# FOOD
## STANDARDS
Australia New Zealand

## The 23rd Australian Total Diet Study

# Foreword

Food Standards Australia New Zealand (FSANZ) is an independent Australian Government agency responsible for ensuring a safe food supply, protecting and supporting the health of people in Australia and New Zealand. FSANZ is responsible for developing food standards, which includes establishing limits for the levels of chemicals in foods where appropriate.

In order to determine the level of chemicals in food, FSANZ conducts a number of food surveys, gathering analytical data and estimating 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.

The first ATDS, formerly known as the 'Market Basket Survey', was conducted in 1970 by the National Health and Medical Research Council. Since then, the Australian Government has conducted regular ATDS estimating consumer exposure to chemicals in the food supply, with the last seven studies managed by FSANZ (formerly the Australia New Zealand Food Authority). The first twenty studies examined dietary exposure to pesticide residues and contaminants, with each study concluding that the Australian food supply is safe for consumers.

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

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 23<sup>rd</sup> Australian Total Diet Study as part of FSANZ's commitment to ongoing monitoring of the Australian food supply, ensuring it continues to be one of the safest food supplies in the world.

Ms Philippa Smith AM
CHAIRMAN

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

## Acknowledgements

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FSANZ would also like to thank Dr Gerald Moy (formerly of the World Health Organization, Geneva and an expert in total diet studies) for his valuable assistance in peer-reviewing this study.

# Abbreviations

| | |
|---|---|
| ADI | Acceptable Daily Intake |
| AES | Atomic Emission Spectroscopy |
| AI | Adequate Intake |
| ALARA | As Low As Reasonably Achievable |
| APVMA | Australian Pesticides and Veterinary Medicines Authority |
| ATDS | Australian Total Diet Study |
| BMD | Benchmark Dose |
| BMDL | Benchmark Dose Lower Confidence Limit |
| bw | body weight |
| CCCF | Codex Committee on Contaminants in Foods |
| DIAMOND | Dietary Modelling of Nutritional Data – FSANZ's Dietary Modelling computer program |
| EAR | Estimated Average Requirement |
| ECD | Electron Capture Detector |
| FAO | Food and Agriculture Organization of the United Nations |
| FSANZ | Food Standards Australia New Zealand |
| GC-MS | Gas Chromatography – Mass Spectrometry |
| HPLC | High Performance Liquid Chromatography |
| ICP | Inductively Coupled Plasma |
| kg | kilograms |
| JECFA | Joint FAO/WHO Expert Committee on Food Additives |
| LC | Liquid Chromatography |
| LOAEL | Lowest Observed Adverse Effect Level |
| LOD | Limit of Detection |
| LOQ | Limit of Quantification |
| LOR | Limit of Reporting |
| mg | milligram (one thousandth of a gram) |

| mg/kg | milligrams per kilogram |
| ML | Maximum Limit |
| MOE | Margin of Exposure |
| MRL | Maximum Residue Limit |
| MS | Mass Spectrometry |
| NHMRC | National Health and Medical Research Council |
| NNS | National Nutrition Survey |
| NOAEL | No Observed Adverse Effect Level |
| NPD | Nitrogen-Phosphorus Detector |
| NRV | Nutrient Reference Value |
| NZ MOH | New Zealand Ministry of Health |
| PFPD | Pulse Flame Photometric Detector |
| PMTDI | Provisional Maximum Tolerable Daily Intake |
| PTDI | Provisional Tolerable Daily Intake |
| PTMI | Provisional Tolerable Monthly Intake |
| PTWI | Provisional Tolerable Weekly Intake |
| RDI | Recommended Dietary Intake |
| µg | Microgram (one millionth of a gram) |
| UL | Upper Level of Intake |
| WHO | World Health Organization |

Note: Definitions for some of these abbreviations can be found in Appendix 1.

# 23<sup>rd</sup> ATDS Key Findings

**Key Findings from this study:**

*Overview*

- The current ATDS investigated agricultural and veterinary chemicals, contaminants, nutrients and selected mycotoxins in a range of foods and beverages.

- The current study has reaffirmed the Australian food supply as safe for consumers.

- FSANZ has identified a number of areas for further work in the future.

*Agricultural and veterinary chemicals:*

- Estimated dietary exposures for agricultural and veterinary chemicals were all below relevant reference health standards, which is consistent with the findings from previous studies.

- Some agricultural and veterinary chemicals were detected that are not approved for use in Australia.

- The level of some chemicals that are approved for use exceeded the maximum residue limits (MRLs) established in the Australia New Zealand Food Standards Code (the Code).

- Although FSANZ identified no safety risks from exceeding the MRLs, a notice of exceedances or possibly non-approved use has been provided to the relevant state or territory for follow up action.

*Mycotoxins (toxins produced by fungi):*

- Aflatoxins (B1, B2, G1, G2 and M1), deoxynivalenol, fumonisins (B1 and B2), ochratoxin A, patulin and zearalenone, were not detected in any foods analysed.

*Contaminants:*

- For all contaminants, estimated dietary exposures were below the relevant reference health standards for all population groups at both the mean and 90th percentile consumption levels (high consumers).

*Nutrients:*

- The ATDS only provides a general indication of nutrient intake amongst the Australian population. These indicators will inform further studies such as national nutrition surveys that will investigate and further define nutrient adequacy.

- **Molybdenum and selenium**

  — The prevalence of intakes of molybdenum and selenium below the Estimated Average Requirement (EAR) was low.

- **Copper, fluoride, selenium & zinc**

  — A proportion of children aged 2-3 years exceeded the Upper Level of Intake (UL) for these nutrients. Given the ULs for children are set on a highly conservative basis, these findings are not considered to pose a human health and safety risk.

  — 9 month old infants may also exceed the UL for these nutrients, excluding copper, at the 95th percentile (high consumers) of intake. Assessment of nutrient intakes for 9 month old infants is theoretical and based on extrapolations from a model diet. As such, any conclusions around nutrient intake for this population group should take this into account.

*Conclusion:*

- The 23rd ATDS confirms the current safety of the Australian food supply in relation to the levels of agricultural and veterinary chemicals, contaminants, selected mycotoxins and nutrients.

# Executive Summary

The 23rd ATDS examined dietary exposure for the Australian population to 214 agricultural and veterinary chemicals, 9 contaminants 12 mycotoxins, and 11 nutrients found in 92 foods and beverages. Foods and beverages commonly consumed in the Australian diet were sampled during January/February and June/July 2008. The dietary exposure was estimated by determining the concentration of the substance in 92 foods and beverages multiplied by the amount of food consumed by various age and gender groups, as reported in the two most recent Australian national nutrition surveys (NNS). The dietary exposure to agricultural and veterinary chemicals, contaminants and nutrients was assessed against available reference health standards to determine any potential human health and safety risks. Where there were no Australian health standards, internationally accepted reference health standards or Margins of Exposure (MOE) were used.

The estimated dietary exposures to agricultural and veterinary chemical residues were all below the relevant reference health standards. This is consistent with the findings from previous ATDS. In addition, there were no detections of mycotoxins in any of the foods analysed.

For contaminants, estimated dietary exposures were below the relevant health standards for all population groups at both the mean and 90th percentile consumption levels (high consumers).

For nutrients assessed in this study with EARs, molybdenum and selenium, dietary intakes were considered adequate. In the case of nutrients with Adequate Intakes (AIs), mean estimated dietary intakes for most age-gender groups were similar to, or exceeded their respective AIs. In order to make an accurate assessment of nutrient adequacy amongst the Australian population, larger scale and more comprehensive surveys such as NNSs, which include biological measurements, are more appropriate for this purpose in comparison to the ATDS.

In relation to nutrients assessed with established ULs, some age-gender groups, particularly young children, exceeded these values at varying magnitudes, specifically for copper, fluoride, selenium and zinc. Given that both the ULs for children and the theoretical intake estimates for infants are highly conservative; these exceedances are not considered to pose a human health and safety risk.

In relation to fluoride, the current UL values provide a theoretical estimate of fluoride intake which is not based on actual food consumption data. The apparent discordance between the theoretical and actual intakes without an increase in the adverse clinical sign of moderate dental fluorosis suggests that the existing UL may need to be reconsidered.

Due to the absence of established ULs in Australia for chromium, cobalt, manganese and potassium, conclusions around excessive intakes for the Australian population could not be drawn.

FSANZ recommends that future ATDS continue to monitor levels of agricultural and veterinary chemicals to ensure that regulatory measures for these chemicals in food continue to provide adequate protection of the Australian population.

Continued monitoring of contaminants in foods for which maximum levels have been established, and are likely to be major contributors to dietary exposure, should also be undertaken.

Nutrients will be investigated in future ATDS, particularly those for which fortification and addition to food and water has been mandated or permitted (e.g. iron, calcium and fluoride).

FSANZ will also continue to monitor the international developments relating to contaminants in food and use this to inform the selection of chemicals and foods for future ATDS.

# Part A – Background

The purpose of the ATDS is to estimate dietary exposure (or 'intake' when referring to nutrients) of the Australian population to a variety of agricultural and veterinary chemicals, contaminants, nutrients and food additives from food. Dietary exposure to these substances is estimated by determining the analytical concentration in food samples and multiplying the concentration by the amount of those foods consumed by various age and gender groups. In the past, the ATDS has focused on estimating dietary exposure to agricultural and veterinary chemicals and contaminants for the Australian population. In more recent studies, the scope of the ATDS has changed to include the assessment of dietary exposure to a broader range of substances, including food additives (FSANZ, 2005) and nutrients (FSANZ, 2008).

## Origin of the study

In 1969, the National Health and Research Council (NHMRC) recommended that a 'market basket' survey be conducted to analyse the concentrations of agricultural and veterinary chemicals and metal contaminants in foods that represent a significant part of the Australian diet. Accordingly, the first ATDS was commissioned by the NHMRC in 1970. A further 15 studies were conducted by the NHMRC until the predecessor of FSANZ, the National Food Authority, gained responsibility in 1991. The 23ʳᵈ ATDS is the eighth study to be conducted by FSANZ and its predecessor.

Originally, the ATDS was conducted every two years, focussing on the assessment of dietary exposure to a range of agricultural and veterinary chemicals and contaminants. However, these studies consistently indicated that dietary exposure to these chemicals for the Australian population were well below relevant health standards, and therefore, did not present a risk to public health and safety. Due to these consistent findings, in 2003, the scope of the ATDS was broadened to encompass a wider range of food chemicals, including food additives and nutrients. The 21ˢᵗ ATDS, which examined dietary exposure to the food additives benzoates, sulphites and sorbates, was the first study conducted under the new format (FSANZ, 2005). The 22ⁿᵈ ATDS continued to expand the approach, assessing the dietary intake of a number of nutrients in the Australian population (FSANZ, 2008). The expansion of the ATDS has provided greater flexibility; focusing these studies on a wider range of substances to assess a variety of public health and safety issues.

Since the 22nd iteration of this study, the ATDS is part of the Food Regulation Standing Committee's (FRSC)[1] Implementation Sub-Committee's[2] (ISC) coordinated food survey plan (the Plan). The aim of the Plan is to identify efficiencies and foster collaboration in the planning, implementation, co-ordination and consistent management of food surveys. The ATDS is now a national survey activity on the Plan and is conducted with the participation of all states and territories in Australia.

## Using information from the study

The revised scope of the ATDS has provided greater flexibility to target a wider range of substances to inform the food standards setting process. This data, in conjunction with information from other sources, provides a valuable evidence base to draw upon when developing, renewing or amending relevant food standards. The results of the study also provide important public health information on a national level and are a source of information for Australia's contribution to the World Health Organization (WHO) Global Environmental Monitoring System (GEMS) Food Contamination Monitoring and Assessment Program, the Joint WHO/FAO Expert Committee on Food Additives (JECFA), the Codex Committee on Food Additives (CCFA), the Codex Committee on Contaminants in Food (CCCF), the Joint FAO/WHO Meeting on Pesticide Residues (JMPR) and independent researchers in both government and non-government agencies.

## Chemicals assessed in this study

The 23rd ATDS primarily returned to a more traditional approach examining a range of agricultural and veterinary chemicals and contaminants. It also included a number of nutrients for investigation, producing a hybrid-style ATDS.

## Agricultural and Veterinary Chemicals

Agricultural and veterinary chemicals are used for the control of unwanted insects, mites, fungi, rodents, weeds, nematodes and other pests, and for the control of diseases in farm animals and crops. The use of these chemicals has been common practice in world agriculture for many years, and provides greater availability and improved quality and variety in our food supply.

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[1]   FRSC is responsible for the coordination and provision of policy advice to the Australia and New Zealand Food Regulation Ministerial Council in relation to food regulation.

[2]   ISC is a sub-committee of FRSC and is responsible for overseeing a consistent approach to the implementation and enforcement of food regulations and standards across jurisdictions.

Although the use of agricultural and veterinary chemicals presents significant benefits, there may be risks associated with their use if Good Agricultural Practices (GAP) are not followed by industry to minimise levels of residues in foods. In order to ensure safe use, the Australian Pesticide and Veterinary Medicines Authority (APVMA) set maximum residue limits (MRLs) after assessing the various safety aspects of the specific agricultural and veterinary chemical prior to approval for use in Australia. The MRL is the highest concentration of an agricultural and veterinary chemical residue that is legally permitted or accepted in a food or animal feed. The MRL does not indicate the amount of chemical that is always present in a treated food but it does indicate the highest residue that could result from the registered conditions of use.

FSANZ works with the APVMA to list MRLs in the *Australia New Zealand Food Standards Code* (the Code). As part of the assessment, FSANZ considers the dietary exposure of consumers to agricultural and veterinary chemical residues from all foods in the diet by comparing the dietary exposure with relevant health standards. FSANZ must be satisfied that the residues of these chemicals do not present a risk to public health and safety before their inclusion in the Code. The MRLs listed in the Code are monitored and enforced by state and territory agriculture or food agencies.

The 23ʳᵈ ATDS examined a range of agricultural and veterinary chemicals in a variety of foods.  The range of agricultural and veterinary chemicals tested included:

- chlorinated organic pesticides
- organophosphorus pesticides
- carbamate pesticides
- synthetic pyrethroid pesticides
- herbicides
- fungicides

*Chlorinated organic pesticides*

Chlorinated organic pesticides, in general, are highly stable compounds that are not susceptible to chemical and biological degradation (Hanberg, 1996). As a result, these compounds persist in the environment, particularly in soil, with human exposure to these chemicals through food.

Chlorinated organic pesticides are also fat soluble and can accumulate in the fatty tissue of humans and animals (Hanberg, 1996). The use of chlorinated organic pesticides in developed countries has been heavily restricted since it was shown that some of these compounds were health and environmental hazards.

*Organophosphorus pesticides*

Organophosphorus pesticides are a diverse group of insecticides, used for a variety of agricultural purposes. Organophosphorus pesticides generally degrade rapidly and do not concentrate in the food chain (Gan *et al*., 2010).

Organophosphorus pesticides act on the central nervous system of insects, animals and humans. They act by inhibiting the enzyme acetylcholinesterase which metabolises acetylcholine, a chemical essential for nerve function (Mileson *et al*., 1998).

*Carbamate pesticides*

Similar to organophosphorus pesticides, carbamate pesticides are mostly biodegradable, and therefore, do not accumulate in the food chain like chlorinated organic pesticides. They also act on the central nervous system of humans and animals, but generally do not accumulate in the human body due to degradation in the liver (Fishel, 2008a).

*Synthetic pyrethroid pesticides*

Synthetic pyrethroid pesticides are insecticides, which have a similar chemical structure to the naturally occurring pyrethrins found in the flowers of some chrysanthemum species. Synthetic pyrethroids act rapidly on the nervous system of insects, but have relatively low mammalian toxicity (Fishel, 2008b). They are generally biodegradable and as such, tend not to persist in the environment.

