

Statement on the effects of lead on maternal health

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Introduction and Background - Statement on the effects of lead on maternal health

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Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment

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Introduction

1. The Scientific Advisory Committee on Nutrition (SACN) last considered maternal diet and nutrition in relation to offspring health in its reports on 'The influence of maternal, fetal and child nutrition on the development of chronic disease in later life' (SACN, 2011) and on 'Feeding in the first year of life' (SACN, 2018). In the latter report, the impact of breastfeeding on maternal health was also considered.
2. In 2019, SACN agreed to conduct a risk assessment on nutrition and maternal health focusing on maternal outcomes during pregnancy, childbirth and up to 24 months after delivery; this would include the effects of chemical contaminants and excess nutrients in the diet.
3. SACN agreed that, where appropriate, other expert Committees would be consulted and asked to complete relevant risk assessments e.g., in the area of food safety advice. This subject was initially discussed by COT during the horizon scanning item at the January 2020 meeting with a scoping paper being presented to the Committee in July 2020. This included background information on a provisional list of chemicals proposed by SACN. It was noted that the provisional list of chemicals was subject to change following discussion by COT who would be guiding the toxicological risk assessment process: candidate chemicals or chemical classes can be added or removed as the COT considered appropriate. The list was brought back to the COT with additional information in September 2020. Following a discussion at that meeting, the COT agreed that papers on a number of components should be prioritised and to this end, papers on iodine, vitamin D and dietary supplements have been or will be presented to the Committee. The remaining list of compounds were to be triaged on the basis of toxicity and exposure.
4. Following discussion of the first prioritisation paper on substances to be considered for risk assessment by the COT, the Committee decided that each of the heavy metals (lead, mercury, cadmium and arsenic) should be considered in separate papers. The following paper discusses the risks posed to maternal health by lead in the diet and the environment.

Background

5. The Merck Index (15th edition, 2013) describes lead (Pb) as a bluish-white-to-silvery grey group 14 metal, with atomic number 82 and a relative atomic mass of its most abundant isotope of 208. It occurs naturally in the earth's crust at an abundance of about 0.002%, chiefly as lead sulfide (PbS). It is relatively soft and malleable, has a high density, low melting point, and is relatively inert. These properties led to a long history of use in a variety of applications, including in domestic articles such as drinking vessels and plates and in water and drainage pipes (plumbing, from "plumbum", the Latin word for lead). More recently it has been used in paints, ceramic pigments, cosmetics, insecticides, hair dyes, lead-acid batteries, and in the "anti-knock" agent tetraethyl lead in petrol (to improve spark-plug efficiency). Due to its long-known toxicity, many of these uses have been substituted with less toxic alternatives but lead is still used in various applications such as car batteries, pigments, ammunition, cable sheathing, weights for lifting, weight belts for diving, lead crystal glass, some solders and as radiation shielding in the nuclear industry.

6. The Joint FAO/WHO Committee on Food Additives (JECFA) (FAO/WHO, 2011) state that lead contamination of food arises mainly from the environment or from food processing, handling and packaging. Atmospheric lead can contaminate food through deposition on agricultural crops. Water is another source of lead contamination of food. Although lead exists in both organic and inorganic forms, only inorganic lead has been detected in food. Specifically, the major contributors to lead exposure are: cereal products, potatoes, cereal grains (except rice), cereal-based mixed dishes and leafy vegetables.

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Previous evaluations

7. The safety of lead in food has previously been evaluated by the European Food Safety Authority (EFSA) Panel on Contaminants in the Food Chain (CONTAM Panel) (EFSA, 2010) and JECFA (2011). The US Agency for Toxic Substances and Disease Registry has also reviewed the toxicity of lead (ATSDR, 1999). These evaluations are discussed in more detail in the discussion paper for Lead in the Maternal Diet (COT, 2022).

ADME

8. Lead absorption has been determined in a number of studies, and in adult humans is approximately 10 % of the ingested dose (Rabinowitz *et al.*, 1976). Lead absorption from the gastrointestinal tract appears to be higher in infants and children than in adults, with an average lead absorption in infants of about 42 % of intake (Ziegler *et al.*, 1978). This is supported by animal studies which indicate that gastrointestinal absorption rates for lead are greater in the very young than in older animals (Forbes *et al.*, 1972; McMichael *et al.*, 1986).

9. Approximately 95 % of lead in adult tissues and 70 % in children resides in mineralised tissues such as bones and teeth. This reflects changing turnover rates throughout an individual's lifetime, with a slower turn-over of lead in the bones of adults than those of children. The lead which has accumulated in adult bone, can replenish lead eliminated from the blood by excretion, long after the external exposure has ended. It can also be a source of lead transfer to the fetus when the maternal skeleton is resorbed for the production of the fetal skeleton. Gulson *et al.* (1997) found that increases in blood lead (bPb) of 20 % were detected in the mother during pregnancy. Additionally, it is worth noting that previous studies have suggested that bPb levels increased only in the second

half of pregnancy, however, Gulson (1997) found two subjects with increased bPb levels in the first trimester of pregnancy. Skeletal contribution to bPb level was $31 \pm 19\%$ (mean \pm SD), with the remaining increase suggested to be due to increased absorption of dietary lead and decreased elimination of lead. These results showed that mobilisation from long-term stores (i.e., bone) was a significant contributor to bPb levels during pregnancy. However, it is pertinent to clarify that lead levels in bone accumulate over a period of many years before pregnancy and are largely determined by the pre-maternal rather than maternal diet and as such fall beyond the remit of this paper.

10. Bolan *et al* (2021) examined the intestinal permeability/bioaccessibility of lead, as influenced by gut microbes and chelating agents using an *in vitro* gastrointestinal/Caco-2 cell intestinal epithelium model. In the presence of gut microbes and chelating agents, there was a significant decrease of 7.9 % in the permeability coefficient of lead, indicative of a decrease in lead absorption from the gut.

11. Rădulescu and Lundgren (2019) reviewed the recent pharmacokinetic models for lead. Absorption takes place via ingestion, inhalation and to a lesser extent through the skin. The effectiveness of gastrointestinal absorption depends on the quantity and type of food consumed prior to lead ingestion. The efficiency of gastrointestinal absorption of water-soluble lead is also age-dependent and is higher in children than in adults. The authors cited several older studies regarding the different distributions of lead in human soft tissues, highlighting that the major organ is the liver (Barry, 1975; Gross *et al.* 1975; Schroeder, H. A. & Tipton, I. H. 1968; Barregård *et al.* 1999; and Gerhardsson *et al.* 1995).

