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Results of the BfR-MEAL study on lead in food recovered food groups substantially contributing to total intake in the German population

Lead is released into the environment through both natural processes and human activity, which is how it enters the environment and the food chain. The highest concentrations of lead in adults are found in the liver, kidneys and bones. Lead can remain in the bones for decades, but under certain circumstances it can also be released again. The toxic effects of lead impact parts of the body including the kidneys, the cardiovascular system and the central nervous system. The general population comes into contact with lead through various sources, such as food, water, air and dust. Exposure can also occur through ceramics containing lead or through tobacco (smoke).

In this opinion, the German Federal Institute for Risk Assessment (BfR) addresses intake via food. The data on the occurrence of lead in food come from the BfR-MEAL study (**M**ahlzeiten für die **E**xpositionsschätzung und **A**nalytik von **L**ebensmitteln – "Meals for Exposure Assessment and Analysis of Food"), the first total diet study (TDS) in Germany. For this study, food was purchased, prepared in a typical household manner, and then analysed for its ingredients, including lead. These concentrations were combined with data from various consumption studies in order to better estimate the possible dietary intake of lead.

In all age groups, "grain and grain products" and "water and water-based beverages" make the highest contribution to average lead intake, at 15 % to 20 % respectively. These are followed by "composite dishes" at 8 % to 11 %. In adults, the group "coffee, cocoa, tea" also contributes to average exposure, accounting for around 16 %. Coffee is the main contributing foodstuff here. In addition to the estimated lead intake in children, blood lead concentrations were also used for the assessment. Lead is associated with a risk for developmental neurotoxic effects, particularly in younger age groups (infants aged 1 to 2 years). In this context, it should be noted that the estimated lead intake of children through food consumption is higher (up to four to five times higher) than that of adolescents and adults. As there is no safe intake level according to current knowledge, lead intake should continue to be minimised as far as possible.

1 Subject of the assessment

The subject of the present opinion is an exposure assessment based on the results of the BfR-MEAL study on lead, including a comparison with appropriate reference points from the dose-effect relationship for the health effects associated with chronic lead exposure (Benchmark Dose Lower Confidence Limit, BMDL).

2 Results

Lead is a metal that occurs ubiquitously. It can enter the environment naturally, but since ancient times release into the environment – and thus the food chain – has occurred primarily through anthropogenic activities. The present opinion deals exclusively with external exposure due to the occurrence of lead in food. Critical consequences of chronic lead exposure include health effects on the central nervous system, the cardiovascular system and the kidneys. As the available study data do not indicate a blood lead concentration at which no health effects are to be expected, lead intake should be minimised as far as possible.

Dietary lead exposure was calculated for children, adolescents and adults in Germany. The most recent representative consumption studies, the "Children's Nutrition Study to Record Food Consumption" (KiESEL study), the nutrition study as a KiGGS module (EsKiMo II) and the National Nutrition Survey II (LOD II), were used as the basis for consumption. The occurrence data for lead is based on the National Total Diet Study (BfR-MEAL study). The BfR-MEAL study is characterised by its coverage of > 90 % of consumption and its consideration of preparation-related changes in levels.

According to the exposure assessment, "grain and grain products" and "water and water-based beverages" make the highest contribution to average lead intake in all age groups, each accounting for 15 % to 20 %. The main group, "composite dishes", also accounts for a high proportion of average exposure, at 8 % to 11 %. In adults, the group "coffee, cocoa, tea" also contributes substantially to average exposure, at around 16 %. Coffee in particular is a major contributor here.

For lead, the margin of exposure (MOE) was calculated based on exposure estimates for different age groups through the consumption of food and using the EFSA reference points for renal toxicity in adults ($BMDL_{10}$ 0.63 µg/kg bw/day) or for developmental neurotoxic effects in children ($BMDL_{01}$ 0.5 µg/kg bw/day). The MOE approach serves to prioritise the urgency of risk management measures. The benchmark dose lower confidence limit (BMDL) values for external exposure were calculated by EFSA (EFSA, 2010) from BMDL values for internal exposure using kinetic models.

According to estimates of external exposure for **children,** the intake of lead through food consumption is of the same order of magnitude as the $BMDL_{01}$ of 0.5 µg/kg bw/day for developmental neurotoxic effects of lead. For example, the intake for children < 1 year is 0.27 µg/kg bw/day (P50, upper bound) or 0.35 µg/kg bw/day (P95, upper bound). The lowest MOE values are thus calculated for children < 1 year of age at 1.8 (P50, UB[1]) and 1.4 (P95, UB) and for children aged 1-2 years at 2.3 (P50, UB) and 1.4 (P95, UB). Based on

[1] Upper Bound

internal exposure data by the German Environmental Survey for Children and Adolescents GerES V (2014-2017) the internal exposure in highly exposed children and adolescents (P95) of all age groups is above the $BMDL_{01}$ of 12 µg/L for developmental neurotoxic effects. Even with blood lead levels below the $BMDL_{01}$, effects on cognitive development parameters in children exposed to lead cannot be ruled out, although EFSA considers the health risk at MOE values > 1 as likely to be low.

Exposure is far lower for **adolescents and adults** than for children (up to four to five times). This results in values between 0.08 and 0.09 µg/kg bw/day (P50, upper bound) and 0.14-0.016 µg/kg bw/day (P95, upper bound). The MOE values resulting from the comparison of exposure and the reference point for renal toxicity ($BMDL_{10}$ 0.63 µg/kg bw/day) are between 6.9-7.6 (P50, UB) and 3.8-4.4 (P95, UB) for all age groups between 14 and > 65 years. With regard to the increase in systolic blood pressure and renal toxicity, a level of exposure without effects could not be identified in epidemiological studies. In this respect, exposure cannot be considered harmless, as also pointed out by EFSA, which nevertheless assesses the risk as probably very low at MOE values > 1.

The results of the present internal and external exposure assessments show that, despite the substantial decline in internal lead exposure, there is still a need for action to further reduce exposure to the achievable minimum, especially for younger age groups.

3 Rationale

3.1 Risk assessment

3.1.1 Hazard identification

Lead is a soft and malleable metal (element symbol Pb) with a low melting point (327.5 °C) and a high density (11.34 g/cm³), which has a high level of occurrence in the Earth's crust compared to other heavy elements. Since ancient times, it has been released primarily through anthropogenic activities such as mining, smelting and the manufacture of utensils like pots, ointments and paints. More recently, it has been primarily released through the combustion of fossil fuels and the manufacture and use of products which contain lead, such as batteries. There are also natural sources of release, specifically rock erosion and volcanism. Lead in soil comes mainly from atmospheric deposition of, for example, combustion residues. On agricultural land, additional inputs occur mainly through the application of sewage sludge and other organic fertilisers (Nicholson et al., 2003). Lead is used in many different areas, such as in the manufacture of batteries and bullet cores, for metal products such as fishing weights, solder and pipes, and for equipment used to shield against X-rays. Due to the health risks, the use of lead in many products has been increasingly restricted worldwide. Leaded petrol has been gradually banned in Germany since the late 1980s, followed by a complete ban throughout the European Union in 2000 (Directive 98/70/EC). Since 2021, the fuel is no longer sold worldwide (UNEP, 2021). The ban on leaded petrol and leaded paint has eliminated the main sources of lead entering the environment (ATSDR, 2020; EFSA, 2010).

Lead does not degrade in the environment and can be found in various chemical forms in all environmental spheres (ATSDR, 2020). The predominant form of lead in the environment is inorganic lead in the oxidation state +2 (e.g. lead phosphate and lead carbonate), with lead

usually occurring in compounds with two or more other elements. In industrially synthesised organic lead compounds such as alkyl lead compounds (e.g. tetramethyl lead and tetraethyl lead), lead is almost always present in the oxidation state +4. Both substances are highly volatile, fat-soluble liquids to which humans are mainly exposed in occupational settings (EFSA, 2010).

