ABSTRACT

Lead is a natural environmental contaminant, but its ubiquitous occurrence is the result of anthropogenic activities like the use in the past of lead in water pipes, paint and petrol. The general population is exposed to lead via food, water, air, soil and dust. Food is the major source of exposure to lead, although for children ingestion of soil and dust can also be an important contributor. Lead accumulates in the body, primarily in the skeleton. Half-life for inorganic lead in blood is approximately 30 days, while in bone it is between 10 and 30 years.

SUMMARY

Lead is a natural environmental contaminant, but its ubiquitous occurrence is the result of anthropogenic activities like the use in the past of lead in water pipes, paint and petrol. The general population is exposed to lead via food, water, air, soil and dust. Food is the major source of exposure to lead, although for children ingestion of soil and dust can also be an important contributor. Lead accumulates in the body, primarily in the skeleton. Half-life for inorganic lead in blood is approximately 30 days, while in bone it is between 10 and 30 years.
Due to its long half-life in the body, chronic toxicity of lead is of most concern when considering the potential risk to human health. The central nervous system is the main target organ for lead toxicity. In adults, lead-associated neurotoxicity was found to affect central information processing and short-term verbal memory, to cause psychiatric symptoms and to impair manual dexterity. There is considerable evidence demonstrating that the developing brain is more vulnerable to the neurotoxicity of lead than the mature brain. A number of studies in adults have identified an association between blood lead concentration, elevated systolic blood pressure and chronic kidney disease, at relatively low blood lead levels. The International Agency for Research on Cancer (IARC) classified inorganic lead as probably carcinogenic to humans (Group 2A) in 2006.

Legislative control measures have been taken to remove lead from paint, petrol, food cans and water pipes in Europe since the 1970s. Leaded petrol was banned from 2000 with exemptions possible until 2005.

International and European health-based guidance values for lead exposure have been amended several times as new information has come to light. In 2010, the European Food Safety Authority’s (EFSA) Panel on Contaminants in the Food Chain concluded that the provisional tolerable weekly intake (PTWI) of 25 µg/kg b.w. set by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) in 1986 and endorsed by the European Commission’s Scientific Committee for Food (SCF) in 1990 was no longer appropriate and that, as there was no evidence for a threshold for a number of critical endpoints including developmental neurotoxicity and adult nephrotoxicity, it would not be appropriate to derive a PTWI. This conclusion was confirmed by JECFA in 2010, while also expressing a concern that there was potential at current levels of exposure for lead to affect neurodevelopment in infants, children and the foetus of pregnant women.

Using an alternative measure, the 2010 EFSA opinion identified a 95th percentile lower confidence limit of the benchmark dose of 1 % extra risk (BMDL01) of 0.50 µg/kg b.w. per day for developmental neurotoxicity in young children. It also lists cardiovascular effects and nephrotoxicity in adults as potential critical adverse health effects of lead with respective BMDL01 and BMDL10 of 1.50 and 0.63 µg/kg b.w. per day.

In light of the particular concern for lead exposure in children, it is important to better identify major dietary sources of lead. The current report provides updated information on the levels of lead found in a range of foods on the European market and estimates exposure using detailed individual data from the Comprehensive European Food Consumption Database covering seven age groups from infants to the very elderly.

The 144,206 lead occurrence results retained in the current study were sorted into the four different levels of the FoodEx 1 classification system. More than half of the foods tested had levels of lead at less than detection or quantification limits. The mean lead levels varied between 0.3 µg/kg for infant follow-on formulae to 4,300 µg/kg for dietetic products with an overall median across all categories of 21.4 µg/kg. Eighty-two food categories out of 734 at FoodEx level 3 with quantified discrete results had mean lead levels exceeding 100 µg/kg. The highest individual sample maximum of 232,000 µg/kg was found in game meat, followed by 155,000 µg/kg in seaweed, 117,000 µg/kg in edible offal from game animals and 59,900 µg/kg in dietary supplements.

The annual mean results of the analysis of lead in food across Europe can be very much influenced by the type of food tested and the inclusion of special investigations in a particular year. Nevertheless, an attempt was made to evaluate a potential trend in the occurrence of lead in food over the years covered by the data submitted to EFSA. Lead levels were estimated to have been reduced by about 23 % between 2003 and 2010. However, this should only be interpreted as indicative given the caveats mentioned above.

Mean lifetime dietary exposure to lead was estimated to be 0.68 µg/kg b.w. per day in the overall European population based on middle bound mean lead occurrence. Exposure was highest for
toddlers and other children with 1.32 and 1.03 µg/kg b.w. per day, respectively, while the two infant surveys ranged between 0.83 and 0.91 µg/kg b.w. per day. Adult exposure was estimated at 0.50 µg/kg b.w. per day in the current study or 31% lower than the exposure calculations presented in the EFSA opinion of 2010, mainly due to modelling differences and more accurate inputs in the present study. The elderly and very elderly population groups had similar profiles to the adult age group, while adolescents had slightly higher estimated dietary exposure.

The highest individual contributor to dietary lead exposure at FoodEx level 3 was tap water (6.1%) followed by wheat bread and rolls (3.7%), regular beer (3.0%), pastries and cakes (2.8%), iodised salt (2.4%) and potatoes consumed boiled (2.2%). Aggregating products to FoodEx level 2, major contributors included bread and rolls (8.5%) followed by different tea beverages (6.2%), tap water (6.1%), all potatoes and potato products (4.9%), fermented milk products (4.2%) and beer and beer-like beverages (4.1%). Looking at the highest level of aggregation of food at FoodEx level 1, the major contributing food category to lead exposure was grains and grain-based products (16.1%), followed by milk and dairy products (10.4%), non-alcoholic beverages (10.2%) and vegetables and vegetable products (8.4%).

The lower dietary exposure assessment results of the current study compared to the estimates presented in the EFSA opinion of 2010 can be attributed in part to the more accurate calculation methods used and the better matching of occurrence and food consumption results, but also to some extent to the decreasing lead levels in food.

It will be important to confirm the seemingly decreasing lead levels in food by future testing. A standardised data collection system now in place for reporting of European analytical test results of chemicals in food to EFSA will facilitate a future trend analysis.