Toxicity

12. The acute effects of lead, from exposure of short duration, can manifest as muscle pain, fatigue, abdominal pain, headache, vomiting, seizures, and coma. While there are insufficient data to establish a dose-response relationship for acute toxicity relative to bPb, anecdotally, symptoms that cause individuals to seek medical intervention can occur at bPb levels of $\sim 30 \mu\text{g/dL}$ with signs and symptoms increasing in severity with increasing bPb. Chronic lead poisoning from low level, repeated exposure can result in clinical signs of persistent vomiting, encephalopathy, lethargy, delirium, convulsions and coma. Depending on the location or organ in question, chronic adverse effects can occur at bPb levels $\leq 5 \mu\text{g/dL}$ (ATSDR, 2020).

13. The International Agency for Research on Cancer (IARC) has classified lead compounds as probably carcinogenic to humans (Group 2A) on the basis of limited evidence of carcinogenicity in humans and sufficient evidence in animals. Organic lead compounds were considered not to be classifiable as to their carcinogenicity to humans (Group 3) because there was inadequate evidence for carcinogenicity in humans and animals. The mechanisms of lead-induced cancer in experimental systems are complex, appearing to involve oxidative stress, interaction with zinc finger proteins, altered cell signalling pathways and interactions with cellular genetic machinery by high affinity lead-binding proteins (IARC, 2006).

14. Flora *et al.* (2012) and Wani *et al.* (2015) reviewed the toxicity of lead. They determined that the central nervous system, erythropoietic system and the kidneys are the most affected systems but overall, all bodily systems are adversely affected by the presence of this metal.

15. It has been estimated that systolic pressure is approximately 1 mm Hg higher for each doubling of bPb level, without any clearly identifiable threshold (EFSA, 2010). In the dose response (DR) modelling for cardiovascular effects, EFSA selected a 1 % change in systolic blood pressure as a benchmark response (BMR), which was within the range that could have significant consequences for human health at a population level. An average BMDL01 of 36 µg/L bPb was calculated from two longitudinal and two cross-sectional studies (Glenn *et al.*, 2003; Vupputuri *et al.*, 2003; Nash *et al.*, 2003; Glenn *et al.*, 2006).

16. Both reduced glomerular filtration rate (GFR) associated with exposures to average bPb levels of <200 µg/L and increased serum creatinine in subjects with blood lead levels below 100 µg/L have been observed. EFSA (2010) selected a 10 % increase in the prevalence of chronic kidney disease as a BMR for renal effects and a BMDL10 of 15 µg/L bPb was calculated using data from a cross-sectional study conducted in the USA (Navas-Acien *et al.*, 2009).

17. The COT (2013), like EFSA (2010), determined that neurodevelopmental effects represent the most sensitive endpoint for effects in the developing fetus whilst also being protective of the other toxicological end points in the mother. The study used for the benchmark dose modelling undertaken by EFSA (2010) is described in the following paragraph.

18. Data from a study by Lanphear *et al.* (2005), were used by both EFSA and JECFA for DR modelling of neurodevelopmental effects (EFSA, 2010; and FAO/WHO, 2011). EFSA commissioned their own analysis of the raw data from the

respective author's study in contrast to JECFA who used the dose-response data presented in the publication. The Lanphear *et al.* (2005) study was a pooled analysis of data from seven prospective cohort studies concerning the quantitative relationship between performance on IQ tests and measures of bPb concentration, among children who have had regular follow-ups from infancy. The primary outcome measure was full-scale IQ, assessed at an age between four years 10 months and 10 years. This was related to four measures of bPb: concurrent bPb (the most recent measurement before IQ was assessed), maximum bPb (the highest concentration of bPb that had been measured at any time before IQ was assessed), average lifetime bPb (the mean of bPb measurements from age 6 months up to the time that IQ was assessed), and early childhood bPb (the mean of measurements between 6 and 24 months of age). After adjustment for covariates, IQ was inversely related to each of these measures of bPb (Lanphear *et al.*, 2005). A reduction in IQ of 1 point was selected as the BMR and this corresponds to a BMDL01 of 0.5 µg lead/kg bw/day.

19. The toxicology of lead specifically in the context of pregnancy outcomes and its effects on maternal health have also been previously reviewed and the results are briefly described below.

20. Maternal lead exposure is associated with multiple poor birth outcomes including pre-term delivery and small for gestational age (SGA) births (Chen, (2006); Jelliffe-Pawlowski *et al.* (2006); Taylor *et al.* (2015); Vigeh *et al.* (2011); and Zentner *et al.*, (2006)).

21. Low to moderate lead exposure may also increase the risk of spontaneous abortion during early pregnancy (Hertz-Picciotto, 2000; Ou *et al.* 2020). Additionally, Lamadrid-Figueroa *et al.* (2007) determined that women in the upper tertile of the plasma/blood Pb ratio had twice the incidence rate for spontaneous abortion than those in the lower tertile ($p = 0.02$). Borja-Aburto *et al.* (1999) evaluated the risk of spontaneous abortion from low or moderate Pb exposures during the first trimester. The odds ratio for spontaneous abortion was 1.8 for every 5 µg/dL increase in bPb. Conversely, however, Vigeh *et al.* (2011) did not find a significant difference between spontaneous abortion cases and ongoing pregnancies, suggesting that in apparently healthy women, low bPb levels (mean <5 µg/dL) in early pregnancy may not be a risk factor for spontaneous abortion. Based on available data, there is a concern that long-term lead exposure may adversely affect fetal viability as well as fetal and early childhood development, as lead is reported to cross the placenta readily.

22. Hu *et al.* (2006) found that both maternal plasma and whole blood lead, during the first trimester (but not in the second or third trimester), were significant predictors ($p < 0.05$) of poorer Mental Development Index (MDI) scores. Postnatal blood lead levels in the offspring were less strongly correlated with MDI scores indicating that the adverse effect of lead exposure on neurodevelopment may be most pronounced during the first trimester.

23. Poropat *et al.* (2017, from abstract) and Ikechukwu *et al.* (2012, from abstract) found a correlation between an increase in blood lead and the development and progression of preeclampsia. Ikechukwu *et al.* (2012) further state that the increases observed in lead levels were paralleled by decreases in serum calcium and phosphorus levels. Conversely, Liu *et al.* (2019) did not find an association between the development of preeclampsia and bPb levels.

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24. The DR modelling and establishment of an HBGV have been reviewed and summarised in the COT statement (2013). The COT discussed the three endpoints assessed by EFSA (cardiovascular, renal and neurodevelopmental effects) and concluded that the most relevant were neurodevelopmental effects. This is summarised in brief in the following paragraphs.