The general population's lead exposure occurs through food and water, as well as through the air and, especially in children, through household dust and soil (EFSA, 2010). Outdoor air particles can be another important source. This applies to areas that are exposed to heavy motor vehicle traffic or are located on or near industrial sites. For individuals, ceramic and lead glass containers can be significant sources, as lead from the glazes can migrate into food (the BfR, 2020; Heinemeyer & Bösing, 2020). Lead is also a component of tobacco and tobacco smoke.

However, this opinion deals exclusively with external lead exposure due to the occurrence of lead in food. External lead exposure from other sources is not considered in this opinion.

As lead is ubiquitous in the air, soil and water, it enters the human food chain via plants, fungi and food of animal origin. The occurrence of lead in plants is mainly the result of atmospheric deposition. This means that lead is carried to the surface of fruits and leaves via dust particles and aerosols and is partially absorbed and distributed within the plant. Accordingly, lead concentrations in leaves correlate with atmospheric concentrations (EFSA, 2010). The intake of lead via the roots is rather low, as only a small proportion of the lead in the soil is available to plants. In most plant species, the vast majority of the lead absorbed through the roots remains there, but for some species, translocation of lead from the root system to the above-ground parts of the plant has been demonstrated (ATSDR, 2020). Fungi are more capable of absorbing and accumulating lead from the soil, so that lead concentrations in fungi can exceed those in green crops, fruit and vegetables (Damodaran et al., 2013; Orywal et al., 2021). Comparatively high lead levels are also frequently detected in marine algae. Various marine algae show a high capacity for passive intake of metals, which can lead to lead intake of up to 270 mg lead/g biomass (Holan & Volesky, 1994). Among edible macroalgae, brown algae have the highest metal binding capacity due to physiological characteristics (Besada et al., 2009).

Lead can also enter animal food products via lead-containing plant-based feed, but also via the intake of soil particles, especially by grazing animals (Adamse et al., 2017; Laurent et al., 2012). Plants and animals can therefore accumulate lead, but there is no enrichment in aquatic or terrestrial food chains. This can be explained in part by the fact that lead is primarily stored in the bones of vertebrates, which limits the probability of it being transferred to other species in the food chain (EFSA, 2010). Lead in drinking water is usually the result of release from lead pipes. Significant levels of lead can also enter the food chain during the production and storage of food and beverages through the use of equipment or containers which contain lead (the BfR, 2020; WHO, 2021). Game killed with lead ammunition is also a potential source of exposure for people who regularly consume this meat (the BfR, 2010; WHO, 2021). Depending on how the game meat is prepared in the kitchen, e.g. by marinating, the availability of lead from ammunition can be substantially influenced (Schulz et al., 2021).

3.1.2 Hazard characterisation

Detailed information on the hazard potential of lead can be found in opinions issued by international committees (e.g. EFSA 2010, EPA 2013, WHO/FAO 2011, ATSDR 2020). Lead has no obvious physiological function in the human body (WHO, 2021).

3.1.2.1 Toxicokinetics

The main routes of lead exposure are oral uptake and inhalation (WHO, 2021). This opinion considers lead exposure through food.

The absorption of lead from the gastrointestinal tract after oral intake is influenced by both physiological factors (e.g. age, nutritional status, pregnancy) and physicochemical properties (e.g. lead species, solubility, size). Gastrointestinal absorption of ingested lead is higher in children than in adults. In adults, the absorption rate of lead is generally 3-10 %, while in infants and young children it can be as high as 50 % (ATSDR, 2020; EFSA, 2010).

After absorption, lead is transported via the bloodstream, predominantly bound to red blood cells. The highest concentrations of lead in adults are found in the liver, kidneys and bones. With chronic lead exposure, bones contain > 90 % of the body burden in adults and > 70 % in children. Lead forms stable complexes with phosphate and can replace calcium in hydroxyapatite, a phosphate that forms the main crystalline matrix of bones. Lead is therefore deposited in the bones during growth and remodelling. This means that lead in the bones represents a pool from which small amounts are mobilised over the course of a lifetime and return to the bloodstream. This occurs particularly during phases of physiological or pathological bone demineralisation, such as during pregnancy, breastfeeding or due to osteoporosis in older age. Lead can be transferred from the mother to the foetus and via breast milk to the infant (ATSDR, 2020; EFSA, 2010).

The metabolism of inorganic lead consists of the formation of complexes with a variety of protein and non-protein ligands. Organic lead compounds are actively metabolised in the liver by P-450 enzymes (oxidative dealkylation) (ATSDR, 2020).

Regardless of the route of exposure, lead is mainly excreted through urine and faeces, and to a lesser extent through sweat, saliva, hair, nails, breast milk and seminal fluid. The elimination of lead is multiphase, reflecting the different residence times in different parts of the body. The apparent elimination half-life ($t_{1/2}$) in blood varies with age and exposure history, ranging from one week to two years. Lead is eliminated from bone with an apparent elimination half-life of one to two decades (ATSDR, 2020).

3.1.2.2 Biomarkers

The biomarkers for exposure used in practice today are measurements of total lead levels in body fluids or tissues such as blood, bone or urine.

The most commonly used biomarker is the (whole) blood lead level, which is considered the most reliable biomarker for general clinical use and public health surveillance (ATSDR, 2020). This is due to the fact that there is extensive evidence linking blood lead concentrations to clinical effects, and also validated analytical methods as well as reliable blood quality control and reference materials are available (WHO, 2021). Blood lead levels reflect a combination of short-term and long-term exposure, as lead absorbed through the intestine and lead from

soft tissue ($t_{1/2}$ 20 to 40 days), lead from the bones also passes into the blood ($t_{1/2}$) up to 30 years) (EFSA, 2010).

Lead concentrations in plasma and serum are similar, but are very low and close to the limit of quantification (LOQ) of most analytical methods (ATSDR, 2020; EFSA, 2010).

Lead in urine primarily reflects the amount of lead recently ingested. There is a correlation between lead concentrations in urine and blood, but the variations in urine are too large to allow individual blood lead concentrations to be predicted based on lead concentrations in urine (EFSA, 2010). For this reason, and because of the considerable risk of external contamination during sampling, measuring lead in urine is not recommended for routine assessment of lead exposure (EFSA, 2010; Lauwerys & Hoet, 2001).

Unlike lead in blood and urine, lead concentration in bones reflects long-term intake and total body burden, as more than 90 % of the body's lead burden is found in the skeleton. Lead concentration in bone can be determined using non-invasive methods based on X-ray fluorescence. The determination is possible for the tibia, calcaneus and patella. Lead is also incorporated into teeth during tooth formation. Accordingly, lead concentration in bones and teeth is a good measure for epidemiological studies requiring retrospective assessment of exposure (EFSA, 2010).

Hair has been used occasionally for biomonitoring of lead exposure. However, due to the possibility of external contamination, it is not a reliable indicator of lead intake into the body (EFSA, 2010; WHO, 2007).

3.1.2.3 Toxicology

There is a wealth of data on concentration-response relationships in humans for lead. This data comes from studies of occupationally exposed groups, accidents and the general population. Data are available on mutagenicity, carcinogenicity, and neurological, cardiovascular, renal, endocrine, gastrointestinal, immunological and haematological effects, as well as effects on the musculoskeletal system, reproduction and development in humans. Most of these endpoints have also been investigated in animal experiments.

The following is a summary of information on the neurological, renal and cardiovascular effects, reproductive and developmental toxicity, genotoxicity and carcinogenicity of lead.

