25th Australian Total Diet Study

June 2019

Foreword

Food Standards Australia New Zealand (FSANZ) is an independent Australian Government agency responsible for ensuring a safe food supply that protects and supports the health of people in Australia and New Zealand. FSANZ is responsible for developing food standards and sets limits in the Australia New Zealand Food Standards Code for chemicals when it is appropriate to do so.

To determine the level of chemicals in food, FSANZ conducts food surveys, gathering data and estimating the dietary exposure of the Australian population to these chemicals. The Australian Total Diet Study (ATDS) is the most comprehensive analytical food survey conducted in Australia for this purpose. If needed, risk management options are considered to manage any risks identified by the ATDS.

The first ATDS, formerly known as the 'Australian Market Basket Survey', was conducted in 1970 by the National Health and Medical Research Council (NHMRC). Since then, the Australian Government has conducted regular surveys estimating consumer exposure to chemicals in the food supply, with the last nine studies managed by FSANZ. The first 20 studies examined dietary exposure to pesticide residues and contaminants.

The ATDS has evolved over the past 40 years in its scope and frequency, with more recent studies focussing on a wider range of food chemicals such as additives, nutrients, processing contaminants and food packaging chemicals. Broadening the scope of the ATDS has been invaluable in gathering data to assess the dietary exposure of the Australia population to a wider range of food chemicals, and determining and managing whether there are any public health concerns.

The 25th ATDS has a more traditional focus, looking at agricultural and veterinary chemicals, and metal contaminants.

I extend my thanks to the staff of FSANZ and other agencies who have contributed to a successful outcome. I am pleased to present the 25th ATDS as part of FSANZ's commitment to the ongoing monitoring of the Australian food supply, ensuring it continues to be one of the safest food supplies in the world.

Ms Robyn Kruk AO

Chair

The appendices are available on the Food Standards Australia New Zealand website www.foodstandards.gov.au.

Acknowledgments

FSANZ would like to thank:

- officers from state and territory food regulatory agencies for collecting samples and dispatching them to the laboratory
- Symbio Laboratories and Hill Laboratories for preparing and analysing samples

Glossary

Abbreviations used in the report	
2011-12 NNPAS	2011-12 Australian National Nutrition and Physical Activity Survey
ACT	Australian Capital Territory
ADI	Acceptable Daily Intake
ALARA	As Low As Reasonably Achievable
APVMA	Australian Pesticides and Veterinary Medicine Authority
ARPANSA	Australian Radiation Protection and Nuclear Safety Agency
ATDS	Australian Total Diet Study
AUSNUT	AUStralian Food and NUTrient Database
BMD	Benchmark Dose
BMDL	Benchmark Dose Lower Confidence Limit
Bw	Body weight
CCCF	Codex Committee on Contaminants in Food
CCFA	Codex Committee on Food Additives
ECDLO	Electron Capture Detection
EFSA	European Food Safety Authority
EWG	Electronic Working Group
FAO	Food and Agricultural Organisation of the United Nations
FSANZ	Food Standards Australia New Zealand
GC	Gas Chromatography
GEMS	Global Environmental Monitoring System
HBGV	Health Based Guidance Values
Hg	Mercury
HPLC	High Performance Liquid Chromatography
IANZ	International Accreditation New Zealand
ICP	Inductively Coupled Plasma
ISFR	Implementation Subcommittee for Food Regulation
IQ	Intelligence Quotient
JECFA	Joint FAO/WHO Expert Committee on Food Additives
JMPR	Joint FAO/WHO Meeting on Pesticide Residues
LC	Liquid Chromatography

Abbreviations used in the report	
LOD	Limit of Detection
LOQ	Limit of Quantitation
LOR	Limit of Reporting
ML	Maximum Limit
MOE	Margin of Exposure
MRL	Maximum Residue Limit
MS	Mass Spectrometry
NATA	National Association of Testing Authorities, Australia
nd	Non-detected
NHMRC	National Health and Medical Research Council
NOAEL	No Observed Adverse Effect Level
NRS	National Residues Survey
P50	50th percentile (median)
P90	90th percentile
PFPD	Pulse Flame Photometric Detector
PMTDI	Provisional Maximum Tolerable Daily Intake
PTMI	Provisional Tolerable Monthly Intake
PTWI	Provisional Tolerable Weekly Intake
The Code	Australia New Zealand Food Standards Code
USA	United States of America
WHO	World Health Organization

25th Australian Total Diet Study (ATDS) key findings

Overview

- The 25th ATDS investigated levels of agricultural and veterinary chemicals, and metal contaminants arsenic, cadmium, lead and mercury, in a broad range of foods.

- Dietary exposure was estimated for the general Australian population and there are no public health and safety concerns for most substances.

- FSANZ has identified areas for future work including possible risk management options to ensure that the Australian food supply remains safe.

Agricultural and veterinary chemicals

- Concentrations of agricultural and veterinary chemicals were generally low, with a large proportion of food samples containing no detectable residues.

- Estimated dietary exposures for all but one of these chemicals were below the relevant acceptable daily intakes (ADIs) indicating no public health and safety concerns.

- Estimated dietary exposures for the organophosphorus insecticide prothiofos exceeded the ADI for some population age groups. FSANZ notified the Australian Pesticides and Veterinary Medicines Authority (APVMA), the Australian government regulator of agricultural and veterinary chemicals. The APVMA subsequently worked with industry who voluntarily changed the way prothiofos is used to ensure that risks for Australian consumers are acceptably low.

Metal contaminants

- Concentrations of arsenic, cadmium, lead and mercury, and estimated dietary exposure for the Australian population were consistent with those reported in the international scientific literature.

Inorganic arsenic

- There is no health-based guidance value (HBGV) for inorganic arsenic as international assessments have been unable to establish a safe level of human exposure.

- Estimated dietary exposures to inorganic arsenic were calculated with data for a limited number of foods. Major dietary contributors are rice and rice products; fish and seafood, including crustacea and sushi, and infant cereal products.

- Dietary exposures to inorganic arsenic were determined to be below levels associated with adverse health effects.

Cadmium

- Major dietary contributors to cadmium exposure are root vegetables, savoury snacks including crisps, grain type breads, cakes and baked goods, and berries.

- Dietary exposures to cadmium were compared to the Provisional Tolerable Monthly Intake (PTMI), which determined that there are no public health and safety concerns for Australian consumers.

Lead

- There is no HBGV for lead as international assessments have been unable to establish a safe level of human exposure.

- Major dietary contributors to lead exposure are wide ranging and include water, sweetened soft drinks, baked goods, some dried and tinned fruits, pork, some deli meats, honey, chocolates and fudge.

- Dietary exposures to lead for most Australian consumers are lower than levels considered to be of negligible risk of causing adverse health effects in human populations. For this reason, risks for Australian consumers are considered to be acceptably low.

Mercury

- Seafood is the major dietary contributor to inorganic mercury and organic (methyl)mercury exposure.

Inorganic mercury

- Dietary exposures to inorganic mercury were compared to the Provisional Tolerable Weekly Intake (PTWI), which determined that there are no public health and safety concerns for Australian consumers.

Methylmercury

- Estimated dietary exposures to methylmercury were below the PTWI for all population age groups except 2 to 5 year olds. The most sensitive subgroup–women of child bearing age–had dietary exposure below the PTWI.

- These results indicate that dietary exposures for most Australian consumers are acceptably low. Exceedances of the PTWI for children aged 2 to 5 years should be considered in the context of the known benefits of fish consumption.

- FSANZ already publishes consumer advice to manage dietary exposure to mercury through fish consumption while highlighting the health benefits of fish consumption. This advice will continue to be updated as required to reflect future work on the issue.

Conclusion

- The 25th ATDS confirms the current safety of the Australian food supply for the general population in relation to the levels of agricultural and veterinary chemicals and selected metal contaminants in a broad range of foods.

- FSANZ has identified a number of areas for further work including risk management options to ensure that the Australian food supply remains safe. As part of this, FSANZ will continue to monitor domestic and international developments related to chemicals in food to prioritise future survey work as required.

- For contaminants, in particular, focus will be given to foods known to contribute significantly to dietary exposure, including for those groups identified as being at higher risk (such as infants and children).

Executive summary

The 25th ATDS investigated a wide range of Australian foods for the presence of a number of agricultural and veterinary chemicals, and four metal contaminants (arsenic, cadmium, lead and mercury). A total of 88-different food types were sampled from all Australian states and territories over two sampling periods (May 2013 and February 2014).

Concentrations of agricultural and veterinary chemicals and metal contaminants were generally low, with a large proportion of food samples containing no detectable residues. Detections of agricultural and veterinary chemicals were generally consistent with approved conditions of use. Metal contaminants were detected in a limited number of food samples associated with the known environmental distribution of these substances, including seafood. Some cereal and vegetable crops, such as rice and starchy vegetables, were also determined to contain relatively higher concentrations of arsenic and cadmium, respectively. Levels of metal contaminants were also comparable, or lower, than those found internationally.

Dietary exposure for various age groups representing the general Australian population was estimated by multiplying food chemical concentrations analysed in this study by food consumption amounts recorded in the most recent Australian National Nutrition Survey. These estimated dietary exposures were compared to HBGVs to help characterise the risks for Australian consumers. In cases where no HBGVs could be established, estimated dietary exposure was compared to health-based endpoints using the Margin of Exposure (MOE) approach.

Estimated dietary exposures were below the relevant acceptable daily intakes (ADIs) for all but one agricultural and veterinary chemical indicating that there are no public health and safety concerns. However, as a result of several detections in table grapes, estimated dietary exposures to the organophosphorus insecticide prothiofos exceeded the ADI for mean consumers aged 2 to 5 and 6 to 12 years. All consumers for population sub-groups above 2-years exceeded the ADI at the 90th percentile of exposure. FSANZ notified the APVMA, the regulator of agricultural and veterinary chemical use in Australia, of these results. The APVMA subsequently commenced consultation with industry who have voluntarily cancelled label approvals for the use of prothiofos on grapes. This industry driven outcome ensures that risks for Australian consumers associated with potential prothiofos exposure are acceptably low.

Many metal contaminants occur naturally in the environment which means dietary exposure is largely unavoidable. The Food Standards Code (the Code) contains limits for these contaminants in specific commodities that are major contributors to dietary exposure to ensure that levels are kept as low as reasonably achievable (ALARA) in order to protect public health and safety. Results for metal contaminants indicated a high level of consistency with our regulatory standards and were generally lower or comparable with levels determined in previous ATDS surveys and internationally.

In this study, estimated dietary exposures to arsenic (and the more toxicologically relevant inorganic arsenic) were consistent with, or lower than levels reported internationally. For inorganic arsenic, dietary exposures were estimated based on analytical data for a limited range of foods expected to contribute most significantly to dietary exposure and were determined to be below levels associated with adverse health effects. Due to limitations in the scope of the analytical testing program and uncertainties associated with the available toxicology data, FSANZ considers that further data are required to fully characterise dietary exposure and any potential risks to consumers.

Estimated dietary exposures to cadmium were consistent with, and generally lower, than international estimates. These were compared to the PTMI, which determined that there are no public health and safety concerns for Australian consumers. There was a slight exceedance for infants aged 9 months at the 90th percentile of exposure (which ranged from 25–130% of the PTMI depending on the modelling scenario used). However, this temporary exceedance is not considered to be of concern due to the highly conservative method of assessment and nature of potential health effects which would only be associated with high levels of long-term exposure over many years.

Estimated dietary exposures to lead for Australian consumers were consistent with, and generally lower, than international estimates. These were compared with levels associated with effects in human populations using the MOE approach. Dietary exposure to lead for most Australian consumers was lower than levels found to be of negligible risk of causing adverse health effects. For these reasons, risks for Australian consumers are considered to be acceptably low.

Dietary exposure assessments were performed for both inorganic and methylmercury due to variation in their potential effects on human health. Estimated dietary exposures to inorganic mercury were compared to the PTWI, which determined that there are no public health and safety concerns for Australian consumers. There was an exceedance for infants aged 9 months at the 90th percentile of exposure (which ranged from <1–170% of the PTWI depending on the modelling scenario used). This temporary exceedance in the context of a lifetime of exposure is not considered to be of concern due to the highly conservative method of assessment and nature of potential health effects, which are associated with high levels of long-term exposure over many years. Estimated dietary exposures to inorganic mercury were also consistent with levels reported in the international scientific literature.

Estimated dietary exposures to methylmercury through the consumption of fish were below the PTWI for all age groups, except for consumers aged 2 to 5 years at the mean and high 90th percentile of exposure (up to 110% and 220% of the PTWI respectively). Women of child-bearing age, who represent the most sensitive window of exposure, had dietary exposures below the PTWI. The significance of temporary exceedances of the PTWI in early childhood are not clear. In its most recent assessment, JECFA (2007) was unable to confirm whether prenatal vulnerability through maternal exposure extends into the postnatal period. However, the potential for methylmercury exposure through consumption of fish to adversely affect young children is counterbalanced by the benefits of omega-3 fatty acids, which are also present in fish and are essential for normal development of the rapidly growing brain and eyes. Overall, dietary exposures for most Australian consumers, including pregnant women, are acceptably low. Temporary exceedances of the PTWI for children aged 2 to 5 years should be considered in the context of the known health benefits of fish consumption.

FSANZ will continue to monitor and contribute to international assessment and regulatory activities relating to agricultural and veterinary chemicals and metal contaminants. This work will contribute to FSANZ's current review work on mercury in fish and inform future regulatory considerations for arsenic, cadmium and lead. While these substances are detected at low levels in a broad range of foods, a limited number of commodities such as seafood are known to contribute significantly to dietary exposure. To reduce dietary exposure to these substances, consumers are encouraged to follow good dietary practices, including eating a balanced diet.

Part A–Background

Introduction

The ATDS provides a general indication of the concentrations of a range of substances, including agricultural and veterinary chemicals, metal contaminants, nutrients and other substances (such as food processing contaminants, food additives, packaging chemicals and naturally occurring toxins), in a broad range of Australian foods and beverages. The primary purpose of the ATDS is to estimate dietary exposure for the general Australian population to these substances. For this reason, foods are prepared to a table ready state for analysis to reflect how they are typically consumed, including cooking and removing inedible portions. Results are used to inform a risk assessment of Australian consumers to ensure that food regulatory measures continue to provide adequate protection of human health and safety.

Information from the ATDS is used to support FSANZ's administration of the Code, including the development of food regulatory and non-regulatory measures. The ATDS is also a trusted source of national public health information which is used in a variety of international work such as the World Health Organisation (WHO) Global Environmental Monitoring System (GEMS), the Joint Food and Agricultural Organisation of the United Nations (FAO)/WHO Meeting on Pesticide Residues (JMPR), the Joint FAO/WHO Expert Committee on Food Additives (JECFA), the Codex Committee on Food Additives (CCFA), the Codex Committee on Contaminants in Food (CCCF) as well as independent researchers in both government and nongovernment organisations.

Scope of the 25th ATDS

The 25th ATDS involved the sampling and analysis of a broad range of Australian foods and beverages for agricultural and veterinary chemicals, and metal contaminants. FSANZ has routinely monitored these substances in ATDS surveys since the 1970s. FSANZ has an ongoing commitment to ensure that food regulatory measures continue to provide adequate protection for the Australian public. The monitoring of dietary exposure to agricultural and veterinary chemicals, and metal contaminants forms an important part of this surveillance work.

Regulation of agricultural and veterinary chemicals, and metal contaminants

In Australia, the APVMA is responsible for regulating the use of agricultural and veterinary chemicals and setting of Maximum Residue Limits (MRLs) in agricultural produce. The MRL is the maximum concentration of a residue resulting from the registered use of an agricultural or veterinary chemical which is legally permitted in a food, agricultural commodity or animal feed. MRLs for agricultural and veterinary chemicals in food available for sale in Australia are administered by FSANZ and specified in Schedule 20 of Standard 1.4.2 in the Code. The MRL does not indicate the amount of chemical typically present in a treated food but it does reflect the highest legal residue concentration allowed from the approved conditions of registered use. FSANZ works with the APVMA to list MRLs in the Code. FSANZ must be satisfied that the residues of these chemicals do not present a risk to public health and safety before they are included in the Code. The MRLs listed in the Code are monitored and enforced by the relevant state and territory agriculture or food agencies.

For Australian and New Zealand foods, FSANZ sets maximum limits (MLs) for various contaminants (including arsenic, inorganic arsenic, cadmium, lead and mercury), which are specified in Schedule 19 of Standard 1.4.1 of the Code. MLs are only established for contaminants that present a significant risk to human health and foods that are major contributors to dietary exposure. MLs are established at levels which are ALARA while achieving the public health and safety objectives. Australia's criteria for setting MLs for

contaminants are consistent with international standards including the Codex Standard for Contaminants and Toxins in Food and Feed (FSANZ, 2017a).

Agricultural chemicals

Agricultural chemicals play a vital role in supporting Australia's farming industry by protecting crops and livestock, and boosting production. These chemicals may be present at low levels in the environment (and sometimes food) as a result of legitimate uses for the control of pests, diseases and other conditions for agricultural, commercial and domestic purposes. Human exposure, while undesirable, may occur at low levels through consumption of produce treated in accordance with strict conditions of use. These substances are thus a high priority for ongoing monitoring. A number of factors determined what would be included in the 25th ATDS such as outcomes of other survey activity, including previous ATDS surveys, Australian jurisdictional surveys and the Department of Agriculture and Water Resources National Residues Survey (NRS) (DAWR, 2017). Consideration was also given to other assessment work such as that undertaken as part of the APVMA Chemical Review Program (APVMA, 2017a), or internationally through other government authorities or working groups such as the JMPR. Some chemicals were considered a high priority from a public interest perspective, such as various persistent organic pollutants, which have not been approved in Australia for many years due to human health and environmental concerns. Foods were tested for 206 agricultural chemical residues including:

- carbamate insecticides
- fungicides
- herbicides
- organochlorine insecticides
- organophosphorus insecticides
- synthetic pyrethroid insecticides.

Various other miscellaneous agricultural chemical categories were also included. These are outlined in further detail below.

Carbamate insecticides

Carbamates are synthetic insecticides derived from carbamic acid and are commonly used to control household pests. Low exposures to residues in foods may occur where carbamates are used for agricultural purposes. Carbamate pesticides are mostly biodegradable and therefore do not accumulate in the food chain. They act on the nervous system of humans and animals but, as they are degraded in the liver, they do not generally accumulate in the human body (Fishel, 2015).

Fungicides

Fungicides are compounds that are toxic to fungi. The modes of action for fungicides can vary including inhibition of hyphal growth or spore germination, or limiting the development of spores on the plant. Fungicides can be applied to the seed, soil, fertiliser or foliage to assist in the control of disease. In field crops, fungicides are most commonly used as a seed dressing or via application to foliage (Crop Pro, 2014).

Herbicides

Herbicides are used to protect agricultural crops by killing or controlling the growth of weeds. The most common varieties of herbicides are generally broad spectrum and nonselective (Vats, 2015). These include glyphosate, paraquat and diquat. Glyphosate is used extensively worldwide. As a consequence of the high use of glyphosate, the herbicide paraquat has an

important role in controlling glyphosate-resistant weeds. Diquat is often used in combination with paraquat in managing glyphosate resistance.

Organochlorine insecticides

Organochlorine insecticides are generally highly stable compounds that are not susceptible to chemical and biological degradation (FAO, 2017). As a result, these compounds are persistent and bioaccumulate in the environment, particularly in soil. Human exposure to these chemicals can occur through the food supply. Organochlorine insecticides are fat soluble and can accumulate in adipose tissue of plants, animals and humans (Androutsopoulos et al., 2013).

The use of these chemicals in developed countries has been heavily restricted due to human health and environmental concerns. Some organochlorine insecticides have been identified as persistent organic pollutants including aldrin, chlordane, endrin, dieldrin, heptachlor, dichlorodiphenyltrichloroethane (DDT), campheclor, mirex and hexachlorobenzene (Stockholm Convention, 2008).

Organophosphorus insecticides

Organophosphorus pesticides are a diverse group of insecticides, used for a variety of agricultural purposes. Organophosphorus pesticides generally break down rapidly and do not accumulate in the food chain (Gan et al., 2010). These chemicals act by interrupting a chemical essential for nerve function in humans and in animals (Roberts and Reigart, 2013).

Synthetic pyrethroid insecticides

Pyrethroid pesticides are synthetic insecticides which are generally of lower toxicity to humans than other insecticides such as pyrethrins, carbamates and organophosphorus substances (Department of Health and Human Services, Victoria, 2017). These insecticides are effective against a wide range of pests and act rapidly to cause overstimulation of the nervous system. These chemicals are generally biodegradable and as such tend not to persist in the environment.

Other agricultural chemicals

A range of other agricultural chemicals were included in the 25th ATDS analytical screen including various insecticides/acaricides belonging to neonicotinoid, pyrrole, benzoylurea, organometal, phenylpyrazole, oxadiazine, sulphite, pyrazolium and diphenyl chemical classes.

Other chemicals classes investigated as part of the 25th ATDS included rodenticides, triazole plant growth regulators and polychlorinated biphenyls, which were used extensively as industrial chemicals until the late 1970s and may still be generated and released into the environment as unintended by-products of chemical manufacturing and incineration (DEE, 2017a).

Veterinary chemicals

Veterinary chemicals are used to prevent, diagnose, cure or alleviate disease or infection in an animal (Australian Government, 2016). Foods were tested for two classes of veterinary chemicals including parasiticides (anthelmintics) and antimicrobials (beta-lactams).

Anthelmintics

Anthelmintics are a class of veterinary chemicals used to control parasites without causing significant damage to the host. In farming practices, anthelmintics are used to assist in the control of intestinal worms in grazing livestock (DPI, 2011).

