National Institute for Public Health
and the Environment
Ministry of Health, Welfare and Sport

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RIVM Letter report 2016-0206
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Synopsis

Dietary exposure to lead in the Netherlands

Uptake from the soil is the main route by which lead ends up in food. Lead in soil has its origin in both natural and anthropogenic sources. The lead concentration in food has decreased over the last decennia by the use of unleaded petrol and paint, and the replacement of lead water pipes.

RIVM has assessed the intake of lead via food in the Netherlands. The calculated intakes showed that detrimental health effects cannot be excluded in a part of children up to age seven, pregnant women and adults. The number of persons actually at risk cannot be quantified. The food groups cereals, milk, fruit, non-alcoholic beverages (including tea and fruit juices) and vegetables contributed most to the total lead intake (about 70 percent).

The intake of too much lead may have a negative effect on brain development (quantified as the loss of one IQ point) in children up to age seven, as well as in the developing foetus via lead ingestion of the mother. In adults, the negative effects of a high lead intake are on the kidney. Too much lead can also result in negative effects on blood pressure, but that risk is very low at all calculated intakes via food.

The intake calculations were performed with the most recent information on lead concentrations in food combined with food consumption data from Dutch food consumption surveys, and calculated with a calculation model with which currently the best intake estimations can be obtained. Data on lead concentrations in some food products were limited. Therefore, concentration data from other European countries were also used. Additionally, lead concentrations in certain food products, including milk (products) and bread, were so low that they were difficult to quantify.

The European Food Safety Authority (EFSA) has evaluated at which intake level of lead no detrimental health effects occur. This evaluation was used to determine if the lead intake results in possible health risks.

Keywords: lead, young children, children, adults, concentration data, long-term intake, statistical modelling

Publiekssamenvatting

De inname van lood in Nederland via voedsel

Lood komt in voedsel terecht doordat planten en gewassen het uit de bodem opnemen. Lood kan van nature in de bodem zitten, maar kan er ook in komen door menselijk handelen. De concentratie van lood in voedsel is de laatste decennia afgenomen door het gebruik van loodvrije benzine en verf, en de vervanging van loden drinkwaterleidingen.

Het RIVM heeft berekend hoeveel lood we in Nederland binnen kunnen krijgen via voedsel. Op basis van de berekende innamen blijkt dat bij een deel van de kinderen tot en met 7 jaar, zwangere vrouwen en volwassenen schadelijke effecten niet kunnen worden uitgesloten. Bij hoeveel mensen er sprake is van een daadwerkelijk risico, is niet aan te geven. De voedselgroepen granen, melk, fruit, non-alcoholische dranken (waaronder thee en vruchtendranken) en groenten dragen het meeste bij aan de totale loodinname (circa 70 procent).

Als kinderen tot en met 7 jaar te veel lood binnenkrijgen kan dat effect hebben op hun hersenontwikkeling (gekwantificeerd als het verlies van 1 IQ-punt). Dit geldt ook voor de zich ontwikkelende foetus via de loodinname van de moeder. Bij volwassenen kan een te hoge inname van lood effecten hebben op de nieren. Te veel lood kan ook schadelijk zijn voor de bloeddruk, maar dat risico is bij alle berekende innamen via voedsel zeer laag.

De innameberekeningen zijn gebaseerd op de meest recent beschikbare informatie over loodconcentraties in voedsel gecombineerd met voedselconsumptiegegevens van de Nederlandse voedselconsumptiepeiling en berekend met een rekenmodel waarmee de beste innameschattingen op dit moment kunnen worden verkregen. Gegevens over loodconcentraties in sommige voedselproducten bleken beperkt beschikbaar. Daarom zijn ook concentratiegegevens uit andere Europese landen gebruikt. Verder waren loodgehalten in bepaalde voedingsmiddelen, namelijk melk(producten) en brood, dermate laag dat ze moeilijk waren te meten.

De European Food Safety Authority (Europese Autoriteit voor Voedselveiligheid, EFSA) heeft geëvalueerd bij welke loodinname er in elk geval geen schadelijke effecten optreden. Deze evaluatie is in dit rapport gebruikt om te bepalen of er sprake is van een mogelijk gezondheidsrisico door loodinname.

Kernwoorden: lood, jonge kinderen, kinderen, volwassenen, concentratiedata, langetermijninname, statistisch modelleren

Contents

1 Introduction

Lead is a heavy metal occurring as an environmental contaminant with its origin in both natural (soil) and anthropogenic sources, such as the (past) presence of lead in water pipes, paint and petrol. Lead exists both in organic and inorganic forms. Inorganic lead predominates in the environment, including food. Food is the major source of lead exposure in the non-smoking population. In children, the intake via dust and soil can also be a factor, especially for those living in contaminated areas (EFSA, 2010; Otte et al., 2015). Exposure to organic lead is generally limited to the working environment (EFSA, 2010). In this report, the term "lead" refers to inorganic lead.

In 2010, the Scientific Panel on Contaminants in the Food Chain (CONTAM) of the European Food Safety Authority (EFSA) re-evaluated the then applicable provisional tolerable weekly intake (PTWI) for lead of 25 µg/kg body weight (bw) per week (equivalent to 3.6 µg/kg bw per day) (FAO/WHO, 1987). The CONTAM panel concluded that the derivation of a PTWI was no longer correct, due to lack of evidence for a threshold for a number of critical endpoints of lead. The calculation of margins of exposure (MOEs) to support the risk characterisation was decided to be more appropriate (EFSA, 2010). For this, the panel determined different 95th percentile (P95) lower confidence limits of the benchmark dose (BMDL) for dietary lead intake based on three adverse effects:

- $BMDL_{01}$ (1% extra risk) for developmental neurotoxicity (loss of one Intelligence Quotient (IQ) point)) of 0.5 µg/kg bw per day in children up to age seven. As this effect is also relevant for the developing foetus, a corresponding $BMDL_{01}$ of 0.54 µg/kg bw per day was derived based on a foetal/maternal cord blood lead concentration ratio of 0.9. For the calculation of the corresponding MOE, lead intake of pregnant women should be used;
- $BMDL_{01}$ for cardiovascular effects (systolic blood pressure) of 1.50 µg/kg bw per day in adults;
- $BMDL_{10}$ (10% extra risk) for nephrotoxicity (chronic kidney disease) of 0.63 µg/kg bw per day in adults.

CONTAM Panel considered a MOE of 10 or greater of negligible public health concern (EFSA, 2010). The BMDLs were based on human dose-response data (EFSA, 2010). A MOE of 10 can therefore be interpreted as being equal to the intraspecies assessment factor. At lower MOEs, but greater than one, the risk was considered to be very low for cardiovascular effects and nephrotoxicity, whereas for neurodevelopmental effects the risk was assumed to be low, 'but not such that it could be dismissed as of no potential concern' (EFSA, 2010).

In 2012, the same Panel estimated the exposure to lead via food in several European countries, including the Netherlands (EFSA, 2012b). In the same year, also a Dutch study into the intake of lead in children aged 2 to 6 was published (Boon et al., 2012). Both studies showed that the (mean, median and high) exposure in Dutch children aged 2 to 6

resulted in MOEs below one for developmental neurotoxicity. In the EFSA study, the mean exposure in Dutch adults resulted in MOEs greater than one for nephrotoxicity and cardiovascular effects, whereas the high (P95) exposure resulted in MOEs below one for nephrotoxicity and above one for cardiovascular effects. None of the calculated MOEs was at least 10.

Given these high calculated exposures to lead and because country-specific exposure estimates to food contaminants, such as lead, reported by EFSA may not represent the true exposure within a country (Sprong & Boon, 2015; Boon et al., 2014), a national exposure assessment of lead was performed in persons aged 7 to 69. For this population group, recent food consumption data of a survey conducted in 2007 to 2010 was available (van Rossum et al., 2011). The consumption data were combined with monitoring and surveillance concentration data of lead from 2010-2015. To include as many ages as possible, also the exposure assessment performed in 2012 among children aged 2 to 6 was updated.

In this report, the terms exposure and intake are used alternatively, referring both to the ingestion of lead via food. Furthermore, only the exposure to lead via food was addressed in the current assessment.

2 Intake calculations

2.1 Food consumption data

Exposure calculations for children aged 2 to 6 were performed using food consumption data from the Dutch National Food Consumption Survey (DNFCS)-Young children (Ocké et al., 2008). The survey covered the dietary habits of young children aged 2 to 6 and was conducted in 2005 and 2006. Calculations for the population aged 7 to 69 were performed using food consumption data from the DNFCS 2007-2010 (van Rossum et al., 2011). For a more detailed description of both surveys, see Appendix A.

2.2 Concentration data

Lead concentration data used in the exposure calculations were obtained from Dutch monitoring programmes performed by the Netherlands Food and Consumer Product Safety Authority (NVWA), the Institute for Marine Sources & Ecosystem Studies, Fytolab and the Dutch Dairy Association. These data covered the period 2010-2015 and were stored in the Quality of Agricultural Products (KAP) database. Also monitoring data available in the BioKAP database were included in the analyses. This database contains concentrations of different food chemicals analysed in organically grown products. BioKAP is an initiative of the Dutch trading and processing association (VBP). Many concentrations in the BioKAP database were reported for concentrates. These concentrations were converted to concentrations in the product as such using conversion factors provided by the data supplier. Lead concentrations obtained from the BioKAP database were analysed in fruit, vegetables, cereals and seeds.

Lead analyses were predominantly performed in raw agricultural commodities (RACs), including vegetables, fruit, cereals, milk, fish, liver and kidney (Appendix B). Only few concentration data were available for meat of game, including deer and rabbit. For the other animals whose meat is consumed in the Dutch diet, including bovine animal, pig, poultry, sheep and goat, lead concentrations in meat were estimated based those analysed in liver (poultry and pig) and kidney (bovine animal, sheep and goat). For this, the proportion of lead in meat, liver and kidney was estimated based on animal-specific mean lead concentrations reported in these products by EFSA (2012b). Based on these data, the proportion of lead in meat:liver for poultry was estimated at 1:1.5. For the other animals, the proportion of lead in meat:liver:kidney was estimated at 1:4:8. In addition, the consumption of liver is reported in both DNFCSs, including poultry, pork and calf's liver. Lead is only analysed in poultry and calf's liver within the Dutch monitoring programmes. To obtain lead concentrations in pork liver, the proportion of lead in meat:liver:kidney of 1:4:8 was also used to derive lead concentrations in pork liver based on those analysed in kidney of pork.

Concentrations of lead in drinking water were obtained from the Centre for Sustainability, Environment and Health[1] (part of RIVM) covering the period of 2012-2015. These years resulted in a large enough sample to estimate the lead concentration in drinking water.

In 2013, a mycotoxin-dedicated total diet study (mTDS) was conducted in the Netherlands (Sprong et al., 2016). In this survey, individual food products were collected from Dutch supermarkets and specialist shops, prepared as consumed based on information from both DNFCSs, and subsequently pooled to a sample representing a certain food product. This study included samples representing bread, cereal products (including rice and pasta), breakfast cereals and biscuits. These samples were analysed for lead by RIKILT Wageningen UR and used in the current study (Appendix B).

In case no Dutch concentration data were available for foods or food ingredients which may contain lead based on the 2012 lead exposure report of EFSA (2012b), average concentrations per food or food ingredient were obtained from that study. In this way, possible underestimation of the exposure due to neglecting potential sources of exposure was avoided. Concentrations from foods or food ingredients available in Europe were thus assumed representative for those available on the Dutch market.

For an overview of the concentration data used in the exposure assessment and the source of the data, see Appendix C. Because the lead concentrations in the BioKAP database were comparable to those analysed in similar conventionally grown products (data not shown) and there was no reason to assume that lead concentrations would differ between conventionally and organically grown products, the concentrations obtained from the KAP and BioKAP database are reported together as 'Dutch monitoring data' in Appendix C, and referred to as such in this report.

2.3 Food mapping

Mapping is the process of matching the analysed products to the foods recorded in food consumption databases. For the current exposure assessment two types of food mapping could be distinguished:

- Indirect mapping via RAC
- Direct mapping between an analysed product (in some cases after preparation as consumed) and a food recorded in the food consumption database.

Both types are described in detail below.