Benchmark Dose Modelling

25. A study by Lanphear *et al.* (2005) (paragraph 28), was used by both EFSA and JECFA for DR modelling of neurodevelopmental effects (EFSA, 2010; and FAO/WHO, 2011). The raw data generated in the Lanphear *et al.* (2005) study was obtained and modelled by EFSA for use in their benchmark calculations. JECFA based their analysis on the pooled data presented by Lanphear *et al.* (2005) for their benchmark calculations. The DR modelling was previously described in a COT statement in 2013 relating to Pb in the infant diet and has been summarised in the following paragraphs (COT, 2013).

26. Budtz-Jørgensen (2010) was commissioned by EFSA to calculate a BMDL for the association of lead with the development of intellectual function, by modelling of the raw data by Lanphear *et al.* (2005). The benchmark calculations used regression models with full IQ score as the dependent variable, and adjustment for birth weight, Home Observation for Measurement of the Environment (HOME) score (The HOME Inventory is an index that reflects the quality and quantity of emotional and cognitive stimulation in the home environment (Lanphear *et al.*, 2005)), maternal education and maternal IQ, all of which were significantly associated with IQ in the dataset. BMD and BMDL values were calculated for a 1 % change in full scale IQ score (a decrease in IQ by 1 point), taking concurrent blood lead, maximum blood lead, average lifetime blood lead and early childhood blood lead as alternative exposure metrics. The dose-response models applied were logarithmic, linear, and a piecewise linear function with breakpoint at 100 µg/L.

27. For the assessment of risk, EFSA took as a point of departure, the BMDL01 value of 12 µg/L from the piecewise linear dose-response model for concurrent blood lead. Concurrent blood lead concentration exhibited the strongest relationship with IQ, and the piecewise linear model showed a better fit to the data than the linear model. The logarithmic model generally gave an even better fit than the piecewise linear model, but the differences were small, and EFSA preferred the latter because, taking into account the mathematical properties of the models, they considered that it provided “less uncertain

estimates of the BMDL01". Using the US Environmental Protection Agency's (EPAs) **Integrated Exposure and Uptake Biokinetic** (IEUBK) model, the blood lead BMDL01 of 12 µg/L was estimated to correspond to a dietary lead exposure in infants and children of 0.5 µg lead/kg bw/day (EFSA, 2010).

28. JECFA (FAO/WHO, 2011) also used data from the Lanphear *et al.* (2005) analysis for DR modelling based on the authors' pooled analysis. Models were based on concurrent blood lead levels since they showed the highest correlation with IQ. Initially, six different models were considered – four with linear form and two sigmoidal. From these, a bilinear model (unlike the piecewise linear model used by EFSA, this did not constrain the inflexion in the dose-response relationship to be at a pre-specified blood lead concentration) was chosen to characterise the relationship of blood lead to IQ, since it provided a better fit than four of the other models, and it was considered that it would give better estimates of effect than the one other model with similar fit, when non-dietary exposures to lead were unknown or highly variable. Using this model, the chronic dietary exposure of a 20 kg child corresponding to a decrease of 1 IQ point was estimated to be 0.6 µg/kg bw/day with a 90 % confidence interval of 0.2 - 7.2 µg/kg bw/day.

29. The differences between the EFSA and JECFA analyses are small and reflect inevitable uncertainties in the specification of the mathematical models. The COT (COT, 2013) noted that both were influenced by an apparently steep dose-response at low levels of lead exposure (blood lead levels less than 75 µg/L), which was based on few data from a single study in Rochester, USA, and may have rendered the BMDL values conservative. The COT had based its risk characterisation on the EFSA BMDL01, which is between the EFSA BMD01 and the lower 90 % confidence limit for the BMDL01 calculated by JECFA. The EFSA BMDL01 corresponds to a dietary exposure of 0.5 µg/kg bw/day (COT, 2013).

30. As the BMDL was for a small effect (a one-point difference in IQ), derived from a pooled analysis of multiple cohort studies of exposures in infants and children, and is likely to be conservative (paragraphs 26-27), EFSA therefore concluded that a margin of exposure (MOE) of 10 or greater should be sufficient to ensure that there was no appreciable risk of a clinically significant effect on IQ. At lower MOEs, but greater than 1.0, the risk is likely to be low, but not such that it could be dismissed as of no potential concern. (EFSA, 2010)

31. In relation to women of childbearing age, the COT agreed that the BMDL01 and corresponding dietary exposure of 0.5 µg/kg bw per day should be used in the current risk assessment for risk characterisation purposes.

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Exposure from food

32. The FSA Exposure Assessment Team has provided dietary exposure data on lead for women of childbearing age (16 – 49 years of age) (Table 1, Appendix 1). The food commodities that result in the highest exposures to lead are green vegetables, miscellaneous cereals and other vegetables with mean exposure values of 0.0088, 0.0080 and 0.0063 µg/kg bw/day and 97.5th percentile values 0.034, 0.028 of 0.019 µg/kg bw/day, respectively. The total exposures via food were calculated as 0.12 µg/kg bw/day (mean) and 0.23 µg/kg bw/day (97.5th percentile).

Exposure from drinking water

33. Data on concentrations of lead in water had previously been provided by the Drinking Water Inspectorate (DWI) (for England and Wales), the Drinking Water Quality Regulator (DWQR) for Scotland and Northern Ireland Water. The concentration data from 2019 for lead in drinking water are given in Table 2, Appendix 1.

34. The FSA Exposure Assessment Team has provided values for water consumption for women of child-bearing age of 8 (median) and 32 (97.5th percentile) g (ml) of water per kg bodyweight per day. Using the upper bound mean lead concentration values in drinking water (2.15, 0.48 and 1.1 for England/Wales, Scotland and Northern Ireland respectively), the calculated exposures to lead from drinking water are shown in Table 1.

Table 1. Calculated mean and 97.5th percentile exposures for women of childbearing age to lead from drinking water, using the mean upper bound concentration values ($\mu\text{g}/\text{kg bw}/\text{day}$).

Region	N (number of women)	Median **	97.5th percentile **
England and Wales*	10967	0.00024	0.00098
Scotland	436	0.000054	0.00021
Northern Ireland	122	0.00013	0.00050

*Using 99th percentile lead concentration.

** Average body weight of 70.3 kg for women of childbearing age used for exposure calculation. Value provided by the FSA Exposure Assessment Team from years 1 – 11 of the rolling National Diet and Nutrition Survey, NDNS (Bates et al., 2014, Bates et al., 2016, Roberts et al., 2018).

Exposure from the air

35. Defra provide data on air pollution throughout the UK. An interactive map (Defra, 2020) shows that the majority of the country in 2020 had an average air lead concentration of $<10 \text{ ng}/\text{m}^3$, with major urban centres in England and Wales having concentrations of $10 - 50 \text{ ng}/\text{m}^3$.