Beta-lactams

Beta-lactams are a class of veterinary chemicals containing antibiotic agents, which include a beta-lactam ring within their molecular structure. The active beta-lactam ring binds to penicillin-binding proteins in bacterial cell walls, inhibiting cell wall synthesis. This results in death of the bacterial cell caused by autolysis or osmotic instability (Michigan State University, 2011).

Metal contaminants

A broad range of foods and beverages were tested for arsenic (total and inorganic), cadmium, lead and mercury (total, inorganic and methyl).

These substances are considered an ongoing priority for inclusion in the ATDS as they are ubiquitous environmental contaminants often present in foods in low amounts. They are naturally present in the environment and distributed through erosion, water and air including as a result of human mining, industrial and some food processing activities. Arsenic, cadmium, lead and mercury were prioritised for inclusion in the 25th ATDS for the purposes of estimating dietary exposure and conducting a risk assessment for Australian consumers with consideration to contemporary scientific literature, including several recent assessments from JECFA in 2011. These assessments led to the reconsideration and withdrawal of HBGVs for inorganic arsenic and lead. The data will also support FSANZ's consideration and contribution to international standards development such as the ongoing work of CCCF.

Arsenic

Arsenic is a metalloid that occurs in various organic and inorganic forms. It is naturally present in the environment and widely distributed in rocks, soil, water and the air. Human activity also contributes to the environmental distribution of arsenic through mining and a variety of industrial processes including the manufacture of materials such as metals, glass, pigments, textiles, paper and ammunition. Arsenic is also used in timber preservatives and some pesticides (JECFA, 2011a; WHO, 2017a). Arsenic is found at low levels in a broad range of foods. Seafood and rice are known to contribute substantially to dietary exposure. Due to the wide variety of potential sources of arsenic in the environment, small amounts are found in some food and drinks. Foods were measured for both total arsenic and inorganic arsenic content as part of the 25th ATDS.

Recent international assessments have been unable to establish a safe level of human exposure to inorganic arsenic, with carcinogenic effects identified from studies of human populations.

Cadmium

Cadmium is a metallic element which is naturally present at low levels in the environment. Human activities such as tobacco smoking and various industrial processes including mining, manufacture of metals, fossil fuel combustion, manufacturing of phosphate fertilisers and processing of cadmium-containing waste, are also considered to be significant contributors to environmental cadmium levels. Cadmium is distributed throughout the environment in soil, water and air and can accumulate in the food chain, particularly in aquatic organisms such as

oysters, scallops, mussels and crustaceans. Lower levels of cadmium have been associated with certain crops including cereals and starchy root vegetables (WHO, 2010a; Rebelo & Caldas, 2016).

High levels of long term cadmium exposure have been associated with adverse effects on the human kidneys including impaired renal function.

Lead

Lead is one of the most common natural components of the earth's crust and its extensive use by humans has resulted in widespread environmental contamination and human exposure (WHO, 2011). Lead is found at low levels in a broad range of foods of both terrestrial and aquatic origins, reflecting its widespread distribution in the environment. Lead is used for many industrial and domestic purposes, and as a result, mining and metal manufacture are significant contributors to the levels of lead found in the environment. Lead is used as a component of batteries, paints and numerous household items and industrial materials (DEE, 2017b). Since the restriction of lead use in fuels, human exposure mainly occurs through contaminated food, dust and dirt. Environmental sources have been associated with the deposit of atmospheric lead on crops as well as water-based contamination. Canned foods were previously thought to be a potential dietary source of lead exposure, although this is no longer the case as cans used in Australia no longer have lead soldered seams (Department of Health Western Australia, 2017).

High levels of lead exposure have been associated with adverse cognitive effects (including reduced IQ) in children and cardiovascular effects (including increased blood pressure) in adults. Recent international assessments have been unable to establish a safe level of human exposure to lead.

Mercury

Mercury occurs naturally in the environment and is widely distributed via natural processes such as volcanic activity and erosion as well as human activity, including mining, coal-based power production and waste incineration (WHO, 2017b). Mercury is found in three primary forms; namely, elemental mercury, inorganic mercury and organic mercury—with methylmercury representing an important form from a human dietary perspective. Elemental and inorganic forms of mercury have been used industrially for various purposes including gold mining, vinyl chloride and chloralkali production and as components of dental fillings, electric household items and thermometers (WHO, 2008). Due to concerns regarding toxicity, its use in clinical applications is being phased out in favour of safer alternatives and the WHO is engaged in a global initiative to promote the safer alternatives to mercury-based medical devices (JECFA, 2011b).

Mercury is found at low levels in the food supply, with most dietary exposure coming from seafood. Methylmercury is largely produced when inorganic mercury circulating in the environment is dissolved into water and subject to bacterial transformation.

Methylmercury bioaccumulates in marine and freshwater organisms through the food chain, resulting in large predatory fish, such as swordfish and shark commonly having the highest levels (Hong et al, 2012). Methylmercury is considered to be a major human health concern. High levels of long-term methylmercury exposure have been associated with adverse developmental effects in unborn children. High levels of long term inorganic mercury exposure have been associated with adverse effects on kidney health.

Foods were tested for total mercury, inorganic mercury and methylmercury as part of the 25th ATDS.

Part B–Conducting the study

Food sample purchasing, preparation and analysis were undertaken in accordance with detailed instructions outlined in a survey procedures manual. Food preparation instructions for those analysed in the 25th ATDS are summarised in Appendix 5.

Food purchasing

A total of 88 foods and beverages, including tap water, were included in the 25th ATDS. For each of the 88 foods, 3 primary (individual) sample purchases were collected from between 4-8 different Australian states or territories. A total of 1524 individual food samples were purchased and combined into a total of 508 composite samples for analyses. Each analysed composite sample was made up of three individual samples from a single state or territory.

A full list of foods surveyed is provided in Appendix 1. Foods were selected if they were:

- suspected or known to contribute significantly to dietary exposure for the chemical analysed, and/or
- represented current patterns of food and beverage consumption in Australia.

Foods in the sample list were classified as either regional or national foods. Higher numbers of regional food samples were collected to account for the increased potential for regional variation in composition.

Regional foods were defined as those that might be expected to be sourced regionally and show geographical variation in chemical concentrations. These foods included milk, tap water, fish, fruit, vegetables, red meat and red meat products, chicken, bread and bakery items, wine and selected takeaway foods. For each regional food, eight composite samples were analysed, each consisting of three primary purchases collected from each Australian state and territory.

National foods were defined as foods distributed nationwide and therefore expected to show little regional variation in chemical concentrations. These included breakfast cereals, processed meats, infant foods, tea, coffee, sugar and a variety of canned and other shelf-stable packaged foods. For each national food, four composite samples were analysed, each consisting of three primary purchases collected from four Australian state and territory jurisdictions.

Sampling took place across Australia over two sampling periods, from 13 to 24 May 2013 (autumn sampling period) and 17 to 28 February 2014 (summer sampling period). Due to the large number of samples collected, purchasing took place over several days within the time periods specified above. Samples were sent to a coordinating analytical laboratory as soon as practicable after purchase. In instances where the analytical laboratory was located outside the city of sampling, all perishable samples (e.g. fruits, vegetables and meat) were sent overnight in a frozen or chilled state to the laboratory, reflecting how these products would typically arrive at the home.

Food-analyte combinations

The complete list of foods analysed in the 25th ATDS can be found in Appendix 1. All foods were analysed for most agricultural and veterinary chemicals and metal contaminants.

Several chemicals were only analysed in a subsample of foods to reflect likely sources of contamination and dietary exposure, and analytical limitations for some food matrices. For

example, veterinary chemicals were only tested in selected foods of animal origin. Details for analytes measured in various subsamples of foods are provided in Table 1.

Table 1 Details for analytes measured in various subsamples of foods

Substances	Foods analysed
Flumethrin (synthetic pyrethroid insecticide) and methylpyrrolidine (herbicide)	All foods with the exception of mango and nectarine
Guazatine (fungicide)	Fruit juice and oranges
Selected herbicides (amitrole, chlormequat, diquat, glufosinate, glyphosate (and metabolite aminomethylphosphonic acid (AMPA), paraquat)	Almonds; biscuits, savoury; bread (both multigrain and white); breakfast cereals (both rice and wheat/corn single grain based); chocolate cake; infant cereal; oats; pasta; rice and tap water
Veterinary chemicals	Bacon; beef mince, lean; chicken breast; eggs; ham; liver pate (chicken); full cream milk and sausages
Inorganic arsenic	Fish fillets, plain from takeaway; fish portions, frozen from supermarket; mussels; prawns; rice; sushi and tuna canned in brine
Inorganic and methylmercury	Fish fillets, plain from takeaway; fish portions, frozen from supermarket; mussels; prawns and tuna canned in brine

Food and sample preparation

All primary food samples were prepared to a ready-to-eat state by the sample preparation laboratory, Symbio Laboratories Pty Ltd (Symbio Laboratories). For example, sausages were grilled before analysis. A number of purchased food samples, such as peanut butter and infant desserts, were in a ready-to-eat state when purchased and therefore did not require additional cooking or preparation. Perishable foods were all prepared within 48 hours of purchase. Frozen and shelf-stable foods were prepared as soon as practicable within a week of purchase. Specific details on food preparation procedures are outlined in Appendix 5. After preparation to a table ready state, a standard amount from each primary sample was taken and combined to form composite samples for each jurisdiction, and food, for analyses. Samples requiring further transport for analyses in a different laboratory were freighted in a stable frozen state as soon as practicable.

Food analyses

Symbio Laboratories tested for agricultural chemicals and metal contaminants at their Brisbane laboratory. The testing for veterinary chemicals was undertaken by Hill Laboratories, Christchurch, New Zealand. Full details of the analytical methods and limits are reported in Table 2.

Testing of agricultural chemicals and metal contaminants were undertaken according to National Association of Testing Authorities (NATA) accredited methods, with the exception of inorganic arsenic and organic mercury (methodology fully validated and pending accreditation at the time). Symbio Laboratories became accredited by NATA for both of these tests (NATA, 2017) after testing in 2013/2014.

Hill Laboratories tested samples of animal origin for veterinary chemicals using tandem liquid chromatography mass spectrometry (LC MS/MS). The method is fully validated, but not accredited by International Accreditation New Zealand (IANZ).

Table 2: Summary of analytical methods and limits

Analytes		Technique	Limit of Reporting (LOR)* (mg/kg or mg/L)
Agricultural chemicals		GC MS/ECD/PFPD/ Headspace LC MS/MS LC HR/MS	0.010-0.20
Veterinary chemicals		LC MS/MS	0.005-0.010
Metal contaminants	Cadmium, lead, mercury, arsenic	ICP MS acid digest preparation	0.005 Drinking Water–0.0001
	Inorganic arsenic	HPLC ICP MS freeze dried and enzymatic digest preparation	0.01
	Inorganic and methylmercury	HPLC/ICP MS freeze dried preparation	0.01 (inorganic) 0.05 (methyl)

GC–Gas Chromatography
LC–Liquid Chromatography
MS–Mass Spectrometry
ECD–Electron Capture Detector
PFPD–Pulsed Flame Photometric Detector
ICP–Inductively Coupled Plasma
HPLC–High Performance Liquid Chromatography
* For agricultural chemicals and metal contaminants analytical tests, the LOR[1] was equal to the LOD[2] and the LOQ[3] was 5-times the LOD. For veterinary chemicals, the LOR was equal to the LOQ.

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[1] LOR: The lowest concentration of an analyte reported by the laboratory.
[2] LOD: The lowest concentration of an analyte in a sample that can be detected with acceptable reliability.
[3] LOQ: The lowest concentration of an analyte in a sample that can be quantitatively determined with acceptable accuracy and precision.

Part C–Estimating dietary exposure

What is dietary modelling?

Dietary modelling is a tool used to estimate the dietary exposure of a population or subpopulation group to contaminants, agricultural and veterinary chemicals and other substances. To estimate dietary exposure to food chemicals, food consumption data are combined with food chemical concentration data as shown in Equation 1.

Equation 1: Dietary exposure calculation

Dietary Exposure = food chemical concentration x food consumption amount

International expert bodies have used dietary modelling techniques for many years to determine if dietary exposures to specific food chemicals pose a potential risk to public health and safety.

Dietary modelling is an important part of the ATDS as it translates the chemical concentration data for individual foods into dietary exposure estimates that can be compared to relevant HBGVs in order to assess any risks to public health and safety. The HBGVs used in the 25th ATDS are specified in Appendix 10.

While dietary modelling is a systematic scientific methodology, the accuracy of the dietary exposure estimates depends on the quality of the chemical concentration and food consumption data available for use. For detailed information about the procedures used for dietary modelling in the 25th ATDS, refer to the supplementary information provided in Appendix 7.

Food chemical concentrations used for modelling

A number of composite samples were analysed for each food in the 25th ATDS. From these, the mean concentrations of agricultural and veterinary chemicals were used to calculate dietary exposure. Where a high number of results are below the limit of reporting (LOR), such as in the case of agricultural and veterinary chemicals, the mean concentration is a more conservative indicator of residue levels than the median concentration.

The median concentrations of metal contaminants in each food were used to calculate dietary exposure. The median concentration is typically used for estimating dietary exposure to ubiquitous environmental contaminants, such as trace metals, which may be consistently present in foods at low levels. In these cases, the median generally represents the most likely concentration in a given food and reduces potential over estimation of exposure. This is because median concentrations are less likely to be affected by a small number of high detections or 'outliers'. Using the median concentration may also remove uncertainty about dealing with results below the LOR as it may reflect an actual quantified result. This would be the case when foods have 50% or more positive detections (above the LOR). However, in cases where less than 50% of results are above the LOR for a given food, the median concentration for modelling purposes is a 'non-detect', whereas the mean concentration may have a numerical value.

Treatment of analytical results below the LOR

Some analytical results were 'not detected,' or in other words, were below the LOR for the analytical method. In order to account for these results in the dietary exposure assessment, a numerical concentration value needs to be assigned to these samples. Assumptions were

made about the concentration of substances in food samples where the analytical results were below the LOR.

For agricultural and veterinary chemicals, analytical concentrations below the LOR were assumed to be zero when calculating mean concentrations in foods. This method reflects the use profile of these substances, which are selectively applied to agricultural produce as required, and assumes that they are not present in foods if not detected.

In the case of contaminants that occur naturally in the environment, it is not reasonable to assume that the contaminant is not present in the food when the analytical concentrations are less than the LOR. In the case of the metal contaminants assessed, the LOR was reported as equal to the LOD. Actual concentrations below the LOR could in reality be anywhere between zero and the LOR. To allow for this uncertainty, the results for dietary exposure to metal contaminants were presented as a range. The lower end of the range was calculated based on the assumption that results below the LOR are equal to zero. The upper end of the range, representing a conservative 'worst-case' estimate, was calculated on the assumption that results below the LOR are equal to the LOR.

Food consumption data used for modelling

The dietary modelling used the most recent food consumption data available for the Australian population aged 2 years and above from the 2011–2012 Australian National Nutrition and Physical Activity Survey (2011–12 NNPAS) component of the 2011–2013 Australian Health Survey, conducted by the Australian Bureau of Statistics. Further information on the 2011–12 NNPAS is provided in Appendix 7.

Food mapping

Mapping is the process of matching the foods analysed in the 25th ATDS to the foods consumed in the 2011–12 NNPAS. Given that the ATDS could not survey all foods consumed in the 2011–12 NNPAS, mapping is a major step in the dietary modelling process to ensure the total diet is captured in the estimates of dietary exposure. Mapping can be based on the composition or likely contamination of a food. Dietary exposure results have been presented in terms of the group of foods that the ATDS food represents, rather than as the individual analysed ATDS food itself (e.g. the ATDS food 'apples' is referred to as 'pome fruits' in the dietary exposure assessment results).

Three types of mapping were used:

  – *Direct mapping* matched the ATDS foods to the same food and to similar foods from the 2011–12 NNPAS (e.g. the 2011–12 NNPAS foods '*Apple, royal gala, unpeeled, raw*' and '*Pear, peeled, stewed, unsweetened, no added fat*' were mapped to the ATDS food 'Apples', using the assumption that the food chemicals present in apples are the same in all pome fruits. This food group was then called 'Pome fruits').
  – Mapping using *factors* took place where the ATDS food was in a different form to that consumed in the 2011–12 NNPAS (e.g. the ATDS food 'coffee, instant' was analysed in its hydrated (or ready-to-drink) form but some respondents in the 2011–12 NNPAS reported consuming dry instant coffee powder). The NNPAS food was mapped to the ATDS food after which the food consumption amount reported in the 2011–12 NNPAS was multiplied by a 'factor' to convert the food to the same form as analysed in the ATDS (e.g. 2 grams of instant coffee powder was converted to 226 grams of ready-to-drink black instant coffee). The converted consumption amount for the food was then assigned the analysed concentration for the ATDS food in the dietary exposure calculation.

- *Recipes* mapping was used where a food consumed in the 2011–12 NNPAS was composed of more than one analysed ATDS food (e.g. the 2011–12 NNPAS food '*Fruit drink, orange juice, commercial*' was made up of the ATDS foods 'Sugar, white', 'Water, tap' and 'Juice, fruit'). A recipe was used to disaggregate the consumption of the mixed food to the relevant components.

Table A 8.1 in Appendix 8 contains full details of the 25th ATDS sampled foods that were matched to 2011–12 NNPAS foods.

Population groups assessed

Dietary exposures to agricultural and veterinary chemicals and contaminants were estimated for a range of population groups. These groups were 9 month old infants, children aged 2 to 5 years and 6 to 12 years, teenagers 13 to 18 years, adults aged 19 years and above and the general population aged 2 years and above. Dietary exposure assessments were conducted for infants and children as separate groups as they generally have higher exposures on a body weight basis because they consume more food per kilogram of body weight when compared to adults. Additional population groups were assessed where there was a specific concern, such as for methylmercury exposure (due to potential effects on the fetus) where women of child bearing age (16 to 44 years) were used as a proxy for pregnant women.

Dietary exposures for infants were estimated using a model diet. Dietary exposures for all other population groups were estimated using the 2011–12 NNPAS. Further details can be found in Appendix 7.

Food contribution calculations

Throughout the report, information about the major food contributors to the dietary exposure to particular chemicals has been presented. To obtain an indication of the contribution each food group made to total estimated exposures, the sum of all individuals' exposures from one food group was divided by the sum of all individuals' exposures from all foods containing the food chemicals assessed, and multiplied by 100. All contributions were calculated using the lower bound (nd=0) scenario. The lower bound (nd=0) scenario represents the situation where analytical results listed as 'not detected' are assumed to have a concentration of zero.

There is no direct association between the analytical concentration of a chemical in an ATDS food and its identification as a major contributor to dietary exposure. Even if a food contained a relatively high concentration of a particular chemical, there are many other factors which may contribute to dietary exposures, including the amount of the food consumed, the mapping process used, the number of individuals that were exposed to the chemical and the level to which they were exposed.

Assumptions and limitations in dietary exposure assessment

The aim of dietary exposure assessments is to make estimates of dietary exposure to the food chemicals of interest as realistic as possible.

Dietary exposure assessments based on the 2011–12 NNPAS provide the best available estimates of actual consumption of all foods and the resulting estimated dietary exposure to a food chemical for the population. Nevertheless, limitations still exist in dietary exposure assessment methods as well as in the data. There are a number of limitations relating to food consumption and chemical concentration data. These include:

- Diets derived from two 24-hour food recall surveys were used as the basis for drawing conclusions on lifetime eating patterns. This normally leads to conservative dietary exposure assessments, particularly where exposure arises from the consumption of non-habitually eaten foods.
- The 2011–12 NNPAS data did not include information about food products introduced to the market after it was conducted.
- Participants in 24 hour food recalls may over, or under, report food consumption, particularly for certain types of foods.
- Dietary exposure to the chemicals of interest from dietary supplements has not been taken into account.
- The model diet used for 9 month old infant group is not as specific as the data derived for other population groups from the 2011–12 NNPAS that use distributions of food consumption data of individuals. Additionally, a model diet reports dietary exposures for all respondents, irrespective of whether they are exposed to the chemical of interest or not.
- The list of analysed foods is only a sample of the foods consumed by the population and may not accurately represent the whole diet.
- Only a small number of each food was sampled and analysed, and these were then used to represent that food or a range of similar foods, therefore limiting the potential range of variability in food chemical concentrations.

Assumptions made in the dietary exposure assessment for the 25th ATDS include:

- The food chemical concentration in the analysed food represented the concentration of that chemical in all of the other foods to which it was mapped.
- The chemical concentration in a particular analysed food and foods to which it was mapped were carried over to all of the mixed foods in which they were used as an ingredient.

Part D–Results and risk analysis

Agricultural chemicals

Hazard identification

Human exposure to agricultural chemicals, while undesirable, may occur through consumption of residues in agricultural produce arising from their intended use. In order to assess and manage the use and potential exposure to these chemicals, the APVMA (2017b) maintains a database of HBGVs for substances, which may be present in agricultural produce. These HBGVs include ADIs, expressed on a milligram per kilogram body weight basis, which represent an estimate of the amount of a particular substance in food and drinking water that can be ingested over a lifetime without appreciable risk to human health. ADIs are in most cases derived from controlled toxicological studies in laboratory animals in which the chemical in question is administered in the diet or by oral gavage. These studies are used to determine a No Observed Adverse Effect Level (NOAEL), which is the highest administered dose which does not cause any adverse effects. The NOAEL for a chemical in the most sensitive species is then used to estimate the ADI. The ADI is calculated by dividing the overall NOAEL from a suitable study by an appropriate safety factor. The magnitude of the safety factor is selected to account for uncertainties in extrapolation of animal data to humans, intra-species variation, the completeness of the toxicological database and the nature of the potential toxicologically-significant effects (APVMA, 2017b).