Acute toxicity

Although the high doses required make it rare, symptoms of acute lead poisoning, especially in cases of occupational exposure or other high lead intake, include abdominal pain (colic), constipation, nausea, vomiting and anorexia. In children, high acute exposure may also lead to toxic encephalopathy.

In general, oral intake of lead as a contaminant from food is not expected to reach levels that lead to acute toxicity. Due to the long elimination half-life of lead from the body, the assessment of health risks due to chronic exposure is therefore the main focus when assessing the health risks of lead in food (EFSA, 2010).

Chronic toxicity

Potential toxicity following chronic lead exposure affects many organ systems and is sometimes observed even at blood lead levels below 50 µg/L (ATSDR, 2020).

Neurological effects

The developing nervous system has been identified as the most sensitive target organ for the toxic effects of lead in children. The potential neurotoxic effects of lead have been investigated in many studies, meaning that the evidence base is quite solid. There is consistent evidence of inverse relationships between effects on cognitive and neuromotor/neurosensory development parameters and blood lead concentrations in children and adults. In children, these correlations have been observed at blood lead concentrations of 50 µg/L and above, although there is also evidence of neurological effects below 50 µg/L. In adults, neurological impairments have been observed in connection with blood lead levels of 100 µg/L and above, although there is also evidence of effects at blood lead levels of ≤ 50 µg/L.

Overall, based on current knowledge, there is no evidence of lead exposure without adverse effects on the developing nervous system.

Renal effects

The effects of lead on the kidneys have been documented in various studies. The available data show evidence of kidney damage and impaired renal function in connection with blood lead levels in the range of 50–100 µg/L. Similar to the neurological effects, several studies indicate renal effects that have been observed even at blood lead levels below 50 µg/L. Impairments of renal function include proteinuria, impaired transport of organic anions and glucose, and reduced glomerular filtration rate (GFR). At higher blood lead levels (> 300 µg/L), lead-induced nephrotoxicity is characterized by proximal tubular nephropathy, glomerular sclerosis, interstitial fibrosis and tubular necrosis. Lead-induced impairment of renal function can lead to increased body burden due to reduced lead excretion (ATSDR, 2020).

Cardiovascular effects

Epidemiological studies in adults suggest the possibility of cardiovascular effects at blood lead levels in the range of 50–500 µg/L. Some studies suggest that the occurrence of cardiovascular effects may also occur at blood lead levels below 50 µg/L. The most frequently studied effects are those on blood pressure, with studies showing an increase in systolic and diastolic blood pressure. Other cardiovascular effects of lead include an increased risk of heart disease, arteriosclerosis, altered cardiac conduction, and increased mortality due to cardiovascular disease (ATSDR, 2020).

Effects on reproduction and development (other than neurological development)

Lead also has effects on the reproductive system and is classified as a category 1A reprotoxic substance (“May damage fertility. May damage the unborn child.” – Repr. 1A) and as "Lact." ("May cause harm to breastfed children.") in accordance with the Regulation on Classification, Labelling and Packaging (CLP Regulation) of chemicals in the EU (Regulation (EC) No 1272/2008)."). The observed effects impact both the male and female reproductive systems. For example, human observational studies indicate that lead can, for example, cause changes in serum levels of reproductive hormones or damage sperm.

Genotoxicity

Numerous *in vivo* and *in vitro* studies have been conducted to investigate the genotoxicity of lead. The data on genotoxicity indicate that lead is not directly genotoxic. Numerous studies on workers report a positive association between blood lead concentration and the occurrence of clastogenic effects such as an increased number of cells with micronuclei, an increased number of mutated T-cell receptors or a higher rate of DNA damage observed in the comet assay. Whether lead in the blood is actually the cause of the observed effects could not be conclusively proven. A weak indirect genotoxic effect is suspected due to the formation of reactive oxygen species and a reduction in DNA repair activity (EFSA, 2010).

Carcinogenicity

In rodents, high lead exposure compounds has been associated with the development of tumours in a variety of organs – particularly the kidneys, lungs, prostate and adrenal glands. It has also been found to promote the formation of kidney tumours (EFSA, 2010). This is consistent with the assessment by other committees, which classify lead and lead compounds as potentially carcinogenic to humans at high concentrations (NTP, 2021).

3.1.2.4 Concentration-response relationships and reference points for risk characterisation

Modelling of the concentration-response relationships of lead for the health risk assessment of chronic effects in humans is based on epidemiological studies (ATSDR, 2020; EFSA, 2010; EPA, 2013; FAO/WHO, 2011). Various committees have identified developmental neurotoxicity in children up to at least seven years of age, as well as cardiovascular effects and nephrotoxicity in adults, as critical effects of (inorganic) lead that should be used for risk characterisation (EFSA, 2010).

The most sensitive and relevant endpoint for children is intellectual deficits, which can be measured as a reduction in IQ (full scale IQ). As a reference point for risk characterisation, EFSA (2010) determined a $BMDL_{01}$ of 12 µg/L lead in blood (lower limit of the 95 % confidence interval of the benchmark dose (BMD_{01})[2], which is associated with a calculated decrease in IQ of 1 %, corresponding to a decrease of one point on the IQ scale). The BMDL represents the dose associated with the lower limit of the 95 % confidence interval belonging to the so-called "benchmark dose" (BMD), in this case the BMD_{01}. The data basis for this modelling was an evaluation of the relationships between blood lead levels and full-scale IQ scores in 1,333 children aged 5 to 7 or 10 years who had participated in seven international population-based longitudinal cohort studies (Lanphear et al., 2005). The relationship between dietary lead intake and blood lead levels in children up to the age of seven was estimated using the Integrated Exposure Uptake Biokinetic (IEUBK) model[3]. Using the IEUBK model, a $BMDL_{01}$value of 0.50 µg/kg body weight (BW) per day was calculated for dietary lead intake (**Table 1**) (EFSA, 2010).

The sensitivity of the foetus to the effects of lead on neurological development is unknown. EFSA assumes that the developing foetus is at least as sensitive to this lead exposure as a young child (EFSA 2010). Since the ratio between foetal and maternal blood lead concentrations in the umbilical cord is approximately 0.9, EFSA calculates a maternal blood

[2] Benchmark dose (BMD): dose determined using mathematical dose-response modelling, which is associated with a specific effect strength in the studies underlying the modelling (in the case of BMD 1, for example, an increase in the effect of 1%).
[3] Available at: https://www.epa.gov/superfund/lead-superfund-sites-software-and-users-manuals

lead level of 13 µg/L, which corresponds to the $BMDL_{01}$ for effects on neurodevelopment (12 µg/L). This corresponds to a dietary exposure of 0.54 µg/kg bw/day.

For the EFSA (2010) concentration-effect analysis with regard to cardiovascular effects in adults, blood pressure was found to be the most sensitive endpoint. A 1 % increase in systolic blood pressure (SBP) per year or on average in the general population was considered critical for public health, as this would lead to increased risks of cardiovascular mortality due to coronary heart disease (CHD) at the population level. Based on SBP in four epidemiological studies (longitudinal and cross-sectional studies), a mean $BMDL_{01}$ value of 36 µg/L lead in blood was calculated (EFSA, 2010; Glenn et al., 2003; Glenn et al., 2006; Nash et al., 2003; Vupputuri et al., 2003). The relationship between dietary lead intake and blood lead levels in adults was estimated using the model developed by Carlisle and Wade (1992) (Carlisle & Wade, 1992). Using this model, a BMDL value for dietary lead intake in adults of 1.50 µg/kg bw/day was calculated for lead-related effects on the cardiovascular system (**Table 1**) (EFSA, 2010).