*Herbicides*

Herbicides are used to protect agricultural crops by killing or controlling the growth of weeds. Herbicides are broken down in the environment by both chemical and microbial degradation, but are relatively stable once they enter the subsoil (DPI VIC, 2010). The majority of synthetic herbicides present low acute toxicity to animals and humans as they specifically target particular metabolic pathways within plants (Hamilton and Crossley, 2004).

*Fungicides*

Fungicides are applied to plants and are used to treat animals to control diseases caused by fungi, including moulds and yeasts. Residues of fungicides in food most often arise from their post-harvest use (De Waard *et al*., 1993; QLD DEEDI, 2005). Fungicides are considered to present low acute toxicity to humans (Penn State, 2009).

## Contaminants

Contaminants, including metals and non-metals, are often present in foods in trace amounts. Contaminants can enter the food supply through environmental elements, such as air, soil and water, or during food processing and cooking. The Code stipulates maximum limits (MLs) for specific contaminants in foods that may contribute significantly to the dietary exposure to the contaminant. MLs are set consistent with good food handling practices and protecting public health and safety.

A variety of contaminants have been analysed in this study primarily for the purposes of estimating public health and safety risk, however some contaminants such as strontium and vanadium have also been analysed to inform the FSANZ Food Composition program and enhance our evidence base for these contaminants.

*Aluminium*

Aluminium is the most abundant metallic element comprising about 8% of the earth's crust (WHO, 1997). In nature, aluminium exists exclusively in compounds with other elements.  As a metal, it is widely used in industries, including aircraft, ships, motor vehicles, construction and electrical applications. It is also widely used in the packaging of food and beverages, food processing equipment, cookware and kitchen utensils.  Other uses of aluminium include antacid medicines, jewellery and antiperspirant products.

The major sources of dietary exposure to aluminium are through food grown in soil with naturally occurring aluminium and from aluminium containing food additives. Exposure may also occur via transfer of aluminium from cooking implements as well as containers, such as aluminium drink cans. Drinking water may also contain aluminium, both naturally occurring and as a result of treatment with aluminium containing chemicals.

*Arsenic*

Arsenic is found naturally in both organic and inorganic forms. Inorganic arsenic is the more toxic and is considered carcinogenic in humans (WHO, 2010b). The major use of arsenic is in agricultural and veterinary medicines, and industrial applications (WHO, 2001b). Inorganic arsenic is registered for use in timber preservatives and to control termites. There are no registered uses of inorganic arsenic in food crops or for animal production.

Most foods generally contain low concentrations of inorganic arsenic due to its wide distribution in the environment. Dietary arsenic, particularly from seafood, represents the major source of arsenic exposure for the majority of the population (Borak and Hosgood, 2007).

*Cadmium*

Cadmium is a metallic element that occurs naturally at low concentrations in the environment, but at high concentrations in volcanic soils (WHO, 1992). Additional cadmium has been added to the environment through industrial processes such as cadmium metal production. Cadmium has also been added to agricultural soils through the use of phosphate fertilisers (WHO, 1992), and certain organic fertilisers based on manures.

Food represents the major source of cadmium exposure, although tobacco smoking adds significantly to the body's burden (WHO, 1992).

*Lead*

Lead is a toxic metal found widely in the environment (Tong *et al*., 2000). It is used for a number of industrial and domestic purposes, such as batteries and lead-based paints (JECFA, 2000). A significant source of exposure to lead is the diet (JECFA, 2000) as lead can contaminate food during processing. Canned foods used to be a source of lead if lead solder has been used in the seam; however, all cans now used in Australia have welded seams.

*Mercury*

Mercury occurs naturally in the environment and can be found in various forms: elemental, organic and inorganic forms (U.S EPA, 2010). The form of most concern to humans is organic, the most common being methylmercury. Methylmercury is largely produced from the methylation of inorganic mercury by microbial activity (WHO, 2000). Marine and freshwater organisms bioaccumulate methylmercury in the food chain so that large predatory fish commonly have the highest levels.

In general, the diet is the major source of exposure to methylmercury, with seafood containing much higher concentrations of mercury than other foods (Jarup, 2003). Most other foods contain very low concentrations of mercury, which is almost entirely in the inorganic form.

*Strontium*

Strontium occurs naturally in the earth's crust (WHO, 2010a). Elemental strontium reacts quickly with water and oxygen, and so it is only found in nature as a compound. Strontium is used in the manufacture of ceramics and glass products, in particular the glass used in television tubes (WHO, 2010a). Strontium compounds are also used in the manufacture of pyrotechnics, paint pigments and medicines.

For adults without occupational strontium exposure, the primary exposure sources are food and drinking water. The contribution from air is insignificant by comparison (WHO, 2010a).

*Vanadium*

Vanadium is a metallic element that occurs widely in the environment. The most abundant source of vanadium in the environment is from the combustion of oil and coal, in which vanadium pentoxide is produced (WHO, 2001a). Vanadium pentoxide is used in the manufacture of alloys, pigments and inks (WHO, 2001a).

For adults without occupational exposure to vanadium pentoxide, the main sources of exposure are air inhalation and the consumption of contaminated food and water (CECBP, 2008).

## Nutrients

Nutrients are naturally present in foods and are essential for numerous structural and functional roles in the body. Australian national dietary guidelines recommend consuming a range of foods in order to ensure adequate nutrient intake from the diet (NHMRC, 2005). Assessing nutrient intakes is essential in examining nutrition from a public health perspective, as adverse health effects can arise from both inadequate and excessive intakes. This is in contrast to agriculture and veterinary chemicals and contaminants where only high intakes may have health implications.

It is noteworthy that several food chemicals assessed in the 23rd ATDS, namely copper, selenium and zinc, are recognised as contaminants as well as nutrients. For the purposes of this study, these chemicals were assessed as nutrients rather than contaminants.

*Calcium*

Calcium is essential in the diet for its primary role in the development and maintenance of the structure of bones and teeth. Non-skeletal calcium is required for neural transmission, muscle contraction and cardiac function (Institute of Medicine, 2006).

Milk and milk-based products are the best source of bioavailable calcium. Smaller amounts are present in bony fish, legumes, certain nuts, and foods fortified with calcium (Institute of Medicine, 2006). Some low-oxalate leafy green vegetables, such as bok choy, kale and broccoli, contain appreciable amounts of bioavailable calcium (Heaney *et al*., 1993).

*Chromium*

Chromium is an essential element involved in the action of insulin (Cefalu and Hu, 2004). Only a small amount (0.4-2.5%) of ingested chromium is absorbed. Absorbed chromium binds to plasma proteins, such as transferrin and is transported to the liver. Vitamin C appears to increase the absorption of chromium. Urinary excretion of chromium may be increased by aerobic exercise, and in those with diets very high in simple sugars (35% or greater of total energy). Absorbed chromium is excreted rapidly in the urine, while unabsorbed chromium is excreted in the faeces (Institute of Medicine, 2006). In humans, chromium is stored in bone, soft tissue, and organs such as the liver, kidneys and spleen (Lim *et al*., 1983).

Chromium is abundant in the environment as either trivalent chromium or hexavalent chromium, with the chromium present in foods in the trivalent state. Chromium is found at low concentrations in a wide range of foods, including egg yolk, wholegrain products, coffee, nuts, some vegetables, meat, and brewer's yeast (Cefalu and Hu, 2004). Processing and geochemical factors can greatly affect the chromium content of foods.

*Cobalt*

Cobalt is a trace element and a constituent of vitamin $B_{12,}$ which is essential for folate and fatty acid metabolism. Cobalt is found in most tissues in the human body, particularly the liver (WHO, 2006a). Gastrointestinal absorption of cobalt is approximately 50% and depends upon the dose; very low doses are almost completely absorbed and high doses are less well absorbed. Cobalt absorption is reduced by amino acids, and enhanced in individuals with iron deficiency (Expert Group on Vitamins and Minerals, 2003; WHO, 2006b).

The cobalt contained in vitamin $B_{12}$ represents only a small amount of cobalt intake, as vitamin $B_{12}$ only occurs in foods of animal origin (WHO, 2006a). In general, the richest sources of cobalt include fish, nuts, green leafy vegetables and fresh cereals (Barceloux, 1999a; Expert Group on Vitamins and Minerals, 2003). In a study of 150 Australian food and beverages, the items with the highest cobalt concentrations included yeast and yeast products, coffee, nuts, seeds, grains, chocolate and condiments (Hokin *et al*., 2004a).

*Copper*

Copper functions as a component of several metalloenzymes, which act as oxidases in a variety of biological reactions. An example is the ferroxidases, which are involved in ferrous iron oxidation and are needed to bind iron to transferring (Institute of Medicine, 2006). Copper is absorbed primarily in the small intestine and absorption decreases with increasing intake (Turnlund, 1998). Very high levels of iron and zinc in the form of supplements can impair copper absorption (NHMRC, 2006; Wapnir, 1998). Almost two-thirds of the copper in the body is stored in the skeleton and muscles (NHMRC, 2006).

Copper is found in a wide range of foods, with major dietary sources being offal, seafood, legumes, nuts and seeds, and to a lesser extent, wheat bran cereals and wholegrain products (Barceloux, 1999b).

*Fluoride*

Fluoride is necessary for the mineralisation of teeth and bones with approximately 99% of fluoride in the body found in calcified tissues. It can also stimulate the formation of new bone. Fluoride has been classified as essential to human health due to its role in the prevention of dental caries (tooth decay) (NHMRC, 2006).

Water-soluble fluoride is almost completely absorbed, however the bioavailability may be impaired by calcium, magnesium and aluminium (Cerklewski, 1997). The retention of absorbed fluoride decreases with increasing age, from approximately 80% in young children to 50% in adults. Fluoride in the body is eliminated via the kidneys and excreted in urine (NHMRC, 2006).The main effect of low fluoride intake is an increased risk of dental caries (NHMRC, 2006).

Fluoride intake from most foods is low. However, higher concentrations of fluoride are present in fluoridated water and products containing fluoridated water (e.g. beverages such as tea, specific marine fish and infant formula (NHMRC, 2006). In Australia, fluoridation of water is regulated by state and territory governments.

*Iron*

Iron is an important component of several proteins, including the haem proteins (haemoglobin, myoglobin, and cytochromes) and several enzymes in the body. Almost two-thirds of iron is found in haemoglobin, which is required for the transport of oxygen to tissues throughout the body. Body iron stores, which are readily mobilised, contain another quarter as ferritin or haemosiderin, and most of the remaining iron is found as myoglobin in muscle tissue (NHMRC, 2006).

The primary sources of iron in the Australian diet are wholegrain cereals, meat, fish and poultry (NHMRC, 2006). Iron absorption is influenced by the iron status of the individual, the form of the dietary iron, and inhibiting or promoting substances present in the diet.

*Manganese*

Manganese is important for carbohydrate, cholesterol and amino acid metabolism and bone formation. Manganese is an important constituent of several metalloenzymes (NHMRC, 2006). Less than 5% of ingested manganese is absorbed by the body, where it is taken up from the blood by the liver and transported to tissues (NHMRC, 2006). Low serum ferritin concentration is associated with increased manganese absorption (Finley, 1999), and manganese from drinking water and supplements may be more bioavailable than from food.

The major contributors to manganese in the diet are cereal products, tea and vegetables (NHMRC, 2006).

*Molybdenum*

Molybdenum is involved in several body functions, including energy metabolism and the formation of blood, bone and cartilage. It is a cofactor for the enzymes sulphite oxidase, xanthine oxidase and aldehyde oxidase which are necessary for the catabolism of amino acids, purines and pyrimidines (NHMRC, 2006).

The richest sources of molybdenum are legumes, grains and nuts, with the molybdenum concentration of the soil influencing the level of the foods produced. Fruits, vegetables and animal products tend to be low in molybdenum (Barceloux, 1999c; NHMRC, 2006). Molybdenum is absorbed efficiently across a range of dietary intake amounts and urinary excretion is directly related to intake (Turnlund *et al*., 1995a; Turnlund *et al*., 1995b).

*Potassium*

Potassium is the main cation of intracellular fluid and is necessary for normal cellular function (NHMRC 2006). Nerve transmission and muscle contractions are affected by the ratio of extra- to intra-cellular potassium (Institute of Medicine, 2006). In healthy individuals there is a high correlation between dietary potassium intake and urinary excretion, where approximately 85% of ingested potassium is absorbed, and 80-90% is excreted in the urine (Holbrook *et al*., 1984). Climate, physical activity, the use of diuretics, and the intake of other electrolytes (particularly sodium) can affect the body's requirements for potassium (NHMRC, 2006).

Good sources of potassium include vegetables, particularly leafy green and root vegetables and vine fruits. Moderate sources of potassium include beans, peas, tree fruits such as apples, bananas and oranges, dairy products and meat (NHMRC, 2006).

*Selenium*

The trace element selenium is required for the synthesis and expression of over 35 selenoproteins, including iodothyronine 5'deiodinase, thioredoxin reductase, and several forms of the glutathione peroxidise enzymes. These proteins are involved in redox regulation, thyroid hormone regulation, energy metabolism, cell membrane maintenance, immune response, and protection against DNA damage (Brown and Arthur, 2001). The retention and use of dietary selenium in the human body from food likely depends on its chemical form (Hunt, 2003), and absorption is estimated to be approximately 55-70% (NHMRC, 2006).

The primary dietary sources of selenium in Australia are seafood, poultry, eggs, and muscle meats. Cereals can be an important contributor to selenium in the diet, but the selenium content of plant foods is influenced by the geographical region and soil concentration in which they are produced (Thomson, 2004). There is significant variation in the selenium concentration of soil and food crops in many countries, including Australia (Lyons et al., 2005a; Thomson, 2004). For example, in a survey conducted between 2000 and 2002, most wheat grown in South Australia had a selenium concentration that ranged from 70 to 280 μg/kg (Lyons *et al*., 2005b).

*Zinc*

Zinc is necessary for growth and development and its many functions can be divided into catalytic, structural and regulatory. It is a constituent of several enzymes that are involved in the metabolism of protein, carbohydrate and fat, protein structure maintenance, and gene expression regulation (NHMRC, 2006).

Zinc is present in food as a complex, and dietary factors can greatly affect zinc's solubility and absorption in the gut, thus affecting its bioavailability. The type and amount of protein affects bioavailability as zinc absorption tends to be higher in diets rich in animal protein compared to diets high in plant protein. High intakes of iron in supplemental form may decrease zinc absorption (Krebs, 2000). The majority (>85%) of total body zinc is stored in skeletal muscle and bone (NHMRC, 2006).

Zinc is found in a wide range of foods. The main dietary sources are meat, fish and poultry, with cereals and dairy products also making a significant contribution (NHMRC, 2006).

# Part B – Conducting the study

The aim of the 23rd ATDS was to assess the concentrations of, and dietary exposure to a range of agricultural and veterinary chemicals, contaminants and nutrients in the Australian food supply. These dietary exposures were compared to the relevant reference health standards to assess public health and safety.

FSANZ funded and coordinated the study with the assistance of the Australian state and territory government food regulatory agencies. Each state and territory nominated a representative liaison officer to co-ordinate the collection of food samples, packaging and transport to the appointed laboratory for analysis.

## Foods included in the study

A total of 92 foods and beverages, including tap and bottled water, were surveyed in this study, with a total of 570 composite samples analysed. Each composite sample was made up of three primary purchased (individual) samples from a single state or territory. A full list of foods surveyed is provided at Appendix 2.

