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# Report on a Total Diet Study carried out by the Food Safety Authority of Ireland in the period 2012 – 2014

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# REPORT ON A TOTAL DIET STUDY CARRIED OUT BY THE FOOD SAFETY AUTHORITY OF IRELAND IN THE PERIOD 2012 – 2014

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## SUMMARY

As part of its statutory responsibility to ensure the safety of food consumed, distributed, produced and sold on the Irish market, the Food Safety Authority of Ireland (FSAI) periodically carries out Total Diet Studies (TDS) to measure the dietary exposure of the population to particular chemicals that may pose a risk to health if taken into the body in excessive amounts. In carrying out a TDS, the most commonly consumed foods in Ireland, based on food consumption data, are analysed for particular chemical contaminants, food additives and nutrients present in the food. Dietary exposure to each chemical is then estimated using the food consumption data and the level of the particular chemical present in each food. This report presents the findings of the most recent TDS carried out in Ireland.

The food consumption data used in the FSAI TDS were derived from the National Adult Nutrition Survey (NANS), which investigated habitual food and beverage consumption in a representative sample (n=1,500) of adults aged 18 years and over in the Republic of Ireland during 2008 - 2010 and the National Children's Food Survey (NCFS) which investigated habitual food and drink consumption in 594 children, aged 5 - 12 years, from the Republic of Ireland during 2003 - 2004.

The chemicals analysed were the contaminant metals aluminium, arsenic (total and inorganic), cadmium, chromium, lead, mercury and tin, the essential nutrients iodine and selenium, the food additives nitrates and nitrites, the food contaminants acrylamide, mycotoxins, polycyclic aromatic hydrocarbons (PAH), pesticide residues and bisphenol A and phthalates, which can be found in some food contact materials (FCMs). Fluoride was originally also included in the TDS, however, the need for a more detailed study was identified, and a separate project is currently underway to assess population exposure to fluoride and will be published as separate study in 2016.

The exposure estimates obtained in the study were compared with health-based guidance values established by international risk assessment bodies such as the European Food Safety Authority (EFSA), the Joint Food and Agriculture Organization (FAO)/World Health Organization (WHO) Expert Committee on Food Additives (JECFA) and the former European Union Scientific Committee on Food (SCF), in order to characterise the risk to Irish consumers from the presence of chemicals in the food they eat. These health-based guidance values include the Acceptable Daily Intake (ADI), for chemicals such as food additives and pesticides, and the Tolerable Daily (or Weekly) Intake (TDI/TWI) for contaminants found in food.

The ADI/TDI (TWI) is the amount of a food additive/potentially harmful contaminant that can be consumed on a daily (weekly) basis over a lifetime without appreciable risk to health. For substances, which are both genotoxic and carginogenic, or for substances where the toxicological database was insufficient to set a tolerable intake, the Margin of Exposure (MoE) approach was applied in the risk characterisation. Nutrients were compared against the Recommended Daily Allowance (RDA), Adequate Intake (AI) and/or the Tolerable Upper Limit (UL) to evaluate potential deficiency or excessive intake.

The following paragraphs provide a summary of the main findings for each substance tested.

Exposure to aluminium was found to be below the EFSA TWI of 1 mg/kg bw for both adults and children. Major contributors to dietary exposure were non-alcoholic beverages, i.e. tea at 40%, and cereals (mainly bread and fine bakery ware) (33%) for adults and cereals (mainly bread and fine bakery ware and breakfast cereals) (54%) for children, respectively.

Exposure to inorganic arsenic in both the adult and child populations was found to be below the range of values for the 95% lower confidence limit of the benchmark dose of 1% extra risk ($BMDL_{01}$ 0.3 to 8 µg/kg bw per day) for cancers of the lung, skin and bladder as well as skin lesions. Major contributors to dietary exposure for both adults and children were cereals (81 and 94%, respectively). In an effort to lower overall exposure to inorganic arsenic at EU population level, the European Commission (EC) has recently introduced maximum limits for rice and rice products in tandem with a monitoring recommendation for other important dietary contributors with a view to potentially introduce further maximum limits.

Exposure to cadmium was found to be below the EFSA TWI of 2.5 µg/kg bw for both the average adult and child populations, however, slight exceedances were observed at the 97.5$^{th}$ percentile in both population groups. Exposure estimates for adults were found to be appreciably lower than those estimated in the previous TDS, most likely due to a change in dietary behaviour. Based on the findings of a biomarker study undertaken on urine samples collected from subjects partaking in the most recent adult food consumption survey, and which reflect long term chronic exposure to cadmium, the levels of cadmium in the Irish diet do not present an unacceptable risk to the consumer.

Exposure to chromium was found to be well below the EFSA TDI of 300 µg/kg bw for both adults and children and is considered not to be of concern. Major dietary contributors to exposure were found to be meat (26%) and vegetables (31%) for adults and meat (16%), vegetables (26%), cereals (17%) and fruit juices (16%).

Exposure to lead was compared against a range of lower 95% confidence limits of benchmark Bench Mark Doses (BMDLs) relating to developmental neurotoxicity, effects on systolic blood pressure and effects on prevalence of chronic kidney disease. Estimates of dietary exposure to lead in both population groups were lower than the BMDLs of relevance and the calculated MoEs indicate that risks from lead in foods are likely to be low. Major dietary contributors to lead exposure in adults were alcoholic beverages (28%), cereals (22%) and vegetables (12%), whereas for children it was found to be cereals (37%), non-alcoholic beverages (19%) and vegetables (12%). Tap water tested in this survey which was taken from individual households across the country, did not contain detectable levels of lead. However, the past use of lead as a material for water pipes in many older houses may result in unacceptably high levels in water supplies. In 2015, Irish Water confirmed that there are still properties with lead piping in Ireland, and occupants of such premises might therefore, be exposed to additional lead from their water supply (Irish Water, 2015). In 2013, the Health Service Executive (HSE) and the Environmental Protection Agency (EPA) issued a joint position paper summarising the issues in relation to lead in drinking water including health, legislation and interventions (EPA/HSE, 2013). There is now a *National Strategy to Reduce Exposure to Lead in Drinking Water* published by the Department of Health and Department of the Environment, Community and Local Government which is used by the EPA to track progress. Irish Water has also published a National Implementation Plan to reduce levels of lead in drinking water. EPA enforcement activities have also resulted in all lead distribution mains being removed; however, lead remains in 5 - 10% of lead communication or service pipes. Data for 2014 (EPA, 2015) indicated that there was 98.7% compliance with the new limit of 10 µg/L for lead in drinking water. This covered 1,337 water supply zones and 3,010 samples in total.

Exposure to mercury was found to be well below the EFSA Provisional Tolerable Weekly Intake (PTWI) for both adults and children. For both the adult and child populations, intake of white fish (52% and 59% respectively) and canned fish (29% and 36% respectively) were found to be the major contributors to dietary exposure. Fish, particularly predatory fish (such as shark, marlin, swordfish and fresh tuna) are recognised to be the major source of exposure to mercury in the diet, and since fish consumption in Ireland is below the EU average, Irish consumers are unlikely to be at risk from this source. Nevertheless, the FSAI provides advice on fish consumption for children, pregnant women and women of reproductive age with regard to mercury exposure.

Exposure to tin was estimated to be low in both population groups, i.e. less than 4% of PTWI of 14 mg/kg bw established by JECFA, and is not considered to be of concern.

Iodine and selenium are both essential nutrients in the body. Results of this TDS indicate that generally, the Irish population is neither likely to be deficient in either iodine or selenium, nor at risk from the toxic effects of excess iodine and/or selenium in their diet.

Exposure to acrylamide was compared against a range of BMDLs relating to non-neoplastic and neoplastic effects, and although human studies have not demonstrated acrylamide to be a human carcinogen, the calculated MoEs indicate a concern with respect to neoplastic effects. The same observations were made by EFSA in its most recent risk assessment (EFSA, 2015). In this study, for adults, MoE values for neoplastic effects ranged from 452 – 1,030 for the mean exposure and from 166 - 333 for the 97.5$^{th}$ percentile exposure. For children, MoE values ranged from 238 - 298 at the mean and 119 - 136 at the 97.5$^{th}$ percentile. Major contributors to dietary exposure were found to be cereals (49%), snacks (28%) and vegetables (23%) for adults and cereals (47%), snacks (38%) and vegetables (15%) for children. Given the toxicity of acrylamide, EFSA in tandem with other international bodies has concluded that efforts should be made to reduce acrylamide concentrations in food, and more rigorous risk management measures are likely to be implemented by the European Commission in 2016.

Exposure to nitrate (from both use as additive and natural occurrence) was below the ADI of 3.7 mg/kg bw in both population groups, with natural occurrence in vegetables being the more important contributor to dietary exposure. Nitrite was only detected in one foodgroup (hams) and exposure estimates based on this finding as well as estimates taking into account other foodgroups in which nitrite was tested but not detected, were also below the TDI of 0.06 mg/kg bw.

Exposure to mycotoxins was estimated for aflatoxins, ochratoxin A, patulin and fusarium toxins. Fusarium toxins were not detected in any of the samples tested. However, more sensitive methodologies are required for future analysis of fusarium toxins as part of a TDS in order to fully characterise the potential risk from exposure to these toxins. Exposure estimates for ochroatoxin A and patulin were below established health-based guidance values for both population groups and are not of concern. No health-based guidance value has been established for aflatoxins, and risk characterisation is based on the MoE concept, which is considered appropriate for substances which are both genotoxic and carcinogenic. For both the adult and child populations, calculated MoEs were found to be low, which is in line with the findings by EFSA and indicate a potential concern. Regarding aflatoxins and ochratoxin A, cereal-based products were the major source of exposure for both adults and children (>80%) whereas for patulin, the major contributors were alcoholic beverages and non-alcoholic beverages. Mycotoxins are subject to stringet controls within the EU and at international level to reduce population exposure to as low as reasonably achievable.

Exposure to polycyclic hydrocarbons (PAHs) was evaluated following the MoE concept based on the bench mark dose lower confidence limit for a 10% increase in the number of tumour bearing animals compared to control animals ($BMDL_{10}$ of 0.34 mg/kg bw). For both population groups, the calculated MoEs were sufficiently high to conclude that there is low concern for human health. For both population groups, cereals were found to be the main contributor to dietary exposure.

Exposure to BPA was estimated to be low in both population groups and was well below the temprorary TDI (t-TDI) of 4 µg/kg bw/d. The findings are in line with estimates derived by EFSA (EFSA, 2015) and indicate that exposure to BPA is of low concern. The main food groups contributing to dietary BPA exposure were non-alcoholic beverages (48%), vegetables (21%) and meat (14%) for adults, and vegetables (42%), meat (20%) and soups and sauces (19%) for children, respectively.

Exposure to phthalates was estimated to be low in both population groups and average as well as above average exposure to phthalates was found to be well below the respective TDIs set by EFSA. These results are in line with exposure estimates derived by the UK FSA in a Total Diet Study conducted in 2007 (FSA, 2007), and are of low concern.

Overall, the outcome of this analysis showed that the Irish population is generally not at risk from intakes from food of the majority of the chemicals and to a lesser degree for lead, analysed in the study. Potential concern was identified for exposure to acrylamide and aflatoxins. However, these findings are not specific to Ireland, rather they are of concern worldwide and continuous efforts are being made by risk managers to reduce exposure to these substances to as low as reasonably achievable, bearing in mind that zero exposure is impossible. While these results are not of immediate concern, the FSAI reiterates that continued surveillance of the Irish food supply for contaminants, residues, food additives and essential nutrients by food business operators and by other competent bodies including environmental health professionals and public analysts is essential, in order to ensure the continuing safety of Irish food.

Global trade in food necessitates harmonised control and risk management actions at European wide level to reduce exposure of the European population to contaminants and pesticide residues. This is realised via harmonised European Commission food contaminants and residues legislation within Europe. Ireland participates in all relevant EU Expert Working Groups and provides food consumption and occurrence data to EFSA to ensure that the safety of Irish consumers is accounted for.

## GLOSSARY

| | |
|---|---|
| Adenoma | A generic term for a benign epithelial tumour composed of glands and/or glandular elements |
| ADI | Acceptable Daily Intake |
| AI | Adequate Intake |
| AR | Average Requirement |
| BMD | The Benchmark Dose is based on a mathematical model being fitted to the experimental data within the observable range and estimates the dose that causes a low but measurable response (the benchmark response BMR) typically chosen at a 5 or 10% incidence above the control. |
| BMDL | Benchmark Dose Lower Limit (see BMD). The BMD lower limit (BMDL) refers to the corresponding lower limits of a one-sided 95% confidence interval on the BMD. Using the lower-bound takes into account the uncertainty inherent in a given study, and assures (with 95% confidence) that the chosen BMR is not exceeded. |
| BMR | Benchmark Response (see BMD) |
| bw | Bodyweight |
| Carcinogenic | Causing cancer |
| Carcinoma | A malignant new growth made up of epithelial cells tending to infiltrate surrounding tissues and to give rise to metastases |
| DAFM | Department of Agriculture, Food and the Marine |
| EC | European Community |
| EFSA | European Food Safety Authority |
| EGVM | Expert Group on Vitamins and Minerals (UK) |
| EPA | Environmental Protection Agency |
| EU | European Union |
| FAO | Food and Agriculture Organization |
| FCM | Food Contact Material |
| FSAI | Food Safety Authority of Ireland |
| Genotoxic | Damaging to DNA and capable of causing mutations or cancer |
| HSE | Health Service Executive |
| IARC | International Agency for Research into Cancer |
| JECFA | Joint WHO/FAO Expert Committee on Food Additives and Contaminants |

## GLOSSARY (continued)

| | |
|---|---|
| Kg | Kilogram |
| LB | Lowerbound (<LOD=0) |
| LOD | Limit of Detection |
| LOQ | Limit of Quantitation |
| mg | Milligram = 10-3 part of a kg (0.001 g) |
| MoE | Margin of Exposure (MoEs are calculated by dividing the BMDL values derived from dose-response data for the different endpoints by the estimates of dietary exposure) |
| MI | Marine Institute |
| µg | Microgram = 10-6 part of a g (0.000001 g) |
| NANS | National Adult Nutrition Survey |
| Neoplastic | An abnormal new growth of tissue; a tumor |
| ng | Nanogram = $10^{-9}$ part of a g (0.000000001 g) |
| NTP | National Toxicity Programme |
| P97.5 | 97.5$^{th}$ Percentile of a Distribution |
| PAH | Polycyclic Aromatic Hydrocarbons |
| PMTDI | Provisional Maximum Tolerable Daily Intake |
| PRI | Population Reference Intake |
| PTWI | Provisional Tolerable Weekly Intake |
| RDA | Recommended Daily Allowance |
| SCF | Scientific Committee for Food |
| SCOOP | Scientific Cooperation Task (EC) |
| SFPA | Sea-Fisheries Protection Authority |
| SML | Specific migration limits (defined as the maximum permitted amount of a given substance that can be released from a material or article into food or food simulant) |
| TDI | Tolerable Daily Intake |
| t-TDI | Temporary Tolerable Daily Intake |
| TDS | Total Diet Study |
| TWI | Tolerable Weekly Intake |
| UB | Upperbound (<LOD=LOD) |
| UL | Tolerable Upper Intake Level |
| WHO | World Health Organization |

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## 1. INTRODUCTION

The Food Safety Authority of Ireland (FSAI) has a statutory responsibility to ensure the safety of food consumed, distributed, produced and sold on the Irish market. In this respect, the FSAI co-ordinates the collation of food safety surveillance information from laboratories operated by its official agents, the HSE, the Department of Agriculture, Food and the Marine (DAFM), the Sea-Fisheries Protection Authority (SFPA), the Marine Institute (MI) and the local authorities. The FSAI also conducts targeted food safety surveillance in areas where potential food safety issues have been identified and/or on food contaminants for which there are currently no testing facilities in Ireland.

A Total Diet Study (TDS) is considered to be a good complement to existing food monitoring or surveillance programmes to estimate population dietary exposure to beneficial and harmful chemical substances across the entire diet. The World Health Organization (WHO), the lead United Nations agency for health, supports the undertaking of TDSs as one of the most cost-effective means for assuring that people are not exposed to unsafe levels of toxic chemicals through food (WHO, 2005a). At the beginning of 2010, EFSA formed a working group of experts on TDS aiming at reviewing the state of the art on TDSs worldwide with a particular emphasis on activities in Europe and developed a guidance document for a harmonised approach of TDS in collaboration with the WHO (EFSA/WHO/FAO, 2011).

A comparison of the actual dietary intake of chemicals present in food, estimated via a TDS, with their corresponding health-based guidance values, such as the ADI, TDI or TWI gives a realistic estimate of exposure for risk assessment purposes. TDS results can be indicators of contamination of food from the environment and can be used to assess the effectiveness of specific risk management measures to control the levels of such chemicals in food. A TDS may also be used to provide an estimate of intake of food additives present in the diet, such as sweeteners or preservatives, or of intake of key nutrients such as vitamins. The results of a TDS can be used as a priority-setting tool to enable risk managers to focus their limited resources on those chemicals which are considered to pose the greatest risks to public health.

The overall aim of a TDS is to provide a snapshot in time of exposure of a given population to contaminants or other food chemicals of interest. It aims to estimate exposure of the general population and does not represent unusual exposure scenarios, e.g. above average exposure to chemicals due to environmental contamination in specific geographic regions.

In carrying out a TDS at national level, the most commonly consumed foods in the country are selected, based on food consumption data, and the respective predominant food preparation methods are determined. The selected foods representative of the normal diet consumed by the population over a given period of time are collected from defined surveillance regions and consist of a number of sub-samples per sample (for example, a bread sample could contain five different sub-samples of the particular type of bread being analysed). The samples are prepared following the most commonly used kitchen preparation practice and are subsequently analysed as they would be consumed for the chemicals of interest. The chemical occurrence data are then combined with consumption data to calculate the exposure estimates of the population to the chemicals from the selected foods. A TDS explicitly takes into account the kitchen preparation of foods to assess the levels of contaminants in foods as consumed, as these may change during preparation and cooking.

This report provides the results of a TDS undertaken by the FSAI during the period of 2012 - 2014, and is the second such study carried out in Ireland.

## 2. METHODOLOGY

Chemical exposure estimates were calculated by combining food consumption data with chemical occurrence data for representative foods as prepared for consumption, e.g. cooked, grilled, baked, etc.

Planning and co-ordination of this project as well as sampling of the foods of interest was undertaken by FSAI staff; food preparation and analysis of the samples was undertaken under contract by the Food and Environment Research Agency (FERA) in the UK.

The general approach for selection of foods and food preparation methods was developed as part of the previous TDS (FSAI, 2011).

### 2.1. Food Consumption Data

The food consumption data used in this TDS were derived from NANS (IUNA, 2011) and the NCFS (IUNA, 2006). These surveys investigated habitual food and beverage consumption in a representative sample (n=1,500) of adults aged 18 years and over in the Republic of Ireland during 2008 - 2010 and in 594 children, aged 5 - 12 years from the Republic of Ireland during 2003 - 2004. The extensive electronic databases which have been compiled from these surveys have been used to obtain information on food consumption and food preparation habits.

## 2.2. List of Analysed Food Contaminants, Additives and Nutrients

The majority of contaminants included in this TDS were analytes selected in the previous TDS (FSAI, 2011). The list was extended with additional analytes of interest/concern to the FSAI (see Table 1).

Fluoride was originally also included in the TDS, however, the need for a more detailed study was identified, and a separate project is currently underway to assess population exposure to fluoride and will be published as separate study in 2016.

Details on methods of analysis and analytical sensitivity are detailed in Annex I; details on analytes determined by food are listed in Annex II, a summary of analytical results are listed in Annex III.

### Table 1. List of food additives, contaminants, food contact materials and nutrients analysed in the FSAI TDS

| | | |
|---|---|---|
| Aluminium | Mercury | Phthalates |
| Arsenic | Selenium | Pesticides multi-screen (see Annex I) |
| Inorganic Arsenic | Tin | Aflatoxins |
| Cadmium | Acrylamide | Fumonisins |
| Chromium | Nitrates | Ochratoxin A |
| Iodine | Nitrites | Patulin |
| Lead | PAHs | Trichothecenes |
| | Bisphenol A (BPA) | Zearalenone |

## 2.3. Selection of Foods for Analysis

The choice of foods for this TDS was based on the list as determined in the previous TDS (FSAI, 2011) and additionally informed based on information available from more recent food consumption surveys, in particular brand information available in the most recent adult food consumption survey. The complete food list is shown in Table 2.

## 2.4. Shopping List/Food Sampling

For each foodstuff, a number of sub-samples (typically five) were purchased. The selection of brands was based on interrogation of the brand information in the food consumption databases. The quantity of each foodstuff purchased was dependent on the contaminants to be analysed for and the minimum sample size required for the various types of analysis to be performed on that foodstuff. This latter information was obtained from the laboratory contracted to carry out the food preparation and analysis.

Sampling of the foods was conducted by the FSAI in autumn of 2012 and a total of 141 samples (comprising 1,043 sub-samples) were sent for preparation and analysis. Food was mainly purchased in the major retailers located in Dublin. Tap water was sourced from a variety of private households attached to the public water supply.

## 2.5. Food Preparation

Where required, foods were prepared ready for consumption by the laboratory before analysis. Information on most commonly used kitchen preparation practices was obtained as part of the analysis of the food consumption database for the food list. A detailed description of the required method of food preparation was provided to the laboratory.

## Table 2. List of foods for sampling and analysis in the FSAI TDS

| Foodgroup | Foods within Foodgroup |
|---|---|
| **CEREALS** | White Flour, Wholemeal Flour, White Bread/Rolls, Granary/Wholegrain Breads, Brown Bread and Rolls, Plain Biscuits, Chocolate Biscuits, Other Biscuits, Cakes, Other Cakes, Buns and Pastries, Pasta, Rice, Cornflakes, Bran Flakes, Wheat-type Cereals, Muesli, Oat Flakes, Rice-type Cereals |
| **DAIRY** | Whole Milk, Low-fat, Skimmed & Fortified Milks, Cream, Cheese (hard), Cheese (continental style), Cheese (soft and Semi-soft), Yogurts, Custard, Vanilla Ice-cream, Butter, Dairy Spreads, Non-dairy Spreads, Other Ice-creams, Other Milk |
| **EGGS** | Eggs (fried) |
| **MEAT** | Pork, Ham, Pork Sausage, Bacon Rashers, Beef, Beef Mince, Beef Burger, Chicken, Turkey, Lamb, Offal (kidney), Offal (liver), Pudding (black and white) |
| **FISH** | Cod and other White Fish, Oily Fish other than Salmon, Salmon, Canned Tuna, Tinned Fish (excl. salmon & tuna), Tinned Salmon, Smoked Salmon, Smoked Fish (excl. salmon), Mussels, Prawns, Crab |
| **POTATOES** | Potatoes without Skin (boiled), Potatoes with Skin (microwaved), Chips (homemade, from frozen pre-prepared) |
| **VEGETABLES** | Onion (fried), Tomatoes, Canned Tomatoes, Tomato Canned/Concentrate, Peppers, Cucumber, Mushrooms, Canned Sweet Corn, Carrots (boiled), Carrots, Celery, Peas, Canned Peas, Green Beans, Baked Beans, Legumes (excl. peas), Canned Legumes (excl. Peas), Cabbage (raw), Cabbage (boiled), Broccoli, Cauliflower, Root Vegetables (excl. carrots), Stir-fry Vegetables, Lettuce, Other Leafy Vegetables |
| **FRUIT** | Apples, Oranges, Bananas, Grapes, Pears, Peaches and Nectarines, Canned Peaches, Plums, Berries, Other Fruit, Canned Fruit (excl. peaches) |
| **FRUIT DRIED** | Dried Raisins, Dried Fruit (excl. raisins) |
| **NUTS SEEDS** | Nuts, Seeds |
| **HERBS SPICES** | Herbs, Spices |
| **SOUPS** | Stock Cubes, Beef & Vegetable Concentrates, Soup (tetrapak), Soups (canned), Soups (dried packet) |
| **SAUCES** | Tomato Sauce, Mayonnaise, Gravy, Cook-in Sauces (other), Cook-in Sauces Tomato-based, Other Sauces and Condiments, Soy Sauce |
| **SUGAR AND PRESERVES** | Sugar & Sugar Substitutes, Marmalade, Jam, Honey |
| **CONFECTIONERY** | Chocolate Confectionery, Non-chocolate Confectionery |
| **BEVERAGES** | Lager, Stout, White/Red Wine, Spirits, Alcoholic Drinks (apple-based), Carbonated Soft Drinks, Squashes, Apple Juice, Orange Juice, Other Fruit Juices, Tea, Instant Coffee, Filter Coffee, Herbal Tea, Bottled Water, Tap Water |
| **FATS OILS** | Olive Oil, Vegetable Oil, Fat, Hard Cooking Fat |
| **SNACKS** | Crisps, Other Savoury Snacks |
| **COMPOSITE** | Pizza |

## 2.6. Exposure Assessment

The NANS and NCFS databases are structured using the McCance and Widdowson Food Code System (Holland *et al.* 1988, 1989, 1991a, 1991b, 1992a, 1992b, 1993; Chan *et al.* 1994, 1995, 1996; McCance and Widdowson, 1991, 2002) and classify foods based on this system.

To enable exposure assessment to the various chemicals in the food, the food consumption databases were restructured and recoded, thereby splitting composite foods, e.g. dishes, into ingredients and regrouping them accordingly.

The databases were re-categorised into 141 food groups in accordance with the food list. The relevant food groups were then matched with the associated chemical occurrence levels determined in the foods contained in the food list. Occurrence data for those food groups not on the food list were extrapolated from comparable foods for which data were available, e.g. the concentrations obtained in prawns were used to estimate substance exposure from comparable shellfish, such as lobster and shrimps, for which no contaminant occurrence data were available.

Furthermore, as not every single food on the food list was likely to contain every single contaminant covered by this survey, not all contaminants were routinely included in the analysis. The selection of contaminants to be analysed in each food was dependent on the type of food and the type of contaminant, e.g. particular mycotoxins are not expected to occur in meat products but rather in cereals, nuts and dried fruit products, whereas heavy metals on the other hand occur in almost all foodstuffs. An overview of analytes covered per foodgroup is listed in Annex II.

The recoded food consumption data and chemical occurrence data were combined using the probabilistic web-based Creme software (Creme Food). The Creme software allows estimation of exposure to food additives, pesticide residues, nutrients, food packaging migratory compounds, intentionally added flavouring substances and novel foods including genetically modified foods, and delivers probabilistic estimates of dietary exposure to hazards. For the purpose of this survey, a semi-probabilistic approach was used, i.e. the single aggregate-sample-based occurrence levels of contaminants, nutrients or additives were combined with population food intake distribution data.

Results are expressed as lower-bound (LB) and upper-bound (UB) values (see Section 2.7 for a description of how these were treated mathematically). Both UB and LB values were expressed as average intake and above average intake (97.5$^{th}$ percentile (P97.5) exposure) of the particular contaminant, additive or nutrient, together with average intake per kg bodyweight (kg bw) and above average intake per kg bw for the total population to reflect average and above average consumers.

Per kg bw calculations take into account the distribution of weights of individuals within the survey population and are included for direct comparison with health based guidance values such as the ADI, TDI, TWI or other reference value, which are expressed on a kg bw basis.

## 2.7. Mathematical Treatment of UB and LB Analytical Results

For LB calculations/estimates as presented in this report, analytical results below the limit of detection (<LOD) were set at zero (<LOD=0), whereas for UB calculations, analytical results recorded as below the LOD were assumed to be present at the limit of detection (<LOD=LOD). In either model, values above the LOD were taken as the actual level of the analyte in the food. UB values therefore reflect the worst-case (highest) exposure scenario and LB values the best-case (lowest) exposure scenario. The true level of estimated exposure, obtained by combining either the UB or LB estimate of the level of analyte in the food with data on consumption of that particular food by the population, will be between the two values.

# 3. RESULTS AND DISCUSSION

An overall summary of the analytical results for each chemical (contaminant, additive or nutrient) analysed in this survey is provided in Annex III. Exposure and potential health effects are discussed in the following pages.

For each chemical, both LB and UB average mean and 97.5$^{th}$ percentile exposure estimates for the Irish adult and child populations, as derived from the TDS, are provided. Results are also provided adjusted for individual body weights of all survey participants and expressed on a per kg/bw basis. The derived exposure estimates are then compared to existing health-based guidance values.

## 3.1. Aluminium

### 3.1.1. Sources of exposure to aluminium

Aluminium occurs naturally in the environment, and is the most abundant metallic element in the earth's crust (WHO, 1997; EFSA, 2008a). Some sources of aluminium exposure are:

- Geological sources
- Mining and industrial uses in the production of aluminium metal and other aluminium compounds
- Aluminium-containing food additives and release to food from aluminium-containing food contact materials

Aluminium is a ubiquitous component of many foods, e.g. fruit, vegetables, cereals, seeds and meat. According to EFSA (2008a), the major route of exposure to aluminium for the general population is through food, and an additional minor exposure may arise from aluminium in drinking water. At neutral pH, aluminium concentration in drinking water ranges from 1 to 50 µg/litre, but in more acidic water this can rise to 500 – 1,000 µg/litre (WHO, 1997). As reported by EFSA (2008a), most unprocessed foods typically contain less than 5 mg aluminium/kg, but higher concentrations can be found in breads, cakes and pastries, some vegetables, glacé fruits, dairy products, sausages, offal, shellfish, farinaceous products and flours. Foods with high mean concentrations include tea leaves, herbs, cocoa and cocoa products, and spices (EFSA, 2008a).

### 3.1.2. Health effects of aluminium

**The TWI established by EFSA for ingested aluminium from all sources is 1 mg/kg bw (EFSA, 2008a).**

Aluminium is of lower toxicity than many other metals. It is very poorly taken up from the gastrointestinal tract following ingestion. Like other metals however, once absorbed into the body, it is persistent, with the whole-body, half-life of aluminium in humans being estimated to be 50 years (JECFA, 2007; EFSA, 2008a). The main health effect of aluminium is neurotoxicity, which has been demonstrated in experimental animal studies and also in patients undergoing dialysis, who are chronically exposed to aluminium via dialysis water. It has also been suggested that aluminium may be a causative factor in Alzheimer's disease and may be associated with other neurodegenerative diseases in humans, although these hypotheses are controversial and remain unproven (EFSA, 2008a). The developing nervous system appears to be particularly at risk, as demonstrated in a number of developmental neurotoxicity studies in animals, and there is also evidence of effects on the reproductive system (developmental toxicity and effects on fertility) (JECFA, 2007; EFSA, 2008a). The EFSA TWI for aluminium from all sources including food additives was in agreement with the 2007 provisional tolerable weekly intake (PTWI) of JECFA (JECFA, 2007). However, in 2011 JECFA increased its PTWI for aluminium to 2 mg/kg bw (JECFA, 2011).

### 3.1.3. Dietary exposure to aluminium

Table 3 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile aluminium exposure of the Irish adult and child populations from all food groups.

## Table 3. Estimated aluminium exposure of the Irish adult and child populations from all food groups, expressed as mg/d, mg/kg bw and % of the EFSA TWI

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake mg | | | | Daily Intake mg/kg bw (weekly intake in parenthesis) | | | | % of EFSA TWI (1 mg/kg bw/week) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 3.73 | 3.78 | 8.54 | 8.60 | 0.05 (0.35) | 0.05 (0.35) | 0.12 (0.83) | 0.12 (0.84) | 35% | 35% | 83% | 84% |
| **CHILDREN** | | | | | | | | | | | |
| Daily Intake mg | | | | Daily Intake mg/kg bw (weekly intake in parenthesis) | | | | % of EFSA TWI (1 mg/kg bw/week) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 1.60 | 1.63 | 3.20 | 3.23 | 0.05 (0.36) | 0.05 (0.37) | 0.11 (0.74) | 0.11 (0.75) | 36% | 37% | 74% | 75% |

As can be seen from Table 3, for adults the average mean intake of aluminium from food was estimated at 0.05 mg/kg bw/day, which is equivalent to 0.35 mg/kg bw/week.The above average (97.5$^{th}$ percentile) intake was estimated at 0.12 mg/kg bw/day. The estimated intakes for aluminium determined in this TDS compare well with estimates of the previous TDS (FSAI, 2011).

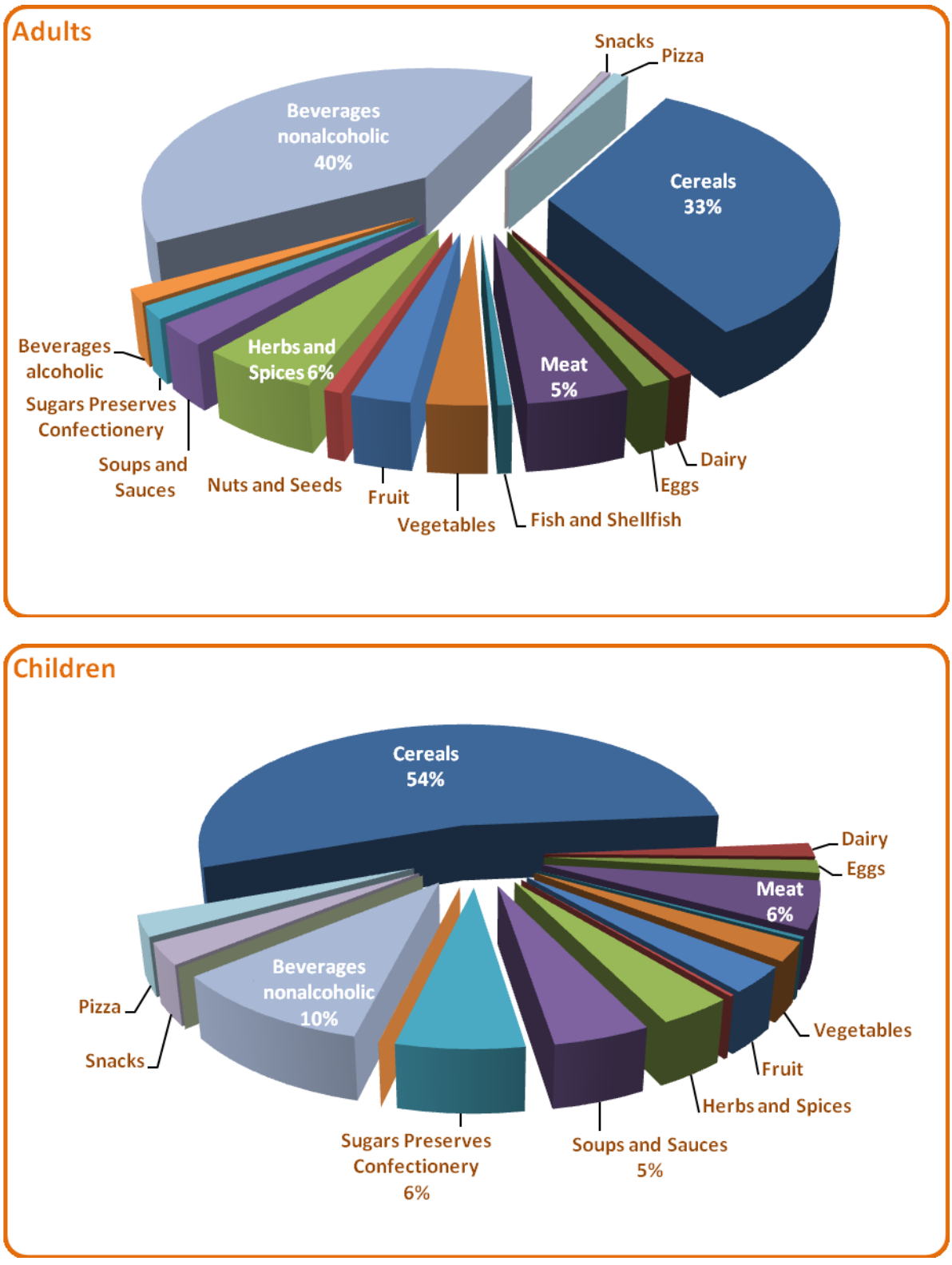
The results for daily intake are also within the range of 1.6 to 13 mg/day estimated by EFSA based on TDSs (EFSA, 2008).

For children, average intake of aluminium from food was estimated at 0.05 mg/kg bw/day, which is equivalent to 0.37 mg/kg bw/week. The above average (97.5$^{th}$ percentile) intake was estimated at 0.11 mg/kg bw/day, equivalent to 0.75 mg/kg bw.

Aluminium was detected in 84% of all samples analysed.

Figure 1 shows the main contributing food groups to dietary aluminium exposure, based on LB measurements, revealing that non-alcoholic beverages (40%) and cereals (33%) were the major contributors to dietary intake for adults, and for children also cereals (54%) and non-alcoholic beverages (10%) were found to be the major contributors.

**Figure 1. Contribution of the various food groups in which aluminium was detected (LB) as a percentage of total aluminium intake in adults and children**



### 3.1.4. Risk characterisation

As can be seen from Table 3, for adults the average mean intake of aluminium from food corresponds to 35% of the EFSA TWI, and above average (97.5$^{th}$ percentile) intake to 84% of the EFSA TW, respectively.

For children, the average intake of aluminium from food corresponds to 37% of the EFSA TWI and above average (97.5$^{th}$ percentile) intake to 75% of the EFSA TWI, respectively.

In establishing a TWI for aluminium, EFSA estimated that the daily dietary exposure to aluminium in the general population, assessed in several European countries, varied from 0.2 to 1.5 mg/kg bw/week at the mean and was up to 2.3 mg/kg bw/week in highly exposed consumers (EFSA, 2008a). The intakes for the population resident in Ireland (both adults and children) lie towards the lower end of this range for both the mean and the 97.5$^{th}$ percentile, and do not exceed the EFSA TWI. Therefore, exposure to aluminium from food in Ireland is of no health concern.

## 3.2. Arsenic

### 3.2.1. Sources of exposure to arsenic

Arsenic is a naturally occurring element, present in soil, ground water and plants. Arsenic is a metalloid element, displaying different valences (-3, 0, +3, +5), resulting in a broad variety of inorganic and organic arsenic compounds with diverse chemical characteristics. Inorganic and organic forms of arsenic differ significantly in their toxicity, the organic arsenic compounds exhibiting a low toxic potential. Data on arsenic occurrence in food show that fish and seafood account for over 90% of total exposure to arsenic in food. As the carry-over of arsenic in its inorganic form into edible tissue of mammals and poultry is low, food from this source contributes in only a limited way to human exposure (EFSA, 2005a). Data from the EC Scientific Cooperation Task Force (SCOOP) report on exposure to metals in the diet and EFSA show that with the exception of seafood and animal offal, the concentration of arsenic is generally less than 250 µg/kg (EC, 2004; EFSA, 2009b). However, the majority of this arsenic appears to be in the form of the less toxic organic arsenic species, e.g. in shellfish, molluscs and seaweeds the predominant species are arsenosugars (dimethylarsinyl riboside derivatives), while in fish and crustaceans, the predominant arsenic compound is arsenobetaine, a form of arsenic which is considered to be virtually non-toxic.

EFSA in 2009 concluded that "*inorganic arsenic exposures from food and water across 19 European countries, using lower-bound (LB) and upper-bound (UB) concentrations, have been estimated to range from 0.13 to 0.56 µg/kg bw per day for average consumers, and from 0.37 to 1.22 µg/kg bw per day for 95$^{th}$ percentile consumers*" (EFSA, 2009b). In 2014, EFSA re-assessed dietary exposure to inorganic

arsenic based on more precise occurrence data, which led to considerably lower exposure estimates compared to those in the 2009 EFSA opinion (EFSA, 2014). The highest dietary exposure to inorganic arsenic was estimated in the younger population. The mean dietary exposure among infants, toddlers and other children ranged, across the different Member States and surveys, from 0.20 to 0.45 µg/kg bw per day (min - max LB) and from 0.47 to 1.37 µg/kg bw per day (min - max UB), with the maximum value estimated in infants. In the same three age classes, the 95$^{th}$ percentile dietary exposure estimates ranged from 0.36 to 1.04 µg/kg bw per day (min - max LB) and from 0.81 to 2.09 µg/kg bw per day (min - max UB), with the highest level estimated in toddlers. The mean dietary exposure to inorganic arsenic among all surveys in the adult population (including adults, elderly and very elderly) ranged from 0.09 to 0.38 µg/kg bw per day (min LB - max UB) for the mean dietary exposure, and from 0.14 to 0.64 µg/kg bw per day (min LB - max UB) for the 95$^{th}$ dietary exposure.