For nephrotoxicity, the prevalence of chronic kidney disease (CKD) in adults was analysed based on the NHANES study (1999-2006, cross-sectional data) (EFSA, 2010; Navas-Acien et al., 2009). The $BMDL_{10}$ value of 15 µg/L lead in blood calculated by EFSA is based on a 10 % change, defined as a reduction in GFR to values below 60 mL/1.73 m² body surface area per minute. For dietary lead intake, using the above-mentioned model (Carlisle & Wade, 1992), a $BMDL_{10}$ value of 0.63 µg/kg bw per day is calculated for adults as a reference point for the risk characterisation of lead-related effects on the kidneys **(Table 1**) (EFSA, 2010).

Table1 : BMDL values as reference points for risk characterisation for the most sensitive endpoints of the toxic effects of lead after chronic exposure, expressed in µg/L blood lead content and in µg/kg bw/day for dietary lead intake (EFSA, 2010)

Endpoint	Population	Reference point	
		BMDL based on blood lead levels [µg/L]	**Dietary exposure corresponding to the benchmark dose lower confidence limit (BMDL) lead exposure [µg/kg bw per day]**
Developmental neurotoxicity (IQ[a])	Children	12[b]	0.50
	Pregnant women*	13[b]	0.54
Renal toxicity (GFR[c])	Adults	15[d]	0.63
Cardiovascular effects (SBP[e])	Adults	36[f]	1.50

[a]IQ = intelligence quotient

[b] $BMDL_{01}$ for the reduction of the full-scale IQ value by one point

[c] GFR = glomerular filtration rate

[d]$BMDL_{10}$ for a 10 % increase in the prevalence of chronic kidney disease corresponding to a decrease in GFR to a value below 60 mL/1.73 m^2 body surface area per min

[e] SBP = systolic blood pressure

[f]$BMDL_{01}$ for a 1 % increase in SBP corresponding to an increase of 1.2 mm Hg from an SBP of 120 mm Hg in a normotensive adult

* Assuming that the developing foetus is at least as sensitive to the developmental neurotoxic effects of lead as a young child and based on a ratio of foetal to maternal blood lead concentration in the umbilical cord of 0.9 (EFSA 2010).

International committees point out that for various critical endpoints, including developmental neurotoxicity, blood lead concentrations at which no health effects are to be expected could not be identified in epidemiological studies (ATSDR, 2020; EFSA, 2010; FAO/WHO, 2011). It is therefore not possible to derive a health-based guidance value (HBGV). Furthermore, opinions point out that in epidemiological studies, blood lead levels below 10 µg/L may also be associated with observations such as intellectual deficits in children.

The provisional tolerable weekly intake (PTWI) of 25 µg/kg bw derived by JECFA (FAO/WHO, 1993) is no longer considered suitable for risk characterisation of dietary lead intake by international committees, including JECFA itself (FAO/WHO, 2011).

EFSA (2010) recommends calculating the margins of exposure (MOEs[4]) to support risk characterisation. Regarding the interpretation of MOE values, EFSA (2010) concludes that for adults, with respect to the increase of SBP or the reduction of GFR, and for children, with

[4] A margin of exposure (MOE) refers to the quotient of a suitable reference point from the dose-effect relationship and the estimated human exposure to the substance. A benchmark dose lower confidence limit (BMDL) is often used as a reference point, i.e. a dose that is associated with a specific increase in the effect of a substance. An MOE value is not a health-based guidance value (HBGV). Rather, it serves to prioritise the urgency of risk management measures for substances for which, based on current knowledge, no safe intake level can be derived.

respect to effects on IQ, an MOE of 10 or greater would be sufficient to ensure that there is no appreciable risk of a clinically significant effect. Even at MOEs > 1, EFSA estimates the risk of lead-related blood pressure increase or reduction of GFR to be very low and of effects on IQ likely to be low, but not such that it could be dismissed as of no potential concern.

Until updated models of reference points for dietary lead intake are available, the BfR considers the reference points published by EFSA (2010) to be a suitable basis for an initial risk characterisation. The BfR points out that these reference points correspond to the state of scientific knowledge in 2010. As described in EFSA (2010), exposure is compared with the reference points and MOE values are calculated.

3.2 Exposure estimation and exposure assessment

3.2.1 External exposure via food

3.2.1.1 Data basis for consumption

The data basis for consumption by **infants, toddlers and children between 0.5 and < 6 years of age** was provided by the "Children's Nutrition Study to Record Food Consumption" (KiESEL study). A total of 1,104 children aged between six months and five years participated in KiESEL. The survey was conducted between 2014 and 2017. Based on an interview, the parents or legal guardians completed a questionnaire on general nutrition, nutrition in the first year of life, and a Food Propensity Questionnaire on rarely consumed food. Of these, 1,008 children and their parents also participated in the nutrition survey using a weighing/estimation protocol. The children's food consumption was documented in a weighing protocol for three consecutive days and in a 1-day weighing protocol on an independent day. In addition, out-of-home consumption (e.g. in childcare facilities) was recorded using a reduced estimation protocol (Nowak et al., 2022a; Nowak et al., 2022b) . The results from the weighing protocols were used for the evaluation, and only non-breastfed individuals were taken into account (N = 952).

The KiGGS module (EsKiMo II) (Mensink et al., 2021) served as the data basis for consumption among **children between the ages of 6 and < 12**. As part of EsKiMo II, 2,644 children and adolescents aged 6 to < 18 years were surveyed about their food consumption and eating habits between 2015 and 2017. These children and adolescents had previously participated in the second wave of the Robert Koch Institute's "Study on the Health of Children and Adolescents in Germany" (KiGGS) Wave 2. The four-day weighing protocols of 1,190 children between the ages of 6 and < 12 were used for the exposure assessment. Consumption from the age of 14 onwards is covered by NVS II.

The National Consumption Survey II (NVS II) conducted by the Max Rubner Institute (MRI) served as the data basis for consumption among **adolescents and adults between the ages of 14 and 80**. The NVS II is the current representative study on consumption among the German population. The study, in which around 20,000 people aged between 14 and 80 were surveyed about their eating habits using three different methods (dietary history, 24-hour recall and weighing protocol), took place throughout Germany between 2005 and 2006 (Krems et al., 2006; MRI, 2008). The consumption evaluations are based on data from the two independent 24-hour recalls of the NVS II, which were collected in a computer-assisted interview using "EPIC-SOFT". Data from 13,926 people for whom both interviews were available were evaluated.

These consumption studies are suitable for estimating long-term consumption levels. The intake estimates were evaluated according to the age groups listed in **Table 2**.

Table 2: Consumption studies for estimating exposure in the German population

Age group	Consumption study
Infants (0.5 < 1 year)[a]	KiESEL
Toddlers (1 < 3 years)	KiESEL
Children (3 < 6 years)	KiESEL
Children (6 < 10 years)	EsKiMo II
Adolescents (10 < 12 years)	EsKiMo II
Adolescents (14 < 19 years)	NVS II
Adults (19 < 25 years)	NVS II
Adults (25 < 35 years)	NVS II
Adults (35 < 51 years)	NVS II
Adults (51 < 65 years)	NVS II
Older adults and seniors (≥ 65 years)	NVS II

[a] Children who were (partially) breastfed were excluded from the consumption study.

3.2.1.2 Data basis for concentrations in food

Lead was examined in the core module of the BfR-MEAL study in all 356 foods on the MEAL food list[5]. Based on the 24-hour recalls from NVS II for adolescents and adults and the VELS data for children (Banasiak et al., 2005), the MEAL food list covers at least 90 % of the average food intake of different age groups in the German population for each main food group and also takes into account rarely consumed foods with known high concentrations of undesirable substances. The MEAL food was purchased between December 2016 and May 2019 in four different regions across Germany, with the product selection taking into account the different shopping habits of the German population as well as regional and seasonal characteristics. The information underlying the representative composition of the samples was collected through consumer studies and generated from market data. The food was prepared in the MEAL study kitchen, replicating typical consumer behaviour. The food and dishes were then pooled and homogenised (Sarvan et al., 2017). Specific details on data collection and the presentation and discussion of lead concentrations have already been published (Fechner et al., 2022). The highest concentrations were found in the main food group "coffee, cocoa, tea", which is mainly due to the high concentrations in cocoa powder, which was measured without preparation and is therefore undiluted. High concentrations are also found in the main groups "pulses, nuts, oilseeds and spices" and "vegetables and vegetable products". In addition to cocoa powder, the highest lead concentrations were measured in the following food: spices, porcini mushrooms, mussels, liver, herbs and algae.

