration ratio is approximately 0.9. Characteristics of females 20 to 40 years of age were used as a surrogate for pregnant women to assess lead exposure in utero (EFSA, 2010). Dietary exposure to lead in this group ranges from 0.4 to 1.3 µg/kg bw/d for average consumers and 0.7 to 2.6 µg/kg bw/d per day for high consumers. Based on these figures, EFSA concluded that a risk to the developing foetus from maternal lead exposure cannot be excluded. Calculations estimate the exposure to lead from drinking water and water-based beverages using one, 1.5 and two litres per day (WHO default) and a body weight of 60 kg. Lead exposure range from 0.17-0.33 µg/kg bw/d at 10 µg/L of drinking water, 0.33-0.67 µg/kg bw/d at 20 µg/L and 0.50-1.0 µg/kg bw/d at 30 µg/L.

In the EU-RAR, exposure modelling for all possible exposure sources of lead is performed considering inhalation, intake with food, water, and soil and dust using both a typical exposure scenario and a worst-case scenario (VRAR, 2008). In the worst-case scenario, drinking water levels of lead are assumed to be at the future standard of 10 µg/L. This exposure modelling predicts lead blood levels above the “threshold” of 5 µg/dL in the worst-case scenario for children 1 – 2 years of age in both rural and urban areas and for children 5 – 6 years of age in urban areas (Table 3). For the typical scenario, the lead blood levels are predicted to be often close to 5 µg/dL.

Table 3: Predicted average blood levels [µg/dL] of lead in the general population under typical and worst-case conditions in rural and urban environments including the % of the children’s population with blood lead levels above 10 µg/dL (VRAR, 2008).

<table>
<thead>
<tr>
<th>Setting</th>
<th>Typical exposure scenario age 1–2</th>
<th>Worst-case scenario age 1–2</th>
<th>Typical exposure scenario age 5–6</th>
<th>Worst-case scenario age 5–6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rural</td>
<td>2.5 (0.1 %)</td>
<td>5.6 (11 %)</td>
<td>1.3 (0.001 %)</td>
<td>3.8 (2 %)</td>
</tr>
<tr>
<td>Urban</td>
<td>4.4 (4 %)</td>
<td>12.2 (67 %)</td>
<td>2.8 (0.3 %)</td>
<td>8.1 (33 %)</td>
</tr>
</tbody>
</table>

6. SUMMARY AND OVERALL CONCLUSIONS

In view of the available data, SCHER concludes that even at levels below 10 µg of lead/dL, adverse effects on children’s intelligence development can be observed.

EFSA concluded that when using a low concentration of lead in drinking water (2.1 µg/L), the dietary exposure of sensitive subgroups (infants and foetal exposures) to lead results in a Margin-of-Exposure value of less than 1 indicating that risks to young children regarding neurodevelopmental effects cannot be excluded. Therefore, effects may occur even at the proposed new drinking-water standard for lead.
The EU-RAR integrated a drinking water concentration of 10 µg/L in the exposure assessment (table 3). This resulted in blood levels of lead above the “epistemic” threshold of 5 µg/L for worst-case exposure scenarios. Therefore, an increase in the drinking water concentrations from 10 µg/L to 15 or 20µg/L will further increase exposures and may result in blood levels above the “epistemic” threshold even in a typical exposure scenario. A change of the lead standard for drinking water, i.e. relaxing the standard from 10µg/L to 15 up to 30µg/L, will cause additional risk to human health, especially to the mental and neurological development of children aged 0-14 years.

The exposure assessment shows that blood concentrations of lead in populations exposed to drinking water at the level of increased drinking water limits may rise to lead blood levels observed in the 1970s, thus compensating the beneficial effects of the costly reduction of lead exposures of the general population over the last 30 years. Therefore, SCHER sees no scientific basis to support an increase in the lead drinking water standard. In fact, a further reduction in lead intake is warranted for risk reduction.

7. ABBREVIATIONS

BMD Benchmark dose
BMDL Benchmark dose level
bw Body weight
CONTAM Panel Panel on Contaminants in the Food Chain
CSTEE Scientific Committee on Toxicity, Ecotoxicity and the Environment
DG ENV Directorate-General for the Environment
EU European Union
EFSA European Food Safety Authority
IARC International Agency for Research on Cancer
IEGRE Institut Européen pour la gestion raisonnée de l’environnement
IQ Intelligence quotient
IEUBK Integrated Exposure Uptake Biokinetic Model
IPCS WHO International Program on Chemical Safety
NHANES National Health and Nutrition Examination Survey
PTWI Provisional tolerable weekly intake
RAR Risk Assessment Report
TDI Total daily intake
WHO World Health Organization

8. REFERENCES

elevated blood lead during pregnancy in a population surrounding a lead smelter in Kosovo, Yugoslavia. Environ Health Perspect 89, 95-100.