2.3.1 Indirect food mapping via RAC

Indirect mapping via RAC was needed to include the Dutch monitoring data in the exposure assessment. Also, levels of lead in drinking water were included in this manner. For several RACs, the number of analytical values available from monitoring were limited (i.e. less than 10 samples) or absent. Most analysed RACs were therefore grouped according the FoodEx1 classification system (EFSA, 2011) before

[1] Part of the National Institute for Public Health and the Environment (RIVM)

mapping. FoodEx1 is the classification system used by EFSA to assess the exposure to food contaminants (e.g. EFSA, 2015; 2016)). It is a hierarchical classification system consisting of four food group levels, each higher level containing more details about the food. Most foods are classified according to three food group levels. For example, the food 'carrot' is classified as level 1 'Vegetables and vegetable products (including fungi)', level 2 'Root vegetables' and level 3 'Carrots'. In this report, the available concentration data per relevant RAC were grouped in an appropriate food group. The concentration data per food group were subsequently assigned to all RACs belonging to that food group. For example, limited lead concentration data were available for 'globe artichokes', 'asparagus', 'rhubarb' and 'fennel'. These foods belong to FoodEx level 2 food group 'stem vegetables'. Concentrations of these RACs were grouped and assigned to all RACs belonging to the FoodEx level 2 food group, including, for example, 'beetroot', 'celeriac', and 'turnips'. See Appendix C for an overview of the food groups that were defined, listed under 'Dutch monitoring data' and 'Grouped foods'. FoodEx1 was chosen to align the assessment as much as possible to the 2012 lead exposure assessment of EFSA (2012b).

A number of analysed RACs, as well as drinking water, were not grouped, because the number of analysed samples was sufficient. Examples of such RACs were meat, liver and milk. Honey and seaweed were also not grouped despite a limited number of analysed samples. For these RACs, grouping was no option due to lack of comparable foods.

To assess the dietary exposure, the concentrations in RACs and drinking water were converted to concentrations in foods as recorded in the DNFCSs. For this, it is important to realise that foods recorded in food consumption databases include foods consisting of one ingredient (e.g. fruits, vegetables, full-fat milk) and composite foods consisting of more than one ingredient (e.g. pizza and salads). Lead concentrations in RACs and drinking water were converted to concentrations in foods as recorded in both food consumption databases as described below.

Consumed foods consisting of one RAC ingredient
Concentrations in RACs and drinking water, either individually or as belonging to a food group, were assigned directly to single RAC ingredient foods as recorded in the food consumption databases. For example, the concentrations in the FoodEx1 level 2 food group 'pome fruit' were mapped directly to the consumption of apple and pear as such. This type of mapping is similar to direct food mapping (section 2.3.2).

Composite foods
To include exposure via the consumption of composite foods in the assessment, a food conversion model was used. In this model, chemical concentrations per RAC are converted to equivalent concentrations in composite foods (Boon et al., 2009; Geraets et al., 2011; van Dooren et al., 1995). This model first converts composite foods to their corresponding RAC ingredients (including their weight fractions) based on recipe data and conversion factors of processed ingredients to their raw counterparts. For example, pizza is first divided into equivalent

amounts of its ingredients like flour, cheese and tomato. These ingredients are subsequently converted to their raw counterparts (wheat, milk and tomato, respectively) using conversion factors. Then, the chemical concentrations analysed in these RAC ingredients are attributed to these fractions and summed to result in the chemical concentration in pizza. This approach was used to assign lead concentrations to composite foods in the current assessment, which were not covered via direct food mapping (see section 2.3.2). Lead concentrations analysed in drinking water were mapped to foods containing drinking water as an ingredient, such as lemonade.

2.3.2 Direct food mapping

Direct mapping was used for the food samples of the mTDS (Appendix B), as well as for the concentrations obtained from EFSA (2012b). Via direct mapping, the analysed products are mapped as much as possible to identical foods or to appropriately similar foods recorded in the DNFCSs.

The foods 'cheese', 'dried milk', 'condensed milk', and fruit and vegetable juices recorded in the DNFCs were also directly mapped to respective concentrations reported in comparable foods in EFSA (2012b), despite the availability of monitoring data in the relevant RACs: milk, fruit and vegetables, respectively. Assigning lead concentrations to these foods via concentrations analysed in the relevant RAC and the conversion model (section 2.3.1) would have resulted in lower (cheese, and dried and condensed milk) or higher (fruit and vegetable juices) concentrations of lead compared to those analysed directly in these foods (EFSA, 2012b). To avoid a possible under- or overestimation of the exposure, the lead concentration reported by EFSA (2012b) were therefore used. These discrepancies are inherent to using models to estimate concentrations in composite foods based on concentrations analysed in RACs, recipes of composite foods and conversion factors (see section 4.2.3).

2.4 Long-term dietary exposure assessment

The long-term (or usual) dietary exposure to lead was assessed, because for consumers repeated exposure to this compound is most relevant (EFSA, 2010). For this, the Monte Carlo Risk Assessment (MCRA) software, release 8.2 was used (de Boer et al., 2016). This software contains the LogisticNormal-Normal (LNN) model, which was used to assess the long-term exposure.

In this model, daily consumption patterns of individuals were multiplied with the mean lead concentration per consumed food, and summed over foods per day per individual. All daily estimated exposures were adjusted for individual body weight, resulting in a distribution of daily exposures per individual. This distribution was subsequently corrected for the day-to-day variation in exposure within individuals to estimate the long-term exposure distribution. See Appendix D for a description of LNN.

Exposures were expressed in "µg/kg bw per day", and weighted for small deviances in socio-demographic factors and season. The exposure distribution of persons aged 7 to 69 was also corrected for day of the

week. Weights were those used by Ocké et al. (2008) and van Rossum et al. (2011). No weights for day of the week were available within the DNFCS-Young children database. The exposure was calculated for three age groups: children aged 2 to 6, persons aged 7 to 69 and women of childbearing age aged 20 to 40. For this last population group, food consumption data of women aged 20 to 40 in the DNFCS 2007-2010 were used as a proxy for pregnant women, because no food consumption data were available for this population group. The age limits of this population group were taken from EFSA (2012b) for reasons of comparison. The reported percentiles of the long-term exposure distribution were the 50th (median, P50) and 95th (P95).

Lead concentration database contained samples with lead concentrations below the limit of detection (LOD) or quantification (LOQ). In this report, these samples are referred to as non-detect samples and were assigned a lead concentration equal to ½LOD or ½LOQ (medium bound (MB) scenario). Non-detect samples of drinking water were reported as below the limit of reporting (LOR). Since this LOR was very low (maximally 6 µg/kg), we also assume this limit value to be either an LOD or LOQ. To study the sensitivity of the exposure estimates to the concentration assigned to the non-detect samples, two additional scenarios were performed in which either zero (lower bound (LB) scenario) or the limit value itself (upper bound (UB) scenario) was used.

After imputing the non-detect samples with LB, MB or UB values, lead concentrations were subsequently included in the exposure assessment by fitting a lognormal distribution to the samples with observed positive measurements per food (group). The non-detect samples were modelled as a proportion of samples below LOD or LOQ. This approach is recommended in the refined long-term exposure assessment (EFSA, 2012a). To model the concentrations in this way, the 'NonDetectSpike LogNormal' option within MCRA was used (van der Voet et al., 2015; de Boer et al., 2016). For a long-term exposure assessment, a mean concentration was subsequently calculated from both the positive and LB, MB or UB imputed values per food (group) and used in the exposure assessment[2]. Figure 1 shows an example of a NonDetectSpike-LogNormal distribution fitted to the Dutch monitoring data of the food groups 'berries and small fruits' and 'oilseeds'.

For fitting a lognormal distribution to the positive samples, at least two of such samples should be available for a certain food (group). In the present study, this was not true for the concentration data obtained from the mTDS and part of the Dutch monitoring data (Appendix C). These concentrations data were therefore included as such (so-called empirical modelling) in the exposure assessment. Concentrations of EFSA (2012b) were also included via empirical modelling: only available as already calculated LB, MB and UB mean concentrations per food (group). Appendix C lists the mean lead concentrations per food (group) used in the three exposure scenarios.

[2] For example, if a food (group) consists for 60% of non-detect samples, the MB concentration was calculated as 0.6 x MB imputed value + 0.4 x mean concentration of lognormal positive distribution. For the LB and UB concentrations, the LB and UB imputed values were used.

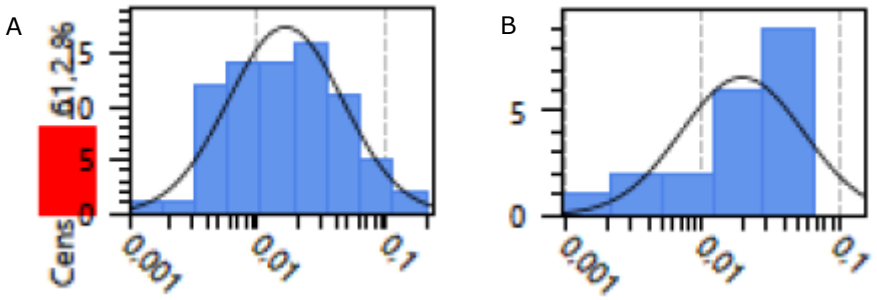

Figure 1. Example of a 'NonDetectSpike LogNormal' distribution fitted to the lead concentrations of the food group 'Berries and small fruits' (A) (61% non-detect samples) and 'Oilseeds' (B) (0% non-detect samples)

In order to evaluate the uncertainty in the dietary exposure assessment due to the sampling size of the concentration and food consumption database, the bootstrap approach was used. Per bootstrap sample of the concentration data, the concentration modelling as described above was repeated. The uncertainty is reported as the 95% confidence interval around the median and P95 of exposure. To quantify the uncertainty due to sampling size of the concentration data with this approach, more than one analysed sample should be available per food (group). Due to this, the majority of the mTDS samples and the concentration data of EFSA could not be addressed in this way, and their uncertainty due to sampling size was therefore not quantified (Appendix C). See Appendix E for a description of the bootstrap.

2.5 Calculation of margins of exposure

To assess if there is a health risk related to the exposure to lead, MOEs were calculated for the median and P95 of long-term exposure. Given the BMDLs derived by the CONTAM Panel (EFSA, 2010) and the available food consumption data, the MOEs were calculated for the following endpoints and population groups:

- Developmental neurotoxicity
  - $BMDL_{01}$ = 0.5 µg/kg bw per day: children aged 2 to 6 and children aged 7
  - $BMDL_{01}$ = 0.54 µg/kg bw per day: women of childbearing age aged 20 to 40
- Nephrotoxicity
  - $BMDL_{10}$ = 0.63 µg/kg bw per day: adults aged 18 to 69
- Cardiovascular effects
  - $BMDL_{01}$ = 1.50 µg/kg bw per day: adults aged 18 to 69

3 Results

3.1 Exposure to lead

Figure 2 shows the median (P50) and P95 of long-term dietary lead exposure in children aged 2 to 6 and in persons aged 7 to 69,

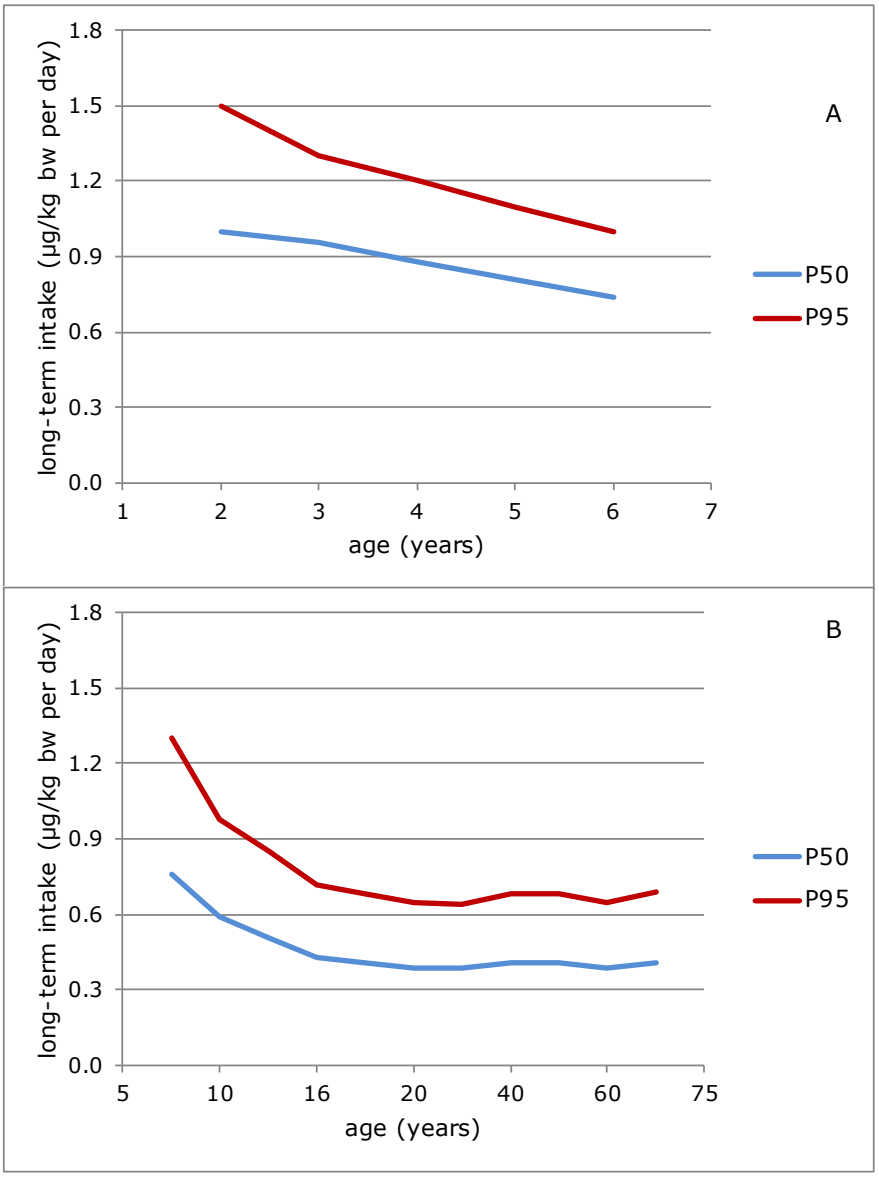

Figure 2. The median (P50) and high (P95) long-term dietary exposure[3,4] to lead as a function of age in young children aged 2 to 6 (A) and in persons aged 7 to 69 (B) in the Netherlands in which samples with a lead concentration below the limit of detection or quantification were assumed to contain lead at half of the relevant limit value (medium bound scenario)

[3] Best (point) estimate of the median exposure within 95% confidence interval (Appendix F).
[4] Best (point) estimate of the P95 of exposure within 95% confidence interval (Appendix F).

respectively, in the MB scenario. The exposure in women of childbearing age was similar to the exposure in person aged 7 to 69 for the ages 20 to 40. Appendix F lists the exposure estimates for all three scenarios, including 95% confidence intervals.