The types of foods selected for inclusion in the survey were based on the following criteria:

- Foods that are representative of current patterns of food and beverage consumption in Australia.

- Foods that are suspected or known to contribute significantly to the dietary exposure for the chemical analysed.

- Resource capabilities of the states and territories to collect samples.

- Cost associated with the purchase, transport and analysis of samples.

Foods were sampled in accordance with a schedule which covered both national and regional foods. This ensured more samples were collected where there was potential for regional variation in composition of the food.

Regional foods were defined as those foods that might be expected to show regional variation of chemical or nutrient concentrations. These foods included fresh fruit and vegetables, red meat, chicken, eggs, bread and other bakery goods, wine and some dairy foods. Two composite samples of these foods, each consisting of three primary purchases, were collected in four to six capital cities, totalling between 8 and 12 composite samples for each regional food.

National foods were defined as those foods that are distributed nationwide and therefore not expected to show chemical or nutrient variation, such as breakfast cereals, tea, coffee, soft drink and canned fruit. Two composite samples of these foods, each consisting of three primary purchases, were collected in two capital cities, making up 4 composite samples for each national food.

Foods were sampled in each state and territory during January/February 2008 (summer sampling period) and June/July 2008 (winter sampling period). This allowed for variation in produce available during different seasons. Due to the large number of samples collected, purchasing took place over several days within a reasonable time period. Samples were sent to the analytical laboratory as soon as practicable after purchase. Where the analytical laboratory resided outside of the city/state conducting the sampling, all perishable samples (e.g. fruits, vegetables and meat) were sent overnight in a chilled state to the laboratory.

## Food-analyte combinations

All foods were analysed for agricultural and veterinary chemicals and a range of contaminants, including aluminium, total arsenic, cadmium, lead, total mercury, strontium and vanadium. In addition, all foods were analysed for the nutrients calcium, chromium, cobalt, copper, fluoride, iron, manganese, molybdenum, potassium, selenium and zinc.

For the remaining analytes, only a selected range of foods were analysed based on the major contributors to dietary exposure. The analytes and specific foods are listed below:

- **Inorganic mercury**, **methylmercury** and **inorganic arsenic** were analysed in battered fish fillets, frozen fish portions, tuna canned in brine and cooked prawns.

- **Antimicrobial residues**, including nitrofurans, nicarbazin, ractopamine, chloramphenicol and triclabendazole were analysed in selected red meat, white meat, seafood, dairy products and egg samples.

- **Aflatoxins** (**B1**, **B2**, **G1** and **G2**) were analysed in almonds, baked beans in tomato sauce, mixed grain breakfast cereals, single grain breakfast cereals, mixed infant cereal, rolled oats, white rice, peanut butter, meat pie, meat and savoury sauce (non-tomato).

- **Aflatoxin M1** was analysed in dairy products, including full fat milk, full fat fruit yoghurt, cheddar cheese, infant formula and milk-based infant dessert.

- **Mycotoxins deoxynivalenol** and **zearalenone** were analysed in savoury biscuits, breads (white and multigrain), breakfast cereals (single grain, mixed grain and infant cereal), rolled oats, pasta and meat pie.

- **The mycotoxin**, **ochratoxin A**, was analysed in all foods listed above for deoxynivalenol and zearalenone as well as baked beans, chocolate (milk), coffee (espresso and instant), dried apricots, canola and olive oils, full fat soy beverage, sultanas and red wine.

- **Fumonisins B1** and **B2** were analysed in baked beans, breakfast cereals (mixed grains, single grain and infant cereal), meat pie and frozen sweet corn kernels.

- **Patulin** was analysed in fruit juice.

## Food preparation

All foods examined in the study were prepared to a 'table ready' state prior to laboratory analysis. For example, chicken breast and lamb chops were grilled prior to analysis. A number of the foods surveyed were in their table ready form at the time of purchase and therefore did not require additional cooking preparation, such as bread and canned products. Further details on food preparation instructions are provided in Appendix 3.

Any perishable foods were prepared within 48 hours of arriving at the laboratory. Where necessary, the preparation of frozen or shelf-stable foods was delayed to ensure perishable foods were analysed within the timeframe. Remaining foods were all analysed within a week of purchase.

## Analysis of food samples

Symbio Alliance Pty Ltd conducted the analysis for the majority of analytes and all were performed in accordance with accredited quality assurance procedures. The analysis of total mercury, methylmercury and inorganic mercury in prawns, fish and canned tuna was conducted by the University of Canberra Ecochemistry Laboratory. All analytical results were provided to FSANZ.

Table 1 provides an overview of analytical methods used, with further details provided in Appendix 4 for agricultural and veterinary chemicals, Appendix 5 for contaminants and in Appendix 6 for nutrients.

*Table 1: Analytical methods used to test food samples in the 23rd ATDS*

| Analyte/s | Method |
|---|---|
| Aluminium, calcium, iron, potassium | ICP-AES |
| Antimicrobials and mycotoxins | LC/MS & HPLC-MS/MS |
| Arsenic, cadmium, lead, strontium, vanadium | ICP-MS |
| Chromium, cobalt, copper, manganese, molybdenum, selenium, zinc | ICP-AES & ICP-MS |
| Fluoride | Ion specific electrode |
| Total mercury, methylmercury, inorganic mercury | Ion chromatography & ICP-MS |
| Agricultural and veterinary chemicals | GC-MS/ECD/NPD/PFPD/Headspace LC/MS/MS |

# Part C – Estimating dietary exposures

## What is dietary modelling?

Dietary modelling is a tool used to estimate the intake of nutrients or dietary exposure to agricultural and veterinary chemical residues, contaminants and other substances from the diet. To estimate dietary exposure[3] to food chemicals, food consumption data are combined with food chemical concentration data Equation 1.

*Equation 1: Dietary exposure calculation*

**Dietary Exposure = food chemical concentration x food consumption amount**

International expert bodies the FAO and WHO 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 (WHO, 2009b).

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 reference health standards. The different types of reference health standards used in the 23rd ATDS are outlined in Table 2.

While dietary modelling is a scientific systematic method for estimating the amounts of food chemicals to which a population may be exposed, the accuracy of these estimates depends on the quality of the chemical concentration and food consumption data available for use.

For detailed information regarding the procedures used for dietary modelling in the 23rd ATDS, refer to the supplementary information provided in Appendix 8.

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[3]     By convention, the term 'intake' is used to refer to estimates of dietary exposure to nutrients.

*Table 2: Reference health standard types used in the 23rd ATDS*

| Food Chemical Type | Reference Health Standard Type |
| --- | --- |
| Agricultural and veterinary chemicals | Acceptable Daily Intake (ADI) |
| Contaminants | Provisional Maximum Tolerable Daily Intake (PMTDI)<br>Provisional Tolerable Monthly Intake (PTMI)<br>Provisional Tolerable Weekly Intake (PTWI) |
| Nutrients | Estimated Average Requirement (EAR)*<br>Upper Level of Intake (UL)^<br>Adequate Intake (AI) |

* There are currently no EARs established in Australia for chromium, cobalt, copper, fluoride, manganese and potassium.
^ There are currently no ULs established in Australia for chromium, cobalt, manganese and potassium.

## Population groups assessed

Dietary exposures to agricultural and veterinary chemicals, contaminants and nutrients were estimated for a range of population groups including infants, children and adults.  The age range assessed differed for nutrients in order to correlate with relevant reference health standards.  For further information regarding the specific population groups for each food chemical, refer to the supplementary information in Appendix 8.

## Food chemical concentrations

The mean concentrations of agricultural and veterinary chemicals were used in dietary modelling for this study. Where a high number of results were below the limit of reporting (LOR), the mean concentration is a more conservative indicator of the detected concentrations of agricultural and veterinary chemical residues than the median concentration.

The concentrations of contaminants used in dietary modelling were the statistical middle value (median), as calculated using the concentrations for each of the composite samples analysed. Where the distribution of concentrations of food chemicals is positively skewed, as is typically found in surveys of food contaminants, then the median concentration is typically used as it better represents the most likely concentration in any given commodity and thus will avoid overestimating exposure.

The median concentration is a more stable central statistic and is not sensitive to skewing by a small number of chemical detections above the normally expected range. The median simplifies calculations for analytical results below the LOR because the position of the median, unlike the mean, is not dependent on the treatment of results below the LOR. Median concentration values have been used in estimating dietary exposure to contaminants in previous ATDS.

As with agricultural and veterinary chemicals, the concentrations of nutrients used in dietary modelling were the mean analytical results derived from the results for each of the composite samples analysed. Some averaging of the concentrations had already occurred during the sample compositing process.

Food chemical concentrations are provided in Appendix 9, Appendix 10 and Appendix 11.

## Food consumption data

The dietary exposure assessment uses food consumption data from two Australian National Nutrition Surveys (NNS): the 2007 Australian Children's Nutrition and Physical Activity Survey (2007 NNS) for children aged 2-16 years, and the 1995 NNS for those aged 17 years and above (Appendix 12).There has not been a more recent NNS for Australian adults. Detailed information about these NNSs and how they are used in DIAMOND[4] is available on the FSANZ website (FSANZ, 2009). There are some differences in survey design between the two NNSs that are highlighted in Appendix 8.

## Food mapping

Mapping is the process of matching the foods analysed in the ATDS to the foods consumed in the 1995 NNS and 2007 NNS. Given that the ATDS could not survey all foods consumed in the NNSs, mapping is a major step in the dietary modelling process. Mapping can be based on nutritional considerations and/or the expected presence of a chemical in a food. For further information on food mapping, refer to Appendix 8 and Appendix 13.

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[4] DIAMOND (**Di**etary **M**odelling **o**f **N**utritional **D**ata) is a custom built computer program designed to automate dietary exposure calculations.

## Food contribution calculations

Throughout the report, information regarding 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.

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 contains a relatively high concentration of a particular chemical, the amount of the food consumed, the mapping process and the number of individuals that were exposed to the chemical, will determine its identification as a major contributor to the diet.

## Assumptions and limitations in dietary modelling

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

Dietary modelling based on the 1995 NNS and 2007 NNS provides the best available estimates of actual consumption of all foods and the resulting estimated dietary exposure to a food chemical for the population. FSANZ considers that, despite the age of the 1995 NNS, consumption of staple foods, such as fruit, vegetables, meat, dairy products and cereal products, which make up the majority of most people's diet, is unlikely to have changed markedly since 1995 (Cook *et al*., 2001a; Cook *et al*., 2001b).

Nevertheless, limitations still exist in dietary modelling methods as well as in the data. Limitations relating to the food consumption and chemical concentration data include:

- Diets derived from one or two 24-hour food recall surveys are used as the basis for drawing conclusions on lifetime eating patterns (Appendix 8). This normally leads to conservative dietary exposure assessments, particularly where exposure arises from the consumption of non-habitually eaten foods.

- The 1995 NNS data do not include information regarding food products that have been introduced to the market since the 1995 NNS was conducted.

- A single food chemical concentration is used in calculating dietary exposure estimates, which does not take into account the level of uncertainty associated with the concentration measurement or the variability of chemical concentrations.

- Participants in 24-hour food recalls may over- or under- report food consumption, particularly for certain types of foods.

- The dietary intake of nutrients from dietary supplements has not been taken into account.

- Contribution of nutrients that have been added to foods (i.e. fortified foods) has not been taken into account except for breakfast cereals and infant cereal. For example, the contribution of the calcium added to calcium-fortified orange juice has not been taken into account.

- The theoretical diet used for 9 month old infants is not as accurate as the data derived for other population groups from the 1995 NNS and 2007 NNS that use food consumption data of individuals.

Assumptions made in the dietary modelling for the 23rd ATDS include:

- The food chemical concentration in the analysed food was a good representation of the concentration of that chemical in all of the other foods to which it was mapped.

- No contribution to dietary exposures was included for medicines or vitamin and mineral supplements.

- The chemical concentration in a particular analysed food was carried over to all of the mixed foods in which it was used as an ingredient.

- Where a food was not tested for a particular food chemical, then the concentration of that chemical in the food was assumed to be equal to zero (e.g. non-seafood samples were not tested for methylmercury; the methylmercury content of these foods was assumed to be zero).

## Treatment of analytical values below the LOR

Assumptions were also made about the value of analytical results below the LOR or where there were no detections. In general for agricultural and veterinary chemicals, the LOR was reported as equal to the limit of detection (LOD). For a small number of agricultural and veterinary chemicals (34), the LOR is equal to two times the LOD or five times the LOD depending on the matrix analysed. Analytical concentrations below the LOR were assumed to be zero when calculating mean concentrations in foods. As these chemicals are intentionally applied to food crops, it was assumed that the chemicals were not used at all when residues were not present at levels greater than the LOR.

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 were less than the LOR. For all contaminants, the LOR was reported as equal to the LOD. Analytical results below the LOR could be anywhere between zero and the LOR. To allow for this uncertainty, the results for dietary exposure to contaminants were presented as a range. The lower end of the range was calculated based on the assumption that results below the LOR were equal to zero. The upper end of the range, representing a very conservative 'worst-case' estimate, was calculated on the assumption that results below the LOR were equal to the LOR.

As both low and high intakes of some nutrients were examined in the ATDS, it was important not to over-estimate or under-estimate the concentrations of nutrients in foods. For all nutrients, the LOR was reported as equal to the LOD. Where nutrient concentrations were reported as being below the LOR, it was assumed that the nutrient concentration was equivalent to half of the LOR.

## A comparison of dietary exposure assessments with reference health standards

Public health and safety risk is characterised by comparing the dietary exposure with relevant reference health standards.

Different types of food chemicals have different types of reference health standards. The reference health standards that were used for the risk characterisation for each group of chemicals investigated in this study are described below.

## Reference health standards for agricultural and veterinary chemicals

For agricultural and veterinary chemicals, the reference health standard used was the Acceptable Daily Intake (ADI) (Appendix 14). The ADI is an estimate of the amount of a substance in food or drinking water, expressed on a body weight basis, which can be ingested daily over a lifetime without appreciable risk to health. With the exception of allethrin, which does not currently have a reference health standard, the ADIs for the chemicals investigated in this study are those assigned by the Office of Chemical Safety and Environmental Health, Department of Health and Ageing (OCS, 2010).

## Reference health standards for contaminants

For contaminants, estimated dietary exposures were compared to the 'tolerable intake' or 'provisional tolerable intake', either in the form of Provisional Tolerable Weekly Intake (PTWI), Provisional Tolerable Monthly Intake (PTMI) or Provisional Maximum Tolerable Daily Intake (PMTDI) (Appendix 15). This value provides an indication of the amount of chemical that can be consumed on a weekly/monthly basis without appreciable risk. The use of the term 'tolerable' indicates that contaminants are not deliberately added to foods. All 'provisional tolerable intakes' were converted to a daily amount to allow comparison with the estimated daily exposures calculated by DIAMOND. These 'provisional tolerable intake' reference health standards for the contaminants investigated in this study are those assigned by JECFA. This is with the exception of strontium, where the reference health standard is a daily estimate, derived in the Concise International Chemical Assessment Document 77 (WHO, 2010a). Table 3 summarises the reference health standards used for each contaminant.

*Table 3: Summary of reference health standards for contaminants*

| Contaminant | Reference Health Standard | Source |
|---|---|---|
| Aluminium | 2 mg/kg bw (PTWI) | (WHO, 2011a) |
| Arsenic | None | (WHO 2010b) |
| Cadmium | 25 µg/kg bw (PTMI) | (WHO, 2010c) |
| Lead | None[1] | (WHO 2010c) |
| Mercury<br>  Inorganic mercury[2]<br>  Methylmercury | <br>4 µg/kg bw (PTWI)<br>1.6 µg/kg bw (PTWI) | <br>(WHO, 2011b)<br>(WHO 2010b) |
| Strontium | 0.13 mg/kg bw (PMTDI) | (WHO 2010a) |

[1] Risk assessment based on a margin of exposure (MOE) approach
[2] Also applicable to dietary exposure to total mercury in foods other than fish and shellfish
Note: vanadium has not been included in Table 3 as there were no detections in any of the foods analysed.