The ADIs for agricultural (and veterinary chemicals) detected in the 25th ATDS are presented in Appendix 10.

Analytical results

The ATDS is used to monitor the general levels of agricultural chemicals in the Australian food supply. Summary information on the concentrations of agricultural chemicals found in the 25th ATDS can be found in Table A 3.9 of Appendix 3.

Out of a total of 206 agricultural chemicals analysed, 56 were detected in this study at concentrations above the LOR. Agricultural chemicals were detected in 42 of the 88 foods analysed. Out of all 508 composite samples analysed, 172 (34%) had detectable residues of one or more of the 206 agricultural chemicals analysed. A total of 336 composite samples (66%) had no detectable residue of any agricultural chemical. As shown in Figure 1, these results indicate that a large proportion of foods consumed in Australia contain no detectable residues of agricultural chemicals.
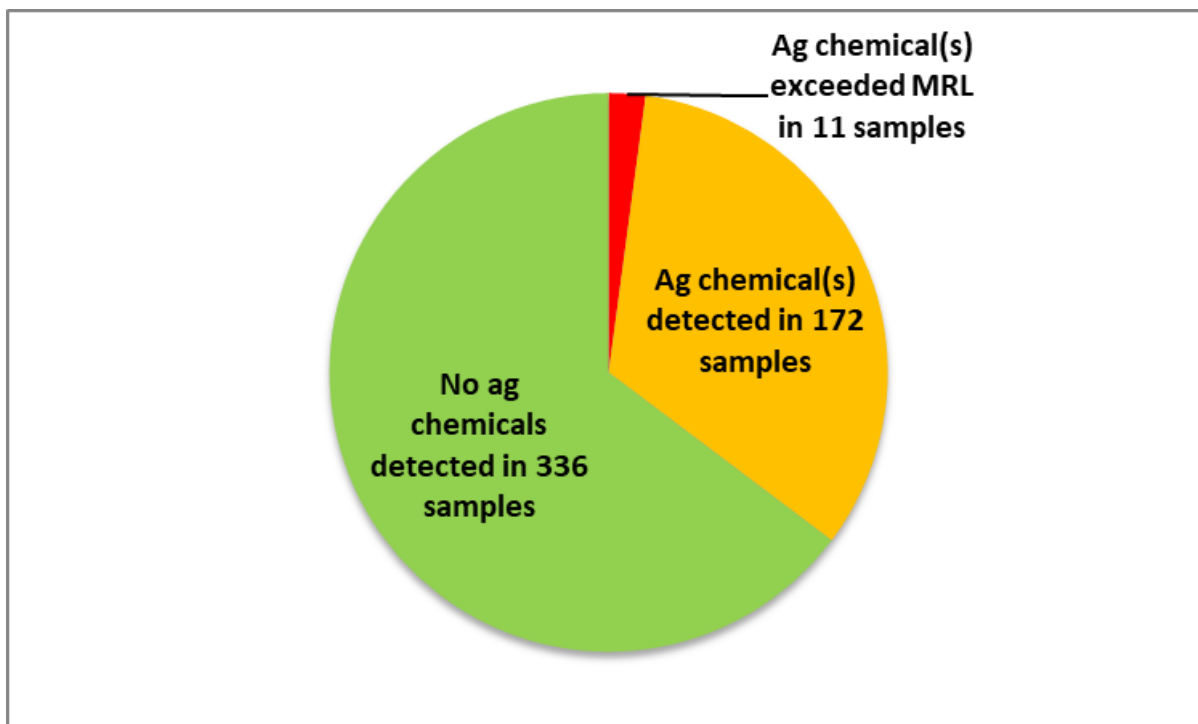
Figure 1 Number of composite food samples with detectable agricultural chemical residues

The most frequently detected agricultural chemicals were piperonyl butoxide (detected in 18 foods), dithiocarbamates (13 foods), iprodione (9 foods), cypermethrin (8 foods), imidacloprid (7 foods), carbendazim, chlorpyrifos and chlorpyrifos methyl (all detected in six foods), and bifenthrin, fluazifop-P-butyl and glyphosate (all detected in five foods). All other agricultural chemicals were detected in four or less foods.

The foods with the highest mean concentrations of piperonyl butoxide (assuming nd=0) were mixed breakfast cereals (0.81 mg/kg), white bread (0.12 mg/kg) and multigrain bread (0.080 mg/kg). These results are consistent with the approved uses of piperonyl butoxide, which may be applied as a spray to control insects in agricultural buildings including storage sheds, milking sheds, poultry sheds and barns which may contain postharvest agricultural produce including cereal grains (APVMA, 2017c). The foods with the highest mean concentrations of dithiocarbamates were broccoli (1.2 mg/kg), grapes (0.79 mg/kg) and cauliflower (0.60 mg/kg). These results are consistent with the approved uses of dithiocarbamate chemicals to manage weeds, soil-borne pests and fungal diseases in a variety of agricultural crops (APVMA, 2017d). All other mean agricultural chemical concentrations were less than 0.20 mg/kg.

The foods which had the highest number of agricultural chemicals detected across all of their respective composite samples were bok choi (16 different agricultural chemicals detected), apples and capsicum (11 agricultural chemicals), cucumber and strawberries (10 agricultural chemicals), grapes, nectarine, sultanas and raw tomatoes (9 agricultural chemicals), green beans and white bread (6 agricultural chemicals), and savoury biscuits, multigrain bread, various breakfast cereals and hamburgers (5 agricultural chemicals). All other foods had four or less different agricultural chemicals detected across all of their respective composite samples.

Comparison against MRLs

A comparison of analytical results for composite samples against corresponding commodity based MRLs was undertaken. It should be noted that ATDS results are not a measure of

compliance with the MRLs specified in the Code. Standard 1.4.2 specifies that MRLs are applicable to 'food for sale' in Australia and enforcement is the responsibility of state and territory departments, agencies and local councils in Australia, and, for food imported into Australia, the Australian Department of Agriculture and Water Resources. However, ATDS results can provide a general indication of areas which may warrant follow-up investigation and management by FSANZ and the food regulatory system.

All agricultural chemicals that were detected in the 25th ATDS are permitted to be present in various food commodities as specified in Schedule 20 of Standard 1.4.2 of the Code. However, foods for sale must not have a detectable amount of an agricultural (or veterinary) chemical, unless expressly permitted by the Code (FSANZ, 2017b). There were a total of eight residue detections for chemicals in composite food samples which have no corresponding commodity based MRL in the Code. There were a total of four composite sample results which exceeded corresponding commodity based MRLs for agricultural chemicals. These are shown in Table 3.

Table 3 Agricultural chemical composite sample results exceeding corresponding commodity based MRLs

Agricultural chemical	Food	Concentration(s) (mg/kg)
Detections in composite samples with no corresponding commodity-based MRL		
Cyhalothrin	Bok choi	0.059
Dimethoate	Bok choi	0.012
	Cucumber	0.015, 0.020
Dithiocarbamates (total)	Prawns	0.16, 0.20
Permethrin	Capsicum	0.019
Triadimenol	Bok choi	0.24
Composite sample results exceeding corresponding commodity-based MRL		
Chlorpyrifos	Cucumber	0.019
	Green beans	0.17
Dithiocarbamates (total)	Broccoli	2.3, 2.5

Conclusion

Overall, the results indicate that the levels of agricultural chemicals in Australian foods are very low, with the vast majority of individual measured results (>99%) at undetectable levels below the LOR. A total of 11 of 508 (2%) composite food samples had agricultural chemical residues exceeding MRLs. These results indicate general consistency with regulatory standards.

Dietary exposure

Dietary exposure assessments were only conducted for the 56 agricultural chemicals that were detected at levels above the LOR in the 25th ATDS. These chemicals are listed by type in Table 4.

Table 4 Agricultural chemicals detected

Chemical Type	Chemical Name
Fungicides	Azoxystrobin
	Captan

Chemical Type	Chemical Name
	Carbendazim
	Cyprodinil
	Difenoconazole
	Diphenylamine
	Dithiocarbamates, Total (mancozeb, thiram, zineb & ziram)
	Imazalil
	Iprodione
	Metalaxyl
	Myclobutanil
	Prochloraz
	Procymidone
	Propiconazole
	Pyrimethanil
	Tebuconazole
	Thiabendazole
	Triadimefon
	Triadimenol
Herbicides	Chlorpropham
	Fluazifop-P-butyl
	Glyphosate
	Haloxyfop-methyl
	Propyzamide
Organophosphorus insecticides	Acephate
	Azinphos-methyl
	Carbofuran
	Chlorpyrifos
	Chlorpyrifos-methyl
	Diazinon
	Dimethoate
	Fenitrothion
	Fenoxycarb
	Fenthion (including fenthion sulphoxide)
	Malathion

Chemical Type	Chemical Name

Chemical Type	Chemical Name
	Methamidophos
	Methomyl
	Omethoate
	Pirimicarb (including dimethyl-pirimicarb)
	Pirimiphos-methyl
	Prothiofos
	Trichlorfon
Synthetic pyrethroids	Bifenthrin
	Cyhalothrin
	Cypermethrin
	Deltamethrin
	Fenvalerate/es/fenvalerate
	Permethrin
Other pesticides	Acetamiprid
	Imidacloprid
	Indoxacarb
	Piperonyl butoxide
	Propargite
	Spinosad

Fungicides

There were 19 fungicides detected in the study. The estimated dietary exposures to individual fungicides can be found in Appendix 11. Estimated mean and 90th percentile (P90) dietary exposures for consumers were less than or equal to 1% of the ADI for 15 of the 19 fungicides. For the other four fungicides (diphenylamine, dithiocarbamates, imazalil, iprodione), the estimated mean dietary fungicide residue exposures were less than or equal to 35% of the ADI and P90 exposures were less than or equal to 80% of the ADI across all of the population groups assessed.

Herbicides

There were detectable residues for five herbicides. The dietary exposures to individual herbicides are outlined in Appendix 11. All mean and P90 estimated dietary exposures to herbicide residues were 2% of the ADI or less.

Organophosphorus insecticides

There were detectable residues for 18 organophosphorus insecticides. The dietary exposures to individual organophosphorus insecticides are discussed in detail in Appendix 11. For 6 of the 18 organophosphorus insecticides (azinphos methyl, carbofuran, dimethoate, fenoxycarb, malathion, pirimiphos methyl), mean and P90 estimated dietary exposures were less than or equal to 1% of the ADI. For 11 organophosphorus insecticides (acephate,

chlorpyrifos, chlorpyrifos methyl, diazinon, fenitrothion, fenthion, methamidophos, methomyl, omethoate, pirimicarb and trichlorfon), mean and P90 estimated dietary exposures were 9%, or less, of the ADI. One organophosphorus insecticide, prothiofos, had estimated dietary exposures which exceeded the ADI for some population sub-groups and is discussed in greater detail below.

Dietary exposures to prothiofos

Estimated mean and P90 dietary exposures to prothiofos for all age groups 2 years and above were 3.6–5.2 µg/day and 7.4–9.6 µg/day respectively. Teenagers 13 to 18 years of age had the highest mean and P90 dietary exposures on a µg/day basis.

On a body weight basis (µg/kg bw/day), estimated mean and P90 prothiofos dietary exposures for all age groups 2 years and above were 0.061–0.20 µg/kg bw/day and 0.14–0.40 µg/kg bw/day. Children aged 2 to 5 years had the highest mean and P90 dietary exposures on a µg/kg bw/day basis.

For infants aged 9 months, mean and P90 dietary exposures were estimated to be 0.16 µg/day and 0.31 µg/day, respectively. On a body weight basis, mean and P90 dietary exposures were 0.017 µg/kg bw/day and 0.035 µg/kg bw/day, respectively.

Between 8% and 23% of the population groups were consumers of prothiofos. For children, 23% of those aged 2 to 5 years and 15% of those aged 6 to 12 years were consumers of prothiofos.

All prothiofos dietary exposures were from the food group Grapes. Refer to Table A 11.44 in Appendix 11 for more information.

Synthetic pyrethroids

There were detectable residues for six synthetic pyrethroids. Dietary exposures to individual synthetic pyrethroids are discussed in detail in Appendix 11. The mean and P90 estimated dietary exposures to synthetic pyrethroids were all 2% of the ADI or less.

Other pesticides

There were detectable residues for six other pesticides. Mean and P90 dietary exposures to these pesticides, except propargite and piperonyl butoxide, were less than 1% of the ADI. Propargite mean and P90 dietary exposures were 8 to 30% of the ADI and 15 to 50% of the ADI respectively across the age groups assessed. Piperonyl mean and P90 dietary exposures were 2% of the ADI or less and 3% of the ADI or less, respectively. Dietary exposures to all other pesticide residues are discussed in detail in Appendix 11.

Risk characterisation

With the exception of prothiofos, estimated dietary exposures to all agricultural chemicals at the mean and P90 were below the ADI for all population groups assessed, indicating a low risk to public health and safety.

Mean estimated dietary exposures to prothiofos, through the consumption of grapes, exceeded the ADI only for children aged 2 to 5 years and 6 to 12 years at up to 200% of the ADI. High level (P90) estimates of dietary exposure exceeded the ADI for all population groups assessed, except 9 month infants, at up to 400% of the ADI (see Figure 2).

FSANZ determined that these results warrant further investigation including consideration of risk management measures.
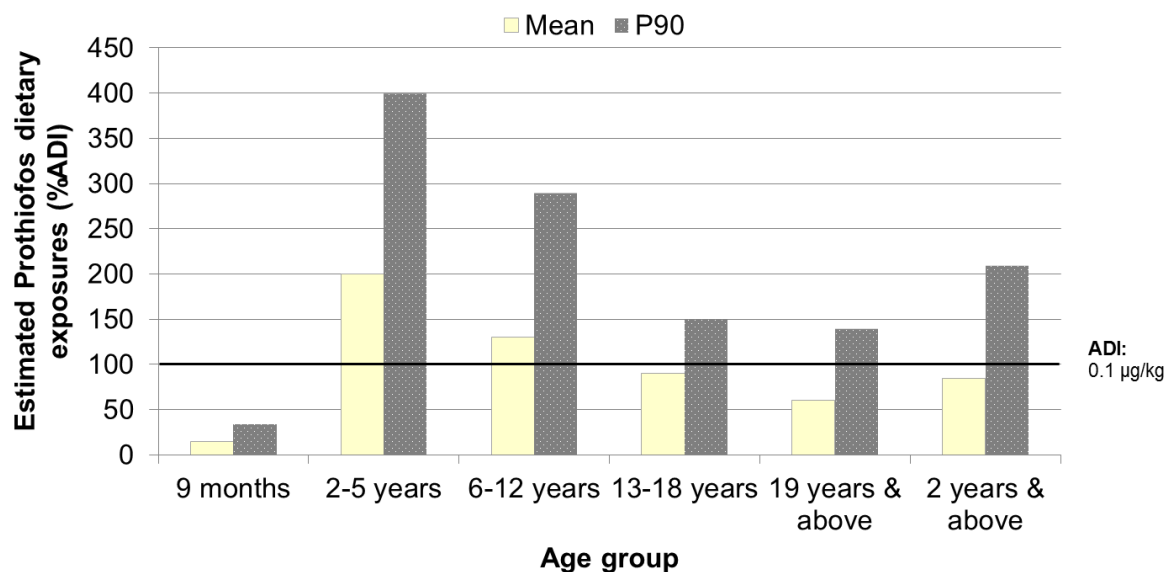

Figure 2 Estimated dietary exposures to prothiofos as a percentage of the ADI

Risk management

Most composite sample results for agricultural chemicals in the 25th ATDS were below corresponding commodity based MRLs indicating general consistency with regulatory standards. Details of any exceedances were provided to the APVMA and relevant state and territory enforcement authorities for information and management as required.

The detections of prothiofos in composite grape samples were all well below the corresponding commodity based MRL and consistent with approved conditions of use at the time of sampling (APVMA, 2017c). FSANZ consulted with key stakeholders, including the APVMA, to investigate potential public health and safety implications relating to exceedances of the ADI. As an outcome of these discussions, the APVMA engaged with industry resulting in the Australian registrant voluntarily cancelling label approvals for the use of prothiofos on grapes.

Veterinary chemicals

Hazard identification

As described previously, the APVMA establishes HBGVs for veterinary chemicals and maintains a database of HBGVs, including ADIs, for agricultural and veterinary chemicals. The ADIs for agricultural and veterinary chemicals detected in the 25th ATDS are presented in Appendix 10.

Analytical results

Foods of animal origin sampled in the 25th ATDS were screened for 20 veterinary chemicals, including anthelmintics and beta-lactams. There were no detections for 19 of the 20 veterinary chemicals, with the parasiticide closantel detected in two composite samples of lamb loin chops. Both of these detections (0.37 and 0.006 mg/kg) were well below the corresponding commodity based MRL. A risk assessment for dietary exposure to closantel was undertaken using these results.

Dietary exposures to closantel

Estimated mean and P90 consumer dietary exposures to closantel for all age groups 2 years and above were 1.4–2.9 µg/day and 2.8–5.8 µg/day respectively. Adults aged 19 years and above had the highest mean dietary exposures on a µg/day basis, with teenagers aged 13 to 18 years having the highest P90 exposures.

On a µg/kg bw/day basis, estimated mean and P90 dietary exposures for all age groups 2 years and above were 0.035–0.088 µg/kg bw/day and 0.067–0.17 µg/kg bw/day respectively. Children aged 2 to 5 years had the highest mean and P90 dietary exposures on a µg/kg bw/day basis.

For infants aged 9 months, mean and P90 respondent dietary exposures were estimated to be 0.095 µg/day and 0.19 µg/day, respectively. On a body weight basis, mean and P90 respondent dietary exposures were 0.011 µg/kg bw/day and 0.021 µg/kg bw/day, respectively.

Refer to Table 11.60 for further details on dietary exposures to closantel for all age groups.

The only food group contributing to the dietary exposure to closantel was 'Lamb, mutton, goat, kangaroo and rabbit' which is shown in Table 11.61 in Appendix 11.

Risk characterisation of closantel dietary exposures

The mean and 90th percentile dietary exposures to closantel were well below the ADI (less than 1%) for all population groups assessed. FSANZ determined that dietary exposure to closantel does not represent an appreciable risk to public health and safety.

Risk management

All veterinary chemical results for composite samples were below corresponding commodity based MRLs indicating a high level of consistency with regulatory standards. As there were no concerns raised from a public health and safety perspective, it is considered that risk management measures are not required in relation to 25th ATDS results for veterinary chemicals. FSANZ will consider the need for testing of different classes of veterinary chemicals in the future, to ensure continued effective monitoring of a broader range of substances.

Metal Contaminants in food

Arsenic

Hazard identification

Arsenic is highly toxic in its inorganic form, and long term exposure from food and drinking water has been associated with cancer, skin lesions, developmental effects, cardiovascular disease, neurotoxicity and diabetes (JECFA, 2011a). The bioavailability of arsenic is variable, with soluble forms readily absorbed via the gastrointestinal tract, as opposed to insoluble forms which have a lower rate of absorption. Arsenic is rapidly cleared by the blood and is generally excreted by the kidneys within a few days. Arsenic can accumulate for longer periods in the bone, skin, hair and nails.

The 72nd JECFA meeting (2011a) concluded that the previously established PTWI of 15 µg/kg body weight for inorganic arsenic was no longer considered to be protective of health, as the calculated Benchmark Dose Lower Confidence Limit (BMDL)$_{0.5}$[4] of 3 µg/kg body weight/day for lung cancer from a large-scale prospective cohort study in north-eastern Taiwan, China, was found to be at a similar level of exposure. Other key points of departure (BMDL$_{0.5}$) established from the dose-analyses of epidemiology studies were 5.2 µg/kg body weight/day for bladder cancer and 5.4 µg/kg body weight/day for skin lesions. In the absence of a threshold of toxicological concern, the PTWI was withdrawn (JECFA, 2011a).

For the purposes of the risk assessment, FSANZ used the most sensitive point of departure established by JECFA (2011a) (BMDL$_{0.5}$ for lung cancer of 3 µg/kg body weight/day) for comparison against estimated dietary exposures. JECFA (2011a) noted that quantitative assessment of cancer risk is limited by lack of information on total exposure in the available epidemiological studies. The BMDL$_{0.5}$ is also subject to uncertainty related to the relevance of extrapolation to other populations, because the studied populations had nutritional factors, such as low protein intake, and other lifestyle factors that may have influenced the study findings.

Total arsenic

Analytical results

Summary information on concentrations of total arsenic found in the 25th ATDS is outlined in Table A 3.2 in Appendix 3. Arsenic was detected in 45 of the 88 foods sampled. Of all 508 composite samples analysed, 142 (28%) had detectable residues of arsenic. A total of 366 composite samples (72%) had no detectable residues of arsenic. As shown in Figure 3 results indicate that a large proportion of foods consumed in Australia contain no detectable residues of arsenic.