### 3.2.2. Health effects of arsenic

**EFSA has not established a PTWI for inorganic arsenic, but recommends that a range of Benchmark Doses (lower confidence limit) ($BMDL_{01}$) of 0.3 to 8 µg/kg bw per day should be used in any risk characterisation for inorganic arsenic. No TDI/TWIs have been established for organic arsenic compounds.**

Inorganic arsenic is significantly more toxic than organic arsenic compounds such as dimethylarsinate, and in turn the trivalent forms of arsenic, e.g. arsenic trichloride, are more toxic than the pentavalent arsenates. The latter are considered to be toxic only after metabolic conversion to the trivalent form of arsenic. This pattern of toxicity is also seen for certain other metallic compounds in the body, e.g. chromium compounds. Exposure to inorganic arsenic is primarily of concern because of its cancer-causing properties. However, arsenic is also more acutely toxic than other metallic compounds and was used in earlier times as a rodenticide, while continual low level exposure to arsenic is associated with cancer, skin lesions, developmental toxicity, neurotoxicity, cardiovascular diseases, abnormal glucose metabolism, and diabetes (EFSA, 2009b). There is evidence of negative impacts on foetal and infant development, particularly reduced birth weights (EFSA, 2009b). Arsenic and inorganic arsenic compounds have been classified by the International Agency for Research into Cancer (IARC) as a human carcinogen (Group 1) on the basis of increased incidence of cancers at several sites, particularly skin, in people exposed to arsenic at work, in the environment or through their diet (IARC, 1987a, IARC 2012a).

JECFA noted that organic forms of arsenic present in seafood needed different consideration from the inorganic arsenic in water. Based on the low toxicity and rapid metabolism of organoarsenicals, and taking into account the nutritious value of fish despite the presence of organoarsenicals, JECFA considered that there was no recommendation to restrict the consumption of fish. In 2009, the EFSA CONTAM Panel noted that since the PTWI of 15 µg/kg bw was established by JECFA, new data had established that inorganic arsenic causes cancer of the lung and urinary tract in addition to skin, and that a range of adverse effects had been reported at exposures lower than those reviewed by JECFA. The Panel further noted that inorganic arsenic is not directly DNA-reactive and there are a number of proposed mechanisms of carcinogenicity, for each of which a thresholded mechanism could be postulated. However, taking into account the uncertainty with respect to the shape of the dose-response relationships, it was not considered appropriate to identify from the human data a dose of inorganic arsenic with no appreciable health risk, i.e. a tolerable daily or weekly intake. Therefore, the MoE should be assessed between the identified reference points from the human data and the estimated dietary exposure to inorganic arsenic in the EU population (EFSA, 2009b). The Panel modelled the dose-response data from key epidemiological studies and selected a benchmark response of 1% extra risk for cancers of the lung, skin and bladder, as well as skin lesions and concluded that the overall range of $BMDL_{01}$[1] values of 0.3 to 8 µg/kg bw per day should be used instead of a single reference point in any risk characterisation for inorganic arsenic. In 2011, JECFA withdrew the TWI of 15 µg/kg bw and established $BMDL_{0.5}$ values of 3 µg/kg bw/day (lung cancer); 5.2 µg/kg bw/day (bladder cancer); 5.4 µg/kg bw per day (skin lesions) (WHO, 2011).

### 3.2.3. Dietary exposure to arsenic

Tables 4 and 5 present the estimated LB and UB daily mean and 97.5$^{th}$ percentile arsenic and inorganic arsenic exposure of the Irish adult and child populations from all food groups. The LB intakes of arsenic are considered to be more representative of exposure of the population resident in Ireland, as the UB estimates, which are considerably higher, reflect the assumption that in food where the arsenic levels were <LOD, arsenic was present at the LOD (see Section 3.2.1 on arsenic occurrence in food).

[1] The BMD approach estimates the dose that causes a low but measurable target organ effect, e.g. a 5% reduction in body or organ weight or a 10% increase in the incidence of kidney toxicity.

As can be seen from Table 4, for adults average intake of total arsenic was estimated to fall between 0.7 - 0.9 µg/kg bw/day and for above average consumers between 3.9 - 4.2 µg/kg bw/day. The average intake of inorganic arsenic was much lower (see Table 5), falling between 0.01 - 0.02 µg/kg bw/day and for above average consumers between 0.06 - 0.08 µg/kg bw/day.

For children (see Table 4) average intake of total arsenic was estimated to fall between 0.6 - 0.9 µg/kg bw/day and for above average consumers between 2.9 - 3.3 µg/kg bw/day. The average intake of inorganic arsenic (see Table 5) was much lower, falling between 0.03 - 0.05 µg/kg bw/day and for above average consumers, between 0.13 - 0.14 µg/kg bw/day.

### Table 4. Estimated daily total arsenic intake of the Irish adult and child populations

| ADULTS | | | | | | | |
|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 56 | 71 | 294 | 310 | 0.7 | 0.9 | 3.9 | 4.2 |
| **CHILDREN** | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 17.9 | 27.3 | 88.5 | 97.5 | 0.6 | 0.9 | 2.9 | 3.3 |

### Table 5. Estimated daily inorganic arsenic intake of the Irish adult and child populations

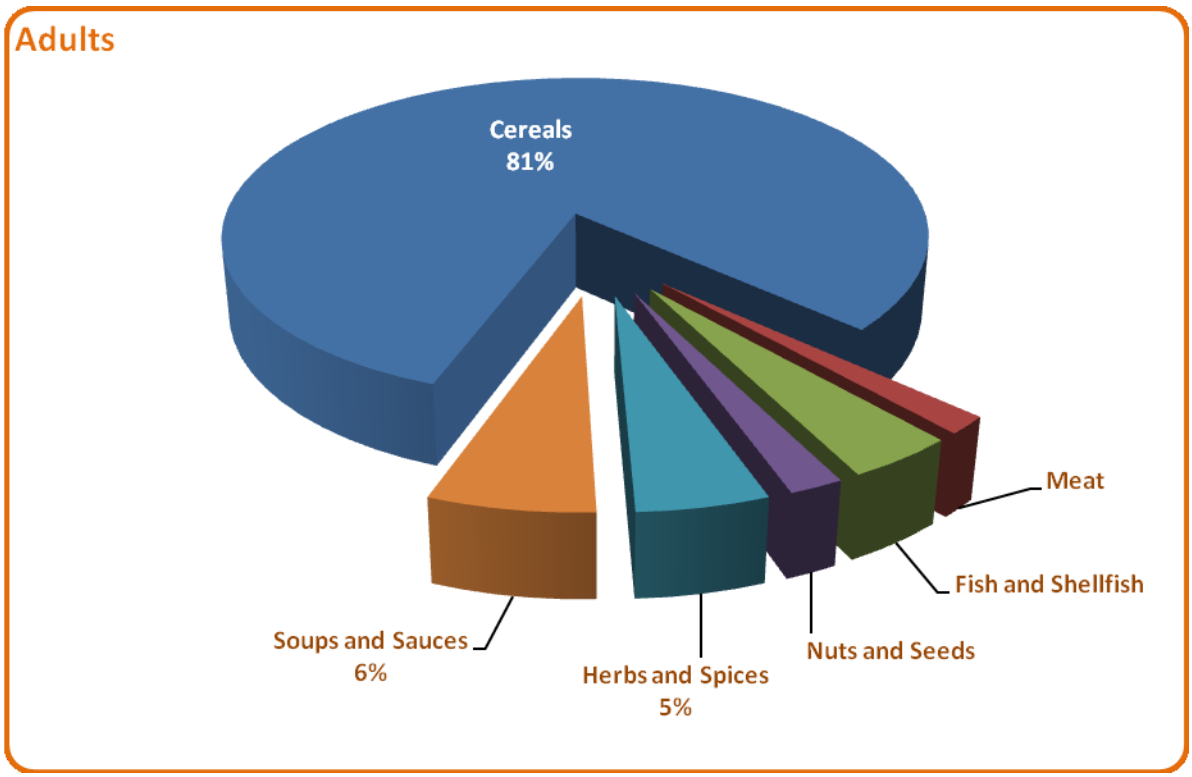
| ADULTS | | | | | | | |
|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 1.03 | 1.7 | 4.3 | 5.7 | 0.01 | 0.02 | 0.06 | 0.08 |
| **CHILDREN** | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 0.98 | 1.48 | 3.81 | 4.14 | 0.03 | 0.05 | 0.13 | 0.14 |

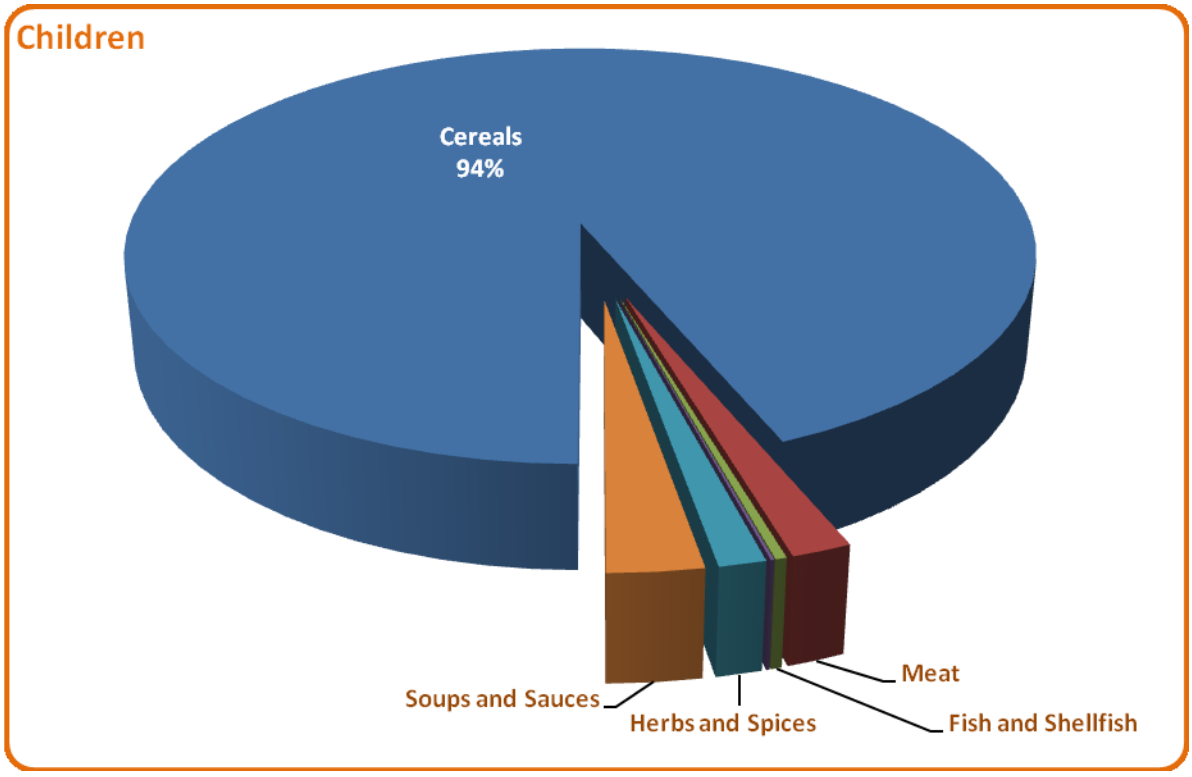
Arsenic was detected above the LOD in 17% of all analysed foods. Those foodstuffs which contained detectable levels of arsenic were further analysed for inorganic arsenic and of these, eight samples (33%) contained levels of inorganic arsenic above the LOD.

Fish and seafood were the main contributors to total arsenic intake in both adults and children, contributing 95% and 89% to overall intake, respectively. However, it should be noted that the arsenic in fish and seafood is primarily in the organic form.

Figure 2 shows that the contribution from fish and seafood to total inorganic arsenic exposure represented only a small proportion (4% in adults and 0.3% in children). The main contributor to inorganic arsenic (81% in adults and 94% in children) was found to be cereals (see Figure 5), and arsenic is known to particularly occur as inorganic arsenic in rice (Laparra *et al.*, 2005).

Figure 2. Contribution of the various food groups in which inorganic arsenic was detected (LB) as a percentage of total inorganic arsenic intake in adults and children



An updated exposure assessment was undertaken by EFSA in 2014 (see Table 6), which resulted in considerably lower dietary arsenic exposure estimates than in the original opinion (EFSA, 2009b). The observed reduction in exposure was due to the use of more refined consumption data and better matching of and increased use of actual concentration data for inorganic arsenic (EFSA, 2014).

**Table 6. Summary statistics of the dietary chronic exposure assessment (µg/kg bw/day) to inorganic arsenic across European dietary surveys reported by EFSA (2014)**

| EU population group | Mean µg/kg bw/day | | 95th percentile µg/kg bw/day | |
|---|---|---|---|---|
| Infants, toddlers and children | 0.20 - 0.45 (min - max LB) | 0.47 - 1.37 (min - max UB) | 0.36 - 1.04 (min - max LB) | 0.81 - 2.09 (min - max UB) |
| Adults, elderly/very elderly | 0.09 to 0.38 (min LB - max UB) | | 0.14 to 0.64 (min LB - max UB) | |

As can be seen from Table 5, estimates for inorganic arsenic intake by adults and children derived in the most recent TDS indicate considerably lower values (adult mean: 0.01 - 0.02 µg/kg bw/day; adult P97.5: 0.06 - 0.08 µg/kg bw/day; children: mean 0.03 - 0.05 µg/kg bw/day; children P97.5: 0.13 - 0.14 µg/kg bw/day) than those calculated by EFSA (see Table 6). The observed difference is most likely due to results for the TDS being derived from actual measured values for inorganic arsenic in the food samples, whereas EFSA, due to lack of occurrence data for inorganic arsenic for all food groups, extrapolated most of the values from total arsenic, assuming a conservative proportion of (generally) 70% inorganic arsenic to the total arsenic measured in food.

### 3.2.4. Risk characterisation

EFSA (EFSA, 2009b), in its most up-to-date risk assessment, has recommended that a range of $BMDLs_{01}$ of 0.3 to 8 µg/kg bw/day should be used in any risk characterisation for inorganic arsenic.

Table 7 provides MoEs for the lower and upper end of the range of $BMDLs_{01}$ calculated by EFSA, derived for both average and above average intake estimates of inorganic arsenic in the Irish adult and child populations.

## Table 7. MoEs for inorganic arsenic derived for Irish adults and children based on BMDLs of 0.3 to 8 µg/kg bw/day as set by EFSA (EFSA, 2009)

| Estimated intake of inorganic arsenic (µg/kg bw/day LB - UB) | | MoE based on $BMDL_{01}$ of 0.3 µg/kg bw/day | MoE based on $BMDL_{01}$ of 8 µg/kg bw/day |
|---|---|---|---|
| Adults Mean | 0.01 - 0.02 | 30 - 15 | 800 - 400 |
| Adults 97.5$^{th}$ percentile | 0.06 - 0.08 | 5 - 4 | 133 - 100 |
| Children Mean | 0.03 - 0.05 | 10 - 6 | 267 - 160 |
| Children 97.5$^{th}$ percentile | 0.13 - 0.14 | 2 - 2 | 62 - 57 |

Estimated dietary intake for both adults and children is below EFSA's benchmark doses with MoEs ranging from 30 - 6 for average consumers (adults and children) and 5 - 2 for above average consumers when measured against the lower end of the $BMDL_{01}$ range of 0.3 µg/kg bw/day.

These MoEs indicate that the exposure to inorganic arsenic in adults and children resident in Ireland is at least two fold lower than the lower limit of the 95$^{th}$ percent confidence interval benchmark dose of 0.3 µg/kg bw/day, associated with a 1% increased risk of developing lung cancer. However, the calculated MoEs between the estimated exposure and BMDLs are low and a further effort to decrease exposure to the population is warranted.

In 2009, EFSA conluded that the estimated dietary exposures to inorganic arsenic for average and high level consumers in Europe are within the range of the $BMDL_{01}$ values identified by the CONTAM Panel and therefore, there is little or no MoE and the possibility of a risk to some consumers cannot be excluded. The CONTAM Panel therefore recommended that dietary exposure to inorganic arsenic should be reduced and further data to produce speciation data for different food commodities to support dietary exposure assessment and dose-response data for the possible health effects, should be collected.

To mitigate the risks of exposure to arsenic, the Commission introduced maximum legislative limits for inorganic arsenic in rice and rice-based products in tandem with a monitoring recommendation covering a wider range of foods to examine the need for further management actions.

## 3.3. Cadmium

### 3.3.1. Sources of exposure to cadmium

Cadmium is a contaminant which may enter the food chain from a number of natural and industrial sources. Cadmium is present at low levels in most foods, found in commodities such as cereals, fruit, vegetables, the largest contributors to dietary exposure being (EFSA, 2009a):

- Meat and fish
- Offal (kidney and liver)
- Mussels, oysters and scallops
- Certain wild mushrooms
- Rice grown in geological areas where the soil is rich in cadmium
- Cereals and cereal products, vegetables, nuts and pulses, starchy roots or potatoes, and meat and meat products

The kidney of food animals is a major source of cadmium although lower levels are found in many foods and due to relative higher consumption of some of these foodstuffs, the latter might be higher contributors to dietary cadmium exposure.

### 3.3.2. Health effects of cadmium

**EFSA has established a TWI for cadmium of 2.5 µg/kg bw. JECFA has established a PTMI of 25 µg/kg bw, approximately 2.5 times higher than the level adopted by EFSA.**

Cadmium is relatively poorly absorbed into the body, but once absorbed is slowly excreted like other metals, having a half-life of 10 - 20 years. Most of the body burden of cadmium is retained in the liver and the kidneys, and the principal toxic effect of cadmium is its toxicity to the kidney, although it has also been associated with lung damage and development of lung tumours and skeletal changes in occupationally exposed populations. Cadmium has been categorised as a Class 1 human carcinogen (IARC, 2012). In March 2009, EFSA published a scientific opinion on possible health risks related to the presence of cadmium in food (EFSA, 2009a). The EFSA CONTAM Panel identified damage to the kidney as the key health effect on which to base its assessment. The Panel concluded that the mean intake for adults across Europe is close to, or slightly exceeding, the TWI of 2.5 µg/kg body weight (bw), based on the available data on cadmium levels in food. They noted that subgroups such as vegetarians, children, smokers and people living in highly contaminated areas may exceed the TWI by about two fold. JECFA concluded that exposure to cadmium through the diet for all age groups, including consumers with high exposure and subgroups with special dietary habits, e.g. vegetarians, would be below the PTMI of 25 µg/kg bw established by that Committee (JECFA, 2001, 2010).

### 3.3.3. Dietary exposure to cadmium

Table 8 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile cadmium exposure of the Irish adult and child populations from all food groups.

**Table 8. Estimated cadmium exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw (weekly intake in parenthesis) | | | | % of EFSA TWI (2.5 µg/kg bw/week) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 11.75 | 16.55 | 22.44 | 29.12 | 0.16 (1.1) | 0.22 (1.5) | 0.33 (2.3) | 0.42 (3) | 44% | 62% | 92% | 118% |
| **CHILDREN** | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw (weekly intake in parenthesis) | | | | % of EFSA TWI (2.5 µg/kg bw/week) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 7.31 | 10.02 | 14.65 | 17.85 | 0.24 (1.7) | 0.32 (2.3) | 0.47 (3.3) | 0.59 (4.1) | 66% | 91% | 132% | 164% |

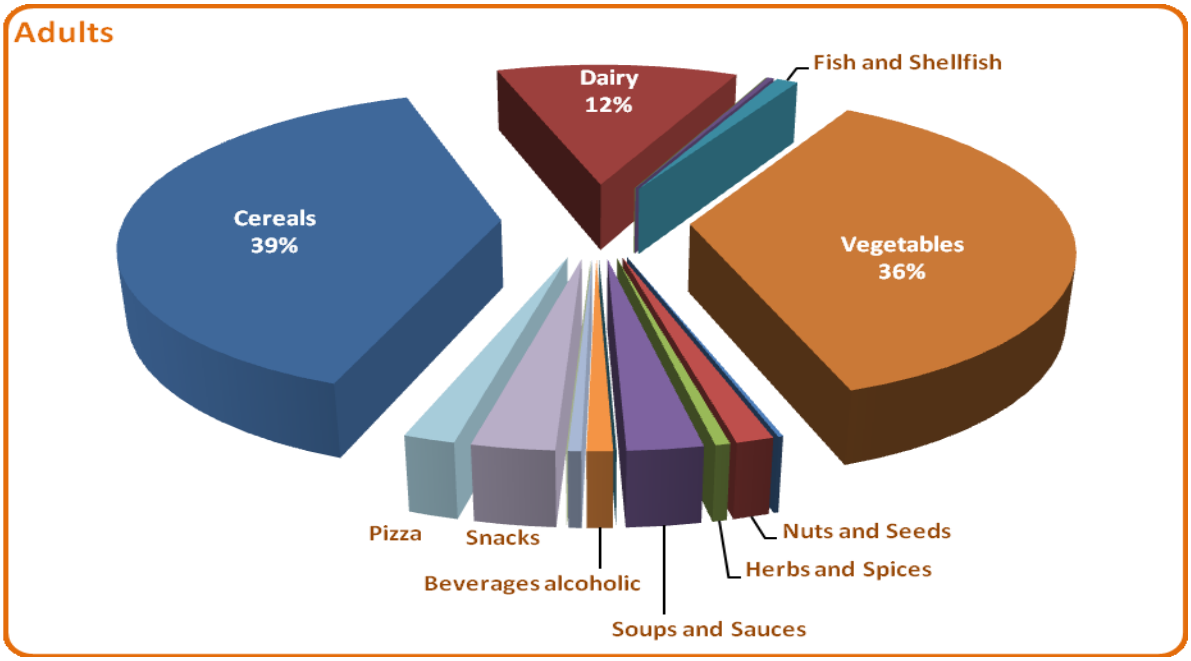
As can be seen from Table 8, for adults, average intake of cadmium was estimated to fall between 1.1 - 1.5 µg/kg bw/week. The above average (97.5$^{th}$ percentile) intake was estimated to fall between 2.3 - 3.0 µg/kg bw/week. This presents a considerable reduction compared to results obtained in the previous TDS (FSAI, 2011). The change observed is mainly due to a change in dietary patterns observed between the two studies, indicating a shift from a predominantly vegetable contribution to cadmium exposure to a more levelled contribution from both cereals and vegetables.

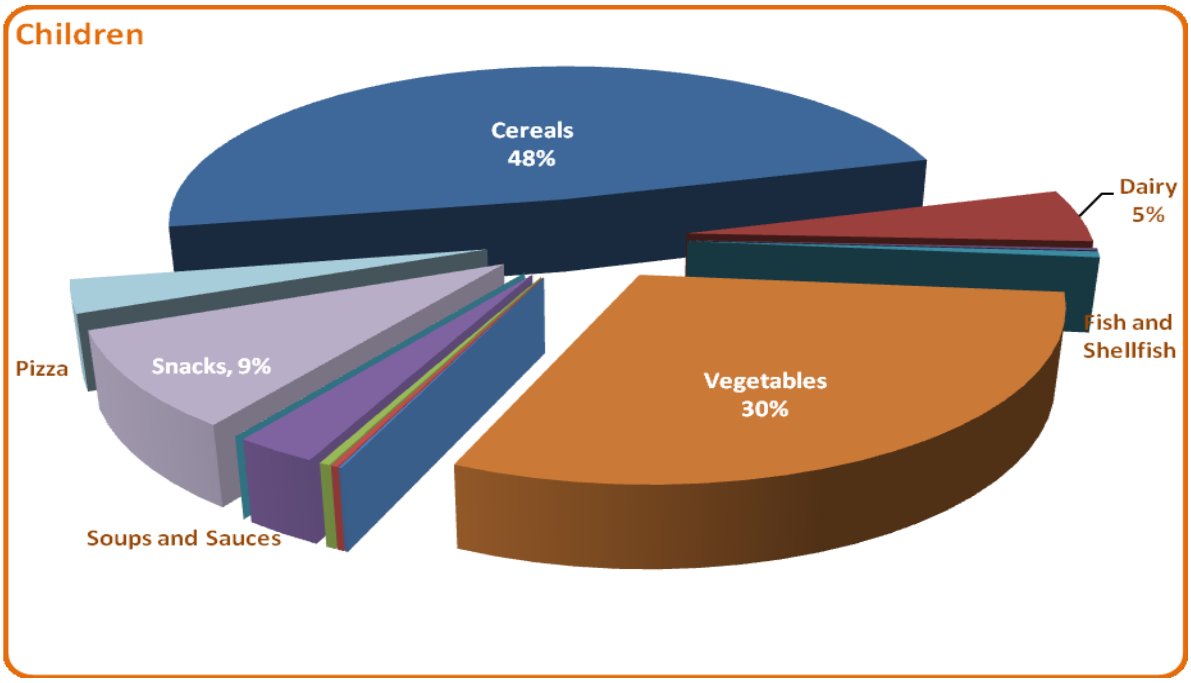
For children, average intake of cadmium was estimated to fall between 1.7 - 2.3 µg/kg bw/week. The above average (97.5$^{th}$ percentile) intake was estimated to fall between 3.3 - 4.1 µg/kg bw/week.

Cadmium was detected in 43% of all samples analysed.

Figure 3 shows that cereals (39% and 48%) and vegetables (36% and 30%) were the major contributing sources in adults and children, respectively.

Figure 3. Contribution of the various food groups in which cadmium was detected (LB) as a percentage of total cadmium intake in adults and children



### 3.3.4. Risk characterisation

Table 8 shows that exposure to cadmium was found to be below the EFSA TWI of 2.5 µg/kg bw for both the average adult and child populations, however, slight exceedances were observed at the 97.5$^{th}$ percentile (92 - 118% (LB - UB) of TWI for adults and 132 - 164% (LB - UB) TWI for children). This presents a considerable reduction compared to results obtained in the previous TDS (FSAI, 2011), which estimated an average % TWI of 95 - 123% and a 97.5$^{th}$ percentile % TWI of 216 - 244% for adults. Cereals (39%) and vegetables (36%) were found to be the major contributing sources for exposure observed in adults. This presents a shift from predominant vegetable contribution (70%) as determined in the previous TDS (FSAI, 2011). Major dietary contributors for the children population were also found to be cereals (48%) and vegetables (30%). Results from the previous TDS (FSAI, 2011) indicated that also average consumers exceeded the TWI and that vegetables, and potatoes in particular were found to be the major contributors to dietary cadmium exposure in adults (children were not included in the previous TDS). The considerable drop in exposure and shift in dietary food group contribution from predominantly vegetables to cereals and vegetables can be attributed to a change in eating pattern observable in adults. A comparison carried out on the first adult food consumption survey (IUNA, 2001) used in the previous TDS and the new NANS (2008 - 2010) used in the most recent TDS has indicated that potato consumption has decreased in the region of 50% over the last ten years. Nonetheless, since the likely source of cadmium in the potatoes is the naturally-occurring high level in the soil associated with the underlying limestone bedrock geology in parts of the country, DAFM in collaboration with Teagasc, UCD and Bord Bia (the Irish Food Board) commenced a national research project in 2013 to examine a number of parameters that may influence uptake of cadmium by potatoes, including potato variety, soil cadmium content and pH, effect of fertiliser use and of zinc application, with a view to developing strategies to mitigate cadmium uptake. The FSAI also undertook a study of urinary cadmium levels in samples from individuals who participated in the NANS published in 2011. This study examined urinary cadmium excretion in women aged >50 years, and also urinary cadmium excretion in the general population. The results of the study show that 95% of the population (including women aged over 50, who are considered to be the most 'at-risk' sub-group) are below the critical value of 1 µg Cd/g creatinine identified by EFSA in their opinion. The FSAI therefore, considers that levels of cadmium in the Irish diet do not present an unacceptable risk to consumers.

## 3.4. Chromium

### 3.4.1. Sources of exposure to chromium

Chromium is widely distributed in the environment, occurring in air, water, soil and biological materials over a large range of concentrations. Chromium compounds are classified according to their valence state, with trivalent chromium (+3 [Cr(III)]) and hexavalent chromium (+6 [Cr(VI)]) being the most common. Almost all of the sources of chromium in the earth's crust are in the elemental or trivalent state, naturally occurring chromium compounds in the hexavalent state being rare. Hexavalent chromium is derived from the industrial oxidation of mined chromium deposits and possibly from the combustion of fossil fuels, wood, paper, etc. products, and is used in industrial manufacturing processes (WHO, 1999).

While Cr(III) is a natural dietary constituent present in a variety of foods and also in dietary supplements, Cr(VI) most commonly occurs in industrial processes and is present in drinking water usually as a consequence of anthropogenic contamination (EFSA, 2014a).

EFSA's CONTAM Panel estimated mean chronic dietary exposure for Cr(III) across the different dietary surveys and age classes in 2014 (EFSA, 2014a). Overall, mean human chronic dietary exposure for the European population ranged from a minimum LB of 0.6 to a maximum UB of 5.9 µg/kg bw per day. The 95$^{th}$ percentile dietary exposure values ranged from 1.1 (minimum LB) to 9.0 (maximum UB) µg/kg bw per day. Among the different age classes, toddlers showed the highest mean chronic dietary exposure to Cr(III) with minimum LB of 2.3 and maximum UB of 5.9 µg/kg bw per day. The adult population (>18 years of age) showed lower exposure to Cr(III) than the younger populations. The mean chronic dietary exposure to Cr(III) varied between 0.6 µg/kg bw per day and 1.6 µg/kg bw per day (minimum LB and maximum UB, adults in both cases). The 95$^{th}$ percentile chronic dietary exposure ranged from 1.1 µg/kg bw per day (minimum LB) and 2.6 µg/kg bw per day (maximum UB) (EFSA, 2014a). These estimates are in agreement with assessments carried out by individual EU Member States. The TDS in the UK in 1997 reported that the mean chromium intake of adults was 100 µg/day and 170 µg/day at the 97.5$^{th}$ percentile (Ysart *et al.*, 2000), while the French TDS of 2001 indicated a mean intake of 77 µg/day for adults > 15 years, and 68 µg/day for children of 3 - 14 years, and at the 97.5$^{th}$ percentile, of 126 µg/day for adults and 124 µg/day for children (Leblanc *et al.*, 2005). In duplicate diets in Germany, Sweden and Spain, mean intakes of adults varied from 53 to 160 µg/day (SCF, 2003).

Trivalent chromium is widely accepted as an essential element (Cefalu and Hu, 2004), levels in multivitamin and mineral tablets generally providing an intake below 100 µg chromium/day. However,

commonly used trivalent chromium food supplements can provide an additional intake of 100 to 600 µg chromium/day (Cefalu and Hu, 2004). The WHO considered that supplementation of chromium should not exceed the level of 250 µg/day (WHO, 1996).

The CONTAM Panel estimated separately the exposure of the European population to Cr(VI) in all types of drinking water. The mean chronic exposure to Cr(VI) from consumption of all types of drinking water ranged from 0.7 (minimum LB) to 159.1 ng/kg bw per day (maximum UB). The 95$^{th}$ percentile exposure ranged from 2.8 (minimum LB) to 320.2 (maximum UB) ng/kg bw per day. The highest exposure to Cr(VI) through the consumption of all types of drinking water was estimated in the youngest populations (infants and toddlers).

### 3.4.2. Health effects of chromium

**No RDA or Safe Upper Intake Levels (SUL) for total chromium have been established in Ireland or the EU. EFSA has established a TDI of 0.3 mg/kg bw per day for Cr(III).**

Trivalent chromium (Cr(III)) has been postulated to be necessary for the efficacy of insulin in regulating the metabolism of carbohydrates, lipids and proteins. However, the mechanism(s) for these roles and the essential function of Cr(III) in metabolism have not been substantiated. In 2014, the EFSA Expert Panel on Dietetic Products, Nutrition and Allergies (NDA) considered the evidence for setting Dietary Reference Values for chromium, however, due to lack of sufficient evidence on the essentiality of Cr(III), the Panel concluded that no Average Requirement and no population Reference Intake for chromium could be defined. The Panel further considered that there is no evidence of beneficial effects associated with chromium intake in healthy subjects and concluded that the setting of an AI for chromium is also not appropriate (EFSA, 2014b).

The valence state of chromium is a critical factor in determining its toxicity (ASTDR, 1993; EGVM, 2002). The reported acute and chronic effects of chromium are primarily associated with the hexavalent form, acute toxicity being characterised by gastrointestinal haemorrhage, and severe liver and kidney damage and may lead to death. Chronic exposure to hexavalent chromium is reported to induce renal failure, anaemia, haemolysis and liver failure. Where follow-up was carried out symptoms were reversible and returned to normal parameters within a year (EGVM, 2002). Exposure to hexavalent chromium is a particular concern due to its well-established carcinogenic effects. After oral exposure, Cr(VI) has also been shown to be genotoxic in some in vivo studies (EFSA, 2014a). IARC has classified hexavalent chromium, on the basis of combined results of epidemiological studies and carcinogenicity studies in experimental animals, into Group 1, i.e. it is a known lung carcinogen, and trivalent chromium into Group 3, i.e. it is not classifiable as to its carcinogenicity in humans, (IARC, 1997; IARC, 2012).

As recommended for substances which are both genotoxic and carcinogenic, the CONTAM Panel adopted an MoE approach for the risk characterisation of neoplastic effects of Cr(VI). To this end, lower 95% confidence limit for a benchmark response of 10% extra risk ($BMDL_{10}$) values were derived from the two year carcinogenicity study of the National Toxicology Programme (NTP) investigating oral intake of Cr(VI) (as sodium dichromate dihydrate) via drinking water in male and female rats and mice. In a conservative approach, the CONTAM Panel selected a lowest $BMDL_{10}$ of 1.0 mg Cr(VI)/kg bw per day for combined adenomas and carcinomas of the small intestine in male and female mice as reference point for estimation of MoEs for neoplastic effects. The EFSA Scientific Committee has concluded that for substances that are both genotoxic and carcinogenic, an MoE of 10,000 or higher, based on a $BMDL_{10}$ from an animal study, is of low concern from a public health point of view.

The MoEs calculated for all age groups of the European population on the basis of the mean chronic exposure to Cr(VI) via consumption of drinking water indicated a low concern (MoE values > 10,000) for all age groups with the exception of infants at UB exposure estimates (maximum UB - minimum LB MoEs of 6,300 – 71,000) (EFSA, 2014b).

Cr(III) compounds present low oral toxicity because they are poorly absorbed. Cr(III) compounds have the potential to react with DNA in acellular systems, however restricted cellular access limits or prevents genotoxicity. The CONTAM Panel decided to use the data from the chronic toxicity studies of the NTP on chromium picolinate monohydrate to derive a health based guidance value for the risk characterisation of Cr(III). In the two year NTP chronic oral toxicity study in rats and mice, no carcinogenic or other adverse effects have been observed. The lowest no-observed-adverse-effect level (NOAEL) value derived from these studies amounted to 286 mg/kg bw per day in rats, which was the highest dose tested. The Panel derived a TDI of 300 µg Cr(III)/kg bw per day, applying a default uncertainty factor of 100 to account for species differences and human variability and an additional uncertainty factor of ten to account for the absence of adequate data on reproductive and developmental toxicity.

Under the assumption that all chromium in food is Cr(III), the CONTAM Panel noted that the mean dietary exposure levels across all age groups are well below the TDI of 300 µg Cr(III)/ kg bw per day (EFSA, 2014a).

### 3.4.3. Dietary exposure to chromium

Table 9 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile total chromium exposure of the Irish adult and child populations from all food groups.

**Table 9. Estimated chromium exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % TDI (300 µg Cr(III)/kg bw/d) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 51 | 89 | 98 | 147 | 0.7 | 1.2 | 1.4 | 2.1 | 0.2% | 0.4% | 0.5% | 0.7% |
| **CHILDREN** | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % TDI Limit (300 µg Cr(III)/kg bw/d) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 38 | 56 | 72 | 91 | 1.2 | 1.8 | 2.5 | 3.2 | 0.4% | 0.6% | 0.6% | 1.1% |

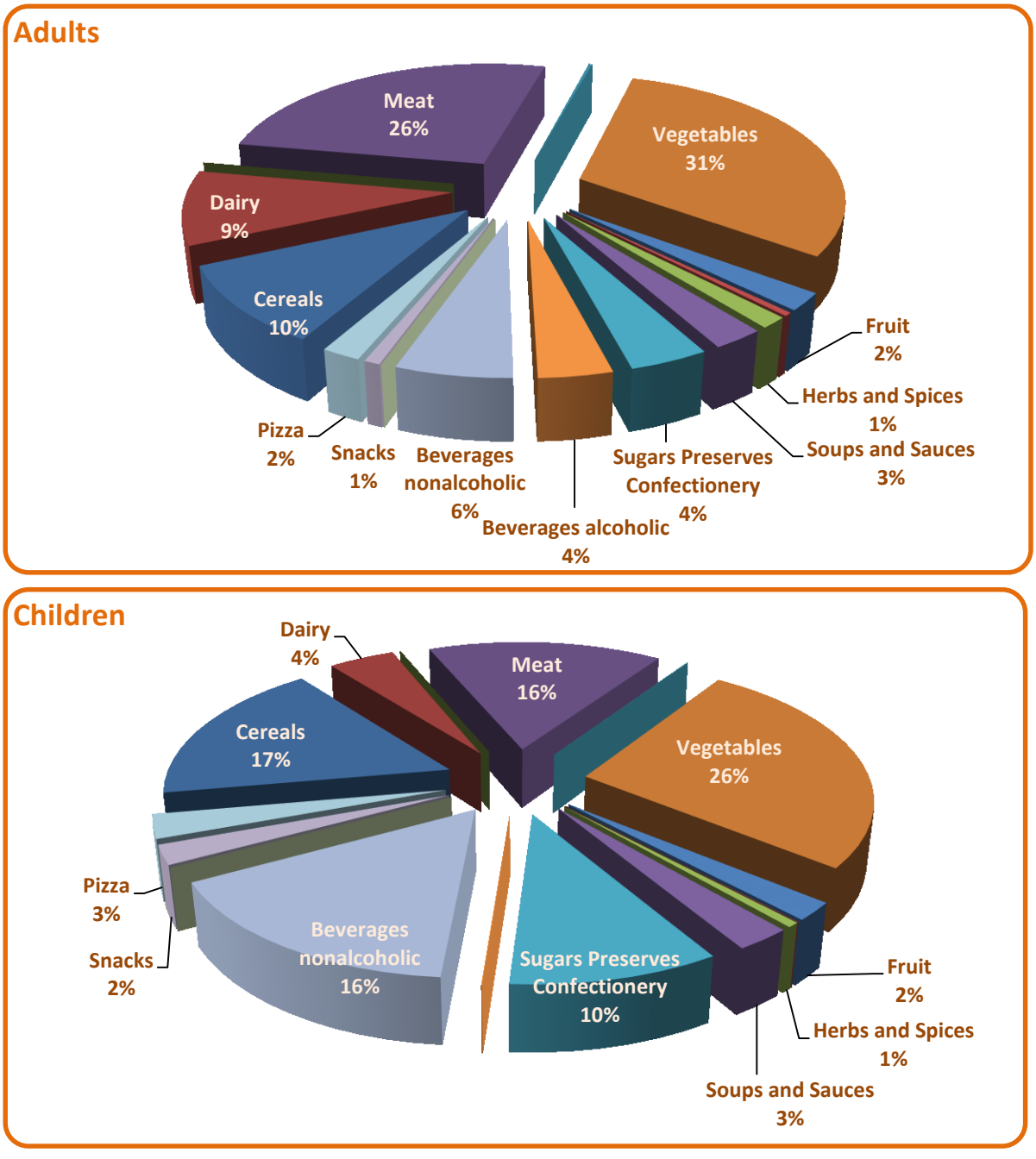
As can be seen from Table 9, for adults, the average intake of total chromium was estimated to fall between 0.7 - 1.2 µg/kg bw/day. The above average (97.5$^{th}$ percentile) intake was estimated to fall between 1.4 - 2.1 µg/kg bw/day. The intakes are similar to those reported by EFSA and the UK and French TDSs, as given in Section 3.4.1. above and are in good agreement with results reported in the previous Irish TDS (FSAI, 2011).

For children, the average intake of total chromium was estimated to fall between 1.2 - 1.8 µg/kg bw/day. The above average (97.5$^{th}$ percentile) intake was estimated to fall between 2.5 - 3.2 µg/kg bw/day. The intakes are similar to those reported by EFSA and the UK and French TDSs, as given in Section 3.1.4.1 above.

Chromium was detected in 48% of all samples analysed.

Figure 4 shows the main contributing food groups to dietary chromium intake, based on LB measurements, revealing that vegetables, meat and cereals were the main contributors to chromium intake for adults, contributing 31%, 26% and 10% of total intake, respectively. For children, vegetables, meat and cereals were the main contributors to chromium intake, contributing 26%, 16% and 17% of total intake, respectively.

Figure 4. Contribution of the various food groups in which chromium was detected (LB) as a percentage of total chromium intake in adults and children



### 3.4.4. Risk characterisation

Almost all of the sources of chromium in the earth's crust are in the elemental or trivalent state and Cr(III) therefore present the natural dietary constituent present in foods. Naturally occurring chromium compounds in the hexavalent state are rare and are present in drinking water usually as a consequence of anthropogenic contamination (EFSA, 2014a).

For the purpose of this risk characterisation, it has therefore been assumed that all chromium detected in the food samples corresponds to Cr(III).