## Reference health standards for nutrients

For nutrients, several reference health standards were used based on the Nutrient Reference Values (NRVs) established by the NHMRC (2006) (Appendix 16). NRVs are a collection of reference values for both lower and upper intakes of a range of nutrients, and therefore can be used to assess both nutrient adequacy and nutrient excess (NHMRC, 2006). The NRVs used in this survey were:

- Estimated average requirement (EAR) – this is the daily nutrient level estimated to meet the requirements of half the healthy individuals in a particular life stage and gender group. In this report, the prevalence of nutrient inadequacy for molybdenum and selenium in each age-gender group was estimated as the proportion with a usual intake below the EAR using the EAR cut-point method (Institute of Medicine, 2000).

- Adequate Intake (AI) – this is the average daily nutrient intake level based on observed or experimentally determined approximations or estimates of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate. Using an AI to assess nutrient adequacy is limited and can only be applied with confidence when the AI is representative of the mean or median intake of a healthy population group similar to that being evaluated. In this case, when a population group has a mean nutrient intake at or above the AI, this suggests there is a low prevalence of inadequate intake. When other criteria have been used to set the AI, similar conclusions can be made, but with less confidence. Caution must be applied to the interpretation of population intakes in comparison with the AI because it is not known how far below the AI the unknown EAR is located. Assumptions cannot be made about the prevalence of inadequate intakes when the mean intake of a group is less than the AI.

- Upper Level of Intake (UL) – the highest average daily nutrient intake level likely to pose no adverse health effects to almost all individuals in the general population. As intake increases above the UL, the potential risk of adverse effects increases (NHMRC, 2006). In this report, the UL was used to estimate the percentage of the population at potential risk of adverse effects from excessive nutrient intake (NHMRC, 2006).There is no pre-determined cut-off for an acceptable proportion of a population to exceed the UL. ULs are generally estimated by applying uncertainty factors to levels of intake in studies of the effects of high intakes. Sometimes ULs are extrapolated from animal studies and have large uncertainty factors to allow for inter-species differences.

With respect to the nutrients examined in the 23rd ATDS, calcium, iron, molybdenum selenium and zinc have EARs and ULs. It should be noted that the proportions of the population with estimated dietary intakes of calcium, iron and zinc below the relevant EAR have not been reported in this study. The reasoning for this is that intake estimates for these three nutrients have been published in the 1995 NNS and the 2007 NNS, in which both surveys include the nutrient profile and consumption information for thousands of individual foods. Given that a much smaller number of foods have been analysed in this study (92 foods), caution should be exercised in using this study to consider the adequacy of nutrient intakes.

Additionally this study may not have sampled and analysed foods that are fortified with nutrients and that may make substantial contributions to nutrient intakes in some population groups. For example, some common fortified beverage bases that contain added minerals were not sampled and dietary supplements were not included. Estimates of population nutrient intakes from national nutrition surveys are a more accurate representation of nutrient adequacy as these consider nutrient intakes from all sources with nutrient values for all foods.

For infants aged less than one year, calcium, molybdenum and selenium have an AI rather than an EAR. Copper and fluoride have an AI and a UL, whereas chromium, manganese and potassium have an AI only. Cobalt has neither value.

Several food chemicals assessed in the 23rd ATDS, namely copper, selenium and zinc, are recognised as contaminants as well as nutrients. For the purposes of this study, these chemicals were assessed as nutrients against the relevant NRVs.

## Chemicals without reference health standards

The agricultural and veterinary chemical residue allethrin, and the contaminants arsenic and lead have no formal reference health standard. Previous reference health standards for arsenic and lead were withdrawn as they were considered to be no longer appropriate (WHO, 2010b; WHO, 2010c).

As such, results were presented in the appendices in micrograms per kilogram of bodyweight per day (mg/kg bw/day) only and the margin of exposure (MOE) approach to risk assessment was employed. It should be understood that the MOE approach is unlike a comparison with a reference health standard and therefore should not be interpreted as giving a precise estimate of potential risk.

Typically the MOE approach compares the margin between a dose or an exposure causing an adverse effect in animals or humans with the estimated human exposure to that chemical. The calculation usually involves a reference point value (also called a point of departure) derived from the hazard assessment that is then divided by an estimate of human dietary exposure to give a dimensionless ratio that is the MOE. Frequently several MOE are calculated for an individual substance if estimates of exposure vary within the human population.

The MOE approach is most commonly applied to genotoxic carcinogens, such as arsenic, but also has some applicability for other compounds, such as lead, for which no clear reference dose has been identified to derive a reference health standard (e.g. PTWI). As for all contaminants, it is always preferable to minimise exposure by trying to adhere to the ALARA (as low as reasonably achievable) principle.

# Part D – Results and risk characterisation

## Introduction

The results section of this report has been divided into three sections: the first addresses agricultural and veterinary chemicals, the second contaminants and the third nutrients. Selected mycotoxins were also investigated in this study, however there were no detections of mycotoxins in any of the foods analysed. Therefore a dietary exposure estimate and subsequent risk characterisation were not conducted.

It should be noted that a hazard characterisation for each analyte has not been revisited in this report. FSANZ has used the ADIs for agricultural and veterinary chemicals summarised in Appendix 14, the JECFA assigned reference health standards for contaminants in Appendix 15 and the NRVs for nutrients in Appendix 16. These reference health standards are also summarised on pages 27-30 of this report. The adverse effect/ hazard for each contaminant and function of each nutrient has also been summarised in this section of the report.

Estimated dietary exposures are reported for all consumers of the particular chemical in the 1995 NNS and 2007 NNS for their respective age-gender group, as opposed to 'all respondents'. The average body weights for each age-gender category and the mean consumption amounts of each food are provided in Appendix 8 and Appendix 12 respectively.

The analytical results for agricultural and veterinary chemicals, contaminants and nutrients are summarised in Appendix 9, Appendix 10 and Appendix 11. All analytical results are expressed in milligrams per kilogram (mg/kg) of the edible portion of the food prepared for consumption, unless otherwise stated. The estimated dietary exposures for chemicals are displayed in Appendix 17 for agricultural and veterinary chemicals, Appendix 18 for contaminants and Appendix 19 for nutrients.

Data were derived from nutrition surveys using different methodologies or using a model diet, as outlined in Appendix 8. Therefore caution should be used when comparing exposure for infants, children and adults.

Despite the different approaches used to estimate dietary exposures in different age groups, the results for all population groups have been presented together in the figures.

## Agricultural and veterinary chemicals results and risk characterisation

Dietary exposures were estimated only when an agricultural or veterinary chemical was detected in a food. Some of these chemicals were not detected in any food and consequently their estimated dietary exposures were zero.

In this study, the estimated dietary exposure to agricultural or veterinary chemicals was calculated at:

- The mean and 90th percentile levels of dietary exposure in µg/day for all age groups (Appendix 17).

- The 90th percentile of dietary exposure in µg/kg bw/day and as a percentage of the relevant reference health standard (ADI).

The concentrations of agricultural and veterinary chemicals reported in this study are provided in Table A9.1, sorted by food and Table A9.2, sorted by chemical, of Appendix 9.

Results for dietary exposure estimates of agricultural and veterinary chemicals, where they were detected in foods, are tabulated in Appendix 17. Additionally, the foods that had no agricultural or veterinary chemical residues detected are listed in Appendix 9.

The major food contributors to dietary exposure of each agricultural and veterinary chemical are presented in Appendix 20.

Forty-six agricultural and veterinary chemicals were detected in this study. Of these, allethrin (detected in mushroom and beef sausage) was the only chemical not approved for use in any foods in the Code.

There were also detections of residues of the following agricultural and veterinary chemicals in foods, which are not listed in the Code for the identified foods:

- 2-phenylphenol – mushrooms
- Azoxystrobin – celery
- Chlorpyrifos – cucumber
- Guazatine – celery
- Iprodione – oranges, cucumber
- Nicarbazin – eggs
- Pirimiphos-methyl – avocado

- Tebufenpyrad – nectarine
- Triclopyr – cereal grains

The level of iodosulfuron-methyl detected in biscuits and savoury sauce exceeded the MRLs in the Code for all approved commodities including milk, wheat and eggs.

For those chemicals that were detected but not approved for use or are approved but detected at levels that exceeded the MRL specified in the Code, the relevant state or territory was notified for follow up action.

The estimated dietary exposures to all forty-six agricultural and veterinary chemicals were below their respective ADIs at the 90th percentile of exposure or all age groups assessed.

Seven agricultural and veterinary chemicals had dietary exposures at 10% of the ADI or greater, for one or more age group assessed at the 90th percentile, still well below their respective reference health standard (Figure 1 to Figure 7). These were chlorpyrifos, dieldrin, diphenylamine, dithiocarbamates, iprodione, methamidophos and propargite.

For all age groups, the highest dietary exposure to an agricultural and veterinary chemical residue at the 90th percentile of exposure, as compared to the ADI, was for propargite. Propargite is one of the acaricide groups of pesticides, which are used for controlling mites on a variety of crops. Dietary exposures to propargite at the 90th percentile ranged from 20% of the ADI for people aged 17 years and above, up to 60% of the ADI for 2-5 year old children.

The ATDS food with the highest reported propargite concentration was apples, with a mean concentration of 0.12 mg/kg. The major food group contributor to propargite exposure across all age groups was apples and quinces, accounting for greater than 90% of the estimated dietary exposure (Table A9.2 in Appendix 9 and Table A17.1 in Appendix 17).

Estimated dietary exposures to agricultural and veterinary residues tended to be highest for the 2-5 year age group. This is consistent with results from the 20th ATDS (FSANZ, 2003) and is due to this population group's high food consumption relative to body weight. An exception to this was methamidophos, where the estimated 90th percentile dietary exposures were highest for people aged 17 years and above, at 15% of the ADI. The only food group contributor to methamidophos exposure for all age groups was capsicum, chillies and spices. Because of their relatively low consumption of these foods, children did not have the highest dietary exposures.

Given the estimated dietary exposures for all 46 agricultural and veterinary chemicals were below respective ADIs at the 90th percentile, there are no public health and safety issues associated with current dietary exposures to agricultural and veterinary chemical residues in food for the Australian population. This finding is consistent with the results of the 19th and 20th ATDS (FSANZ, 2001; FSANZ, 2003).

*Figure 1: Estimated 90th percentile dietary exposures to chlorpyrifos, as a percentage of the ADI*
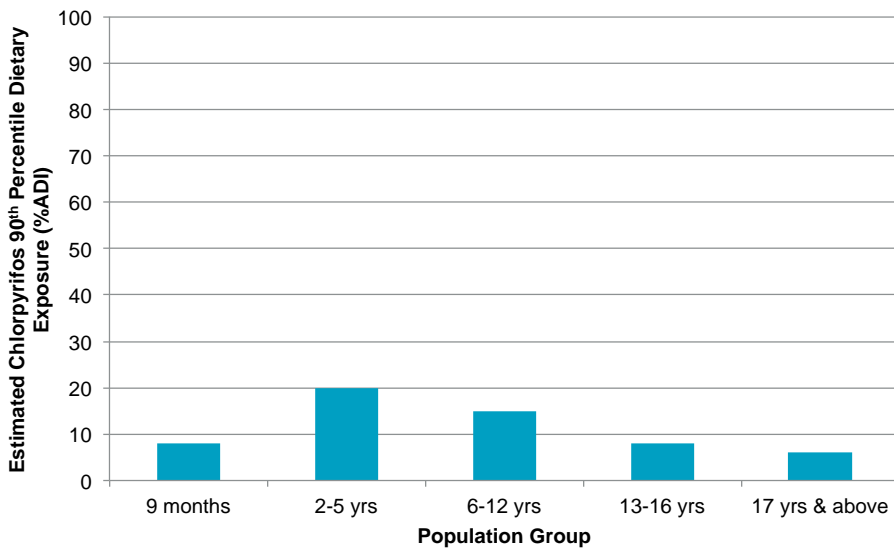
Figure 2: Estimated 90th percentile dietary exposures to dieldrin, as a percentage of the ADI
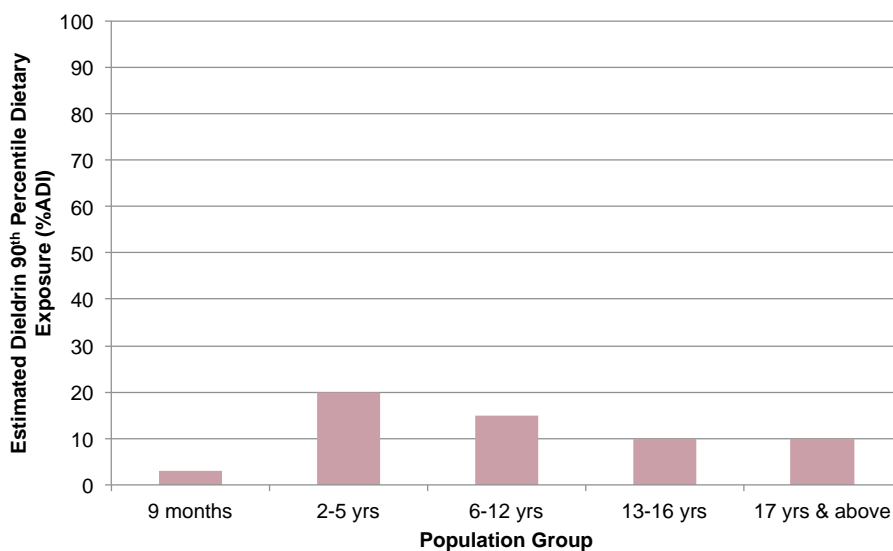


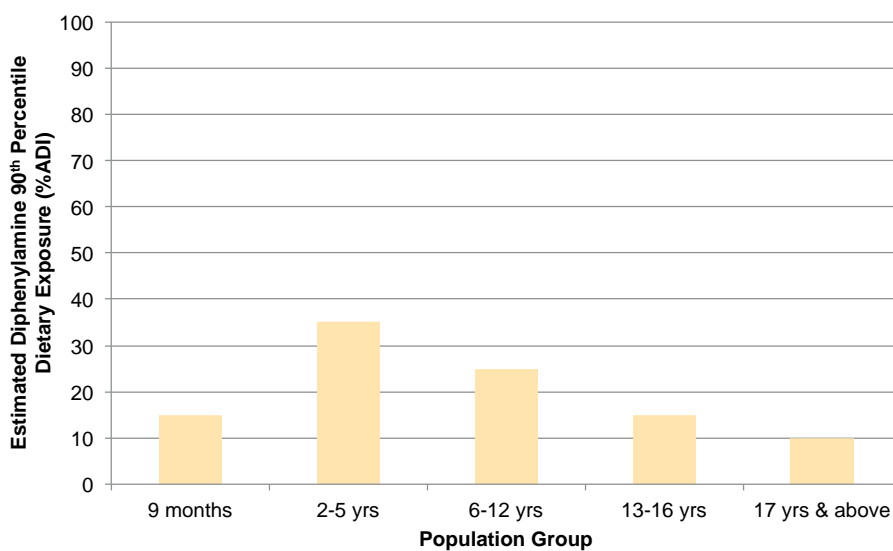Figure 3: Estimated 90th percentile dietary exposures to diphenylamine, as a percentage of the ADI