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[4] Benchmark dose for 0.5% increased incidence of cancer over background.
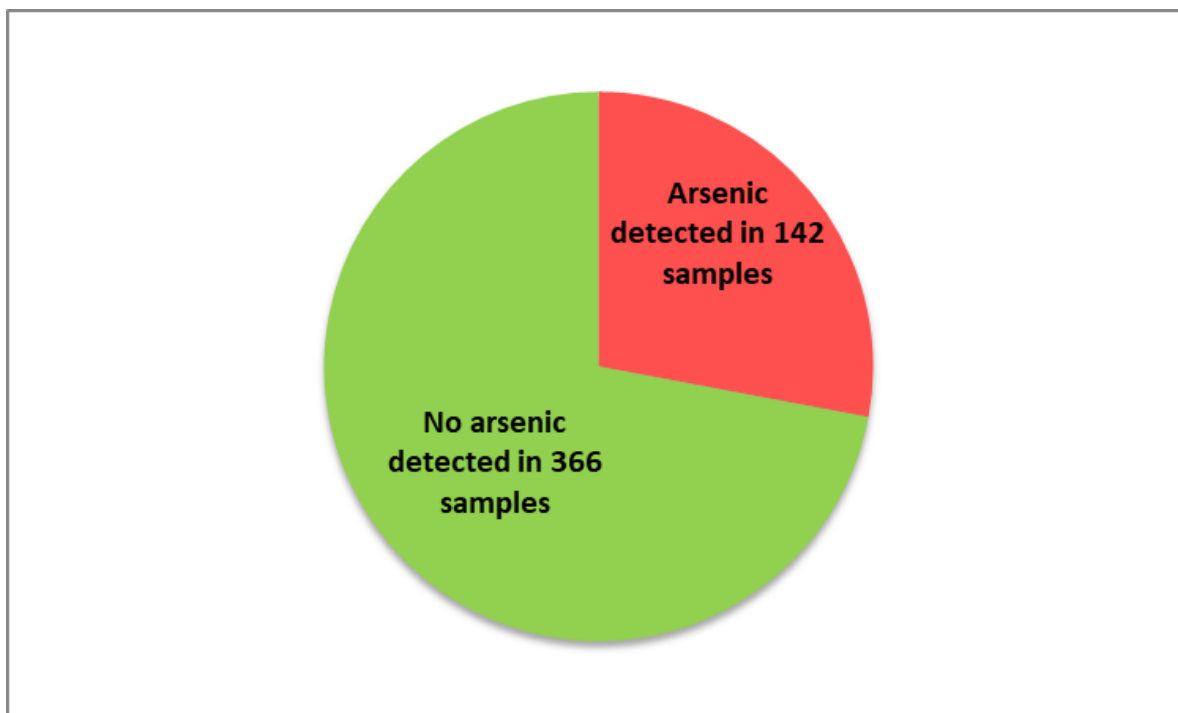
Figure 3 Number of composite food samples with detectable total arsenic residues

Arsenic was detected in several foods in all of their respective composite samples including breakfast cereals (both rice-based and wheat/corn based), fish (both takeaway fillets and frozen portions from supermarkets), garlic, infant cereal, mussels, prawns, rice and canned tuna. The highest mean concentrations of arsenic were found in various types of seafood. These results are not unexpected as fish and other seafood are widely cited in international scientific literature to be a large dietary source of arsenic. This study determined the highest mean concentrations (assuming nd=LOR) of arsenic in prawns (2.9 mg/kg), mussels (2.7 mg/kg), takeaway fish fillets (2.2 mg/kg), canned tuna (0.92 mg/kg) and frozen fish portions (0.88 mg/kg). The highest mean concentration of total arsenic to come from a non-seafood source was rice-based breakfast cereal (0.26 mg/kg).

Comparison against MLs and international levels

A comparison between the 25th ATDS results and MLs for contaminants as specified under Schedule 19 of Standard 1.4.1 was undertaken. There were no composite sample results which exceeded their corresponding commodity based ML for total arsenic in this study. These results indicate consistency with regulatory (the Code) requirements.

FSANZ undertook a review of international data investigating total arsenic levels in foods and compared these to results for foods determined to have the highest mean concentrations in the 25th ATDS. These results are shown in Table 5. Mean arsenic levels determined in the 25th ATDS were generally consistent with those reported in the international scientific literature, with fish and seafood having the highest levels.

Table 5 International mean concentrations of total arsenic in selected foods

ATDS food/beverage	Mean arsenic (total) concentration mg/kg (nd=LOR)				
	25th ATDS	23rd ATDS[1]	USA[2]^	EFSA[3]	NZ[4]*
Breakfast cereal, rice	0.26	0.037	0.16	0.17	-
Breakfast cereal, wheat or corn, single grain	0.017	0.037	0.001	0.030	0.029
Fish portions, frozen	0.88	0.80	0.50	1.5	1.0
Fish, plain from takeaway	2.2	3.9	0.50	1.5	2.7
Garlic	0.022	-	-	0.019	-
Infant cereal	0.020	0.027	0.0004-0.042	0.16	0.012
Mushrooms	0.033	0.54	0.051	0.070	0.19
Mussels	2.7	-	-	3.4	2.2
Prawns	2.9	1.4	0.32	5.7	-
Rice	0.041	0.088	0.066	0.17	0.033
Sausages, beef	0.010	0.020	0.001	0.017	0.002
Sultanas	0.021	0.025	-	0.027	0.024
Tuna	0.92	1.1	1.0	1.5	0.62
Water (tap)	0.0002	0.0006	-	0.002	0.001

1 FSANZ, 2011a

2 US FDA, 2017 ^lower bound values

3 EFSA, 2009a

4 NZ MAF, 2011 *middle bound values (values <LOR assigned a value ½ the LOR)

- Not analysed

Dietary exposure

Dietary exposures to total arsenic are expressed as a range; the lower end of the range (nd=0) represents where analytical results that were listed as 'not detected' are assumed to have a concentration of zero; the upper end of the range (nd=LOR) represents where analytical results that were listed as 'not detected' are assumed to have a concentration equal to the LOR. These results are shown in Figure 4.

At the lower bound (nd=0), estimated mean total arsenic dietary exposures for consumers were 0.37–0.90 µg/kg bw/day, with P90 dietary exposures being 1.3–2.8 µg/kg bw/day. At the upper bound (nd=LOR), estimated mean total arsenic dietary exposures were 0.49–1.2 µg/kg bw/day, with P90 dietary exposures being 1.4–3.1 µg/kg bw/day. The highest mean and P90 consumer dietary exposures to total arsenic, on a µg/kg bw/day basis, were for children aged 2 to 5 years. Teenagers aged 13 to 18 years had the lowest mean and P90 dietary exposures to total arsenic.

For infants aged 9 months, respondent lower bound (nd=0) mean and P90 dietary exposures to total arsenic were 0.6 μg/kg bw/day and 1.2 μg/kg bw/day, respectively. At the upper bound (nd=LOR), respondent mean and P90 dietary exposures were 1.0 μg/kg bw/day and 2.1 μg/kg bw/day, respectively.
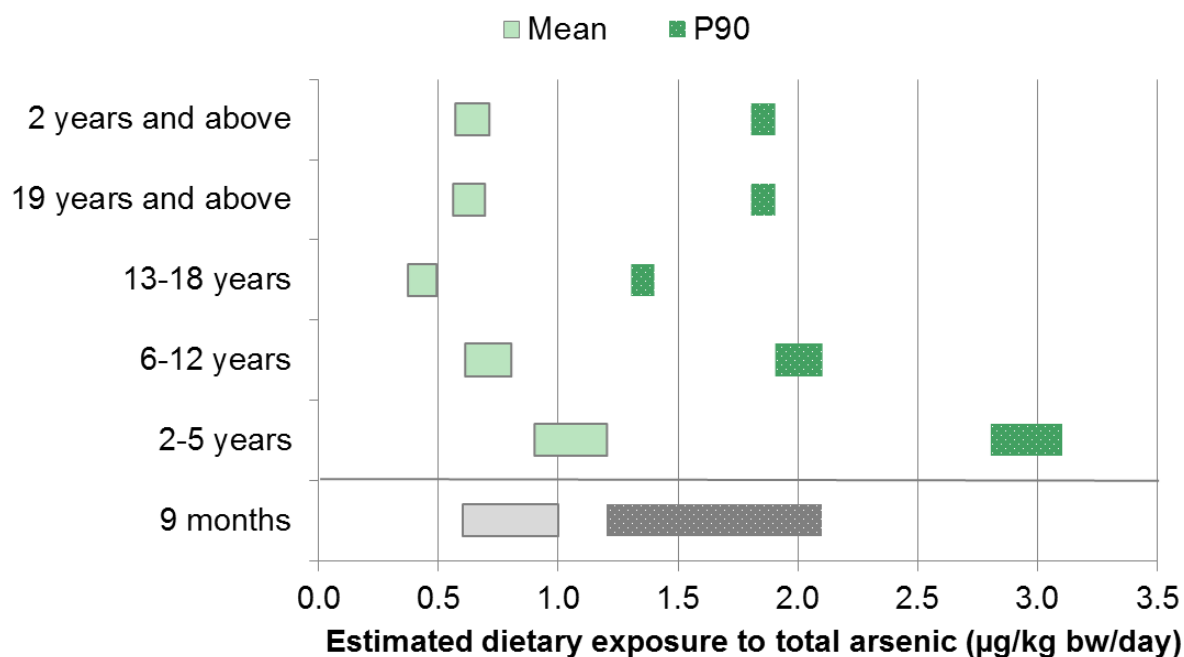

Figure 4 Estimated dietary exposures to total arsenic

Notes:
- *The lower end of each bar is for the lower bound (nd=0) scenario; the upper end of each bar is for the upper bound (nd=LOR) scenario.*
- *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant has consumed foods that contain total arsenic or not).*
- *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain total arsenic).*

Major food contributors

The major food categories contributing to total arsenic dietary exposures were 'meat, poultry, seafood and eggs' (66–87%) and 'cereals and cereal products' (8–20%) for all age groups assessed. 'Infant products' was a major contributing food category (20%) for infants aged 9 months, with 'Takeaway foods and snacks' being a major contributor (6%) for children aged 2 to 12 years and teenagers 13 to 18 years.

In the 'Meat, poultry, seafood and eggs' category, seafoods were the major contributing food group, with Plain fish (25–39%), Crumbed/battered fish and seafood (13–30%), and Tuna (all forms) and canned and smoked seafood (7–17%) the major contributing food groups to total arsenic dietary exposures for all age groups. Crustacea (5–16%) was a major contributing food group for all population groups, except for children aged 2 to 5 years.

In the 'Cereal and cereal products food category', Rice and rice products (5–9%) was the major contributing food group for all age groups. Rice-based breakfast cereals, flours and crackers was a major contributing food group (7–8%) for children aged 2 to 12 years. Infant cereals (20%) was the major contributing 'Infant food' to total arsenic dietary exposures for infants aged 9 months.

In the 'Takeaway foods and snacks category', Sushi roll (<1–5%) was the major contributing food group.

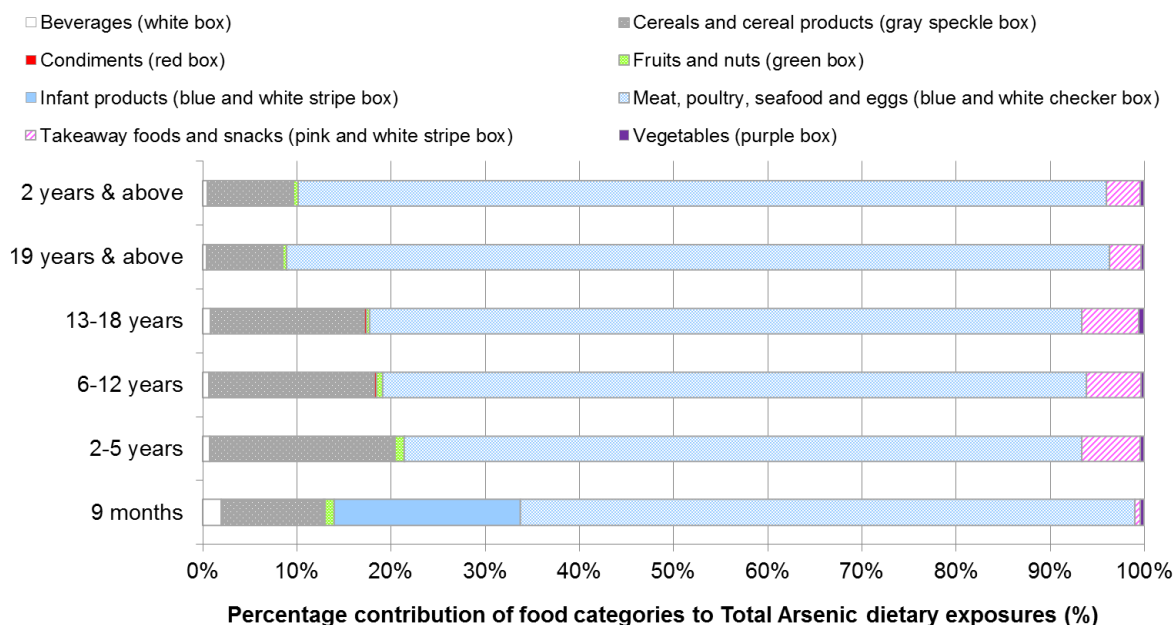For further information, refer to Table A 12.5 in Appendix 12 and Figure 5.

Figure 5 Contributing food categories to estimated total arsenic dietary exposures

Inorganic arsenic

Analytical Results

Inorganic arsenic was analysed in a limited number of food types including likely sources of dietary exposure. It was detected in three foods with mean concentrations (nd=LOR) in mussels of 0.28 mg/kg, white rice of 0.03 mg/kg and sushi rolls (nori) of 0.01 mg/kg. Other types of seafood including takeaway fish fillets, frozen fish portions, prawns and canned tuna had no detectable inorganic arsenic. These results were broadly consistent with the range of concentrations reported by JECFA (2011a) in seaweed (0.1–130 mg/kg), rice (0.01–0.51 mg/kg) and fish and fish products (0.001–1.2 mg/kg).

There were no exceedances of corresponding MLs for inorganic arsenic in composite samples.

Dietary exposure

In the 25th ATDS, inorganic arsenic dietary exposures were estimated using concentration data for the small number of foods analysed specifically for inorganic arsenic. This method, referred to as the *analysed samples only* method included various types of seafood, rice and sushi only. These foods are known to be high contributors to inorganic arsenic dietary exposure. In this case, when a food is not analysed for inorganic arsenic, it is assumed to have a concentration of zero for both the lower bound (nd=0) and upper bound (nd=LOR) scenarios.

Total arsenic concentrations in foods have also been used to estimate inorganic arsenic dietary exposures assuming that a proportion of total arsenic is inorganic (JECFA, 2011a). This method was used by FSANZ assuming a proportion (10%) of the total arsenic concentration as measured in all ATDS foods was inorganic. Estimated dietary exposures

were compared with the *analysed samples only* method to investigate the potential degree of uncertainty in the estimates. JECFA (2011a) noted that the proportion of inorganic arsenic in some foods can vary widely, and stated that dietary exposures to inorganic arsenic should be based on actual data rather than using conversion factors from total arsenic. For this reason this scenario is not discussed further here, and further details can be found in Appendix 7 and results in Appendix 12.

When using the *analysed samples only* method, the lower bound (nd=0), mean inorganic arsenic dietary exposures for consumers were 0.054–0.10 µg/kg bw/day, with P90 dietary exposures being 0.12–0.26 µg/kg bw/day. At the upper bound (nd=LOR), consumer mean inorganic arsenic dietary exposures were 0.042–0.092 µg/kg bw/day, with P90 dietary exposures being 0.10–0.23 µg/kg bw/day. These results are shown in Figure 6. The highest mean and P90 consumer dietary exposures to inorganic arsenic, on a µg/kg bw/day basis, were for children aged 2 to 5 years. The population aged 19 years and above had the lowest mean and P90 consumer dietary exposures to inorganic arsenic.

At the lower bound (nd=0), 35 to 41% of the population were consumers of inorganic arsenic, depending on the age group. At the upper bound (nd=LOR), 46 to 59% of the population were consumers of inorganic arsenic.

For infants aged 9 months, respondent lower bound (nd=0) mean and P90 dietary exposures to inorganic arsenic were 0.020 µg/kg bw/day and 0.040 µg/kg bw/day, respectively. At the upper bound (nd=LOR), respondent mean and P90 dietary exposures were 0.023 µg/kg bw/day and 0.046 µg/kg bw/day, respectively.
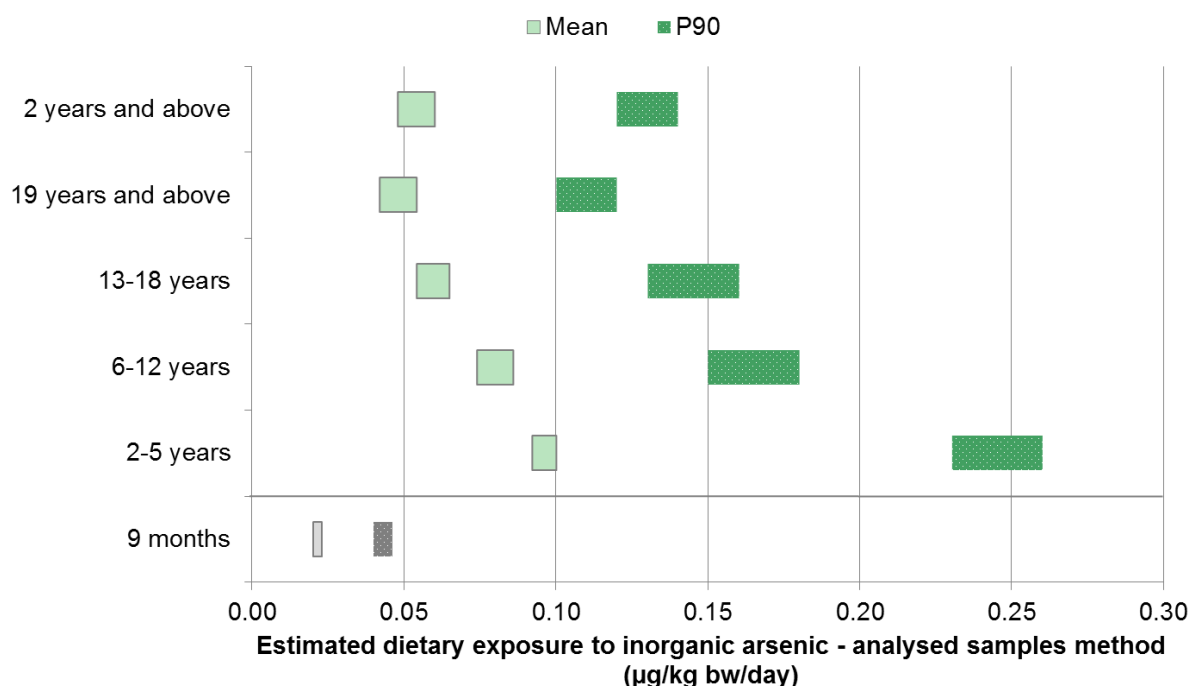

Figure 6 Estimated dietary exposures to inorganic arsenic (analysed samples method)

Notes:
- *For infants aged 9 months, the lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario. For all other age groups, the lower end of each bar is for the nd=LOR scenario; the upper end of each bar is for the nd=0 scenario.*
- *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain inorganic arsenic or not).*
- *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain inorganic arsenic).*

Major food contributors

The major contributing food category to inorganic arsenic dietary exposures based on the *analysed samples only* scenario was Cereals and cereal products (97–100%) for all age groups, with all of this being contributed by Rice and rice products. Meat, poultry, seafood and eggs, specifically Molluscs, were minor contributors to inorganic arsenic dietary exposures (<1–3%). The high contribution from rice reflects its relatively high consumption in the Australian diet compared to mussels, which was the only other food with detectable inorganic arsenic.

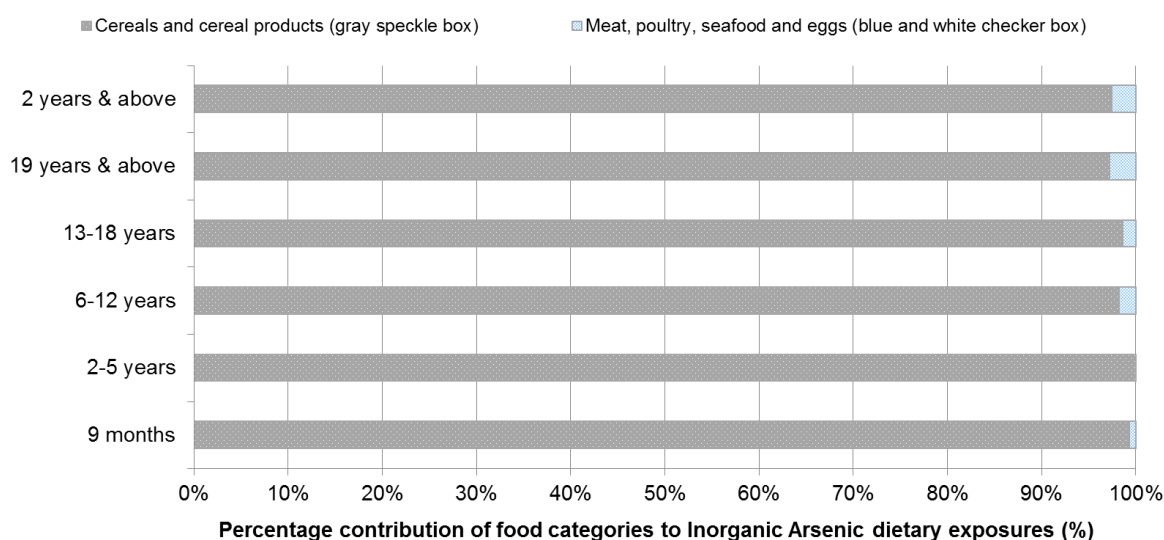For further information, refer to Table A 12.6 in Appendix 12 and Figure 7.

Figure 7 Contributing food categories to estimated inorganic arsenic dietary exposures (analysed samples method)

Risk characterisation

Arsenic is an environmental contaminant present in a wide range of foods, which means some level of dietary exposure is likely for the entire population. Levels of arsenic (total and inorganic) in foods sampled in the ATDS and estimates of dietary exposure for Australian consumers were generally consistent with those reported internationally. Organic arsenic compounds have very low toxic potential (EFSA 2005), in contrast to inorganic forms of arsenic which have been associated with a range of potential adverse effects.