36. The WHO estimates that the average inhalation rate for a 70 kg adult is 20 m³/day (WHO, 2000).

37. As a worst-case scenario, if an adult female were to be constantly exposed to an air concentration of 50 ng lead/m³ then this would result in a daily exposure to 1000 ng of lead from the air. For women with an average body weight of 70 kg, (value provided by the FSA Exposure Assessment Team from years 1 - 11 of the rolling National Diet and Nutrition Survey, NDNS (Bates *et al.*, 2014, Bates *et al.*, 2016, Roberts *et al.*, 2018) this gives an exposure of 14 ng/kg bw (0.014 µg/kg bw/day).

38. This assumes that there is full absorption of all lead in the particles inhaled, but this depends upon particle sizes and since some of the inhaled dose may become trapped in parts of the nasopharynx, these inhalation values are probably an overestimate, but may contribute a small amount to ingested lead.

Exposure from soil and dust

39. People may be exposed to lead through swallowing dirt that contains lead. Ingestion of contaminated soil is often as a result of “hand-to-mouth” activity and while being a more important route of exposure for toddlers and children, soil and dust still present a potential source of intake in adults, for example, from the surface of unwashed vegetables.

40. Lead concentrations in soil are influenced both by underlying lithological lead concentrations and by anthropogenic release of lead. Lead was measured in topsoil from England from a depth of 0-15 cm as part of a Defra-commissioned project (Ander *et al.*, 2011).

41. Table 2 shows the lead exposures from soil for women of child-bearing age. Mean and 75th percentile lead concentrations from soil in regions classified as rural, semi-urban or urban were used to assess potential exposures of adults through soil ingestion. An ingestion rate of 50 mg soil/day was assumed based on the rate used by the Environment Agency in their Contaminated Land Exposure Assessment (CLEA) model (Environment Agency, 2009) and was based on a consensus value from studies by USEPA (1997) and Otte *et al.* (2001). It is a combined value for soil and dust as most of the evidence used to determine the ingestion rate does not differentiate between soil and household dust. Furthermore, the evidence base for selecting a representative soil ingestion rate for adults is much smaller than that for children and as such USEPA (1997) cautioned that the value is highly uncertain and based on a low level of

confidence.

Table 2. Median and 75th percentile exposure values for women of childbearing age to lead from soil. Soil lead concentrations taken from the Defra-commissioned contaminants in the soils of England report (Ander *et al.* 2011) and an ingestion of 50 mg soil/day provided by the Environment Agency (2009).

Mean/ 75th percentile	Region	Soil concentration of lead (mg/kg)	Lead ingestion (μg/kg bw/day)*
Mean	Rural	35	0.025
Mean	Semi-Urban	57	0.041
Mean	Urban	166	0.118
75th percentile	Rural	46	0.033
75th percentile	Semi-Urban	100	0.071
75th percentile	Urban	322	0.229

* Average body weight for women of childbearing age used for ingestion rate = 70.3 kg, value provided by the FSA Exposure Assessment Team from years 1 - 11 of the rolling National Diet and Nutrition Survey, NDNS (Bates *et al.*, 2014, Bates *et al.*, 2016, Roberts *et al.*, 2018).

42. The data presented are representative of lead concentrations in the soil in England only. There have been no individual studies investigating the lead levels in soils of Wales, Scotland or Northern Ireland.

43. No recent data were available for levels of lead measured in household dust in the UK.

44. Pica behaviour is described as the craving for and intentional ingestion of substances that are not described as food. Globally, it is thought to affect up to 28 % of pregnant women, albeit with a high degree of geographic variability (Fawcett *et al*, 2016). Therefore, pica presents a potential route of exposure to lead from soil. However, pica has not been considered as part of this statement due to the lack of data available for the consumption of soil as part of pica behaviour.

Aggregate Exposure

45. Aggregate exposure to lead from food, drinking water, soil and dust, and air was estimated by considering a number of scenarios based on available data. Table 3 shows scenarios of aggregate exposure from the sources listed above and includes estimates of average and high exposure from these sources as indicated below.

46. Average and high exposure for food and drinking water represent the mean and 97.5th percentile exposure as described in paragraphs 30 - 32. Data for exposure from drinking water in England and Wales were used as this represented the highest exposure compared to Scotland and Northern Ireland. The contribution from air in all scenarios is based on average inhalation rates and the maximum concentration from a range reported for England and Wales. For exposure from soil and dust, the average and high exposure represent the mean and 75th percentile exposure respectively for the region with the highest exposure (i.e., urban region as shown in Table 2 and paragraphs 37 - 41).

Table 3. Aggregate exposure to lead from food, drinking water, soil, dust and air*.

Scenarios	Aggregate exposure (µg/kg bw/day)
Average exposure from all sources ^a	0.25
High exposure from all sources ^b	0.49
High exposure from food and mean exposure from all other sources ^c	0.36

High exposure from drinking water and mean from other sources^d 0.26

High exposure from soil and dust and mean from other sources^e 0.36

a This scenario represents a summation of average exposure from food, water and soil and a value for air*.

b Exposure is based on summation of 97.5th percentile estimates for food and water, 75th percentile for dust and soil and a value for air*.

c Exposure is based on summation of 97.5th percentile estimates for food and the averages for water, dust and soil and a value for air*

d Exposure is based on summation of 97.5th percentile estimates for drinking water and the averages for food, dust and soil and a value for air*

e Exposure is based on summation of 75th percentile estimate for soil and dust and averages for food, water and a value for air*.

*The contribution from air in all scenarios is based on average inhalation rates and the maximum concentration identified for England and Wales as shown in paragraphs 33 - 34.

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Risk characterisation - Statement on the effects of lead on maternal health

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47. Potential risks from maternal exposures to lead were characterised by margins of exposure (MOEs), calculated as the ratio of the BMDL of 0.5 µg/kg bw/day to estimated exposures from diet, soil and air. As the BMDL was for a small effect (a one-point difference in IQ), derived from pooled analysis of multiple cohort studies of exposures in infants and children, and is likely to be conservative (see paragraph 26-27), EFSA therefore concluded that a margin of exposure of 10 or greater should be sufficient to ensure that there was no appreciable risk of a clinically significant effect on IQ. At lower MOEs, but greater than 1.0, the risk is likely to be low, but not such that it could be dismissed as of no potential concern. (EFSA, 2010).

48. In 2013, the COT further concluded that an MOE of >1 can be taken to imply that at most, any risk is likely to be small. MOEs <1 do not necessarily indicate a concern, but scientific uncertainties (e.g. because of potential inaccuracies in the assessment of exposures, failure to control completely for confounding factors, and the possibility that the samples of children studied have been unrepresentative simply by chance) mean that a material risk cannot be ruled out. This applies particularly when MOEs are substantially <1 (COT, 2013).