[5] https://www.bfr-meal-studie.de/cm/343/Lebensmittelliste_Deutsch_2021_Web_the_BfR_final_1.pdf

3.2.1.3 Estimation of long-term exposure across all food

Methodology

For each participant in the aforementioned consumption studies, the average long-term consumption per MEAL food was determined in relation to individual body weight and linked to the respective measured lead concentration. Levels below the limit of detection or quantification (LOQ) were treated according to the *modified lower bound* (mLB) and *upper bound* (UB) approach. In the mLB approach, results below the limit of quantification (LOQ) (< LOQ) were assigned the value of the limit of detection (LOD) and results below the limit of detection (< LOD) were assigned the value zero. In the UB approach, results below the limit of quantification (< LOQ) were assigned the value of the respective limit of quantification (LOQ) and results below the limit of detection were assigned the value of the respective limit of detection (LOD).

The exposure assessment was differentiated to the characteristics of organic or conventional production. This means that if MEAL foods were sampled separately according to their production type, they were not averaged but assigned to two separate evaluations. All food that was not differentiated according to production type (seasonal, regional or unspecified[6]) was included in the scenario of consumption of primarily conventionally produced food together with conventionally produced food, and all exclusively organically produced food was included in the scenario of consumption of primarily organically produced food. A total of 105 of the 356 MEAL food samples examined were differentiated by production type. In both exposure scenarios, it was assumed that all individuals consumed either exclusively organically produced or conventionally produced products, provided that a differentiation was made in the food list. Differences in exposure are based solely on differences in occurrence data, as no differentiation was made in the consumption data.

Total dietary exposure was determined on the basis of all respondents who participated in the dietary survey. The median (P50) and the 95th percentile (P95) of the resulting exposure distribution are shown. Exposure is given in µg/kg bw per day.

Long-term lead exposure across all foods
Table 3 shows lead intake for primarily conventional product selection. Depending on age groups and percentiles, exposure in the UB approach is between 13 % and 60 % higher than when using the mLB approach. As it represents the more conservative scenario, the following comments refer only to the results using the UB approach. The figures using the mLB approach are also listed in the tables.

On average, **children** under one year of age show the highest exposure at 0.27 µg/kg BW per day. In the case of the 95th percentile, children aged 1 to < 3 years show an exposure of 0.36 µg/kg BW per day. Across all age groups considered by KiESEL, the median calculated exposure is between 0.17 and 0.27 µg/kg BW per day. Lead exposure decreases with increasing age. For **children** aged 6–11 years with median exposure, lead intake is between 0.11 and 0.14 µg/kg BW per day. Lead intake in P95 for this age group is between 0.18 and 0.23 µg/kg BW per day.

[6] Non-specific MEAL foods were not divided into further pool samples according to season, region or production type. Only one measurement result is available.

Lower lead intake relative to body weight is found for median exposed **adolescents and adults** over the age of 14, in the range of 0.08 to 0.09 µg/kg BW/day, and for the P95 of this age group, in the range of 0.14 to 0.16 µg/kg BW/day.

"Grain and grain products" and "water and water-based beverages" make the highest contribution to average lead intake in all age groups, accounting for 15 % to 20 % respectively. Among these, wheat products such as white bread/rolls, puff pastry snacks, wholemeal and brown bread, and drinking water or mineral water are the main contributors. However, cow's milk, butter and apples are also among the ten food items with the highest contribution to lead exposure in all consumption studies considered. The main group, "composite dishes", also accounts for a high proportion of average exposure, at 8 % to 11 %. In adults, the group "coffee, cocoa, tea" also contributes substantially to average exposure, at around 16 %. Coffee in particular is a major contributor here.

Depending on their age group, consumers who primarily chose organic foods consumed 3–8 % more lead than consumers who primarily chose conventional products. This was solely due to differences in occurrence data, as the same consumption data was used. It should be noted that no fundamentally higher tendency was observed across all organic food. Rather, differences were noticeable in individual foods (e.g. olives or culinary herbs), and no tendency was observed within the individual main groups (Fechner et al., 2022).

Intake estimates for the organic scenario are presented in the appendix (**Table 7**).

Table 3: Long-term lead exposure [µg/kg bw/day] for children, adolescents, and adults in the German population, with the assumption of consumption of **mainly conventionally produced food**.

			Exposure [µg/kg bw/day]			
			mLB		**UB**	
Consumption study	**Age group (years)**	**N**	**P50**	**P95**	**P50**	**P95**
KiESEL	**0.5 < 1**	57	0.24	0.31	0.27	0.35
	1 < 3	308	0.17	0.30	0.22	0.36
	3 < 6	588	0.13	0.24	0.17	0.29
EsKiMo II	**6 < 10**	789	0.11	0.18	0.14	0.23
	10 < 12	401	0.08	0.14	0.11	0.18
NVS II	**14 < 19**	937	0.05	0.11	0.08	0.15
	19 < 25	1,200	0.06	0.11	0.09	0.15
	25 < 35	1,961	0.06	0.12	0.09	0.15
	35 < 51	4,311	0.06	0.13	0.09	0.16
	51 < 65	2,860	0.06	0.13	0.09	0.16
	> 65	2,657	0.06	0.12	0.09	0.14

N: Total number of individuals

Comparison of exposure assessments from the European region

In a European comparison (**Table 4**), lead exposure in infants, toddlers and children is of the same order of magnitude as the intake levels reported in the French TDS. Children and adolescents aged 3 to 17 years consumed an average of 0.27 µg/kg bw daily (Arnich et al., 2012). In the Spanish TDS, the mean daily intake for children and adolescents aged 6 to 15 years was in the range of 0.06 µg/kg bw/day (LB) to 0.12 µg/kg bw/day (UB), which is also similar to the exposure assessment for children and adolescents based on the EsKiMO II study (Marín et al., 2017). By contrast, the results from the EFSA exposure assessment show substantially higher lead intake levels. Depending on the age group and analytical limit considered, this ranged between 0.73 and 1.54 µg/kg bw/day. The data used for this assessment does not come from a TDS but instead from various monitoring programmes of the Member States (EFSA, 2012), which, due to the selection of food, the date of collection of the occurrence data and the analytical methods used, and the sampling of predominantly unprocessed food, lead to differing results, as was to be expected (Kolbaum, 2022).

A similar picture emerges from a comparison of adolescents and adults. The values from the Spanish TDS (0.03 (LB)–0.06 (UB) µg/kg bw per day) are of the same order of magnitude as the exposures estimated here (Marín et al., 2017), while the exposure assessments from the French TDS (0.2 µg/kg bw/day (MB)) are above the values from Germany and Spain (Arnich et al., 2012). Once again, the results from the EFSA opinion are higher than those from the various TDS. Here, average exposures in the range of 0.42–0.63 µg/kg bw/day were determined.

Table 4: Comparison of lead exposure for **children, adolescents and adults** based on data from the BfR-MEAL study with exposure assessments from Europe over the past ten years and the latest EFSA opinion.