Figure 4: Estimated 90th percentile dietary exposures to dithiocarbamates, as a percentage of the ADI
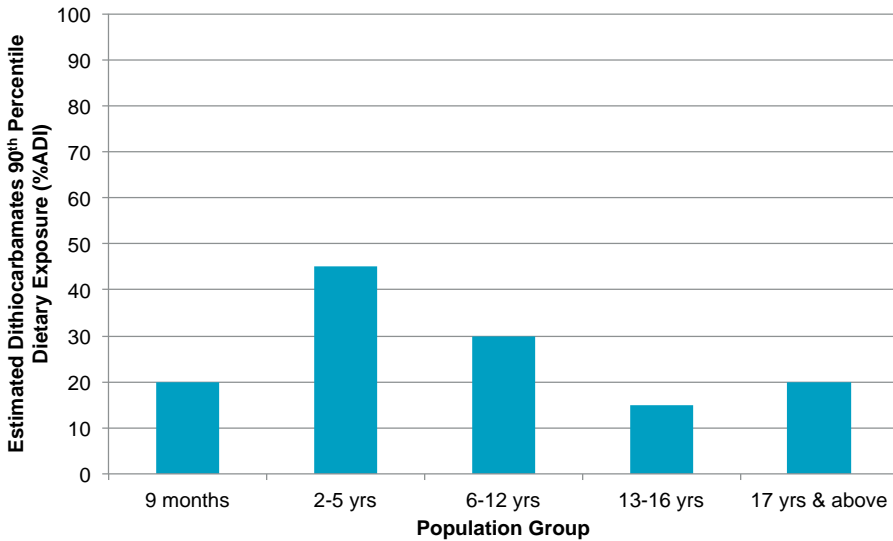


Figure 5: Estimated 90th percentile dietary exposures to iprodione, as a percentage of the ADI
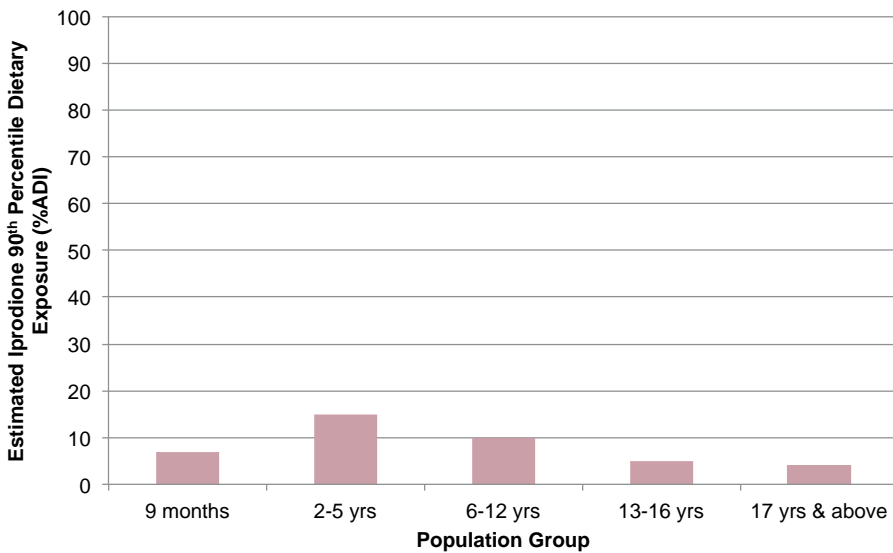
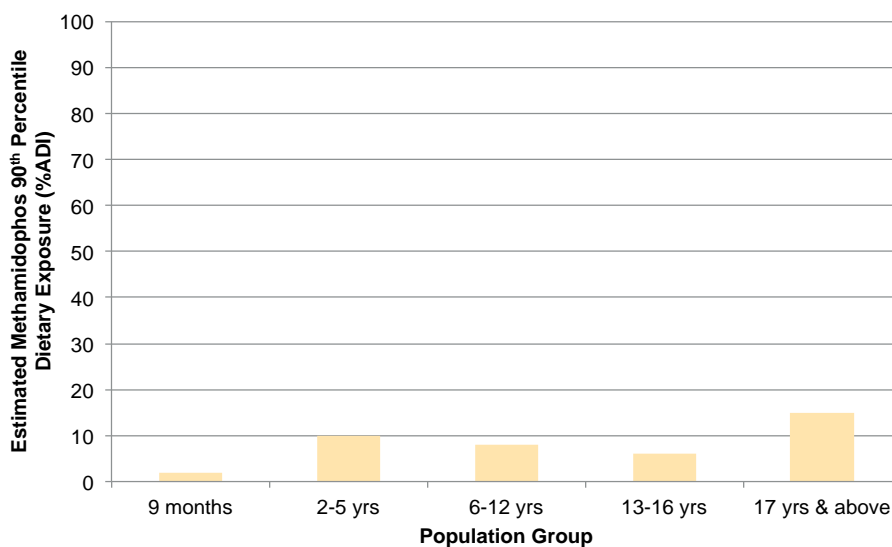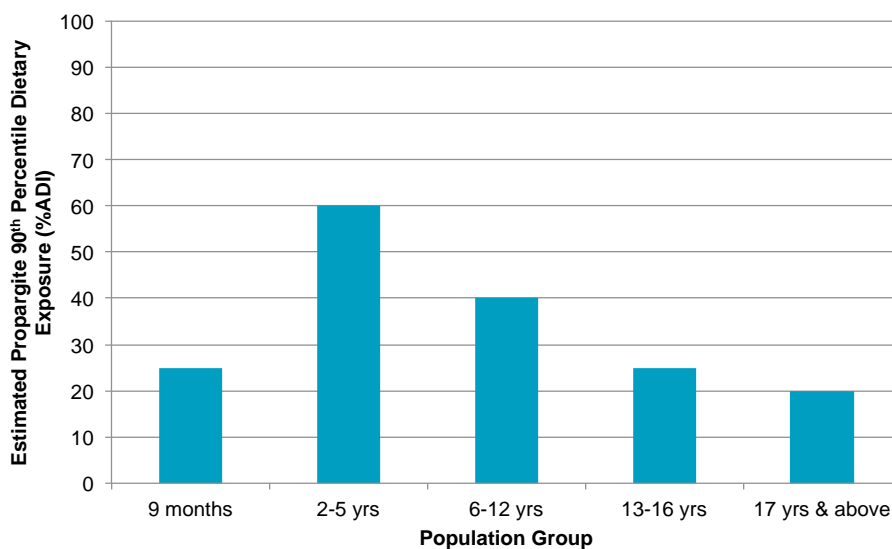*Figure 6: Estimated 90th percentile dietary exposures to methamidophos, as a percentage of the ADI*



*Figure 7: Estimated 90th percentile dietary exposures to propargite, as a percentage of the ADI*

**Agricultural and veterinary chemical results – comparisons with previous studies**

Agricultural and veterinary chemicals have not been surveyed since the 20th ATDS in 2001. While similar types of foods were surveyed in the 20th and 23rd ATDS, the latter survey included a wider range of foods within the various food groups.

In the 20th ATDS, the results were presented as a percentage of the ADI for the mean estimated dietary exposure. In the current study, results are presented as a percentage of the ADI for the 90th percentile of dietary exposure. Therefore, dietary exposure results are not directly comparable between the two surveys. In addition, with the small number of samples collected, relative to the large range of foods available in the marketplace, it is difficult to draw any definite conclusions about any differences in dietary exposures over time. However, both studies have independently shown that there are no human health and safety risks with regard to current dietary exposures to agricultural and veterinary chemical residues in food available to the Australian population.

There were no detections of agricultural and veterinary chemical residues in any infant foods analysed in this study. In the 20th ATDS, residues of pirimiphos-methyl at concentrations close to the LOD were found in two of the nine analytical samples of mixed infant cereal. These results indicate that on rare occasions infant foods are found to contain agricultural and veterinary chemical residues, although in this case the exposure to infants for this chemical was estimated to be low at 0.01% of the ADI.

The organochlorine residues that were detected in this study were dicofol, dieldrin, and endosulfan.

Dicofol was detected in capsicum and strawberries, with mean levels of exposure of ≤1% of the ADI. Dicofol was also detected in the 19th ATDS in beef, grapes, lettuce, rockmelon and tomatoes but was not detected in any of these samples in the 20th ATDS. Mean levels of exposure were 8-30% of the ADI in the 19th ATDS.

Dieldrin was detected in this study in one of eight samples of pumpkin, with mean exposure levels of 2-9% of the PMTDI. Dieldrin was not detected in any sample in the 19th or 20th surveys. However, residues of dieldrin were detected in chicken wings and cucumber in the 18th ATDS, with mean levels of exposure of 1-3% of the PMTDI.

Endosulfan was detected in samples of tomatoes in this study with mean levels of exposure less the 1% of the ADI. Endosulfan was also detected in the 19th and 20th ATDS. This residue was detected in tomatoes, lettuce and several other foods in the 19th ATDS and tomatoes and green beans in the 20th ATDS. Mean levels of exposure in the 19th and 20th ATDS were all 1% of the ADI or less.

## Contaminant results and risk characterisation

The contaminants examined in this survey were aluminium, arsenic, cadmium, lead, mercury (inorganic mercury and methylmercury) strontium and vanadium. Copper, selenium and zinc were also examined; however, even though they may produce adverse effects if exposure exceeds certain levels, these elements are essential for human health. As such, they have been reported as nutrients, with exposure assessed against NRVs in a later section of this report.

The concentrations of contaminants reported in the foods analysed in this study are provided in Appendix 10, with each contaminant presented separately. It should be noted that there were no detections of contaminants in any foods above the relevant MLs stipulated in the Code. Also, given there were no detections of vanadium in any of the foods analysed, a dietary exposure estimate and subsequent risk characterisation were not conducted for vanadium.

The estimated dietary exposures to each contaminant are presented in Appendix 18. Major food contributors to dietary exposure of each contaminant are presented in Appendix 21.

For this study, the estimated dietary exposures for contaminants were calculated using median concentration data at:

- The mean and 90th percentile levels of exposure in mg/day for all age groups, for the lower and upper end of the range (Appendix 18).

- The mean and 90th percentile of exposure as a percentage of the relevant reference health standard, for the lower and upper end of the range.

Estimated levels of exposure, as a percentage of the relevant reference health standard are provided in Figure 8 to Figure 17. Each contaminant is represented separately, and results are shown by age groups.

All dietary exposure estimates were below the relevant reference health standard where established for the contaminants examined.

The risk assessment for lead was based on a MOE approach, as there is currently no reference health standard for this contaminant. Mean estimated dietary exposures for lead were below the level considered by JECFA to have a low risk of reducing the population IQ for children or increasing the systolic blood pressure in adults.

**Aluminium**

*Adverse effects*

The JECFA recently reviewed the PTWI for aluminium (WHO, 2011a). The Committee established a PTWI of 2 mg/kg bw on the basis of adverse effects on reproduction and development in laboratory animals (WHO, 2011a).

There have been questions about the possible role of aluminium in the development of Alzheimer's disease, however conclusive evidence to support this association has not been demonstrated (WHO, 2007b).

*Analytical and dietary exposure results*

The mean, median, minimum and maximum concentrations of aluminium found in the foods analysed are presented in Table A10.1 of Appendix 10. The food with the highest median aluminium concentration was chocolate cake with icing, with a concentration of 87.9 mg/kg.

The majority of foods analysed in this study contained quantifiable concentrations of aluminium in at least half of the composite samples analysed. White sugar, bananas, mango, orange, and peaches in natural juice did not contain quantifiable concentrations of aluminium in any of the analysed composites.

The estimated dietary exposures to aluminium for each age group are presented in Table A18.1 and Table A18.2 of Appendix 18.

Figure 8 indicates that estimated dietary exposures were below the PTWI for all age groups at both the median and 90th percentile consumption levels (high consumers).

*Figure 8: Range of mean and 90th percentile estimated dietary exposure to aluminium, as a percentage of the PTWI, derived using median analytical concentrations§*
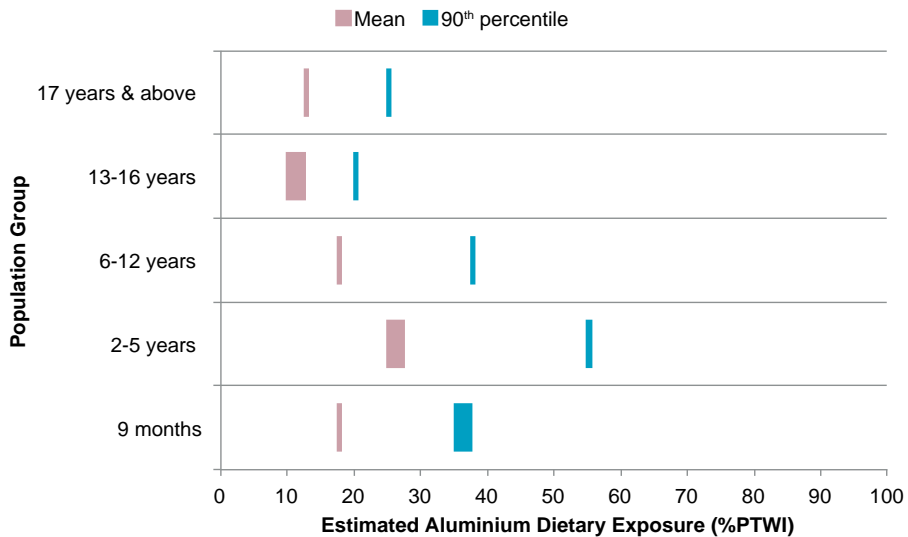


§ Lower end of the range represents where all 'not detected' analytical results have been assigned a concentration of zero; the upper end of the range represents where all 'not detected' analytical results have been assigned a concentration equal to the LOR.

*Major food contributors*

The food type with the major contribution to aluminium dietary exposures was cereal and grain based foods for all age groups (Figure 9). Non-alcoholic beverages (excluding waters and milk) were a major contributor for those aged 17 years and above and infant formula for 9 month old infants.

More specific details on the major food contributors to aluminium dietary exposures are presented in Table A21.1 and Table A21.2 of Appendix 21. The food group 'cakes, muffins and puddings' (categorised under cereal and grain based foods) was the major source of aluminium exposure for children aged 2-16 years. This food group contributed 32-38% of the estimated dietary exposure to aluminium. These results may be partially explained by the presence of aluminium containing additives such as sodium aluminium phosphate (food additive number 541), which are commonly used as leavening agents in a number of products in this food group.

For 9 month old infants, infant formula was the major contributor (29%) to aluminium dietary exposures, followed by 'cakes, muffins and puddings' (19%).

For Australians aged 17 years and above, tea (35%) was the major contributor to aluminium dietary exposures followed by 'cakes, muffins and puddings' (23%).