Dietary exposure estimates for inorganic arsenic, based on a limited number of foods, were below the most sensitive point of departure established by JECFA (2011a), the $BMDL_{0.5}$ of 3.0 μg/kg bw/day, for all population subgroups assessed. Uncertainties associated with the $BMDL_{0.5}$ related to the lack of information on total exposure in the available epidemiological studies, and the relevance of extrapolation to other populations limit the utility of this data for further quantitative risk assessment.

Effects in human populations have generally been observed in populations exposed to high levels of inorganic arsenic in the drinking water (≥50-100 μg/L). JECFA (2011a) concluded that adverse effects of inorganic arsenic in water and food would be difficult to detect in human populations if the level in water is less than 50 μg/L. Levels of arsenic in Australian

reticulated water are low[5] such that it can be concluded that at the levels of inorganic arsenic in the Australian diet, it would be difficult to detect any adverse effects in epidemiological studies.

Further work would be required to collect more accurate information on the inorganic arsenic content of a broader range of foods as they are consumed in order to improve the assessment of dietary exposure to inorganic arsenic.

Risk management

Risk management measures are already in place for arsenic via MLs in the Code. Cereal grains and milled cereal products (including rice) are one of the groups of foods that have a 1 mg/kg ML for arsenic. This means that the arsenic level in any rice product available for sale in Australia and New Zealand must not be greater than 1 mg/kg.

FSANZ is aware that international analytical surveys of food, including infant foods, have detected the presence of arsenic in rice-based foods, however, there is little evidence of arsenic being detected in infant formula. The 25th ATDS found no detectable residues of arsenic in infant formula samples. In the US Food & Drug Administration (USFDA) summary of results from TDS market baskets from 2006 to 2013, total arsenic was measured in 32 samples of infant formula (milk based, iron fortified ready-to-feed), with no detections. It was also measured in ten samples of infant formula (milk based, low iron) with no detections. Additionally in September 2013 the USFDA reviewed infant formula in a survey of inorganic arsenic in rice and rice products. A total of 10 samples of infant formula were analysed and showed extremely low levels of arsenic.

FSANZ is currently contributing to targeted analytical survey work on arsenic in rice based products including rice-based infant formula and these results will be available in the future for consideration by FSANZ.

Conclusion

As arsenic is naturally present in a wide range of foods it is not possible to completely eliminate dietary exposure. As such, FSANZ advises eating a mixed diet with a variety of foods to limit exposure. FSANZ notes that the Codex Alimentarius Commission finalised a Code of Practice for the prevention and reduction of arsenic contamination in rice (at Step 5/8 [CL 2017/25-CF]). When fully implemented, this will assist with keeping arsenic levels in rice to ALARA.

Cadmium

Hazard identification

Cadmium exposure is associated with adverse effects on the kidney, skeletal and respiratory systems and it is also classified as a human carcinogen (WHO, 2018). The absorption of ingested cadmium through the gastrointestinal tract of humans is reported to be in the range of 1 to 10%, with the composition of the diet known to affect bioavailability. Animal studies indicate that cadmium is distributed to the liver and kidneys where it accumulates over time, accounting for 50 to 75% of total body burden, with an additional 20% typically found in muscle. Cadmium does not accumulate at significant levels in the bone and is not associated with demineralisation (JECFA, 2011c).

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[5] The level of arsenic specified in the 2011 Australian Drinking Water Guidelines version 3.4; updated October 2017 (NHMRC 2011) is 10 µg/L.

The 73rd JECFA meeting (2011c) considered contemporary toxicology data including a meta-analysis of several epidemiological studies reporting the correlation between ß_2-microglobulin (ß2MG), a marker of impaired renal function, and cadmium levels in urine. Considering that the half-life of cadmium in the human kidney is approximately 15 years, the dose analysis was restricted to individuals aged 50 years and over, who were expected to exhibit a steady-state of biomarkers in the urine. It was concluded that urinary excretion of cadmium at less than 5.24 µg/g of creatinine, corresponding with an estimated dietary exposure of 0.8 µg/kg bw/day, was not associated with increased excretion of ß2MG.

Due to the long half-life of cadmium, it was considered that exposure should be assessed on at least a monthly basis, resulting in the establishment of a PTMI of 25 µg/kg bw (JECFA, 2011c). FSANZ has used this PTMI for the risk assessment including comparison against estimated dietary exposures.

Analytical results

A summary of results for cadmium for foods analysed in the 25th ATDS can be found in Table A 3.3 in Appendix 3. Cadmium was detected in 43 of the 88 foods analysed. Out of all 508 composite samples analysed, 149 (29%) had detectable residues of cadmium. A total of 359 composite samples (71%) had no detectable residues of cadmium. As shown in Figure 10 these results indicate that a large proportion of foods consumed in Australia contain no detectable residues of cadmium.
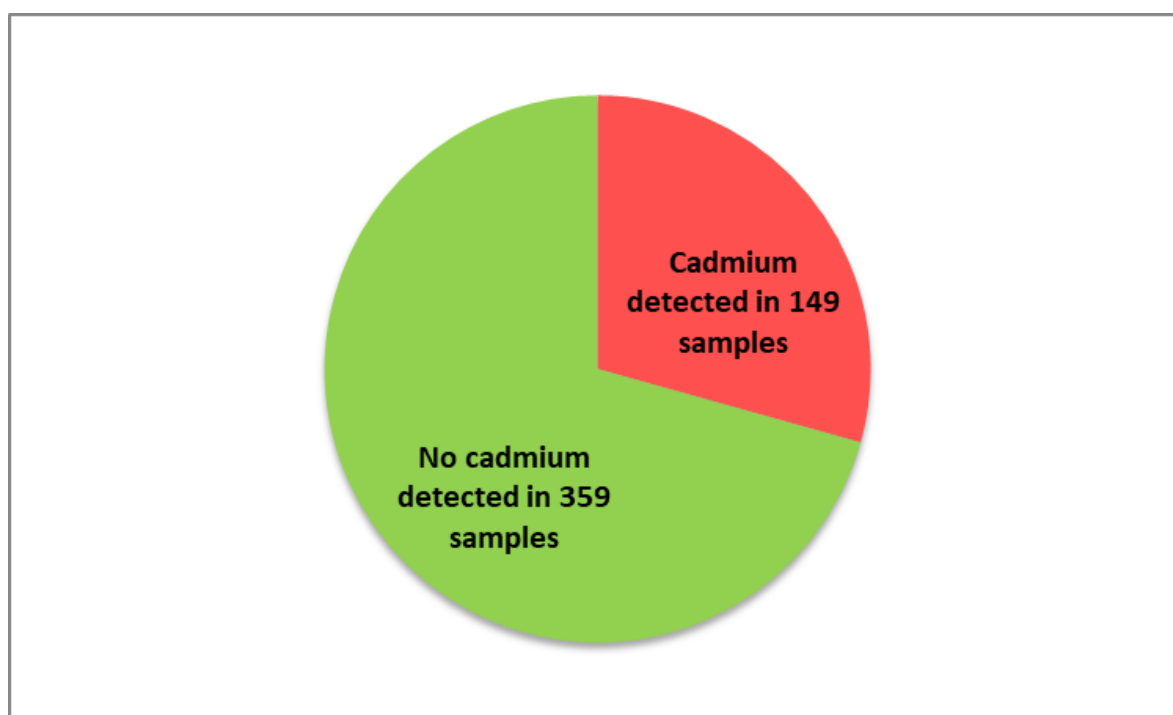

Figure 8 Number of composite food samples with detectable cadmium residues

A total of 14 of the 43 foods determined to contain cadmium had detections in all of their respective composite samples. These included mussels, potato chips, potato, peanut butter, desiccated coconut, garlic, chocolate cake, chocolate, multigrain bread, canned beetroot, canned tuna, carrots, canned tomatoes and pizza.

The highest mean concentrations of cadmium (assuming nd=LOR) were found in mussels (mean concentration 0.20 mg/kg), prawns (0.065 mg/kg), potato crisps (0.058 mg/kg) and potato (0.026 mg/kg). All other mean concentrations of cadmium were less than 0.020 mg/kg. These results are consistent with the known distribution of cadmium in the environment, with

highest concentrations in foods typically associated with shellfish, crustaceans and to a lesser extent, starchy vegetables.

Comparison against MLs and international levels

A comparison between 25th ATDS results and the MLs for cadmium in Schedule 19 of Standard 1.4.1 of the Code was undertaken. There were no composite sample results which exceeded their corresponding ML for cadmium in this study, indicating consistency with regulatory requirements.

FSANZ reviewed recent international data investigating cadmium concentrations in food and compared these to foods determined to have the highest concentrations as part of the 25th ATDS, shown in Table 6. Mean cadmium concentrations determined in the 25th ATDS were generally consistent with those reported in the international scientific literature. The 25th ATDS foods with the highest mean concentrations of cadmium, including mussels and prawns, were also reported to have relatively higher cadmium concentrations in Europe and New Zealand. Mean cadmium concentrations in potatoes and potato crisps were generally on the lower end of the international scale with higher levels reported in Europe and New Zealand. Other foods with results of note from the international literature included beetroot, chocolate and peanut butter which had relatively higher cadmium concentrations compared to those found in the 25th ATDS.

Table 6 International mean concentrations of cadmium in selected foods

ATDS food/beverage	Mean cadmium concentration mg/kg (nd=LOR)				
	25th ATDS	23rd ATDS[1]	US FDA[2]	EFSA[3]	NZ[4*]
Avocado	0.014	0.029	0.012	0.009	0.021
Beetroot, canned	0.015	0.009	0.018	0.068	0.011
Bread multigrain	0.015	0.019	0.022	0.016	0.017
Cake, chocolate, iced	0.016	0.025	0.015	0.020	0.005
Chocolate, milk	0.015	0.042	0.021	0.021	0.039
Coconut, desiccated	0.018	0.019	-	-	-
Garlic	0.017	-	-	0.018	-
Mussels	0.20	-	-	0.32	0.19
Peanut butter	0.019	0.027	0.039	0.038	0.054
Potato	0.026	0.033	0.021	0.068	0.040
Potato crisps	0.058	0.11	0.054	0.027	0.13
Prawns	0.065	0.014	0.006	0.14	-
Sauce, tomato	0.016	0.015	0.013	0.011	0.015
Strawberries	0.015	0.029	0.015	0.005	0.004
Tuna, canned	0.012	0.012	0.015	0.021	0.008
Water	0.0001	0.0002	0.0001	0.0004	0.0005

1 FSANZ, 2011a
2 US FDA, 2017 ^lower bound values
3 EFSA, 2012a
4 NZ MAF, 2011 *middle bound values (values <LOR assigned a value ½ the LOR)

Dietary exposure

Dietary exposures to cadmium are expressed as a range; the lower end of the range (nd=0) represents where analytical results that were listed as 'not detected' are assumed to have a concentration of zero; the upper end of the range (nd=LOR) represents were analytical results that were listed as 'not detected' are assumed to have a concentration equal to the LOR.

At the lower bound (nd=0), mean cadmium dietary exposures for consumers were 2.0–5.5 µg/kg bw/month, with P90 dietary exposures of 3.7–9.9 µg/kg bw/month. At the upper bound (nd=LOR), mean dietary exposures were 5.8–14 µg/kg bw/month, with P90 dietary exposures of 8.8–20 µg/kg bw/day.

The highest mean and P90 dietary exposure, on a µg/kg bw/month basis, were for children aged 2 to 5 years. Adults aged 19 years and above had the lowest mean and P90 dietary exposures to cadmium.

For infants aged 9 months, respondent lower bound (nd=0) mean and P90 dietary exposures were 2.8 µg/kg bw/month and 5.7 µg/kg bw/month, respectively. At the upper bound (nd=LOR), respondent mean and P90 dietary exposures were 16 µg/kg bw/day and 33 µg/kg bw/month, respectively.

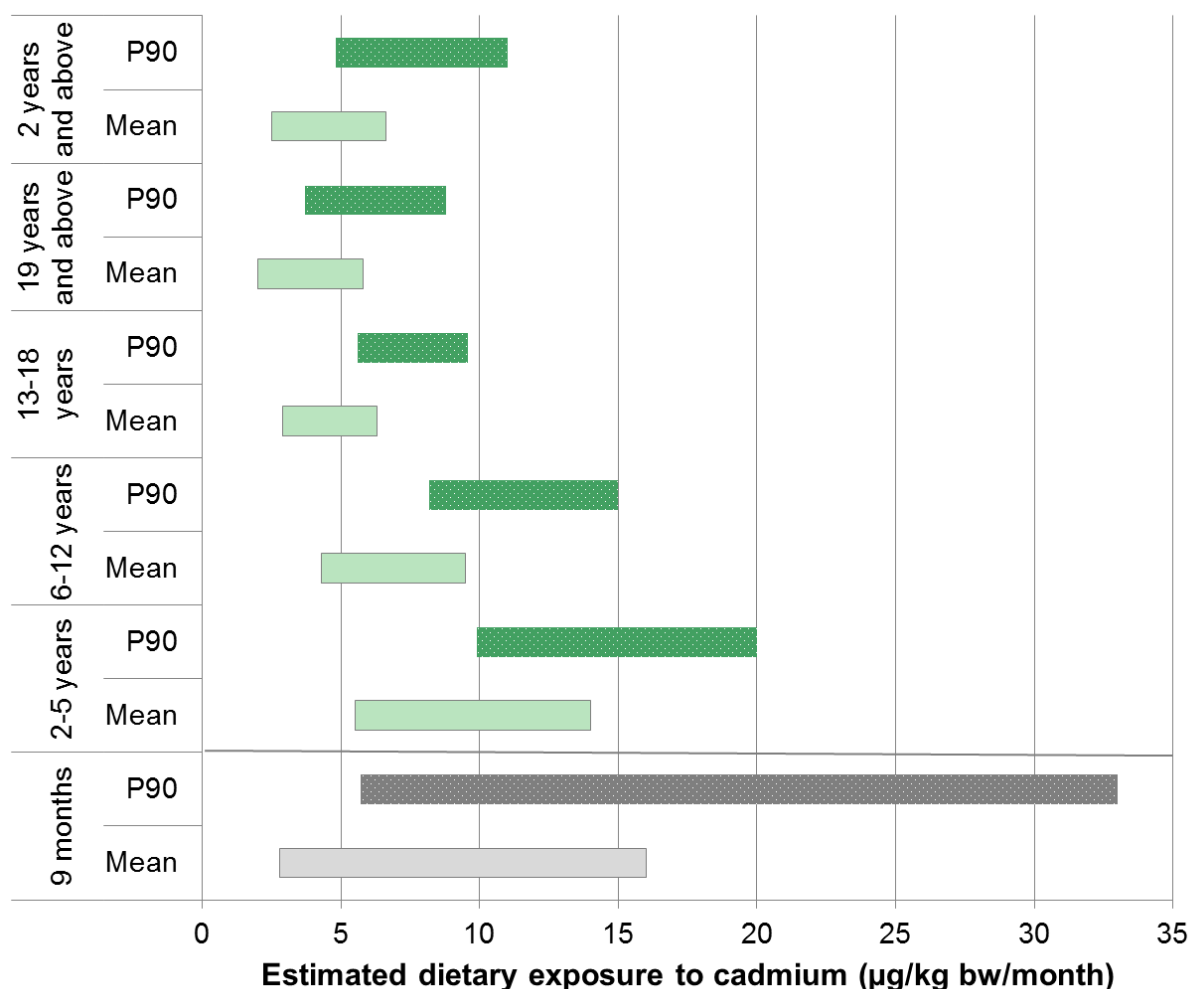Further details are provided in Table A 12.2 in Appendix 12 and in Figure 9.

Figure 9 Estimated dietary exposures to cadmium

Notes:
- *The lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario.*
- *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain cadmium or not).*
- *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain cadmium).*

Major food contributors

The major contributing food categories to cadmium dietary exposures were vegetables (38–50%), cereals and cereal products (22–35%) and takeaway foods and snacks (8–17%) for all age groups assessed. Meat, poultry, seafood and eggs were major contributors for adults aged 19 years and above and for the general population aged 2 years and above (6%). Fruits and nuts were a major contributing food category for infants aged 9 months, children aged 2 to 5 years and adults aged 19 years and above (5–9%).

Beverages (2–4%), condiments (2%), infant products (0 to <1%) and sugars and confectionary (1–2%) were minor contributing food categories to cadmium dietary exposures for all population groups.

In the vegetables category, root vegetables (starchy) (30–42%) was the major contributing food group for all age groups. Root vegetables (starchy) was a major contributing food category for infants aged 9 months. Beetroot (<1%), garlic (<1%), leafy vegetables and herbs (<1 to 1%), onions, shallots, spring onions and leeks (<1%), and tomatoes/eggplant/okra (cooked or processed) (2–3%) were minor contributing food groups for all age groups.

In the cereals and cereal products category, cakes, muffins, pudding & doughnuts (6–11%) was a major contributing food group for all age groups assessed. Multigrain, wholemeal, spelt and rye breads (6–12%) was a major contributing food group for all age groups except teenagers 13 to 18 years. Commercial biscuits and crackers (<1–2%), pasta, noodles (except rice) and couscous (3–4%), rice and rice products (2–3%), rice-based breakfast cereal, flours and crackers (<1 to 1%), wheat- and non-rice based breakfast cereals and flours (0–2%) and White breads (including high-fibre white) (2–3%) were minor contributing food groups.

In the takeaway foods and snack category, crisps (chips) and savoury snacks (5–12%) was a major contributing food groups for all age groups. Hamburgers (all meat types) (1–3%), pizzas (1–2%) and sushi roll (<1 to 1%) were minor contributing food groups to cadmium dietary exposures for all age groups.

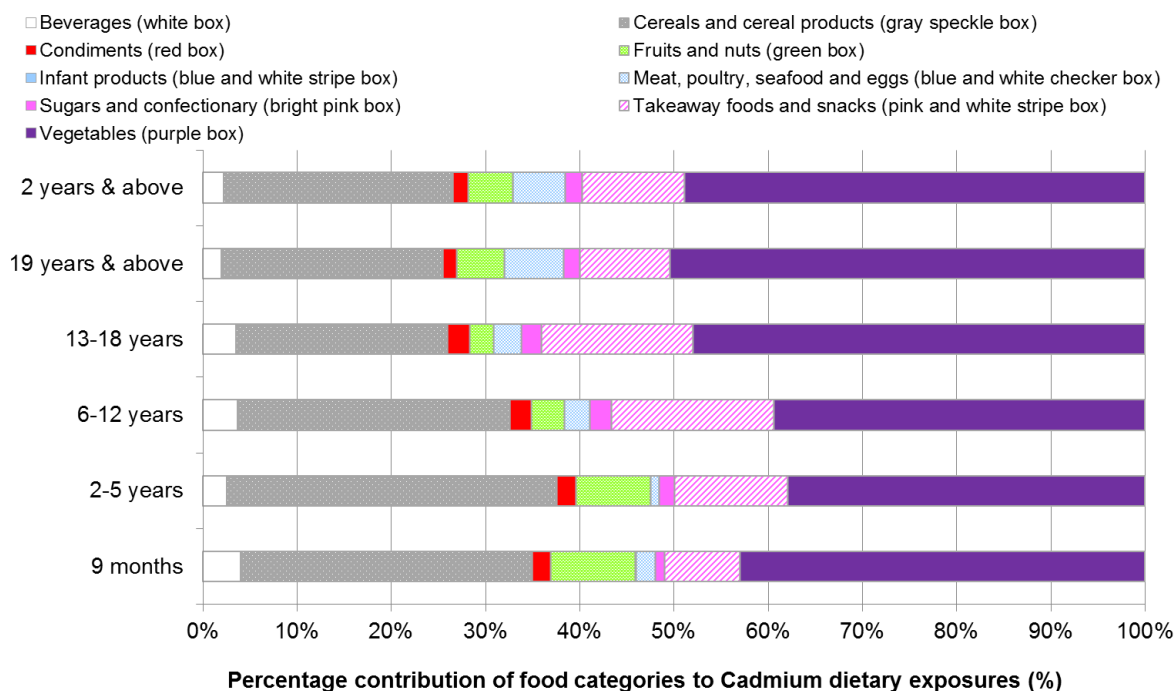For further information, refer to Table A 12.8 in Appendix 12 and Figure 10.

Figure 10 Contributing food categories to estimated cadmium dietary exposures

Risk characterisation

Cadmium is an environmental contaminant and is present in a wide range of foods, which means some level of dietary exposure is likely for the entire population. Levels of cadmium in foods sampled in the ATDS and estimates of dietary exposure for Australian consumers were generally consistent with those reported internationally.