In 2- to 6-year olds, the exposure decreased with age (Figure 2A). The MB median exposure decreased from 1.0 µg/kg bw per day at age 2 to 0.74 µg/kg bw per day at age 6. Corresponding estimates for the high (P95) exposure were 1.5 and 1.0 µg/kg bw per day. The MB estimates of the median and high (P95) exposure in the whole age group equalled 0.88 and 1.3 µg/kg bw per day, respectively. Considering the uncertainty around the exposure estimates due to sampling size of the concentration and consumption database (section 2.4), the high (P95) exposure to lead could be as high as 1.8 µg/kg bw per day in 2-year olds.

In persons aged 7 to 69, exposure decreased further with age (Figure 2B). The MB median exposure ranged from 0.76 µg/kg bw per day in 7-year olds to 0.39-0.43 µg/kg bw per day in persons from age 16 onwards. Corresponding estimates for the high (P95) exposure were 1.3 and 0.64-0.72 µg/kg bw per day. Overall, the MB estimates of the median and high (P95) exposure in persons aged 7 to 69 equalled 0.41 and 0.74 µg/kg bw per day, respectively. Considering the sampling size uncertainty around these exposure estimates, the high (P95) exposure to lead could be as high as 1.4 µg/kg bw per day in 7-year olds.

The exposure to lead in women of childbearing age was stable across the ages. The MB median and high (P95) exposures were 0.41 and 0.76 µg/kg bw per day, respectively (Appendix F). The overall high (P95) exposure could be as high as 0.80 µg/kg bw per day considering the sampling size uncertainty.

3.2 Contribution of food groups

Figure 3 shows the food groups that contributed at least 5% to the MB total long-term exposure to lead in the three population groups. In children aged 2 to 6, the food groups ‘grains and grain-based products’, ‘fruit and fruit products’, ‘milk and dairy products’, ‘sugar and confectionary’ and ‘vegetables and vegetables products’ contributed at least 10% to the overall exposure to lead (Figure 3A). Together, these food groups contributed in total about 74% to the exposure. Within the food group ‘grains and grain-based products’, bread contributed most to the exposure (56%). Within the other three food groups, apple (38%), milk (85%), chocolate (cocoa) products (43%), and brassica (23%) and leaf vegetables (22%) were the main contributors, respectively.

In persons aged 7 to 69, four food groups contributed for at least 10% to the MB total long-term exposure to lead: ‘grains and grain-based products’, ‘non-alcoholic beverages’, ‘vegetable and vegetable products’ and ‘milk and dairy products’ (Figure 3B). Together, they contributed 61% to the exposure. In women of childbearing age, the same food groups contributed at least 10% to the MB long-term exposure to lead, adding up to 69% (Figure 3C). In both population groups, the contribution of the food group ‘non-alcoholic beverages’ was mainly due

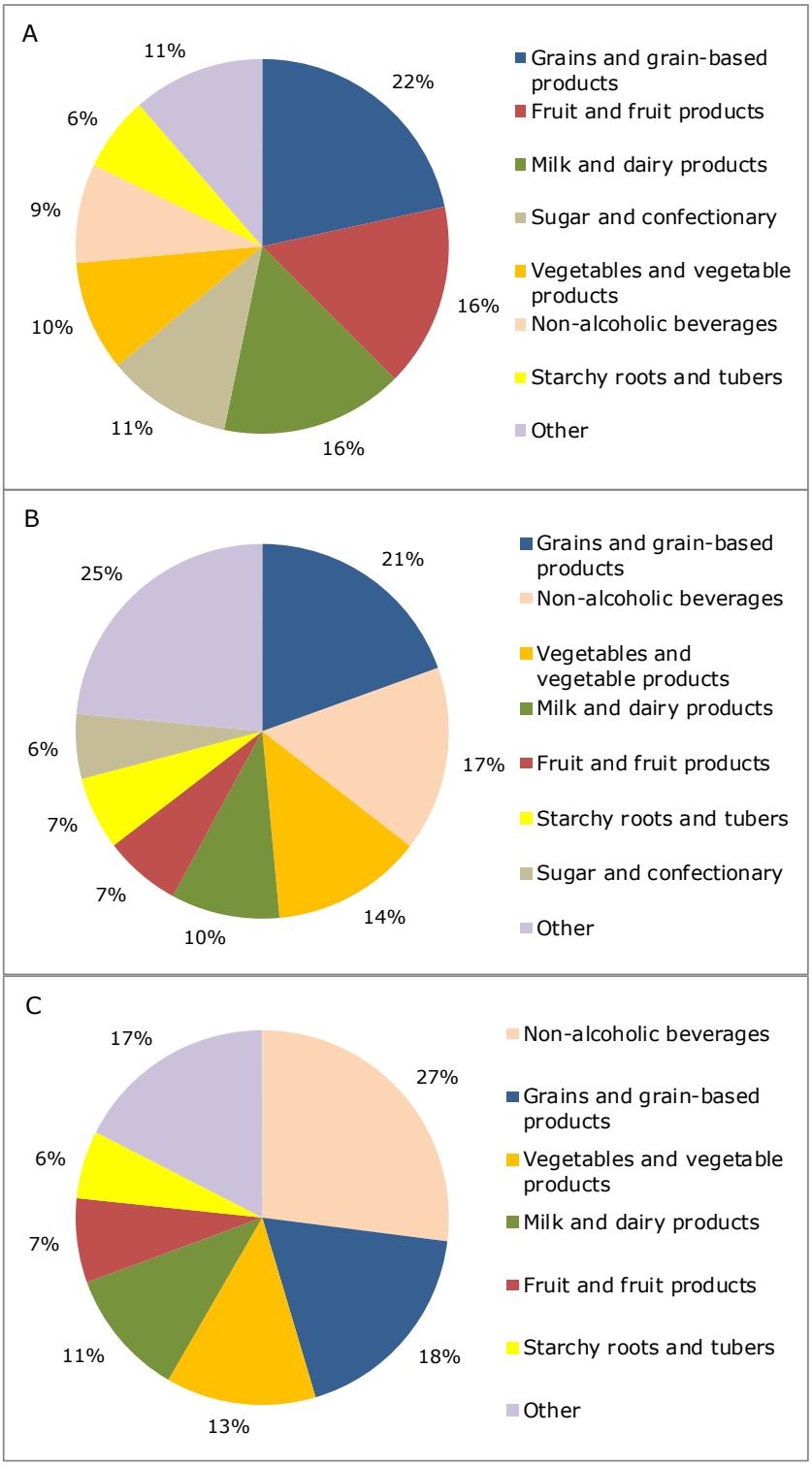

Figure 3. Contribution (%) of food groups, with a contribution of at least 5%, to the total long-term dietary exposure to lead in children aged 2 to 6 (A), persons aged 7 to 69 (B) and women of childbearing age (C) in the Netherlands in which samples with a lead concentration below limit of detection or quantification were assumed to contain lead at half of the relevant limit value (medium bound scenario)

to the consumption of tea (on average at least 65% of the food group). All vegetables contributed to the lead exposure. In the other two food groups, i.e. 'grains and grain-based products' and 'milk and dairy based products', the foods contributing most to the exposure within a particular food group were the same as those in children aged 2 to 6: bread and milk, respectively.

3.3 Calculation of margins of exposure

Table 1 lists the calculated MOEs belonging to the MB median and P95 level of exposure per population group. None of the calculated MOEs was larger than 10.

In children aged 2 to 6 and 7, for which developmental neurotoxicity was the most critical effect, the MOEs were lower than one for both the MB median and high (P95) levels of exposure (Table 1). This was also true for the high lead intake of women of childbearing age. In adults, the MOEs for cardiovascular effects were higher than one for both percentiles of exposure in the MB scenario (Table 1). The MOEs for nephrotoxicity were higher than one at the median exposure level, but below one at the high exposure estimate (Table 1). Considering the sampling size uncertainty around the estimated MOEs, the MOEs could

Table 1. Estimated margins of exposure (MOE) for the median and P95 long-term exposure[a] to lead in children aged 2 to 6, children aged 7, adults and women of childbearing age living in the Netherlands using the relevant 95th percentile lower confidence limit of the benchmark dose (BMDL) for three toxicological endpoints

Population and endpoint[b]	Margin of exposure	
	Median	P95
Children aged 2 to 6		
Developmental neurotoxicity	0.57 [0.51-0.60]	0.38 [0.33-0.42]
Children aged 7		
Development neurotoxicity	0.66 [0.61-0.68]	0.38 [0.36-0.42]
Adults		
Cardiovascular effects	3.7 [3.3-3.9]	2.1 [1.9-2.3]
Nephrotoxicity	1.5 [1.4-1.6]	0.90 [0.79-0.95]
Women of childbearing age[c]		
Development neurotoxicity	1.3 [1.3-1.4]	0.71 [0.68-0.76]

Note: Between brackets, the MOEs corresponding with the 2.5% lower and 97.5% upper confidence limit of the medium bound estimates of exposure are reported.
[a] Samples with a lead concentration below limit of detection or quantification were assumed to contain lead at half of the relevant limit value (medium bound scenario)
[b] For developmental neurotoxicity, the MOE were calculated by dividing the $BMDL_{01}$ of 0.50 µg/kg bw per day by the dietary exposure estimates in children aged 2 to 6 (overall) and 7, and by dividing the $BMDL_{01}$ of 0.54 µg/kg bw per day by the overall dietary exposure estimates in women of childbearing age (Appendix F). For cardiovascular effects and nephrotoxicity, the $BMDL_{01}$ of 1.50 µg/kg bw per day and the $BMDL_{10}$ of 0.63 µg/kg bw per day, respectively, were divided by the overall dietary exposure estimates in adults aged 18 to 69.
[c] Proxy for pregnant women; covered food consumption data of women aged 20 to 40.

be as low as 0.33 at the high level of exposure in children aged 2 to 6 and as high as 3.9 at the median exposure level in adults for cardiovascular effects.

4 Discussion

The current study describes the dietary exposure to lead in the population aged 2 to 69 in the Netherlands. Below, the results are discussed in relation to those reported by EFSA (2012b) and estimated within a national exposure assessment study (Boon et al., 2012), both published in 2012 (section 4.1), and in relation to the results of total diet studies performed in France, Ireland and the UK (section 4.2). In addition, the methodology and input data used in the assessment are discussed (section 4.3), as well as the estimated margins of exposure (section 4.4).

4.1 Comparison with lead intake reported by EFSA, Boon et al (2012) and three total diet studies

EFSA (2012b)

In 2012, EFSA reported on the exposure to lead via food in several European countries, including the Netherlands (EFSA, 2012b). Exposure estimates for the Netherlands were based on food consumption data from the DNFCS 2003 (Ocké et al., 2005) and the DNFCS-Young Children of 2005/2006 (Ocké et al., 2008) combined with lead concentrations of at least 25 European countries. Exposure results are reported in Table 2, including the estimates of the current study. For reasons of comparison, also the estimated exposures of adults are reported. The comparison shows that the exposure in both children aged 2 to 6 and adults tended to be lower in the current study (Table 2)[5]. These differences in lead exposure can be due to three factors: 1) the calculation model used, 2) the food classification system used, and/or 3) the concentration database used.