Food

49. Using the estimated dietary exposure of 0.5 µg/kg bw day, equivalent to the BMDL01 of 12 µg/L blood Pb concentration, for effects of Pb on developmental neurotoxicity (EFSA, 2010), the MOEs for women of childbearing age from the highest-lead-containing food groups in the total diet study are given in Tables 3 and 4 for the highest measured mean and 97.5th percentile lead levels, respectively.

Table 3. Calculated MOEs for lead in the food groups with the highest measured mean lead concentrations (upper bound) for the total diet in women aged 16 to

49 years of age.

Commodity	Mean lead exposure ($\mu\text{g}/\text{kg}$ bw/day)*	MOE for 0.5 $\mu\text{g}/\text{kg}$ bw/day
Green vegetables	0.0088	57
Misc. cereals	0.0080	63
Other vegetables	0.0063	79
Total in all food	0.12	4.2

The calculated exposures were compared to the dietary intake value of 0.5 $\mu\text{g}/\text{kg}$ b.w. per day which corresponds to the blood BMDL01 of 12 $\mu\text{g}/\text{L}$ for developmental neurotoxicity. "Total" was obtained by summing the individual upper bound estimates for all foods assessed (see Table 1, Appendix 1).

* Average body weight for women of childbearing age used for exposure = 70.3 kg, value provided by the FSA Exposure Assessment Team from years 1 - 11 of the rolling National Diet and Nutrition Survey, NDNS (Bates *et al.*, 2014, Bates *et al.*, 2016, Roberts *et al.*, 2018).

Table 4. MOEs for lead in the dietary commodities with the highest measured 97.5th percentile lead concentrations (upper bound) and for the total diet in women aged 16 to 49 years of age.

Commodity	97.5th percentile lead exposure ($\mu\text{g}/\text{kg}$ bw/day)*	MOE for 0.5 $\mu\text{g}/\text{kg}$ bw/day
Green vegetables	0.034	15

Misc. cereals	0.023	22
Other vegetables	0.019	26
Total in all food	0.23	2.2

The calculated exposures were compared to the dietary intake value of 0.5 µg/kg b.w. per day which corresponds to the blood BMDL01 of 12 µg/L for developmental neurotoxicity. “Total” was obtained by summing the individual upper bound estimates for all foods assessed (see Table 1, Appendix 1).

*Average body weight for women of childbearing age used for exposure = 70.3 kg, value provided by the FSA Exposure Assessment Team from years 1 – 11 of the rolling National Diet and Nutrition Survey, NDNS (Bates et al., 2014, Bates et al., 2016, Roberts et al., 2018).

50. Neither the mean nor the 97.5th percentile exposure MOEs for the foods with the highest measure of lead, nor for the total amount of lead in food as a whole as reported by the NDNS, has a value of 1 or lower, indicating that any risk of toxicity from lead in food is likely to be small.

Drinking water

51. The MOEs for lead in drinking water are shown in Table 5.

Table 5. MOEs for lead in drinking water using the concentration data provided by the water regulators for England and Wales, Scotland and Northern Ireland and consumption data provided by the FSA Exposure Assessment Team.

Region	97.5th percentile lead exposure (µg/kg bw/day) **	MOE for 0.5 µg/kg bw/day
England and Wales *	0.00098	510

Scotland	0.00021	2400
Northern Ireland	0.00050	1000

The calculated exposures were compared to the dietary intake value of 0.5 µg/kg b.w. per day which corresponds to the blood BMDL01 of 12 µg/L for developmental neurotoxicity MOEs rounded to 2 s.f.

*Using 99th percentile lead concentration.

**Average body weight for women of childbearing age used for exposure = 70.3 kg, value provided by the FSA Exposure Assessment Team from years 1 - 11 of the rolling National Diet and Nutrition Survey, NDNS (Bates *et al.*, 2014, Bates *et al.*, 2016, Roberts *et al.*, 2018).

52. The MOEs for intake of lead from drinking water from Scotland and Northern Ireland are above 10, indicating that there was no appreciable risk from lead in drinking water. The MOE for intake of lead from drinking water in England and Wales is greater than 1, therefore indicating that any risk of toxicity from lead in drinking water is likely to be small.

Air

53. The inhaled exposure level would have minimal impact upon total lead exposure. Relative to the BMDL01 corresponding dietary intake value derived by EFSA, a conservative intake from air gives an MOE of 36 for developmental neurotoxicity.

Soil and Dust

54. The MOEs for exposures from lead in soil are shown in Table 6 and Table 7.

Table 6. MOEs for lead in soil from regions in England using the mean concentrations of lead. Soil lead concentration data are taken from Defra (Ander *et al.* 2011) and a soil ingestion rate from the Environment Agency (2009).

Region	Mean lead exposure ($\mu\text{g}/\text{kg bw}/\text{day}$) *	MOE for 0.5 $\mu\text{g}/\text{kg bw}/\text{day}$
Rural	0.025	20
Semi-Urban	0.041	12
Urban	0.118	4

The calculated exposures were compared to the dietary intake value of 0.5 $\mu\text{g}/\text{kg b.w.}$ per day which corresponds to the blood BMDL01 of 12 $\mu\text{g}/\text{L}$ for developmental neurotoxicity.

* Average body weight for women of childbearing age used for ingestion rate = 70.3 kg, value provided by the FSA Exposure Assessment Team from years 1 - 11 of the rolling National Diet and Nutrition Survey, NDNS (Bates *et al.*, 2014, Bates *et al.*, 2016, Roberts *et al.*, 2018).

Table 7. MOEs for lead in soil from regions in England using the highest measured (75th percentile) lead concentrations. Soil lead concentration data taken from Defra (Ander *et al.* 2011) and a soil ingestion rate from the Environment Agency (2009).

Region	75th Percentile lead exposure ($\mu\text{g}/\text{kg bw}/\text{day}$) *	MOE for 0.5 $\mu\text{g}/\text{kg bw}/\text{day}$
Rural	0.033	15
Semi-Urban	0.071	7
Urban	0.229	2

The calculated exposures were compared to the dietary intake value of 0.5 $\mu\text{g}/\text{kg bw}/\text{day}$ which corresponds to the blood BMDL01 of 12 $\mu\text{g}/\text{L}$ for developmental

neurotoxicity.

* Average body weight for women of childbearing age used for ingestion rate = 70.3 kg, value provided by the FSA Exposure Assessment Team from years 1 - 11 of the rolling National Diet and Nutrition Survey, NDNS) (Bates *et al.*, 2014, Bates *et al.*, 2016, Roberts *et al.*, 2018).