Dietary exposures to cadmium were below the PTMI for all population subgroups assessed, with the exception of the 90th percentile for infants aged 9 months, for which there was a slight exceedance (130%) at the upper bound of the dietary exposure estimate. Adverse effects of cadmium on the kidney in human studies are associated with bioaccumulation over

many years. Therefore, the slight and short term exceedance in infancy is unlikely to represent a significant public health and safety concern.
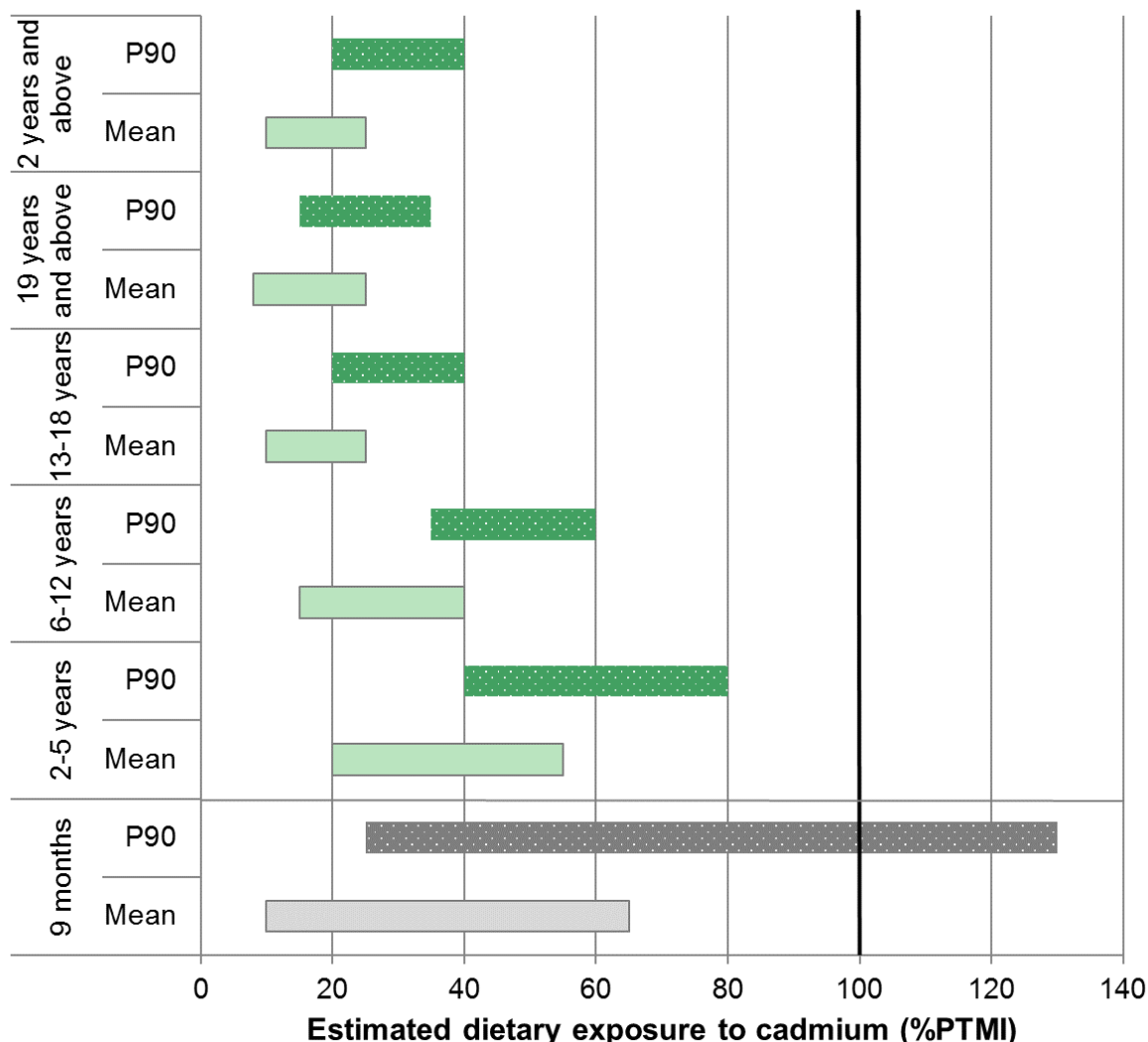

Figure 11 Estimated dietary exposures to cadmium as percentage of the PTMI

> Notes:
> - The lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario.
> - Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain cadmium or not).
> - Dietary exposures for age groups aged 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain cadmium).

Risk management

There are already MLs in place for cadmium in the Code and no additional risk management measures are needed at this point in time.

Cadmium contained in soil and water can be taken up by certain crops and aquatic organisms and accumulate in the food chain. The major dietary sources of cadmium in Australia are the following foods:

- root vegetables (starchy)
- crisps (chips) and savoury snacks
- multigrain, wholemeal, spelt and rye breads

- cakes, muffins, puddings & doughnuts
- root vegetables (nonstarchy) (except beetroot)
- berries.

The presence of cadmium in vegetables may be due to the absorption of cadmium through the roots. Atmospheric fallout can also contribute, especially for leafy vegetables. In general, cadmium accumulates in the leaves of plants and therefore high levels can be found in leafy vegetables such as spinach.

An adequate amount of vegetable intake is an essential component of healthy eating. Maintaining a balanced diet with a variety of leafy and non-leafy vegetables can avoid excessive exposure to cadmium from a small range of food items. In addition, washing vegetables and peeling roots and tubers can reduce cadmium contamination to some extent.

Due to their high consumption, cereals, including wheat and rice as well as their derived products, are one of the major dietary sources of cadmium. Cadmium can mainly be found in the outer parts of the grain, which are wholly or partly removed in the milling process. Nevertheless, consumption of whole grains is generally recommended as part of a healthy diet due to the presence of important nutrients such as dietary fibre. All in all, it is essential to eat a balanced and varied diet.

Lead

Hazard identification

Lead exposure is associated with a wide range of adverse effects on multiple body systems, with young children particularly susceptible to harmful effects on the brain and central nervous system. After exposure, lead is distributed to the brain, liver, kidneys and accumulates in the bone and teeth. The majority of metabolism studies indicate age-dependent differences in uptake from the gastrointestinal tract, with children generally absorbing more ingested lead than adults. Apart from neurodevelopmental effects in children, which are considered to be the most sensitive toxicological endpoint, lead exposure is also associated with cardiovascular disease, impaired renal function, hypertension, impaired fertility and adverse reproductive outcomes (JECFA, 2011d).

The 73rd JECFA meeting (2011d) evaluated data from a meta-analysis of seven longitudinal cohort studies conducted in the USA, Mexico, Kosovo and Australia and found that exposure to lead at the previously established PTWI of 25 µg/kg body weight was associated with an estimated population decrease of at least 3 IQ points in children and an increase in systolic blood pressure of approximately 3 mm Hg in adults. With regard to these findings, the Committee concluded that the PTWI could no longer be considered adequately protective of human health and it was therefore withdrawn. A dose analysis of the epidemiological data was undertaken, with a dietary exposure of 0.3 µg/kg bw/day associated with a population decrease of 0.5 IQ points in children. Estimated points of departure were established including 0.6 µg/kg body weight/day for children (associated with a decrease of 1 IQ point) and 1.2 µg/kg body weight/day for adults (associated with a 1 mm Hg increase in blood pressure) (JECFA, 2011d). For the purposes of the risk assessment, FSANZ used toxicological endpoints established by JECFA (2011d) (including 0.3 µg/kg body weight/day for decreased IQ in children and 1.2 µg/kg body weight/day for increased blood pressure in adults) for comparison against estimated dietary exposures.

Analytical results

A summary of results for lead in the 25th ATDS can be found in Table A 3.4 of Appendix 3. Lead was detected in 36 of the 88 foods sampled. Of all 508 composite samples analysed,

76 (15%) had detectable residues of lead. A total of 432 composite samples (85%) had no detectable residues of lead. As shown in Figure 14 these results indicate that a large proportion of foods consumed in Australia contain no detectable residues of lead.
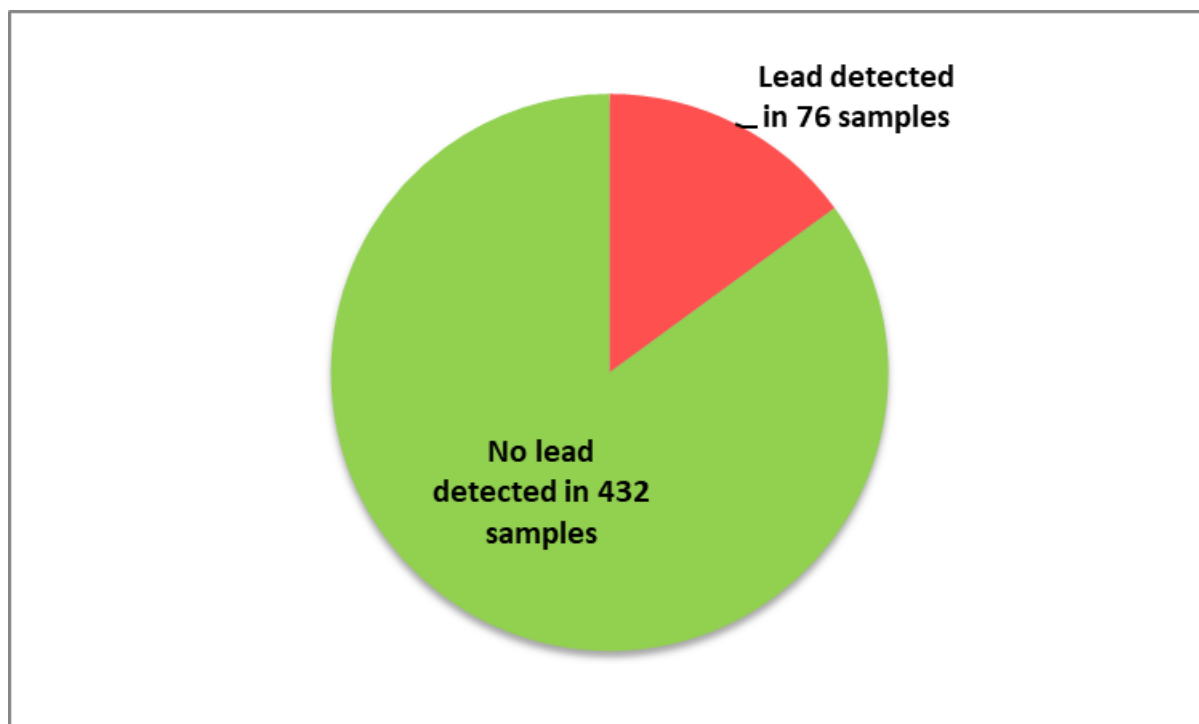

Figure 12 Number of composite food samples with detectable lead residues

Several foods were determined to have lead in all of their respective composite samples including mussels, sultanas, chocolate cake, honey, peaches (packaged in natural juice), canned pineapple and chocolate. The highest mean concentrations (assuming nd=LOR) were detected in mussels (mean concentration 0.074 mg/kg), sultanas (0.037 mg/kg), chocolate cake (0.026 mg/kg) and honey (0.024 mg/kg). All other foods had mean lead concentrations less than 0.020 mg/kg.

Comparison against MLs and international levels

FSANZ compared the 25th ATDS results to the MLs for lead in Schedule 19 of Standard 1.4.1. No composite sample results exceeded the corresponding ML in this study. These results indicate that dietary exposure to lead is acceptable.

FSANZ reviewed international data investigating lead levels in foods and compared these to results for foods found to have the highest concentrations in the 25th ATDS as shown in Table 7. Mean concentrations of lead in mussels and chocolate were consistently well below those reported in the previous 23rd ATDS, the United States, Europe and New Zealand. However, the mean concentration of lead in sultanas found in the 25th ATDS was higher than those reported in the international literature.

Table 7 International mean concentrations of lead in selected foods

ATDS food/beverage	Mean lead concentration mg/kg (nd=LOR)				
	25th ATDS	23rd ATDS[1]	US FDA[2^]	EFSA[3]	NZ[4*]
Cake, chocolate, iced	0.026	0.023	0.006	0.022	-

ATDS food/beverage	Mean lead concentration mg/kg (nd=LOR)				
	25th ATDS	23rd ATDS[1]	US FDA[2]^	EFSA[3]	NZ[4]*
Chocolate, milk	0.006	0.028	0.011	0.039	0.027
Honey	0.024	0.052	0.004	0.041	0.010
Mussels	0.074	-	-	0.23	0.10
Peach, natural juice	0.019	0.017	0.012	0.025	0.037
Pineapple, natural juice	0.010	0.024	0.007	0.025	0.016
Sultanas	0.037	0.013	0.004 (raisins)	0.022	0.023
Water	0.0004	0.006	0.0004	0.0006	0.0002

1 FSANZ, 2011a

2 US FDA, 2017^lower bound values

3 EFSA, 2012b

4 NZ MAF, 2011*middle bound values

Dietary exposure

Dietary exposures to lead are expressed as a range; the lower end of the range (nd=0) represents where analytical results that were listed as 'not detected' are assumed to have a concentration of zero; the upper end of the range (nd=LOR) represents were analytical results that were listed as 'not detected' are assumed to have a concentration equal to the LOR for that matrix.

At the lower bound (nd=0), mean dietary exposures were 0.016–0.048 µg/kg bw/day, with P90 dietary exposures of 0.032–0.10 µg/kg bw/day. At the upper bound (nd=LOR), mean dietary exposures were 0.16–0.38 µg/kg bw/day, with P90 dietary exposures of 0.23–0.56 µg/kg bw/day. The highest mean and P90 dietary exposures, on a µg/kg bw/day basis, were for children aged 2 to 5 years. Teenagers aged 13 to 18 years had the lowest mean and P90 dietary exposures to lead.

For infants aged 9 months, respondent lower bound (nd=0) mean and P90 dietary exposures were 0.040 µg/kg bw/day and 0.079 µg/kg bw/day, respectively. At the upper bound (nd=LOR), respondent mean and P90 dietary exposures were 0.51 µg/kg bw/day and 1.0 µg/kg bw/day, respectively.

Further details are provided in Table A 12.3 in Appendix 12 and in Figure 15.
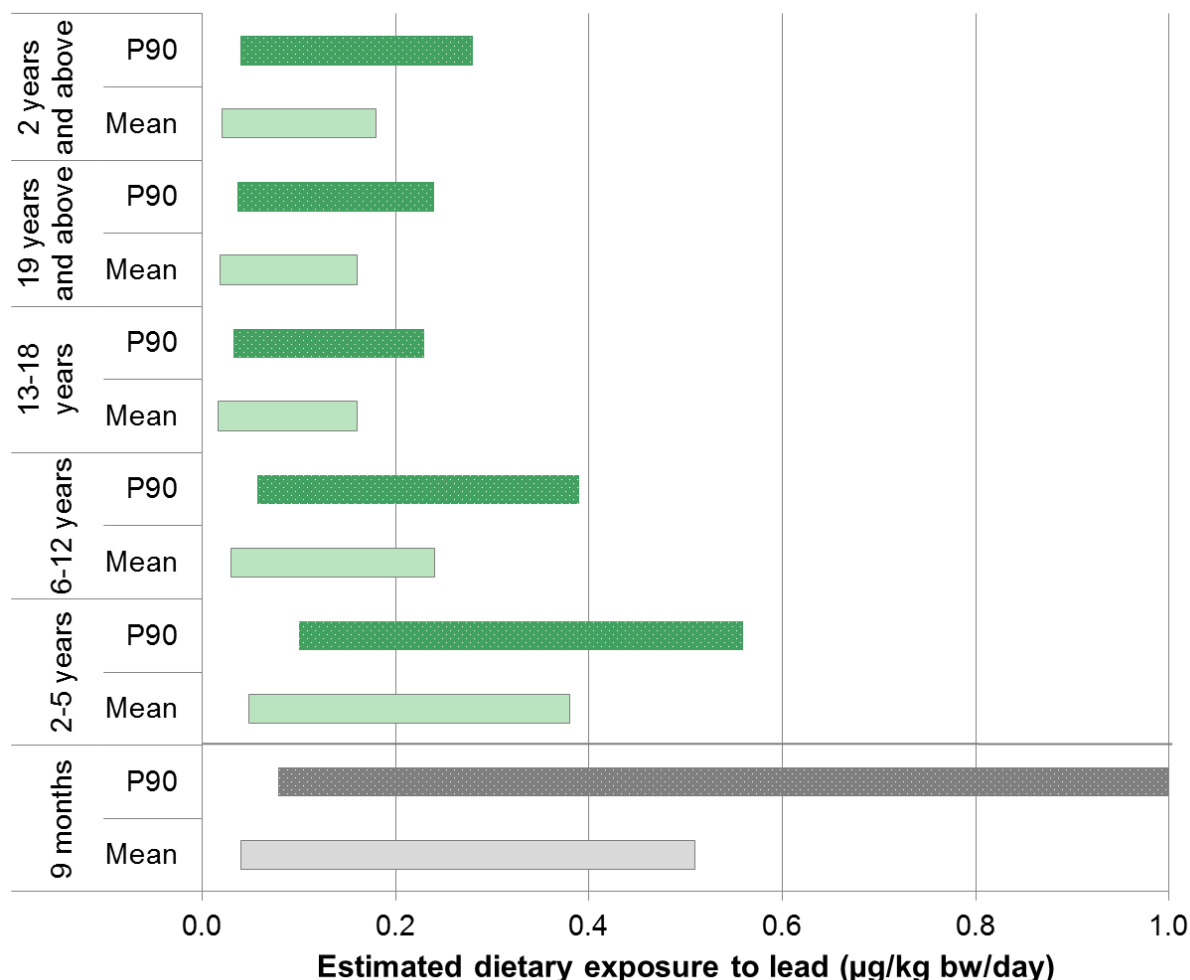
Figure 13 Estimated dietary exposures to lead

Notes:
- *The lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario.*
- *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain lead or not).*
- *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain lead).*

Major food contributors

The major contributing food categories to lead exposures were 'Beverages' (28–57%), 'Fruits and nuts' (14–36%) and 'Cereals and cereal products' (9–29%) for all age groups assessed. 'Meat, poultry, seafood and eggs' (8–10%) was a major contributor for those aged 6 years and above and for the general population aged 2 years and above. 'Sugars and confectionary' (6–9%) was a major contributing food category for all population groups aged 2 years and above. 'Vegetables' was a minor contributor (2–4%) for all population groups.

In the 'beverages category', water (all sources) and intensely sweetened soft drinks (28–57%) were a major contributing food group for all age groups. Wine and wine products was a major food category contributor for adults aged 19 years and above (14%), and also when included as part of the total dietary exposure for the general population aged 2 years and above (12%).

In the 'cereals and cereal' products category, all dietary exposure contributions were from cakes, muffins, puddings and doughnuts (9–29%).

Dried grapes/figs/dates and prunes were a major contributing food group for all age groups assessed (8–24%). Canned fruits (excluding pineapple) (5–9%) was a major contributor to lead exposures for all age groups except for teenagers 13 to 18 years. Pineapple and jackfruit (2–3%) was a minor contributor to lead dietary exposures for all age groups.

Within the 'meat, poultry, seafood and eggs' category, pork (except bacon) and deli meats (except frankfurts and poultry-based) was the major contributing food group (6–7%) for those aged 6 years and above and for the general population aged 2 years and above. Crustacea (<1%) and Molluscs (<1 to 4%) were minor contributing food groups to lead dietary exposures.

In the 'sugars and confectionary' food category, chocolates and fudge was a minor contributing food group for all age groups except for teenagers 13 to 18 years (5%). Honey was a minor contributing food group for all age groups except for children aged 2 to 5 years (6%).

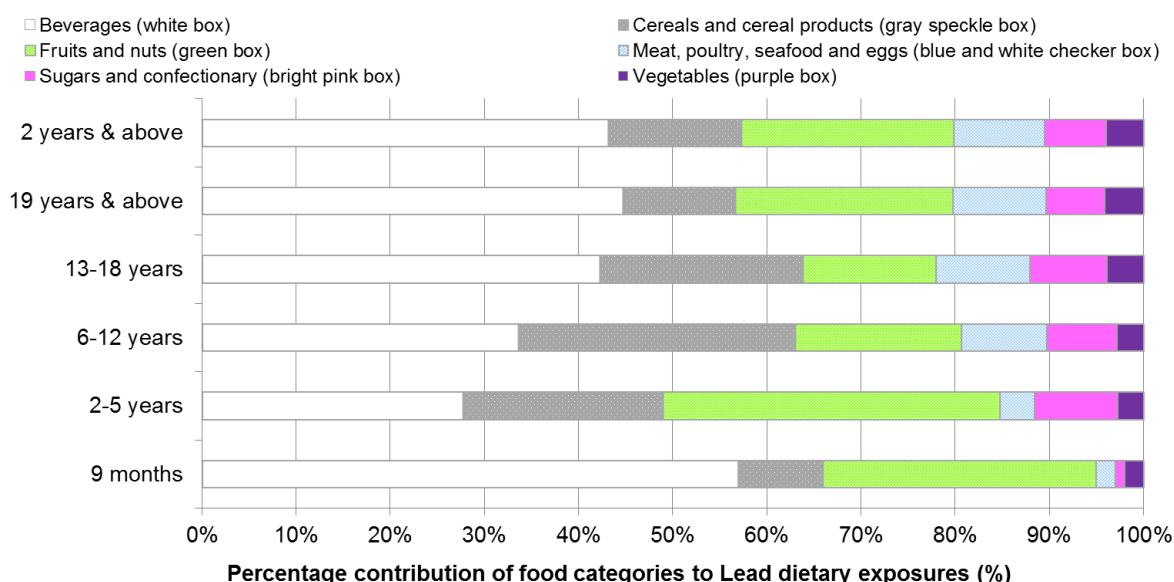For further information, refer to Table A 12.9 in Appendix 12 and Figure 16.

Figure 14 Contributing food categories to estimated lead exposures

Risk characterisation

Lead is an environmental contaminant and present in a wide range of foods in the Australian diet meaning that some level of dietary exposure is likely for the entire population. Levels of lead in foods sampled in the ATDS and estimates of dietary exposure for Australian consumers were generally consistent with those reported internationally.

In 2010, JECFA concluded that for children aged 1 to 4 years of age, a lead exposure of 0.3 µg/kg bw/day could result in a population decrease of 0.5 IQ points. For adults, an exposure of 1.2 µg/kg bw/day could result in a population increase in systolic blood pressure of 1 mm Hg.