As can be seen from Table 9, for adults, the average intake of chromium corresponds to between 0.2 and 0.4% of the TDI for Cr(III) and above average (97.5$^{th}$ percentile) intake to between 0.5 and 0.7% of the TDI, respectively. For children, the average intake of chromium corresponds to between 0.4 - 0.6% of the TDI and above average (97.5$^{th}$ percentile) intake to between 0.6 - 1.1% of the TDI, respectively.

Exposure to chromium was found to be well below the EFSA TDI of 300 µg/kg bw for both adults and children and is not of concern.

## 3.5. Iodine

### 3.5.1. Sources of exposure to iodine

Sources of elemental iodine in the environment include the following:

- The earth's crust
- Sea water (50 µg iodide/L)

The weathering of rock, volcanic activity, decay of vegetation, and human activities all contribute to the deposition of iodine in soil, with subsequent uptake into the food chain (SCF, 2002a; WHO, 2009). It is more commonly found in food or the environment as salts with other elements, such as iodides and iodates. Elemental iodine is volatile and can evaporate from seawater into the atmosphere, with subsequent deposition in rainfall on land surfaces. Metabolism of iodine/iodide by, e.g. marine algae can result in formation of organic forms of iodine such as methyl iodide and other alkyl iodides. This continuous leaching and recycling of iodine can result in soils that are low in iodide, particularly at long distances from the sea, resulting in iodine deficiency disorders. The iodides in the sea accumulate in seaweeds, seafish and shellfish. On land, small amounts of iodide are taken up by plants, the plants being subsequently ingested by herbivores. Iodine and its salts are present at levels of 3 - 50 ng/m$^3$ in the atmosphere as a result of vaporisation from seawater, unpolluted surface water contains <3 µg

iodide/L, drinking water approximately 4 - 15 µg/L. The major natural food sources are marine fish (mean 1,220 µg/kg, up to 2.5 mg/kg), shellfish (mean 798 µg/kg, up to 1.6 mg/kg), marine algae, seaweed (1,000 – 2,000 µg/kg) and sea salt (up to 1.4 mg/kg) (SCF, 2002a). Dairy products are also important sources with mean levels in UK winter milk of 210 µg/kg and 90 µg/kg in summer milk, and mean 93 µg/kg in eggs. Other food sources are cereals and cereal products (mean 47 µg/kg), freshwater fish (mean 30 µg/kg), poultry and meat (mean 50 µg/kg), fruits (mean 18 µg/kg), legumes (mean 30 µg/kg) and vegetables (mean 29 µg/kg) and use of iodised salt (SCF, 2002a; EGVM, 2002; WHO, 2009). Examples of anthropogenic sources are medicinal products, sanitising solutions and iodophores.

Total iodine intakes reported by the SCF (2002a) included the following: Germany median daily iodine intake was approximately 60 - 118 (mean 45.3) µg/day for males and females aged 4 - 75 years; in Denmark, the median intake was about 119 µg/day for males and 92 µg/day for females, while in the Netherlands, the median intake was about 145 µg/day for males and 133 µg/day for females (SCF, 2002a). In Great Britain, the median dietary intake from all sources was 226 µg/day for males and 163 µg/day for females, the 97.5$^{th}$ percentile reaching 434 µg/day in males and 359 µg/day in females. Survey data in young children aged 1½ - 4½ years show for high milk consumers in winter, 247 µg/day to 309 µg/day, approximately 16.5 – 20 µg/kg bw/day assuming a body weight of 15kg, suggesting that some pre-school children are likely to have intakes exceeding the JECFA PMTDI of 17 µg/kg bw/day (EGVM, 2002).

### 3.5.2. Health effects of iodine

**The SCF has established a UL for iodine for adults of 600 µg/day, while the RDA for iodine/iodide for Irish adults aged 18-64 excluding pregnant women is 130 µg/day. EFSA has proposed an AI of 150 µg/day for adults, AIs ranging from 70 µg/day to 130 µg/day for infants aged 7–11 months and children and an AI of 200 µg/day for pregnant women.**

Iodine is an essential dietary element for mammals, being required for the synthesis of the thyroid hormones thyroxine (T4, 3,5,3',5'-tetraiodothyronine), containing 65% by weight of iodine, and its active form T3 (3,5,3'-triiodothyronine), containing 59% by weight of iodine, as well as the precursor iodotyrosines. A number of health effects can be attributed to both deficiency and excess of iodine in the diet. An inadequate intake of iodine can lead to hypothyroidism and compensatory increase in the size of the thyroid gland (goitre), while excessive intake of iodine can be associated with both hyperthyroidism and hypothyroidism (WHO, 2009). Inflammatory reaction of the thyroid, or thyroiditis, has been described after excessive iodine intake. An increased incidence of thyroid cancer has been observed both in association with endemic hypothyroidism and with increased dietary iodine intake in endemic goitre areas (WHO, 2009). The RDA for iodine/iodide for Irish adults aged 18 - 64 excluding pregnant women is 130 µg/d, while the WHO-recommended intake (population requirement) of iodine is 150 µg/day for adults, 200 µg/day during pregnancy and lactation, and 50, 90, and 120 µg/day for children 1–12 months, 1–6 years, and 7–12 years of age, respectively (WHO/UNICEF/ICCIDD, 2001; WHO, 2004b). EFSA has proposed AIs of 150 µg/day for adults, a range of 70 µg/day to 130 µg/day for infants aged 7–11 months and children and an AI of 200 µg/day for pregnant women (EFSA, 2014c). According to the SCF (1992), the LTI is 70 µg/day, below which thyroid adaption may become inadequate with a risk of dysfunction of suboptimal operation, while the SCF has established a UL for iodine for adults of 600 µg/day, while noting that a UL is not a threshold of toxicity but may be exceeded for short periods without an appreciable risk to the health of the individuals concerned. JECFA has established a PMTDI for iodine in adults of 1 mg/day, or 17 µg/kg bw/day (WHO, 1989).

### 3.5.3. Dietary exposure to iodine

Table 10 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile iodine exposure of the Irish adult and child populations from all food groups.

**Table 10. Estimated iodine exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % SCF UL for adults (600 µg/day) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 147 | 153 | 354 | 359 | 2.0 | 2.0 | 4.8 | 4.9 | 24.5% | 25.4% | 58.9% | 59.8% |
| CHILDREN | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % SCF UL for adults (600 µg/day) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 162 | 167 | 363 | 368 | 5.4 | 5.5 | 12.7 | 12.9 | 27.1% | 27.8% | 60.4% | 61.3% |

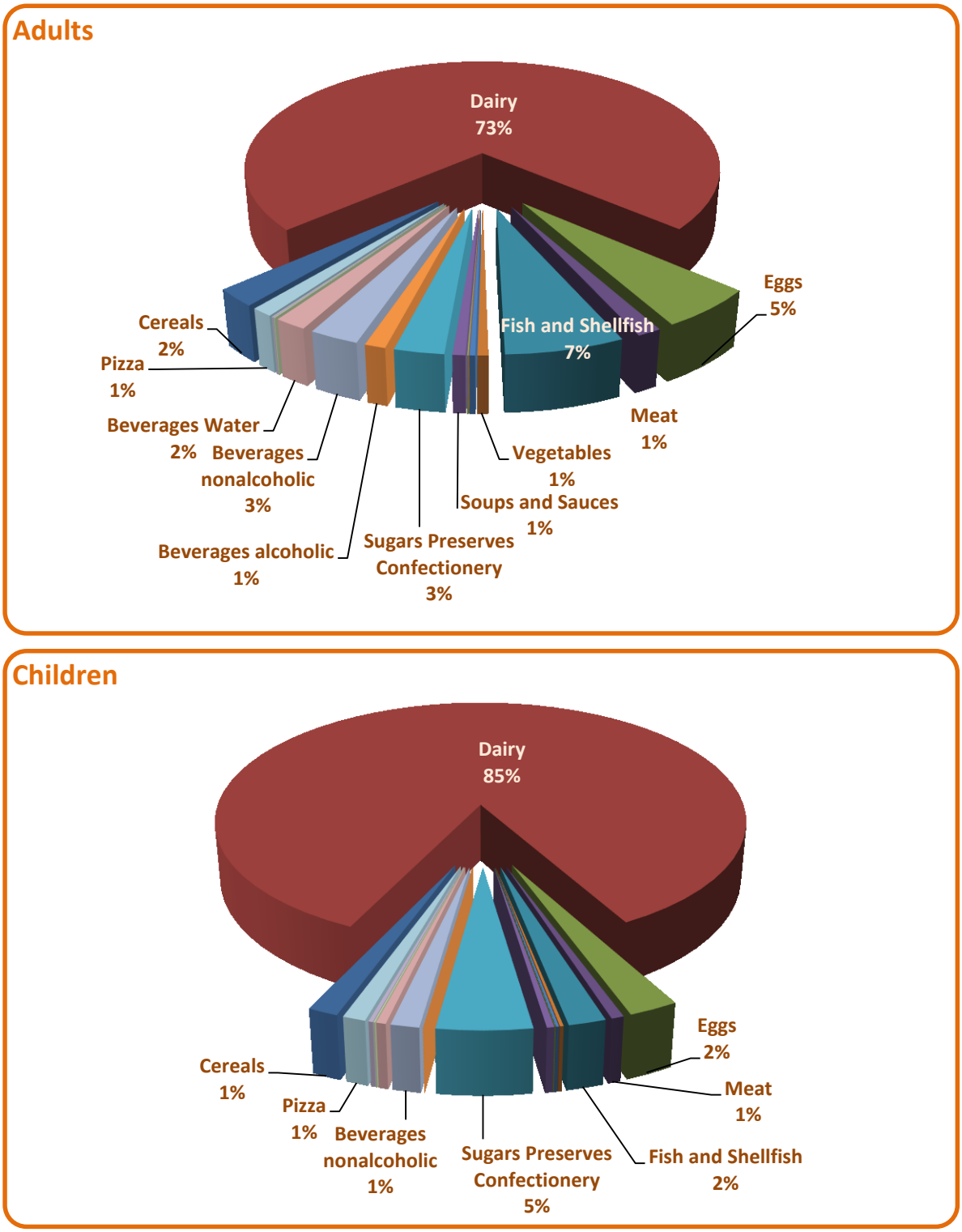
As can be seen from Table 10, for adults the average intake of iodine was estimated to fall between 147 - 153 µg/day. The above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 354 - 359 µg.

For children, the average intake ranged from 162 - 167 µg/day and above average intake ranged from 363 - 368 µg/day.

Iodine was detected in 60% of all foods analysed.

Figure 5 shows that for both adults and children, dairy products are the most important contributors of iodine to the diet, contributing 73% and 85% to overall intake, respectively.

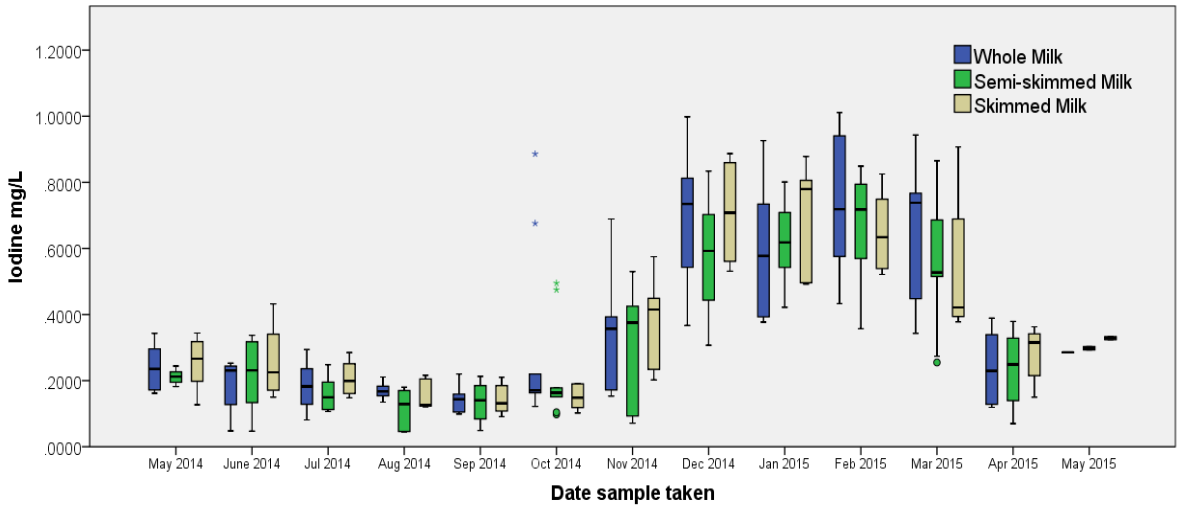
**Figure 5. Contribution of the various food groups in which iodine was detected (LB) as a percentage of total iodine intake in adults and children**




Milk and dairy products can contain relatively high amounts of iodine due to the use of hygiene products or supplements used in the dairy industry. Seasonal variations in milk taken at different times

of the year have also been observed. The differences observed between iodine content in milk sampled in summer versus milk sampled in winter can possibly be attributed to the use of iodinated cattle feed supplements during the winter, when the cattle are predominantly housed indoors, while during the summer when cattle are grazing, there is less need for supplementation. This assumption is supported by a finding of a survey on iodine content in Irish milk conducted June 2001 to April 2002 reported in the previous TDS (FSAI, 2011) and a survey on seasonal differences in milk iodine content conducted in the UK (MAFF, 2000). A new survey conducted during 2014 and 2015 again confirms this finding (see Figure 6) (FSAI, unpublished data).

**Figure 6. Distribution of iodine concentration mg/L in milk samples in Ireland over the period of 13 months (2014/2015) (source: FSAI)**




Milk samples analysed in this TDS were sampled in October/November 2012 and showed concentrations of 0.36 and 0.45 mg iodine/L in semi-skimmed/skimmed and whole milk, respectively.

### 3.5.4. Risk characterisation

Iodine is an essential nutrient in the body. As can be seen from Table 10, the intakes of iodine estimated in this study were in line with the RDA for iodine/iodide for Irish adults aged 18 - 64 of 130 µg/d and the EFSA AI of 130 µg/d for children. Exposure in both population groups was below the UL of 600 µg/d set by the SCF and the JECFA PMTDI of 17 µg/kg bw/day (WHO, 1989).

Results of this TDS indicate that generally, the Irish population is neither likely to be deficient in iodine, nor at risk from the toxic effects of excess iodine in their diet. However, seasonal fluctuation in milk iodine concentration can lead to occasional deficiency in or excessive intake of iodine, as reported in the previous TDS (FSAI, 2011).

## 3.6. Lead

### 3.6.1. Sources of exposure to lead

Food and water represent the major sources of exposure to lead for the general population (EFSA, 2010a; JECFA, 2000a). Lead contamination of food and water arises as a result of industrial releases to the environment, such as from mining, smelting, battery manufacturing and the now diminished use of leaded petrol. Data from EFSA and the SCOOP report on heavy metals show that levels of lead in most commonly consumed foodstuffs are generally low (EFSA, 2010a; EC, 2004). However, like mercury, lead can accumulate in fish and shellfish and in addition, can be found at higher levels in the offal (liver and kidney) of food animals. Consumers eating diets rich in these foods may therefore, be exposed to an unacceptable level of lead. Levels of lead in these particular foods and in fruit and vegetables generally are stringently regulated in the EU. A further source of lead in the diet is from food containers containing lead, e.g. storage in lead-soldered cans, ceramic vessels with lead glazes and leaded crystal glass. The first of these has now been largely discontinued, at least in the EU, and the second is also strictly regulated under EU legislation related to food contact materials. However, there are repeated instances of food dishes, utensils or other materials manufactured outside the EU that release lead into food at levels above those permitted in the EU. Finally, the past use of lead as a material for water pipes in many older houses may result in unacceptably high levels in water supplies. The 1998 Drinking Water Directive, in line with the WHO's recommendations, sets a limit of 10 µg/l for lead in drinking water. In 2015, Irish Water estimated that lead pipework is in up to 200,000 residential properties in Ireland as well as many of the commercial and public buildings (Irish Water, 2015b). Occupants of such premises might therefore be exposed to additional lead from their water supply. However, tap water tested in this survey, which was taken from individual households across the country, did not contain detectable levels of lead.

### 3.6.2. Health effects of lead

**Both EFSA in 2010 and JECFA in 2011 concluded that the previously established PTWI of 25 µg/kg bw was no longer appropriate as there is no evidence for a threshold for critical lead-induced effects. EFSA established BMDLs for: developmental neurotoxicity, a $BMDL_{01}$ of 0.50 µg/kg bw, for effects on systolic blood pressure, a $BMDL_{01}$ of 1.50 µg/kg bw and for effects on prevalence of chronic kidney disease, a $BMDL_{10}$ of 0.63 µg/kg bw (EFSA, 2010a).**

The toxic effects of lead have been principally established in studies on people exposed to lead in the course of their work. Lead is a cumulative poison and produces a continuum of effects, primarily on the haematopoietic system, the nervous system, and the kidneys (EFSA, 2010a; JECFA, 2011).

Short-term exposure to high levels of lead can cause brain damage, paralysis (lead palsy), anaemia and gastrointestinal symptoms. Longer-term exposure can cause damage to the kidneys, reproductive and immune systems in addition to effects on the nervous system. The most critical effect of low-level lead exposure is on intellectual development in young children and, like mercury, lead crosses the placental barrier and accumulates in the foetus. Infants and young children are more vulnerable than adults to the toxic effects of lead, and they also absorb lead more readily. Even short-tem, low-level exposures of young children to lead is considered to have an effect on neurobehavioral development (EFSA, 2010a; JECFA, 2011). Lead and inorganic lead compounds are possibly carcinogenic to humans (Group 2B) (IARC, 2006).

The CONTAM Panel identified developmental neurotoxicity in young children and cardiovascular effects and nephrotoxicity in adults as the critical effects for the risk assessment. The Panel concluded that the previously set provisional tolerable weekly intake (PTWI) of 25 µg/kg bw was no longer appropriate as there was no evidence for a threshold for critical lead-induced effects. Therefore, a MoE approach was applied to risk characterisation.The respective BMDLs derived from blood lead levels in µg/L (corresponding dietary intake values in µg/kg bw per day) were: developmental neurotoxicity $BMDL_{01}$, 12 µg/L (0.50 µg/kg bw); effects on systolic blood pressure $BMDL_{01}$, 36 µg/L (1.50 µg/kg bw); effects on prevalence of chronic kidney disease $BMDL_{10}$, 15 µg/L (0.63 µg/kg bw) (EFSA, 2010a). The Panel noted that in adults, children and infants, the MoEs between BMDLs and the estimated intake from food and water were such that the possibility of an effect from lead in some consumers, particularly in children from 1 - 7 years of age, could not be excluded (EFSA, 2010a).

### 3.6.3. Dietary exposure to lead

Table 11 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile lead exposure of the Irish adult and child populations from all food groups.

**Table 11. Estimated lead exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | |
|---|---|---|---|---|---|---|---|
| Daily Intake µg/d | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 2.61 | 8.72 | 7.90 | 15.04 | 0.04 | 0.12 | 0.11 | 0.22 |
| **CHILDREN** | | | | | | | |
| Daily Intake µg/d | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 1.27 | 5.10 | 2.7 | 7.7 | 0.04 | 0.17 | 0.09 | 0.27 |

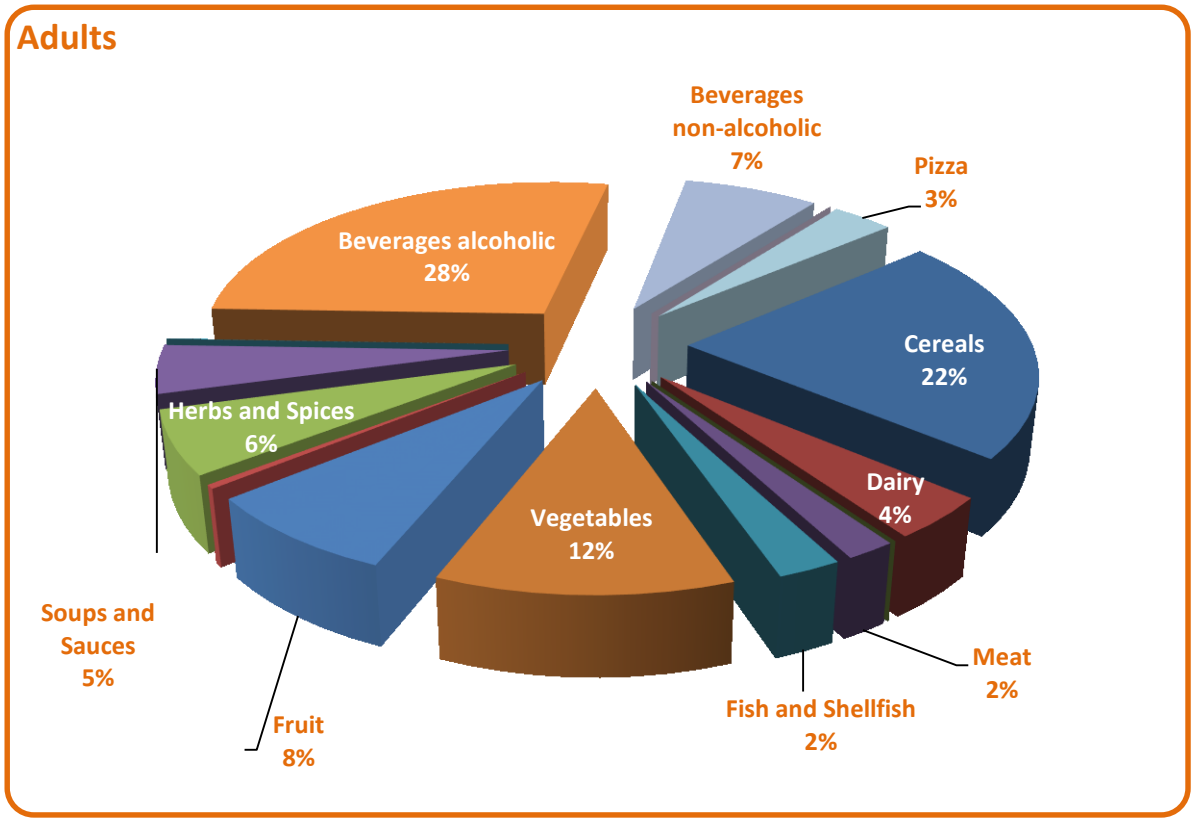
As can be seen from Table 11 for adults, the average intake of lead was estimated to fall between 0.04 - 0.12 µg/kg bw/day. The above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 0.11 - 0.22 µg/kg bw/day. These estimates are in line with results reported in the previous TDS (FSAI, 2011).

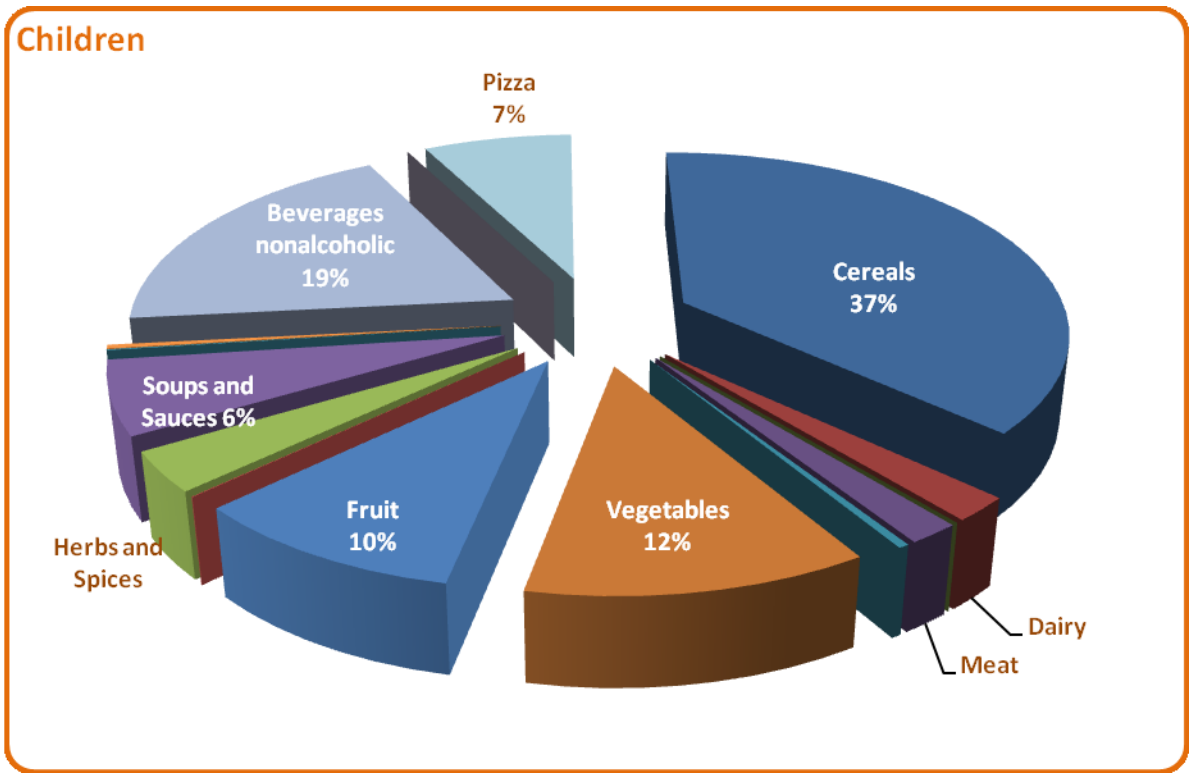
For children, the average intake of lead was estimated to fall between 0.04 - 0.17 µg/kg bw/day. The above average (97.5$^{th}$ percentile) intake was estimated to fall between 0.09 - 0.27 µg/kg bw/day.

EFSA reported that in average adult consumers over the whole EU, lead dietary intake ranges from 0.36 to 1.24 µg/kg bw/day, and up to 2.43 µg/kg bw/day in high consumers in Europe (EFSA, 2010a), which are much higher than the intake levels found for Irish adults.

Lead was detected above the LOD in 29% of all samples analysed. Figure 7 shows the main contributing food groups to dietary lead exposure, based on LB measurements, revealing that alcoholic beverages, cereals and vegetables were the major contributors (28%, 22% and 12% of total intake, respectively) in adults. Childrens' cereals, beverages and vegetables were found to be the major contributors (37%, 19% and 22% of total intake, respectively).

Figure 7. Contribution of the various food groups in which lead was detected (LB) as a percentage of total lead intake in adults and children



### 3.6.4. Risk characterisation

As a consequence of the withdrawal of the PTWI of 25 µg/kg bw/week, EFSA (2012) considered it appropriate to calculate MoEs to characterise the risk from lead exposure.

Table 12 provides MoEs derived for both average and above average intake estimates of lead in the Irish adult and child populations, based on the EFSA $BMDL_{01}$ of 0.50 µg/kg bw/day for developmental neurotoxicity, $BMDL_{01}$ of 1.50 µg/kg bw/day for effects on systolic blood pressure and the $BMDL_{10}$ of 0.63 µg/kg bw/day for effects on prevalence of chronic kidney disease.

**Table 12. MoEs for lead derived for Irish adults and children based on BMDLs as set by EFSA (EFSA, 2010a)**

| Estimated intake of lead (µg/kg bw/day LB - UB) | | MoE based on $BMDL_{10}$ (0.63 µg/kg bw/day) for CKD[1] | MoE based on $BMDL_{01}$ (1.50 µg/kg bw/day) for SBP[1] | MoE based on $BMDL_{01}$ (0.5 µg/kg bw/day) for DNT[2] |
|---|---|---|---|---|
| Adults Mean | 0.04 – 0.12 | 16 - 5 | 38 - 13 | |
| Adults 97.5$^{th}$ percentile | 0.11 – 0.22 | 6 - 3 | 14 - 7 | |
| Children Mean | 0.04 – 0.17 | | | 13 - 3 |
| Children 97.5$^{th}$ percentile | 0.09 – 0.27 | | | 6 - 2 |
| [1] Critical for adults<br>[2] Critical for children | | | | |

Estimates of dietary exposure to lead in adults are lower than both the BMDL intake value for effects on systolic blood pressure (1.50 µg/kg bw per day) and the BMDL intake value for effects on the prevalence of chronic kidney disease (0.63 µg/kg bw per day). The respective MoEs for the lower set BMDL for chronic kidney disease range from 5 - 18 and from 3 - 6 for the mean and 97$^{th}$ percentile consumer, respectively. Estimated exposure in children was below the $BMDL_{01}$ intake level of 0.50 µg/kg bw per day for neurodevelopmental effects. The MoE in average consumers ranged from 3 - 12 and from 2 - 5 in 97.5$^{th}$ percentile consumers.

The Panel concluded that a margin of exposure of ten or greater would be sufficient to ensure that there was no appreciable risk of a clinically significant effect on systolic blood pressure or of a clinically significant change in the prevalence of chronic kidney disease, and that overall, the risk at MoEs of greater than 1.0 would be very low.

With regard to effects on IQ, EFSA concluded that that a MoE of ten or greater should be sufficient to ensure that there was no appreciable risk of a clinically significant effect on IQ. At lower MoEs, but greater than 1.0, the risk is likely to be low, but not such that it could be dismissed as of no potential concern (EFSA, 2010b). In conclusion, risks from lead in foods are likely to be low however, given EFSA's conclusion that exposure margins between 1-10 should not be dismissed as of no potential concern with regard to effects on IQ, further efforts to reduce exposure should be made.

In 2010, EFSA noted that MoEs between BMDLs and the estimated intake from food and water were such that the possibility of an effect from lead in some consumers, particularly in children from 1 - 7 years of age, could not be excluded (EFSA, 2010a). Consequently, in 2015[2], the European Commission undertook measures to reduce the dietary exposure to lead in food by lowering existing maximum levels and setting additional maximum levels for lead in relevant commodities.

## 3.7. Mercury

### 3.7.1. Sources of exposure to mercury

Sources of mercury exposure include:

- Natural occurrence of the element
- Anthropogenic sources (mining operations, industrial processes, combustion of fossil fuels (especially charcoal), production of cement, and incineration of municipal, chemical, and medical wastes)

Data from EFSA and the SCOOP report on heavy metals on exposure of the European population to heavy metals in its diet, showed that mercury is relatively widely distributed in food at very low levels, and primarily in the less toxic inorganic form, but that the most toxic form of mercury, methylmercury, is found at significant levels only in fish and seafood, in particular top predatory fish such as swordfish and marlin (EFSA, 2004a; EC, 2004; JECFA, 2004; EFSA, 2012). It has been estimated that methylmercury comprises 75–100% of the total mercury in seafood, occurring as a consequence of industrial releases of inorganic mercury into marine environments, followed by uptake into marine microorganisms which then convert the less toxic inorganic mercury into the more toxic methyl mercury. The methylmercury then accumulates through the food chain due to its low rate of breakdown, reaching potentially toxic levels in species at the top of the food chain which may then form part of the human diet. The amount of methylmercury in fish and shellfish correlates with a number of factors including the size and age of the fish, the species and the level of mercury in the

[2] Commission Regulation (EU) 2015/1005 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of lead in certain foodstuffs

waters that form their primary habitat. Larger, older, predatory species such as shark, marlin, swordfish and fresh tuna usually contain higher levels than other marine fish. Canned tuna on average has been found to contain half the amount of mercury of fresh tuna. This is because different species and smaller more immature fish are used for canning. Shellfish, particularly filter feeders such as mussels and scallops can also take up mercury from their environment, accumulating it in their viscera and hence, may contribute significantly to dietary exposure.

Individuals consuming a diet containing a high content of predatory fish and/or shellfish may exceed the PTWI, for methylmercury established by JECFA in 2003, of 1.6 µg/kg body weight and may therefore, be at risk. In its 2004 evaluation of mercury and methylmercury, EFSA's CONTAM Panel looked at exposure of the European population to mercury in its diet. The estimated intakes of mercury in Europe varied by country, depending on the amount and the type of fish consumed. The mean intakes were in most cases, below the PTWI, but high intakes exceeded the PTWI. In 2012, the CONTAM Panel re-evaluated mercury and methylmercury and established a TWI for methylmercury of 1.3 µg/kg bw, and a TWI for inorganic mercury of 4 µg/kg bw, both expressed as mercury and confirmed previous findings with regard to population exposure.

EFSA concluded that methylmercury toxicity has been demonstrated at low exposure levels, and exposure to this compound should therefore be minimised, but if measures to reduce methylmercury exposure are considered, the potential beneficial effects of fish consumption should also be taken into account (EFSA, 2004a; EFSA, 2012).

### 3.7.2. Health effects of mercury

**A TWI for methylmercury of 1.3 µg/kg bw, and for inorganic mercury of 4 µg/kg bw, both expressed as mercury, were established by EFSA. The PTWI established by JECFA for methylmercury is 1.6 µg/kg bw and for inorganic mercury 4 µg/kg bw.**

Excessive exposure to mercury is associated with a wide spectrum of adverse health effects including damage to the central nervous system (neurotoxicity), the kidney and potentially the cardiovascular system.

Different forms of mercury, i.e. mercury metal, inorganic mercury salts such as mercuric chloride and organic forms of mercury such as methylmercury, produce different patterns of toxicity. The main concern in relation to the toxicity of mercury in the general population exposed to low levels of mercury in their diet relates to the potential neurotoxicity of organic forms of mercury, e.g. methylmercury, in young children. Organic forms of mercury can cross the placental barrier between

the mother and the unborn baby, and epidemiological studies in exposed populations of humans and toxicological studies in animals have shown that this can result in a range of neurological disturbances from impaired learning to obvious brain damage. In its 2004 evaluation, the CONTAM Panel concluded, in line with JECFA and other evaluations, that the developing brain should be considered the most sensitive target organ for methylmercury (EFSA, 2004a). Both expert Committees also noted that there is an increasing body of data indicating that increased exposure to methylmercury may augment the risk of cardiovascular morbidity and mortality (EFSA, 2004a; JECFA, 2004). The reported associations between methylmercury exposure and cardiovascular disease were again addressed by JECFA in its update in 2006 (FAO/WHO, 2007) and EFSA in 2012 (EFSA, 2012). The importance of taking the beneficial effects of fish consumption into account when studying cardiovascular outcomes of methylmercury has become evident. Although the observations related to myocardial infarction, heart rate variability and possibly blood pressure are of potential importance, they are still not conclusive. Consequently, after carefully considering endpoints other than neurodevelopmental outcomes, particularly cardiovascular disease, the CONTAM Panel concluded that associations between methylmercury exposure and neurodevelopmental outcomes after prenatal exposure still form the best basis for derivation of a health-based guidance value for methylmercury (EFSA, 2012).

Maternal hair mercury concentration determined in human cohort studies was used as the basis for derivation of a health-based guidance value. By application of a maternal hair to maternal blood ratio of 250, the maternal hair mercury concentration with no appreciable adverse effect (11.5 mg/kg) was converted into a maternal blood mercury concentration of 46 µg/L. Using a one-compartment toxicokinetic model, the value of 46 µg/L in maternal blood was converted to a daily dietary mercury intake of 1.2 µg/kg bw. A data-derived uncertainty factor of 2 was applied to account for variation in the hair to blood ratio. In addition, a standard factor of 3.2 was applied to account for interindividual variation in toxicokinetics, resulting in a total uncertainty factor of 6.4. A TWI for methylmercury of 1.3 µg/kg bw expressed as mercury, was established (EFSA, 2012).

The critical target for toxicity of inorganic mercury is the kidney. Other targets include the liver, nervous system, immune system, reproductive and developmental systems. Having considered the experimental animal data on inorganic mercury, including some recent studies not reviewed by JECFA in its evaluation of 2010, the Panel in 2012 (EFSA, 2012) agreed with the rationale of JECFA in setting a health-based guidance value using kidney weight changes in male rats as the pivotal effect. Based on the $BMDL_{10}$ of 0.06 mg/kg bw per day, expressed as mercury and an uncertainty factor of 100 to account for inter and intra species differences, with conversion to a weekly basis and rounding to one significant figure, the Panel established a TWI for inorganic mercury of 4 µg/kg bw, expressed as mercury.

IARC has concluded that methylmercury compounds are possibly carcinogenic to humans (Group 2B) (IARC, 1993).

### 3.7.3. Dietary exposure to mercury

Table 13 presents the estimated LB and UB mean and 97.5$^{th}$ percentile mercury exposure of the Irish adult and child populations from all food groups. Of all the foods analysed, only 11% were found to contain mercury. Of these, 97% were fish and seafood samples, and the LB intakes of mercury, as presented in Table 13, reflect this source. The UB estimates which are considerably higher, reflect the assumption that in food where the mercury levels were <LOD, mercury was present at the LOD.

## Table 13. Estimated mercury exposure of the Irish adult and child populations from all food groups

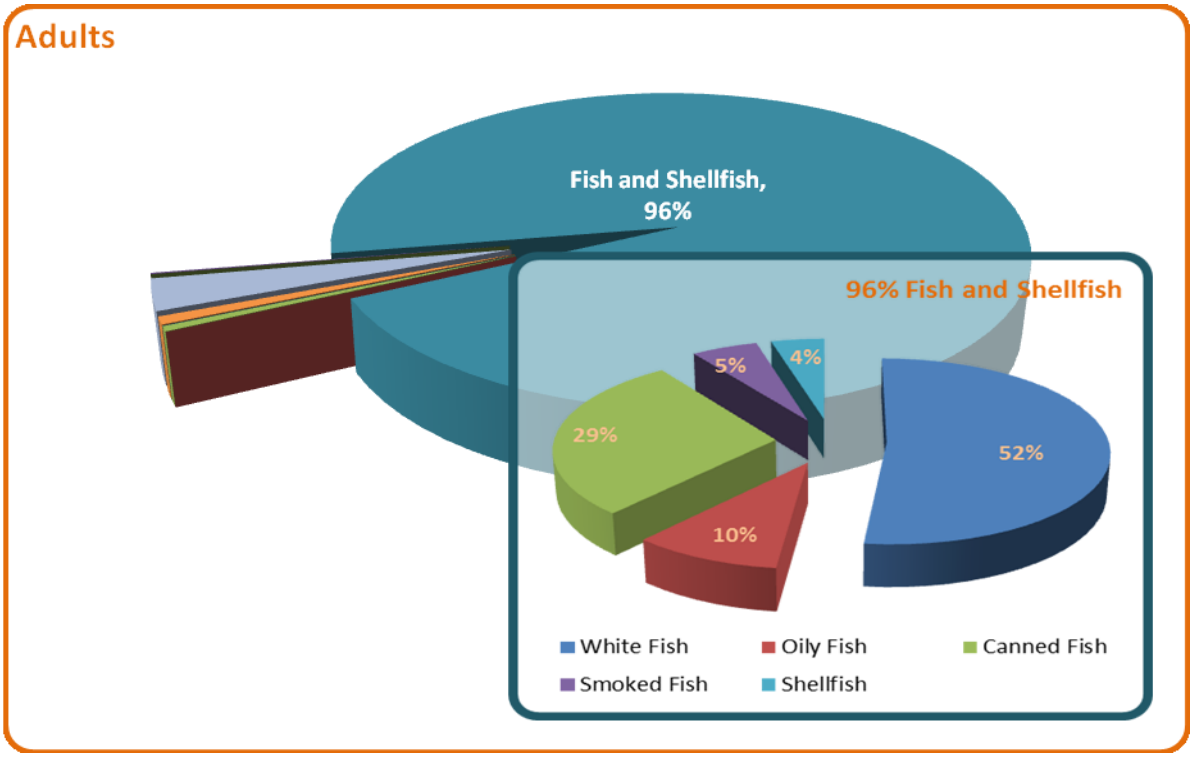
| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw (weekly intake in parenthesis) | | | | % of EFSA PTWI (1.6 µg/kg bw/week) for methylmercury | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 1.78 | 9.00 | 9.47 | 18.00 | 0.02 (0.17) | 0.12 (0.84) | 0.12 (0.84) | 0.25 (1.74) | 10% | 52%* | 53% | 109%* |
| **CHILDREN** | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw (weekly intake in parenthesis) | | | | % of EFSA PTWI (1.6 µg/kg bw/week) for methylmercury | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.61 | 5.20 | 3.17 | 8.31 | 0.02 (0.14) | 0.17 (1.19) | 0.11 (0.77) | 0.31 (2.18) | 9% | 74%* | 48% | 136%* |
| *Since methylmercury predominantly occurs in fish, UB values, which take into account all foodgroups in which methylmercury was not detected, i.e. non-fish foodgroups, have not been taken into account in the risk characterisation of methylmercury | | | | | | | | | | | |

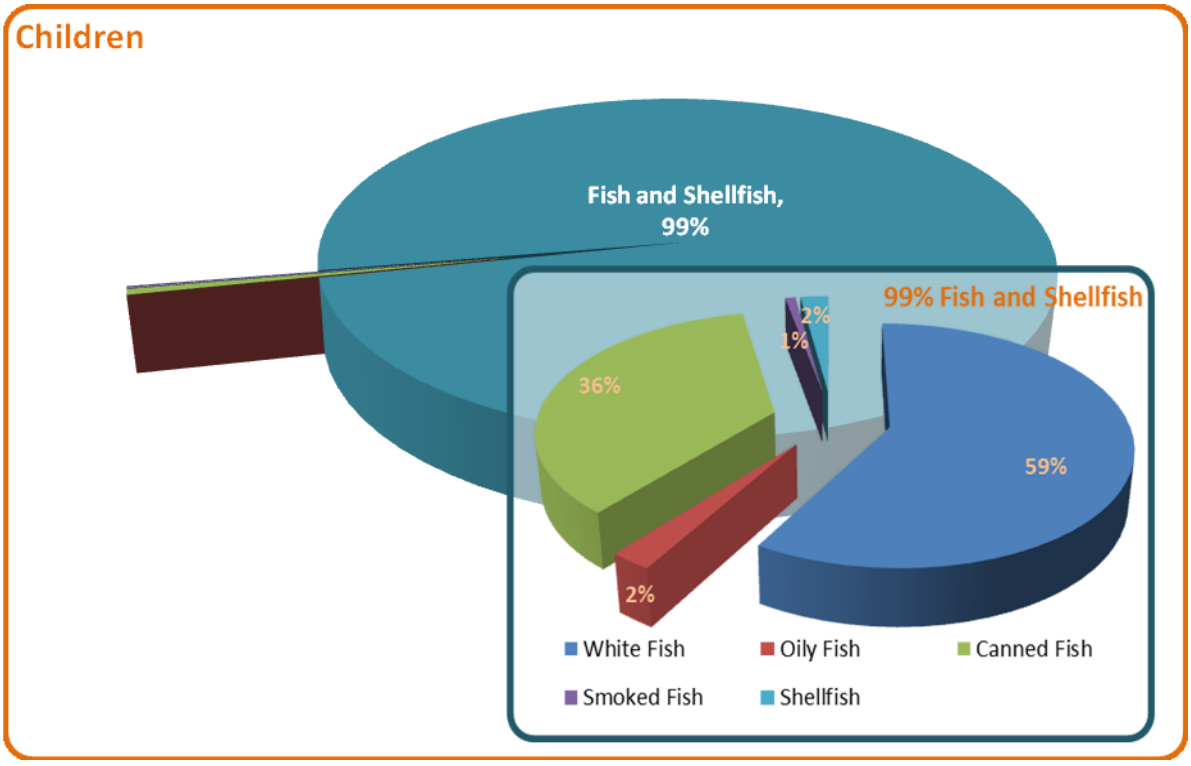
Assuming that all mercury in fish is present in the form of methylmercury, the average intake of methylmercury, based on LB estimates (the most valid comparison, since this represents intake from fish, likely to be the only source of mercury in the diet) in adults was estimated at 0.17 µg/kg bw per week and above average intake was estimated at 0.84 µg/kg bw per week (see Table 13). These levels compare well with estimates derived in the previous TDS (FSAI, 2011).