*Figure 9: Major food contributors to aluminium dietary exposures*
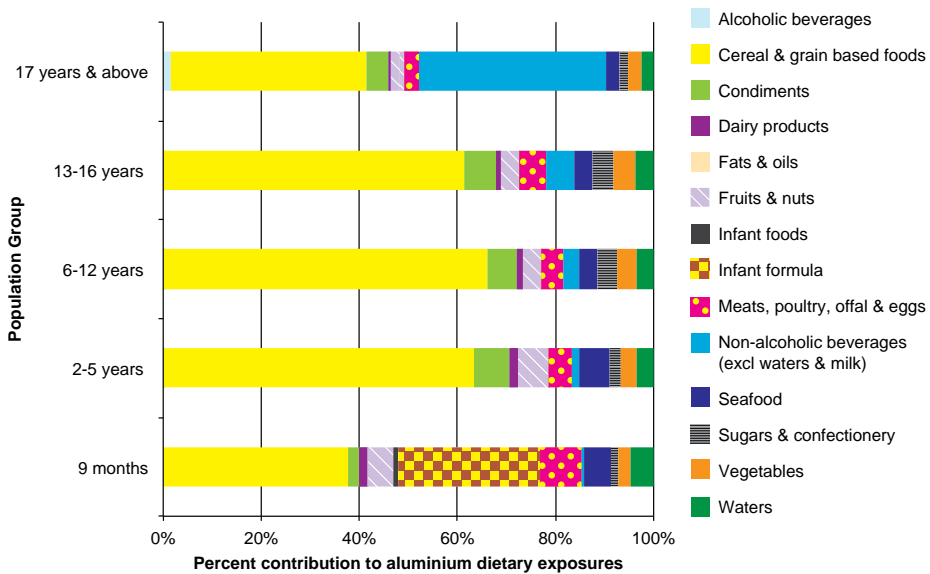


*Risk characterisation*

JECFA recently reconsidered the PTWI for aluminium. The Committee established a PTWI of 2 mg/kg bw based on adverse effects on reproduction and development in laboratory animals. The previous PTWI of 1 mg/kg bw was withdrawn (WHO, 2011a).

The mean dietary exposures to aluminium in this study were lower than the JECFA established PTWI of 2 mg/kg bw across all population groups. Therefore, estimated dietary exposure to aluminium does not pose a risk to human health and safety.

FSANZ is also undertaking additional aluminium analysis as part of the 24th ATDS and will update exposure assessments in the future.

*Comparisons with previous studies*

Aluminium was not investigated in the 19th or 20th ATDS (conducted in 1998 and 2001 respectively). Therefore, it is not possible to comment on any differences in estimated dietary exposures over time.

*Summary table*

| Contaminant | Reference health standard | Adverse effect | Major food contributors | Risk characterisation |
|---|---|---|---|---|
| Aluminium | 2 mg/kg bw (PTWI) | • Adverse effects on reproduction and development in laboratory animals | • Cakes, muffins and puddings | • Estimated dietary exposures were below the PTWI for all population groups |

**Arsenic**

*Adverse effects*

The previous reference health standard for inorganic arsenic was withdrawn by JECFA because it was not possible to establish a safe level of exposure. Therefore there is currently no reference health standard for inorganic arsenic.

Acute toxicity as a result of high exposure to inorganic arsenic can result in gastrointestinal disturbances such as vomiting (WHO, 2001). Chronic toxicity from high exposure to inorganic arsenic from drinking water has been associated with cancer (WHO, 2001).

People who consume large amounts of seafood may ingest significant amounts of arsenic, however the arsenic in seafood is primarily in the organic, less toxic form (Borak and Hosgood, 2007).

*Analytical and dietary exposure results*

This study examined total arsenic in all foods and inorganic arsenic in all seafood sampled, namely, battered fish fillets, frozen fish portions, prawns, and canned tuna. Inorganic arsenic was only measured in seafood because of the generally higher concentrations of arsenic in these foods, and to identify the quantities of the more toxic inorganic arsenic.

The mean, median, minimum and maximum concentrations of inorganic arsenic and total arsenic found in the foods analysed are presented in Table A10.2 and Table A10.3 of Appendix 10. The seafood samples surveyed contained the highest concentrations of total arsenic in comparison to the other foods surveyed. The median concentration of total arsenic present in seafood was between 0.71 and 2.5 mg/kg. There were no detections of inorganic arsenic in any of the sampled seafoods, at an LOR of <0.05 mg/kg.

The majority of foods contained quantifiable concentrations of arsenic in at least one of the samples analysed. Approximately 15% of foods did not contain quantifiable concentrations in any of the analysed samples. Some of these foods included raw green beans, tea, sugar, soft drink, almonds and watermelon.

The estimated 90th percentile dietary exposures to total arsenic for each age category were between 1.0 and 2.8 µg/kg bw/day where no detections were assigned a value of zero, and between 1.2-3.2 µg/kg bw/day where no detections were assigned the value of LOR. In the absence of reference health standards for total and inorganic arsenic, and given that there were no detections of inorganic arsenic in selected samples, only the estimated dietary exposures to total arsenic for each age category were calculated. Results are provided in Table A18.1 of Appendix 18.

*Major food contributors*

Cereal and grain based foods and seafood were the food types that were the major sources of arsenic dietary exposure for all age-gender groups (Figure 10).

More specific details on the major food contributors to arsenic dietary exposures are presented in Table A21.3 of Appendix 21. In this study, the food group fish (including unbattered, uncrumbed or canned) was the major source of total arsenic dietary exposure for all age groups. This food group contributed to 32-41% of total arsenic exposure. Crumbed seafood contributed to 15% of total arsenic dietary exposure in the diets of 9 month old infants and 2-5 year old children, while battered seafood contributed to approximately 20% of the exposure to total arsenic in the diets of those aged 6 years and above. Another food group contributing to total arsenic exposure across all age groups was rice and rice products (11-13%). For 9 month old infants, infant cereals were also major sources of arsenic exposure, contributing to 7% of total exposure.

*Figure 10: Major food contributors to arsenic dietary exposures*
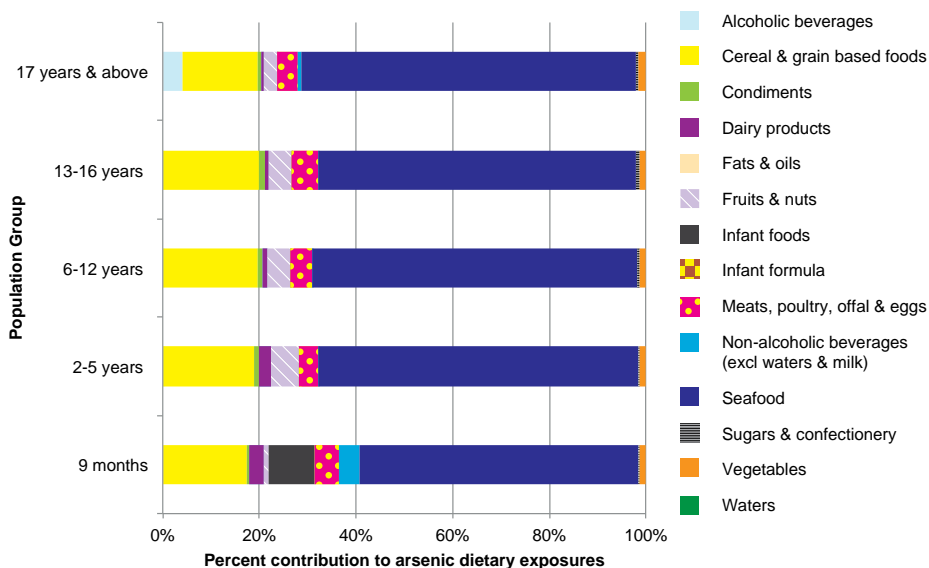


*Risk characterisation*

Total arsenic was detected in a variety of foods. However as the data do not differentiate between organic and the more toxicologically-relevant, inorganic arsenic, and as there is no reference health standard against which to compare the intakes, no comments can be made on the current exposures to total arsenic. However, the more toxicologically relevant inorganic arsenic was below the LOR in the analysed seafood samples. This suggests that there is unlikely to be any public health and safety issues in relation to inorganic arsenic on the basis of the seafood sampled in this survey.

*Comparisons with previous studies*

Total arsenic was reported in both the 19th and 20th ATDS. Consistent with this latest survey, seafood contained the highest concentrations of total arsenic in comparison to the other foods surveyed.

In the 19th ATDS, estimated mean dietary exposures to total arsenic were 0.48-1.7 µg/kg bw/day, depending on the population group being assessed whereas in the 20th ATDS exposures were 0.28-1.4 µg/kg bw/day. In this study estimated mean dietary exposures to total arsenic were similar at 0.42-1.4 µg/kg bw/day.

*Summary table*

| Contaminant | Reference health standard | Adverse effect | Major food contributors | Risk characterisation |
|---|---|---|---|---|
| Arsenic | None | • Not possible to establish a safe level of exposure | • Seafood<br><br>• Cereal and grain based foods | • Given there were no detections of inorganic arsenic above the LOR, there is unlikely to be a human health and safety risk |

**Cadmium**

*Adverse effects*

The JECFA assigned reference health standard for cadmium is a PTMI of 25 µg/kg bw and is based on kidney toxicity (WHO, 2010c). High concentrations of cadmium may also lead to considerable accumulation in the liver (WHO, 1992).

*Analytical and dietary exposure results*

The mean, median, maximum and minimum concentrations of cadmium found in the foods analysed are presented in Table A10.4 of Appendix 10. The food with the highest median cadmium concentrations was potato crisps, with a concentration of 0.11 mg/kg. The majority of foods contained quantifiable concentrations of cadmium in at least one of the samples analysed. Approximately 10% of foods did not contain quantifiable concentrations in any of the analysed samples and included apples, bananas, beer, butter, coffee, ice cream and chicken breast.

The estimated dietary exposures to cadmium for each age category are presented in Table A18.1 and Table A18.2 of Appendix 18. Figure 11 indicates that dietary exposures to cadmium at the 90th percentile of exposure were below the PTMI of 25 µg/kg bw for all age groups, and were consequently within acceptable safety standards. The highest 90th percentile exposure to cadmium was for 2-5 year olds due to their high food consumption relative to body weight. This exposure ranged from 50-60% of the PTMI.

*Figure 11: Range of mean and 90th percentile estimated dietary exposure to cadmium, as a percentage of the PTMI¶*
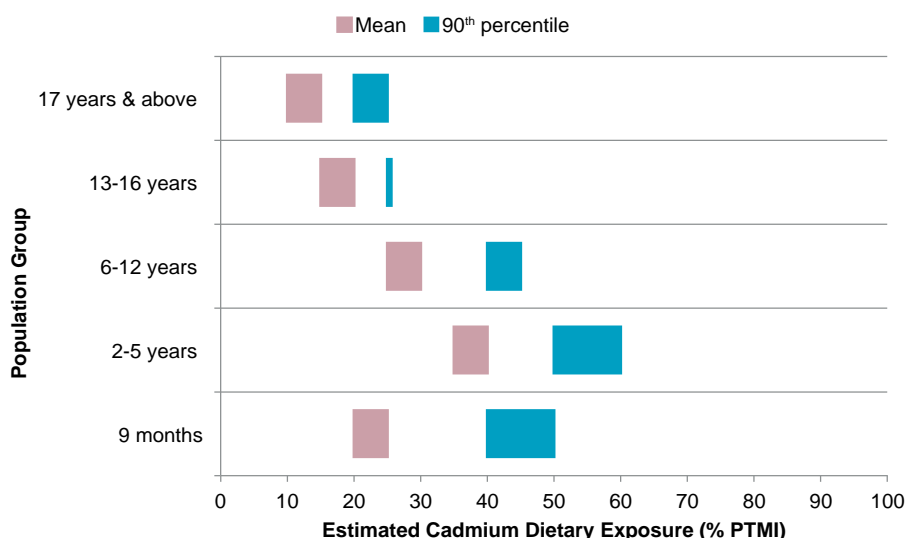


¶ Lower end of the range represents where all 'not detected' analytical results have been assigned a concentration of zero; the upper end of the range represents where all 'not detected' analytical results have been assigned a concentration equal to the LOR.

*Major food contributors*

As shown in Figure 12, cereal and grain based foods and vegetables were the food types that were the major sources of cadmium dietary exposure for all age-gender groups.

More specific details on the major food contributors to cadmium dietary exposures are presented in Table A21.4 of Appendix 21. The food group 'root vegetables (starchy)' (e.g. potatoes) was the major source of cadmium exposure for all age groups. This food group contributed 21-27% of total cadmium exposures. Another major contributor was savoury snacks (categorised under cereal and grain based foods), contributing 6-17% to total dietary exposures. These results may possibly reflect the high concentrations found in potato crisps. The food group 'pasta, noodles (except rice) and couscous' (categorised under cereal and grain based foods) was another major contributor to total cadmium exposure in the diets of infants and children aged 2-16 years (6-8% of total dietary exposures). The food group 'multigrain, wholemeal and rye breads' was a major source of cadmium exposure for infants and children below 6 years of age and for the population group aged 17 years and above, contributing to 7% of total exposures.

*Figure 12: Major food contributors to cadmium dietary exposures*
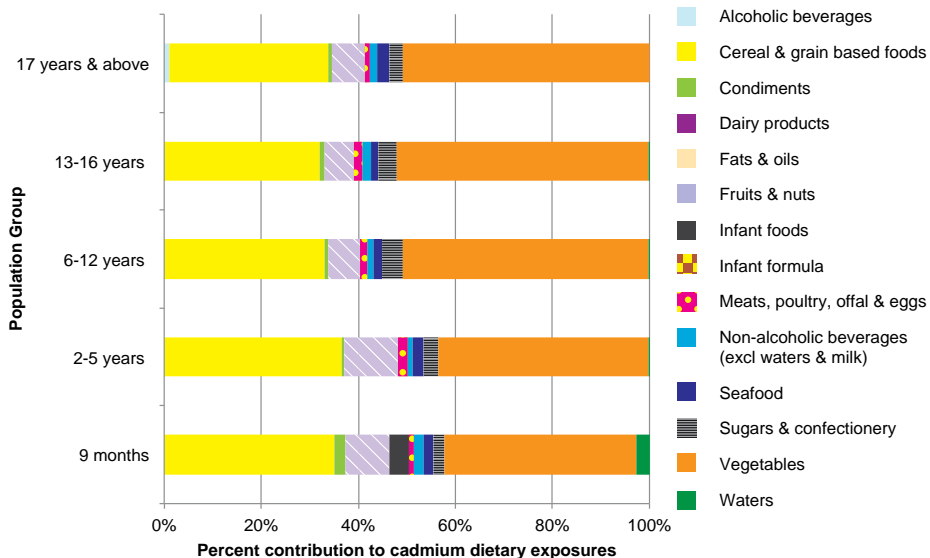


### Risk characterisation

Dietary exposures to cadmium were below the respective reference health standard. On this basis, there are no public health and safety issues with regard to current intakes of cadmium by Australian consumers.

### Comparisons with previous studies

This contaminant was reported in both the 19th and 20th ATDS. Consistent with this latest survey, all estimated dietary exposures to cadmium were below the tolerable limit. Previous studies reported the mean levels of exposure (rather than 90th percentile levels) against a PTWI of 7 µg/kg of bw/week for different age groups. As such, it is not appropriate to make any direct comparisons regarding changes to dietary exposure, as a percentage of the PTWI, of cadmium over time.

In the 19th ATDS, mean dietary exposures to cadmium were estimated to be 0.08-0.58 µg/kg bw/day, depending on the population group being assessed. The 20th ATDS estimated the range of mean dietary exposures to be 0.07-0.68 µg/kg. The estimated range of mean dietary exposures to cadmium in this study was 0.09-0.33 µg/kg bw/day depending on the population group; however different population groups were examined in this study compared to the 19th and 20th ATDS.

*Summary table*

| Contaminant | Reference health standard | Adverse effect | Major food contributors | Risk characterisation |
|---|---|---|---|---|
| Cadmium | 25 µg/kg bw (PTMI) | • Kidney toxicity | • Root vegetables e.g. potato<br><br>• Savoury snacks<br><br>• Pasta, noodles and couscous | • Estimated dietary exposures to cadmium were below the reference health standard for all age-gender groups |

**Lead**

*Adverse effects*

There is currently no reference health standard for lead, so the risk assessment has been based on the MOE approach. For adults, there is consistent evidence that the first significant effect seen at lower blood lead concentrations is an increase in systolic blood pressure (WHO 2010c).