	Country	Lead intake [µg/kg bw/day]	Parameter	Age group (years)	References
Children	France	0.27 (MB)	MW	3 - 17	(Arnich et al., 2012)
	Spain (Valencia)[a]	0.06 – 0.12[a]	MW	6 - 15	(Marín et al., 2017)
	Europe/EFSA[b]	0.73 (LB) – 1.09 (UB)	MW	<1	EFSA, 2012
		1.10 (LB) – 1.54 (UB)	MW	1 - <3	
		0.87 (LB) – 1.46 (UB)	MW	3 - <10	
	Germany[c]	0.13 (mLB) – 0.27 (UB)	Median	0.5 - 5	Present opinion, based on KIESEL
		0.08 (mLB) – 0.14 (UB)	Median	6	Current opinion, based on EsKiMo II
Adolescents and adults	France	0.2 (MB)	MW	> 18	(Arnich et al., 2012)
	Spain (Valencia)[a]	0.03 (LB) – 0.06 (UB)	MW	> 15	(Marín et al., 2017)
	Europe/EFSA[b]	0.46 (LB) – 0.63 (UB)	MW	10 - <18	EFSA, 2012
		0.43 (LB) – 0.57 (UB)	MW	18 - <65	
		0.42 (LB) – 0.55 (UB)	MW	65 - <75	
	Germany[c]	0.05 (mLB) - 0.09 (UB)	Median	15 - 80	Present opinion; based on NVS II

[a] "Optimistic" and "pessimistic" scenarios[7b] Range between lowest and highest determined lead intake in different German federal states ("Laender") [(c)] Conventional scenario

3.2.1.4 Uncertainties

The concept of a TDS involves drafting a food list (TDS food list) comprising foods that represent at least 90 % of the consumption in the target population. This means a reduction in uncertainties regarding the occurrence data of a TDS compared to other data collections, such as food monitoring. From a methodological point of view, another advantage is that in a TDS, ingredient level determinations are made on the basis of prepared and ready-to-eat foods, thus taking into account any changes in levels that may occur during the preparation of meals. A TDS provides a representative data set with regard to the average consumption levels of a wide range of foods and thus provides a very good basis for determining long-term exposure (Kolbaum, 2022).

It should be noted that none of the pooled samples analysed in the BfR-MEAL study exceeded the legally specified maximum levels. However, based on the analysis of pooled samples it is not possible to estimate the proportion of individual food samples that would exceed a specified maximum value. This means that the occurrence data collected in the

[7] "Optimistic": All non-detects are assigned a value of "0" and only food with a detection rate of ≥ 20 % are included in the evaluation. "Pessimistic": All non-detects are assigned the value of the LOQ and all food are included in the evaluation.

course of a TDS are not suitable for monitoring maximum levels, and it cannot be ruled out that individual samples within the pool samples may have exceeded the maximum level.

For an exposure assessment, occurrence data must be combined with consumption data. The data for children (KiESEL, EsKiMo II) was collected as part of the KiGGS wave 2 between 2014 and 2017. The consumption data for adolescents and adults (NVS II) was collected in 2005/2006. It cannot be ruled out that consumption habits in the German population have changed since then.

Due to the consideration of > 90 % (but < 100%) of consumption within a TDS, the lead exposure determined here may underestimate the actual exposure across all food consumed. However, as food that is consumed in large quantities and food with known high lead content were included in the estimate, the possible underestimation is considered to be low.

The intake estimate used the average consumption over the protocol days. This may lead to an overestimation of exposure at the margins of the distribution, as intra-individual variability was not sufficiently reflected. However, compared to the EFSA recommendation to use at least two protocol days, KiESEL and EsKiMO II already provide improved coverage of intra-individual variability.

3.2.2 Internal exposure (human biomonitoring)

The focus of the German Environmental Survey GerES V (2014-2017) was human biomonitoring of a representative sample of children and adolescents in Germany aged 3 to 17 years (UBA, 2023). Among other things, lead concentrations in the blood of 720 children and adolescents were examined (Vogel et al., 2021). In all individuals examined, blood lead concentrations were above the limit of quantification (LOQ). Children aged 14 to 17 had slightly lower median and P95 blood lead concentrations than younger children, but not in terms of maximum concentrations. The highest internal exposure was found in children in the youngest age group examined, aged 3 to 5 years. Lower concentrations were observed in girls than in boys (data not shown). A comparison with earlier studies showed a downward trend in blood lead concentrations. The concentrations in the blood of children and adolescents were 38 % lower than in GerES IV in the period 2003–2006. The statistical indicators of the results for lead concentrations in blood from the GerES V study are shown in Table 5.

Table 5: Statistical parameter on blood lead concentrations in subpopulations of GerES V participants (UBA, 2023)

		Lead concentration in blood [µg/L]		
Age group [years]	**Number of participants**	**P50**	**P95**	**Maximum**
3 - 5	138	9.6	23.1	32.1
6 - 10	231	10.8	20.3	48.4
11 - 13	143	8.3	17.5	23.0
14 - 17	208	7.9	15.6	129

The Human Biomonitoring (HBM) Commission of the German Federal Environment Agency has derived reference values for lead in the blood of children from the GerES V study (2014-2017). These values are 15 µg/L (girls aged 3 to 17, boys aged 11 to 17) and 20 µg/L (boys aged 3 to 10). In its latest update of the reference values for adults, the Commission derived preliminary reference values of 30 µg/L for women and 40 µg/L for men from data from the Environmental Specimen Bank 2010-2015 (UBA, 2019). Reference values are determined statistically and describe the average exposure of the population or a specific population group at a given point in time. This does not imply any health risk assessment. The HBM Commission reevaluated the effects of lead in blood and suspended the HBM values for lead for the toxicological assessment of internal lead exposure in whole blood due to the lack of a threshold effect, the reassessment of carcinogenic potential and recent research findings (UBA, 2009).

3.3 Risk characterisation

Based on exposure assessments for different age groups to lead through food consumption, the respective MOE values were calculated using the reference points for renal toxicity in adults ($BMDL_{10}$ 0.63 µg/kg bw per day) and for developmental neurotoxic effects in children ($BMDL_{01}$ 0.5 µg/kg bw per day) (**Table 6**). For risk characterisation, the exposure assessment was based on the assumption that mainly conventionally produced food was consumed. The resulting MOE values based on the exposure assessment on the consumption of mainly organically produced food are presented in the appendix (**Table 8)**.

Table 6: Margins of Exposure for children, adolescents and adults in the German population, with the assumption of consumption **of mainly conventionally produced food** and based on the $BMDL_{01}$ for developmental neurotoxic effects of 0.50 µg/kg bw/day for children and adolescents and the $BMDL_{10}$ for renal toxic effects of 0.63 µg/kg bw/day for adults.

		Margin of Exposure				
			mLB		UB	
Consumption study	**Age group (years)**	**N**	**P50**	**P95**	**P50**	**P95**
KiESEL	**0.5 < 1**	57	2.1	1.6	1.8	1.4
	1 < 3	308	3.0	1.7	2.3	1.4
	3 < 6	588	3.7	2.1	2.9	1.7
EsKiMo II	**6 < 10**	789	4.6	2.7	3.5	2.2
	10 < 12	401	6.1	3.5	4.4	2.7
NVS II	**14 < 19**	937	11.5	5.6	7.6	4.3
	19 < 25	1,200	11.3	5.9	7.2	4.3
	25 < 35	1,961	10.6	5.4	6.9	4.1
	35 < 51	4,311	10.2	5.0	6.9	3.9
	51-<65	2,860	10.1	4.8	6.9	3.8
	> 65	2,657	10.4	5.5	7.3	4.4

N: Total number of individuals

Children

According to estimates of external exposure for children, lead intake through food consumption is of the same order of magnitude as the $BMDL_{01}$ of 0.5 µg/kg bw/day. The lowest MOE values are calculated for children < 1 year of age with 1.8 (P50, UB) and 1.4 (P95, UB) and for children aged 1–2 years with 2.3 (P50, UB) and 1.4 (P95, UB), meaning that for the youngest age group, the MOE is a maximum of 2. A small proportion of the youngest age groups exceeds the benchmark dose lower confidence limit $BMDL_{01}$ (1 to 2 % of children < 1 to 2 years of age, data not shown).