For children, based on the same assumptions, average exposure was estimated at 0.14 µg/kg bw per week and above average intake at 0.77 µg/kg bw per week.

For both adults and children, white fish was found to be the main contributor to mercury intake, as shown in Figure 8.

Figure 8. Contribution of the various food groups in which mercury was detected (LB) as a percentage of total mercury intake in adults and children



These findings are in line with EFSA's risk assessment of 2012 that fish muscle meat was the main contributor to methylmercury dietary exposure for all age classes, followed by fish products (EFSA, 2012).

### 3.7.4. Risk characterisation

Assuming that all mercury in fish is present in the form of methylmercury, the average intake of methylmercury, based on LB estimates (the most valid comparison, since this represents intake from fish, likely to be the only source of mercury in the diet) in adults, corresponds to 10% of the PTWI and above average intake corresponds to 53% of the PTWI (Table 13). For children, based on the same assumptions, exposure corresponds to 9% of the PTWI and above average intake corresponds to 48% of the PTWI.

Estimated (LB) dietary exposure for both adults and children did also not exceed the TWI for for inorganic mercury (for which the TWI is three fold higher).

These findings are supported by the results of a recent pilot biomonitoring study (Cullen *et al*., 2014), entitled "*Demonstration of a study to Coordinate and Perform Human Biomonitoring on a European Scale (DEMOCOPHES)".* For this study, hair mercury concentrations were determined from 120 Irish mother/child pairs, in order to determine the extent of mercury exposure among mothers and their children in Ireland, and to identify factors associated with elevated levels. Average levels in mothers (0.262 µg/g hair) and children (0.149 µg/g hair) did not exceed the US EPA guidance value (1.0 µg/g). Although hair mercury levels were significantly higher in those who frequently consumed fish, these were also below guidance values.

Exposure to mercury was found to be well below the EFSA PTWI of 1.6 µg/kg bw for both adults and children. For both adult and child populations, intake of white fish (52% and 59%, respectively) and canned fish (29% and 36%, respectively) were found to be the major contributors to dietary exposure. Fish, particularly predatory fish (such as shark, marlin, swordfish and fresh tuna), are recognised to be the major source of exposure to mercury in the diet, and since fish consumption in Ireland is below the EU average, Irish consumers are unlikely to be at risk from this source.

## 3.8. Selenium

### 3.8.1. Sources of exposure to selenium

Selenium is distributed widely in nature and is found in most rocks and soils at concentrations between 0.1 and 2.0 ppm (ASTDR, 2003). Selenium is released into the environment from both natural and industrial sources, with the principal releases of selenium occurring as a consequence of human activities as a result of the combustion of coal. Like other metallic elements it occurs in different valence states, the -2 (selenides), 0 (selenium), +4 (selenites), and +6 (selenates) valence states all being found in nature. The behaviour of selenium in the environment is influenced to a large degree by its oxidation state and the consequent differences in the behaviour of its different chemical compounds (ASTDR, 2003). The soluble selenates are readily taken up by plants and are converted to organic compounds such as selenomethionine, selenocysteine, dimethyl selenide, and dimethyl diselenide.

For the general population, the primary exposure pathways, in order of decreasing relative proportions, are food, water, and air. Selenium is a natural component of the diet and is present particularly in fish (0.32 mg/kg), offal (0.42 mg/kg), brazil nuts (0.25 mg/kg), eggs (0.16 mg/kg) and cereals (0.02 mg/kg), generally as the amino acid derivates selenomethionine and selenocysteine (EFSA, 2009c).

EFSA estimated dietary intake of selenium from food consumption data from the EFSA Comprehensive Food Consumption Database combined with data on the selenium content of foods from the EFSA nutrient composition database (EFSA, 2014). The data covered all age groups from infants to adults aged 75 years and older. Estimates were based on food consumption only, i.e. without dietary supplements. Average selenium intake ranged from 17.2 to 36.3 µg/day in children aged 1 to < 3 years, from 20.6 to 45.9 µg/day in children aged 3 to < 10 years, from 33.9 to 60.3 µg/day in adolescents (10 to < 18 years) and from 31.0 to 65.6 µg/day in adults (≥ 18 years). Average intake was slightly higher in males than in females, mainly owing to the larger quantities of food consumed per day. The main food groups contributing to selenium intake were milk and dairy products, meat and meat products, grains and grain-based products and fish and fish products. Differences in the main contributors to selenium intake between the sexes were minor (EFSA, 2014).

The amount of selenium available in the soil for plant growth and corresponding variations in the intake of selenium by humans differs considerably among regions and countries (SCF, 2000). Use of food supplements and mineral and vitamin preparations can contribute significantly to selenium intake, commonly used selenium supplements providing an additional intake of 100 to 400 µg selenium/day (SCF, 2002).

### 3.8.2. Health effects of selenium

**The SCF has established a UL for selenium of 300 µg/day while the UK Expert Committee on Vitamins and Minerals (EVM) derived a SUL of 450 µg/day for total selenium (EVM, 2003). The US Food and Nutrition Board (FNB) estimated a UL of 400 µg /day (FNB, 2000). In 2014, EFSA set an AI of 70 µg per day for adults (EFSA, 2014d).**

Selenium is regarded as an essential micronutrient for humans and animals, being an essential component of a number of seleno-proteins and enzymes playing a role in physiological functions such as antioxidant defence, reduction of inflammation, thyroid hormone production, DNA synthesis, prevention of cancer, fertility and reproduction (Rayman, 2000). In 2014, EFSA set an AI of 70 µg per day for adults; for infants and children up to 3, an AI of 15 µg/day; for children 4 - 6 years of age, an AI of 20; for children aged 7 - 20 years, an AI of 35 and for children 11 - 14 years, an AI of 55 was derived (EFSA, 2014d).

However, although selenium is an essential element, selenium compounds are toxic at high intakes and show a very steep dose-response curve. Acute oral exposure to extremely high levels of selenium, e.g. several thousand times more than normal daily intake, produces nausea, vomiting and diarrhoea in both humans and laboratory animals (ASTDR, 2003).

Acute oral exposure of humans to high levels of selenium has been reported to cause cardiovascular symptoms, such as tachycardia, while in laboratory animals, acute-and intermediate-duration oral exposure to very large amounts of selenium (approximately 100 times normal human intake) has produced myocardial degeneration (ASTDR, 2003). Chronic oral intake of very high levels of selenium (10–20 times more than normal) can produce selenosis in humans, the major effects of which are dermal and neurological. In selenium-rich areas of China, chronic dietary exposure to excess levels of selenium has caused diseased nails and skin and hair loss, as well neurological problems, including unsteady gait and paralysis. The average intakes in the low-, medium- and high-selenium areas studied

were 70, 195 and 1,438 µg/day for adult males. Selenium compounds have also been reported to have effects on reproduction and offspring in a number of animal species in the presence of maternal toxicity and nutritional deprivation. A number of studies have reported on the health of volunteers taking selenium supplements or bread made with selenium-enriched wheat; no signs of selenium-related toxicity were recorded in these volunteers, who would have been receiving doses in the range 200 - 400 µg/day (EFSA, 2009c). The SCF (2000) adopted the value of 300 µg/day as a UL for adults including pregnant and lactating women, on the basis of a NOAEL of 850 µg/day for clinical selenosis and applying an uncertainty factor of 3, supported by three studies reporting no adverse effects for selenium intake between about 200 and 500 µg/day. As there were no data to support a derivation of a UL for children, the SCF (2000) extrapolated the UL from adults to children on the basis of reference body weights. The proposed UL values range from 60 µg/day (1 - 3 years) to 250 µg selenium/day (15 - 17 years).

### 3.8.3. Dietary exposure to selenium

Table 14 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile selenium exposure of the Irish adult and child populations from all food groups.

**Table 14. Estimated selenium exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | Daily intake in µg as % of SCF UL (300 µg/day) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 58.3 | 68.4 | 111 | 123 | 0.77 | 0.91 | 1.50 | 1.68 | 19% | 23% | 37% | 41% |
| CHILDREN | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | Daily intake in µg as % of SCF UL (90 µg/day (4 - 6 years of age)) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 34.7 | 39.0 | 59.9 | 66.0 | 1.13 | 1.27 | 2.05 | 2.28 | 39% | 43% | 67% | 73% |

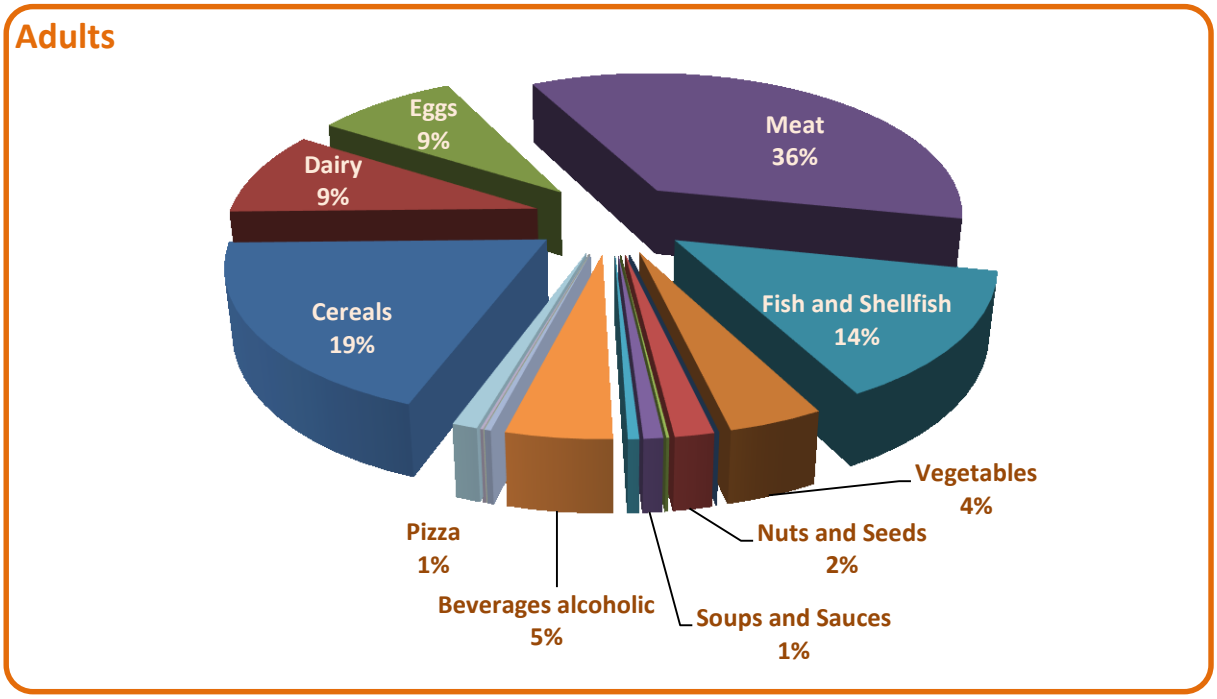
As can be seen from Table 14, for adults, average intake of selenium from food was estimated to fall between 58.3 µg - 68.4 µg/day. The above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 111 and 123 µg/day. The estimates derived for selenium in this study are in line with results of the previous TDS (FSAI, 2011).

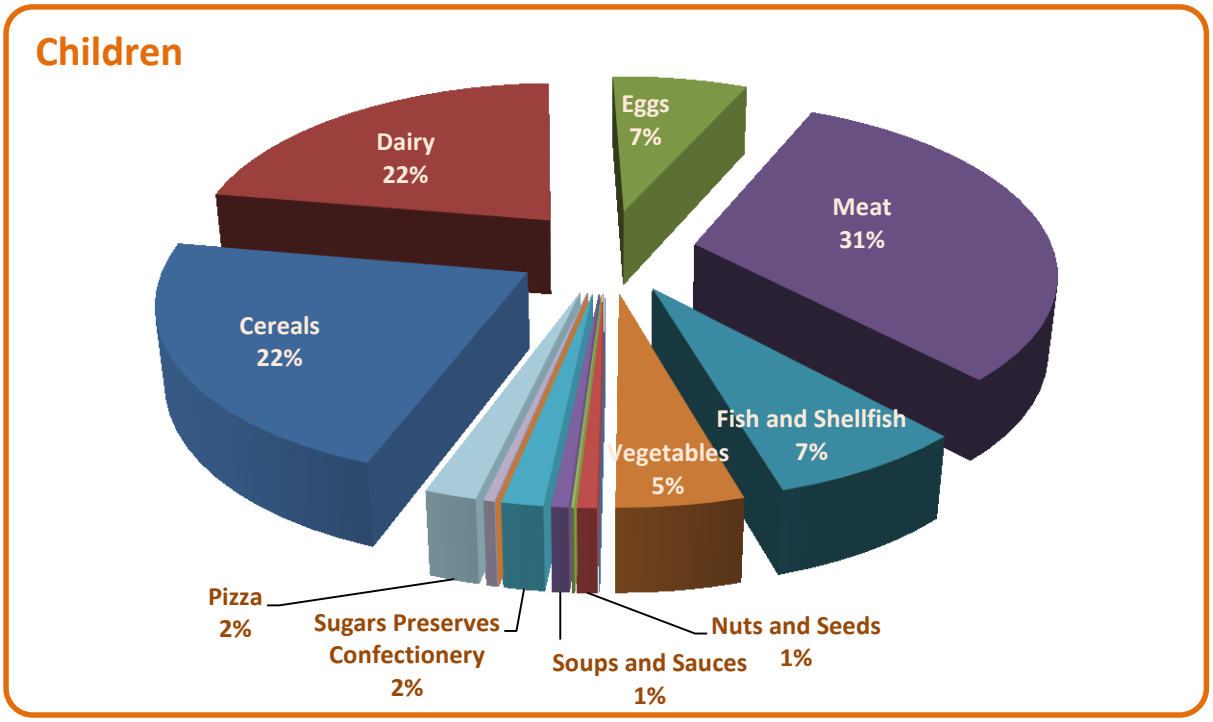
For children, average intake of selenium from food was estimated to fall between 34.7 - 39 µg/day. The above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 59.9 - 66 µg/day.

Selenium was detected above the LOD in 52% of all samples analysed.

Figure 9 shows the main contributing food groups to dietary selenium intake, based on LB measurements, revealing that, for adults, meat was the major contributing source of selenium (36% of total intake), followed by cereals (19% of total intake), fish and fish products (14% of total intake), dairy produce (9% of total intake) and eggs (9% of total intake). For children, meat was the major contributing source of selenium (31% of total intake), followed by cereals (22% of total intake), dairy produce (22% of total intake), fish and fish products (7% of total intake) and eggs (7% of total intake).

Figure 9. Contribution of the various food groups in which selenium was detected (LB) as a percentage of total selenium intake in adults and children



### 3.8.4. Risk characterisation

Selenium is an essential nutrient in the body. As can be seen from Table 14, average intake of selenium from food was very close to the AI of 70 µg/day set by EFSA in 2014 for adults and was also in good agreement with AIs set for children (20 µg/d for children children 4 - 6 years of age, 35 µg/d for children 7 - 10 years of age and 55 µg/d for children 11 - 14 years of age). Intakes of both population groups were well below the UL of 300 µg established by the SCF in 2000. These results indicate that the Irish population is not likely to be selenium-deficient, nor at risk from the toxic effects of excess selenium in their diet.

## 3.9. Tin

### 3.9.1. Sources of exposure to tin

Tin (Sn) is a metallic element obtained chiefly from the mineral cassiterite, where it occurs as tin dioxide, $SnO_2$. The major tin-producing countries are China, Indonesia, Peru, Bolivia, Brazil, and Australia and its main uses are in electrical/electronic and general industrial applications (WHO, 2005b). Over 50% of the world's tin production is used for plating steel or other metals, including food cans, resulting in a potential for exposure via food. Tin is a component of many soils and may be released in dusts from wind storms, roads, and agricultural activities. In general, tin occurs only in trace amounts in natural waters, and for the general population, the main source of exposure to inorganic tin is from the diet.

As reported by the WHO (2005b), inorganic (and total) tin levels are generally less than 1 mg/kg in most unprocessed foods. Higher concentrations can arise as tin(II) in canned foods due to dissolution of the tin coating or tin plate. Tin levels are usually below 25 mg/kg in lacquered food cans, but may exceed 100 mg/kg in unlaquered cans, and increase with storage time, temperature and other factors (JECFA, 2001; Blunden & Wallace, 2003).

Within the EU, stannous chloride is a permitted food additive (E512) for bottled and canned white asparagus only (25 mg Sn/kg). Tin concentrations of vegetables, fruits and fruit juices, nuts, dairy products, meat, fish, poultry, eggs, beverages, and other foods not packaged in metal cans are generally below 2 mg/kg, while tin concentrations in pastas and breads have been reported to range from <0.003 to 0.03 mg/kg. As reported by the WHO (2005b), intake from the diet is dependent on the type and amount of canned food consumed (JECFA, 2001). JECFA has concluded that mean tin intakes in seven countries (excluding Ireland) ranged from <1 up to 15 mg/day per person, but maximum daily intakes could reach 50–60 mg for certain consumers who routinely consume canned fruit, vegetables, and juices from unlacquered cans could ingest 50–60 mg tin daily. In the United

Kingdom, results from total diet studies suggest that tin intake has been falling (mean daily intakes of 4.4, 2.4, and 1.8 mg in 1976, 1994, and 1997, respectively), possibly due to use of an increasing proportion of lacquered cans (Ysart *et al.*, 2000; WHO, 2005b).

### 3.9.2. Health effects of tin

**JECFA has established a PTWI for tin of 14 mg/kg body weight. EFSA concluded in 2005 that the available data from human and animal studies were insufficient to derive a UL for tin.**

In both man and experimental animals, gastrointestinal effects are the main acute manifestation of toxicity associated with ingestion of tin. These are caused by the irritant action of soluble inorganic tin compounds on the mucosa of the gastrointestinal tract. In humans, acute effects resulting from consumption of tin-contaminated foods and drinks have resulted in gastrointestinal symptoms, including abdominal distension and pain, vomiting, diarrhoea, and headache. The balance of evidence suggests that the concentration of tin in contaminated foods is critical to the development of acute gastrointestinal effects, and that tin concentrations of 250 mg/kg in canned foods and 150 mg/kg in canned beverages are more likely to be associated with this (EFSA, 2005c).

### 3.9.3. Dietary exposure to tin

Table 15 presents the estimated LB and UB daily mean and 97.5th percentile tin exposure of the Irish adult and child populations from all food groups.

**Table 15. Contribution of the various food groups in which tin was detected (LB) as a percentage of total tin intake in adults and children**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake mg | | | | Daily Intake mg/kg bw (weekly intake in parenthesis) | | | | Weekly Intake as % of JECFA PTWI of 14 mg/kg bw for tin | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.35 | 0.42 | 3.13 | 3.21 | 0.005 (0.03) | 0.01 (0.04) | 0.04 (0.28) | 0.04 (0.29) | 0.24% | 0.29% | 2.03% | 2.07% |
| CHILDREN | | | | | | | | | | | |
| Daily Intake mg | | | | Daily Intake mg/kg bw (weekly intake in parenthesis) | | | | Weekly Intake as % of JECFA PTWI of 14 mg/kg bw for tin | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.23 | 0.28 | 1.98 | 2.02 | 0.01 (0.05) | 0.01 (0.06) | 0.07 (0.48) | 0.07 (0.49) | 0.38% | 0.5% | 3.4% | 3.5% |

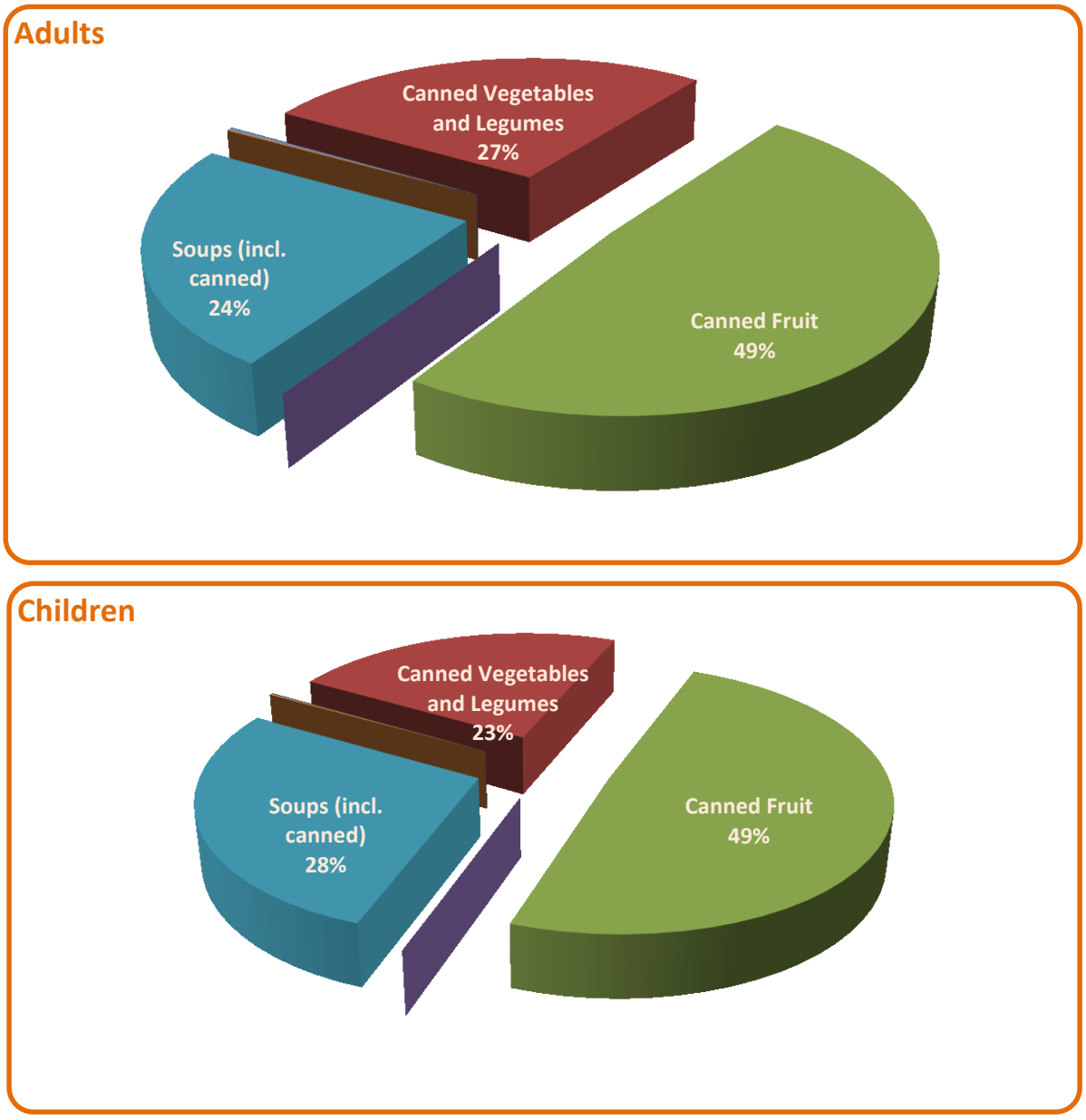
As can be seen from Table 15, for adults the intake of tin (LB) was estimated to be 0.03 mg/kg bw/week at the mean and 0.28 mg/kg bw/week at the 97.5th percentile (see Table 15). These levels compare well with estimates derived in the previous TDS (FSAI, 2011).

For children, the intake of tin was estimated to be 0.05 mg/kg bw/week at the mean and 0.48 mg/kg bw/week at the 97.5th percentile.

Of all the foods analysed, only 9% was found to contain tin. Of this 9%, 73% was canned foods and the LB intake of tin, as presented in Table 15, therefore reflects this source. The UB estimates are considerably higher, since it reflects estimated intake from all sources and the analytical assumption made in assigning a concentration of an analyte to a food where the levels are below the LOD (see Section 2.7.).

Figure 10 shows that, for adults, the major sources of tin in the diet were canned fruit (49%), canned vegetables (27%) and canned soups (24%). For children, the major sources of tin in the diet were canned fruit (49%), canned vegetables (23%) and canned soups (28%).

Figure 10. Percentage contribution of the various food groups in which tin was detected (LB) to total tin intake




### 3.9.4. Risk characterisation

As can be seen from Table 15, for adults, intake of tin (LB) corresponds to 0.24% and 2% of the PTWI for mean and above average consumers, respectively. For children, intake of tin corresponds to 0.38% and 3.4% of the PTWI for mean and above average consumers, respectively.

In conclusion, exposure to tin was estimated to be low in both population groups, i.e. less than 4% of PTWI of 14 mg/kg bw/week established by JECFA, and is not considered to be of concern.

## 3.10. Acrylamide

### 3.10.1. Sources of exposure to acrylamide

Acrylamide is an industrial chemical used in the manufacture of polyacrylamides, which have applications in water treatment and mining, in grouting agents, as a laboratory reagent and in cosmetics. In 2002, Swedish scientists and the Swedish National Food Authority reported the existence of acrylamide in a variety of fried and baked foods, particularly in potato products such as chips and this finding was subsequently confirmed by many other food scientists throughout the world. Research into acrylamide in food shows that the chemical is formed during the frying, roasting or baking of a variety of foods, particularly starchy foods such as potatoes and cereal products, as a side product of the Maillard reaction.

Several large databases of acrylamide occurrence data have been compiled, including the EU's acrylamide monitoring database, the United States Food and Drugs Administration's (FDA) acrylamide survey data and the WHO Summary Information and Global Health Trends database for acrylamide. All these databases show that acrylamide is most prevalent in fried potato products (such as French fries (chips) and potato crisps), cereals, crispbreads, biscuits and other bakery wares, and coffee.

Acrylamide was most recently evaluated by EFSA in 2015. EFSA's CONTAM Panel evaluated a total of 43,419 analytical results from food commodities collected and analysed since 2010 and reported by 24 European countries and six food associations. Acrylamide was found at the highest levels in 'coffee substitutes (dry)' (average medium bound (MB) levels of 1,499 µg/kg) and 'coffee (dry)' (average medium bound (MB) levels of 522 µg/kg). However, due to dilution effects, lower levels are expected in 'coffee beverages' and 'coffee substitutes beverage' as consumed by the European population. High levels were also found in 'potato crisps and snacks' (average MB level of 389 µg/kg) and 'potato fried products (except potato crisps and snacks)' (average MB level of 308 µg/kg). Lower acrylamide levels were found in 'processed cereal-based baby foods' (average MB level of 73 µg/kg), 'soft bread' (average MB level of 42 µg/kg) and 'baby foods, other than cereal-based' (average MB level of 24 µg/kg) (EFSA, 2015).

Mean and 95th percentile dietary acrylamide exposures across all European surveys and age groups were estimated at 0.4 to 1.9 µg/kg bw per day and 0.6 to 3.4 µg/kg bw per day, respectively. The main contributor to total dietary exposure was generally the category 'potato fried products (except potato crisps and snacks)'. European infants, toddlers and other children were found to be the most exposed population groups (EFSA, 2015). Exposure estimates for acrylamide in the diet reported by JECFA, range from 0.3 to 2.0 µg/kg bw/day for mean consumers and 0.6 to 3.5 µg/kg bw/day for high-level consumers (JECFA, 2006).

### 3.10.2. Health effects of acrylamide

**Since acrylamide is a probable human carcinogen, no regulatory bodies have established Tolerable Intakes for acrylamide. In 2015, EFSA derived BMDLs for non-neoplastic ($BMDL_{10}$ 0.43 mg/kg bw) and neoplastic effects ($BMDL_{10}$ 0.17 mg/kg bw).**

The main concern regarding possible health effects of acrylamide in food is its carcinogenicity and genotoxicity (DNA-damaging effects). It causes tumours in laboratory rats, and since its discovery in food, detailed studies have been carried out to establish whether it causes cancer in humans, but as yet there is no definitive evidence that this is the case. However, it is classified by IARC as a probable human carcinogen (IARC, 1994). It has also been shown to be neurotoxic in humans and may affect reproductive processes. The risks to the health of consumers of acrylamide in food have been assessed by many international bodies including EFSA, the FAO and the WHO. EFSA's 2015 scientific opinion confirmed previous evaluations that based on animal studies, acrylamide in food potentially increases the risk of developing cancer for consumers in all age groups. In addition to carcinogenicity, neurotoxicity, adverse effects on male reproduction and developmental toxicity were identified as possible critical endpoints for acrylamide toxicity from experimental animal studies. The CONTAM Panel selected $BMDL_{10}$ values of 0.43 mg/kg bw per day for neurotoxicity in rats and 0.17 mg/kg bw per day for neoplastic effects in mice. The Panel concluded that the current levels of dietary exposure to acrylamide are not of concern with respect to non-neoplastic effects. However, although the human studies have not demonstrated acrylamide to be a human carcinogen, the MoEs across dietary surveys and age groups indicate a concern with respect to neoplastic effects (EFSA, 2015). Given the toxicity of acrylamide, EFSA in tandem with other international bodies, has concluded that efforts should be made to reduce acrylamide concentrations in food.

### 3.10.3. Dietary exposure to acrylamide

Table 16 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile acrylamide exposure of the Irish adult and child populations from those food groups that were analysed in the study. However, only those foods that were anticipated to contain acrylamide, cereals and cereal products, meat products, potatoes/potato products and specific vegetables, beers, spirits, coffee and savoury snacks were analysed, thus the intakes given in Table 16 are only derived from these foods. They are however, anticipated to be the major contributors of acrylamide in the diet, based on the published literature.

**Table 16. Estimated acrylamide exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | |
|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 12.46 | 28.84 | 37.50 | 84.82 | 0.16 | 0.38 | 0.51 | 1.03 |
| **CHILDREN** | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB |
| 17.38 | 21.76 | 36.43 | 40.55 | 0.57 | 0.71 | 1.25 | 1.42 |

As can be seen from Table 16, for adults the average intake of acrylamide from food was estimated to fall between 0.16 - 0.38 µg/kg bw/day, while the above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 0.51 - 1.03 µg/kg bw/day. The mean intakes derived in this study are in the lower part of the range of 0.4 to 0.9 µg/kg bw/day for adults (>18 years) reported by EFSA in 2015.

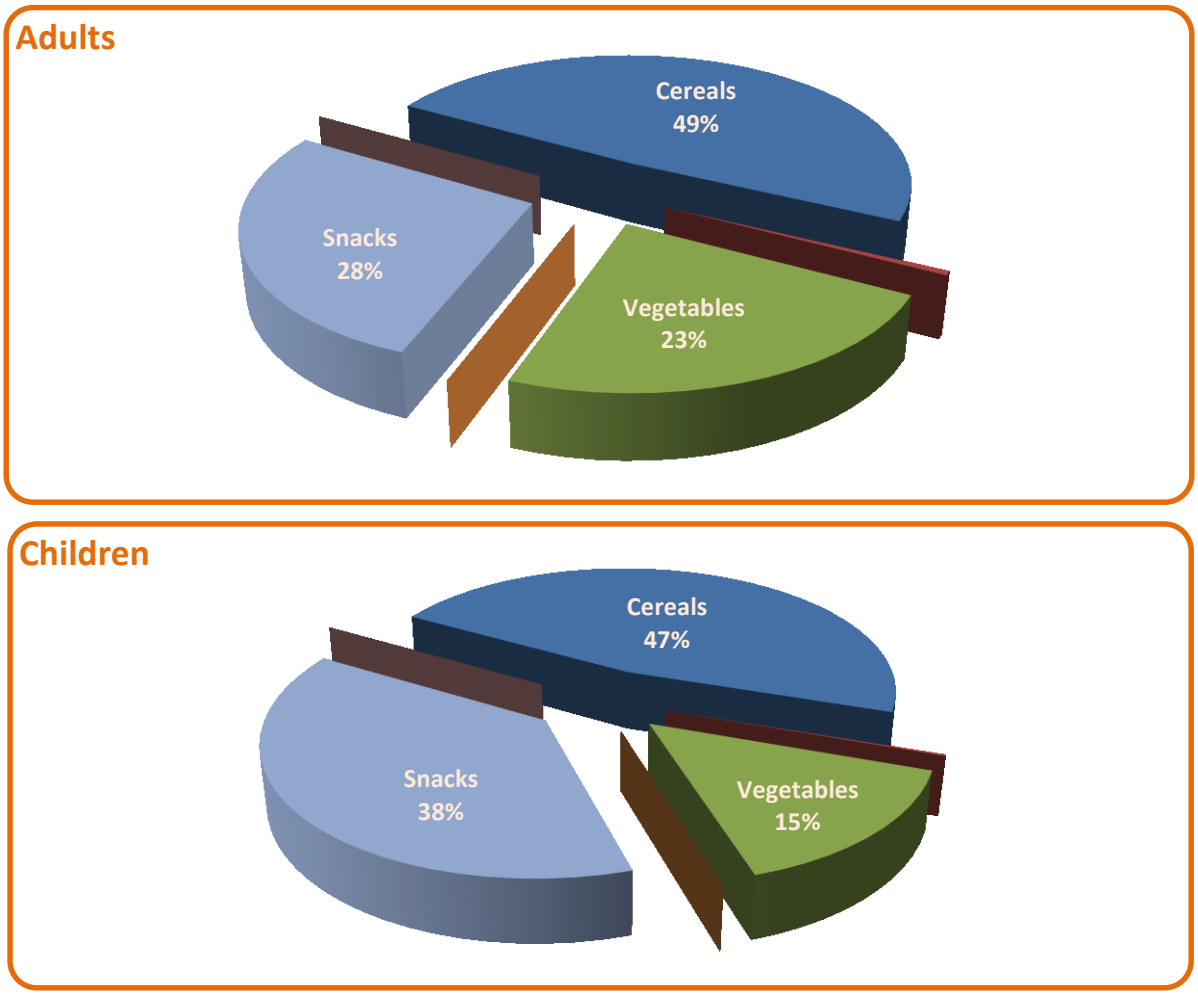
For children, the average intake of acrylamide from food was estimated to fall between 0.57 - 0.71 µg/kg bw/day, while the above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 1.25 - 1.42 µg/kg bw/day. The daily mean intakes derived in this study are in the lower part of the range of 0.5 and 1.9 µg/kg bw per day for children reported by EFSA in 2015 (EFSA, 2015).

As already indicated, acrylamide was only analysed in a number of specific foods, namely those anticipated to contain the contaminant based on published literature. A total of 27 foods were analysed, and acrylamide was detected above the LOD in 44% of these.

Figure 11 shows the relative contribution of these foods (shown as the major food groups to which they belong) to dietary acrylamide intake, based on LB measurements, revealing that, for adults, the cereal group (biscuits, cereals, cake) contributed 49% of total intake, with 23% coming from

vegetables, i.e. potatoes, and 28% coming from savoury snacks, e.g. crisps. In terms of the individual foods in these groups, the exposure analysis confirmed that consumption of fine bakery ware and snacks provided the largest contribution to dietary intake of acrylamide. For children, the cereal group (biscuits, cereals, cake) contributed 47% of total intake, with 15% coming from vegetables, i.e. potatoes, and 38% coming from savoury snacks, e.g. crisps. In terms of the individual foods in these groups, the exposure analysis confirmed that consumption of snacks and breakfast cereals provided the largest contribution to dietary intake of acrylamide.

**Figure 11. Contribution of the various food groups in which acrylamide was detected (LB) as a percentage of total acrylamide intake in adults and children**




### 3.10.4. Risk characterisation

$BMDL_{10}$ values of 0.43 mg/kg bw per day for peripheral neuropathy in rats and of 0.17 mg/kg bw per day for neoplastic effects in mice were established by EFSA (2014d). Table 17 provides MoEs for the $BMDLs_{10}$ calculated by EFSA, derived for both average and above average intake estimates of acrylamide in the Irish adult and child populations.

## Table 17. MoEs for acrylamide derived for Irish adults and children based BMDLs set by EFSA (EFSA, 2014d)

| Estimated intake of acrylamide | | MoE based on $BMDL_{10}$ of 0.43 mg/kg bw/day | MoE based on $BMDL_{10}$ of 0.17 mg/kg bw/day |
|---|---|---|---|
| Adults Mean | 0.16 - 0.38 | 2,606 – 1,143 | 1,030 - 452 |
| Adults 97.5$^{th}$ percentile | 0.51 - 1.03 | 842 - 419 | 333 - 166 |
| Children Mean | 0.57 - 0.71 | 753 - 603 | 298 - 238 |
| Children 97.5$^{th}$ percentile | 1.25 - 1.42 | 344 - 302 | 136 - 119 |

Risk characterisation for non-neoplastic effects was performed using the MoE approach based on the $BMDL_{10}$ value of 0.43 mg/kg bw per day for the most relevant and sensitive endpoint for neurotoxicity. For adults, MoE values for the neurotoxic effects ranged from 1,143 – 2,606 for the mean exposure, and from 419 - 842 for the 97.5$^{th}$ percentile exposure. For children, MoE values ranged from 603 - 753 at the mean and from 302 - 344 at the 97.5$^{th}$ percentile. Usually, for non-genotoxic compounds, unless there are major gaps in the toxicological database, a MoE of 100 is considered sufficient to conclude that there is no health concern (EFSA SC, 2012a). Therefore, these MoEs are not of concern.

For the risk characterisation for neoplastic effects, the MoE approach for compounds that are both genotoxic and carcinogenic is considered appropriate, using as the reference point the $BMDL_{10}$ of 0.17 mg/kg bw per day. For adults, MoE values for neoplastic effects ranged from 452 – 1,030 for the mean exposure and from 166 - 333 for the 97.5$^{th}$ percentile exposure. For children, MoE values ranged from 238 - 298 at the mean and 119 - 136 at the 97.5$^{th}$ percentile.

According to the EFSA Scientific Committee, for substances that are both genotoxic and carcinogenic, an MoE of 10,000 or higher, based on a $BMDL_{10}$ from an animal study, and taking into account overall uncertainties in the interpretation, would be of low concern from a public health point of view. The MoEs calculated in this study therefore, indicate a potential concern with respect to neoplastic effects. However, available human studies have not demonstrated acrylamide to be a human carcinogen.