Dietary exposure to lead for the Australian population, presented in Table 8, reveals that the mean lower bound exposures are below the levels considered by JECFA to have a low risk of reducing the population IQ for children or increased blood pressure in adults. Estimated

MOEs based on mean lower bound exposures were between 6 and 80 for all population sub-groups assessed. For the other dietary exposure scenarios (mean upper bound, P90 lower and upper bound) the MOEs were lower (at between 0.5 to 40). MOEs were only less than 1 at the P90 upper bound dietary exposures for the children aged 2–12 years. Overall, these results indicate that dietary exposures to lead for most Australian consumers are lower than levels found to be of negligible risk of causing adverse health effects. P90 dietary exposures for some population sub-groups with lower MOEs are not considered to be of concern due to the high-level of conservatism in the calculations. Dietary exposures are likely to be lower than those estimated at the upper bound as they are based on where a not detected analytical result for any food is assigned a concentration equal to the LOR. In reality, lead concentrations and therefore exposures will be somewhere between the lower and upper bound. Only two days of food consumption data are available for estimating long term exposure. More days of data have the effect of bringing in the tails of the exposure distributions and lowering P90 exposures. Dietary exposures will fluctuate day to day and are not likely to be at P90 level of exposure over a number of years.

For these reasons, risks for Australian consumers are considered to be acceptably low.

Risk management

Risk management measures are already in place for lead via MLs in the Code. No exceedance of the current lead MLs were found in foods sampled for the 25th ATDS, suggesting that levels of lead are ALARA.

Table 8 Mean and P90 lead dietary exposures on a body weight basis and expressed as MOEs

Population Group	Consumer Dietary Exposure (µg/kg bw/day)				Margin of Exposure (MOE)			
	Mean (nd=0)	Mean (nd=LOR)	P90 (nd=0)	P90 (nd=LOR)	Mean (nd=0)	Mean (nd=LOR)	P90 (nd=0)	P90 (nd=LOR)
2-5 yearsʸ	0.048	0.380	0.100	0.560	6	0.8	3	0.5
6-12 yearsʸ	0.029	0.240	0.057	0.390	10	1	5	0.8
13-18 years*	0.016	0.160	0.032	0.230	80	8	40	5
19 years and above*	0.018	0.160	0.036	0.240	70	8	30	5

ʸ   dose estimate for lead for children aged 1–12 years is 0.3 µg/kg bw/ day
*   dose estimate for lead for Australians aged 13 years and above is 1.2 µg/kg bw/ day

Mercury

Total mercury

Hazard identification

Mercury is considered by WHO to be a major health concern, and has the potential to adversely affect the central and peripheral nervous systems, the digestive and immune systems, and the lungs and kidneys (WHO, 2017b). Due to differences in biological effects and occurrence in foods; inorganic mercury and methylmercury are considered separately for the purposes of the risk assessment.

Analytical results

A summary of results for total mercury in the 25th ATDS is provided in Table A 3.7 in Appendix 3. Mercury was detected in 14 of the 88 foods sampled, with six of these being seafood. Out of all 508 composite samples analysed, 40 (8%) had detectable residues of mercury. A total of 468 composite samples (92%) had no detectable residues of mercury. As shown in Figure 17, these results indicate that a large proportion of foods consumed in Australia contain no detectable residues of mercury.
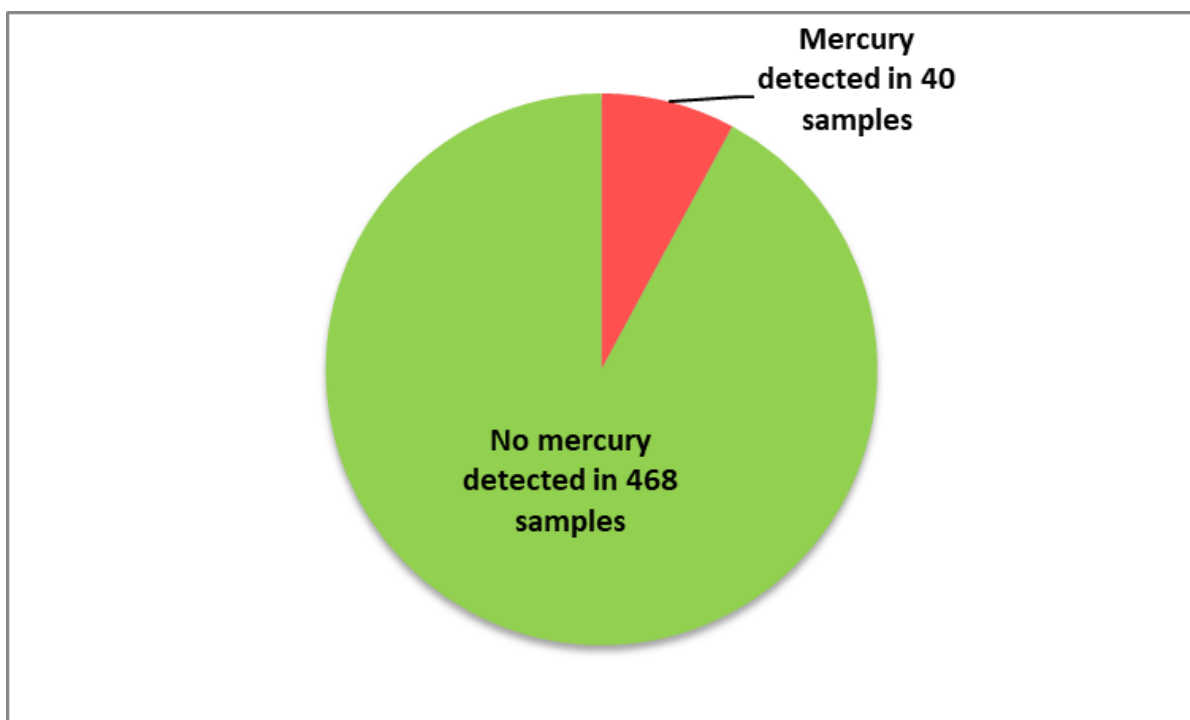

Figure 15 Number of composite food samples with detectable total mercury residues

A limited number of foods were determined to contain mercury in all of their respective composite samples including takeaway fish fillets, frozen fish portions, canned tuna and mussels. This study found the highest mean concentrations (assuming nd=LOR) of mercury in takeaway fish fillets (0.13 mg/kg), frozen fish portions (0.048 mg/kg) and also canned tuna (0.046 mg/kg). These results are consistent with the international scientific literature and the well-known tendency of mercury to concentrate in fish and seafood (particularly species high in the food chain).

Comparison against MLs and international levels

A comparison between the 25th ATDS results and MLs for mercury in Schedule 19 of Standard 1.4.1 of the Code was undertaken. There were no composite sample results which exceeded their corresponding ML for mercury in this study. These results indicate consistency with regulatory requirements.

FSANZ reviewed international data that investigated total mercury levels in foods and these results appear in Table 9. Mean mercury levels reported in the 25th ATDS were consistent with international data, with the highest concentrations reported in fish and other seafood.

Table 9 International mean concentrations of total mercury in selected foods

ATDS food/beverage	Mean mercury (total) concentration mg/kg (nd=LOR)				
	25th ATDS	23rd ATDS[1]	USA[2]^	EFSA[3]	NZ[4]*
Fish fillets, plain from takeaway	0.13	0.15	0.021	0.10	0.27
Fish portions, frozen	0.048	0.10	0.004	0.10	0.06
Mussels	0.010	-	-	0.041	0.017
Prawns	0.015	0.042	0.006	0.050	-
Tuna, canned in brine	0.046	0.057	0.14	0.10	0.034
Water	0.0001	-	-	-	0.00004

1 FSANZ, 2011a

2 US FDA, 2017^LB values

3 EFSA, 2012c

4 NZ MAF, 2011 *MB values

Dietary exposure

Dietary exposures to mercury (total, inorganic and methyl) are expressed as a range; the lower end of the range (nd=0) represents the situation where analytical results that were listed as 'not detected' are assumed to have a concentration of zero; the upper end of the range (nd=LOR) represents the situation where analytical results that were listed as 'not detected' are assumed to have a concentration equal to the LOR.

At the lower bound (nd=0), mean total dietary exposures were 0.32–1.1 µg/kg bw/week, with P90 dietary exposures being 0.73–2.4 µg/kg bw/week. At the upper bound (nd=LOR), consumer mean total mercury dietary exposures were 1.1–2.7 µg/kg bw/week, with P90 dietary exposures being 1.7–4.1 µg/kg bw/week.

The highest mean and P90 dietary exposures to total mercury, on a µg/kg bw/week basis, were for children aged 2 to 5 years. Teenagers 13 to 18 years had the lowest mean and P90 dietary exposures to total mercury.

For infants aged 9 months, respondent lower bound (nd=0) mean and P90 dietary exposures to total mercury were 0.15 µg/kg bw/week and 0.29 µg/kg bw/week, respectively. At the upper bound (nd=LOR), respondent mean and P90 dietary exposures were 3.6 µg/kg bw/week and 7.1 µg/kg bw/week, respectively.

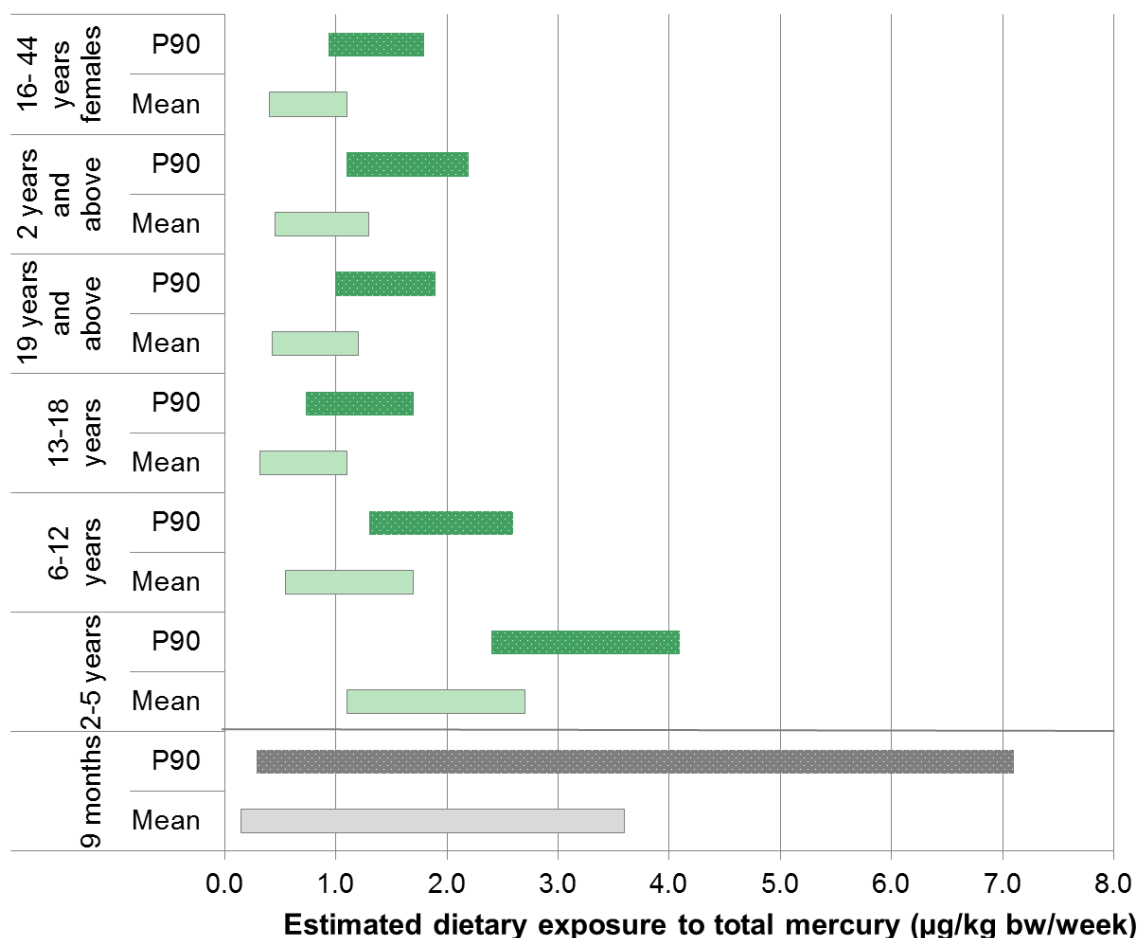Further details are provided in Table A 12.4 in Appendix 12 and in Figure 16.

Figure 16 Estimated dietary exposures to total mercury

> *Notes:*
> - *The lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario.*
> - *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain total mercury or not).*
> - *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain total mercury).*

Major food contributors

The major contributing food category to total mercury exposure was 'meat, poultry, seafood and eggs' (98–100%). 'Takeaway foods and snacks' (<1 to 2%) made a minor contribution. Within the 'meat, poultry, seafood and eggs' category, plain fish (51–71%), crumbed/battered fish and seafood (15–34%) and tuna (all forms) and canned and smoked seafood (11–26%) were the major contributing food groups for all age groups assessed. Crustacea (<1 to 3%) and molluscs (<1%) made a minor contribution to total dietary exposures for all population groups assessed.

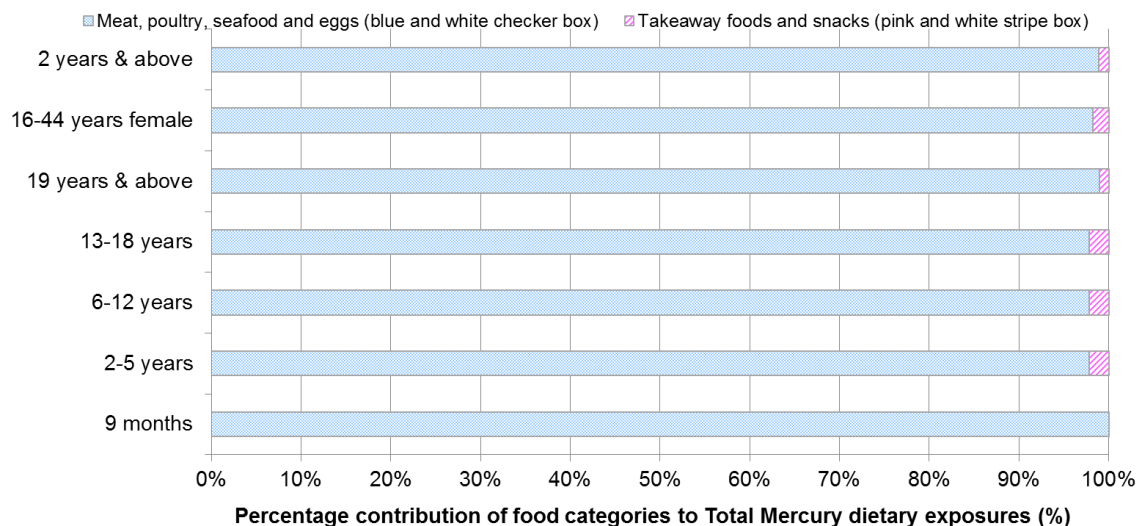For further information, refer to Table A 12.10 in Appendix 12 and Figure 19.

Figure 17 Contributing food categories to estimated total mercury dietary exposures

Inorganic mercury

Hazard identification

The absorption and bioavailability of inorganic mercury varies significantly between the many chemical forms which are ingested. Information from various human volunteer studies found that absorption through the gastrointestinal tract ranged from as low as 0.04% to 5 to 10% depending on the form of mercury and the vehicle used for oral dosing. Despite wide variation in bioavailability, the distribution of inorganic mercury is relatively consistent, with the retained dose accumulating predominantly in the kidneys (JECFA, 2011b).

The 72nd JECFA meeting (2011b) considered the toxicity of inorganic mercury and established a PTWI of 4 µg/kg bw based on a $BMDL_{10}$[6] of 0.06 mg/kg bw/day for relative kidney weight increase in male rats exposed to a five day/week dosing schedule for six months. The PTWI was calculated by adjusting the five day/week dose to an average daily dose and applying a 100-fold uncertainty factor to account for intraspecies and interspecies variation (JECFA, 2011b). FSANZ has used this PTWI for the purposes of the risk assessment for the 25th ATDS, including comparison against estimated dietary exposures.

Analytical results

Inorganic mercury was analysed in a limited number of seafood types with a focus on likely sources of dietary exposure. There were no detections in these foods for the 25th ATDS.

Dietary exposure

Inorganic mercury dietary exposures were estimated using concentration data for selected seafood which were analysed for inorganic mercury (fish, crustacean and molluscs) and total mercury concentrations for all other foods.

At the lower bound (nd=0), mean inorganic mercury dietary exposures for consumers were 0.055–0.19 µg/kg bw/week, with P90 dietary exposures being 0.09–0.32 µg/kg bw/week. At the upper bound (nd=LOR), mean inorganic mercury dietary exposures were 1.0–2.4 µg/kg bw/week, with P90 dietary exposures being 1.5–3.6 µg/kg bw/week.

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[6] Benchmark dose associated with 10% increase in relative kidney weight.

The highest mean and P90 consumer dietary exposures to inorganic mercury, on a µg/kg bw/week basis, were for children aged 2 to 5 years.

For infants aged 9 months, respondent lower bound (nd=0) mean and P90 dietary exposures to inorganic mercury were 0.00003 µg/kg bw/week and 0.000061 µg/kg bw/week, respectively. At the upper bound (nd=LOR), respondent mean and P90 dietary exposures were 3.4 µg/kg bw/week and 6.8 µg/kg bw/week, respectively.

Further details are provided in Table A 12.4 in Appendix 12 and in Figure 20.
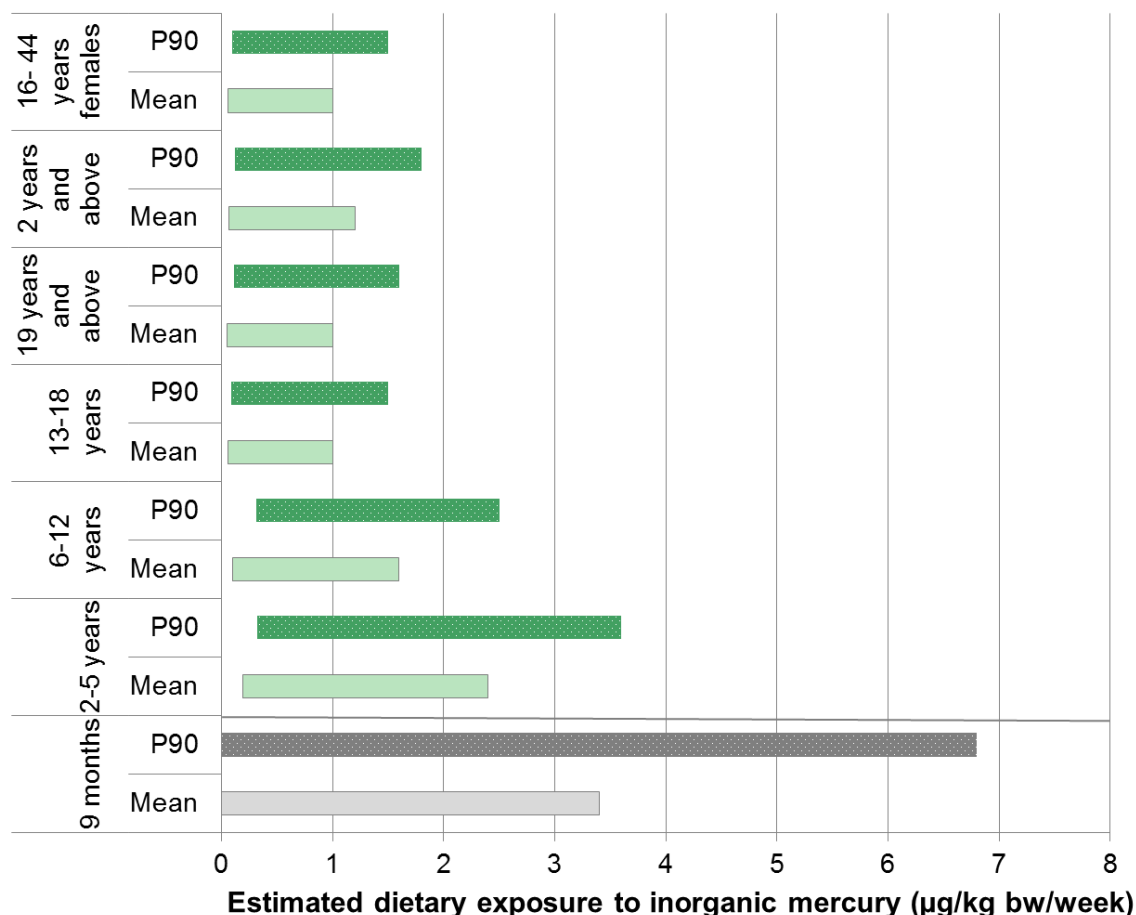

Figure 18 Estimated dietary exposure to inorganic mercury

> *Notes:*
> - *The lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario.*
> - *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant consumed foods that contain inorganic mercury or not).*
> - *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain inorganic mercury).*

Food contributors

The sole contributing food category to inorganic mercury exposures was 'Takeaway foods and snacks', with all of this contribution being from the food group Sushi roll. This was due to this food being the only one with detectable concentrations of total mercury which for this assessment was assumed to be inorganic mercury.

For further information, refer to Table A 12.11 in Appendix 12.

Risk characterisation

JECFA established the current PTWI of 4 µg/kg bw based on effects on kidney weight in rats dosed with inorganic mercury compounds for six months (JECFA 2011b).

Dietary exposures were below the PTWI for all population subgroups assessed except at the high exposure (P90) for 9 month old infants whose exposures ranged between <1 to 170% of the PTWI at the lower and upper bound respectively. This wide range indicates a considerable amount of uncertainty and that non-detects contributed to the upper bound estimates where they are assigned a value equal to the LOR. Estimated dietary exposures for 9 month old infants are based on a model diet, and the P90 exposure is based on a doubling of the mean exposure. The use of dietary survey data of individuals in this age group would be required to assist in refining the exposure estimates and reducing uncertainty.