55. The MOEs for soil ingestion from regions across England are all greater than 1, therefore, any risk of toxicity from lead in soil is likely to be small. Furthermore, the soil ingestion rate could be an overestimate, particularly as it is a combined value for soil and dust. The ingestion rate is also highly uncertain as it is based upon a small and variable evidence base. Consequently, as the assumptions in the exposure estimates are conservative, the actual soil ingestion rate and lead exposure through this route could be much lower.

Aggregate Characterisation

56. A combined exposure assessment, considering exposure to lead from all sources, relative to the estimated dietary exposure of 0.5 µg/kg bw day, equivalent to the BMDL01 of 12 µg/L blood Pb concentration, for effects of Pb on developmental neurotoxicity (EFSA, 2010), gives a MOE in the range of 0.9-2 depending on the individual contribution to the total from each source (food, drinking water, soil/dust). In a scenario where there are high exposures to lead from all sources (food, drinking water, soil/dust) the MOE is 0.9, and in a scenario where there are average levels of exposure to each source, the MOE is 2. In all aggregate scenarios, any risk of toxicity from lead is likely to be small.

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57. Lead is a heavy metal pollutant that is ubiquitous in the environment and is thus present in the diet of the general population, including women of childbearing age. Levels have, nonetheless, fallen since the phasing out of lead in petrol, plumbing and paints.

58. Lead in pregnant women can cause increased blood pressure and may be associated with preeclampsia and premature birth.

59. Lead accumulates in the body, therefore, adverse effects can occur from long term dietary exposures at levels below those which cause acute toxicity. Neurotoxicity has been identified at lower levels of exposure than for other effects, and the developing brain appears to be more vulnerable than the mature brain. It has not been possible to demonstrate a threshold level of exposure below which the neurodevelopmental effects of lead do not occur.

60. EFSA (2010, updated 2013) derived BMDLs for effects on neurodevelopment, renal function and systolic blood pressure and provided values as both $\mu\text{g/L}$ in blood and the corresponding calculated dietary intake values in $\mu\text{g/kg bw/day}$. The COT determined that the most relevant reference point was the BMDL01 for neurodevelopmental toxicity as this value is for the most sensitive effect and hence will be protective for the other endpoints in the mother.

61. Exposure of women of childbearing age to lead, in food at the mean and 97.5th percentile of consumption of commodities with the highest concentrations and from soil and dust give MOEs exceeding 1, for effects on developmental neurotoxicity relative to the dietary intake value of 0.5 $\mu\text{g/kg bw/day}$ corresponding to the BMDL01. These MOEs indicate that any risk of toxicity from lead in food is likely to be small.

62. There is no appreciable risk of a significant effect on IQ in children following maternal exposure to lead in air in the UK.

63. The calculated MOEs for soil exposure indicate that in semi-urban and urban areas in the 75th percentile of measured lead levels, the risk to human health is low, however, this is based upon ingestion rates of high uncertainty.

64. Toxicity will depend on total exposure to lead from all sources, and it is therefore important to consider this to determine an overall likely level of risk. A scenario in which there are high levels of exposure to lead from food, drinking water and soil/ dust would result in an MOE of 0.9, however, this assumes a worst-case for exposure from all sources for a prolonged period of time. A scenario in which there are average levels of exposure to lead from food, drinking water and soil/ dust would result in an MOE of 2. These MOE values indicate that any aggregate risk of toxicity from lead in relation to the maternal diet together and other potential sources of maternal exposure is likely to be small.

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Abbreviations

AGA	Adequate for gestational age
ATSDR	Agency for Toxic Substances and Disease Registry
BMD	Benchmark Dose
BMDL01	Benchmark Dose Lower Limit for 1% change in effect
BPb	Blood lead concentration
CI	Confidence interval
CLEA	Contaminated Land Exposure Assessment
COT	Committee on Toxicity
DNA	Deoxyribonucleic acid
DWI	Drinking Water Inspectorate
DWQR	Drinking Water Quality Regulator
EFSA	European Food Safety Authority
FSA	Food Standards Agency
IQ	Intelligence Quotient

JECFA	Joint FAO/WHO Committee on Food Additives
kPa	KiloPascals
L	Litre
MDI	Mental development index
mmHg	Millimetres of mercury
MOE	Margin of exposure
NDNS	National Diet and Nutrition Survey
OR	Odds ratio
Pb	Lead
SBP	Systolic blood pressure
SD	Standard deviation
SGA	Small for gestational age
µg	Microgram

Search terms

The references cited in this discussion paper are of publications found in PubMed searches and references therein, using the following search terms:

Pb AND

Maternal health

Pre-conception

Conception

Post-partum

Toxicity

Mechanism

ADME

Toxicokinetics

Absorption

Distribution

Metabolism

Excretion

Biomarker

Exposure

Pre-eclampsia

Abortion

Pica

Pica AND Pregnancy

Soil AND Pregnancy

References

ATSDR (2020) Toxicological profile for lead (update). (Agency for Toxic Substances and Disease Registry.) U.S. Department of Health and Human Services, Atlanta, GA.: [ATSDR Lead Tox Profile \(cdc.gov\)](https://www.cdc.gov/ATSDR/lead/toxprofile/)

Ander EL, Johnson CC, Cave MR, and Palumbo-Roe B. 2011. Normal background concentrations of contaminants in the soils of England. Available data and data exploration. British Geological Survey Commissioned Report, CR/11/145. 124pp: [Normal background concentrations of contaminants in English and Welsh soils - British Geological Survey \(bgs.ac.uk\)](#)

Barregård, L. et al. Cadmium, mercury, and lead in kidney cortex of the general Swedish population: a study of biopsies from living kidney donors. Environmental Health Perspectives 1999 **107**(11), 867.

Barry PS. A comparison of concentrations of lead in human tissues. Occupational and Environmental Medicine 1975 **32**(2): 119-139.

Bolan S, Seshadri B, Keely S, Kunhikrishnan A, Bruce J, Grainge I, Talley NJ, Naidu R. Bioavailability of arsenic, cadmium, lead and mercury as measured by intestinal permeability, Scientific Reports. 2021 **11**(1):14675.

Borja-Aburto VH, Hertz-Picciotto I, Rojas Lopez M, Farias P, Rios C, Blanco J. Blood lead levels measured prospectively and risk of spontaneous abortion. American Journal of Epidemiology 1999 Sep 15;150(6):590-7.

Bound J, Harvey P, Francis B, Awwad F, Gatrell A. Involvement of deprivation and environmental lead in neural tube defects: a matched case-control study Archives of Disease in Childhood. 1997 **76**(2): 107-112.