In the current study, a statistical model was used to assess long-term exposure (section 2.4), whereas EFSA used an approach in which the mean exposure over the recording days per individual is taken as a proxy for long-term exposure. This last approach is known to result in an overestimation of the upper percentiles of the exposure distribution, whereas the average exposure levels are not influenced (Boon & van der Voet, 2015). Secondly, EFSA used the FoodEx1 classification system to map the foods analysed to those consumed (section 2.3.1). This system consists largely of broad food categories and mapping may thus result in imprecise exposure results, especially for heterogeneous food groups (Boon et al., 2014). How this has affected the exposure calculated by EFSA (2012b) demands a critical examination of the data used. In practice however, use of broad food categories to assess the exposure results habitually in overestimations, due to conservative choices during mapping. Lastly, also the used concentration data may have resulted in a lower exposure reported in the current study. The concentration data used in the current assessment were partly derived from Dutch

[5] Note that EFSA (2012b) reported mean exposures as opposed to median exposures in the current study. Given the symmetrical distribution of the intake of lead in our study (Appendix F), the median exposure will closely resemble the mean exposure.

Table 2. Mean, median (P50) and high (P95) lead dietary exposure[a] in children aged 2 to 6 and adults in the Netherlands as estimated by EFSA (2012b), Boon et al. (2012) and current study

Age (years)	Dutch National Food Consumption Survey	N[b]	Exposure (µg/kg bw per day)	
			Mean[c] / P50[d]	P95
EFSA (2012b)				
2	2005/2006	322	1.5 [1.3-1.7]	2.6 [2.3-2.9]
3-6	2005/2006	957	1.2 [1.1-1.4]	2.0 [1.8-2.2]
19-30	2003	750	0.57 [0.49-0.65]	0.99 [0.83-1.2]
Boon et al. (2012)				
2-6	2005/2006	1279	0.53-0.76[e,f] [0.23-1.3]	0.73-1.0 [0.33-2.1]
Current study				
2-6	2005/2006	1279	0.88 [0.83-0.99]	1.3 [1.2-1.5]
7-69	2007/2010	3819	0.41 [0.40-0.44]	0.74 [0.71-0.82]
18-69	2007/2010	2230	0.41 [0.39-0.45]	0.70 [0.66-0.80]

Note: Estimates between brackets relate to the corresponding lower (LB) and upper bound (UB) estimates of exposure in which samples with a lead concentration below limit of detection (LOD) and quantification (LOQ) were assumed to contain no lead (LB) or lead at the limit value (UB).

[a] Samples with a lead concentration below LOD or LOQ were assumed to contain lead at half of the relevant limit value (medium bound (MB))

[b] N = number of individuals

[c] Estimates of EFSA (2012b) are mean levels

[d] Estimates of Boon et al. (2012) and the present study are median (P50) levels

[e] Boon et al. (2012) reported the exposure per age. The results presented here are the range of MB estimates of exposure across the six ages. Between brackets, the LB and UB dietary exposure estimates across the ages are reported.

[f] The exposure estimates in Boon et al. (2012) are lower than those reported in the current study, mainly because not all sources of exposure were considered. For more details, see text.

monitoring data, supplemented with data derived from EFSA (Appendix C). Examining the MB mean lead concentrations of the food groups contributing largely to the lead exposure (section 3.2) showed that concentrations in especially fruit and fruit products (except citrus fruit) and breakfast cereals were higher in the current study, whereas those in milk and bread, two important contributors to the exposure in children, were comparable. The concentrations in vegetables were higher, lower or comparable to those reported by EFSA (2012b). For an overview of the MB mean concentrations per food group, see Table 3. Together with the inclusion of concentrations from EFSA (2012b) for a large number of food sources (Appendix C), it is not likely that a difference in concentrations has contributed significantly to lower exposure estimates in the current study. The lead levels in non-alcoholic beverages (including tea, mainly due to the presence of lead in tea leaves), which contributed largely to the exposure in persons aged 7 to 69 and women of childbearing age, were derived from EFSA (2012b).

Table 3. Comparison of the medium bound (MB)[a] concentrations used in the present study and those used by EFSA (2012b) for the food groups contributing largely to the overall exposure to lead and for which Dutch monitoring data were used in the assessment.

Food (group)	Concentration (mg/kg)	
	Dutch monitoring	EFSA (2012b)
Fruit and fruit products		
Berries and small fruits	0.026	0.015
Citrus fruits	0.005	0.012
Miscellaneous fruits	0.022	0.011
Pome fruits	0.020	0.012
Grains and grain-based products		
Biscuits	0.025	0.02
Breakfast cereals	0.102[b]	0.025
Bread	0.025	0.029
Grain milling products	0.019	0.029
Pasta	0.025	0.008
Rice	0.025	0.026
Milk and dairy products[c]		
Milk	0.0053	0.004
Vegetables and vegetable products		
Brassica vegetables	0.042	0.013
Bulb vegetables	0.024	0.031
Fruiting vegetables	0.023	0.011
Fungi, cultivated	0.019	0.057
Leaf vegetables	0.038	0.041
Legume vegetables	0.009	0.026
Legumes, beans, dried	0.031	0.034
Root vegetable	0.039	0.019
Seaweed	1.23	2.7
Stem vegetables	0.022	0.021

[a] Samples with a lead concentration below limit of detection or quantification were assumed to contain lead at half of the relevant limit value
[b] Mean concentration of mTDS samples breakfast cereals (Brinta/Bambix) and breakfast cereals (cornflakes) (Appendix C)
[c] Concentration of dairy products was obtained from EFSA (2012b)

Boon et al (2012)

In 2012, also a national Dutch study into the exposure to lead in children aged 2 to 6 was published (Boon et al., 2012). Compared to this national study, the exposure in children aged 2 to 6 tended to be higher in the present study (Table 2). Given that the consumption data used in both studies were the same and both studies used a statistical model[6] to assess the long-term exposure, the difference in exposure was due to the concentration data used. In the current assessment, more possible sources of exposure were considered than in Boon et al. (2012), such as nuts, tea, and a larger group of vegetables and fruits.

[6] Boon et al (2012) used the BetaBinomial-Normal (BBN) model to estimate the long-term exposure to lead. BBN gives usually results that are very similar to LNN in cases with no correlation between intake frequency and intake amount (Boon & van der Voet, 2015), as was assumed in the current assessment (Appendix D).

In Boon et al (2012), the possible sources of exposure not included in the Dutch monitoring data were only supplemented with data of wheat and eggs from EFSA (2010). Furthermore, products with only lead monitoring levels below the LOD or LOQ were assumed to contain no lead in the MB and UB scenario, even if they belonged to a food group that included products that were likely to contain lead. Examples of such products were lambs lettuce, banana, Chinese cabbage and oranges. This may also have contributed to lower MB and UB exposure estimates in Boon et al (2012).

Total diet studies

In France, Ireland and the UK, total diet studies (TDSs) have been performed to assess the exposure to lead (including other substances) (Arnich et al., 2012; FSAI, 2016; Rose et al., 2010). In these studies, the exposure to lead was estimated based on lead concentrations analysed in a wide range of representative national composite samples of specified food groups. The estimated exposures to lead are listed in Table 4. In these studies, the non-detect samples were addressed in the same way as in the current study.

The exposures estimated in the TDSs were significantly lower than those of the current study, but not more than about a factor of five. A comparison of lead concentrations showed that this difference was at least partly due to lower levels of lead in comparable food groups analysed in the TDSs (Table 5). Lead levels in the food groups bread, breakfast cereals, fruit and tea were lower in the TDSs. Since these

Table 4. Mean, median and high (P95 and P97.5) exposure to lead in children and adults estimated in the current study and in three total diet studies (TDS).

Study and age group (years)	Scenario[a]	Exposure (µg/kg bw per day)		
		Median[b] / mean	P95	P97.5
the Netherlands (current study)				
2-6	LB-UB	0.43-1.3	0.70-2.0	-
18-69	LB-UB	0.24-0.58	0.46-0.97	-
TDS France[c]				
3-17	MB	0.27	0.57	-
18-79	MB	0.20	0.35	-
TDS UK[d]				
1.5-4.5	LB-UB	0.21-0.25	-	0.38-0.42
4-18	LB-UB	0.13-0.15	-	0.26-0.30
16-64	LB-UB	0.09-0.10	-	0.17-0.18
TDS Ireland[e]				
5-12	LB-UB	0.04-0.17	-	0.09-0.27
≥ 18	LB-UB	0.04-0.12	-	0.11-0.22

[a] LB (lower bound): samples with a lead concentration below limit of detection (LOD) or quantification (LOQ) (non-detect samples) were assumed to contain no lead; UB (upper bound): non-detect samples were assumed to contain lead at the relevant limit value. In the French TDS MB scenario, samples with a lead concentration below LOD were assumed to contain lead at ½LOD and those below the limit of quantification (LOQ) at ½LOQ. No LB and UB exposure estimates for lead were reported in this study, because for all food groups considered at least 40% of samples contained lead at concentrations > LOD.
[b] Estimates of the present study are median (P50) levels
[c] Arnich et al., 2012
[d] Rose et al., 2010
[e] FSAI, 2016

Table 5. Mean lead concentrations[a] (mg/kg) per food group used in the current study and in three total diet studies[b] (TDS) to estimate the exposure to lead via food.

Food group	Lead concentration (mg/kg)			
	Current study	TDS France	TDS Ireland	TDS UK
Bread	(0.025)	0.017	-	(0.011)
Breakfast cereals	0.102[c]	0.005	0-0.01	(0.007)
Dairy products	0.07-0.044[d]	0.006	0-0.01	<0.003
Drinking water	0.0007	0.002	0-0.001	-
Eggs	0.012	0.004	0-0.01	<0.003
Fats & oils	0.016-0.023	0.0039	0-0.01	<0.006
Fish	0.021	0.004	0-0.22	(0.004)
Fruit	0.005-0.026	0.005	0-0.01	(0.002)
Meat	0.003-0.009[e]	0.008	0-0.03	(0.005)
Milk	0.0053	0.006	-	(0.001)
Nuts	0.019-0.081	-	0-0.01	-
Offal	0.013-0.065	0.020	-	0.065
Poultry	0.017	0.004[f]	-	<0.003
Tea & coffee	0.012/0.002[g]	0.003	0-0.00	-
Vegetables	0.009-0.042	0.008	0-0.01	0.009[h]

Note: Parentheses mean that the measured concentrations are below the limit of detection (LOD) or quantification (LOQ)
[a] Concentrations relate to the medium bound scenario: samples with a lead concentration below LOD or LOQ were assumed to contain lead at half the relevant limit value, except for the French TDS. In this TDS, samples with a lead concentration below LOD were assumed to contain lead at ½LOD and those below LOQ at ½LOQ in the medium bound scenario.
[b] For the references of the three TDSs, see footnote c-e of Table 4.
[c] Mean concentration of mTDS samples breakfast cereals (Brinta/Bambix) and breakfast cereals (cornflakes) (Appendix C)
[d] Lead concentrations in cheese (Appendix C)
[e] Range of lead concentrations in beef, pork and mutton (Appendix C)
[f] Concentration of food group 'poultry and game'
[g] Lead concentration of coffee as a beverage based on a lead concentration of 0.043 mg/kg in coffee beans (Appendix C) and a dilution factor of 18 (EFSA, 2012b)
[h] Mean concentration of food groups 'green vegetables' (0.004 mg/kg) and 'other vegetables' (0.013 mg/kg)

foods belonged to the food groups that contributed largely to the exposure in the present study (section 3.3); this may have contributed to the lower exposure levels in the TDSs. In these studies, foods were prepared before consumption, if relevant. This has very likely not resulted in lower lead concentrations compared to the current study. In none of the three studies, as well as in EFSA (2012b), effects of processing on lead levels are mentioned. The approach to assess the exposure was similar to the approach taken in the current study in the Irish TDS. In the French and the UK TDS, a similar approach was used as by EFSA (2012b), resulting very likely in overestimations of the higher percentiles of exposure. Differences in food consumption may also have contributed to the observed differences in exposure.

4.2 Uncertainties in the exposure assessment

The exposure estimates of lead presented in this report are influenced by different sources of uncertainty. The most important sources are summarized in Table 6, including the direction and magnitude of the uncertainty relative to the exposure estimate, using the format as

proposed by EFSA (2006). The most important sources are discussed in detail below.

4.2.1 *Food consumption data*

The food consumption data used in the exposure assessment to lead were the most recent food consumption data available for the Netherlands (Appendix A). However, especially the food consumption data of children aged 2 to 6 were collected more than 10 years ago. Presently, a new DNFCS is being conducted among persons aged 1 to 79. Preliminary results of this survey gathered in the period of 2012-2014 show that consumption patterns are changing[7]. For example, looking at relevant food groups for the intake of lead, the consumption of dairy products and meat has decreased, of non-alcoholic beverages (mainly coffee, tea and drinking water) has increased, and of vegetables and cereal products has been unchanged compared to the consumption levels used in the present study. In children, also the consumption of fruit has increased. These changes in consumption patterns will very likely affect the exposure to lead.