For the toxicological assessment of internal lead exposure in children, the blood lead levels of the relevant age groups can be compared with the $BMDL_{01}$ of 12 µg lead/L blood as a reference point for the developmental neurotoxic effects of lead in children (Chapter3.2.2). EFSA (2010) uses the corresponding $BMDL_{01}$ for the assessment of external exposure only for children up to 7 years of age. According to the results from GerES V, the high internal exposure (P95) of children up to 7 years of age exceeds the blood level of 12 µg lead/L (**Table 5).** The high internal exposure of older children is also above the blood level of 12 µg/L. The reference values for lead in the blood of children derived by the HBM Commission on the basis of GerES V data of 15 µg/L (girls aged 3 to 17, boys aged 11 to 17) and 20 µg/L (boys aged 3 to 10) therefore also exceed the benchmark dose lower confidence limit $BMDL_{01}$ of 12 µg lead/L blood. In children with blood lead levels in the range of the median,

internal exposure is close to the $BMDL_{01}$, especially in the younger age groups (3 to 10 years), but does not exceed it.

Overall, the data show that external lead exposure in children, especially in the younger age groups, through food consumption is in the order of magnitude of the $BMDL_{01}$ for developmental neurotoxic effects of lead, and that internal exposure in highly exposed children (P95) of all age groups exceeds this value. The latter reflects not only dietary lead exposure, but exposure from all sources. Even with blood lead levels below the $BMDL_{01}$, effects on cognitive development parameters in children exposed to lead cannot be ruled out, although EFSA considers the health risk at MOE values > 1 likely to be low.

Adolescents and adults

Lead intake by adolescents and adults through food consumption is below the $BMDL_{10}$ of 0.63 µg/kg bw/day and hardly differs between the age groups considered (14 - > 65 years). The MOE values resulting from the comparison of exposure and the reference point for renal toxicity ($BMDL_{10}$ 0.63 µg/kg bw/day) are between 6.9 and 7.6 (P50, UB) and 3.8 and 4.4 (P95, UB) for all age groups between 14 and > 65 years.

Based on the assumption that the developing foetus is at least as sensitive to the effects of lead as a small child, EFSA (2010) used a lower $BMDL_{01}$ of 0.54 µg/kg bw/day for developmental neurotoxicity of lead in pregnant women compared to the endpoint of renal toxicity. A comparison of this $BMDL_{01}$ with the dietary lead intake of pregnant women cannot be made in the context of this opinion, as no consumption data for pregnant women are available. When considering the exposure of adults aged 18 to 35 years as representative of pregnant women in relation to the $BMDL_{01}$ of 0.54 µg/kg bw/day (**Table 3),** MOE values would be in a similar range to those for (non-pregnant) adolescents and adults (data not shown).

Recommendation for action

The results of the present internal and external exposure assessments show that, despite the substantial decline in internal lead exposure, there is still a need to minimise lead intake, especially for younger age groups.

Further information on the BfR website

Lead occurrence data from the MEAL study
https://www.bfr-meal-studie.de/en/a-z_microsite_index/lead-309596.html#fragment-3

Questions and answers on the consumption of game shot with lead ammunition (in German)
https://www.bfr.bund.de/fragen-und-antworten/thema/fragen-und-antworten-zum-verzehr-von-wild-das-mit-bleihaltiger-munition-geschossen-wurde/

4 References

Adamse P, Van der Fels-Klerx HJI, de Jong J (2017). Cadmium, lead, mercury and arsenic in animal feed and feed materials – trend analysis of monitoring results. Food Addit Contam Part A Chem Anal Control Expo Risk Assess, 34 (8): 1298-1311. https://doi.org/10.1080/19440049.2017.1300686

Arnich N, Sirot V, Riviere G, Jean J, Noel L, Guerin T, Leblan JC (2012). Dietary exposure to trace elements and health risk assessment in the 2nd French Total Diet Study. Food and Chemical Toxicology, 50 (7): 2432-2449. https://doi.org/10.1016/j.fct.2012.04.016

ATSDR (2020). Toxicological profile for lead. Agency for Toxic Substances and Disease Registry. Available online at: https://www.atsdr.cdc.gov/toxprofiles/tp13.pdf. Accessed on 14 June 2023.

Banasiak U, Heseker H, Sieke C, Sommerfeld C, C V (2005). Estimation of the intake of plant protection product residues in food using new consumption levels for children. Federal Health Gazette – Health Research – Health Protection, 1: 98. https://doi.org/10.1007/s00103-004-0949-6

Besada V, Andrade JM, Schultze F, González JJ (2009). Heavy metals in edible seaweeds commercialised for human consumption. Journal of Marine Systems, 75 (1-2): 305-313.

BfR (2010). Lead contamination of game meat due to the use of lead ammunition in hunting. (Opinion No. 040/2011). German Federal Institute for Risk Assessment (BfR). Retrieved online, available at: https://www.bfr.bund.de/cm/343/bleibelastung-von-game-meat-durch-verwendung-von-bleimunition-bei-der-jagd.pdf. Accessed on 14 June 2023.

BfR (2020). Ceramic tableware: BfR recommends lower release levels for lead and cadmium. (Opinion No. 043/2020). German Federal Institute for Risk Assessment (BfR). https://doi.org/10.17590/20200921-112429

Carlisle JC & Wade MJ (1992). Predicting blood lead concentrations from environmental concentrations. Regul Toxicol Pharmacol, 16 (3): 280-289. https://doi.org/10.1016/0273-2300(92)90008-w

Damodaran D, Balakrishnan RM, Shetty VK (2013). The uptake mechanism of Cd(II), Cr(VI), Cu(II), Pb(II), and Zn(II) by mycelia and fruiting bodies of *Galerina vittiformis*. Biomed Res Int, 2013: 149120. https://doi.org/10.1155/2013/149120

EFSA (2010). Scientific Opinion on Lead in Food. EFSA Journal, 8 (4): 1570. https://doi.org/10.2903/j.efsa.2010.1570

EFSA (2012). Lead dietary exposure in the European population European Food Safety Authority. EFSA Journal, 10 (7). https://doi.org/ARTN283110.2903/j.efsa.2012.2831

EPA (2013). Integrated Science Assessment (ISA) for Lead (Pb). US EPA Research Triangle Park, NC. Retrieved online, available at: https://ordspub.epa.gov/ords/eims/eimscomm.getfile?p_download_id=518908. Accessed on 14 June 2023.

FAO/WHO (1993). Evaluation of certain food additives and contaminants. Series: WHO Technical Report Series, Vol. 837. Retrieved online, available at:

https://apps.who.int/iris/bitstream/handle/10665/36981/WHO_TRS_837.pdf?sequence=1&isAllowed=y. Accessed on 14 June 2023.

FAO/WHO (2011). Safety evaluation of certain food additives and contaminants – Lead. Series: WHO Food Additives Series. Report: 64. Retrieved online, available at: https://apps.who.int/iris/bitstream/handle/10665/44521/9789241660648_eng.pdf?sequence=1&isAllowed=y. Accessed on 14 June 2023.