Braun J. Erythrocyte zinc protoporphyrin. Kidney International Supplement 1999 **69**: S57-60.

Budtz-Jørgensen E. 2010. Scientific/technical report submitted to EFSA. An international pooled analysis for obtaining a benchmark dose for environmental lead exposure in children. (Question No. EFSA-Q-2009-01078) <https://doi.org/10.2903/sp.efsa.2010.EN-47>

Chen PC, Pan IJ, Wang JD. Parental exposure to lead and small for gestational age births. American Journal of Industrial Medicine. 2006 49(6):417-22.

COT, 2013, Statement on the potential risks from lead in the infant diet, [cotstatlead.pdf \(food.gov.uk\)](#)

COT, 2022, Discussion paper on the effects of lead on maternal health, [Effects of Lead on Maternal Health \(food.gov.uk\)](#)

Defra, 2020, [UK Ambient Air Quality Interactive Map \(defra.gov.uk\)](https://defra.gov.uk). Accessed: 20.04.22.

EFSA Panel on Contaminants in the Food Chain (CONTAM); Scientific Opinion on Lead in Food. **EFSA Journal** 2010; 8(4):1570. [151 pp.]. doi: [10.2903/j.efsa.2010.1570](https://doi.org/10.2903/j.efsa.2010.1570).

Environment Agency, 2009, [Microsoft Word - 0901115 CLEA Report for publication.doc \(publishing.service.gov.uk\)](https://publishing.service.gov.uk). Accessed 20.04.22.

Fawcett E, Fawcett J, Mazmanian D. A meta-analysis of the worldwide prevalence of pica during pregnancy and the postpartum period, *International Journal of Gynecology & Obstetrics* 2016 133(3): 277-283.

Flora G, Gupta D, Tiwari A. Toxicity of lead: A review with recent updates. *Interdisciplinary Toxicology* 2012 **5**(2):47-58.

Forbes G, Reina J, Effect of Age on Gastrointestinal Absorption (Fe, Sr, Pb) in the Rat, *The Journal of Nutrition* 1972 **102**(5): 647-652.

Garza A, Vega R, Soto E. Cellular mechanisms of lead neurotoxicity *Medical Science Monitor* 2006; **12**(3): RA57-65.

Gerhardsson, L. et al. Lead in tissues of deceased lead smelter workers. *Journal of Trace Elements in Medicine and Biology* 1995 **9**(3), 36-143.

Glenn BS, Bandeen-Roche K, Lee BK, Weaver VM, Todd AC, Schwartz BS. Changes in systolic blood pressure associated with lead in blood and bone. *Epidemiology*. 2006 **17**: 538-544.

Glenn BS, Stewart WF, Links JM, Todd AC, Schwartz BS. The longitudinal association of lead with blood pressure. *Epidemiology*. 2003 **14**: 30-36

Gross, S. B., Pfitzer, E. A., Yeager, D. W. & Kehoe, R. A. Lead in human tissues. *Toxicology and Applied Pharmacology* 1975 **32**(3), 638-651.

Gulson BL, Jameson CW, Mahaffey KR, Mizon KJ, Korsch MJ, Vimpani G. Pregnancy increases mobilization of lead from maternal skeleton *Journal of Laboratory and Clinical Medicine*. 1997; **130**(1):51-62.

Hertz-Picciotto I. The evidence that lead increases the risk for spontaneous abortion. *American Journal of Industrial Medicine* 2000 **38**:300-309.

Hu H, Téllez-Rojo MM, Bellinger D, Smith D, Ettinger AS, Lamadrid-Figueroa H, Schwartz J, Schnaas L, Mercado-García A, Hernández-Avila M. Fetal lead exposure at each stage of pregnancy as a predictor of infant mental development. *Environmental Health Perspectives*. 2006 **114**(11):1730-5.

Ikechukwu IC, Ojareva OIA, Ibhagbemien, AJ, Okhoaretor OF, Oluwatomi OB, Akhalufo OS, Oluwagbenga AT, Chigaekwu MN. Blood Lead, Calcium, and Phosphorus in Women With Preeclampsia in Edo State, Nigeria. *Archives of Environmental & Occupational Health* 2012 67(3): 63-69.

JECFA (2011) Evaluation of certain food additives and contaminants: seventy-third report of the Joint FAO/WHO Expert (WHO technical report series; no. 960)1. Meeting (73rd: 2010, Geneva, Switzerland). IV.Series.ISBN 978 92 4 120960 (NLM classification: WA 712) ISSN 0512-3054.

Jelliffe-Pawlowski LL, Miles SQ, Courtney JG, Materna B, Charlton V. Effect of magnitude and timing of maternal pregnancy blood lead (Pb) levels on birth outcomes. *Journal of Perinatology* 2006; **26**: 154-162.

Karri SK, Saper RB, Kales SN. Lead encephalopathy due to traditional medicines *Current Drug Safety* 2008 Jan;3(1):54-9.

Lamadrid-Figueroa H, Téllez-Rojo MM, Hernández-Avila M, Trejo-Valdivia B, Solano-González M, Mercado-García A, Smith D, Hu H, Wright RO. Association between the plasma/whole blood lead ratio and history of spontaneous abortion: a nested cross-sectional study. *BMC Pregnancy Childbirth* 2007 **7**:22.

Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, Canfield RL, Dietrich KN, Bornschein R, Greene T, Rothenberg SJ, Needleman HL, Schnaas L, Wasserman G, Graziano J and Roberts R. Low-level environmental lead exposure and children's intellectual function: An international pooled analysis. *Environmental Health Perspectives* 2005. 113(7): 894-899.

Liu T, Zhang M, Guallar E, Wang G, Hong X, Wang X, Mueller NT, Trace Minerals, Heavy Metals, and Preeclampsia: Findings from the Boston Birth Cohort. *Journal of the American Heart Association*. 2019 **8**(16): e012436.

Nash D, Magder L, Lustberg M, Sherwin RW, Rubin RJ, Kaufmann RB, Silbergeld EK. Blood lead, blood pressure, and hypertension in perimenopausal and postmenopausal women. *Journal of the American Medical Association*. 2003 **289**: 1523-1532.

Navas-Acien A, Tellez-Plaza M, Guallar E, Muntner P, Silbergeld E, Jaar B, Weaver V. Blood cadmium and lead and chronic kidney disease in US adults: a joint analysis. *American Journal of Epidemiology*.2009 **170**: 1156-1164.

Naylor, K.E., Iqbal, P., Fledelius, C., Fraser, R.B. and Eastell, R., The Effect of Pregnancy on Bone Density and Bone Turnover. *Journal of Bone and Mineral Research* 2009, **15**: 129-137.