4.2.2 *Concentration data*

The main limitation of the present study was the concentration data used in the assessment. The Dutch monitoring data available to assess the exposure was limited or not available for certain foods or food groups. This was addressed in two ways: 1) analysed RACs were grouped in food groups according to the FoodEx1 classification, and 2) data were supplemented with data used in EFSA (2012b). By grouping RACs in food groups before mapping them to foods recorded in the DNFCS, the number of analytical data per RAC within a food group was increased. Furthermore, by mapping these concentrations to all consumed foods belonging to the relevant food group, including often foods for which no lead concentrations were available, underestimation of the exposure was minimised. This improves the exposure assessment if all RACs within the food group contain similar lead concentrations. If the mean concentration of the available RACs is systematically higher or lower than the (unknown) mean concentration of a food group, this may potentially result in an over- or underestimation of the exposure, respectively. The extent by which the exposure results were affected by this is not clear. The data available were too limited to ascertain this.

Missing lead concentrations were further supplemented with "European" concentrations published in EFSA (2012b). These "European" data covered the period of 2003 up to 2011, and were from 20 EU Member States and Norway. No data from the Netherlands were included. These data were used in the current study assuming that due to open trading of foods between EU Member States, products available on the Dutch market will very likely have comparable mean lead levels. Despite this, the use of "European" data instead of national data may have introduced uncertainty in the reported exposure estimates. For example, locally produced food products may have a diverging mean lead concentration due to differences in soil lead levels. The "European" data were

[7] Factsheet 'Voedselconsumptie in Nederland. Wat, waar en wanneer? (www.rivm.nl/Onderwerpen/V/Voedselconsumptiepeiling)

Table 6. Sources, direction and magnitude of uncertainty in dietary exposure assessment to lead.

Source of uncertainty[a]	Direction & magnitude[b]	Section[c]
Food consumption data		
Food consumption data of 2005-2006 and 2007-2010	+/-	4.2.1
Concentrations		
Grouping of monitoring data in food groups	+	4.2.2 and 4.2.3
Samples with a concentration < LOD or LOQ were assumed to contain • No lead (lower bound) • Lead at half the limit value (medium bound) • Lead at the limit value (upper bound)	 - ++ +++	4.2.2
Representativity samples for consumed foods	-/+	4.2.2 and 4.2.3
Use of lead concentrations of 2003-2011	+	4.2.2
Food mapping via RACs and food groups	-/+	4.2.3
Model uncertainty		
LNN	•	4.2.4
Exposure via other sources		
Not included in the present assessment	•	4.2.5
Overall assessment: Based on this qualitative evaluation of different uncertainty sources it was concluded that the exposure to lead in the MB scenario might be conservative due to the use of ½LOD or ½LOQ in the assessment, mapping at food group level and the use of concentrations of 2003-2010.	+	

LOD: limit of detection; LOQ: limit of quantification; RAC: raw agricultural commodity; LNN: LogisticNormal-Normal

[a] Apart from the listed sources of uncertainty, also the uncertainty due to sampling size of the concentration and food consumption data was quantified via a bootstrap analysis (Appendix E). This uncertainty was quantified as the 95% confidence interval around the estimated percentiles of exposure (section 2.4).

[b] Key to direction and magnitude

+, ++, +++ = uncertainty likely to cause small, medium or large overestimation of exposure

-, --, --- = uncertainty likely to cause small, medium or large underestimation of exposure

• = uncertainty likely to cause a negligible effect on exposure estimate

[c] Section in which the uncertainty source is discussed

furthermore older than the Dutch monitoring data. Ideally, all concentration data used in the assessment would have covered the period 2010-2015.

Due to the use of unleaded petrol and paint, and the replacement of lead water pipes, the presence of lead in the environment has decreased in the last decades (Otte et al., 2015). EFSA evaluated the trend in lead concentrations in food over the sampling period of 2003-2010, excluding 2011 due to too few results (EFSA, 2010). An overall decrease in lead levels by 23% was observed. If this trend is extended to the present day, the use of the "European" data may have resulted in an overestimation of the exposure. Also comparing the monitoring data used in Boon et al (2012) to those used in the present study showed that in some foods the lead levels were decreased. For example, the MB mean lead concentrations in drinking water and liver of pig were about

50% lower (0.0007 versus 0.0018 mg/kg and 0.013 versus 0.028 mg/kg, respectively). No lower exposure estimates were however observed in the current study compared to the 2012 study due to the inclusion of more possible food sources of exposure (section 4.1).

Additionally, monitoring data refer to concentrations analysed in samples that are obtained to monitor compliance with maximum limits set in legislation. These samples may therefore be targeted to RACs that are suspected to exceed these limits, and may thus not represent the concentrations to which people are daily exposed. In the current assessment, only samples that were not labelled as being obtained via targeted sampling were included in the assessment. We therefore judge that this is of limited relevance in the current study.

Another important source of uncertainty related to the concentration data was the large number of non-detect samples (lead concentration below LOD or LOQ). To quantify this uncertainty in the exposure estimates, the exposure was assessed according to three exposure scenarios: lower (LB), medium (MB) and upper bound (UB) scenario (section 2.4). The exposure differed largely between the three scenarios (Appendix F), mainly due to the non-detect samples belonging to the food group 'grains and grain-based products' and 'milk and dairy products'. In children aged 2 to 6, the contribution of the food group 'grains and grain-based products' to the total lead exposure increased from 6% in the LB scenario to 22% in the MB scenario. For persons aged 7 to 69, the increase in contribution was comparable: 5% and 21%, respectively. In children aged 2 to 6, also the contribution of 'milk and dairy products' increased significantly: < 5% in the LB scenario up to 16% in the MB scenario. All samples of several analysed foods within the food group 'grains and grain products', such as bread, pasta and biscuits (Appendix B), as well as all milk samples (Appendix B) were reported to contain lead at levels below LOD and/or LOQ. Due to their relatively high consumption, the exposure increased in the MB and UB scenarios compared to the LB scenario.

Since all milk samples and the majority of the mTDS samples belonging to the food group 'grains and grain products' were non-detect samples (Appendix B and C), an additional exposure scenario was calculated for children aged 2 to 6. This scenario was similar to the MB scenario, except that all milk and mTDS samples (except for breakfast cereals (cornflakes) and muesli (Appendix B)) were assumed to contain no lead. In this scenario, the median and P95 exposure decreased to 0.60 and 0.96 µg/kg bw per day. These exposure estimates still result in MOEs < 1 (0.83 and 0.52, respectively). Given the observation that lead may be present in grains and grain-based products and milk (EFSA, 2012b), assuming that lead is not present in these foods, may underestimate the exposure.

4.2.3 Food mapping

The concentrations were mapped as much as possible to the foods recorded in the DNFCSs. To achieve this, either mapping via a food conversion model or direct mapping was used (section 2.3). Food mapping is potentially a large source of uncertainty in an exposure assessment, since the foods analysed are often not those actually

consumed. Lead is analysed in RACs within monitoring programmes to establish if maximum limits, set in Commission Regulation (EC) Nr 1881/2006, are met. These analyses are performed as part of different monitoring obligations prescribed in legislation and therefore available every year. However, these data can only be used in a complete exposure assessment via a food conversion model. Advantage of this is that concentrations analysed in RACs are mapped to consumed amounts of composite foods, which contain these RACs as ingredient. These composite foods are thus included in the assessment without the need to analyse them separately. A disadvantage of this approach is that there is no direct link between analysed and consumed composite foods, as well as with prepared foods consisting of single ingredients. This last disadvantage is especially relevant for chemicals analysed in RACs of which the concentration is affected by preparation (e.g. cooking and peeling). As a result, there is always an uncertainty whether the calculated concentrations in consumed foods via the food conversion model are representative for the concentrations in those actually consumed. In the current study, lead concentrations in some foods estimated with the food conversion model differed largely from those analysed directly in the relevant foods (EFSA, 2012b). These estimated concentrations were therefore replaced by those reported in EFSA (2012b) (section 2.3.1). In addition, the composition of foods may change over time. These likely changes are presently not updated and therefore the composition may not be representative for the foods currently on the market. Furthermore, in the food conversion model variation in both composition and conversion factors is not addressed.

Direct mapping was used for the concentrations of the mTDS samples, of EFSA (2012b) and of RACs that were consumed as such (e.g. fruit) (section 2.3.2). Also for this type of mapping, assumptions were made to include all consumed foods that may potentially contain lead in the exposure assessment. For example, the overall mean lead concentration of the food group 'condiments' was mapped to all the different types of sauces recorded in the DNFCSs. Another example is the food group 'vegetable oils'. The average concentration of this food group was mapped to all the different types of vegetable oils recorded in the DNFCs.

Both types of mapping may have resulted in over- or underestimates of the exposure per food (group). However, given the large number of mapped foods, we estimate that overall the uncertainties may have levelled out in the final estimates.

4.2.4 Modelling of exposure

LNN is the preferred model to assess the long-term exposure, since this model corrects for the within-person's variation in exposure (Boon & van der Voet, 2015). This approach results in more realistic exposure estimates at the tails of the exposure distribution than without correction (Dodd et al., 2006; Hoffmann et al., 2002; Slob, 1993). However, the within-person's variation can only be removed when the daily positive exposure distribution is normally distributed after transformation. If this condition is not met, the use of LNN to assess the long-term exposure might be debatable. Normality can be checked by using the normal quantile–quantile (q–q) plot, a graphical display of

observed vs. theoretical residuals (de Boer et al., 2009). Examination of the q-q plots showed that the daily positive exposure distributions of lead in the three population groups could be considered close to normal (Appendix F), justifying the use of LNN to assess the long-term exposure.

The high (P95) exposure to lead was higher in 7-year olds than in 6-year olds and comparable to that in children aged 3 to 5 (Figure 2, Appendix F). The median exposure in this age group was estimated to be similar to that in 6-year olds, and lower than in 2- to 5-year olds. A high exposure in 7-year olds compared to children aged 2 to 6 was also observed in similar exposure assessments of cadmium (Sprong & Boon, 2015) and 3-MCPD (Boon & te Biesebeek, 2016). Due to differences in study design between the food consumption survey in children aged 2 to 6 and that of persons aged 7 to 69, this result is very likely due to methodological issues rather than real differences in exposure. In the exposure assessment to 3-MCPD, the underlying food consumption data were examined in more detail and no differences in consumption could be detected to explain the observed difference in exposure to 3-MCPD (Boon & te Biesebeek, 2015). The new DNFCS will cover ages 1 to 79 (section 4.2.1), foreclosing possible differences in intakes between age groups due to differences in study design.

4.2.5 Other sources of exposure

Children and adults are also exposed to lead through ingestion of dust and soil, and outdoor air contaminated with lead (EFSA, 2010). The CONTAM Panel calculated that the exposure via outdoor air was maximally 0.003 µg/kg bw per day in adults. Intake via soil may be an important health factor for children, especially in areas (inner cities) with lead contaminated soil (Otte et al., 2015). For such areas, municipalities are advised to reduce exposure to soil to a level as low as possible (Otte et al., 2015).

4.2.6 Summary

The different issues contributing to the uncertainty in the exposure estimates are summarized in Table 6. Overall, the estimated exposure to lead in the MB scenario is very likely overestimated due to the use of ½LOD or ½LOQ and the use of "European" data from 2003-2010. In addition, mapping at food group level for many foods may have resulted in an overestimation of the exposure.

4.3 Risk analysis

The CONTAM Panel has derived BMDLs based on cardiovascular effects, nephrotoxicity and developmental neurotoxicity (loss of one IQ point) to assess possible health risks related to the dietary exposure to lead (EFSA, 2010). These BMDLs were used in this report to calculate the margins of exposure (MOEs) related to the median (P50) and high (P95) intake of lead in the relevant population groups (section 3.3). A MOE of 10 or greater was considered to be of negligible public health concern (EFSA, 2010). At lower MOEs, but greater than one, the risk was considered to be very low for cardiovascular effects and nephrotoxicity, whereas for neurodevelopmental effects the risk was considered to be low, 'but not such that it could be dismissed as of no potential concern' (EFSA, 2010).

In adults, the estimated MOEs for cardiovascular effects for both the median and P95 levels of exposure were higher than one in the MB scenario, but below 10 (Table 1), indicating that risks from exposure to lead in foods for these effects are likely to be very low. For nephrotoxicity, the MB median exposure resulted in MOEs higher than one but below 10, whereas the MOE of the P95 was below one (0.85). The BMDLs for nephrotoxicity and cardiovascular effects were based on studies in adults, i.e. after prolonged exposure, and hence cannot be related to manifest disease during childhood (EFSA, 2010).

In children aged 2 to 6, the exposure to lead resulted in MOEs for neurodevelopmental neurotoxicity, the critical effect in this population group, below one: 0.38 at the P95 and 0.57 at the median exposure level (Table 1). In children aged 7, the MOEs for the same critical effect were also below one: 0.38 and 0.66, respectively. The developing foetus (through *in utero* exposure) may also experience loss of at least one IQ point at the P95 exposure of its mother (MOE = 0.71).