Overall, these results indicate that dietary exposures to inorganic mercury for most Australian consumers are acceptably low.
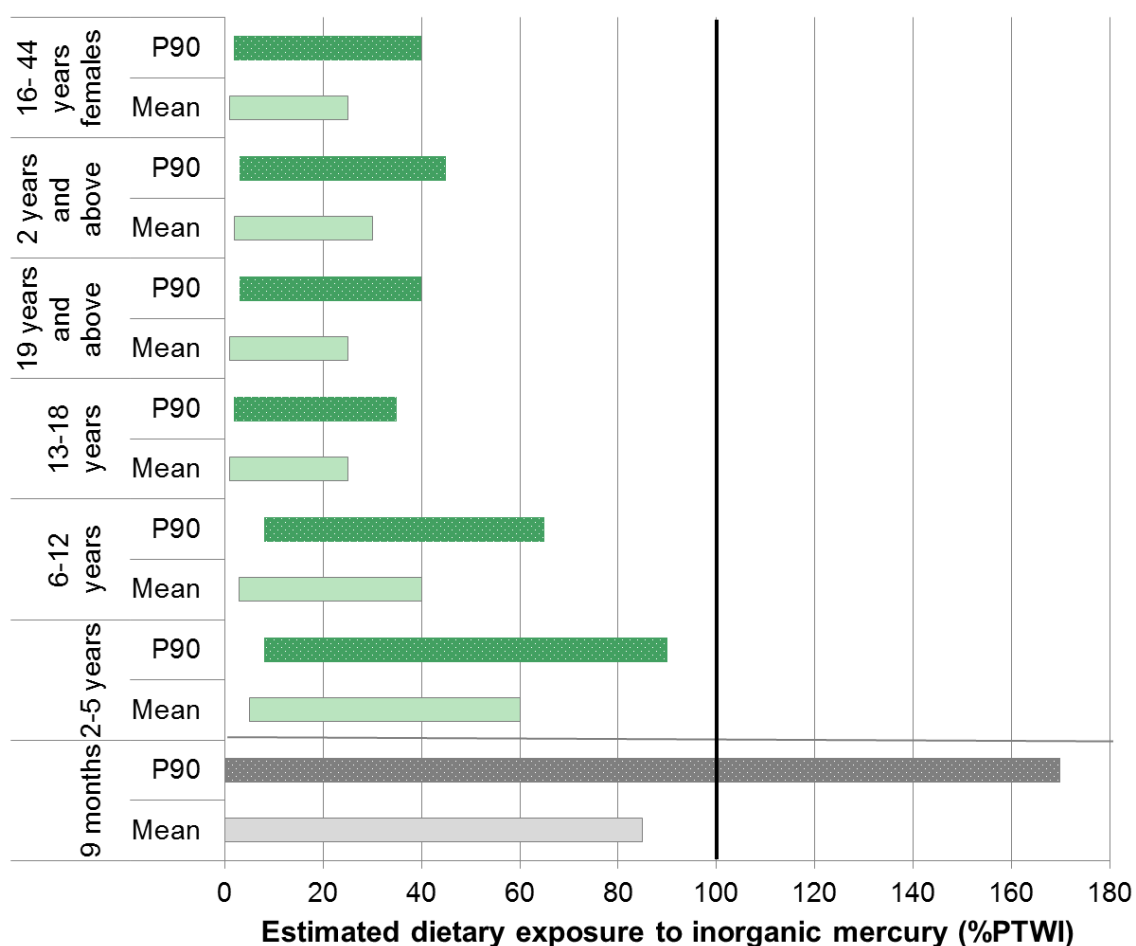

Figure 19 Estimated dietary exposure to inorganic mercury

Notes:
* The lower end of each bar is for the nd=LOR scenario; the upper end of each bar is for the nd=0 scenario
* Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain inorganic mercury or not).
* Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain inorganic mercury).

Methylmercury

Hazard identification

Methylmercury is readily absorbed from the gastrointestinal tract and can cross the blood-brain barrier and placenta, with the potential to concentrate at higher levels in the fetus compared to the mother (JECFA, 2007). It is considered that developmental neurotoxicity of the embryo and fetus is the most sensitive toxicological endpoint in humans. On this basis, the 67th JECFA meeting (2007) reconfirmed the PTWI of 1.6 μg/kg/body weight, based on a dose-response analysis of epidemiological studies, which established a maternal NOAEL of 1.5 μg/kg body weight/day for adverse neurodevelopmental effects in children (including decreased IQ and impaired performance in various neurobehavioral and cognitive tests). The PTWI was calculated by applying an uncertainty factor of 6.4 to the NOAEL to account for inter-human variation in converting maternal blood concentration to long term dietary intake (JECFA, 2007). FSANZ has used this PTWI for the purposes of risk assessment including comparison against estimated dietary exposures.

Analytical results

Methylmercury was analysed in a limited number of seafood types, with a focus on likely sources of dietary exposure. It was detected in three foods, with mean concentrations (nd=LOR) in takeaway fish fillets (0.14 mg/kg), frozen fish portions (0.06 mg/kg) and canned tuna (0.05 mg/kg). Methylmercury was not detected in mussels and prawns. These results are consistent with the known properties of methylmercury including propensity to bioaccumulate at the higher end of the marine food chain.

Dietary exposure

At the lower bound (nd=0), mean methylmercury dietary exposures were 0.55–1.7 μg/kg bw/week, with P90 dietary exposures of 1.2–3.5 μg/kg bw/week. At the upper bound (nd=LOR), mean organic mercury dietary exposures were 0.38–1.2 μg/kg bw/week, with P90 dietary exposures of 0.8–2.4 μg/kg bw/week. The lower bound (nd=0) estimated dietary exposures to methylmercury were higher than those for the upper bound (nd=LOR) scenario due to the different number of consumers between the two scenarios.

For females aged 16 to 44 years, who were used as a proxy group for pregnant women, mean lower bound (nd=0) dietary exposures were 0.61 μg/kg bw/week, with P90 dietary exposures of 1.4 μg/kg bw/week. At the upper bound (nd=LOR) consumer mean dietary exposures were 0.43 μg/kg bw/week, with P90 exposures being 0.87 μg/kg bw/week. The highest mean and P90 consumer dietary exposures to methylmercury, on a μg/kg bw/week basis, were for children aged 2 to 5 years.

For infants aged 9 months, respondent lower bound (nd=0) mean and P90 dietary exposures to methylmercury were 0.089 μg/kg bw/week and 0.18 μg/kg bw/week, respectively. At the upper bound (nd=LOR), respondent mean and P90 dietary exposures were 0.14 μg/kg bw/week and 0.29 μg/kg bw/week, respectively.

Further details are provided in Table A 12.4 in Appendix 12 and in Figure 22.
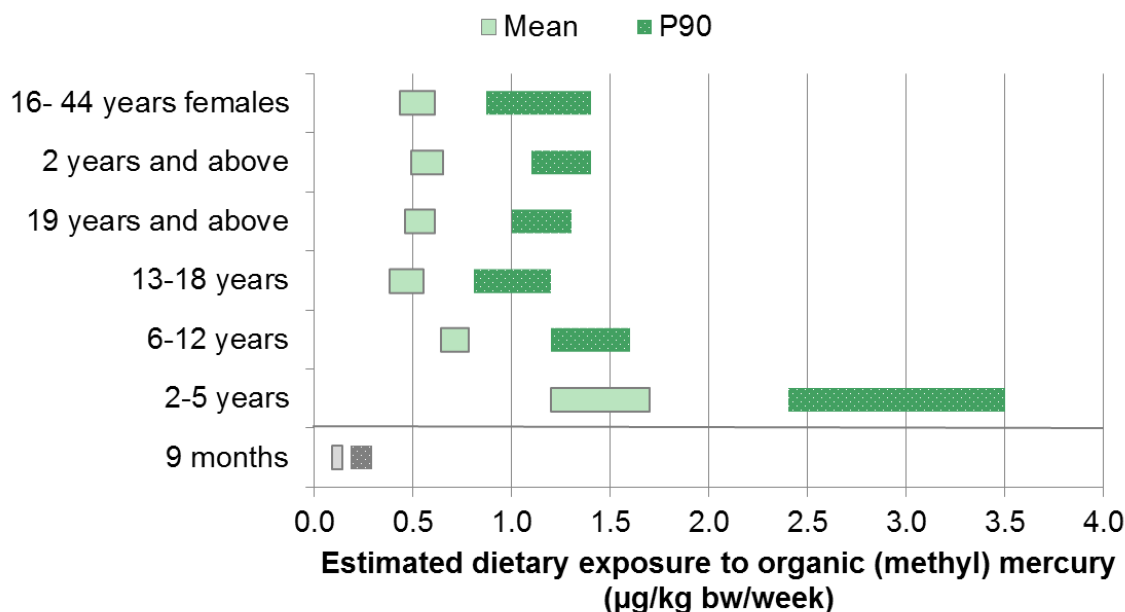
Figure 20 Estimated dietary exposures to methylmercury

Notes:
- *For infants aged 9 months, the lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario. For all other age groups, the lower end of each bar is for the nd=LOR scenario; the upper end of each bar is for the nd=0 scenario.*
- *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain methylmercury or not).*
- *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain methylmercury).*

Food contributors

The only contributing food category to methylmercury exposures was 'Meat, poultry, seafood and eggs', with Plain fish being the only contributing food group for all age groups assessed.

For further information, refer to Table A 12.12 in Appendix 12.

Risk characterisation

Methylmercury is a developmental neurotoxin, and the most sensitive developmental stage is the fetus. Exposure to methylmercury exposure by pregnant women is therefore of greatest concern to human health. The PTWI established by JECFA is 1.6 µg/kg bw, based on epidemiological evidence of adverse effects on neurological development in children following prenatal exposure.

Estimated dietary exposures to methylmercury for women of childbearing age and all other population subgroups assessed, except children 2–5 years, did not exceed the PTWI. The exceedances for children aged 2 to 5 years were up to 110% and 220% of the PTWI for mean and P90 consumers, respectively. Further details on the comparison of dietary exposures with the PTWI can be found in Table A 12.4 in Appendix 12 and in Figure 23.

JECFA (2007) concluded that there was no clear evidence that prenatal vulnerability extends into postnatal exposure, however the sensitivity of 2–5 year olds to adverse effects of methylmercury is not clearly defined. This uncertainty must be weighed against the potential benefits of moderate fish consumption including omega-3 fatty acid intake, which is understood to have beneficial effects on brain and eye development in children. Fish also

contains high quality protein, iodine and vitamin D, which are important for their growth and development needs.

Overall, these results indicate that dietary exposures to inorganic mercury for most Australian consumers are acceptably low. Exceedances of the PTWI for certain subpopulations should be considered in the context of the health benefits of fish consumption in accordance with the FSANZ Mercury in Fish: Advice on fish consumption (FSANZ, 2004a; FSANZ, 2004b).
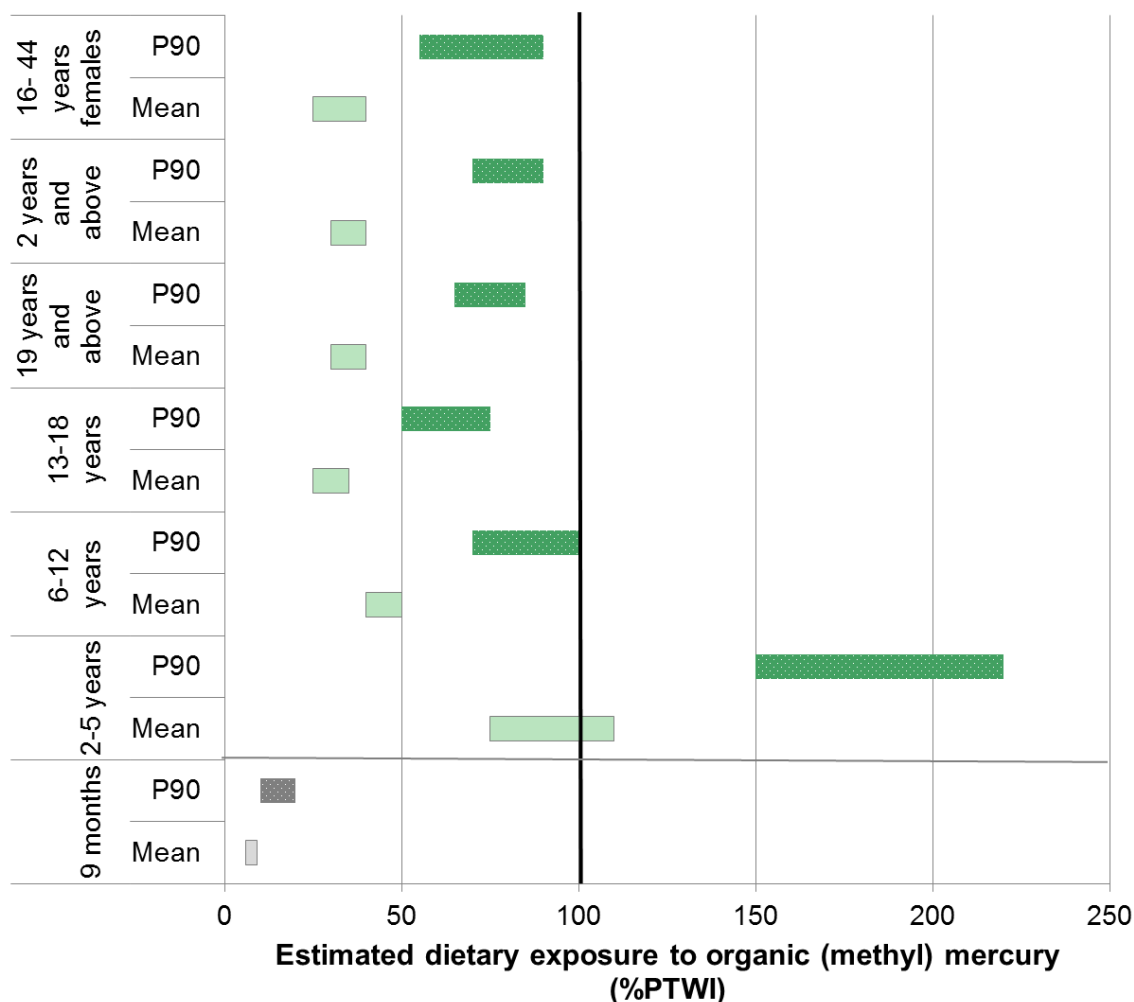

Figure 21 Estimated dietary exposure to methylmercury

Notes:
- *For infants aged 9 months, the lower end of each bar is for the nd=0 scenario; the upper end of each bar is for the nd=LOR scenario. For all other age groups, the lower end of each bar is for the nd=LOR scenario; the upper end of each bar is for the nd=0 scenario.*
- *Dietary exposures for infants aged 9 months is for all respondents (i.e. exposures irrespective of whether a survey participant was consumed foods that contain methylmercury or not).*
- *Dietary exposures for all age groups 2 years and above are for consumers only (i.e. exposures only for those survey participants who consumed foods that contain methylmercury).*

Risk management

Risk management measures are already in place for mercury via MLs in the Code. No exceedance of the current mercury MLs were found for foods sampled for the 25th ATDS, suggesting that levels of methylmercury are ALARA.

The potential for exceedances of the PTWI result from the consumption of fish, which is known to be a significant source of dietary exposure, especially in relation to long living,

predatory species. Results indicate that dietary exposure for Australian consumers is consistent with levels found to be of concern internationally. As such, FSANZ will continue to participate in international assessment and standards-setting work through Codex. These activities, in combination with the current FSANZ assessment work on methylmercury will inform the need for revised regulatory measures. It is noted that FSANZ has published consumer advice to limit exposure to methylmercury through fish consumption. This will continue to be updated as required to reflect future FSANZ work on the issue (FSANZ, 2017c).

Risk management measures for contaminants

Legislative requirements in Australia and New Zealand, including state and territory food Acts, aim to keep food safe and suitable. State and territory food Acts and the New Zealand Food Act contain general provisions that make it an offence to sell food that is unsafe, and food businesses must comply with requirements in the Code.

The Code contains food standards that have been developed, approved and gazetted by FSANZ. The Code applies to all food sold or prepared for sale in Australia and New Zealand (except where specified 'Australia or New Zealand only'). In accordance with state, territory and New Zealand food legislation, it is an offence to supply food that does not comply with the Code.

In March 1999, FSANZ reviewed the provisions for Maximum Permitted Concentrations (MPCs[7]) of metal contaminants in food in order to develop Standard 1.4.1. The approach agreed to by the then Australia New Zealand Ministerial Council[8] was that MLs would be set in the following circumstances:

- only for those contaminants that present a significant risk to public health and safety
- only for those foods that significantly contribute to the dietary exposure of the contaminant
- to ensure that levels are as low as reasonably achievable
- consistent with Codex levels, where possible, however, harmonisation with Codex is secondary to measures put in place to protect the public health and safety of Australians and New Zealanders.

These principles underpin the MLs that are currently in the Code.

There is no indication to date that the Code needs to be revised to amend the current MLs.

Decisions on the acceptability of an MOE are made on a case-by-case basis depending on the level of public health protection needed or desired and the extent and nature of the population of people being exposed.

Although FSANZ has not established that regulatory measures are required to increase the MOEs calculated for arsenic and lead, or the marginal exceedances of HBGVs for cadmium and mercury, a number of non-regulatory measures will assist with keeping exposure ALARA as shown in Table 10.

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[7] Now known as maximum limits (MLs)
[8] Now known as the Australia and New Zealand Ministerial Forum on Food Regulation

Table 10 Non-regulatory measures for contaminants for the 25th ATDS

Contaminant	Public health issue to address	Proposed measures
Arsenic	Withdrawal of HBGV by JECFA Insufficient data to characterise risk for Australian consumers.	Monitoring other risk management measures being employed in other countries via the International Food Chemical Safety Liaison Group (IFCSLG) and considering their suitability for Australia and New Zealand. FSANZ encourages use of the Code of practice for the prevention and reduction of arsenic contamination in rice (CL 2017/25-CF). FSANZ to consider whether any other risk management measures are needed to reduce arsenic exposure following consideration of the results of future planned survey work for arsenic in rice.
Cadmium	No specific public health and safety concerns. Levels in food and dietary exposure should be ALARA.	Monitoring other risk management measures being employed in other countries via the IFCSLG and considering their suitability for Australia and New Zealand.
Lead	Withdrawal of HBGV by JECFA No specific public health and safety concerns. Levels in food and dietary exposure should be ALARA.	FSANZ to consider the development of new standards for lead by Codex and consider if current MLs in the Code need revising in order to reduce exposure to ALARA.
Inorganic mercury	No specific public health and safety concerns. Levels in food and dietary exposure should be ALARA.	As the temporary exceedance in a small group of infants is not considered a public health and safety concern, no risk additional management measures are needed at this stage.
Methylmercury	For children aged 2-5 years, estimated P90 dietary exposures to methylmercury >HBGV (up to 220%).	FSANZ to consider the development of new standards for methylmercury by Codex and consider if current MLs in the Code need revising in order to reduce exposure to ALARA. FSANZ to update consumer advice for mercury in fish as required, via the separate review project for methylmercury being undertaken by FSANZ.

Part E–Conclusions and recommendations

The 25th ATDS confirms the current safety of the Australian food supply for the general population in terms of the levels of agricultural and veterinary chemicals and selected metal contaminants.

Agricultural and veterinary chemicals

A significant proportion of the 88 different food types foods sampled contained no detectable residues of the 226 agricultural and veterinary chemicals surveyed. Estimated dietary exposures for the general population aged 9-months and over were below the ADIs for all but one agricultural chemical. Estimated mean dietary exposure to the organophosphorus insecticide prothiofos, exceeded the ADI for 2 to 5 and 6 to 12 year olds, and the 90th percentile exposure for all age groups above 2 years. FSANZ briefed the APVMA on these results, who consulted with the product registrant resulting in the voluntary cancellation of label approvals for the use of prothiofos on grapes.

Metal contaminants

The levels of arsenic, cadmium, lead and mercury found in composite food samples in the 25th ATDS were all below corresponding commodity based MLs in the Code. These levels and estimated dietary exposure for the Australian population were also consistent with those reported in international literature.

Foods were measured for total and inorganic arsenic content. Inorganic arsenic is considered a genotoxic carcinogen and international bodies have been unable to establish a safe level of exposure. FSANZ calculated inorganic arsenic dietary exposure with limited analytical data using highly conservative methods and dietary exposures were below levels associated with adverse effects in human populations. However, FSANZ considers that that there is insufficient data to fully characterise the risk for Australian consumers. The major contributors of arsenic to the Australian diet are seafood and rice.

Cadmium is found at low levels in a broad range of foods. Seafood, and to a lesser extent, cereals and starchy root vegetables, are major contributors to the Australian diet. FSANZ estimated dietary exposures to cadmium of Australian consumers to be within the range of, and generally lower than, international estimates. A slight exceedance of the relevant HBGV for high consuming infants aged 9 months is not considered to be a significant public health and safety concern due to the highly conservative nature of the assessment.

Dietary exposure assessments were performed for inorganic and methylmercury. As the mean and 90th percentile exposure for studied population subgroups were all considerably below the HBGV, except for high consuming (90th percentile exposures) 9 month old infants, it can be concluded that there are no health concerns for Australian consumers with respect to dietary exposure to inorganic mercury. International estimates also support this conclusion, indicating that dietary exposure of the Australian population is within the range seen internationally. With respect to methylmercury, estimated dietary exposures were below the relevant HBGV for all age groups except mean and 90th percentile consumers aged 2 – 5 years. It is important to note that omega 3 fatty acids are present in fish and are essential for normal development of rapidly growing brain and eyes. Importantly, in the most sensitive age group–women of child bearing age–estimated exposures were below the HBGV. FSANZ has published consumer advice to limit mercury exposure through fish consumption which will continue to be reviewed and updated to reflect future work in the area.

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