In conclusion, exposure to acrylamide was compared against a range of BMDLs relating to non-neoplastic ($BMDL_{10}$ 0.43 mg/kg bw) and neoplastic effects ($BMDL_{10}$ 0.17 mg/kg bw). The calculated MoEs for non-neoplastic effects were not of concern for either population group. However, although the human studies have not demonstrated acrylamide to be a human carcinogen, the MoEs indicate a concern with respect to neoplastic effects. The same observations were made by EFSA in its most recent risk assessment (EFSA, 2015). Given the toxicity of acrylamide, EFSA in tandem with other

international bodies, has concluded that efforts should be made to reduce acrylamide concentrations in food, and more rigorous risk management measures are likely to be implemented by the European Commission in 2016.

## 3.11. Nitrites and Nitrates

### 3.11.1. Sources of exposure to nitrates and nitrites

Nitrate ($NO_3$) is a naturally occurring form of nitrogen found in soil and is essential to all life. Due to its high solubility, nitrate also can leach into groundwater, thus drinking water is an additional source of human exposure. Human activities such as intensive agriculture (with use of nitrogenous fertilisers), concentrated livestock and poultry farming can result in high levels of nitrate in drinking water, these levels are however, tightly controlled (to 50 mg/l) under the EC legislation on drinking water standards. As part of the nitrogen cycle, nitrates are fixed in the soil by the action of microorganisms on decaying plants or other organic residues. Other common sources of nitrate include:

- Fertilisers and manure
- Animal feedlots
- Municipal wastewater
- Sludge and septic tanks
- Use as food additive

Nitrogen in the soil is taken up by plants to satisfy nutrient requirements, this nitrate then accumulates in plant leaves and stems. Vegetables are therefore a major source of human exposure to nitrates, through the diet, accounting for between 50 - 75% of nitrate intake (EFSA, 2008b). Higher levels of nitrate tend to be found in leaves whereas lower levels occur in seeds or tubers, and lettuce, rocket, spinach and cabbage in particular, contain relatively high concentrations of nitrate. Beetroot and potatoes may also contribute significantly to dietary exposure. Nitrates and the related nitrites are also used as food additives in the processing of meat products because of their antimicrobial action and their ability to give meat a characteristic pink colour, texture and flavour, although nitrate itself has no effect on meat colour and preservation and its effect is mediated by conversion to nitrite by bacterial action during processing and storage.

Food is the major source of exposure of consumers to nitrite, whether used directly as a food additive or produced from nitrate in, e.g. vegetables, drinking water or use as a food additive via bacterial action. It has been estimated that 5-8% of the nitrate from the diet may be reduced to nitrite by the microflora in the oral cavity (JECFA, 2003; EFSA, 2008b). Some natural occurrence of nitrite in vegetables has also been reported.

### 3.11.2. Health effects of nitrates and nitrites

**An ADI for nitrate of 3.7 mg/kg bw was established by the SCF (SCF, 1997). A similar ADI had been set by JECFA. The SCF has derived an ADI of 0-0.06 mg/kg bw for nitrite (SCF, 1997), while JECFA has set an ADI of 0-0.07 mg/kg bw for nitrite (JECFA, 2003).**

Nitrates are themselves relatively nontoxic, and healthy adults can consume large amounts of nitrate with few known health effects. Most dietary nitrate is readily absorbed, undergoes a number of metabolic interconversions, and is recycled between the saliva and the gut and the bile and the gut, with some excretion in the urine (SCF, 1997; JECFA, 2003; EFSA, 2008b). The concern regarding adverse health effects of nitrates relate to their metabolic conversion to the related nitrites, since as indicated above it has been estimated that 5–8% of the nitrate from the diet may be reduced to nitrite by the microflora in the oral cavity (SCF, 1997; JECFA, 2003; EFSA, 2008b). In the stomach, under acidic conditions, nitrite will be transformed to nitric oxide and other metabolites, and nitric oxide reacts with the haemoglobin in the blood, oxidising its divalent iron to the trivalent form and creating methaemoglobin. This methaemoglobin cannot bind oxygen, which decreases the capacity of the blood to transport oxygen so less oxygen is transported from the lungs to the body tissues, thus causing a condition known as methaemoglobinemia. Pregnant women, adults with reduced stomach acidity, and people deficient in the enzyme that changes methemoglobin back to normal haemoglobin are all susceptible to nitrite-induced methemoglobinemia. Methaemoglobin is produced normally with background levels of 1 - 3%. Levels of 10% or more have been shown clinically to reduce oxygen transport. At levels above 20%, cyanosis and hypoxia can occur and an increase to 50% methaemoglobin can prove fatal. However, this is not likely to result in adverse effects except at very high (accidental) nitrate intakes. The more serious health consideration is the development of ‘blue baby’ syndrome in very young children exposed to high levels of nitrate. The stomach acid of an infant is not as strong as in older children and adults, causing an increase in bacteria that can readily convert nitrate to nitrite, and resulting in a higher level of methaemoglobin. Infants younger than three months of age are more susceptible to methaemoglobinaemia than adults as they lack key enzymes which convert methaemoglobin back to haemoglobin, and this can result in ‘blue baby’ syndrome and death. A number of factors are critical to methaemoglobin formation including the presence of increased nitrite and intestinal infection together with inflammation of the stomach (EFSA, 2008b). A further health concern linked with nitrate exposure is the recognition that nitrites react with secondary amines in food to form nitrosamines, many of which are carcinogenic in experimental

animals and exert other toxic effects. Studies of people exposed to high levels of nitrate or nitrite have not however, provided convincing evidence of an increased risk of cancer.

An ADI for nitrate of 3.7 mg/kg bw/day, equivalent to 222 mg nitrate per day for a 60 kg adult was established by the SCF and was reconfirmed by JECFA in 2002. In 2008, the EFSA CONTAM Panel noted that no new data were identified that would require a revision of the ADI (EFSA, 2008).

### 3.11.3. Dietary exposure to nitrates

Table 18 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile nitrate exposure of the Irish adult and child populations from all food groups that were analysed in the study.

**Table 18. Estimated nitrate exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake mg | | | | Daily Intake mg/kg bw | | | | Daily intake as % of the SCF group ADI for nitrate of 3.7 mg/kg bw/day | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 33.8 | 63.9 | 110.8 | 148.0 | 0.46 | 0.86 | 1.62 | 2.20 | 12.4% | 23.2% | 43.7% | 59.4% |
| **CHILDREN** | | | | | | | | | | | |
| Daily Intake mg | | | | Daily Intake mg/kg bw | | | | % of the SCF group ADI for nitrate of 3.7 mg/kg bw/day | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 14.1 | 28.1 | 40.2 | 55.1 | 0.46 | 0.91 | 1.30 | 1.82 | 12.3% | 24.6% | 35.3% | 49.1% |

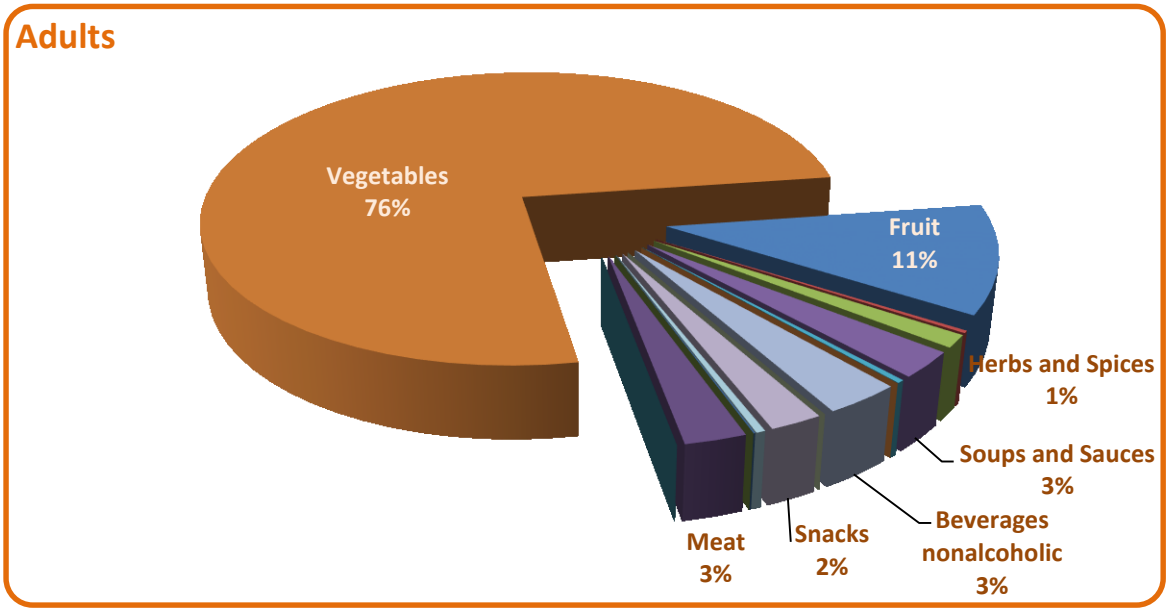
As can be seen from Table 18, for adults, average intake of nitrates from food was estimated to fall between 0.46 - 0.86 mg/kg bw/day. The above average (97.5$^{th}$ percentile) intake was estimated to fall between 1.62 - 2.20 mg/kg bw/day. The levels reported in this TDS are comparable to somewhat higher than levels reported in the previous TDS. However, the previous TDS did not include the intake attributable to tap (drinking) water.

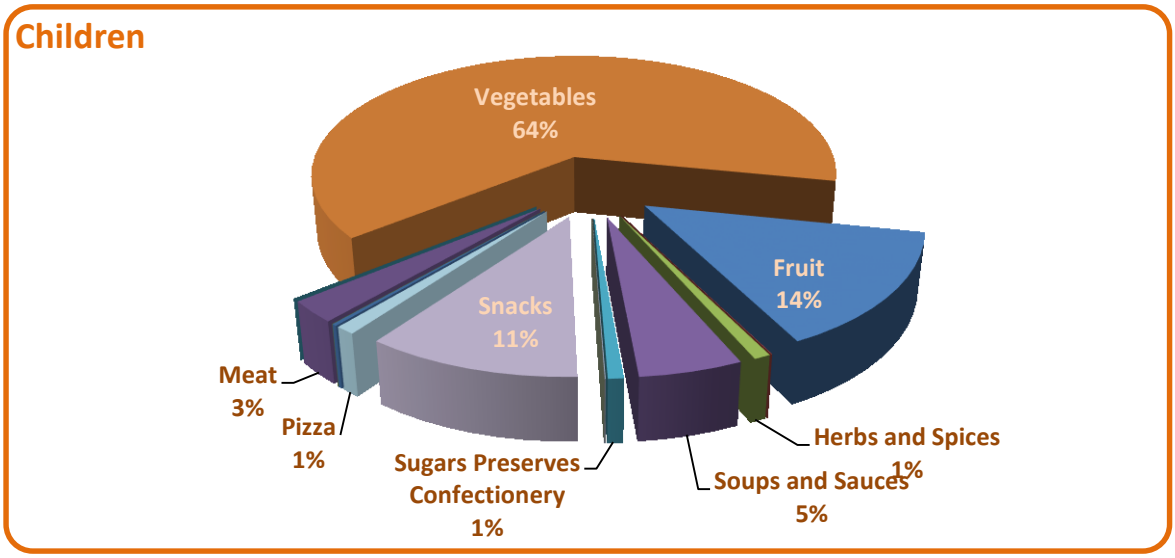
For children, average intake of nitrates from food was estimated to fall between 0.46 - 0.91 mg/kg bw/day. The above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 1.30 - 1.82 mg/kg bw/day.

Nitrates were detected above the LOD in 25% of all samples analysed.

Figure 12 shows the main contributing food groups to dietary nitrate intake, based on LB measurements showing, as anticipated, that for adults, vegetables represent a major contributing source (76% of total intake), followed by fruit (11% of total intake). As nitrate was not detected in water and some of the non-alcoholic beverages, these foodgroups are not depicted in Figure 12. However, they provide a relative higher importance of 11% and 20% respectively when based on UB calculations, with an associated proportionate decrease in the vegetable group contribution. For children, also vegetables represent a major contributing source (64% of total intake), followed by fruit (14% of total intake) and snacks (11%). As for adults, water and some of the non-alcoholic beverages provide a relative higher importance of 9% and 13% respectively when based on UB calculations with according decrease in the vegetable group contribution.

Figure 12. Contribution of the various food groups in which nitrate was detected (LB) as a percentage of total nitrate intake in adults and children



### 3.11.4. Dietary exposure to nitrites

Table 19 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile nitrite exposure of the Irish adult and child populations from those food groups that were analysed in the study. In relation to dietary exposure to nitrite in this TDS, nitrite was only detected in one food, i.e. ham, out of 19 analysed.

**Table 19. Estimated nitrite exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake mg | | | | Daily Intake mg/kg bw | | | | % of SCF ADI (0.06 mg/kg bw/d) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.1 | 0.8 | 0.3 | 1.9 | 0.001 | 0.01 | 0.004 | 0.02 | 1.5% | 17.7% | 6.9% | 39.1% |
| CHILDREN | | | | | | | | | | | |
| Daily Intake mg | | | | Daily Intake mg/kg bw | | | | % of SCF ADI (0.06 mg/kg bw/d) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.05 | 0.5 | 0.2 | 1.0 | 0.002 | 0.02 | 0.01 | 0.04 | 2.6% | 26.7% | 13.4% | 59.7% |

As can be seen from Table 19 for adults, average intake of nitrite from food was estimated to fall between 0.001 - 0.01 mg/kg bw/day. The above average (97.5$^{th}$ percentile) intake was estimated to fall between 0.004 - 0.02 mg/kg bw/day.

For children, the estimated daily mean and 97.5$^{th}$ percentile nitrite intake (in mg/kg bw/day) were estimated to fall between 0.002 - 0.02 mg/kg bw/day and 0.01 - 0.04 mg/kg bw/day, respectively.

### 3.11.5. Risk characterisation

As can be seen from Table 18, for adults the average intake of nitrates from food corresponds to 12.4 - 23.2% of the ADI. The above average (97.5$^{th}$ percentile) intake corresponds to 43.7 - 59.4% of the ADI. For children, the average intake of nitrates from food corresponds to 12.3 - 24.6% of the ADI. The above average (97.5$^{th}$ percentile) daily intake corresponds to 35.3 - 49.1% of the ADI.

With regard to nitrite (see Table 19), for adults, the average intake of nitrite from food corresponds to 1.5 - 17.7% of the ADI. The above average (97.5$^{th}$ percentile) intake corresponds to 6.9 - 39.1% of the ADI. For children, the estimated daily mean and 97.5$^{th}$ percentile nitrite intakes (in mg/kg bw/day) correspond to 2.6 - 26.7 and 13.4 – 59.7% of the ADI, respectively.

In conclusion, exposure to nitrate (from both use as additive and natural occurrence) was below the ADI of 3.7 mg/kg bw in both population groups, with natural occurrence in vegetables being the more important contributor to dietary exposure. Nitrite was only detected in one foodgroup (hams) and exposure estimates based on this finding (LB) as well as estimates taking into account other foodgroups in which nitrite was tested but not detected (UB) were also below the TDI of 0.06 mg/kg bw. Results from this study indicate that exposure to nitrates and nitrites from food is not of concern.

## 3.12. Mycotoxins

### 3.12.1. Sources of exposure to mycotoxins

Mycotoxins are natural chemicals produced by certain fungi which occur as contaminants of some food crops, either in the field or during post-harvest storage. It is estimated that 25% of the world's food crops overall are contaminated by mycotoxins (Bhat, 1999). Considering that these food crops include cereals, nuts, fruit and vegetables which comprise a significant part of the European consumer's diet, there is potentially a significant exposure to mycotoxins. Exposure of consumers to mycotoxins is mainly via plant foods. However, an additional potential exposure may be via foods of animal origin such as milk, cheese and meat, as a result of consumption of contaminated feed by food animals. Available data on the incidence of mycotoxins in various foodstuffs indicate that the situation is very different for different mycotoxins. Aflatoxins occur mainly in commodities imported from the tropics and sub-tropics, in particular pistachio nuts, groundnuts (peanuts), other edible nuts such as Brazil nuts, dried figs, spices and maize, and products derived from these commodities. While individually none of these commodities may be major contributors to the diet, the range of commodities in which the aflatoxins are found means that there is a significant potential for exposure. The consumer may also be exposed indirectly to aflatoxin M1 and M2, the hydroxylated metabolites of aflatoxin B1 and B2, through milk from cows fed aflatoxin-containing feed. The main contributor to the dietary intake of ochratoxin A seems to be cereals and cereal products, but the contaminant has been detected at relatively high levels in dried vine fruits such as raisins and has been also reported in coffee, beer, wine and nuts. Additionally, exposure may occur as a consequence of consumption of meat from pigs fed ochratoxin-containing feed. Human exposure to ochratoxin A has been demonstrated in several European countries in blood and human milk. Patulin is found in a variety of mouldy fruits, vegetables and cereals. Major sources of exposure are products such as juice derived from apples and pears, and exposure of young children, for whom these food items may represent an important component of the diet, is of particular concern. The fusarium toxins such as the trichothecenes, zearalenone and the fumonisins, occur mainly in cereals grown in more moderate climates including Ireland. While these contaminants may be of lower toxicity than the aflatoxins,

ochratoxin A, etc., their occurrence in food commodities that are eaten more widely by consumers means that levels must also be rigorously controlled in food and feed.

### 3.12.2. Health effects of mycotoxins

**EFSA has established a TWI for ochratoxin A of 120 ng/kg bw, a TDI of 0.25 µg/kg bw for zearalenone, a group TDI of 100 ng/kg bw for the sum of T-2 and HT-2 toxins and a TDI of 1.2 µg/kg for nivalenol. JECFA has established a PMTDI for patulin of 0.4 µg/kg bw, a group TDI for the fumonisins of 2 µg/kg bw and a PMTDI of 1 µg/kg bw for deoxinivalenol. Since aflatoxins are human carcinogens, no Tolerable Intakes have been set for this group of mycotoxins.**

Mycotoxin-related illnesses have been recognised for centuries, e.g. 'St. Anthony's Fire' was a recognised disease, caused by eating rye contaminated with ergot alkaloids produced by the fungus *Claviceps purpurea*, as far back as 1,000 AD. Only in the last century were mycotoxins identified as being the causative agents of illness, both in humans and animals. Mycotoxins vary widely in their toxicity and the toxic effects may be both acute (after a single exposure) and chronic (after repeated exposure). The aflatoxins are considered to be the most toxic of the mycotoxins, aflatoxins B1, B2, G1 and G2 being the principal aflatoxins of concern. Long-term low level exposure to aflatoxins has been associated with liver diseases such as cancer, cirrhosis, hepatitis and jaundice in humans and animals and they are regarded both as genotoxic (DNA-damaging) carcinogens and as immunosupressants. Ochratoxin A also has immunosuppressant, teratogenic (reproductive) and carcinogenic effects, and a clear connection has been shown between nephropathy (kidney disease) and exposure to ochratoxin A in humans and animals. Other penicillium mycotoxins such as penicillic acid and citrinin have been found to enhance the toxic effect (synergism) of ochratoxin A on liver and kidney carcinogenesis in animals. Patulin is a potent protein synthesis inhibitor and is also regarded as genotoxic. In animal toxicity studies, the effects observed include reduced weight gain, impaired kidney function and intestinal effects. Citreoviridin is a neurotoxin in animals, resulting in paralysis and muscular atrophy. Trichothecenes at relatively high levels give rise to acute symptoms of vomiting, diarrhoea and allergic reactions in humans. These mycotoxins are also associated with reduced weight gain (failure to thrive) in animals and immune dysfunction. Zearalenone is an oestrogenic substance with relatively low overall toxicity but it has been shown to have uterotrophic (anti-reproductive) effects in pigs. The effects of this mycotoxin in humans are not clearly established. The fumonisins may have neurotoxic effects in some animals, and carcinogenicity in humans has been proposed but not proven. In assessing the toxicity of mycotoxins to humans, a number of considerations are important. The main

issue is the potential carcinogenic effect of a number of the mycotoxins, which is considered to be mediated via a so-called genotoxic (DNA-damaging) mechanism, meaning that in theory no safe level can be established for this effect. For other effects of the mycotoxins, the level of exposure and the period of exposure may affect toxicity. Furthermore, since more than one mycotoxin may be present in a food, additive and/or synergistic effects (where one mycotoxin enhances the toxicity of another) may be important. Also, the immunosuppressant effect of a range of mycotoxins may impact on already immune-compromised individuals. Animals are likely to be exposed to much higher levels of mycotoxins, via contaminated animal feed, and have shown symptoms such as higher mortality, reproductive failures, reduced feed efficiency and reduced productive capacity, e.g. decreased liver weight, milk yield, etc. The toxins can carry through into products from these animals.

### 3.12.3. Dietary exposure to mycotoxins

Aflatoxins, ochratoxin A, patulin and the fusarium toxins were measured in a proportion of the 141 different foods selected for analysis in this TDS, but only in those foods in which they were anticipated to occur, e.g. cereals, nuts, dried fruit, etc. Fusarium toxins were not detected in any of the samples tested however, the respective LODs were relatively high (20 µg/kg for fumonisins, 10 µg/kg for zearalenone and 50 µg/kg for all remaining fusarium toxins), providing unrealistically high upper-bound estimates and are hence, excluded from further analysis.

Table 20 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile for total aflatoxins, ochratoxin A and patulin exposure of the Irish adult and child populations from those food groups that were analysed in the study.

As can be seen from Table 20, for adults the average intake of total aflatoxins (sum of AFB1+AFB2+AFG1+AFG2) was estimated to fall between 0.23 - 10.6 ng/kg bw/day and intake at the 97.5$^{th}$ percentile between 0.78 - 26.9 ng/kg bw/day. For children, the average intake of total aflatoxins was estimated to fall between 0.6 - 6.8 ng/kg bw and intake at the 97.5$^{th}$ percentile between 1.5 - 12.5 ng/kg bw. Of the four aflatoxins tested, only aflatoxin B1 was detected in some of the samples tested (21%), which explains the large span between LB and UB estimates. EFSA estimated an average total aflatoxin exposure of 0.69 ng/kg bw/day (EFSA, 2007) for adults, which is somewhat higher than the LB estimate of 0.23 ng/kg bw/day derived for adults in this study.

Exposure to patulin in the adult population was estimated to fall between 0.01 - 0.02 µg/kg bw/day at the mean and at 0.11 µg/kg bw/day at the 97.5$^{th}$ percentile. For children, the average and above average exposure were estimated to fall between 0.04-0.06 µg/kg bw/day and 0.25-0.28 µg/kg bw/day, respectively.

**Table 20. Estimated intake of total aflatoxins, ochratoxin A and patulin by the Irish adult population from all food groups**

| Estimated total aflatoxins exposure | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ADULTS | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake ng/kg bw | | | | MoE based on $BMDL_{10}$ of 170 ng/kg bw per day | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.02 | 0.8 | 0.06 | 2 | 0.23 | 10.6 | 0.78 | 26.9 | 734 | 16 | 218 | 6 |
| CHILDREN | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake ng/kg bw | | | | MoE based on $BMDL_{10}$ of 170 ng/kg bw per day | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.02 | 0.21 | 0.05 | 0.4 | 0.6 | 6.8 | 1.5 | 12.5 | 303 | 25 | 115 | 14 |

| Estimated patulin exposure | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ADULTS | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % of SCF PMTDI of 0.4 µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.87 | 1.2 | 8.05 | 8.65 | 0.01 | 0.02 | 0.11 | 0.11 | 2.8% | 3.9% | 26.4% | 26.6% |
| CHILDREN | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % of SCF PMTDI of 0.4 µg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 1.28 | 1.85 | 8.15 | 8.99 | 0.04 | 0.06 | 0.25 | 0.28 | 10.8% | 15.5% | 62.7% | 70.9% |

| Estimated ochratoxin A exposure | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| ADULTS | | | | | | | | | | | |
| Daily Intake ng | | | | Daily Intake ng/kg bw (weekly intake in parenthesis) | | | | % of EFSA TWI (120 ng/kg bw/week) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 35 | 236 | 85 | 553 | 0.5 (3.3) | 3.1 (21.8) | 1.2 (8.3) | 7.3 (51) | 2.7% | 18.2% | 6.9% | 42.5% |
| CHILDREN | | | | | | | | | | | |
| Daily Intake ng | | | | Daily Intake ng/kg bw (weekly intake in parenthesis) | | | | % of EFSA TWI (120 ng/kg bw/week) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 36 | 72 | 71 | 133 | 1.2 (8.1) | 2.3 (16.4) | 2.3 (16.0) | 4.7 (33.0) | 6.8% | 13.7% | 13.4% | 27.5% |

Exposure to ochratoxin A in the adult population was estimated to fall between 3.3 - 21.8 ng/kg bw/week. Above average intake was estimated to fall between 8.3 - 51 ng/kg bw/week. For children, the average and above average exposure were estimated to fall between 8.1 – 16.4 ng/kg bw/week and 16 – 33 ng/kg bw/week, respectively.

Mycotoxins were only analysed in foods making up the cereal group, in meat and meat products, eggs, dried fruit, alcoholic and non-alcoholic beverages and some other miscellaneous foods, e.g. confectionery, i.e. foods in which mycotoxins are anticipated to occur. Aflatoxin B1 was found in 21% of 39 foods analysed, including biscuits, fine bakery ware, breakfast cereals, nuts and pizza, while none of the other aflatoxins were detected. Ochratoxin A was detected in 28% of 40 foods analysed, in flour, biscuits, bread, fine bakery ware, breakfast cereals, seeds and pizza. Patulin was only analysed in eight foods (apples, pears, berries, jams, fruit juices and cider) and was detected in pears, cider and fruit juices. Fusarium toxins (fumonisins, trichothecenes, and zearalenone) were not detected in any foods analysed. Regarding aflatoxins and ochratoxin A, cereal-based products were the major source of exposure for both adults and children (>80%) whereas for patulin, the major contributors were alcoholic beverages and non-alcoholic beverages, respectively.

### 3.12.4. Risk characterisation

Exposure to mycotoxins was estimated for aflatoxins, ochratoxin A, patulin and fusarium toxins. Fusarium toxins were not detected in any of the samples tested however, the respective LODs were relatively high (20 µg/kg for fumonisins, 10 µg/kg for zearalenone and 50 µg/kg for all remaining fusarium toxins) and UB estimates as a consequence were unrealistically high and in some cases above the respective health-based guidance values. Therefore, more sensitive methodologies are required for future analysis of fusarium toxins as part of a TDS in order to fully characterise the potential risk from exposure to these toxins.

No health-based guidance value has been established for aflatoxins, and risk characterisation is based on the MoE concept, which is considered appropriate for substances which are both genotoxic and carcinogenic. For adults, using the $BMDL_{10}$ (10% extra cancer risk) value of 170 ng/kg bw per day as the reference point, MoE values falling between 734 - 16 for the mean exposure and between 218 - 6 for the 97.5$^{th}$ percentile exposure were calculated. This finding is in line with the EFSA estimated MoE of 247 for an average total aflatoxin exposure of 0.69 ng/kg bw/day (EFSA, 2007), indicating a potential concern regarding aflatoxin intakes in all regions of the EU, including Ireland.

For children, the MoE values were estimated to fall between 303 - 25 at the mean exposure and between 115 - 14 at the 97.5$^{th}$ percentile exposure. These MoEs are low, taking into account that the aflatoxins are genotoxic carcinogens and are of potential concern.

Estimated exposure to patulin in the adult population corresponds to 2.8 - 3.8% of the PMTDI. Above average intake corresponded to 26.4 - 26.6% of the PMTDI. For children, average and above average exposure corresponded to 10.8 - 15.5% and 62.7 - 70.9% of the PMTDI, respectively.

Estimated exposure to ochratoxin A in the adult population corresponds to 2.7 - 18.2% of the TWI. Above average intake corresponded to 6.9 - 42.5% of the TWI. For children, the average and above average exposure corresponded to 6.8 - 13.7% and 3.4 - 27.5% of the TWI, respectively.

Exposure estimates for ochroatoxin A and patulin were below established health-based guidance values for both population groups and are not of concern.

In conclusion, exposure estimates for ochroatoxin A and patulin are not of concern. More sensitive methodologies are required for future analysis of fusarium toxins in order to fully characterise the potential risk from exposure to these toxins. Regarding aflatoxin, the findings of this study are in line with EFSA's observations, indicating a potential concern regarding aflatoxin intakes in all regions of the EU, including Ireland.

## 3.13. Polycyclic Aromatic Hydrocarbons (PAHs)

### 3.13.1. Sources of exposure to PAHs

PAHs are a class of complex chemicals that are formed and released during incomplete combustion or pyrolysis (burning) of organic matter such as waste or food, during industrial processes and other human activities (WHO, 1998). PAH compounds are emitted from a number of environmental sources, such as processing of coal, crude oil, petroleum, and natural gas, production of aluminium, iron and steel, heating in power plants and residences (oil, gas, charcoal-fired stoves, wood stoves), combustion of refuse, fires including wood fires, motor vehicle exhaust and used motor lubricating oil (WHO, 1998). Soils, surface waters, precipitations and sediments may be contaminated by PAHs due to atmospheric fallout, urban runoff, deposition from sewage, and certain wastes, such as oil or gasoline spills, and there is potential for food crops to become contaminated as a result. For the general population, the major routes of exposure to PAHs are from food and inhaled air (WHO, 1998; EFSA, 2008c).

In food, PAHs may be formed during processing and domestic food preparation, such as barbecuing, smoking, drying, roasting, baking, frying or grilling (EFSA, 2008c). Direct fire-drying and heating processes used during the production of some oils of plant origin and in particular olive pomace oil (oil extracted from olive pulp after the first press) can result in high levels of PAHs. Effective refining of olive pomace oils can remove PAHs to ensure that the products are safe. However, alternative methods that avoid the initial formation of PAHs should be used wherever possible. Vegetables may be contaminated by the deposition of airborne particles or by growth in contaminated soil. Raw meat, milk, poultry and eggs will normally not contain high levels of PAHs due to rapid metabolism of these compounds in the species of origin. However, some marine organisms, such as bivalve molluscs, e.g. mussels, oysters, are known to absorb and accumulate PAHs from water. In 2008, EFSA calculated dietary exposure to PAHs for average and high consumers based on data supplied by 17 European countries including Ireland (EFSA, 2008c). Exposure varied between 235 ng/day and 389 ng/day for average and high consumers respectively for benzo[a]pyrene alone, rising to 1,729 ng/day and 3,078 ng/day respectively for the sum of eight of the most critical PAHs. The two highest contributors to the dietary exposure were found to be cereals and cereal products, and seafood and seafood products, it should be noted however, that little data were available on goods with potentially high PAH content such as barbecued and roast meat products (EFSA, 2008c).

### 3.13.2. Health effects of PAHs

**Since PAHs are considered to be probable human carcinogens, no regulatory bodies have established TDI or TWIs for these contaminants in food. EFSA derived a $BMDL_{10}$ of 0.34 mg/kg bw per day for PAH4 as a marker for the carcinogenic PAHs in food.**

Although studies in experimental animals on individual PAHs, mainly on benzo[a]pyrene, have shown various toxicological effects, such as haematological effects, reproductive and developmental toxicity and immunotoxicity, it is the carcinogenic and genotoxic (DNA-damaging) potential of these compounds that has attracted most attention. A number of PAHs have shown carcinogenicity in experimental animals and genotoxicity and mutagenicity in vitro and in vivo. In 2012, IARC concluded that benzo[a]pyrene is a human carcinogen (IARC, 2012b). Some other PAHs have also been identified as being carcinogens, with possible genotoxic properties. Although the PAHs are lipophilic chemicals like the dioxins and PCBs, they are metabolised or broken down faster than the latter chemicals, both in the human body and in the environment, and thus persistence for long periods is not such a major problem.

In 2002, the SCF carried out a risk assessment on 33 PAHs originally evaluated by the International Programme on Chemical Safety (IPCS) in 1998 (SCF, 2002b). They considered on the basis of the available toxicological information that benzo[a]pyrene could be used as a marker of the occurrence and effect of the carcinogenic PAHs in food and that 15 out of the 33 PAHs evaluated showed clear evidence of mutagenicity/genotoxicity in somatic cells in experimental animals in vivo (SCF, 2002b). With the exception of benzo[ghi]perylene, they have also shown clear carcinogenic effects in various types of bioassays in experimental animals. Although only benzo[a]pyrene has been adequately tested using dietary administration, in the opinion of the SCF these compounds should be regarded as potentially genotoxic and carcinogenic to humans. The SCF recommended that, in view of the non-threshold effects of these genotoxic substances, the levels of PAHs in foods should be reduced to as low as reasonably achievable (the ALARA principle) (SCF, 2002). JECFA also concluded that PAHs are clearly genotoxic and carcinogenic (JECFA, 2005). Except for benzo[ghi]perylene and cyclopenta[cd]pyrene, the PAHs of concern were the same as those stated by SCF. JECFA indicated that benzo[a]pyrene could be used as a marker of exposure to PAHs. They applied an MoE approach to assessing the possible risk of PAHs in food and concluded that estimated intakes of PAHs, based on available exposure data, were of low concern for human health. A similar approach was applied in

2008 by the EFSA CONTAM Panel to assess the possible risk of PAHs in food. However, The CONTAM Panel was of the opinion that benzo[a]pyrene is not a suitable indicator for the occurrence of PAHs in food and concluded that, based on the currently available data relating to occurrence and toxicity, the sum of four PAHs (PAH4: benzo[a]pyrene, chrysene, benz[a]anthracene and benzo[b]fluoranthene) were the most suitable indicators of PAHs in food (EFSA, 2008c).

### 3.13.3. Dietary exposure to PAHs

In accordance with the EFSA opinion (EFSA, 2008c), the four PAHs deemed indicative for PAH presence were included in the study. Table 21 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile PAH4 exposure of the Irish adult and child populations from those food groups that were analysed in the study.

**Table 21. Estimated PAH 4 SUM (LB - UB) exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | MoE based on $BMDL_{10}$ of 0.34 mg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.08 | 0.31 | 0.17 | 0.52 | 0.001 | 0.0041 | 0.002 | 0.0075 | 326,393 | 82,330 | 39,788 | 45,045 |
| **CHILDREN** | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | MoE based on $BMDL_{10}$ of 0.34 mg/kg bw | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.07 | 0.17 | 0.12 | 0.26 | 0.002 | 0.01 | 0.004 | 0.01 | 59,671 | 63,199 | 91,460 | 37,775 |

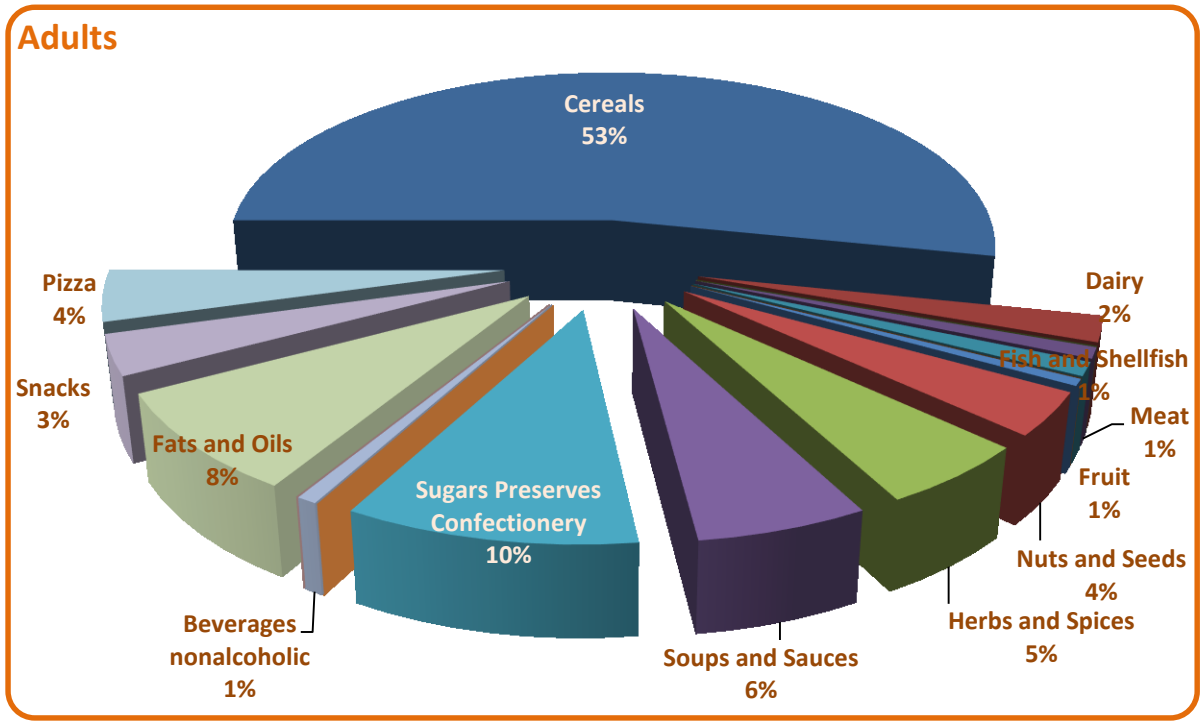
As can be seen from Table 21, for adults the average intake of PAH4 from food was estimated to fall between 0.001 - 0.0041 µg/kg bw/day, while the above average (97.5$^{th}$ percentile) daily intake was estimated to fall between 0.002 - 0.0075 µg/kg bw/day. These intake estimates are somewhat lower than results obtained in the previous TDS (FSAI, 2011) and are also lower than those calculated by EFSA for average and high consumers (EFSA, 2008c), which provided exposure estimates of between 1.17 µg/day and 2.07 µg/day for average and high consumers respectively for the sum of PAH4.

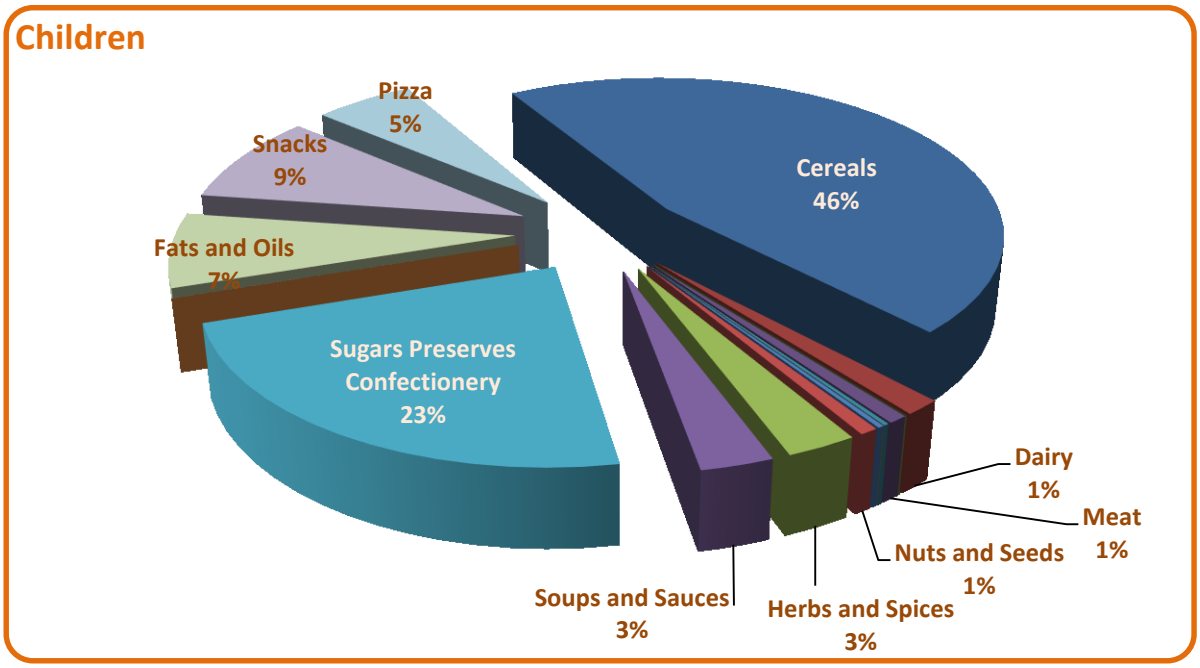
For children, theaverage intake of PAH4 from food was estimated to fall between 0.002 - 0.01 µg/kg bw/day, while for above-average (97.5$^{th}$ percentile) consumers the intakes were estimated to fall between 0.004 - 0.01 µg/kg bw/day. These intakes estimates are also lower than those calculated by EFSA for average and high consumers (EFSA, 2008c), which provided exposure estimates of between 1.17 µg/day and 2.07 µg/day for average and high consumers respectively for the sum of PAH4.

PAHs were analysed in all 141 samples, with varying detection rates of individual PAH congeners. Only in three samples, were all four PAHs detected.

Figure 13 shows that for adults, the main contributing food group to dietary PAH intake, based on LB measurements, was cereals (53%). Confectionery (10%), fats and oils (8%), soups and sauces (6%), herbs and spices (5%) were the next highest contributors, with other foods contributing 4% or less. For children, cereals (46%) were also were the major contributing source of PAHs. Confectionery (23%), snacks (9%), fats and oils (7%), pizza (5%) were the next highest contributors, with other foods contributing 4% or less.