Infants and children are considered particularly vulnerable to the potential neurotoxic effects of lead exposure (Tong *et al*., 2000). This is due to their higher energy requirements, their higher fluid, air and food intake per unit of body weight, and the immaturity of their kidneys, liver, nervous and immune systems.

*Analytical and dietary exposure results*

The mean, median, maximum and minimum concentrations of lead found in the foods analysed are presented in Table A10.5 of Appendix 10. The food with the highest median concentration was honey, with a concentration of 0.04 mg/kg of lead.

The majority of foods contained quantifiable concentrations of lead in at least one of the samples analysed. Olive and canola oil and white sugar were the only foods that did not have quantifiable concentrations of lead in any of the composite samples analysed.

The estimated dietary exposures to lead for each age category are provided in Table A18.1 and Table A18.2 of Appendix 18.

*Major food contributors*

As shown in Figure 13 cereal and grain-based foods, waters and fruits and nuts were the food types that were the major sources of lead dietary exposure for all age-gender groups. Infant formula was also a major source of lead dietary exposure for 9 month old infants and non-alcoholic beverages (excluding waters and milk) for those aged 17 years and above.

More specific details about the major contributing foods to lead dietary exposures are presented in Table A21.5 of Appendix 21. In this study, water (non-bottled) or beverages reconstituted with water were the major source of lead exposure for all age groups. Water (non-bottled) contributed to 14% of total lead intake for children aged 2-5 years, 6-12 years and 13-16 years and was also a major contributor to lead exposure in other age groups. For 9 month old infants, infant formula contributed to 28% of total lead exposure. For those 17 years and above, coffee (from ground) contributed to 15% of total lead exposure.

*Figure 13: Major food contributors to lead dietary exposures*
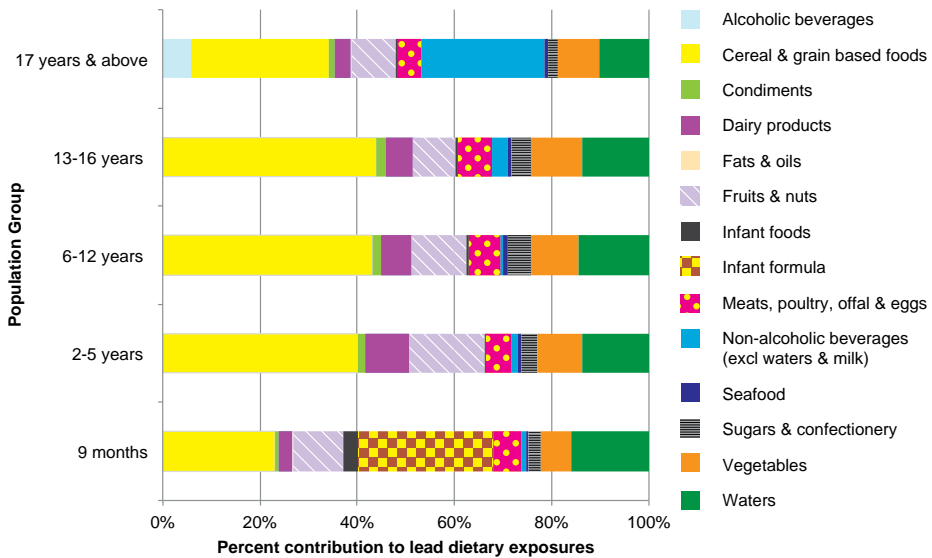
*Risk characterisation*

In 2010, JECFA concluded that for children aged 1-4 years of age, a lead exposure of 0.3 µg/kg bw/day may result in a population decrease of 0.5 IQ points, while an exposure of 0.6 µg/kg bw/day may result in a population decrease of 1 IQ points. For adults, an exposure of 1.2 µg/kg bw/day may result in a population increase in systolic blood pressure of 1 mm Hg. Safe intakes of lead for 9 month old infants were not calculated. As described previously (see under 'Reference health standards for nutrients') these dose estimates (or reference points) are not reference health standards but approximate estimates of where the risk of an adverse effect is considered to be low. Hence if the exposure is equal or lower than this level, the risk is considered to be acceptably low (i.e. MOE >1).

An inspection of dietary lead exposure (assigning a zero value where there were no detections) for the Australian population, presented in Table 4, reveals that the mean 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.  It is important to recognise that, in contrast to a reference health standard, it is not appropriate to compare a 90th percentile dietary exposure against the dose-response curve that compares a population mean reduction in IQ levels with lead exposure because it would overestimate the risk for a sub-population.

Lead levels will continue to be surveyed in further ATDS. The Codex Committee on Contaminants in Food (CCCF) has a working group reviewing current MLs for lead. FSANZ will remain aware of the progress of this CCCF working group.

*Table 4: Mean and 90th percentile lead intakes*

| Age group | Mean exposure (µg/kg bw/day) | MOE at mean |
|---|---|---|
| 2-5 yrs[1] | 0.27 | 1.1 |
| 6-12 yrs[1] | 0.18 | 1.7 |
| 13-16 yrs[2] | 0.12 | 10 |
| ≥17 yrs[2] | 0.13 | 9.2 |

Results are derived from – [1] relative to the JECFA exposure of 0.3 µg/kg bw/day for 1-4 year olds; [2] relative to the JECFA exposure of 1.2 µg/kg bw/day for adults

*Comparisons with previous studies*

Dietary lead exposures reported in both the 19th and 20th ATDS were below the then JECFA assigned PTWI of 25 µg/kg bw.

It is re-assuring that over the last decade since the 19th ATDS (2001), the dietary exposure to lead from food for the Australian population has been reduced approximately 7-fold (i.e. 1.92 to 0.27 µg/kg bw/day) as a result of a range of effective risk management strategies.

*Summary table*

| Contaminant | Reference health standard | Adverse effect | Major food contributors | Risk characterisation |
|---|---|---|---|---|
| Lead | None^ | • Reduction in IQ points in children<br><br>• Increase in systolic blood pressure in adults | • Water (non -bottled) and beverages reconstituted with water<br><br>• Ground coffee | • Estimated mean dietary exposures to lead were below the level considered by JECFA to have a low risk of reducing the population IQ for children or increasing the systolic blood pressure in adults |

^ Risk assessment based on the MOE approach

**Mercury**

*Adverse effects*

The JECFA assigned reference health standard for methylmercury is a PTWI of 1.6 µg/kg bw and is based on the estimated exposure that would be expected to have no appreciable adverse effects on children (WHO, 2004).

The ability of methylmercury to cross the blood-brain barrier and blood-placenta barrier, places vulnerable groups such as pregnant women, infants and children at particular risk of adverse health effects (WHO, 2007).

For inorganic mercury, the PTWI of 4 µg/kg bw is based on adverse effects on the kidneys in laboratory animals (WHO, 2011b).

*Analytical and dietary exposure results*

Mercury was analysed in all foods sampled and included both inorganic and organic mercury components. Analysis of methylmercury (the organic form) and inorganic mercury was undertaken in seafood samples only, noting that almost all dietary exposure to methylmercury is from fish and seafood. For all other foods the total mercury content was assumed to be comprised entirely of the inorganic mercury component.

The mean, median, maximum, and minimum concentrations of inorganic mercury, total mercury and methylmercury in foods analysed are presented in Table A10.6, Table A10.7 and Table A10.8 of Appendix 10.

Mercury was detected in all but three foods (full strength beer, canola and olive oil composite and full fat soy beverage). The food with the highest median total mercury concentration was battered fish fillets, with a concentration of 0.14 mg/kg.

Low concentrations of methylmercury were found in all of the seafood samples. Concentrations ranged from 0.014 to 0.11 mg/kg at the median concentration for prawns (only type of shell fish analysed) and battered fish fillets, respectively. For inorganic mercury, very low concentrations were found in all seafood samples. Concentrations ranged from 0.011 to 0.020 mg/kg at the median concentration for prawns and frozen fish portions, respectively.

*Inorganic mercury*

The estimated dietary exposures to inorganic mercury for each age group are presented in Table A18.1 and Table A18.2 of Appendix 18. Figure 14 indicates that dietary exposures for all age groups at the 90th percentile were below the PTWI of 4 µg/kg of bw, and were consequently within acceptable safety standards. The highest exposure at the 90th percentile to inorganic mercury was for 9 month old infants, with 90th percentile dietary exposure estimated to be 25-40% of the PTWI. Dietary exposures for the 2-5 year old age group were estimated to be 20-30% of the PTWI.

*Figure 14: Range of mean and 90th percentile estimated dietary exposure to inorganic mercury, as a percentage of the PTWI§*
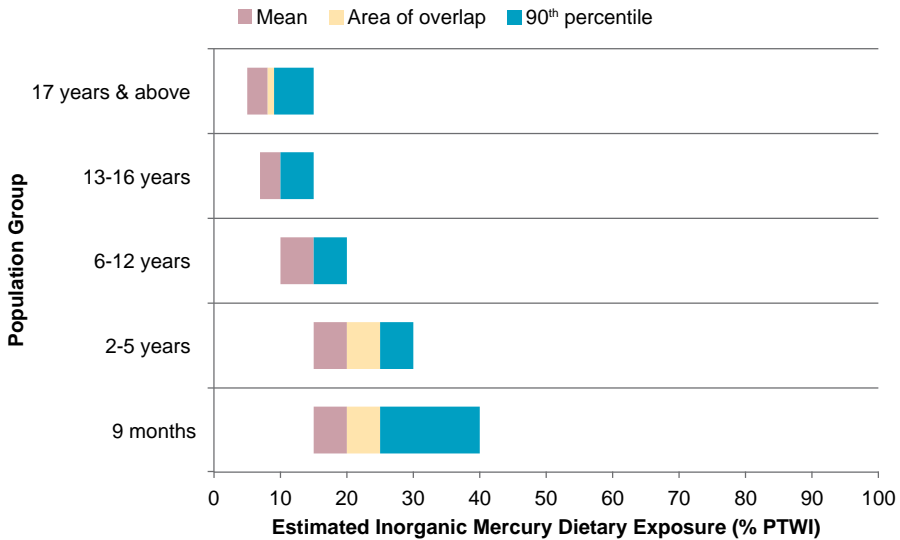


§ Lower end of the range represents where all 'not detected' analytical results have been assigned a concentration of zero; the upper end of the range represents where all 'not detected' analytical results have been assigned a concentration equal to the LOR.

Note: the upper end of the mean range and the lower end of the 90th percentile range overlap. This area of overlap is represented by the colour green on the figure.

*Major food contributors*

Figure 15 indicates that cereal and grain based foods, fruits and nuts and vegetables were major sources of inorganic mercury dietary exposure for all age-gender groups. Infant formula was also a major source of inorganic mercury dietary exposure for 9 month old infants.

More specific details about the major contributing foods to inorganic mercury dietary exposures are presented in Table A21.6 of Appendix 21. For all population groups aged 2 years and above, the food group white breads (including high-fibre white) was the greatest contributor (11-13%) to inorganic mercury exposure. For 9 month old infants, infant formulas were the major contributor to dietary exposure, contributing to 22% of total exposure. Another major source of dietary exposure for all age groups was water (non-bottled), contributing to approximately 10% of exposure.

*Figure 15: Major food contributors to inorganic mercury dietary exposures*
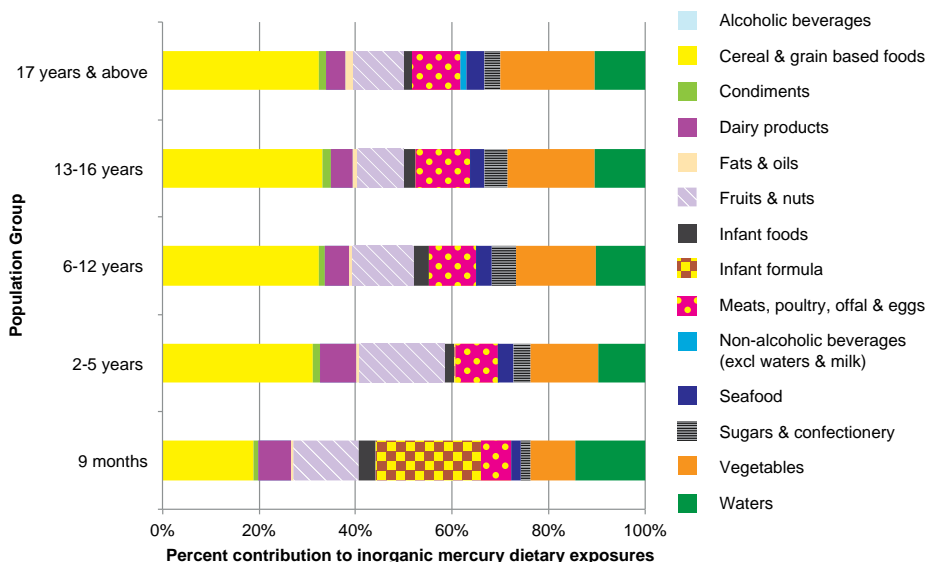


### Risk characterisation

Dietary exposures to inorganic mercury were below the respective reference health standard. On this basis, there are no public health and safety issues with regard to current exposure to inorganic mercury by Australian consumers.

### Comparisons with previous studies

Total mercury, rather than inorganic mercury, was investigated in the 19th and 20th ATDS. In the 19th ATDS, mean levels of dietary exposure exceeded the assigned reference health standard (5 µg/kg bw/week) for two year olds and 9 month old infants, where exposure ranged from 2% up to 150% of the tolerable limit. However, it is likely that limitations with the analytical method were responsible for the large range in results obtained, extending above the reference health standard. In the 20th ATDS, the estimated mean dietary exposures were below the tolerable limit for all age-gender groups. Refinements to the analytical methods and subsequently a lower limit of reporting assisted in producing more refined exposure estimates.

*Methylmercury*

The estimated dietary exposures to methylmercury for each age category are presented in Table A18.1 and Table A18.2 of Appendix 18.

Figure 16 indicates that estimated dietary exposures to methylmercury are below the PTWI of 1.6 µg/kg bw for all age groups at the 90th percentile, and consequently within acceptable safety standards. The highest level of exposure was for 2-5 year olds at 80% of the PTWI, because of their high food consumption relative to body weight.

**Figure 16: Range of mean and 90th percentile estimated dietary exposure to methylmercury, as a percentage of the PTWI§**
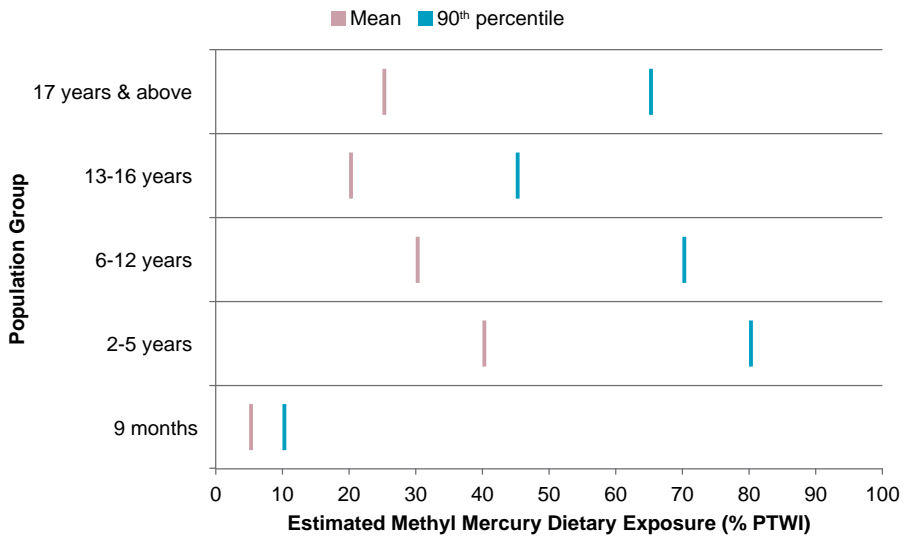


§ Lower end of the range represents where all 'not detected' analytical results have been assigned a concentration of zero; the upper end of the range represents where all 'not detected' analytical results have been assigned a concentration equal to the LOR.