Given the uncertainty related to non-detect samples, also LB and UB estimates were calculated (Appendix F). This analysis showed that if it is assumed that non-detect samples do not contain lead (LB estimates), children aged 2 to 6 and 7 may still experience loss of at least one IQ point at the P95 level of exposure (MOE of around 0.7). In the other population groups, including women of child-bearing age (relevant for the developing foetus), the MOEs would be one or higher in the LB scenario.

Overall, the results show that the health risks of long-term exposure to lead are very low for cardiovascular effects in adults, but cannot be excluded for effects on the kidney in highly exposed adults. Additionally, a decrease in cognitive ability by at least one IQ point cannot be excluded in children up to age seven and in the developing foetus with highly exposed mothers.

4.4 Conclusion

The exposure estimates of lead indicate that health effects in certain population groups cannot be excluded (section 4.3). The current exposure assessment was however hampered by limited concentration data (section 4.2.2). Another uncertainty related to the concentration data was the large number of samples that contained lead at levels below the LOD or LOQ. This resulted in large differences in exposure between the LB, MB and UB exposure scenarios (Appendix F). We showed however that assuming that these samples do not contain lead (LB scenario), a loss of at least one IQ point in children aged 2 to 7 could still not be excluded at the P95 level of exposure. This LB estimate is expected to be lower than the true exposure: it is unlikely that all samples with a reported lead concentration below LOD or LOQ do not contain lead. Assuming that only milk samples and the mTDS samples (except for breakfast cereals (cornflakes) and muesli), which consisted solely of non-detect samples, did not contain lead, and that the other non-detect samples contained lead at a level equal to ½LOD or ½LOQ, resulted in MOEs < 1 at both the median and high level of exposure in children aged 2 to 6. Three total diet studies performed in France,

Ireland and the UK, in which representative samples of foods as consumed were analysed, showed that the exposure was generally lower compared to those calculated in the current study, resulting in MOEs higher than one for neurodevelopmental effects (section 4.1). However, in the French study, the MOE at the P95 level of exposure was lower than one in children aged 3 to 17. Given the decrease in exposure with age as observed in the current study (Figure 2), it is likely that the exposure in the relevant population (children up to age seven), would have been higher. The results of the French study show therefore also that there may be a potential health concern of lead exposure in young children.

To improve the lead intake assessment via food, reliable concentration data, preferably analysed in foods as close as possible to those actually consumed and including all possible food sources, are needed. Due to the possible still continuing decrease of lead concentration in food, these concentrations should be as recent as possible. In addition, the LOD and LOQ should be as low as possible to reduce the uncertainty in the exposure estimates due to non-detect samples, as well as to refine the levels to be assigned to these samples : samples < LOD are assigned a level equal to (a fraction of) the LOD and those ≥ LOD but below LOQ by a level equal to (a fraction of) the LOQ. This is especially relevant for those food groups that contributed largely to the exposure because of their high consumption levels, such as grain products, and milk and milk products. In the recent Irish TDS, an analytical method with an LOQ of 0.017 mg/kg and an LOD of 0.005 mg/kg was used to analyse lead (FSAI, 2016). The corresponding LOD of the mTDS samples was 0.05 mg/kg (Appendix B). Due to the possibility of additional routes of exposure, biomonitoring of lead in certain population groups (e.g. children) might be an alternative.

In the current exposure assessment, the exposure estimates were compared to the BMDLs derived by EFSA (2010) to assess potential health risks. Possible uncertainties of these limit values were not considered in this study. These uncertainties, together with those in the exposure estimates, should be considered in a full risk assessment of lead.

Lead is an environmental contaminant for which maximum limits (MLs) are set in Commission Regulation (EC) No. 1881/2006. In view of changes in food concentrations over time, these maximum limits are regularly evaluated to keep the intake of lead, as well as other contaminants regulated via this regulation, As Low As Reasonably Achievable (ALARA principle). Despite these MLs, and the fact that the used concentrations were predominantly below the relevant MLs, the intake of lead resulted in low MOEs in parts of the Dutch population. It is important to realise that MLs are not only based on the toxicity of the contaminant, but also on the presence of the contaminant in food. When the concentration of a contaminant like lead in food is lower than the relevant MLs, the intake may thus not always result in no health risk, or, in case of higher concentrations than the MLs, in a health risk.

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References

Arnich N, Sirot V, Rivière G, Jean J, Noël L, Guérin T, Leblanc J-C (2012). Dietary exposure to trace elements and health risk assessment in the 2nd French Total Diet Study. Food and Chemical Toxicology 50: 2432-2449.

Boon PE, Ruprich J, Petersen A, Moussavian S, Debegnach F, van Klaveren JD (2009). Harmonisation of food consumption data format for dietary exposure assessments of chemicals analysed in raw agricultural commodities. Food and Chemical Toxicology 47: 2883-2889.

Boon PE, te Biesebeek JD (2016). Preliminary assessment of dietary exposure to 3-MCPD in the Netherlands. Reportnr: 2015-0199. National Institute for Public Health and the Environment (RIVM), Bilthoven. Available online: www.rivm.nl.

Boon PE, te Biesebeek JD, Sioen I, Huybrechts I, Moschandreas J, Ruprich J, Turrini A, Azpiri M, Busk L, Christensen T, Kersting M, Lafay L, Liukkonen K-H, Papoutsou S, Serra-Majem L, Traczyk I, De Henauw S, van Klaveren JD (2012). Long-term dietary exposure to lead in young European children: comparing a pan-European approach with a national exposure assessment. Food Additives and Contaminants: Part A 29: 1701-1715.

Boon PE, van der Voet H (2015). Probabilistic dietary exposure models. Reportnr: 2015-0191. National Institute for Public Health and the Environment (RIVM), Bilthoven. Available online: www.rivm.nl.

Boon PE, van der Voet H, Ruprich J, Turrini A, Sand S, van Klaveren JD (2014). Computational tool for usual intake modelling workable at the European level. Food and Chemical Toxicology 74: 279-288.

de Boer WJ, Goedhart PW, Hart A, Kennedy MC, Kruisselbrink J, Owen H, Roelofs W, van der Voet H (2016). MCRA 8.2 a web-based program for Monte Carlo Risk Assessment. Reference Manual. December, 2016. Biometris, Wageningen UR, Food and Environmmental Research Agency (Fera) and National Institute for Public Health and the Environment (RIVM), Wageningen, Bilthoven, the Netherlands and York, UK.

de Boer WJ, van der Voet H, Bokkers BGH, Bakker MI, Boon PE (2009). Comparison of two models for the estimation of usual intake addressing zero consumptions and non-normality. Food Additives and Contaminants: Part A 26: 1433-1449.

Dodd KW, Guenther PM, Freedman LS, Subar AF, Kipnis V, Midthune D, Tooze JA, Krebs-Smith SM (2006). Statistical methods for estimating usual intake of nutrients and foods: a review of the theory. Journal of the American Dietetic Association 106: 1640-1650.

Efron B (1979). Bootstrap methods: another look at the jackknife. Annals of Statistics 7: 1-26.

Efron B, Tibshirani R (1993). An introduction to the bootstrap. New York: Chapman & Hall.

EFSA (2006). Opinion of the Scientific Committee related to uncertainties in dietary exposure assessment. The EFSA Journal 438: 1-54. Available online: www.efsa.europa.eu.

EFSA (2010). Scientific Opinion on lead in food. EFSA Journal 8(4): 1570. [147 pp.]. Available online: www.efsa.europa.eu.

EFSA (2011). Evaluation of the FoodEx, the food classification system applied to the development of the EFSA Comprehensive European Food Consumption Database. EFSA Journal 9(3): 1970. [27 pp.]. Available online: www.efsa.europa.eu.

EFSA (2012a). Guidance on the Use of Probabilistic Methodology for Modelling Dietary Exposure to Pesticide Residues. EFSA Journal 10(10): 2839. [95 pp.]. Available online: www.efsa.europa.eu.

EFSA (2012b). Lead dietary exposure in the European population. EFSA Journal 10(7): 2831. [59 pp.]. Available online: www.efsa.europa.eu.

EFSA (2015). Scientific Opinion on acrylamide in food. EFSA Panel on Contaminants in the Food Chain (CONTAM). EFSA Journal 13(6): 4104. [321 pp.]. Available online: www.efsa.europa.eu.

EFSA (2016). Scientific opinion on the risks for human health related to the presence of 3- and 2-monochloropropanediol (MCPD), and their fatty acid esters, and glycidyl fatty acid esters in food. EFSA Journal 2016;14(5): 4426. [159 pp.]. Available online: www.efsa.europa.eu.

FAO/WHO (1987). Evaluation of certain food additives and contaminants (Thirtieth report of the Joint FAO/WHO Expert Committee on Food Additives (JECFA)). WHO Technical Report Series, No. 751. World Health Organisation (WHO), Geneva. Available online: www.who.int.

FSAI (2016). Report on a total diet study carried out by the Food Saferty Authority of Ireland in the period 2012-2014. Food Safety Authority of Ireland, Dublin. Available online: www.fsai.ie.

Geraets L, te Biesebeek JD, van Donkersgoed G, Koopman N, Boon PE (2011). The intake of acrylamide, nitrate and ochratoxin A in the population aged 7 to 69 years living in the Netherlands. Reportnr: 2014-0002. National Institute for Public Health and the Environment (RIVM), Bilthoven. Available online: www.rivm.nl.

Goedhart PW, van der Voet H, Knüppel S, Dekkers ALM, Dodd KW, Boeing H, van Klaveren JD (2012). A comparison by simulation of different methods to estimate the usual intake distribution for episodically consumed foods. Supporting Publications 2012:EN-299. [65 pp]. Available online: www.efsa.europa.eu.

Hoffmann K, Boeing H, Dufour A, Volatier JL, Telman J, Virtanen M, Becker W, De Henauw S (2002). Estimating the distribution of usual dietary intake by short-term measurements. European Journal of Clinical Nutrition 56 Suppl. 2: S53-S62.

Ocké MC, Hulshof KFAM, van Rossum CTM (2005). The Dutch national food consumption survey 2003. Methodological issues. Archives of Public Health 63: 227-241.

Ocké MC, van Rossum CTM, Fransen HP, Buurma EJM, de Boer EJ, Brants HAM, Niekerk EM, van der Laan JD, Drijvers JJMM, Ghameshlou Z (2008). Dutch National Food Consumption Survey - Young children 2005/2006. Reportnr: 350070001/2008. National Institute for Public Health and the Environment (RIVM), Bilthoven. Available online: www.rivm.nl.

Otte PF, Bakker MI, Lijzen JPA, Versluijs CW, Zeilmaker MJ (2015). Diffuse loodverontreiniging in de bodem. Reportnr: 2015-0204. Rijksinstituut voor Volksgezondheid en Milieu (RIVM), Bilthoven. Available online: www.rivm.nl.

Rose M, Baxter M, Brereton N, Baskaran C (2010). Dietary exposure to metals and other elements in the 2006 UK Total Diet Study and some trends over the last 30 years. Food Additives and Contaminants: Part A 27: 1380-1404.

Slob W (1993). Modeling long-term exposure of the whole population to chemicals in food. Risk Analysis 13: 525-530.

Sprong RC, Boon PE (2015). Dietary exposure to cadmium in the Netherlands. Reportnr: 2015-0085. National Institute for Public Health and the Environment (RIVM), Bilthoven. Available online: www.rivm.nl.

Sprong RC, de Wit-Bos L, Zeilmaker MJ, Alewijn M, Castenmiller JJM, Mengelers MJB (2016). A mycotoxin-dedicated total diet study in the Netherlands in 2013: Part I – Design. World Mycotoxin Journal 9: 73-87.

van der Voet H, de Boer WJ, Kruisselbrink JW, Goedhart PW, van der Heijden GWAM, Kennedy MC, Boon PE, van Klaveren JD (2015). The MCRA model for probabilistic single-compound and cumulative risk assessment of pesticides. Food and Chemical Toxicology 79: 5-12.

van Dooren MMH, Boeijen I, van Klaveren JD, van Donkersgoed G, 1995. Conversie van consumeerbare voedingsmiddelen naar primaire agrarische produkten (Conversion of consumed foods into raw agricultural commodities). Reportnr: 95.17. RIKILT-Instituut voor Voedselveiligheid, Wageningen UR, Wageningen. Available online: www.rikilt.wur.nl.

van Rossum CTM, Fransen HP, Verkaik-Kloosterman J, Buurma-Rethans EJM, Ocké MC (2011). Dutch National Food Consumption Survey 2007-2010. Diet of children and adults aged 7 to 69 years. Reportnr: 350050006/2011. National Institute for Public Health and the Environment (RIVM), Bilthoven. Available online: www.rivm.nl.