Figure 13. Contribution of the various food groups in which PAH4 was detected (LB) as a percentage of total PAH4 intake in adults and children



### 3.13.4. Risk characterisation

Exposure to PAHs was evaluated using the sum of four PAHs (benzo[a]pyrene, chrysene, benz[a]anthracene and benzo[b]fluoranthene) as indicators of PAHs in food. No health based guidance value has been established for PAHs, and the sum of PAH4 was used to derive MoEs based on the bench mark dose lower confidence limit for a 10% increase in the number of tumour bearing animals compared to control animals ($BMDL_{10}$). EFSA derived a $BMDL_{10}$ of 0.34 mg/kg bw per day for PAH4 as a marker for the carcinogenic PAHs in food.

The EFSA Scientific Committee has concluded that for substances that are both genotoxic and carcinogenic, an MoE of 10,000 or higher based on a $BMDL_{10}$ from an animal study, is of low concern from a public health point of view.

For both population groups, the calculated MoEs (see Table 21) were sufficiently high (>10,000) to conclude that there is low concern for human health.

## 3.14. Bisphenol A (BPA)

### 3.14.1. Sources of exposure to BPA

Bisphenol A (BPA) is an organic chemical used as a building block or monomer in the synthesis of polycarbonate plastics, which are widely used in many household items such as plastic bottles, tableware (plates, mugs, plastic utensils etc.), storage containers, plastic furniture, compact disks (CDs), etc. BPA is also a component of epoxy resins, used as protective coatings and linings for food and beverage cans and vats, in dental materials, as well as many other uses. Another widespread use of BPA is in thermal paper commonly used in till/cash register receipts. Residues of BPA can migrate into food and beverages and be ingested by the consumer and can be absorbed through the skin and by inhalation from other sources including thermal paper, cosmetics and dust.

BPA is regulated as a food contact material. It was first evaluated for this use over 25 years ago by the SCF and since that time, the safety of BPA has been assessed by a number of national and international organisations. In January 2015, EFSA published a comprehensive re-evaluation having considered hundreds of scientific publications in peer-reviewed scientific journals as well as reports from studies submitted by industry.

EFSA reported that diet is the main source of exposure to BPA in all population groups. Specifically, canned food and non-canned meat and meat products are the two main dietary contributors to external BPA exposure in the large majority of European countries and age classes.

Among the European population older than six months, infants (6 - 12 months) and toddlers had the highest estimated external average (0.375 µg/kg bw per day) and high (0.857 µg/kg bw per day) dietary exposure. This was mainly due to their higher consumption of foods and beverages per kilogram body weight. The modelled dietary exposure for European adolescents, adults (including women of childbearing age) and elderly/very elderly ranged from 0.116 to 0.159 µg/kg bw per day for the average external exposure and from 0.335 to 0.388 µg/kg bw per day for the high exposure, respectively (EFSA, 2015).

### 3.14.2. Health effects of BPA

**EFSA has set a temporary tolerable daily intake (t-TDI) of 4 µg/kg of bw/day.**

BPA is known to have oestrogen-like properties and has been characterised as an endocrine-active substance or endocrine-disruptor and has become one of the most studied substances. After weighing up a significant body of new scientific information on its toxic effects, EFSA concluded that high doses of BPA are likely to adversely affect the kidney and liver. Uncertainties surrounding potential health effects of BPA on the mammary gland, reproductive, metabolic, neurobehavioural and immune systems have been quantified and factored in to the calculation of a safe level by EFSA, which is temporary pending the outcome of a long-term study in rats conducted by the US NTP, which will help to reduce the uncertainties about BPA's toxic effects.

The safety of BPA has been repeatedly and comprehensively examined by a number of national and international organisations, including the SCF, the European Union in the context of Council Regulation (EEC) No 793/93 on the evaluation and control of existing substances, the FDA, Health Canada, the French Agency for Food, Environmental and Occupational Health and Safety (ANSES), the WHO and EFSA, the European Union's risk assessment body. In January 2015, EFSA published a comprehensive re-evaluation having considered hundreds of scientific publications in peer-reviewed scientific journals as well as reports from studies submitted by industry and concluded that BPA poses no health risk to consumers of any age group (including unborn children, infants and adolescents) at current exposure levels.

### 3.14.3. Dietary exposure to BPA

Table 22 presents the estimated LB and UB daily mean and 97.5$^{th}$ percentile BPA exposure of the Irish adult and child populations from those food groups that were analysed in the study. However, only those foods that were anticipated to contain BPA, e.g. cereals and cereal products, meat products, potatoes/potato products and specific vegetables, beers, spirits, coffee, tea and savoury snacks were analysed, thus the intakes given in Table 22 are only derived from these foods. They are however, anticipated to be the major contributors of BPA in the diet, based on the published literature.

**Table 22. Estimated BPA exposure of the Irish adult and child populations from all food groups**

| ADULTS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % of EFSA t-TDI (4 µg/kg bw/d) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 2.0 | 6.7 | 6.0 | 19.3 | 0.03 | 0.09 | 0.09 | 0.24 | 0.7% | 2.2% | 2.2% | 6.0% |
| **CHILDREN** | | | | | | | | | | | |
| Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % of EFSA t-TDI (4 µg/kg bw/d) | | | |
| Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| 0.8 | 2.4 | 3.1 | 5.7 | 0.02 | 0.08 | 0.10 | 0.17 | 0.6% | 1.9% | 2.6% | 4.3% |

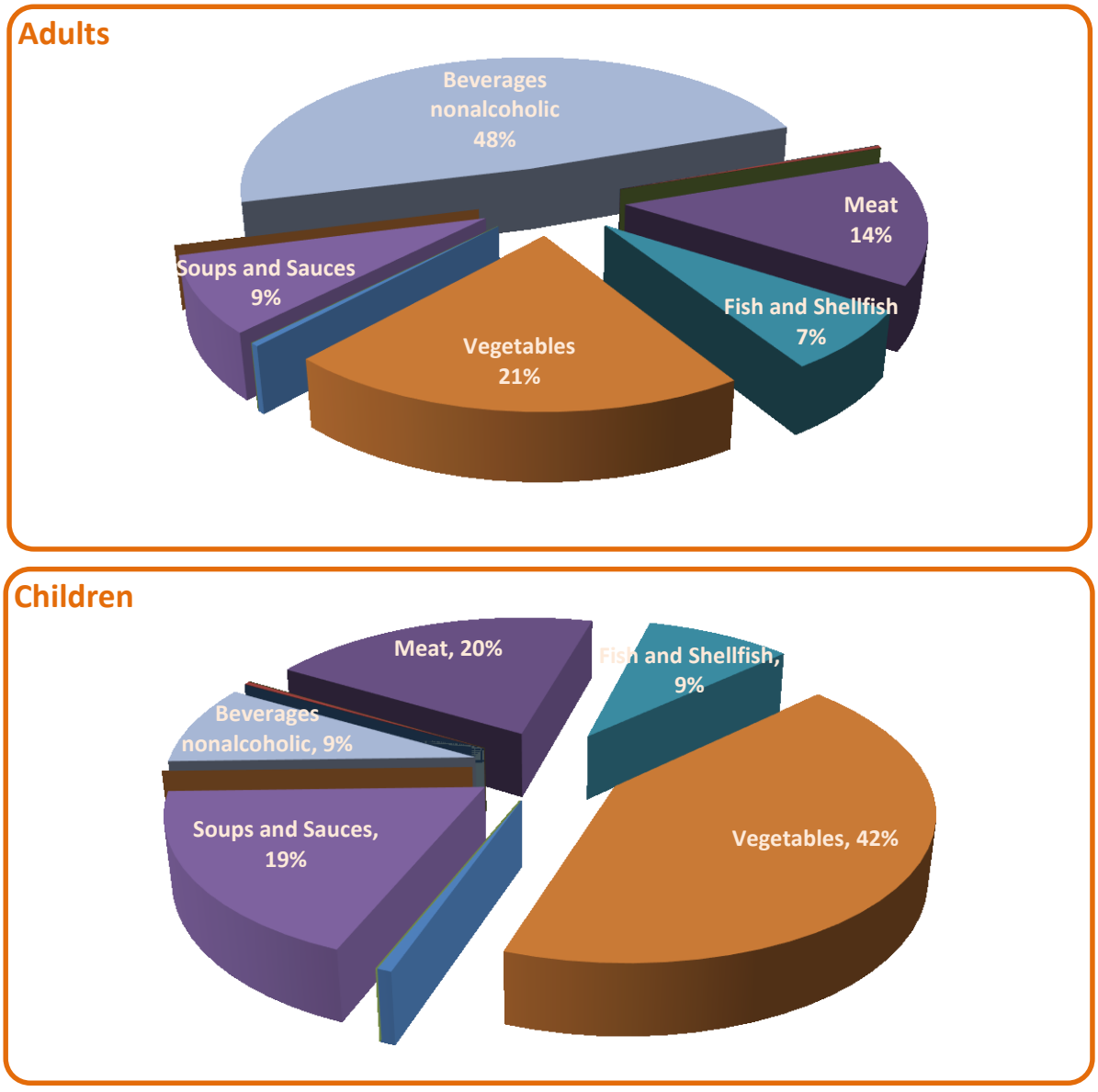
As can be seen from Table 22, for adults, average intake of BPA from food was estimated to fall between 0.03 - 0.09 µg/kg bw/day. The intakes for above average consumers were 0.09 - 0.24 µg/kg bw/day. These results are in line with exposure estimates derived by EFSA (EFSA, 2015).

For children, the average intake of BPA from food was estimated to fall between 0.02 - 0.08 µg/kg bw/day. The intakes for above average consumers were 0.1 - 0.17 µg/kg bw/day.

BPA was detected in 30% of all samples analysed.

Figure 14 shows the main food groups contributing to dietary BPA exposure revealing that, for adults, non-alcoholic beverages, i.e. tea (48%), vegetables (21%) and meat (14%) are the major contributing sources. For children, vegetables (42%), meat (20%) and soups and sauces (19%) were the major contributing sources.

Figure 14. Contribution of the various food groups in which BPA was detected (LB) as a percentage of total BPA intake in adults and children



### 3.14.4. Risk characterisation

As can be seen from Table 22, for adults, average intake of BPA from food corresponds to 0.7 - 2.2% of the t-TDI. The intakes for above average consumers correspond to 2.2 - 6% of the t-TDI. For children, average and above average intake of BPA from food correspond to 0.6 - 1.9% and 2.6 - 4.3% of the t-TDI, respectively.

In conclusion, exposure to BPA was estimated to be low in both population groups and was well below the t-TDI of 4 µg/kg bw/d. The findings are in line with estimates derived by EFSA (EFSA, 2015) and indicate that exposure to BPA from food is of low concern.

## 3.15. Phthalates

### 3.15.1. Sources of exposure to phthalates

Phthalates are di-esters of phthalic acid (1,2-benzenedicarboxylic acid) and are man-made high production volume chemicals used primarily as plasticisers in polyvinylchloride (PVC) products (Hartmann *et al*, 2015; US EPA, 2012) however, phthalates are also used in other products such as paints and glues (Danish EPA, 2013). Some phthalates are used as solvents (dissolving agents) for other materials. They are used in hundreds of products, such as vinyl flooring, adhesives, detergents, lubricating oils, automotive plastics, plastic clothes (raincoats), and personal-care products (soaps, shampoos, hair sprays, and nail polishes) (CDC, 2009).

There are many different types of phthalates and there are indications that they do not all have the same effects on the environment and human health. Phthalates can be divided into high- and low-molecular-weight phthalates. High-molecular-weight phthalates are often defined as phthalates with a carbon backbone in the main alkyl chain consisting of seven or more carbon atoms. These include, e.g. the phthalates di-isononylphthalate (DINP), diisodecylphthalate (DIDP), di(2-propylheptyl) phthalate (DPHP), diundecyl phthalate (DIUP) and ditridecyl phthalate (DTDP). Low-molecular-weight phthalates are often defined as phthalates with a carbon backbone in the main alkyl chain of three to six carbon atoms. These include, e.g. the phthalates di(2-ethylhexyl)phthalate (DEHP), di - butylphthalate (DBP), diisobutyl phthalate (DIBP) and butyl benzyl phthalate (BBP) (Danish EPA, 2013). Phthalates may be present in food due to their widespread presence as environmental contaminants or due to migration from food contact materials. When incorporated into PVC, phthalates are not covalently bound and therefore, are easily released into the environment, leading to animal and human exposure (COT, 2009).

The use of certain phthalates as food contact materials is regulated in the EU via Regulation (EU) No 10/2011 on plastic materials and articles intended to come into contact with food (as amended) (EC, 2011). Restrictions have been imposed on the use of di-butylphthalate (DBP), di(2-ethylhexyl)phthalate (DEHP), butylbenzylphthalate (BBP), di-isononylphthalate (DINP) and diisodecylphthalate (DIDP). The restrictions specify the permitted scope of use and specific migration limit for each compound (EC, 2011).

### 3.15.2. Health effects of phthalates

**EFSA has set TDIs for DBP of 0.01 mg/kg bw, for DEHP of 0.05 mg/kg bw, for BBP of of 0.5 mg/kg bw , for DINP 0.15 mg/kg bw and DIDP at 0.15 mg/kg bw.**

Since the mid-1990s, phthalates have been the object of great attention globally due to their suspected negative effects on the environment and reproduction, as well as their suspected carcinogenic effect. In recent years, their potential endocrine disrupting effects have been the centre of attention (Danish EPA, 2013).

Five phthalates (DEHP, DBP, BBP, DINP and DIDP) have undergone risk assessments by the EU, and these phthalates have been relatively thoroughly investigated for their effects on the environment and human health as well as their use in different types of products.

Most recently in 2005, the EFSA Scientific Panel on Food Additives, Flavourings, Processing Aids and Materials in Contact with Food (AFC) re-evaluated DBP, BBP, DEHP, DINP and DIDP for use in the manufacture of food contact materials. For DBP, DEHP and BBP, the EFSA Panel concluded that effects on reproduction and development were the most sensitive end-points on which to base its risk assessment and set TDIs for DBP of 0.01 mg/kg bw, for DEHP of 0.05 mg/kg bw/day and for BBP of of 0.5 mg/kg bw, respectively (EFSA, 2005a, 2005b, 2005c). For DINP and DIDP, the EFSA Panel concluded that effects on liver, reproduction and development were the endpoints upon which to base its risk assessment and set TDIs for each DINP and DIDP at 0.15 mg/kg bw (EFSA, 2005d, EFSA, 2005e). The latter five phthalates DBP, BBP, DEHP, DINP and DIDP assessed by EFSA have been included in this TDS. Also included was DIBP (di-isobutyl phthalate), however no health-based guidance value has been established for this phthalate by EFSA to date. In 2011, the Danish EPA drafted a Restriction Report on risks from combined exposures to four phthalate esters (DEHP, DBP, DIBP and BBP) and derived a no effect level (DNEL) of 1.25 mg/kg bw based on NOAEL of 125 mg/kg bw for anti-androgenic effects in developmental studies (Danish EPA, 2011). This report was reviewed by the UK COT, who agreed with this reference value (COT, 2011). Therefore, in absence of an EFSA or

international health-based guidance value, the levels for DIBP found in this study are compared against the Danish DNEL.

### 3.15.3. Dietary exposure to phthalates

Table 23 presents the estimated daily mean and 97.5$^{th}$ percentile intake (in µg/day) of the six phthalates measured for the Irish adult and child populations from all food groups.

**Table 23. Estimated intake of phthalates of the Irish adult population from all food groups**

| Adult population | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % of EFSA TDI* | | | |
| | Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| BBP | 2.4 | 18.3 | 8.4 | 35.2 | 0.03 | 0.25 | 0.12 | 0.51 | 0.01% | 0.05% | 0.02% | 0.1% |
| DBP | 5.8 | 30.0 | 32.9 | 66.5 | 0.08 | 0.40 | 0.45 | 0.95 | 0.8% | 4.0% | 4.5% | 9.5% |
| DEHP | 18.7 | 48.1 | 45.2 | 86.5 | 0.25 | 0.64 | 0.64 | 1.20 | 0.5% | 1.3% | 1.3% | 2.4% |
| DIBP | 2.5 | 33.1 | 17.9 | 77.0 | 0.03 | 0.44 | 0.25 | 1.07 | 0.003% | 0.04% | 0.02% | 0.09% |
| DIDP | 2.1 | 165.5 | 19.4 | 314.3 | 0.03 | 2.20 | 0.25 | 4.17 | 0.0% | 1.5% | 0.2% | 2.8% |
| DINP | 77.0 | 209.3 | 553.1 | 725.4 | 1.02 | 2.78 | 7.06 | 8.81 | 0.7% | 1.9% | 4.7% | 5.9% |
| **Children population** | | | | | | | | | | | | |
| | Daily Intake µg | | | | Daily Intake µg/kg bw | | | | % of EFSA TDI* | | | |
| | Mean | | P97.5 | | Mean | | P97.5 | | Mean | | P97.5 | |
| | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB | LB | UB |
| BBP | 1.13 | 6.74 | 3.35 | 12.0 | 0.04 | 0.22 | 0.12 | 0.38 | 0.01% | 0.04% | 0.02% | 0.08% |
| DBP | 0.69 | 9.41 | 2.04 | 15.6 | 0.02 | 0.30 | 0.07 | 0.52 | 0.22% | 3.03% | 0.71% | 5.25% |
| DEHP | 11.6 | 24.3 | 24.5 | 40.7 | 0.37 | 0.79 | 0.82 | 1.45 | 0.75% | 1.58% | 1.63% | 2.90% |
| DIBP | 1.55 | 10.52 | 3.97 | 18.9 | 0.05 | 0.34 | 0.13 | 0.58 | 0.004% | 0.03% | 0.01% | 0.05% |
| DIDP | 0.50 | 122.8 | 3.89 | 216.4 | 0.02 | 4.01 | 0.11 | 7.39 | 0.01% | 2.68% | 0.08% | 4.92% |
| DINP | 74.7 | 173.3 | 343.7 | 457.1 | 2.36 | 5.59 | 11.22 | 14.93 | 1.57% | 3.73% | 7.48% | 9.95% |

* DBP 0.01 mg/kg bw, DEHP 0.05 mg/kg bw/day, for BBP 0.5 mg/kg, DINP 0.15 mg/kg bw, DIDP at 0.15 mg/kg bw, DIBP 1.25 mg/kg bw which is based on a DNEL derived by the Danish EPA (2011)

As can be seen from Table 23, in adults the average intake of individual phthalates from food was estimated to fall between 0.03 - 2.78 µg/kg bw/day; the intakes for above average consumers were 0.12 - 8.8 µg/kg bw/day. For children, the average exposure was estimated to fall between 0.5 - 173.3 µg/kg bw/day and above average exposure between 2.04 - 457.1 µg/kg bw/day. These results are in line with exposure estimates derived by the UK FSA for BBP, DBP, DEHP and DIBP, in a TDS conducted in 2007 (FSA, 2007).

The individual phthalates included in this study were detected in 3 - 22% of samples analysed (DEHP (22%), BBP (15%), DIBP (11%), DINP (8%), DBP (7%), DIDP (3%)) however, results obtained could not always be confirmed. In some cases, a response was observed in the GC-MS chromatograms but the confirmation criteria were not met and as such, it could not be confirmed unequivocally that the response was due to the presence of a phthalate diester. Where a phthalate was detected but not confirmed, it was considered a detect in order to calculate exposures for this TDS and hence, presents an indication of exposure and is likely to be an over-estimation. For this reason, contribution of individual foodgroups to total dietary exposure is not displayed.

### 3.15.4. Risk characterisation

As can be seen from Table 23, exposure to phthalates was estimated to be low in both population groups and average as well as above average exposure to phthalates was found to be well below the respective TDIs set by EFSA. These results are in line with exposure estimates derived by the UK FSA in a TDS conducted in 2007 (FSA, 2007), and are of low concern.

## 3.16. Pesticides

As indicated in the methodological section, all 141 foods were analysed for pesticide residues using multi-residue screens capable of detecting up to 492 pesticides (matrix dependent) (see Annex I). Out of a theoretically maximum possible 55,000 pesticide in food occurrences, only 91 were found. In total, 44 different pesticide residues were detected across 41 samples, all at very low levels and with the exception of two, all were below the legislative Maximum Residue Limit (MRL) set for the respective pesticide/food occurrence at that time. The two observed exceedances were minimal and deemed not of health concern. The very limited occurrence of pesticide residues indicate that the exposure of the Irish population to pesticides in their diet is extremely low, a finding that is supported by a previously conducted exposure assessment of Irish adults and children to selected pesticides undertaken by Connolly *et al.* (2009).

## 4. CONCLUSION

This TDS has provided estimates of dietary exposures (mean and 97.5$^{th}$ percentile) of a representative population of Irish adults (n=1,500, males and females) and Irish children (n=594) to a number of common chemical contaminants, food additives and nutrients that are or may be present in Irish food. The chemicals selected for analysis in food in this study were the contaminant metals: aluminium, arsenic (total and inorganic), cadmium, chromium, lead, mercury and tin, the essential nutrients: iodine and selenium, the food additives: nitrates and nitrites, the food contaminants: acrylamide, mycotoxins, polycyclic aromatic hydrocarbons and the food contact materials: phthalates and Bisphenol A. Pesticide residues were also analysed. The exposure estimates were compared with exposure estimates derived by EFSA or available data from other countries in order to assess whether levels of exposure for Irish consumers are comparable to those found in other countries. The results have also been compared with health-based guidance values derived by EFSA, SCF or JECFA where available, enabling a conclusion to be made regarding any risk to consumers of these foods.

It should be noted that due to the size and structure of this (and any) TDS study, there are certain limitations to the data generated which must be borne in mind in its interpretation:

- Not all foods consumed can be analysed and certain assumptions and extrapolations have to be made
- Analytical sensitivities have to be taken into account and resulting non detected or non-quantifiable values treated accordingly
- Variability in contaminant occurrence cannot be taken into account due to the survey size and estimates are based on single or mean occurrence data and provide a snapshot only

Overall, the outcome of this analysis showed that the Irish population is generally not at risk from intakes from food of the majority of the chemicals analysed in the study. Potential concern was identified for exposure to acrylamide, aflatoxins and to a lesser degree, for lead. These findings are not specific to Ireland, rather they are of concern worldwide and continuous efforts are being made by risk managers to reduce exposure to these substances to as low as reasonably achievable, bearing in mind that zero exposure is impossible.

Where EFSA has identified potential risks to consumers in Europe, the European Commission has implemented risk management actions, such as the reduction of maximum limits for aluminium containing food additives to reduce long term exposure of the population in Europe. To mitigate the risks of exposure to arsenic, the European Commission introduced maximum legislative limits for

inorganic arsenic in rice and rice-based products in tandem with a monitoring recommendation covering a wider range of foods to examine the need for further management actions.

For cadmium, an EU monitoring recommendation on the investigation and/or introduction of mitigation strategies to reduce dietary exposure to cadmium was implemented by DAFM, in collaboration with Teagasc and Bord Bia. Offal from older animals is also a potential source of cadmium for consumers and has consequently been addressed through introduction of official controls.

With regard to lead, in 2015[3], the European Commission undertook measures to reduce the dietary exposure to lead in food by lowering existing maximum levels and setting additional maximum levels for lead in relevant commodities.

In order to reduce the mercury levels in the environment and the consequent human exposure, the European Commission launched the EU Mercury Strategy in 2005. It is a comprehensive plan that includes 20 measures to reduce mercury emissions to reduce the supply and demand for mercury and to protect against exposure. In 2010, the European Commission reviewed the mercury strategy and concluded that the implementation of the strategy is in an advanced stage and almost all actions are delivered. The implementation of these policies is expected to reduce the emissions, although data are not yet available.

The major highlights of the Minamata Convention on Mercury in 2013, an international treaty ratified by delegates from 140 countries, include a ban on new mercury mines, the phase-out of existing ones, control measures on air emissions, and the international regulation of the informal sector for artisanal and small-scale gold mining. This international agreement is anticipated to cause further reduction in mercury levels in the environment over time, thus meeting the objective of protecting human health and the environment. This convention is due for ratification by the EU in 2015.

Maximum legislative limits for mercury in fish are also currently under review in Europe.

While this TDS has found that the risk from dietary exposure to the chemicals under consideration is low for the general population, specific advice to certain sub-population groups is warranted.

[3] Commission Regulation (EU) 2015/1005 of 25 June 2015 amending Regulation (EC) No 1881/2006 as regards maximum levels of lead in certain foodstuffs

The FSAI has thus far provided advice on fish consumption for children, pregnant women and women of reproductive age with regard to mercury exposure and consumption advice relating to arsenic in Hijiki seaweed and rice-based infant formula.

While these results are not of immediate concern, the FSAI reiterates that continued surveillance of the Irish food supply for contaminants, residues, food additives and essential nutrients by food business operators and by other competent bodies including environmental health professionals and public analysts, is essential in order to ensure the continuing safety of Irish food.

Global trade in food necessitates harmonised control and risk management actions at European wide level to reduce exposure of the European population to contaminants and pesticide residues. This is realised via harmonised European Commission food contaminants and residues legislation within Europe. Ireland participates in all relevant EU Expert Working Groups and provides food consumption and occurrence data to EFSA to ensure that the safety of Irish consumers is accounted for.

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## ANNEX I: ANALYTICAL METHODOLOGY

*Pesticide Analysis*

Pesticide Multi-screens analysed via either GC-MSMS, LC-MSMS at stated reporting limits (RL)

| Pesticide Multi-screen: Fruit and Vegetables, Cereals and Honey Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC |
| 1-Naphthyl acetamide | 0.01 | LC | Dichlofluanid | 0.01 | GC | HCH-delta | 0.01 | GC | Phorate | 0.01 | LC |
| 2,4,5-T | 0.01 | LC | Dichlorprop-P | 0.01 | LC | Heptachlor | 0.01 | GC | Phorate sulfoxide | 0.01 | LC |
| 2,4,6-Trichlorophenol | 0.01 | GC | Dichlorvos | 0.01 | GC | Heptachlor-endo-epoxide | 0.01 | GC | Phosalone | 0.01 | LC |
| 2,4-D | 0.02 | LC | Diclobutrazol | 0.01 | LC | Heptachlor-exo-epoxide | 0.01 | GC | Phosmet | 0.01 | GC |
| 2,4-DB | 0.05 | LC | Dicloran | 0.01 | GC | Heptenophos | 0.01 | LC | Phosmet-oxon | 0.01 | LC |
| 3,5-Dichloroaniline | 0.01 | GC | Dicofol | 0.01 | GC | Hexachlorobenzene | 0.01 | GC | Phosphamidon | 0.01 | GC |
| 3-Chloroaniline | 0.01 | GC | Dicrothophos | 0.01 | LC | Hexaconazole | 0.01 | GC | Phoxim | 0.01 | LC |
| 4,4-Dichlorobenzophenone | 0.01 | GC | Dieldrin | 0.01 | GC | Hexaflumuron | 0.01 | LC | Picloram | 0.01 | LC |
| Abamectin | 0.01 | LC | Diethofencarb | 0.01 | LC | Hexythiazox | 0.01 | LC | Picoxystrobin | 0.01 | LC |
| Acephate | 0.01 | LC | Difenoconazole | 0.01 | LC | Imazalil | 0.01 | LC | Piperonyl butoxide | 0.01 | LC |
| Acephate | 0.01 | GC | Diflubenzuron | 0.01 | LC | Imazamox | 0.01 | LC | Pirimicarb | 0.01 | GC |
| Acetamiprid | 0.01 | LC | Dimethenamid | 0.01 | LC | Imazaquin | 0.01 | LC | Pirimicarb desmethyl | 0.01 | GC |
| Acetochlor | 0.01 | LC | Dimethoate | 0.01 | GC | Imazethapyr | 0.01 | LC | Pirimifos-ethyl | 0.01 | LC |
| Acibenzolar-S-methyl | 0.01 | LC | Dimethomorph | 0.01 | LC | Imidacloprid | 0.01 | LC | Pirimifos-methyl | 0.01 | LC |
| Aclonifen | 0.01 | GC | Dimoxystrobin | 0.01 | GC | Indoxacarb | 0.01 | LC | pp DDD | 0.01 | GC |
| Acrinathrin | 0.01 | GC | Diniconazole | 0.01 | LC | Iodofenphos | 0.01 | GC | pp DDE | 0.01 | GC |
| Alachlor | 0.01 | GC | Dinitramine | 0.01 | LC | Iodosulfuron-methyl-sodium | 0.01 | LC | pp DDT | 0.01 | GC |
| Aldicarb | 0.02 | LC | Dinoseb | 0.02 | LC | Ioxynil | 0.01 | LC | Prochloraz | 0.01 | LC |
| Aldicarb sulfone | 0.01 | LC | Dinoterb | 0.02 | LC | Iprodione | 0.01 | GC | Procymidone | 0.01 | GC |
| Aldicarb-sulfoxide | 0.02 | LC | Dioxacarb | 0.01 | LC | Iprovalicarb | 0.01 | GC | Profenophos | 0.01 | GC |
| Aldrin | 0.01 | GC | Diphenamid | 0.01 | LC | Isazofos | 0.01 | GC | Promecarb | 0.01 | LC |
| Ametryn | 0.01 | LC | Diphenylamine | 0.01 | GC | Isocarbofos | 0.01 | LC | Prometon | 0.01 | LC |
| Amidosulfuron | 0.01 | LC | Ditalimfos | 0.01 | LC | Isodrin | 0.01 | GC | Prometryn | 0.01 | LC |
| Aminocarb | 0.01 | LC | Diuron | 0.01 | LC | Isofenphos | 0.02 | LC | Propachlor | 0.01 | LC |
| Anthraquinone | 0.01 | GC | DMSA | 0.02 | LC | Isofenphos | 0.01 | GC | Propamocarb free base | 0.01 | LC |
| Asulam | 0.02 | LC | DMST | 0.02 | LC | Isofenphos-methyl | 0.01 | GC | Propanil | 0.01 | LC |
| Atrazine | 0.01 | LC | DNOC | 0.01 | LC | Isofenphos-oxon | 0.01 | GC | Propaquizafop | 0.01 | LC |
| Atrazine-desethyl | 0.01 | LC | Dodine | 0.01 | LC | Isoprocarb | 0.01 | LC | Propargite | 0.01 | LC |
| Atrazine-desisopropyl | 0.01 | LC | Emamectin benzoate | 0.01 | LC | Isoprothiolane | 0.01 | LC | Propazine | 0.01 | LC |
| Azaconazole | 0.01 | LC | Endosulfan alpha | 0.01 | GC | Isoproturon | 0.01 | LC | Propetamphos | 0.01 | GC |
| Azamethiphos | 0.01 | GC | Endosulfan beta | 0.01 | GC | Kresoxim-methyl | 0.01 | LC | Propham | 0.01 | GC |
| Azinphos-ethyl | 0.01 | GC | Endosulfan ether | 0.01 | GC | Lenacil | 0.01 | GC | Propiconazole | 0.01 | GC |
| Azinphos-methyl | 0.01 | GC | Endosulfan lacton | 0.01 | GC | Lindane | 0.01 | GC | Propoxur | 0.01 | LC |
| Azoxystrobin | 0.01 | LC | Endosulfan sulfate | 0.02 | GC | Linuron | 0.01 | LC | Propoxycarbazone sodium | 0.01 | LC |
| Azoxystrobin | 0.01 | GC | Endosulfan-sulfate | 0.02 | LC | Lufenuron | 0.01 | LC | Propyzamide | 0.01 | LC |
| Benalaxyl | 0.01 | LC | Endrin | 0.01 | GC | Malaoxon | 0.01 | LC | Proquinazid | 0.01 | LC |
| Bendiocarb | 0.01 | LC | EPN | 0.01 | GC | Malathion | 0.01 | LC | Prosulfocarb | 0.01 | LC |
| Bentazone | 0.01 | LC | Epoxiconazole | 0.01 | LC | Mandipropamid | 0.01 | LC | Prosulfuron | 0.01 | LC |
| Benthiavalicarb-isopropyl | 0.01 | LC | EPTC | 0.01 | LC | MCPA | 0.02 | LC | Prothioconazole desthio | 0.01 | LC |
| Benzoximate | 0.01 | LC | Esfenvalerate | 0.01 | GC | MCPA Methyl Ester | 0.01 | GC | Prothiophos | 0.01 | GC |
| Bifenthrin | 0.01 | LC | Ethiofencarb | 0.01 | LC | MCPB | 0.01 | LC | Pymetrozine | 0.02 | LC |

| Pesticide Multi-screen: Fruit and Vegetables, Cereals and Honey Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/ LC |
| Binapacryl | 0.01 | GC | Ethiofencarb sulfone | 0.02 | LC | Mecarbam | 0.01 | GC | Pyraclostrobin | 0.01 | LC |
| Bioresmethrin | 0.01 | LC | Ethiofencarb sulfoxide | 0.02 | LC | Mecoprop-P | 0.01 | LC | Pyrazophos | 0.01 | LC |
| Biphenyl | 0.01 | GC | Ethion | 0.01 | LC | Mefenpyr-diethyl | 0.01 | LC | Pyrethrins | 0.05 | LC |
| Bitertanol | 0.01 | GC | Ethirimol | 0.01 | LC | Mepanipyrim | 0.01 | LC | Pyridaben | 0.01 | LC |
| Bixafen | 0.01 | LC | Ethofumesate | 0.01 | LC | Mephosfolan | 0.01 | LC | Pyridaben | 0.01 | GC |
| Boscalid | 0.01 | LC | Ethoprophos | 0.01 | GC | Mepronil | 0.01 | LC | Pyridaphenthion | 0.01 | LC |
| Boscalid | 0.01 | GC | Etofenprox | 0.01 | LC | Mesosulfuron-methyl | 0.01 | LC | Pyrifenox | 0.02 | GC |
| Bromacil | 0.01 | LC | Etoxazole | 0.01 | GC | Metalaxyl | 0.01 | LC | Pyrimethanil | 0.01 | LC |
| Bromophos-ethyl | 0.01 | GC | Etridiazole | 0.01 | GC | Metamitron | 0.01 | LC | Pyriproxyfen | 0.01 | LC |
| Bromophos-methyl | 0.01 | GC | Etrimfos | 0.01 | LC | Metazachlor | 0.01 | LC | Quinalphos | 0.01 | LC |
| Bromopropylate | 0.01 | GC | Famoxadone | 0.01 | LC | Metconazole | 0.01 | LC | Quinclorac | 0.01 | LC |
| Bromoxynil | 0.01 | LC | Fenamidone | 0.01 | GC | Methacrifos | 0.01 | GC | Quinoxyfen | 0.01 | LC |
| Bromuconazole | 0.01 | LC | Fenamiphos | 0.01 | LC | Methamidophos | 0.01 | LC | Quintozene | 0.01 | LC |
| Bupirimate | 0.01 | LC | Fenamiphos-sulfone | 0.01 | LC | Methamidophos | 0.01 | GC | Quizalfop (free acid) | 0.02 | LC |
| Buprofezin | 0.01 | LC | Fenamiphos-sulfoxide | 0.01 | LC | Methidathion | 0.01 | LC | Quizalofop-ethyl | 0.01 | LC |
| Butocarboxim-sulfoxide | 0.01 | LC | Fenarimol | 0.01 | LC | Methiocarb | 0.01 | LC | Resmethrin | 0.10 | GC |
| Butoxycarboxim | 0.01 | LC | Fenazaquin | 0.01 | GC | Methiocarb sulfone | 0.01 | LC | Rimsulfuron | 0.01 | LC |
| Cadusafos | 0.01 | LC | Fenbuconazole | 0.01 | LC | Methiocarb sulfoxide | 0.01 | LC | Rotenone | 0.01 | LC |
| Captafol | 0.02 | GC | Fenchlorphos | 0.01 | GC | Methomyl | 0.01 | LC | Silthiofam | 0.01 | GC |
| Captan | 0.01 | GC | Fenhexamid | 0.01 | LC | Methoprene | 0.01 | LC | Simazine | 0.01 | LC |
| Carbaryl | 0.01 | LC | Fenitrothion | 0.01 | GC | Methoxychlor | 0.01 | GC | Simetryn | 0.01 | LC |
| Carbendazim | 0.02 | LC | Fenoprop ( 2,4,5 TP) | 0.01 | LC | Methoxyfenozide | 0.01 | LC | Spinosad | 0.01 | LC |
| Carbofuran | 0.01 | LC | Fenothiocarb | 0.01 | LC | Metobromuron | 0.01 | LC | Spirodiclofen | 0.01 | LC |
| Carbofuran-3-hydroxy | 0.01 | LC | Fenoxaprop-P | 0.01 | LC | Metolachlor | 0.01 | LC | Spirodiclofen | 0.01 | GC |
| Carbosulfan | 0.01 | LC | Fenoxycarb | 0.01 | LC | Metosulam | 0.01 | LC | Spiromesifen | 0.01 | LC |
| Carboxin | 0.01 | LC | Fenpiclonil | 0.01 | LC | Metoxuron | 0.01 | LC | Spirotetramat | 0.01 | LC |
| Carfentrazone-ethyl | 0.01 | LC | Fenpropathrin | 0.01 | LC | Metrafenone | 0.01 | LC | Spiroxamine | 0.01 | LC |
| Chlorantraniliprole | 0.01 | LC | Fenpropidin | 0.01 | LC | Metribuzin | 0.01 | LC | Sulfentrazone | 0.01 | LC |
| Chlorbromuron | 0.01 | LC | Fenpropimorph | 0.01 | LC | Metsulfuron-methyl | 0.01 | LC | Sulfotep | 0.01 | LC |
| Chlorbufam | 0.01 | GC | Fenpyroximate | 0.01 | LC | Mevinphos | 0.01 | GC | Sulprofos | 0.01 | LC |
| Chlordane-cis | 0.01 | GC | Fenthion | 0.01 | LC | Mirex | 0.01 | GC | Tebuconazole | 0.01 | LC |
| Chlordane-trans | 0.01 | GC | Fenthion sulfone | 0.01 | LC | Molinate | 0.01 | LC | Tebufenozide | 0.01 | LC |
| Chlorfenapyr | 0.01 | GC | Fenthion sulfoxide | 0.01 | LC | Molinate | 0.01 | GC | Tebufenpyrad | 0.01 | LC |
| Chlorfenvinphos | 0.01 | LC | Fenuron | 0.01 | LC | Monocrothophos | 0.02 | LC | Tecnazene | 0.01 | GC |
| Chlorfluazuron | 0.01 | LC | Fenvalerate | 0.01 | GC | Monolinuron | 0.01 | LC | Teflubenzuron | 0.01 | LC |
| Chloridazon | 0.01 | LC | Fipronil | 0.01 | LC | Myclobutanyl | 0.01 | LC | Tefluthrin | 0.02 | GC |
| Chlorobenzilate | 0.01 | GC | Fipronil desulfinyl | 0.01 | LC | Napropamide | 0.01 | LC | Terbufos | 0.01 | LC |
| Chlorothalonil | 0.01 | GC | Fipronil sulfide | 0.01 | LC | Naptalam | 0.01 | LC | Terbumeton | 0.01 | LC |
| Chlorotoluron | 0.01 | LC | Fipronil sulfone | 0.01 | LC | Neburon | 0.01 | LC | Terbuthylazine | 0.01 | LC |
| Chloroxuron | 0.01 | LC | Flamprop isopropyl | 0.01 | LC | Nicosulfuron | 0.01 | LC | Terbuthylazine-2-hydroxy | 0.01 | LC |
| Chlorpropham | 0.01 | GC | Flazasulfuron | 0.01 | LC | Nitenpyram | 0.01 | LC | Terbuthylazine-desethyl | 0.01 | LC |
| Chlorpyrifos | 0.01 | LC | Flonicamid | 0.01 | LC | Nitrofen | 0.01 | GC | Terbutryn | 0.01 | LC |
| Chlorpyrifos-methyl | 0.01 | LC | Florasulam | 0.01 | LC | Nonachlor-trans | 0.01 | GC | Tetraconazole | 0.01 | LC |
| Chlorsulfuron | 0.01 | LC | Fluazifop (free acid) | 0.02 | LC | Nuarimol | 0.01 | LC | Tetradifon | 0.01 | GC |
| Chlorthal-dimethyl | 0.01 | GC | Fluazifop-P-butyl | 0.01 | LC | Omethoate | 0.01 | GC | Tetramethrin | 0.02 | GC |