Needleman HL. Rabinowitz M. Leviton A. Linn S. Schoenbaum S. The relationship between prenatal exposure to lead and congenital anomalies. *Journal of the American Medical Association* 1984, **251**, 2956–2959.

Ou J, Peng P, Qiu L, Teng L, Li C, Han J, Liu X. Effect of Lead Exposure on Spontaneous Abortion: A Case-Control Study *Clinical Laboratory* 2020 **66**(5).

Otte P, Lijzen J, Otte J, Swartjes F, Versluijs C, Evaluation and revision of the CSOIL parameter set. RIVM Report 711701021. Bilthoven: National Institute of Public Health and Environment 2001.

Poropat AE, Laidlaw MAS, Lanphear B, Ball A, Mielke HW, Blood lead and preeclampsia: A meta-analysis and review of implications, *Environmental Research* 2018 **160** 12–19.

Rădulescu A, Lundgren S. A pharmacokinetic model of lead absorption and calcium competitive dynamics *Anca Scientific Repots* 2019 **9**:14225.

Sanders T, Liu Y, Buchner V, Tchounwou PB. Neurotoxic Effects and Biomarkers of Lead Exposure: A Review [Reviews of Environmental Health. 2009; 24\(1\): 15-45.](#)

Schroeder, H. A. & Tipton, I. H. The human body burden of lead. *Archives of Environmental Health: An International Journal* 1968 **17**(6), 965–978.

Taylor CM, Golding J, Emond AM. Adverse effects of maternal lead levels on birth outcomes in the ALSPAC study: a prospective birth cohort study. *British Journal of Obstetrics and Gynaecology* 2015; **122**:322–328.

UESPA, 1997. Exposure Factors Handbook. August 1997. Washington: United States Environmental Protection Agency.

Vigeh M, Saito H, Sawada S. Lead exposure in female workers who are pregnant or of childbearing age. *Indian Health*. 2011 **49**(2):255-61.

<https://doi.org/10.2486/indhealth.ms1192> Epub 2010 Dec 16.

Vupputuri S, He J, Muntner P, Bazzano LA, Whelton PK, Batuman V. Blood lead level is associated with elevated blood pressure in blacks. *Hypertension*.2003 **41**: 463-468.

Wani AL, Ara A, Usmani JA Lead toxicity: a review, *Interdisciplinary Toxicology* 2015 **8**(2): 55 - 64.

Wells EM, Navas-Acien A, Herbstman JB, Apelberg BJ, Silbergeld EK, Caldwell KL, Jones RL, Halden RU, Witter FR, Goldman LR. Low-level lead exposure and elevations in blood pressure during pregnancy. *Environmental Health Perspectives*. 2011 **119**(5):664-9.

WHO, 2000, WHO air quality guidelines for Europe, 2nd edition. [WHO/Europe | WHO air quality guidelines for Europe, 2nd edition, 2000 \(CD ROM version\)](#). Accessed: 22.04.22.

Zentner LEA, Rondó PHC, Mastroeni SSBS. Lead Contamination and Anthropometry of the Newborn Baby **Journal of Tropical Pediatrics**, 2006 **52** (5) 369-371.

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Lead exposure from food in women of childbearing age

Table 1. Estimated exposure (in $\mu\text{g}/\text{kg bw}/\text{day}$) to lead from foods consumed by women of childbearing age (16-49 years) using data from the total diet study food groups (Bates *et al.*, 2014, 2016; Roberts *et al.*, 2018).

Food Groups	Mean Exposure to lead LB to UB ($\mu\text{g}/\text{kg bw}/\text{day}$)*	97.5th Percentile Exposure to lead LB to UB ($\mu\text{g}/\text{kg bw}/\text{day}$)*
Bread	0.0061	0.015
Miscellaneous Cereals	0.0080	0.023
Carcase meat	0-0.0014	0-0.0065
Offal	0.00057	0.011
Meat products	0.0022	0.010
Poultry	0.0015	0.0055
Fish and seafood	0.0015	0.0071
Fats and oils	0-0.00034	0-0.0010
Eggs	0-0.00052	0-0.0025
Sugars and confectionaries	0.0020	0.0081

Green vegetables	0.0088	0.034
Potatoes	0-0.0045	0-0.013
Other vegetables	0.0063	0.019
Canned vegetables	0.0027	0.013
Fresh fruit	0-0.0047	0- 0.018
Fruit products	0.0041	0.024
Non-alcoholic beverages	0-0.039	0-0.091
Milk	0-0.0037	0-0.014
Dairy products	0.0023	0.0087
Nuts and seeds	0-0.00013	0-0.0011
Alcoholic drinks	0.0053	0.037
Meat substitutes	0.00020	0.0027
Snacks	0.00055	0.0025
Desserts	0.00062	0.0039
Condiments	0.0045	0.016

Tap water	0-0.0048	0-0.021
Bottled water	0-0.0013	0-0.0093
Total	0.057-0.12	0.12-0.23

*Values have been rounded to two significant figures. LB=lower bound; UB=upper bound. "Total" is sum of the values for individual items.

Lead exposures from water in women of childbearing age

Table 2. Concentration of lead in tap water sampled in the nations of the United Kingdom in 2019 ($\mu\text{g/l}$).

Region	N	LB mean	LB SD	UB mean	UB SD
England and Wales*	10967	0.38	0.38	2.15	1.9
Scotland	436	0.34	1.2	0.48	1.2
Northern Ireland	122	0.24	0.79	1.1	0.75

*99th percentile concentration.

LB = lower bound: values below the limit of detection assumed to be zero.

UB = upper bound: values below the limit of detection assumed to be the same as the limit of detection.

References

Bates, B.; Lennox, A.; Prentice, A.; Bates, C.; Page, P.; Nicholson, S.; Swan, G. (2014) National Diet and Nutrition Survey Results from Years 1, 2, 3 and 4 (combined) of the Rolling Programme (2008/2009 - 2011/2012) Available at: [Main heading \(publishing.service.gov.uk\)](http://main.heading.publishing.service.gov.uk)

Bates, B.; Cox, L.; Nicholson, S.; Page, P.; Prentice, A.; Steer, T.; Swan, G. (2016) National Diet and Nutrition Survey Results from Years 5 and 6 (combined) of the Rolling Programme (2012/2013 - 2013/2014): [Main heading \(publishing.service.gov.uk\)](#).

Roberts, C.; Steer, T.; Maplethorpe, N.; Cox, L.; Meadows, S.; Page, P.; Nicholson, S.; Swan, G. (2018) National Diet and Nutrition Survey Results from Years 7 and 8 (combined) of the Rolling Programme (2014/2015 - 2015/2016): [National Diet and Nutrition Survey \(publishing.service.gov.uk\)](#).