Appendix A Description of consumption data used in the exposure assessment of lead

DNFCS-Young Children 2005/2006 (Ocké et al., 2008)
The target population of the DNFCS-Young Children 2005/2006 consisted of boys and girls aged 2 to 6 years living in the Netherlands. Respondents were selected from representative consumer panels of Market Research Agency GfK. Panel characteristics, such as socio-demographic characteristics, are known to GfK. Persons in these panels participate in all types of surveys and were not specially selected on nutritional characteristics. Institutionalised persons were excluded, as well as children whose parents/carers did not have sufficient knowledge of the Dutch language. Per family, only one child was included to avoid correlations in dietary consumption patterns between children of the same family. In total, 1,634 children were invited to participate in the study, of which 1,279 consented (net response of 78%). During recruitment, the representativeness of the study population was monitored and, if necessary, the recruitment was adjusted for age and sex, education of the head of the household, level of urbanisation, place of residence and region. The study population was representative regarding socio-demographic characteristics (including region and education of the head of the household), but densely populated areas were slightly underrepresented.

The food consumption data were collected in the period October 2005 to November 2006 on two non-consecutive days (separated by about 8 to 13 days) via food diary. Parents/carers were visited at home by a trained employee of GfK. During the home visit, survey materials were presented and overall instructions were given.

Portion size of the foods and meals were estimated by using photographs, domestic measures (a small and a large spoon were supplied to standardise estimates), standard units, weight and/or volume. The usual volume of cups and glasses used was measured by the carer. All days of the week were equally represented, but the winter and autumn period were slightly overrepresented compared to the spring and summer period. National and/or religious holidays or holidays of the participants were not included in the survey.

DNFCS 2007-2010 (van Rossum et al., 2011)
The target population of the DNFCS 2007-2010 consisted of people aged 7 to 69 years living in the Netherlands. Pregnant and breast-feeding women, as well as institutionalised people were not included. Respondents were selected from representative consumer panels of GfK. A maximum of one person per household was included in the survey to avoid correlations in dietary consumption patterns between members of the same family. In addition, the panels only included people with sufficient knowledge of the Dutch language. In total, 5,502 individuals were invited to participate in the study, of which 3,819 consented (net response of 69%). Children were overrepresented in the study population and adults underrepresented.

The food consumption data were collected over a 3-year period from March 2007 to April 2010 via two non-consecutive 24-hour dietary recalls (separated by 2 to 6 weeks). Children aged 7 to 15 years were interviewed face to face during home visits in the presence of at least one of the child's parents or carers. Participants aged 16 and over were interviewed by telephone, at dates and times unannounced to the participants.

Portion sizes of foods consumed were quantified in several ways: by means of quantities as shown on photos in a provided picture booklet, or in household measures, standard units, by weight and/or volume. The survey covered all days of the weeks and all four seasons. National and/or religious holidays or holidays of the participants were not included in the survey.

Appendix B Lead concentrations in bread and cereal products derived from the mTDS

Product	Concentration (mg/kg)
Biscuit	<0.050[a]
Breakfast cereals, Bambix-like ones	<0.050
Breakfast cereals, Brinta-like ones	<0.050
Breakfast cereals, cornflakes-like ones	0.179
Gingerbread	<0.050
Gingerbread, wholemeal	<0.050
Macaroni	<0.050
Maize bread	<0.050
Muesli, regular	0.054
Muesli, crunchy	<0.050
Noodles	<0.050
Oatmeal	<0.050
Rice, white	<0.050
Rice, brown	<0.050
Rye bread, dark	<0.050
Spaghetti	<0.050
Spiced biscuit	<0.050
Tortilla	<0.050
Wheat bread	<0.050
Wheat bread roll, soft	<0.050
White bread	<0.050
White bread roll	<0.050
White bread roll, hard	<0.050
Wholemeal bread	<0.050
Wholemeal bread with sunflower seeds	<0.050

[a] Lead concentration below limit of detection

Appendix C Overview of the lead concentrations used in the exposure assessment, including its source

Food (group)	Nr of samples (nr of non-detects)	Concentration (mg/kg)[a]		
		LB	MB	UB
EFSA[b]				
Alcoholic beverages	3554 (2239)	0.019	0.021	0.023
Almond, sweet	170 (99)	0.021	0.027	0.033
Amphibians, reptiles, snails, insects	31 (10)	0.041	0.044	0.047
Animal and vegetable oil and fat	1731 (1108)	0.017	0.020	0.022
Animal fat	566 (357)	0.014	0.016	0.018
Bottled water	2594 (1868)	0.0002	0.001	0.001
Brazil nuts	24 (5)	0.081	0.081	0.081
Cashew nuts	106 (80)	0.009	0.020	0.031
Cassava root	10 (0)	0.181	0.181	0.181
Cheese	1262 (707)	0.017	0.021	0.025
Cheese, Camembert	91 (55)	0.022	0.032	0.042
Cheese, Edam	56 (18)	0.020	0.021	0.021
Cheese, Emmental	8 (3)	0.035	0.037	0.040
Cheese, Gouda	6 (3)	0.041	0.044	0.048
Cheese, Mozzarella	14 (10)	0.026	0.029	0.031
Cheese, processed spreadable	14 (9)	0.005	0.007	0.008
Cheese, processed, plain	180 (50)	0.026	0.027	0.028
Cheese, Roquefort	10 (3)	0.010	0.010	0.011
Chestnuts	58 (48)	0.073	0.080	0.086
Chocolate (cocoa) products	723 (137)	0.053	0.055	0.057
Cider	81 (19)	0.013	0.014	0.014
Cocoa mass	5 (1)	0.053	0.054	0.056
Cocoa powder	349 (45)	0.138	0.139	0.139
Coconuts	125 (51)	0.024	0.026	0.027
Coffee beans	13 (58)	0.041	0.043	0.046
Condensed milk	60 (39)	0.010	0.011	0.012
Condiment	280 (78)	0.052	0.054	0.056
Confectionary (non-chocolate)	398 (239)	0.032	0.037	0.043
Dressing	63 (36)	0.024	0.030	0.037
Dried milk	207 (77)	0.027	0.028	0.029
French fries	27 (10)	0.005	0.005	0.006
Fruit and vegetable juices	2231 (1004)	0.009	0.010	0.012
Game birds	596 (304)	0.264	0.267	0.270
Gelatine	101 (28)	0.063	0.063	0.063
Hare meat	149 (88)	0.149	0.155	0.162
Hazelnuts	126 (58)	0.026	0.031	0.036
Macadamia	80 (75)	0.005	0.019	0.032
Main-crop potatoes	1028 (504)	0.018	0.020	0.023
Margarine and similar products	110 (88)	0.006	0.01	0.013
Margarine, normal fat	52 (42)	0.004	0.006	0.008

Food (group)	Nr of samples (nr of non-detects)	Concentration (mg/kg)[a]		
		LB	MB	UB
Mashed potato powder	126 (105)	0.022	0.030	0.038
Milk and milk product imitates	52 (32)	0.012	0.015	0.017
Molasses and other syrups	100 (58)	0.009	0.009	0.010
Non-alcoholic beverages	1520 (1049)	0.012	0.013	0.014
Other starchy roots and tubers	984 (423)	0.016	0.017	0.018
Pistachios	101 (53)	0.031	0.038	0.045
Popcorn	15 (4)	0.031	0.032	0.033
Potato starch	13 (8)	0.018	0.020	0.022
Potatoes and potato products	1370 (767)	0.016	0.019	0.022
Sauerkraut	76 (9)	0.018	0.018	0.018
Savoury sauces	45 (19)	0.046	0.053	0.059
Stone fruit	826 (537)	0.009	0.012	0.016
Sugars	181 (119)	0.012	0.013	0.014
Sweet potatoes	967 (425)	0.014	0.015	0.016
Tea (infusion)	764 (8)	0.012	0.012	0.012
Tomato puree	84 (22)	0.024	0.024	0.025
Tree nuts	983 (570)	0.026	0.033	0.039
Vegetable fat	114 (47)	0.023	0.023	0.024
Vegetable oil	924 (601)	0.020	0.023	0.026
Walnuts	164 (84)	0.026	0.031	0.036
Whole egg, chicken	1009 (726)	0.009	0.012	0.015
Wine	2302 (506)	0.023	0.025	0.027
Wine grapes	302 (236)	0.005	0.007	0.010
Yeast	32 (14)	0.015	0.016	0.017
Dutch monitoring data[c,d]				
Individual foods[e]				
Beef[f]	356 (176)	0.0066	0.0082	0.0097
Chicken[g]	327 (326)	0.0002	0.017	0.033
Deer (tamed)[h]	25 (24)	0.0004	0.024	0.048
Drinking water[i]	6822 (4968)	0.0004	0.0007	0.0009
Duck (tamed)	374 (351)	0.345	0.362	0.378
Honey	8 (7)	0.01	0.032	0.054
Horse	21 (21)	0	0.025	0.050
Kidney of bovine animal	356 (176)	0.053	0.065	0.078
Liver of poultry	327 (326)	0.0002	0.025	0.050
Liver of pig[j]	512 (510)	0.0002	0.013	0.025
Liver of calf	89 (84)	0.0019	0.014	0.026
Milk	66 (66)	0	0.0053	0.011
Mutton[k]	27 (13)	0.007	0.009	0.01
Ostrich	334 (333)	0.0002	0.017	0.034
Pork/piglet[j]	512 (510)	0.00005	0.003	0.006
Rabbit (domestic)	5 (5)	0	0.025	0.05
Seaweed	24 (2)	1.23	1.23	1.24
Tea powder	1 (0)	0.72	0.72	0.72
Turkey[g]	327 (326)	0.0002	0.017	0.033
Veal	89 (84)	0.0005	0.0034	0.0064

Food (group)	Nr of samples (nr of non-detects)	Concentration (mg/kg)[a]		
		LB	MB	UB
Grouped foods[l]				
Berries and small fruits	196 (120)	0.011	0.026	0.041
Brassica vegetables	26 (21)	0.027	0.042	0.057
Bulb vegetables	11 (10)	0.0014	0.024	0.047
Citrus fruits	10 (1)	0.004	0.005	0.005
Crustaceans	40 (34)	0.013	0.028	0.042
Fish and other seafood	632 (537)	0.006	0.021	0.036
Fruiting vegetables	172 (172)	0	0.023	0.047
Fungi, cultivated	93 (77)	0.011	0.019	0.024
Grain milling products	39 (32)	0.01	0.019	0.028
Herbs	14 (7)	0.059	0.071	0.084
Leaf vegetables	216 (190)	0.016	0.038	0.060
Legume vegetables	8 (5)	0.005	0.009	0.013
Legumes, beans, dried	20 (1)	0.030	0.031	0.032
Miscellaneous fruits	24 (7)	0.022	0.022	0.023
Molluscs	42 (2)	0.21	0.21	0.21
Oilseeds	20 (0)	0.033	0.033	0.033
Pome fruits	58 (33)	0.006	0.020	0.034
Root vegetable	34 (29)	0.018	0.039	0.06
Spices	10 (4)	0.096	0.11	0.12
Stem vegetables	11 (7)	0.009	0.022	0.036
mTDS[m]				
Biscuits	2 (0)	0	0.025	0.05
Bread	7 (0)	0	0.025	0.05
Breakfast cereals (Brinta/Bambix)	1 (0)	0	0.025	0.05
Breakfast cereals (cornflakes)	1 (1)	0.179	0.179	0.179
Macaroni/spaghetti/noodles	2 (0)	0	0.025	0.05
Muesli, crunchy	1 (0)	0	0.025	0.05
Muesli, regular	1 (1)	0.054	0.054	0.054
Rice	2 (0)	0	0.025	0.05
Rye products	1 (0)	0	0.025	0.05

[a] LB (lower bound): samples with a lead concentration below limit of detection (LOD) or quantification (LOQ) (non-detect samples) were assumed to contain no lead; MB (medium bound): non-detect samples were assigned a lead concentration equal to ½LOD or ½LOQ; UB (upper bound): non-detect samples were assigned a lead concentration equal to the relevant limit value

[b] Already calculated mean LB, MB and UB concentrations obtained from EFSA (2012b).

[c] Includes lead concentration data from the KAP and BioKAP databases (see section 2.2).

[d] Mean concentrations as used in the exposure assessment after fitting a NonDetectSpike-LogNormal distribution to the positive concentrations per food (group), including the relevant imputed values for the non-detect samples. For foods or food groups with no or only one positive sample, the available concentrations (including the imputed values) were averaged (see section 2.4).

[e] In the main text, also referred to as raw agricultural commodities (RACs)

[f] Derived from lead concentrations in kidney of bovine animals according to the proportion meat:kidney = 1:8 (section 2.2)

[g] Derived from lead concentrations in liver of poultry according to the proportion meat:liver = 1:1.5 (section 2.2).