| Pesticide Multi-screen: Fruit and Vegetables, Cereals and Honey Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/ LC |
| Chlozolinate | 0.01 | GC | Fluazinam | 0.01 | LC | op DDD | 0.01 | GC | Thiabendazole | 0.01 | LC |
| Clethodim | 0.01 | LC | Flubendiamide | 0.01 | LC | op DDE | 0.01 | GC | Thiacloprid | 0.02 | LC |
| Clodinafop-propargyl ester | 0.01 | LC | Flucycloxuron | 0.01 | LC | op DDT | 0.01 | GC | Thiamethoxam | 0.01 | LC |
| Clofentezine | 0.01 | LC | Flucythrinate | 0.01 | GC | o-Phenylphenol | 0.01 | GC | Thifensulfuron-methyl | 0.01 | LC |
| Clomazone | 0.01 | LC | Fludioxonil | 0.01 | LC | Oxadiazon | 0.01 | LC | Thiobencarb | 0.01 | LC |
| Clopyralid | 0.05 | LC | Flufenacet | 0.01 | LC | Oxadixyl | 0.01 | GC | Thiodicarb | 0.01 | LC |
| Clothianidin | 0.01 | LC | Flufenoxuron | 0.01 | LC | Oxamyl | 0.01 | LC | Thionazin | 0.01 | LC |
| Coumaphos | 0.01 | LC | Fluopicolide | 0.01 | LC | Oxamyl-oxime | 0.01 | LC | Thiophanate-ethyl | 0.01 | LC |
| Cyanazine | 0.01 | LC | Fluopyram | 0.01 | LC | Oxy-chlordane | 0.01 | GC | Thiophanate-methyl | 0.01 | LC |
| Cyanofenphos | 0.01 | GC | Fluquinconazole | 0.01 | LC | Oxyfluorfen | 0.01 | LC | Tolclofos-methyl | 0.01 | GC |
| Cyanophos | 0.01 | GC | Flurochloridone | 0.01 | LC | Paclobutrazol | 0.01 | LC | Tolyfluanid | 0.01 | LC |
| Cyazofamid | 0.01 | LC | Flurtamone | 0.01 | GC | Paraoxon-ethyl | 0.01 | LC | Topramezone | 0.01 | LC |
| Cyclanilide | 0.10 | LC | Flusilazole | 0.01 | LC | Paraoxon-methyl | 0.01 | LC | Triadimefon | 0.01 | GC |
| Cycloate | 0.01 | LC | Flutolanil | 0.01 | LC | Parathion-ethyl | 0.01 | GC | Triadimenol | 0.01 | GC |
| Cycloxydim | 0.05 | LC | Flutriafol | 0.01 | LC | Parathion-methyl | 0.01 | GC | Tri-Allat | 0.01 | LC |
| Cyfluthrin | 0.01 | GC | Fluvalinate-tau | 0.01 | GC | PCB No. 101 | 0.01 | GC | Triasulfuron | 0.01 | LC |
| Cyfluthrin beta | 0.01 | GC | Fluxapyroxad | 0.01 | LC | PCB No. 118 | 0.01 | GC | Triazophos | 0.01 | LC |
| Cyhalothrin lambda | 0.01 | GC | Folpet | 0.01 | GC | PCB No. 138 | 0.01 | GC | Trichlorfon | 0.02 | LC |
| Cymiazole | 0.01 | LC | Fonofos | 0.01 | GC | PCB No. 153 | 0.01 | GC | Triclopyr | 0.01 | LC |
| Cymoxanil | 0.01 | LC | Forchlorfenuron | 0.01 | LC | PCB No. 180 | 0.01 | GC | Tricyclazole | 0.01 | LC |
| Cypermethrin | 0.02 | GC | Formothion | 0.01 | GC | PCB No. 28 | 0.01 | GC | Trifloxystrobin | 0.01 | LC |
| Cyproconazole | 0.01 | LC | Fosthiazate | 0.01 | LC | PCB No. 52 | 0.01 | GC | Triflumizole | 0.02 | LC |
| Cyprodinil | 0.01 | LC | Fuberidazole | 0.01 | LC | Penconazole | 0.01 | LC | Triflumizole | 0.02 | GC |
| DEET | 0.01 | LC | Furalaxyl | 0.01 | GC | Pencycuron | 0.01 | LC | Triflumuron | 0.01 | LC |
| Deltamethrin | 0.01 | GC | Furathiocarb | 0.01 | LC | Pendimethalin | 0.01 | LC | Trifluralin | 0.01 | GC |
| Demeton-s-methyl sulfone | 0.01 | LC | Furmecyclox | 0.01 | LC | Pentachloroanaline | 0.01 | GC | Triflusulfuron-methyl | 0.01 | LC |
| Demeton-s-methyl sulfoxide | 0.01 | LC | Haloxyfop | 0.02 | LC | Permethrin | 0.01 | GC | Triticonazole | 0.01 | LC |
| Desmedipham | 0.01 | LC | Haloxyfop-methyl | 0.01 | LC | Pethoxamid | 0.01 | LC | Vamidothion | 0.01 | LC |
| Diazinon | 0.01 | GC | HCH-alpha | 0.01 | GC | Phenmedipham | 0.01 | LC | Vinclozolin | 0.01 | GC |
| Dichlobenil | 0.01 | GC | HCH-beta | 0.01 | GC | Phenthoate | 0.01 | LC | Zoxamide | 0.01 | LC |
| Dichlofenthion | 0.01 | LC | | | | | | | | | |

| Pesticide Multi-screen: Milk, Eggs and Infant Formula Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/LC | Compound Name | RL (mg/ Kg) | GC/LC | Compound Name | RL (mg/ Kg) | GC/ LC |
| 1-Naphthyl acetamide | 0.01 | LC | Diclobutrazol | 0.01 | LC | Hexachlorobenzene | 0.005 | GC | Phosphamidon-I | 0.005 | GC |
| 2,4,5-T | 0.01 | LC | Dicloran | 0.005 | GC | Hexaconazole | 0.005 | GC | Phosphamidon-II | 0.005 | GC |
| 2,4,6Trichlorophenol | 0.005 | GC | Dicrothophos | 0.01 | LC | Hexaflumuron | 0.01 | LC | Phoxim | 0.01 | LC |
| 2,4-D | 0.02 | LC | Dieldrin | 0.01 | GC | Hexythiazox | 0.01 | LC | Picloram | 0.01 | LC |
| 2,4-DB | 0.05 | LC | Diethofencarb | 0.01 | LC | Imazalil | 0.01 | LC | Picoxystrobin | 0.01 | LC |
| 3,5-Dichloroaniline | 0.01 | GC | Difenoconazole | 0.01 | LC | Imazamox | 0.01 | LC | Piperonyl butoxide | 0.01 | LC |
| 3-Chloroaniline | 0.005 | GC | Diflubenzuron | 0.01 | LC | Imazaquin | 0.01 | LC | Pirimicarb | 0.005 | GC |
| 4,4-Dichlorobenzophenone | 0.005 | GC | Dimethenamid | 0.01 | LC | Imazethapyr | 0.01 | LC | Pirimicarb desmethyl | 0.005 | GC |
| Abamectin | 0.01 | LC | Dimethoate | 0.005 | GC | Imidacloprid | 0.01 | LC | Pirimifos-ethyl | 0.01 | LC |
| Acephate | 0.01 | LC | Dimethomorph | 0.01 | LC | Indoxacarb | 0.01 | LC | Pirimifos-methyl | 0.01 | LC |
| Acephate | 0.05 | GC | Dimoxystrobin | 0.005 | GC | Iodofenphos | 0.005 | GC | ppDDD | 0.005 | GC |
| Acetamiprid | 0.01 | LC | Diniconazole | 0.01 | LC | Iodosulfuron-methyl-sodium | 0.01 | LC | ppDDE | 0.005 | GC |
| Acetochlor | 0.01 | LC | Dinitramine | 0.01 | LC | Ioxynil | 0.01 | LC | ppDDT | 0.01 | GC |
| Acibenzolar-S-methyl | 0.01 | LC | Dinoseb | 0.02 | LC | Iprovalicarb-I | 0.02 | GC | Prochloraz | 0.01 | LC |

| Pesticide Multi-screen: Milk, Eggs and Infant Formula Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC |
| Aclonifen | 0.02 | GC | Dinoterb | 0.02 | LC | Iprovalicarb-II | 0.02 | GC | Prochloraz | 0.05 | GC |
| Acrinathrin | 0.005 | GC | Dioxacarb | 0.01 | LC | Isazophos | 0.005 | GC | Procymidone | 0.005 | GC |
| Alachlor | 0.005 | GC | Diphenamid | 0.01 | LC | Isocarbofos | 0.01 | LC | Profenofos | 0.005 | GC |
| Aldicarb | 0.02 | LC | Diphenylamine | 0.005 | GC | Isodrin | 0.005 | GC | Promecarb | 0.01 | LC |
| Aldicarb sulfone | 0.01 | LC | Ditalimfos | 0.01 | LC | Isofenphos | 0.02 | LC | Prometon | 0.01 | LC |
| Aldicarb-sulfoxide | 0.02 | LC | Diuron | 0.01 | LC | Isofenphos | 0.005 | GC | Prometryn | 0.01 | LC |
| Aldrin | 0.005 | GC | DMSA | 0.02 | LC | Isofenphos-methyl | 0.005 | GC | Propachlor | 0.01 | LC |
| Ametryn | 0.01 | LC | DMST | 0.02 | LC | Isofenphos-oxon | 0.005 | GC | Propachlor | 0.005 | GC |
| Amidosulfuron | 0.01 | LC | DNOC | 0.01 | LC | Isoprocarb | 0.01 | LC | Propamocarb free base | 0.01 | LC |
| Aminocarb | 0.01 | LC | Dodine | 0.01 | LC | Isoprothiolane | 0.01 | LC | Propanil | 0.01 | LC |
| Anthraquinone | 0.005 | GC | Emamectin benzoate | 0.01 | LC | Isoproturon | 0.01 | LC | Propanil | 0.005 | GC |
| Asulam | 0.02 | LC | Endosulfan-alpha | 0.01 | GC | Kresoxim-methyl | 0.01 | LC | Propaquizafop | 0.01 | LC |
| Atrazine | 0.01 | LC | Endosulfan-beta | 0.01 | GC | Lenacil | 0.005 | GC | Propargite | 0.01 | LC |
| Atrazine-desethyl | 0.01 | LC | Endosulfan-ether | 0.005 | GC | Lindane | 0.005 | GC | Propargite | 0.005 | GC |
| Atrazine-desisopropyl | 0.01 | LC | Endosulfan-lacton | 0.02 | GC | Linuron | 0.01 | LC | Propazine | 0.01 | LC |
| Azaconazole | 0.01 | LC | Endosulfan-sulfate | 0.02 | LC | Lufenuron | 0.01 | LC | Propetamphos | 0.005 | GC |
| Azaconazole | 0.005 | GC | Endosulfan-sulfate | 0.02 | GC | Malaoxon | 0.01 | LC | Propham | 0.005 | GC |
| Azamethiophos | 0.01 | GC | Endrin | 0.01 | GC | Malathion | 0.01 | LC | Propiconazole-I | 0.005 | GC |
| Azinphos-ethyl | 0.005 | GC | EPN | 0.005 | GC | Mandipropamid | 0.01 | LC | Propiconazole-II | 0.005 | GC |
| Azinphos-methyl | 0.01 | GC | Epoxiconazole | 0.01 | LC | MCPA | 0.02 | LC | Propoxur | 0.01 | LC |
| Azoxystrobin | 0.01 | LC | EPTC | 0.01 | LC | MCPA methyl ester | 0.005 | GC | Propoxycarbazone sodium | 0.01 | LC |
| Azoxystrobin | 0.01 | GC | Ethiofencarb | 0.01 | LC | MCPB | 0.01 | LC | Propyzamide | 0.01 | LC |
| Benalaxyl | 0.01 | LC | Ethiofencarb sulfone | 0.02 | LC | Mecarbam | 0.005 | GC | Proquinazid | 0.01 | LC |
| Bendiocarb | 0.01 | LC | Ethiofencarb sulfoxide | 0.02 | LC | Mecoprop-P | 0.01 | LC | Prosulfocarb | 0.01 | LC |
| Bentazone | 0.01 | LC | Ethion | 0.01 | LC | Mefenpyr-diethyl | 0.01 | LC | Prosulfuron | 0.01 | LC |
| Benthiavalicarb-isopropyl | 0.01 | LC | Ethirimol | 0.01 | LC | Mepanipyrim | 0.01 | LC | Prothioconazole desthio | 0.01 | LC |
| Benzoximate | 0.01 | LC | Ethofumesate | 0.01 | LC | Mephosfolan | 0.01 | LC | Prothiofos | 0.005 | GC |
| Bifenthrin | 0.01 | LC | Ethoprophos | 0.005 | GC | Mepronil | 0.01 | LC | Pymetrozine | 0.02 | LC |
| Bifenthrin | 0.005 | GC | Etofenprox | 0.01 | LC | Mesosulfuron-methyl | 0.01 | LC | Pyraclostrobin | 0.01 | LC |
| Bioresmethrin | 0.01 | LC | Etoxazole | 0.005 | GC | Metalaxyl | 0.01 | LC | Pyrazophos | 0.01 | LC |
| Biphenyl | 0.005 | GC | Etridazole | 0.005 | GC | Metamitron | 0.01 | LC | Pyrethrins | 0.05 | LC |
| Bitertanol-I | 0.005 | GC | Etrimfos | 0.01 | LC | Metazachlor | 0.01 | LC | Pyridaben | 0.01 | LC |
| Bitertanol-II | 0.005 | GC | Famoxadone | 0.01 | LC | Metconazole | 0.01 | LC | Pyridaben | 0.005 | GC |
| Bixafen | 0.01 | LC | Fenamidone | 0.005 | GC | Methacrifos | 0.005 | GC | Pyridaphenthion | 0.01 | LC |
| Boscalid | 0.01 | LC | Fenamiphos | 0.01 | LC | Methamidophos | 0.01 | LC | Pyrifenox-I | 0.01 | GC |
| Boscalid | 0.02 | GC | Fenamiphos-sulfone | 0.01 | LC | Methamidophos | 0.005 | GC | Pyrifenox-II | 0.01 | GC |
| Bromacil | 0.01 | LC | Fenamiphos-sulfoxide | 0.01 | LC | Methidathion | 0.01 | LC | Pyrimethanil | 0.01 | LC |
| Bromophos-ethyl | 0.005 | GC | Fenarimol | 0.01 | LC | Methiocarb | 0.01 | LC | Pyriproxyfen | 0.01 | LC |
| Bromophos-methyl | 0.005 | GC | Fenarimol | 0.005 | GC | Methiocarb sulfone | 0.01 | LC | Quinalphos | 0.01 | LC |
| Bromopropylate | 0.005 | GC | Fenazaquin | 0.01 | GC | Methiocarb sulfoxide | 0.01 | LC | Quinclorac | 0.01 | LC |
| Bromoxynil | 0.01 | LC | Fenbuconazole | 0.01 | LC | Methomyl | 0.01 | LC | Quinoxyfen | 0.01 | LC |
| Bromuconazole | 0.01 | LC | Fenbuconazole | 0.005 | GC | Methoprene | 0.01 | LC | Quintozene | 0.01 | LC |
| Bupirimate | 0.01 | LC | Fenchlorphos | 0.005 | GC | Methoxychlor | 0.02 | GC | Quintozene | 0.005 | GC |
| Buprofezin | 0.01 | LC | Fenhexamid | 0.01 | LC | Methoxyfenozide | 0.01 | LC | Quizalfop (free acid) | 0.02 | LC |
| Butocarboxim-sulfoxide | 0.01 | LC | Fenitrothion | 0.005 | GC | Metobromuron | 0.01 | LC | Quizalofop-ethyl | 0.01 | LC |
| Butoxycarboxim | 0.01 | LC | Fenoprop ( 2,4,5 TP) | 0.01 | LC | Metolachlor | 0.01 | LC | Resmethrin | 0.10 | GC |
| Cadusafos | 0.01 | LC | Fenothiocarb | 0.01 | LC | Metosulam | 0.01 | LC | Rimsulfuron | 0.01 | LC |

| Pesticide Multi-screen: Milk, Eggs and Infant Formula Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC |
| Carbaryl | 0.01 | LC | Fenoxaprop-P | 0.01 | LC | Metoxuron | 0.01 | LC | Rotenone | 0.01 | LC |
| Carbendazim | 0.02 | LC | Fenoxycarb | 0.01 | LC | Metrafenone | 0.01 | LC | Silthiofam | 0.005 | GC |
| Carbofuran | 0.01 | LC | Fenpiclonil | 0.01 | LC | Metribuzin | 0.01 | LC | Simazine | 0.01 | LC |
| Carbofuran-3-hydroxy | 0.01 | LC | Fenpropathrin | 0.01 | LC | Metribuzin | 0.005 | GC | Simetryn | 0.01 | LC |
| Carbosulfan | 0.01 | LC | Fenpropathrin | 0.005 | GC | Metsulfuron-methyl | 0.01 | LC | Spinosad | 0.01 | LC |
| Carboxin | 0.01 | LC | Fenpropidin | 0.01 | LC | Mevinphos | 0.005 | GC | Spirodiclofen | 0.01 | LC |
| Carfentrazone-ethyl | 0.01 | LC | Fenpropimorph | 0.01 | LC | Mirex | 0.005 | GC | Spirodiclofen | 0.02 | GC |
| Chlorantraniliprole | 0.01 | LC | Fenpyroximate | 0.01 | LC | Molinate | 0.01 | LC | Spiromesifen | 0.01 | LC |
| Chlorbromuron | 0.01 | LC | Fenthion | 0.01 | LC | Molinate | 0.02 | GC | Spirotetramat | 0.01 | LC |
| Chlorbufam | 0.02 | GC | Fenthion sulfone | 0.01 | LC | Monocrothophos | 0.02 | LC | Spiroxamine | 0.01 | LC |
| Chlordane-cis | 0.005 | GC | Fenthion sulfoxide | 0.01 | LC | Monolinuron | 0.01 | LC | Sulfentrazone | 0.01 | LC |
| Chlordane-trans | 0.005 | GC | Fenuron | 0.01 | LC | Myclobutanyl | 0.01 | LC | Sulfotep | 0.01 | LC |
| Chlorfenapyr | 0.02 | GC | Fenvalerate-I | 0.01 | GC | Napropamide | 0.01 | LC | Sulprofos | 0.01 | LC |
| Chlorfenvinphos | 0.01 | LC | Fenvalerate-II | 0.01 | GC | Naptalam | 0.01 | LC | Tebuconazole | 0.01 | LC |
| Chlorfluazuron | 0.01 | LC | Fipronil | 0.01 | LC | Neburon | 0.01 | LC | Tebufenozide | 0.01 | LC |
| Chloridazon | 0.01 | LC | Fipronil desulfinyl | 0.01 | LC | Nicosulfuron | 0.01 | LC | Tebufenpyrad | 0.01 | LC |
| Chlorobenzilate | 0.005 | GC | Fipronil sulfide | 0.01 | LC | Nitenpyram | 0.01 | LC | Tecnazene | 0.005 | GC |
| Chlorothalonil | 0.005 | GC | Fipronil sulfone | 0.01 | LC | Nitrofen | 0.02 | GC | Teflubenzuron | 0.01 | LC |
| Chlorotoluron | 0.01 | LC | Flamprop isopropyl | 0.01 | LC | Nonachlor-trans | 0.005 | GC | Tefluthrin | 0.005 | GC |
| Chloroxuron | 0.01 | LC | Flazasulfuron | 0.01 | LC | Nuarimol | 0.01 | LC | Terbufos | 0.01 | LC |
| Chlorpropham | 0.005 | GC | Flonicamid | 0.01 | LC | Nuarimol | 0.005 | GC | Terbumeton | 0.01 | LC |
| Chlorpyrifos | 0.01 | LC | Florasulam | 0.01 | LC | Omethoate | 0.005 | GC | Terbuthylazine | 0.01 | LC |
| Chlorpyrifos-methyl | 0.01 | LC | Fluazifop (free acid) | 0.02 | LC | opDDD | 0.005 | GC | Terbuthylazine-2-hydroxy | 0.01 | LC |
| Chlorpyriphos-Methyl | 0.005 | GC | Fluazifop-P-butyl | 0.01 | LC | opDDE | 0.005 | GC | Terbuthylazine-desethyl | 0.01 | LC |
| Chlorsulfuron | 0.01 | LC | Fluazinam | 0.01 | LC | opDDT | 0.01 | GC | Terbutryn | 0.01 | LC |
| Chlorthal-dimethyl | 0.005 | GC | Flubendiamide | 0.01 | LC | o-Phenyphenol | 0.005 | GC | Tetraconazole | 0.01 | LC |
| Chlozolinate | 0.005 | GC | Flucycloxuron | 0.01 | LC | Oxadiazon | 0.01 | LC | Tetraconazole | 0.005 | GC |
| Clethodim | 0.01 | LC | Flucythrinate-I | 0.02 | GC | Oxadixyl | 0.005 | GC | Tetradifon | 0.005 | GC |
| Clodinafop-propargyl ester | 0.01 | LC | Flucythrinate-II | 0.02 | GC | Oxamyl | 0.01 | LC | Tetramethrin-I | 0.02 | GC |
| Clofentezine | 0.01 | LC | Fludioxonil | 0.01 | LC | Oxamyl-oxime | 0.01 | LC | Tetramethrin-II | 0.02 | GC |
| Clomazone | 0.01 | LC | Fludioxonil | 0.005 | GC | Oxychlordane | 0.005 | GC | Thiabendazole | 0.01 | LC |
| Clopyralid | 0.05 | LC | Flufenacet | 0.01 | LC | Oxyfluorfen | 0.01 | LC | Thiacloprid | 0.02 | LC |
| Clothianidin | 0.01 | LC | Flufenoxuron | 0.01 | LC | Paclobutrazol | 0.01 | LC | Thiamethoxam | 0.01 | LC |
| Coumaphos | 0.01 | LC | Fluopicolide | 0.01 | LC | Paraoxon methyl | 0.005 | GC | Thifensulfuron-methyl | 0.01 | LC |
| Coumaphos | 0.005 | GC | Fluopyram | 0.01 | LC | Paraoxon-ethyl | 0.01 | LC | Thiobencarb | 0.01 | LC |
| Cyanazine | 0.01 | LC | Fluquinconazole | 0.01 | LC | Paraoxon-methyl | 0.01 | LC | Thiodicarb | 0.01 | LC |
| Cyanofenphos | 0.005 | GC | Flurochloridone | 0.01 | LC | Parathion-ethyl | 0.005 | GC | Thionazin | 0.01 | LC |
| Cyanophos | 0.005 | GC | Flurtamone | 0.005 | GC | Parathion-methyl | 0.005 | GC | Thiophanate-ethyl | 0.01 | LC |
| Cyazofamid | 0.01 | LC | Flusilazole | 0.01 | LC | PCB 101 | 0.005 | GC | Thiophanate-methyl | 0.01 | LC |
| Cyclanilide | 0.10 | LC | Flusilazole | 0.005 | GC | PCB 118 | 0.005 | GC | Tolclofos-methyl | 0.005 | GC |
| Cycloate | 0.01 | LC | Flutolanil | 0.01 | LC | PCB 138 | 0.005 | GC | Tolyfluanid | 0.01 | LC |
| Cycloxydim | 0.05 | LC | Flutriafol | 0.01 | LC | PCB 153 | 0.005 | GC | Tolylfluanid | 0.005 | GC |
| Cyfluthrin | 0.02 | GC | Fluvalinate-tau-I | 0.02 | GC | PCB 180 | 0.005 | GC | Topramezone | 0.01 | LC |
| Cyhalothrin-lambda | 0.005 | GC | Fluvalinate-tau-II | 0.02 | GC | PCB 28 | 0.005 | GC | Triadimefon | 0.005 | GC |
| Cymiazole | 0.01 | LC | Fluxapyroxad | 0.01 | LC | PCB 52 | 0.005 | GC | Triadimenol-I | 0.02 | GC |
| Cymoxanil | 0.01 | LC | Fonofos | 0.005 | GC | Penconazole | 0.01 | LC | Triadimenol-II | 0.02 | GC |
| Cypermethrin | 0.05 | GC | Forchlorfenuron | 0.01 | LC | Pencycuron | 0.01 | LC | Tri-Allat | 0.01 | LC |
| Cyproconazole | 0.01 | LC | Formothion | 0.005 | GC | Pendimethalin | 0.01 | LC | Triasulfuron | 0.01 | LC |
| Cyproconazole | 0.005 | GC | Fosthiazate | 0.01 | LC | Pendimethalin | 0.005 | GC | Triazophos | 0.01 | LC |
| Cyprodinil | 0.01 | LC | Fuberidazole | 0.01 | LC | Pentachloroanilin e | 0.005 | GC | Trichlorfon | 0.02 | LC |
| DEET | 0.01 | LC | Furalaxyl | 0.005 | GC | Permethrin-I | 0.02 | GC | Triclopyr | 0.01 | LC |
| Deltamethrin | 0.02 | GC | Furathiocarb | 0.01 | LC | Permethrin-II | 0.02 | GC | Tricyclazole | 0.01 | LC |

| Pesticide Multi-screen: Milk, Eggs and Infant Formula Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC | Compound Name | RL (mg/Kg) | GC/LC |
| Demeton-s-methyl sulfone | 0.01 | LC | Furmecyclox | 0.01 | LC | Pethoxamid | 0.01 | LC | Trifloxystrobin | 0.01 | LC |
| Demeton-S-methyl sulfone | 0.005 | GC | Haloxyfop | 0.02 | LC | Phenmedipham | 0.01 | LC | Triflumizole | 0.02 | LC |
| Demeton-s-methyl sulfoxide | 0.01 | LC | Haloxyfop-methyl | 0.01 | LC | Phenthoate | 0.01 | LC | Triflumizole | 0.01 | GC |
| Desmedipham | 0.01 | LC | HCH-alpha | 0.005 | GC | Phenthoate | 0.005 | GC | Triflumuron | 0.01 | LC |
| Diazinon | 0.005 | GC | HCH-beta | 0.005 | GC | Phorate | 0.01 | LC | Trifluralin | 0.005 | GC |
| Dichlobenil | 0.005 | GC | HCH-delta | 0.005 | GC | Phorate sulfoxide | 0.01 | LC | Triflusulfuron-methyl | 0.01 | LC |
| Dichlofenthion | 0.01 | LC | Heptachlor | 0.005 | GC | Phosalone | 0.01 | LC | Triticonazole | 0.01 | LC |
| Dichlofluanid | 0.005 | GC | Heptachlor endo epoxide | 0.005 | GC | Phosalone | 0.005 | GC | Vamidothion | 0.01 | LC |
| Dichlorprop-P | 0.01 | LC | Heptachlor exo epoxide | 0.005 | GC | Phosmet | 0.005 | GC | Vinclozolin | 0.005 | GC |
| Dichlorvos | 0.005 | GC | Heptenophos | 0.01 | LC | Phosmet-oxon | 0.01 | LC | Zoxamide | 0.01 | LC |
| **Pesticide Multi-screen: Fats Scope** | | | | | | | | | | | |
| Compound Name | RL(mg/Kg) | GC/LC | Compound Name | RL(mg/Kg) | GC/LC | Compound Name | RL(mg/Kg) | GC/LC | Compound Name | RL(mg/Kg) | GC/LC |
| 2,4,6Trichlorophenol | 0.005 | GC | Dimethomorph | 0.01 | LC | Iprovalicarb-I | 0.02 | GC | Phosalone | 0.005 | GC |
| 3,5-Dichloroaniline | 0.01 | GC | Dimoxystrobin | 0.005 | GC | Iprovalicarb-II | 0.02 | GC | Phosmet | 0.005 | GC |
| 3-Chloroaniline | 0.005 | GC | Diniconazole | 0.02 | LC | Isazophos | 0.005 | GC | Phosphamidon-I | 0.005 | GC |
| 4,4-Dichlorobenzophenone | 0.005 | GC | Diphenylamine | 0.005 | GC | Isodrin | 0.005 | GC | Phosphamidon-II | 0.005 | GC |
| Acephate | 0.05 | GC | Diuron | 0.01 | LC | Isofenphos | 0.01 | LC | Phoxim | 0.01 | LC |
| Aclonifen | 0.02 | GC | Endosulfan-alpha | 0.01 | GC | Isofenphos | 0.005 | GC | Picoxystrobin | 0.02 | LC |
| Acrinathrin | 0.005 | GC | Endosulfan-beta | 0.01 | GC | Isofenphos-methyl | 0.005 | GC | Piperonyl butoxide | 0.01 | LC |
| Alachlor | 0.005 | GC | Endosulfan-ether | 0.005 | GC | Isofenphos-oxon | 0.005 | GC | Pirimicarb | 0.005 | GC |
| Aldrin | 0.005 | GC | Endosulfan-lacton | 0.02 | GC | Isoprocarb | 0.01 | LC | Pirimicarb desmethyl | 0.005 | GC |
| Ametryn | 0.01 | LC | Endosulfan-sulfate | 0.02 | GC | Isoprothiolane | 0.01 | LC | Pirimiphos ethyl | 0.01 | LC |
| Aminocarb | 0.01 | LC | Endrin | 0.01 | GC | Isoproturon | 0.01 | LC | Pirimiphos methyl | 0.01 | LC |
| Anthraquinone | 0.005 | GC | EPN | 0.005 | GC | Kresoxim-methyl | 0.01 | LC | ppDDD | 0.005 | GC |
| Atrazine | 0.01 | LC | Epoxyconazole | 0.01 | LC | Lenacil | 0.005 | GC | ppDDE | 0.005 | GC |
| Azaconazole | 0.005 | GC | Ethiofencarb | 0.05 | LC | Lindane | 0.005 | GC | ppDDT | 0.01 | GC |
| Azamethiophos | 0.01 | GC | Ethiofencarb sulfone | 0.05 | LC | Linuron | 0.01 | LC | Prochloraz | 0.05 | GC |
| Azinphos-ethyl | 0.005 | GC | Ethiofencarb sulfoxide | 0.05 | LC | Lufenuron | 0.05 | LC | Procymidone | 0.005 | GC |
| Azinphos-methyl | 0.01 | GC | Ethion | 0.01 | LC | Malaoxon | 0.01 | LC | Profenofos | 0.005 | GC |
| Azoxystrobin | 0.01 | GC | Ethofumesate | 0.05 | LC | Malathion | 0.01 | LC | Prometryn | 0.01 | LC |
| Benalaxyl | 0.01 | LC | Ethoprophos | 0.005 | GC | MCPA methyl ester | 0.005 | GC | Propachlor | 0.005 | GC |
| Bendiocarb | 0.02 | LC | Etofenprox | 0.02 | LC | Mecarbam | 0.005 | GC | Propanil | 0.005 | GC |
| Bifenthrin | 0.005 | GC | Etoxazole | 0.005 | GC | Mepanipyrim | 0.01 | LC | Propargite | 0.005 | GC |
| Biphenyl | 0.005 | GC | Etridazole | 0.005 | GC | Mepronil | 0.01 | LC | Propetamphos | 0.005 | GC |
| Bitertanol-I | 0.005 | GC | Etrimfos | 0.01 | LC | Metalaxyl | 0.01 | LC | Propham | 0.005 | GC |
| Bitertanol-II | 0.005 | GC | Famoxadone | 0.05 | LC | Metazachlor | 0.01 | LC | Propiconazole-I | 0.005 | GC |
| Bixafen | 0.10 | LC | Fenamidone | 0.005 | GC | Metconazole | 0.02 | LC | Propiconazole-II | 0.005 | GC |
| Boscalid | 0.02 | GC | Fenamiphos | 0.01 | LC | Methacrifos | 0.005 | GC | Propoxur | 0.01 | LC |
| Bromacil | 0.02 | LC | Fenarimol | 0.005 | GC | Methamidophos | 0.005 | GC | Propyzamide | 0.01 | LC |
| Bromophos-ethyl | 0.005 | GC | Fenazaquin | 0.01 | GC | Methidathion | 0.01 | LC | Prothiofos | 0.005 | GC |
| Bromophos-methyl | 0.005 | GC | Fenbuconazole | 0.005 | GC | Methiocarb | 0.01 | LC | Pyraclostrobin | 0.01 | LC |
| Bromopropylate | 0.005 | GC | Fenchlorphos | 0.005 | GC | Methiocarb sulfone | 0.05 | LC | Pyrazophos | 0.01 | LC |
| Bromuconazole | 0.02 | LC | Fenhexamid | 0.01 | LC | Methiocarb sulfoxide | 0.05 | LC | Pyrethrin | 0.05 | LC |
| Bupirimate | 0.01 | LC | Fenitrothion | 0.005 | GC | Methomyl | 0.05 | LC | Pyridaben | 0.02 | LC |
| Buprofezin | 0.01 | LC | Fenoxycarb | 0.01 | LC | Methoxychlor | 0.02 | GC | Pyridaben | 0.005 | GC |

| Pesticide Multi-screen: Milk, Eggs and Infant Formula Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (mg/ Kg) | GC/ LC | Compound Name | RL (mg/ Kg) | GC/LC | Compound Name | RL (mg/ Kg) | GC/LC | Compound Name | RL (mg/ Kg) | GC/ LC |
| Butocarboxim-sulfoxide | 0.05 | LC | Fenpropathrin | 0.005 | GC | Methoxyfenozide | 0.05 | LC | Pyridaphenthion | 0.01 | LC |
| Cadusafos | 0.01 | LC | Fenpropidin | 0.02 | LC | Metobromuron | 0.01 | LC | Pyrifenox-I | 0.01 | GC |
| Carbaryl | 0.01 | LC | Fenpropimorph | 0.05 | LC | Metolachlor | 0.01 | LC | Pyrifenox-II | 0.01 | GC |
| Carbendazim | 0.05 | LC | Fenpyroximate | 0.01 | LC | Metribuzin | 0.005 | GC | Pyrimethanil | 0.01 | LC |
| Carbofuran | 0.05 | LC | Fenthion | 0.05 | LC | Mevinphos | 0.005 | GC | Pyriproxifen | 0.01 | LC |
| Carbofuran 3 hydroxy | 0.05 | LC | Fenthion sulfone | 0.01 | LC | Mirex | 0.005 | GC | Quinalphos | 0.01 | LC |
| Carbosulfan | 0.05 | LC | Fenthion sulfoxide | 0.01 | LC | Molinate | 0.02 | LC | Quinoxyfen | 0.02 | LC |
| Carboxin | 0.02 | LC | Fenvalerate-I | 0.01 | GC | Molinate | 0.02 | GC | Quintozene | 0.005 | GC |
| Chlorbromuron | 0.01 | LC | Fenvalerate-II | 0.01 | GC | Myclobutanil | 0.01 | LC | Quizalofop | 0.05 | LC |
| Chlorbufam | 0.02 | GC | Flamprop-isopropyl | 0.01 | LC | Napropamide | 0.02 | LC | Resmethrin | 0.10 | GC |
| Chlordane-cis | 0.005 | GC | Flucythrinate-I | 0.02 | GC | Nitrofen | 0.02 | GC | Rotenone | 0.02 | LC |
| Chlordane-trans | 0.005 | GC | Flucythrinate-II | 0.02 | GC | Nonachlor-trans | 0.005 | GC | Silthiofam | 0.005 | GC |
| Chlorfenapyr | 0.02 | GC | Fludioxonil | 0.005 | GC | Nuarimol | 0.005 | GC | Simazine | 0.01 | LC |
| Chlorfenvinphos | 0.01 | LC | Flufenacet | 0.01 | LC | Omethoate | 0.005 | GC | Spirodiclofen | 0.02 | GC |
| Chlorobenzilate | 0.005 | GC | Flufenoxuron | 0.02 | LC | opDDD | 0.005 | GC | Spiroxamine | 0.02 | LC |
| Chlorothalonil | 0.005 | GC | Fluquinconazole | 0.05 | LC | opDDE | 0.005 | GC | Tebuconazole | 0.01 | LC |
| Chlorpropham | 0.005 | GC | Flurtamone | 0.005 | GC | opDDT | 0.01 | GC | Tebufenozide | 0.05 | LC |
| Chlorpyriphos | 0.01 | LC | Flusilazole | 0.005 | GC | o-Phenyphenol | 0.005 | GC | Tebufenpyrad | 0.02 | LC |
| Chlorpyriphos-Methyl | 0.005 | GC | Flutolanil | 0.01 | LC | Oxadixyl | 0.005 | GC | Tecnazene | 0.005 | GC |
| Chlorthal-dimethyl | 0.005 | GC | Flutriafol | 0.01 | LC | Oxychlordane | 0.005 | GC | Tefluthrin | 0.005 | GC |
| Chlozolinate | 0.005 | GC | Fluvalinate-tau-I | 0.02 | GC | Paclobutrazol | 0.01 | LC | Terbuthylazine | 0.01 | LC |
| Clofentezine | 0.01 | LC | Fluvalinate-tau-II | 0.02 | GC | Paraoxon ethyl | 0.01 | LC | Tetraconazole | 0.005 | GC |
| Coumaphos | 0.005 | GC | Fonofos | 0.005 | GC | Paraoxon methyl | 0.005 | GC | Tetradifon | 0.005 | GC |
| Cyanazine | 0.01 | LC | Formothion | 0.005 | GC | Parathion-ethyl | 0.005 | GC | Tetramethrin-I | 0.02 | GC |
| Cyanofenphos | 0.005 | GC | Fosthiazate | 0.01 | LC | Parathion-methyl | 0.005 | GC | Tetramethrin-II | 0.02 | GC |
| Cyanophos | 0.005 | GC | Fuberidazole | 0.01 | LC | PCB 101 | 0.005 | GC | Thiabendazole | 0.01 | LC |
| Cyazofamid | 0.01 | LC | Furalaxyl | 0.005 | GC | PCB 118 | 0.005 | GC | Thiacloprid | 0.02 | LC |
| Cyfluthrin | 0.02 | GC | Furathiocarb | 0.01 | LC | PCB 138 | 0.005 | GC | Tolclofos-methyl | 0.005 | GC |
| Cyhalothrin-lambda | 0.005 | GC | HCH-alpha | 0.005 | GC | PCB 153 | 0.005 | GC | Tolylfluanid | 0.005 | GC |
| Cypermethrin | 0.05 | GC | HCH-beta | 0.005 | GC | PCB 180 | 0.005 | GC | Triadimefon | 0.005 | GC |
| Cyproconazole | 0.005 | GC | HCH-delta | 0.005 | GC | PCB 28 | 0.005 | GC | Triadimenol-I | 0.02 | GC |
| Cyprodinil | 0.01 | LC | Heptachlor | 0.005 | GC | PCB 52 | 0.005 | GC | Triadimenol-II | 0.02 | GC |
| Deltamethrin | 0.02 | GC | Heptachlor endo epoxide | 0.005 | GC | Penconazole | 0.01 | LC | Triazophos | 0.01 | LC |
| Demeton-S-methyl sulfone | 0.005 | GC | Heptachlor exo epoxide | 0.005 | GC | Pencycuron | 0.01 | LC | Trifloxystrobin | 0.01 | LC |
| Diazinon | 0.005 | GC | Heptenophos | 0.01 | LC | Pendimethalin | 0.005 | GC | Triflumizole | 0.01 | LC |
| Dichlobenil | 0.005 | GC | Hexachlorobenzene | 0.005 | GC | Pentachloroaniline | 0.005 | GC | Triflumizole | 0.01 | GC |
| Dichlofluanid | 0.005 | GC | Hexaconazole | 0.005 | GC | Permethrin-I | 0.02 | GC | Trifluralin | 0.005 | GC |
| Dichlorvos | 0.005 | GC | Hexythiazox | 0.01 | LC | Permethrin-II | 0.02 | GC | Triticonazole | 0.01 | LC |
| Dicloran | 0.005 | GC | Imazalil | 0.02 | LC | Phenmedipham | 0.01 | LC | Vamidothion | 0.01 | LC |
| Dieldrin | 0.01 | GC | Indoxacarb | 0.01 | LC | Phenthoate | 0.005 | GC | Vinclozolin | 0.005 | GC |
| Diethofencarb | 0.01 | LC | Iodofenphos | 0.005 | GC | Phorate sulfoxide | 0.01 | LC | Zoxamide | 0.01 | LC |
| Difenoconazole | 0.01 | LC | | | | | | | | | |
| Dimethenamid | 0.01 | LC | | | | | | | | | |
| Dimethoate | 0.005 | GC | | | | | | | | | |

| Pesticide Multi-screen: Water Scope | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Compound Name | RL (ng/kg) | GC/LC | Compound Name | RL (ng/kg) | GC/LC | Compound Name | RL (ng/kg) | GC/LC | Compound Name | RL (ng/kg) | GC/LC |
| Abamectin | 1.00 | LC | Fenpropimorph | 1.00 | LC | Pirimicarb | 1.00 | LC | BAC10 | 1.00 | LC |
| Acetamiprid | 1.00 | LC | Flazasulfuron | 1.00 | LC | Pirimicarb desmethyl | 1.00 | LC | BAC12 | 1.00 | LC |
| Ametryn | 1.00 | LC | Flonicamid | 1.00 | LC | Prochloraz | 1.00 | LC | BAC14 | 1.00 | LC |
| Amidosulfuron | 1.00 | LC | Florasulam | 1.00 | LC | Promethryn | 1.00 | LC | BAC16 | 1.00 | LC |
| Atrazine | 1.00 | LC | Fluazifop-P-butyl | 1.00 | LC | Propamocarb | 1.00 | LC | DDAC | 1.00 | LC |
| Atrazine-desethyl | 1.00 | LC | Flucycloxuron | 1.00 | LC | Propaquizafop | 1.00 | LC | 2,4-D | 1.00 | LC |
| Atrazine-desisopropyl | 1.00 | LC | Fluopicolide | 1.00 | LC | Propazine | 1.00 | LC | 2,4-DB | 1.00 | LC |
| Azoxystrobin | 1.00 | LC | Fluopyram | 1.00 | LC | Propiconazole | 1.00 | LC | Asulam | 1.00 | LC |
| Benalaxyl | 1.00 | LC | Flutolanil | 1.00 | LC | Propyzamide | 1.00 | LC | Bentazone | 1.00 | LC |
| Bendiocarb | 1.00 | LC | Flutriafol | 1.00 | LC | Prothioconazole desthio | 1.00 | LC | Bromoxynil | 1.00 | LC |
| Bixafen | 1.00 | LC | Fluxapyroxad | 1.00 | LC | Pymetrozine | 1.00 | LC | Chlorfluazuron | 1.00 | LC |
| Boscalid | 1.00 | LC | Fosthiazate | 1.00 | LC | Pyraclostrobin | 1.00 | LC | Clethodim | 1.00 | LC |
| Bupirimate | 1.00 | LC | Imazalil | 1.00 | LC | Pyrethrins | 1.00 | LC | Clothianidin | 1.00 | LC |
| Carbendazim | 1.00 | LC | Imazamox | 1.00 | LC | Pyrimethanil | 1.00 | LC | Cyclanilide | 1.00 | LC |
| Carboxin | 1.00 | LC | Imazaquin | 1.00 | LC | Quizalofop | 1.00 | LC | Cycloxydim | 1.00 | LC |
| Chlorantraniliprole | 1.00 | LC | Imidacloprid | 1.00 | LC | Quizalofop-ethyl | 1.00 | LC | Dichlorprop-P | 1.00 | LC |
| Chlorotoluron | 1.00 | LC | Indoxacarb | 1.00 | LC | Rimsulfuron | 1.00 | LC | Diflubenzuron | 1.00 | LC |
| Chlorpropham | 1.00 | LC | Isoproturon | 1.00 | LC | Silthiofam | 1.00 | LC | Dinoseb | 1.00 | LC |
| Chlorpyriphos | 1.00 | LC | Lenacil | 1.00 | LC | Simazine | 1.00 | LC | Dinoterb | 1.00 | LC |
| Chlorpyriphos-Methyl | 1.00 | LC | Linuron | 1.00 | LC | Spinosad | 1.00 | LC | DNOC | 1.00 | LC |
| Clofentezine | 1.00 | LC | Mandipropamid | 1.00 | LC | Spiromesifen | 1.00 | LC | Endosulfan-sulfate | 1.00 | LC |
| Clopyralid | 1.00 | LC | Mepanipyrim | 1.00 | LC | Spirotetramat | 1.00 | LC | Fipronil | 1.00 | LC |
| Cyazofamid | 1.00 | LC | Metazachlor | 1.00 | LC | Spiroxamine | 1.00 | LC | Fipronil desulfinyl | 1.00 | LC |
| Cypermethrin | 1.00 | LC | Metconazole | 1.00 | LC | Tebuconazole | 1.00 | LC | Fipronil sulfide | 1.00 | LC |
| Cyproconazole | 1.00 | LC | Metolachlor | 1.00 | LC | Tebufenpyrad | 1.00 | LC | Fipronil sulfone | 1.00 | LC |
| Cyprodinil | 1.00 | LC | Metribuzin | 1.00 | LC | Terbutryn | 1.00 | LC | Fluazifop (free acid) | 1.00 | LC |
| Deltamethrin | 1.00 | LC | Myclobutanil | 1.00 | LC | Terbutylazine | 1.00 | LC | Fluazinam | 1.00 | LC |
| Desmedipham | 1.00 | LC | Napropamide | 1.00 | LC | Terbutylazine-2-hydroxy | 1.00 | LC | Fludioxonil | 1.00 | LC |
| Diazinon | 1.00 | LC | Omethoate | 1.00 | LC | Terbutylazine-desethyl | 1.00 | LC | Haloxyfop | 1.00 | LC |
| Diflufenican | 1.00 | LC | Oxadiazon | 1.00 | LC | Tetramethrin | 1.00 | LC | Iprodione | 1.00 | LC |
| Dimethoate | 1.00 | LC | Oxamyl | 1.00 | LC | Thiabendazole | 1.00 | LC | Hexaflumuron | 1.00 | LC |
| Dimethomorph | 1.00 | LC | Oxamyl Oxime | 1.00 | LC | Thiacloprid | 1.00 | LC | Ioxynil | 1.00 | LC |
| Diuron | 1.00 | LC | Oxyfluorfen | 1.00 | LC | Thiamethoxam | 1.00 | LC | MCPA | 1.00 | LC |
| Epoxyconazole | 1.00 | LC | Pencycuron | 1.00 | LC | Tolclofos-methyl | 1.00 | LC | MCPB | 1.00 | LC |
| EPTC | 1.00 | LC | Pendimethalin | 1.00 | LC | Triadimefon | 1.00 | LC | Mecoprop-P | 1.00 | LC |
| Ethofumesate | 1.00 | LC | Permethrin I | 1.00 | LC | Triadimenol | 1.00 | LC | Methoxyfenozide | 1.00 | LC |
| Famoxadone | 1.00 | LC | Phenmedipham | 1.00 | LC | Tri-Allat | 1.00 | LC | Quizalfop (free acid) | 1.00 | LC |
| Fenamidone | 1.00 | LC | Phorate | 1.00 | LC | Trichlorfon | 1.00 | LC | Sulfentrazone | 1.00 | LC |
| Fenhexamid | 1.00 | LC | Phorate Sulfoxide | 1.00 | LC | Trifloxystrobin | 1.00 | LC | Teflubenzuron | 1.00 | LC |
| Fenoxaprop-ethyl | 1.00 | LC | Phosalone | 1.00 | LC | Triticonazole | 1.00 | LC | Triclopyr | 1.00 | LC |
| Fenpropidin | 1.00 | LC | Picoxystrobin | 1.00 | LC | Zoxamide | 1.00 | LC | Triflumuron | 1.00 | LC |