Fechner C, Hackethal C, Hopfner T, Dietrich J, Bloch D, Lindtner O, Sarvan I (2022). Results of the BfR MEAL Study: In Germany, mercury is mostly contained in fish and seafood, while cadmium, lead and nickel are present in a broad spectrum of foods. Food Chem X, 14: 100326. https://doi.org/10.1016/j.fochx.2022.100326

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

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

Heinemeyer G & Bösing U (2020). Entry routes of lead into the human organism. Health U, Ed. Retrieved from https://www.umweltbundesamt.de/sites/default/files/medien/479/publikationen/eintragspfade_blei_menschlicher_organismus_fkz_3717_62_212_0_abschlussbericht_bf_20191121_final.pdf

Holan ZR & Volesky B (1994). Biosorption of lead and nickel by biomass of marine algae. Biotechnology and bioengineering, 43 (11): 1001-1009.

IARC (2006). IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 87. Inorganic and Organic Lead Compounds. Retrieved online, available at: https://publications.iarc.fr/_publications/media/download/2742/4c6e963acd7ad3b767d0720848796ef7099dd02a.pdf. Accessed on 14 June 2023.

Kolbaum AE, Jäger, A., Ptok, S., Sarvan, I., Greiner, M., Lindtner, O. (2022). Collection of occurrence data in foods – The value of the BfR MEAL study in addition to the national monitoring for dietary exposure assessment. Food Chemistry: X, 13: 100240. https://doi.org/https://doi.org/10.1016/j.fochx.2022.100240

Krems C, Bauch A, Götz A, Heuer T, Hild A, Möseneder J, Brombach C (2006). Methods of the National Nutrition Survey (LOD) II. Ernährungs Umschau, 53 (2): 44-50.

Lanphear BP, Hornung R, Khoury J, Yolton K, Baghurst P, Bellinger DC, Canfield RL, Dietrich KN, Bornschein R, Greene T (2005). Low-level environmental lead exposure and children's intellectual function: an international pooled analysis. Environmental health perspectives, 113 (7): 894-899.

Laurent C, Feidt C, Laurent F (2012). Soil contamination – Transfer from soil to animals: Transfer from soil to animals. EDP Sciences.

Lauwerys RR & Hoet P (2001). Industrial chemical exposure: guidelines for biological monitoring. CRC Press.

Marín S, Pardo O, Báguena R, Font G, Yusà V (2017). Dietary exposure to trace elements and health risk assessment in the region of Valencia, Spain: a total diet study. Food Additives and

Contaminants, Part A: Chemistry, Analysis, Control, Exposure and Risk Assessment, 34 (2): 228–240. https://doi.org/10.1080/19440049.2016.1268273

Mensink GBM, Haftenberger M, Lage Barbosa C, Brettschneider AK, Lehmann F, Frank M, Heide K, Moosburger R, Patelakis E, Perlitz H (2021). Research report: EsKiMo II – The nutrition study as a KiGGS module (p. 164): Robert Koch Institute. https://doi.org/http://dx.doi.org/10.25646/7028.2

MRI (2008). National Nutrition Survey (LOD) II. Results report part 1. The nationwide survey on the nutrition of adolescents and adults. Max Rubner Institute (Ed.), 144 pages. Karlsruhe.

Nash D, Magder L, Lustberg M, Sherwin RW, Rubin RJ, Kaufmann RB, Silbergeld EK (2003). Blood lead, blood pressure, and hypertension in perimenopausal and postmenopausal women. Jama, 289 (12): 1523-1532.

Navas-Acien A, Tellez-Plaza M, Guallar E, Muntner P, Silbergeld E, Jaar B, Weaver V (2009). Blood cadmium and lead and chronic kidney disease in US adults: a joint analysis. American journal of epidemiology, 170 (9): 1156-1164.

Nicholson FA, Smith SR, Alloway BJ, Carlton-Smith C, Chambers BJ (2003). An inventory of heavy metals inputs to agricultural soils in England and Wales. Sci Total Environ, 311 (1-3): 205-219. https://doi.org/10.1016/s0048-9697(03)00139-6

Nowak N, Diouf F, Golsong N, Höpfner T, Lindtner O (2022a). KiESEL – The Children's Nutrition Survey to Record Food Consumption for the youngest in Germany. BMC Nutrition, 8 (1): 64. https://doi.org/10.1186/s40795-022-00527-6

Nowak N, Höpfner T, Rüdiger T, Lindtner O (2022b). Children's Nutrition Study to Record Food Consumption (KiESEL). Research report part 1: Results of the questionnaire. Series: BfR Science (ISBN 978-3-948484-56-9; ISSN 1614-3795 (print) 1614-3841 (online)), 175 pp. Berlin, DE: German Federal Institute for Risk Assessment (BfR). https://doi.org/10.17590/20230120-105243

NTP (2021). Report on Carcinogens, 15th Edition. Lead and Lead Compounds. Retrieved from https://ntp.niehs.nih.gov/sites/default/files/ntp/roc/content/profiles/lead.pdf

Orywal K, Socha K, Nowakowski P, Zoń W, Kaczyński P, Mroczko B, Łozowicka B, Perkowski M (2021). Health risk assessment of exposure to toxic elements resulting from consumption of dried wild-grown mushrooms available for sale. PLoS One, 16 (6): e0252834. https://doi.org/10.1371/journal.pone.0252834

Sarvan I, Bürgelt M, Lindtner O, Greiner M (2017). Exposure assessment of substances in foods: The BfR MEAL study – The first German total diet study. Bundesgesundheitsblatt – health research – Gesundheitsschutz, 60 (7: Contaminants in food): 689–696 [8 pp]. https://doi.org/10.1007/s00103-017-2566-1

Schulz K, Brenneis F, Winterhalter R, Spolders M, Fromme H, Dietrich S, Wolf P, Gremse C, Schafft H, Pieper R (2021). Marinating increases the bioavailability of lead in game meat shot with lead ammunition. Journal of Nutritional Science, 10: e24.

UBA (2009). [2nd addendum to "lead monograph--reference--and "human biomonitoring" values of the "Human Biomonitoring" Council of the Environmental Office]. Federal Health

Gazette Health Research Health Protection, 52 (10): 983-986. https://doi.org/10.1007/s00103-009-0936-z

UBA (2019). Update of reference values for lead in the blood of adults. Federal Health Gazette – Health Research – Health Protection, 10: 1280.

UBA (2023). Results report German Environmental Study on the Health of Children and Adolescents 2014–2017 (GerES V) Part 1: Human biomonitoring. Retrieved online, available at: https://www.umweltbundesamt.de/sites/default/files/medien/479/publikationen/uug_02-2023_deutsche_umweltstudie_zur_gesundheit_von_kindern_und_jugendlichen_2014-2017.pdf. Accessed on 14 June 2023.

UNEP (2021). Era of leaded petrol over, eliminating a major threat to human and planetary health. Website. Retrieved online, available at: https://www.unep.org/news-and-stories/press-release/era-leaded-petrol-over-eliminating-major-threat-human-and-planetary, accessed on 14 June 2023.

Vogel N, Murawski A, Schmied-Tobies M, Rucic E, Doyle U, Kämpfe A, Hoera C, Hildebrand J, Schaefer M, Drexler H (2021). Lead, cadmium, mercury, and chromium in urine and blood of children and adolescents in Germany–human biomonitoring results of the German Environmental Survey 2014–2017 (GerES V). International Journal of Hygiene and Environmental Health, 237: 113822.

Vupputuri S, He J, Muntner P, Bazzano LA, Whelton PK, Batuman V (2003). Blood lead level is associated with elevated blood pressure in blacks. Hypertension, 41 (3): 463-468.

WHO (2007). Health risks of heavy metals from long-range transboundary air pollution. (9289071796). World Health Organisation. Regional Office for Europe. Retrieved online, available at: https://www.who.int/publications/i/item/9789289071796. Accessed on 14 June 2023.

WHO (2021). WHO guideline for clinical management of lead exposure: executive summary. Geneva: World Health Organisation. Retrieved from https://www.who.int/publications/i/item/9789240037045

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