[h] In the exposure calculations, lead concentrations analysed in meat of wild deer were not considered due to very high analysed concentrations (up to 810 mg/kg) making this food group the main contributor of the exposure in the LB scenario. These concentrations were

also much higher than those reported by EFSA (2012b) for venison meat: 0.048 mg/kg (MB)

[i] Obtained from the Centre for Sustainability, Environment and Health (RIVM; 2012-2015)

[j] Derived from lead concentrations in kidney of pig according to the proportion meat:liver:kidney = 1:4;8 (section 2.2). LB, MB and UB concentrations in kidney of pig were 0.0037, 0.025 and 0.050 mg/kg, respectively. These concentrations are not reported in the table, because they were not, as such, used in the exposure assessment: no consumption of kidney of pig, as such or as ingredient, is reported in the food consumption surveys.

[k] Derived from lead concentrations in kidney of sheep according to the proportion meat:kidney = 1:8 (section 2.2). LB, MB and UB concentrations in kidney of sheep were 0.061, 0.073 and 0.085 mg/kg, respectively. These concentrations are not reported in the table, because they were not used in the exposure assessment: no consumption of kidney of sheep, as such or as ingredient, is reported in the food consumption surveys.

[l] Monitoring data were grouped according to the FoodEx1 classification system as used in EFSA (2012b)

[m] mTDS: mycotoxin-dedicated total diet study. See Appendix B for more details

Appendix D Modelling of long-term exposure using LNN

LNN models exposure frequencies and exposure amounts separately, followed by an integration step (Goedhart et al., 2012). For the consumption frequencies, LNN fits a logistic regression model to the number of days with consumption per individual, providing both an estimate of the mean consumption frequency and of the variation between individuals in this frequency (dispersion factor). For the modelling of the positive amounts, LNN first transforms the positive daily exposure distribution into a more normal distribution using a logarithmic or power function. Then, a normal-distribution based variance components model is fitted to remove the within-person's variation. The resulting between-person normal distribution is then back-transformed and combined with the exposure frequency distribution to estimate the long-term dietary exposure distribution. This is achieved by sampling a large number of times from both the exposure frequency distribution and the back-transformed positive exposure distribution (Monte Carlo integration). In this report, a logarithmic transformation for the positive daily exposure distribution was used. The correlation between intake frequency and amount was assumed zero.

Appendix E Description of the bootstrap

There are different sources of uncertainty in dietary exposure assessments. One of these sources is the uncertainty due to the limited size of the dataset. The smaller the dataset, the more uncertain the data are. This uncertainty can be quantified by using the bootstrap method (Efron, 1979; Efron and Tibshirani, 1993).

With this method, a bootstrap database is generated of the same size as the original database for both the food consumption and concentration database by sampling with replacement from the original datasets. These bootstrap databases are considered as databases that could have been obtained from the original population if another sample was randomly drawn. These two bootstrap databases are then used for the exposure calculations and derivation of the relevant percentiles. Repeating this process many times results in a bootstrap distribution for each percentile that allows for the derivation of confidence intervals around it. The bootstrap approach was used in this report by generating 100 food consumption and 100 concentration bootstrap databases and calculating the chronic (with at least 10,000 iterations each) dietary exposure. Of the resulting bootstrap distributions per percentile a 95% uncertainty interval was calculated by computing the 2.5% and 97.5% points of the empirical distribution.

Note that by bootstrapping both the consumption and concentration database in one analysis it is not possible to quantify which part of the uncertainty was due to a limited number of consumption or concentration data.

Appendix F Median (P50) and high (P95) exposure estimates (µg/kg bw per day) to lead per age stratum in children aged 2 to 6, persons aged 7 to 69 and 18 to 69, and women of childbearing age in the Netherlands following three scenarios of substitution of samples with lead concentrations below limit of detection (LOD) or quantification (LOQ)

Age (years)	Percentiles of exposure per scenario (µg/kg bw per day)					
	LB[a]		MB[b]		UB[c]	
	P50	P95	P50	P95	P50	P95
Children aged 2 to 6						
2	0.50 [0.43-0.65]	0.78 [0.69-1.1]	1.0 [0.99-1.2]	1.5 [1.4-1.8]	1.6 [1.5-1.8]	2.2 [2.1-2.5]
3	0.46 [0.39-0.58]	0.73 [0.62-0.99]	0.96 [0.88-1.1]	1.3 [1.2-1.6]	1.5 [1.3-1.6]	2.0 [1.8-2.2]
4	0.43 [0.38-0.54]	0.67 [0.61-0.89]	0.88 [0.82-0.99]	1.2 [1.2-1.4]	1.3 [1.3-1.4]	1.9 [1.7-2.0]
5	0.39 [0.36-0.48]	0.62 [0.57-0.82]	0.81 [0.76-0.93]	1.1 [1.1-1.3]	1.2 [1.2-1.3]	1.7 [1.6-1.8]
6	0.37 [0.32-0.43]	0.57 [0.51-0.74]	0.74 [0.69-0.83]	1.0 [0.98-1.2]	1.1 [1.1-1.2]	1.6 [1.5-1.7]
2-6	0.43 [0.38-0.53]	0.70 [0.62-0.94]	0.88 [0.83-0.99]	1.3 [1.2-1.5]	1.3 [1.3-1.4]	2.0 [1.9-2.2]
Persons aged 7 to 69						
7	0.39 [0.36-0.43]	0.73 [0.67-0.81]	0.76 [0.73-0.82]	1.3 [1.2-1.4]	1.1 [1.1-1.2]	1.9 [1.8-1.9]
10	0.30 [0.29-0.33]	0.57 [0.53-0.62]	0.59 [0.57-0.63]	0.98 [0.94-1.1]	0.87 [0.85-0.91]	1.4 [1.4-1.5]
12	0.27 [0.25-0.29]	0.50 [0.47-0.55]	0.51 [0.50-0.55]	0.85 [0.82-0.93]	0.76 [0.74-0.79]	1.2 [1.2-1.3]
16	0.23 [0.21-0.25]	0.43 [0.40-0.47]	0.43 [0.41-0.45]	0.72 [0.68-0.78]	0.63 [0.61-0.65]	1.0 [0.99-1.1]
18	0.22 [0.20-0.24]	0.41 [0.38-0.45]	0.41 [0.39-0.43]	0.68 [0.64-0.74]	0.59 [0.57-0.62]	0.97 [0.93-1.0]
20	0.21 [0.20-0.23]	0.40 [0.37-0.43]	0.39 [0.37-0.41]	0.65 [0.62-0.71]	0.57 [0.55-0.59]	0.93 [0.89-0.97]
30	0.22 [0.20-0.24]	0.41 [0.38-0.46]	0.39 [0.37-0.41]	0.64 [0.61-0.70]	0.55 [0.53-0.57]	0.90 [0.86-0.94]
40	0.24 [0.22-0.26]	0.44 [0.41-0.50]	0.41 [0.39-0.44]	0.68 [0.65-0.75]	0.58 [0.56-0.61]	0.94 [0.91-1.0]
50	0.24 [0.22-0.26]	0.45 [0.41-0.49]	0.41 [0.39-0.44]	0.68 [0.64-0.76]	0.58 [0.55-0.61]	0.94 [0.90-0.99]
60	0.23 [0.21-0.25]	0.43 [0.40-0.47]	0.39 [0.37-0.42]	0.65 [0.61-0.72]	0.55 [0.53-0.58]	0.89 [0.86-0.94]
69	0.25 [0.22-0.28]	0.46 [0.42-0.53]	0.41 [0.39-0.46]	0.69 [0.64-0.79]	0.58 [0.54-0.63]	0.95 [0.87-1.0]

Age (years)	Percentiles of exposure per scenario (µg/kg bw per day)					
	LB[a]		MB[b]		UB[c]	
	P50	P95	P50	P95	P50	P95
7-69	0.24 [0.22-0.26]	0.46 [0.43-0.51	0.41 [0.40-0.44]	0.74 [0.71-0.82]	0.59 [0.57-0.61]	1.1 [1.0-1.1]
18-69	0.24 [0.22-0.26]	0.46 [0.43-0.5]	0.41 [0.39-0.45]	0.70 [0.66-0.80]	0.58 [0.56-0.60]	0.97 [0.93-1.0]
Women of childbearing age[d]						
20	0.25 [0.20-0.28]	0.53 [0.42-0.61]	0.41 [0.37-0.46]	0.76 [0.68-0.85]	0.58 [0.52-0.66]	1.0 [0.92-1.2]
30	0.25 [0.23-0.28]	0.53 [0.48-0.60]	0.41 [0.39-0.44]	0.76 [0.70-0.82]	0.58 [0.54-0.62]	1.0 [0.95-1.1]
40	0.25 [0.18-0.29]	0.53 [0.38-0.63]	0.41 [0.31-0.45]	0.76 [0.57-0.83]	0.58 [0.45-0.63]	1.0 [0.79-1.1]
20-40	0.25 [0.23-0.27]	0.53 [0.49-0.63]	0.41 [0.39-0.43]	0.76 [0.71-0.80]	0.58 [0.55-0.61]	1.0 [0.97-1.1]

Note: 2.5% lower – 97.5% upper confidence limits of the percentiles of exposure are reported between brackets.

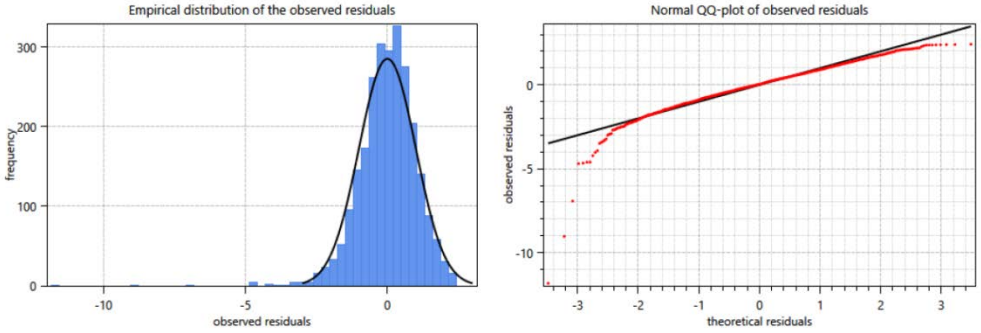
[a] LB (lower bound): samples with a lead concentration below the limit of detection (LOD) or quantification (LOQ) (non-detect samples) were assumed to contain no lead.

[b] MB (medium bound): non-detect samples were assigned a lead concentration equal to half the relevant limit value.

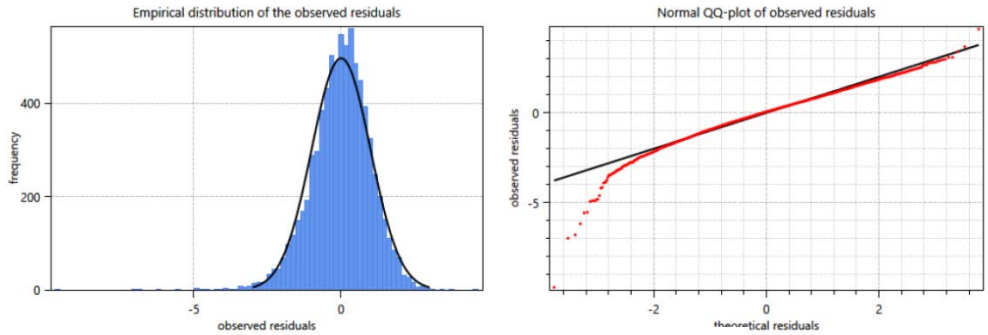
[c] UB (upper bound): non-detect samples were assigned a lead concentration equal to the relevant limit value.

[d] Women of childbearing age covered food consumption data of women aged 20 to 40.

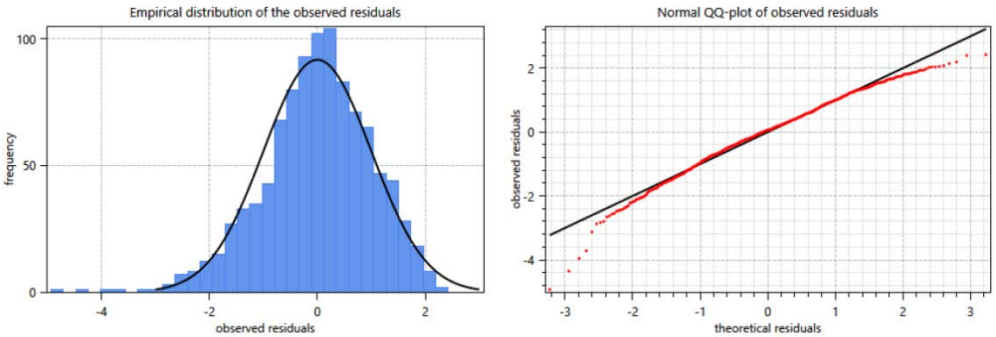
Appendix G Observed vs. theoretical residuals of the positive daily exposure distributions to lead in children aged 2 to 6, persons aged 7 to 69 and women of childbearing age in the Netherlands in which lead concentrations below limit of detection (LOD) or quantification (LOQ) equalled ½LOD and ½LOQ (medium bound scenario)



Children aged 2 to 6



Persons aged 7 to 69



Women of childbearing age

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