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SCIENTIFIC OPINION
Scientific Opinion on Lead in Food
EFSA Panel on Contaminants in the Food Chain (CONTAM)2
On request the European Commission, Question No EFSA-Q-2007-137, adopted on 18 March 2010.

CONCLUSIONS

General

• Lead is a metal that occurs naturally but whose presence in the environment has greatly increased as a result of anthropogenic activities such as mining and smelting and battery manufacturing. Although lead occurs in organic and inorganic forms, it is the inorganic forms that predominate in the environment.
• Control measures taken to regulate lead in paint, petrol, food cans and pipes in Europe since the 1970s have led to a substantial decrease in exposure.

Methods of analysis

• The primary techniques for analysing lead in food samples are based on atomic absorption spectrometry, atomic emission spectrometry and mass spectrometry after digestion of organic material with concentrated acids.

Occurrence and exposure

• Following a call for data, 14 Member States and Norway submitted approximately 140,000 results of lead concentrations in various food commodities and tap water.
• A total of 94,126 results covered the period from 2003 to 2009 and were suitable for calculating lead concentrations in the various food categories. The lead level in approximately two thirds of the samples was below the limit of detection or limit of quantification.
• Mean lead dietary exposure estimates for adults across European countries ranged from 0.36 to 1.24 µg/kg b.w. per day and from 0.73 to 2.43 µg/kg b.w. per day for high consumers, based on lower bound and upper bound assumptions for the level of reporting, respectively.
• Overall, cereals, vegetables and tap water were the most important contributors to lead exposure in the general European population. More specifically, the following food groups were identified as the major contributors to lead exposure: cereal products, followed by potatoes, cereal grains (except rice), cereal-based mixed dishes and leafy vegetables and tap water. Considerable variation between and within countries in the contribution of different food categories/groups exists.
• The available evidence for women of child-bearing age and vegetarians does not indicate a dietary exposure that is different from that of the general adult population.

• Based on limited data, exposure of breast-fed infants was estimated to be 0.21 µg/kg b.w. per day on average or 0.32 µg/kg b.w. per day for high consumers. For infants fed ready-toconsume infant formula, the average exposure estimates range from 0.27 to 0.63 µg/kg b.w. per day, based on lower bound and upper bound assumptions, respectively; for high consumers, lead exposure estimates range from 0.40 to 0.94 µg/kg b.w. per day, respectively.
• For children aged 1 to 3 years mean lead dietary exposure estimates range from 1.10 to 3.10 µg/kg b.w. per day based on lower bound and upper bound assumptions, respectively; for high consumers, lead exposure estimates range from 1.71 to 5.51 µg/kg b.w. per day, respectively.
• For children aged 4 to 7 years mean lead dietary exposure estimates range from 0.80 to 2.61 µg/kg b.w. per day based on lower bound and upper bound assumptions, respectively; for high consumers, lead exposure estimates range from 1.30 to 4.83 µg/kg b.w. per day.
• For adults, non-dietary exposure to lead is likely to be of minor importance for the general population in the EU. House dust and soil can be an important source of exposure to lead for children.
• Lead in blood is considered to be the biomarker of choice for the concentration of lead in soft tissues, and hence recent exposure, although in part it also reflects long term exposure. Bone lead in vivo reflects the long-term uptake and body burden.

Hazard identification and characterisation
• Absorption of lead appears to be highly variable and tends to be higher in children than in adults. It is lower in the presence of food. Absorbed lead is transferred to soft tissues, including liver and kidneys, and to bone tissue, where it accumulates with age.
• Half-lives for lead in blood and bone are approximately 30 days and 10 to 30 years, respectively, and excretion primarily is in urine and faeces.
• The CONTAM Panel identified the following potential adverse effects of lead, the developmental neurotoxicity in young children, cardiovascular effects and nephrotoxicity in adults as the basis for the risk assessment.
• A decrease in Full Scale IQ score was considered to reflect a change in cognitive function in children at ages 4 and higher as it is the most consistently used end-point of cognitive ability assessed in such studies and was used as the critical endpoint for neurodevelopmental effects. An increase in SBP and an increase in the prevalence of CKD as assessed by a decrease in glomerular filtration rate were used as endpoints for adults.
• The computed BMDL were as follows:
Developmental neurotoxicity: BMDL01 = 12 µg/L (B-Pb)
Effects on SBP in adults: BMDL01 = 36 µg/L (B-Pb); 8.1 µg/g (TB-Pb)
Effects on kidney in adults: BMDL10 = 15 µg/L (B-Pb)
• Using the equation of Carlisle and Wade (1992), dietary lead intake values in adults, in whom there is negligible exposure from air and from soil and dust (<1 µg B-Pb/L), corresponding to the respective BMDL dietary intake values were as follows.
Effects on SBP– B-Pb 36 µg/L ~ 90.0 µg/60kg = 1.50 µg/kg per day
Effects on kidney – B-Pb 15 µg/L ~ 37.5 µg/60kg = 0.63 µg/kg b.w. per day.
• Using the IEUBK model, a B-Pb level of 12 µg/L, the BMDL01 dietary intake value for developmental neurotoxicity in 6 year old children, corresponds to a dietary lead intake value of 0.50 µg/kg b.w. per day.
• The CONTAM Panel concluded that the present PTWI of 25 µg/kg b.w. is no longer appropriate and noted that there was no evidence for a threshold for a number of critical endpoints including developmental neurotoxicity and renal effects in adults. Therefore, a margin of exposure approach was applied to risk characterisation.

Risk characterisation
• Dietary exposures to lead based on LB and UB assumptions for average adult consumers in Europe are lower than the BMDL intake value for effects on SBP (1.50 µg/kg b.w. per day),

but vary from above to below the BMDL intake value for effects on the prevalence of CKD, (0.63 µg/kg b.w. per day). The respective MOEs range from 1.2 to 4.2 and from 0.51 to 1.8, respectively. Hence, if exposure were closer to the upper bound estimates, the possibility of effects in some consumers cannot be excluded.

• The limited available evidence does not indicate a different average dietary exposure or risk for vegetarians from the adult population, Consumer groups with higher lead exposure levels include high consumers of game meat (1.98 to 2.44 µg/kg b.w. per day) and high consumers of game offal (0.81 to 1.27 µg/kg b.w. per day). The estimated dietary exposures of these groups are also within, or at the higher end of the range of the respective BMDL intake values.

• Estimated exposure in children up to age seven exceeds the BMDL01 intake level of 0.50 µg/kg b.w. per day for neurodevelopmental effects. The MOE in average 1 to 3 year old child consumers ranged from 0.16 to 0.45, and was only slightly higher in 4 to 7 year old children. Therefore, the possibility of effects in some children cannot be excluded. It was not possible to estimate the potential numbers of children who might be affected, as even in average consumers the MOE was <1.

• Breast-fed 3-month old infants are predicted to have a lead exposure that is below the BMDL01 intake value of 0.50 µg/kg b.w. per day. Lead exposure based on lower bound assumptions in both average and high 3-month old infant consumers of infant formula is below the BMDL01 intake value, but may exceed this level, based on upper bound estimates. Therefore, the possibility of an effect in some infants cannot be excluded.

• Women of 20 to 40 years of age were used as a surrogate for pregnant women to calculate the risk of lead exposure in utero on neurodevelopment in the offspring. Estimates of exposure were at or above the BMDL for neurodevelopmental effects, and the CONTAM Panel concluded that it was not possible to exclude a risk to the developing fetus through exposure of some pregnant female consumers.