*Additive/Contaminant/Food Contact Material Analysis*

| Analyte | Typical LOD (ppb) | Typical LOQ (ppb) | Method (e.g. GC/MS, ICP) | Method accredited Y / N |
|---|---|---|---|---|
| **ADDITIVES** | | | | |
| Nitrites/Nitrates | | | | |
| $NO_2$ | 2500 | 5000 | HPLC | Y |
| $NO_3$ | 2500 | 10000 | HPLC | Y |
| **METALS** | | | | |
| Aluminium | 100 | 333 | ICP-MS | Y |
| Lead | 5 | 17 | ICP-MS | Y |
| Cadmium | 5 | 17 | ICP-MS | Y |
| Mercury (Total) | 5 | 17 | ICP-MS | Y |
| Arsenic (Total) | 10 | 33 | ICP-MS | Y |
| Tin | 50 | 167 | ICP-MS | Y |
| Chromium | 30 | 100 | ICP-MS | Y |
| Selenium | 10 | 17 | ICP-MS | Y |
| Arsenic (Inorganic) | 10 | 33 | ICP-MS | Y |
| **MYCOTOXINS** | | | | |
| Aflatoxin B1 | 0.02 - 0.05 | 0.1 | HPLC | Y |
| Aflatoxin B2 | 0.02 - 0.05 | 0.1 | HPLC | Y |
| Aflatoxin G1 | 0.02 - 0.05 | 0.1 | HPLC | Y |
| Aflatoxin G2 | 0.02 - 0.05 | 0.1 | HPLC | Y |
| Total Aflatoxins | 0.08 - 0.032 | 0.4 | HPLC | Y |
| Aflatoxin M1 | | 0.005 - 0.1 | HPLC | Y |
| Ochratoxin A | 0.02 - 0.05 | 0.2 | HPLC | Y |
| Fumonisin B1 | 10 - 30 | 40 | HPLC | Y |
| Fumonisin B2 | 10 - 30 | 50 | HPLC | Y |
| DON | 10 - 30 | 50 | LC/MS | Y |
| 3-AcDON | 10 - 30 | 50 | LC/MS | Y |
| 15-AcDON | 10 - 30 | 50 | LC/MS | Y |
| DAS | 10 - 30 | 50 | LC/MS | Y |
| T2 | 10 - 30 | 50 | LC/MS | Y |
| HT2 | 10 - 30 | 50 | LC/MS | Y |
| Zearalenone | 2 - 5 | 10 | HPLC | Y |
| Patulin | 1.5 - 4 | 5 | HPLC | Y |
| **HALOGENS** | | | | |
| Iodine/iodide | 20 | 67 | ICP-MS | Y |
| **POLYCYCLIC HYDROCARBONS** | | | | |
| Benz[a]anthracene | 0.01 | 0.01 | GC-MS | Y |
| Benzo[b]fluoranthene | 0.02 | 0.02 | GC-MS | Y |
| Benzo[a]pyrene | 0.04 | 0.04 | GC-MS | Y |
| Chrysene | 0.04 | 0.04 | GC-MS | Y |
| SUM 4PAH | N/A | N/A | GC-MS | Y |
| **Phthalates** | | | | |
| DEHP | < 10 | < 33 | GC-MS | N |
| DBP | < 10 | < 33 | GC-MS | N |

| Analyte | Typical LOD (ppb) | Typical LOQ (ppb) | Method (e.g. GC/MS, ICP) | Method accredited Y / N |
|---|---|---|---|---|
| BBP | < 15 | < 50 | GC-MS | N |
| DIBP | < 15 | < 50 | GC-MS | N |
| DIDP | < 250 | < 830 | GC-MS | N |
| DINP | < 150 | < 500 | GC-MS | N |
| **Other** | | | | |
| Acrylamide | 5 | 30 | GC/MS | Y |
| Bisphenol A | 1 - 10 | 3.3 - 33 | LC-MS/MS | N |

## ANNEX II: ANALYTES INCLUDED IN TDS

| Analyte Abbreviations | | |
|---|---|---|
| Al = Aluminium | Se = Selenium | Trich = Trichothecenes |
| As = Arsenic | $NO_3$ = Nitrate | AA = Acrylamide |
| iAs = Inorganic Arsenic | $NO_2$ = Nitrite | PAHs = PAHs |
| Cd = Cadmium | OTA = Ochratoxin A | BPA=Bisphenol A |
| Cr = Chromium | FBs = Fumonisins | PHTH = Phthalates |
| Sn = Tin | AM1 = AflatoxinM1 | Pest = Pesticides |
| Pb = Lead | Pat = Patulin | |
| Hg = Mercury | AFs = Aflatoxins (B1, B2, G1, G2, Total) | |

| Food | Analytes |
|---|---|
| White flour | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, PAH, BPA, PHTH |
| Wholemeal flour | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, PAH, BPA, PHTH |
| White bread/rolls | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Granary/ Wholegrain breads | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| brown bread and rolls | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Plain Biscuits | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Chocolate Biscuits | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Other biscuits | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Cakes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Other cakes, buns and pastries | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Tap Water | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Pasta | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, PAH, BPA, PHTH |
| Rice | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, FUM, TRICH, PAH, BPA, PHTH |
| Cornflakes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Branflakes | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Wheat type Cereals | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Muesli | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Oat flakes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |

| Food | Analytes |
|---|---|
| Rice-type Cereals | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, FUM, TRICH, AA, PAH, BPA, PHTH |
| Whole Milk | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, FUM, AF, BPA, PHTH |
| Low-fat, Skimmed & Fortified Milks | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, FUM, AF, BPA, PHTH |
| Cream | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, BPA, PHTH |
| Cheese (hard) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, BPA, PHTH |
| Cheese (continental style) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, BPA, PHTH |
| Cheese (soft and semisoft) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, FUM, AF, BPA, PHTH |
| Yogurts | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, FUM, AF, BPA, PHTH |
| Custard | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, BPA, PHTH |
| Vanilla Ice-cream | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, FUM, AF, BPA, PHTH |
| Butter | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, BPA, PHTH |
| Dairy Spreads | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, FUM, AF, PAH, BPA, PHTH |
| Non-dairy Spreads | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, FUM, TRICH, PAH, BPA, PHTH |
| Other Ice-creams | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, FUM, AF, BPA, PHTH |
| Other Milk | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, FUM, TRICH, BPA, PHTH |
| Eggs (fried) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, BPA, PHTH |
| Pork | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Ham | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Pork Sausage | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, OTA, FUM, AA, PAH, BPA, PHTH |
| Bacon Rashers | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Beef | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Beef Mince | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Beef Burger | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, AA, PAH, BPA, PHTH |
| Chicken | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Turkey | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Lamb | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Offal (kidney) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, AF, OTA, ZEA, FUM, TRICH, PAH, BPA, PHTH |
| Offal (liver) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, AF, OTA, ZEA, TRICH, PAH, BPA, PHTH |
| Pudding (black and white) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, AF, OTA, ZEA, TRICH, AA, PAH, BPA, PHTH |
| Cod and Other White Fish | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Oily Fish Other than Salmon | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Salmon | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Canned Tuna | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Tinned Fish (excl. salmon & tuna) | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |

| Food | Analytes |
|---|---|
| Tinned Salmon | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Smoked Salmon | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Smoked Fish (excl. Salmon) | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Mussels | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Prawns | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Crab | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Potatoes without Skin (boiled) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH |
| Potatoes with Skin (microwaved) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AA, PAH |
| Chips (homemade, from frozen prepared) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AA, PAH, BPA, PHTH |
| Onion (fried) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Tomatoes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Canned Tomatoes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, BPA, PHTH |
| Tomato Canned/ Concentrate | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Peppers | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Cucumber | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Mushrooms | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Canned Sweetcorn | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, FUM, TRICH, BPA, PHTH |
| Carrots (boiled) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Carrots | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Celery | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Peas | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Canned Peas | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, BPA, PHTH |
| Green Beans | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Baked Beans | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, FUM, BPA, PHTH |
| Legumes (excl. peas) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, |
| Canned Legumes (excl. peas) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, BPA, PHTH |
| Cabbage (raw) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Cabbage (boiled) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Broccoli | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Cauliflower | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Root Vegetables (excl. carrots) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Stir Fry Vegetables | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Apples | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAT |
| Oranges | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Bananas | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Grapes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, FUM |
| Pears | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAT |

| Food | Analytes |
|---|---|
| Peaches and Nectarines | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Canned Peaches | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, BPA, PHTH |
| Plums | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Berries | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, PAT |
| Other Fruit | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Canned Fruit (other) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, BPA, PHTH |
| Dried Raisins | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, FUM, PAH, BPA, PHTH |
| Dried Fruit (other) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, PAH, BPA, PHTH |
| Nuts | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, PAH, BPA, PHTH |
| Seeds | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, FUM, PAH, BPA, PHTH |
| Herbs | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, PAH, |
| Spices | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, PAH, |
| Stock Cubes, Bovril & Marmite | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Soup, Fresh (tetrapak) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Soups (canned) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, PAH, BPA, PHTH |
| Soups (dried packet) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, AA, PAH, BPA, PHTH |
| Tomato Sauce | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Mayonnaise | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Gravy | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Cooking Sauces (other) | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Cooking Sauces Tomato-based | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Other Sauces and Condiments | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Soy Sauce | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Sugar & Sugar Substitutes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Marmalade | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Jam | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, FUM, PAT, PAH, BPA, PHTH |
| Honey | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Chocolate Confectionery | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, FUM, PAH, BPA, PHTH |
| Non-chocolate Confectionery | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Lager | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, AA, PAH, BPA, PHTH |
| Stout | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, AA, PAH, BPA, PHTH |
| White/Red Wine | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, PAH, BPA, PHTH |

| Food | Analytes |
|---|---|
| Spirits | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Alcoholic Drinks (apple-based) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAT, PAH, BPA, PHTH |
| Carbonated Soft Drinks | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Squashes | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Apple Juice | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAT |
| Orange Juice | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAT |
| Other fruit Juices | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, PAT, |
| Tea | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, PAH, BPA, PHTH |
| Instant Coffee | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, AA, PAH, BPA, PHTH |
| Filter Coffee | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, OTA, AA, PAH, BPA, PHTH |
| Herbal Tea | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, PAH, BPA, PHTH |
| Bottled Water | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, PAH, BPA, PHTH |
| Olive Oil | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, PAH, BPA, PHTH |
| Vegetable Oil | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, PAH, BPA, PHTH |
| Fat, Hard Cooking Fat | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, ZEA, TRICH, PAH, BPA, PHTH |
| Crisps | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, AA, PAH, BPA, PHTH |
| Other Savoury Snacks | Pest, Al, Cr, As, iAs, Se, Cd, Sn, Hg, Pb, I, $NO_3$, $NO_2$, AA, PAH, BPA, PHTH |
| Pizza) | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, AF, OTA, ZEA, TRICH, AA, PAH, BPA, PHTH |
| Lettuce | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$, |
| Other Leafy Vegetables | Pest, Al, Cr, As, Se, Cd, Sn, Hg, Pb, I, $NO_3$ |
| Soya Milk (composite) | PAH |
| Fresh Vegetable (composite) | PAH, BPA, PHTH |
| Tinned Vegetable (composite) | PAH |
| Fresh Fruit (composite) | PAH, BPA, PHTH |
| Canned Fruit (composite) | PAH |

## ANNEX III: ANALYTICAL RESULTS

Analytical results are reported per food category, providing the range of minimum LB to maximum UB values covering the food groups within each category. Table 24 shows the classification of food groups into food categories, for which results have been summarised in Table 25.

### Table 24. TDS Food Groups per Food Category

| Food Category | TDS Food Group | Food Category | TDS Food Group |
|---|---|---|---|
| **Cereals** | White flour | **Breakfast Cereals** | Cornflakes |
| | Wholemeal flour | | Branflakes |
| | White bread/rolls | | Wheat type Cereals |
| | Granary/Wholegrain breads | | Muesli |
| | brown bread and rolls | | Oat flakes |
| | Plain biscuits | | Rice type cereals |
| | Chocolate biscuits | **Dairy Alternatives** | Other ice-creams |
| | Other biscuits | | Other milk |
| | Cakes | | Soya milk (composite) |
| | Other cakes, buns and pastries | **Eggs** | Eggs (fried) |
| | Pasta | **Meat and Meat Products** | Pork |
| | Rice | | Ham |
| **Dairy** | Whole milk | | Pork sausage |
| | Low-fat, skimmed & fortified milks | | Bacon rashers |
| | Cream | | Beef |
| | Cheese (hard) | | Beef mince |
| | Cheese (continental style) | | Beef burger |
| | Cheese (soft and semi-soft) | | Chicken |
| | Yogurts | | Turkey |
| | Custard | | Lamb |
| | Vanilla ice-cream | | Offal (kidney) |
| | Butter | | Offal (liver) |
| | Dairy spreads | | Pudding (black and white) |

## Table 24 continued. TDS Food Groups per Food Category

| Food Category | TDS Food Group | Food Category | TDS Food Group |
|---|---|---|---|
| **Fish and Fishery Products** | Cod and other white fish | **Fish and Fishery Products (canned)** | Canned tuna |
| | Oily fish other than salmon | | Tinned fish (ex salmon & tuna) |
| | Salmon | | Tinned salmon |
| | Smoked salmon | **Vegetables (canned)** | Canned tomatoes |
| | Smoked fish (ex salmon) | | Tomato canned/ concentrate |
| | Mussels | | Canned sweetcorn |
| | Prawns | | Canned peas |
| | Crab | | Baked beans |
| **Vegetables** | Potatoes without skin (boiled) | | Canned legumes (ex peas) |
| | Potatoes with skin (microwaved) | | Tinned vegetable (composite) |
| | Chips (homemade, from frozen pre-prepared) | **Fruit** | Fresh fruit (composite) |
| | Onion (fried) | | Apples |
| | Tomatoes | | Oranges |
| | Peppers | | Bananas |
| | Cucumber | | Grapes |
| | Mushrooms | | Pears |
| | Carrots (boiled) | | Peaches and nectarines |
| | Carrots | | Plums |
| | Celery | | Berries |
| | Peas | | Other fruit |
| | Green beans | **Fruit (dried)** | Dried raisins |
| | Legumes (ex peas) | | Dried fruit (ex raisins) |
| | Cabbage (raw) | **Fruit (canned)** | Canned fruit (composite) |
| | Cabbage (boiled) | | Canned peaches |
| | Broccoli | | Canned fruit (ex peaches) |
| | Cauliflower | **Nuts** | Nuts |
| | Root vegetables (ex carrots) | **Seeds** | Seeds |
| | Stir-fry vegetables | **Herbs & Spices** | Herbs |
| | Lettuce | | Spices |
| | Other leafy vegetables | **Soups, Condiments and Sauces (canned)** | Soups (canned) |
| | Fresh vegetable (composite) | **Sugars, Preseves and Confectionery** | Sugar & sugar substitutes |

## Table 24 continued. TDS Food Groups per Food Category

| Food Category | TDS Food Group | Food Category | TDS Food Group |
|---|---|---|---|
| **Soups, Condiments and Sauces** | Stock cubes, Bovril & Marmite | **Sugars, Preseves and Confectionery** | Marmalade |
| | Soup, fresh (Tetrapak) | | Jam |
| | Soups (dried packet) | | Honey |
| | Tomato sauce | | Chocolate confectionery |
| | Mayonnaise | | Non-chocolate confectionery |
| | Gravy | **Fats & Oils** | Olive oil |
| | Cook-in sauces (other) | | Vegetable oil |
| | Cook-in sauces tomato-based | | Fat, hard cooking fat |
| | Other sauces and condiments | | Non-dairy spreads |
| | Soy sauce | **Snacks** | Crisps |
| **Alcoholic Beverages** | Lager | | Other savoury snacks |
| | Stout | **Composite** | Pizza |
| | White/Red wine | **Non-alcoholic Beverages** | Carbonated soft drinks |
| | Spirits | | Squashes |
| | Alcoholic drinks (apple based) | | Apple juice |
| **Tea & Coffee** | Tea | | Orange juice |
| | Instant coffee | | Other fruit juices |
| | Filter coffee | **Water** | Bottled water |
| | Herbal tea | | Tap water |

## Table 25 Min (LB) – Max (UB) range of results for analytes covered in the TDS

| Food Groups | Aluminium (mg/kg) | | Chromium (mg/kg) | | Arsenic (mg/kg) | | Inorganic Arsenic (mg/kg) | | Selenium (mg/kg) | | Cadmium (mg/kg) | | Tin (mg/kg) | | Mercury (mg/kg) | | Lead (mg/kg) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) |
| Cereals | 0.60 | 47.1 | 0 | 0.15 | 0 | 0.03 | 0 | 0.02 | 0.02 | 0.10 | 0 | 0.14 | 0 | 0.10 | 0 | 0.01 | 0 | 0.01 |
| Water | 0 | 0.03 | 0 | 0.01 | 0 | 0.00 | | | 0 | 0.00 | 0 | 0.001 | 0 | 0.01 | 0 | 0.001 | 0 | 0.001 |
| Breakfast Cereals | 0 | 7.1 | 0 | 0.11 | 0 | 0.15 | 0 | 0.06 | 0 | 0.08 | 0 | 0.10 | 0 | 0.10 | 0 | 0.01 | 0 | 0.01 |
| Dairy | 0 | 1.4 | 0 | 0.10 | 0 | 0.02 | | | 0 | 0.13 | 0 | 0.01 | 0 | 0.10 | 0 | 0.01 | 0 | 0.01 |
| Dairy Alternatives | 0.59 | 3.3 | 0.05 | 0.07 | 0 | 0.01 | | | 0.01 | 0.02 | 0.00 | 0.01 | 0 | 0.05 | 0 | 0.01 | 0 | 0.01 |
| Eggs | 3.0 | 3.0 | 0 | 0.03 | 0 | 0.01 | | | 0.29 | 0.29 | 0 | 0.01 | 0 | 0.05 | 0 | 0.01 | 0 | 0.01 |
| Meat and Meat Products | 0 | 18.0 | 0 | 0.44 | 0 | 4.1 | 0 | 0.01 | 0 | 0.43 | 0 | 0.06 | 0 | 0.05 | 0 | 0.11 | 0 | 0.03 |
| Fish and Fishery Products | 0.10 | 71.7 | 0 | 0.15 | 0.35 | 4.1 | 0 | 0.05 | 0.17 | 0.85 | 0 | 0.10 | 0 | 0.05 | 0.01 | 0.11 | 0 | 0.22 |
| Fish and Fishery Products (canned) | 0.70 | 8.1 | 0 | 0.06 | 0.39 | 2.0 | 0 | 0.01 | 0.29 | 0.73 | 0 | 0.02 | 0 | 0.07 | 0.02 | 0.15 | 0 | 0.02 |
| Vegetables | 0 | 4.0 | 0 | 0.15 | 0 | 0.02 | 0 | 0.01 | 0 | 0.11 | 0 | 0.07 | 0 | 0.05 | 0 | 0.01 | 0 | 0.01 |
| Vegetables (canned) | 0 | 9.5 | 0 | 0.28 | 0 | 0.02 | | | 0 | 0.02 | 0 | 0.06 | 0 | 29.6 | 0 | 0.01 | 0 | 0.03 |
| Fruit | 0.09 | 1.2 | 0 | 0.03 | 0 | 0.01 | | | 0 | 0.01 | 0 | 0.01 | 0 | 0.05 | 0 | 0.01 | 0 | 0.01 |
| Fruit (dried) | 3.0 | 23.6 | 0 | 0.16 | 0 | 0.02 | 0 | 0.02 | 0 | 0.02 | 0 | 0.01 | 0 | 0.10 | 0 | 0.01 | 0 | 0.02 |
| Fruit (canned) | 0.13 | 0.47 | 0.08 | 0.09 | 0 | 0.01 | | | 0 | 0.01 | 0 | 0.00 | 78.8 | 124 | 0 | 0.00 | 0.02 | 0.11 |
| Nuts | 2.0 | 2.0 | 0 | 0.06 | 0 | 0.02 | | | 0.20 | 0.20 | 0.01 | 0.01 | 0 | 0.10 | 0 | 0.01 | 0 | 0.01 |
| Seeds | 24.9 | 24.9 | 0.21 | 0.21 | 0 | 0.02 | 0.02 | 0.02 | 0.35 | 0.35 | 0.12 | 0.12 | 0 | 0.10 | 0 | 0.01 | 0.01 | 0.01 |
| Herbs & Spices | 139 | 639 | 0.58 | 2.1 | 0.04 | 0.19 | 0.03 | 0.16 | 0.09 | 0.14 | 0.09 | 0.12 | 0 | 0.20 | 0.01 | 0.02 | 0.10 | 0.51 |
| Soups, Condiments and Sauces | 0.10 | 5.4 | 0 | 0.12 | 0 | 0.01 | 0.01 | 0.01 | 0 | 0.04 | 0 | 0.02 | 0 | 0.05 | 0 | 0.01 | 0 | 0.01 |
| Soups, Condiments and Sauces (canned) | 0.94 | 0.94 | 0.03 | 0.03 | 0 | 0.01 | | | 0.03 | 0.03 | 0.01 | 0.01 | 22.9 | 22.9 | 0 | 0.00 | 0 | 0.00 |
| Sugars, Preseves and Confectionery | 0 | 5.2 | 0 | 0.20 | 0 | 0.02 | | | 0 | 0.03 | 0 | 0.01 | 0 | 0.10 | 0 | 0.01 | 0 | 0.01 |
| Alcoholic Beverages | 0.08 | 0.60 | 0 | 0.06 | 0 | 0.01 | | | 0 | 0.39 | 0 | 0.02 | 0 | 0.01 | 0 | 0.00 | 0 | 0.01 |
| Non-alcoholic Beverages | 0.13 | 0.53 | 0 | 0.11 | 0 | 0.00 | | | 0 | 0.00 | 0 | 0.001 | 0 | 0.01 | 0 | 0.001 | 0 | 0.00 |
| Tea & Coffee | 0.05 | 3.4 | 0 | 0.05 | 0 | 0.00 | | | 0 | 0.00 | 0 | 0.001 | 0 | 0.01 | 0 | 0.001 | 0 | 0.00 |
| Water | 0 | 0.03 | 0 | 0.01 | 0 | 0.00 | | | 0 | 0.00 | 0 | 0.001 | 0 | 0.01 | 0 | 0.001 | 0 | 0.001 |
| Fats & Oils | 0 | 0.20 | 0 | 0.06 | 0 | 0.02 | | | 0 | 0.02 | 0 | 0.01 | 0 | 0.10 | 0 | 0.01 | 0 | 0.01 |
| Snacks | 3.0 | 3.9 | 0.07 | 0.07 | 0 | 0.02 | 0 | 0.02 | 0 | 0.02 | 0.02 | 0.08 | 0 | 0.10 | 0 | 0.01 | 0 | 0.01 |

## Table 25 continued Min (LB) – Max (UB) range of results for analytes covered in the TDS

| Food Groups | Iodine (mg/kg) | | Nitrate (mg/kg) | | Nitrite (mg/kg) | | AFB1 (µg/kg) | | AFB2 (µg/kg) | | AFG1 (µg/kg) | | AFG2 (µg/kg) | | AFTotal (µg/kg) | | AFM1 (µg/kg) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) |
| Cereals | 0 | 0.12 | 0 | 14.3 | | | 0 | 1.5 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Water | 0.00 | 0.01 | 0 | 10.0 | | | | | | | | | | | | | | |
| Breakfast Cereals | 0 | 0.13 | 0 | 10.0 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Dairy | 0.05 | 0.45 | 0 | 10.0 | | | | | | | | | | | | | 0 | 0.02 |
| Dairy Alternatives | 0.08 | 0.23 | 0 | 10.0 | | | 0 | 0.01 | 0 | 0.01 | 0 | 0.01 | 0 | 0.01 | 0 | 0.02 | 0 | 0.02 |
| Eggs | 0.47 | 0.47 | 0 | 10.0 | | | | | | | | | | | | | | |
| Meat and Meat Products | 0 | 0.75 | 0 | 75.5 | 0 | 7.3 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Fish and Fishery Products | 0.05 | 3.5 | 0 | 10.0 | | | | | | | | | | | | | | |
| Fish and Fishery Products (canned) | 0.13 | 0.74 | 0 | 10.0 | | | | | | | | | | | | | | |
| Vegetables | 0 | 0.03 | 0 | 2312 | | | | | | | | | | | | | | |
| Vegetables (canned) | 0 | 0.01 | 0 | 10.0 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Fruit | 0 | 0.01 | 0 | 143 | | | | | | | | | | | | | | |
| Fruit (dried) | 0.02 | 0.03 | 0 | 10.0 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Fruit (canned) | 0.01 | 0.16 | 0 | 10.0 | | | | | | | | | | | | | | |
| Nuts | 0 | 0.01 | 0 | 10.0 | | | 0.20 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Seeds | 0 | 0.02 | 82.5 | 82.5 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Herbs & Spices | 0.07 | 0.29 | 247 | 1432 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Soups, Condiments and Sauces | 0 | 0.12 | 0 | 55.0 | 0 | 5.0 | | | | | | | | | | | | |
| Soups, Condiments and Sauces (canned) | 0.01 | 0.01 | 12.7 | 12.7 | 0 | 5.0 | | | | | | | | | | | | |
| Sugars, Preseves and Confectionery | 0 | 0.43 | 0 | 41.8 | | | | | | | | | | | | | | |
| Alcoholic Beverages | 0 | 0.01 | 0 | 10.0 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Non-alcoholic Beverages | 0 | 0.02 | 0 | 10.0 | | | | | | | | | | | | | | |
| Tea & Coffee | 0 | 0.00 | 0 | 30.8 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Water | 0.00 | 0.01 | 0 | 10.0 | | | | | | | | | | | | | | |
| Fats & Oils | 0 | 0.02 | 0 | 10.0 | | | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |
| Snacks | 0.03 | 0.03 | 58.3 | 169 | 0 | 5.0 | | | | | | | | | | | | |
| Composite | 0.14 | 0.14 | 10.6 | 10.6 | | | 0.20 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.20 | 0 | 0.80 | | |

## Table 25 continued Min (LB) – Max (UB) range of results for analytes covered in the TDS

| Food Groups | OA (µg/kg) (µg/kg) | | Patulin (µg/kg) | | ZEA (µg/kg) | | Fumonisins (FB1-FB3) (µg/kg) | | Other Fusarium Toxins (µg/kg) (DON, NIV, 3-Ac Don, 15-AcDON, FUS-X,DAS, T-2, HT-2) | | Acrylamide (µg/kg) | | PAH 4 (µg/kg) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) |
| **Cereals** | 0 | 1.5 | | | 0 | 10.0 | 0 | 20.0 | 0 | 50.0 | 0 | 365 | 0 | 3.7 |
| **Water** | | | | | | | | | | | | | 0 | 0.07 |
| **Breakfast Cereals** | 0 | 0.30 | | | 0 | 10.0 | 0 | 20.0 | 0 | 50.0 | 0 | 198 | 0 | 0.60 |
| **Dairy** | | | | | | | 0 | 20.0 | | | | | 0.16 | 0.30 |
| **Dairy Alternatives** | | | | | 0 | 10.0 | 0 | 20.0 | 0 | 50.0 | | | 0 | 0.04 |
| **Eggs** | | | | | | | | | | | | | | |
| **Meat and Meat Products** | 0 | 0.20 | | | 0 | 10.0 | 0 | 20.0 | 0 | 50.0 | 0 | 30.7 | 0 | 0.27 |
| **Fish and Fishery Products** | | | | | | | | | | | | | 0 | 2.9 |
| **Fish and Fishery Products (canned)** | | | | | | | | | | | | | 0 | 0.57 |
| **Vegetables** | | | | | | | | | | | 0 | 88.1 | 0 | 0.25 |
| **Vegetables (canned)** | 0 | 0.20 | | | 0 | 10.0 | 0 | 20.0 | 0 | 50.0 | | | 0 | 0.24 |
| **Fruit** | 0 | 0.20 | 0 | 21.3 | | | 0 | 20.0 | | | | | 0 | 0.23 |
| **Fruit (dried)** | 0 | 0.20 | | | | | 0 | 20.0 | | | | | 0.04 | 0.29 |
| **Fruit (canned)** | | | | | | | | | | | | | 0 | 0.17 |
| **Nuts** | 0 | 0.20 | | | | | | | | | | | 0.34 | 0.34 |
| **Seeds** | 1.8 | 1.8 | | | | | 0 | 20.0 | | | | | 1.8 | 1.8 |
| **Herbs & Spices** | 0 | 0.20 | | | 0 | 10.0 | | | 0 | 50.0 | | | 6.9 | 7.0 |
| **Soups, Condiments and Sauces** | | | | | | | | | | | 0 | 30.0 | 0 | 0.40 |
| **Soups, Condiments and Sauces (canned)** | | | | | | | | | | | | | 0 | 0.09 |
| **Sugars, Preseves and Confectionery** | 0 | 0.20 | 0 | 5.0 | | | 0 | 20.0 | | | | | 0 | 0.83 |
| **Alcoholic Beverages** | 0 | 0.20 | 17.0 | 17.0 | 0 | 10.0 | | | 0 | 50.0 | 0 | 30.0 | 0 | 0.08 |
| **Non-alcoholic Beverages** | 0 | 0.20 | 0 | 31.9 | | | | | | | | | 0 | 0.05 |
| **Tea & Coffee** | 0 | 0.20 | | | 0 | 10.0 | | | 0 | 50.0 | 0 | 30.0 | 0 | 0.07 |
| **Water** | | | | | | | | | | | | | 0 | 0.07 |
| **Fats & Oils** | | | | | 0 | 10.0 | | | 0 | 50.0 | | | 0.71 | 3.1 |
| **Snacks** | | | | | | | | | | | 251 | 765 | 0.36 | 0.62 |
| **Composite** | 0.20 | 0.20 | | | 0 | 10.0 | | | 0 | 50.0 | 0 | 30.0 | 0.27 | 0.27 |

## Table 25 continued Min (LB) – Max (UB) range of results for analytes covered in the TDS

| Food Groups | DiBP (µg/kg) | | DBP (µg/kg) | | BBP (µg/kg) | | DEHP (µg/kg) | | DiNP (µg/kg) | | DiDP (µg/kg) | | BPA (µg/kg) | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) | Min (LB) | Max (UB) |
| Cereals | 0 | 29.9 | 0 | 16.5 | 0 | 24.4 | 0 | 86.4 | 0 | 490 | 0 | 440 | 0 | 4.3 |
| Water | 0 | 37.4 | 0 | 21.6 | 0 | 8.7 | 0 | 14.5 | 0 | 14.5 | 0 | 14.3 | 0 | 0.56 |
| Breakfast Cereals | 0 | 61.4 | 0 | 37.7 | 0 | 4.4 | 0 | 69.9 | 0 | 361 | 0 | 81.2 | 0 | 1.0 |
| Dairy | 0 | 11.4 | 0 | 39.7 | 0 | 7.4 | 0 | 460 | 0 | 818 | 0 | 1669 | 0 | 599 |
| Dairy Alternatives | 0 | 71.8 | 0 | 11.9 | 0 | 7.9 | 0 | 79.1 | 0 | 297 | 0 | 182 | 0 | 2.0 |
| Eggs | 0 | 6.7 | 0 | 3.6 | | | 0 | 17.2 | | | | | 0 | 1.0 |
| Meat and Meat Products | 0 | 9.3 | 0 | 18.0 | 0 | 30.5 | 0 | 74.2 | 0 | 3635 | 0 | 321 | 0 | 19.8 |
| Fish and Fishery Products | | | | | | | | | | | | | | |
| Fish and Fishery Products (canned) | 0 | 9.3 | 0 | 18.0 | 0 | 17.9 | 0 | 77.4 | 0 | 484 | 0 | 226 | 3.2 | 44.0 |
| Vegetables | 0 | 6.1 | 0 | 8.4 | 0 | 5.8 | 0 | 40.4 | 0 | 62.6 | 0 | 61.0 | 0 | 1.3 |
| Vegetables (canned) | 0 | 20.4 | 0 | 14.9 | 0 | 4.8 | 0 | 88.6 | 0 | 49.6 | 0 | 54.0 | 0 | 45.5 |
| Fruit | 0 | 6.1 | 0 | 8.4 | 16.0 | 16.0 | 0 | 40.4 | 0 | 90.1 | 0 | 73.6 | 0 | 0.81 |
| Fruit (dried) | 0 | 15.5 | 0 | 14.9 | 0 | 5.2 | 0 | 88.6 | 0 | 82.6 | 0 | 70.9 | 0 | 0.40 |
| Fruit (canned) | 0 | 15.5 | 0 | 14.9 | 0 | 6.2 | 0 | 88.6 | 0 | 78.7 | 0 | 42.9 | 0.30 | 4.2 |
| Nuts | 0 | 15.5 | 9.2 | 9.2 | 0 | 12.8 | 303 | 303 | 545 | 545 | 0 | 76.7 | 0 | 0.45 |
| Seeds | 23.0 | 23.0 | 26.1 | 26.1 | 0 | 11.6 | 180 | 180 | 1033 | 1033 | 0 | 733 | 0 | 0.40 |
| Herbs & Spices | | | | | | | | | | | | | | |
| Soups, Condiments and Sauces | 0 | 32.7 | 0 | 16.6 | 0 | 24.4 | 0 | 41.8 | 0 | 441 | 0 | 404 | 0 | 29.8 |
| Soups, Condiments and Sauces (canned) | 0 | 3.9 | 0 | 5.8 | 0 | 5.4 | 0 | 30.5 | 0 | 132 | 0 | 92.0 | 47.9 | 47.9 |
| Sugars, Preseves and Confectionery | 0 | 14.3 | 0 | 16.6 | 0 | 12.2 | 0 | 62.2 | 0 | 222 | 0 | 242 | 0 | 0.40 |
| Alcoholic Beverages | 0 | 16.1 | 0 | 89.0 | 0 | 10.3 | 0 | 64.5 | 0 | 93.5 | 0 | 29.8 | 0 | 11.1 |
| Non-alcoholic Beverages | 0 | 16.1 | 0 | 10.3 | 0 | 3.8 | 0 | 8.2 | 0 | 42.9 | 0 | 28.6 | 0 | 5.9 |
| Tea & Coffee | 0 | 37.4 | 0 | 21.6 | 0 | 5.7 | 0 | 14.5 | 0 | 30.0 | 0 | 29.6 | 0 | 12.8 |
| Water | 0 | 37.4 | 0 | 21.6 | 0 | 8.7 | 0 | 14.5 | 0 | 14.5 | 0 | 14.3 | 0 | 0.56 |
| Fats & Oils | 0 | 37.4 | 0 | 21.6 | 0 | 19.7 | 0 | 260 | 0 | 979 | 0 | 537 | 0 | 3.9 |
| Snacks | 0 | 37.4 | 0 | 21.6 | 0 | 5.7 | 0 | 48.3 | 0 | 218 | 0 | 177 | 0 | 0.40 |
| Composite | 0 | 37.4 | 0 | 21.6 | 0 | 4.0 | 65.6 | 65.6 | 4745 | 4745 | 0 | 455 | 0 | 5.0 |


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