*Major food contributors*

Seafoods were major sources of methylmercury exposure for all age groups. Fish (uncrumbed/ unbattered or canned) contributed 42-53% of total methylmercury exposure in children 12 years and younger. Battered seafood contributed 41-44% of total methylmercury exposure for those aged 13 years and over.

Major food contributors to dietary exposure of methylmercury are presented in Table A21.7 of Appendix 21.

*Risk characterisation*

Dietary exposure to methylmercury for all groups was below the respective reference health standard. On this basis, there is no human health and safety risk with regard to current intakes of methylmercury by Australian consumers. Due to the potential adverse effects of methylmercury on vulnerable population groups, such as pregnant women and young children, methylmercury will continue to be monitored in future studies.

*Comparisons with previous studies*

In the 20th ATDS, low concentrations of methylmercury were found in fish portions and canned tuna, but not in fish fillets and prawns. This difference in the results from the 20th ATDS in comparison to the 23rd ATDS is likely to be attributed to sample and species variability.

Estimated dietary exposures to methylmercury as a separate component of total mercury were not investigated in the 19th ATDS. Therefore it is not possible to comment on any differences in estimated dietary exposures over time.

*Summary table*

| Contaminant | Reference health standard | Adverse effect | Major food contributors | Risk characterisation |
|---|---|---|---|---|
| Inorganic mercury | 4 µg/kg bw (PTWI) | • Adverse effects on kidneys in laboratory animals | • White breads (including high-fibre white) <br><br> • Water (non-bottled) | • Dietary exposures to inorganic mercury and methylmercury were below the reference health standards for all age-gender-groups |
| Methylmercury | 1.6 µg/kg bw (PTWI) | • Adverse effects on neurode-velopment in children | • Fish (uncrumbed/ unbattered or canned) <br><br> • Battered seafood | • On this basisthere is no human health and safety risk |

**Strontium**

*Adverse effects*

The JECFA assigned reference health standard for strontium is a PMTDI of 0.13 mg/kg bw and is based on thyroid enlargement in rats (WHO 2010a).

Excess exposure to strontium can affect bone mineralisation and therefore may impact on skeletal development, particularly in vulnerable groups such as infants and children (WHO 2010a).

*Analytical and dietary exposure results*

The mean, median, maximum and minimum concentrations of strontium found in the foods analysed are provided in Table A10.9 of Appendix 10. The food with the highest median strontium concentration was almonds, with a concentration of 24.7 mg/kg.

Strontium was detected in all but one food (white sugar). For the majority of foods, quantifiable concentrations of strontium were detected in more than half of the analysed samples.

The estimated dietary exposures to strontium for each age category are presented in Table A18.1 and Table A18.2 of Appendix 18.

Figure 17 indicates that dietary exposures to strontium for all age groups at the 90th percentile of exposure were below the PMTDI of 0.13 mg/kg of bw/day, and consequently within acceptable safety standards. The highest 90th percentile dietary exposure to strontium was for 9 month old infants, followed by 2-5 year olds because of their high food consumption relative to body weight. The estimated exposures were 85-90% and 80% of the PMTDI, respectively.

*Figure 17: Range of mean and 90th percentile estimated dietary exposure to strontium, as a percentage of the PMTDI[§]*
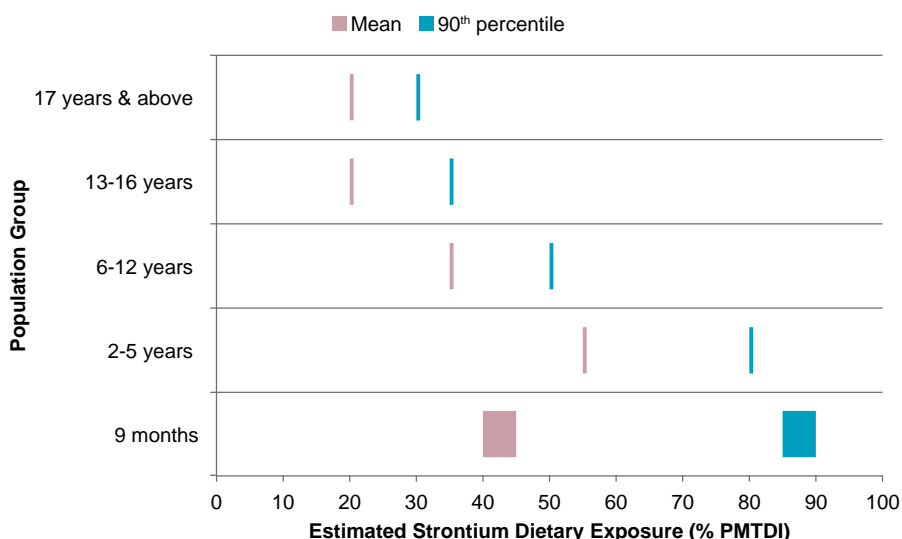


§ Lower end of the range represents where all 'not detected' analytical results have been assigned a concentration of zero; the upper end of the range represents where all 'not detected' analytical results have been assigned a concentration equal to the LOR.

### Major food contributors

Figure 18 illustrates that cereal and grain based foods, dairy products, fruits and nuts and vegetables were major sources of strontium dietary exposure for all age-gender groups. Infant formula was also a major source of strontium dietary exposure for 9 month old infants.

More specific details about the major contributing foods to strontium dietary exposures are presented in Table A21.8 of Appendix 21. The food group 'milks and cream' was the major source of strontium dietary exposure for children aged 2-5 years, 6-12 years and 13-16 years. This food group contributed 11-15% of total exposure. For 9 month old infants, infant formulas were the major source of total strontium dietary exposure, contributing to 36% of exposure. For adults 17 years and above, the major source of exposure was the food group coffee (from instant) and cereal-based beverages, which contributed 10% of dietary exposure. This was closely followed by the food groups, 'milks and cream', and white breads (including high-fibre white), both contributing 7% of total strontium dietary exposure for those over 17 years.

*Figure 18: Major food contributors to strontium dietary exposures*
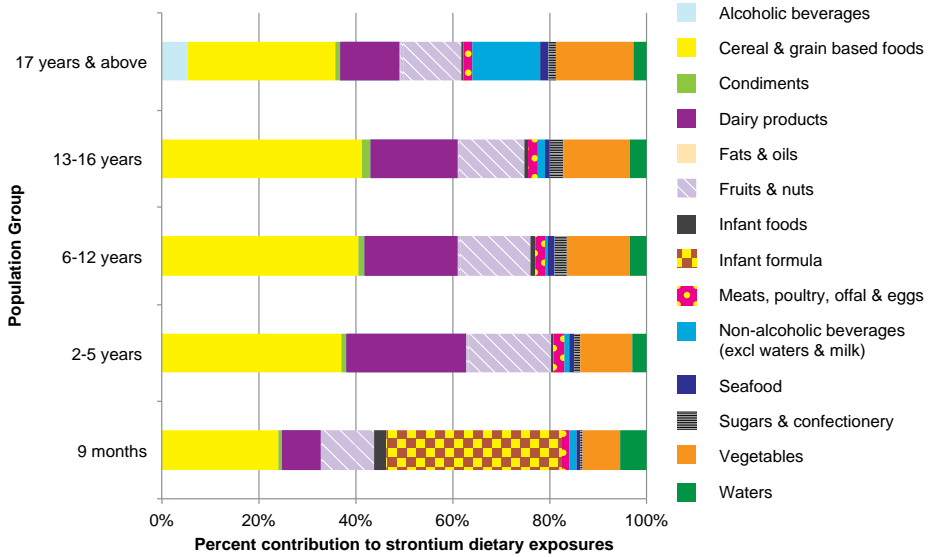


### Risk characterisation

Dietary exposures to strontium were below the respective reference health standard. On this basis, there are no human health and safety risk with regard to current dietary exposures to strontium by Australian consumers.

### Comparisons with previous studies

Strontium was not investigated in the 19th or 20th ATDS hence it is not possible to comment on any differences in estimated dietary exposures over time.

*Summary table*

| Contaminant | Reference health standard | Adverse effect | Major food contributors | Risk characterisation |
|---|---|---|---|---|
| Strontium | 0.13 mg/kg bw (PMTDI) | • Thyroid enlargement in rats | • Milks and cream<br><br>• Instant coffee and cereal based beverages<br><br>• White breads (including high-fibre white) | • Estimated dietary exposures to strontium were below the reference health standard for all age-gender groups<br><br>• Based on this there are no human health and safety risks |

**Vanadium**

*Adverse effects*

There is currently no JECFA assigned reference health standard for vanadium as there is no safe level of exposure that can be established (WHO, 2001a).

*Analytical and dietary exposure results*

There were no detections of vanadium in any of the foods analysed in this study. Therefore estimates of dietary exposure and a subsequent risk characterisation were not conducted.

## Nutrient results and risk characterisation

This section contains information on the estimated dietary intakes of the following eleven nutrients examined in this study: calcium, chromium, cobalt, copper, fluoride, iron, manganese, molybdenum, potassium, selenium and zinc.

The concentrations of nutrients reported in the surveyed foods are presented in Appendix 11 with each nutrient reported separately. The estimated dietary exposures to each nutrient are presented in Appendix 19. Major food contributors to dietary intake of each nutrient are presented in Appendix 22 for all age-gender groups.

Major contributors to nutrient intake have also been presented below in Figure 19 to Figure 29. The food groups presented in Appendix 22 have been categorised into 14 major commodities such as 'cereal and grain-based' foods, dairy products, vegetables etc, providing a simpler pictorial representation of the contributors to nutrient intakes across the food supply.

In this study of nutrients, both essentiality and toxicity needed to be considered. Therefore, an assessment of dietary intake investigated:

- The estimated dietary intakes of nutrients, calculated using mean concentration data, at the mean, 5th and 95th percentile levels of intake (in mg or µg/day) (Appendix 19).

- The proportion of the population with dietary intakes below the EAR (where established) and above the UL (where assigned), calculated using mean concentration data.

It should be noted that for nutrients without established EARs, mean estimated dietary intakes have been compared with AIs (where assigned). This is applicable for chromium, cobalt, copper, fluoride, manganese and potassium. For calcium, iron and zinc, EARs have not been reported as intake estimates have been published in the 1995 NNS and 2007 NNS which include the nutrient profile and consumption for thousands of individual foods. Given the smaller number of foods in this study, an updated assessment of the proportion of the population with dietary intakes below the EAR for these nutrients cannot be determined.

### Calcium

*Health effects*

High intakes of calcium may decrease the absorption of iron and zinc in vulnerable populations (Institute of Medicine, 2006). The adverse health effects of excessive calcium intakes have been observed only when calcium is given in high doses in supplemental form as calcium carbonate. Toxic effects can include kidney stones and milk alkali syndrome (which is characterised by hypercalcaemia with renal calcification and failure) (Institute of Medicine, 2006).

The UL for calcium is 2,500 mg/day for all age-gender groups, excluding infants aged 7-12 months where no UL has been established. The UL for calcium  is based on the above observations of adverse effects on the kidneys (NHMRC, 2006).

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of calcium in the foods analysed are presented in Table A11.1 of Appendix 11. The food with the highest mean calcium content was cheddar cheese with a concentration of 8,000 mg/kg.

All of the foods analysed in this study contained quantifiable concentrations of calcium at an LOR ranging from 0.1-10 mg/kg, dependent on the food matrix type. In addition to cheddar cheese, other dairy products, such as milk, yoghurt and ice cream, contained the highest concentrations of calcium, as did almonds, milk chocolate, pizza and breakfast cereals. Of the foods surveyed in this study, soft drinks, water and olive oil contained the lowest concentrations.

The estimated calcium intakes for all age-gender groups assessed are presented in Table A19.1 of Appendix 19. For all age groups, males had higher mean calcium intakes than females. Among males, mean intake was highest in those aged 17-18 years (1,220 mg/day). Intake was relatively constant with age for females (2 years and above), with mean consumption at its highest point for 14-16 year olds (82 mg/day), declining slightly for 50-69 year olds and those over 70 years of age.

*Major food contributors*

Figure 19 illustrates that dairy products and cereal and grain based foods were major sources of calcium intake for all age-gender groups. This is with the exception of 9 month old infants, where infant formula was the major source of calcium intake.

More specific details regarding the major food group contributors to calcium intake are presented in Table A22.1 and Table A22.2 of Appendix 22. The food group 'milks and cream' contributed to between 32–50% of total calcium intake in the diets of those two years and over. Cheeses were the next major source of calcium intake, contributing up to 20% of total intake. Infant formula contributed 73% of total intake of the diets of 9 month old infants.

*Figure 19: Major food contributors to calcium dietary intakes*
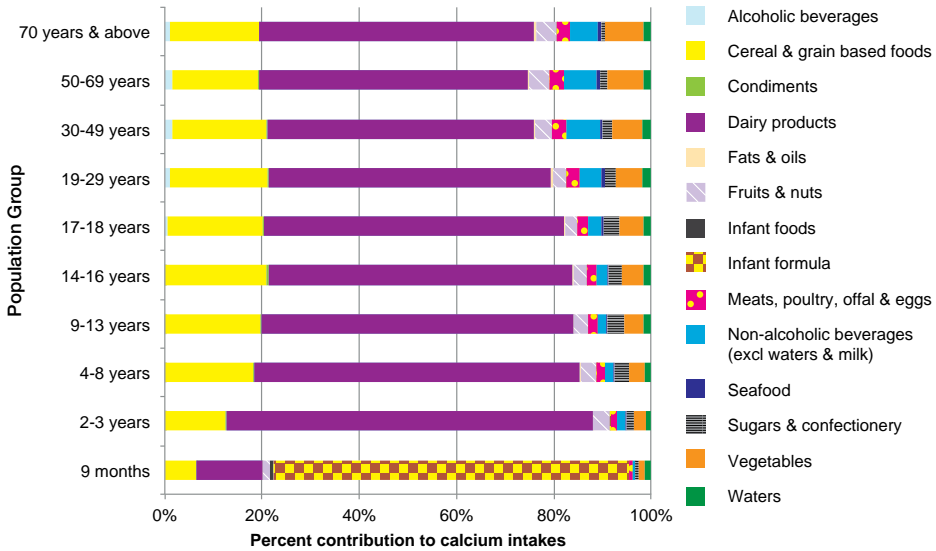


*Risk characterisation*

There is no established UL for calcium for infants in Australia and therefore no risk characterisation was undertaken for this group.

A small proportion of respondents in the 17-18 year group (2% of males and 1% of females) and the 19-29 year group (2% of males) exceeded the UL for calcium (Table 5). The intake of the majority of these respondents exceeded the UL by only a small amount. As the UL for calcium is based on adverse effects (hypercalcaemia and renal calcification) resulting from the ingestion of calcium-containing antacid medication, it is a conservative limit for dietary risk assessment purposes. Indeed there is little expectation that adverse effects could occur through normal dietary exposure to calcium (NHMRC, 2006). On this basis, the exceedances of the UL for calcium in a small proportion of respondents are not considered to represent a human health and safety risk.

*Table 5: Estimated proportion of the population with dietary calcium intakes above the UL*

| Age | Gender | Estimated proportion with dietary calcium intakes >UL (%) ^ |
|---|---|---|
| 2-3 years¤ | Male<br>Female | 0<br><1 |
| 4-8 years¤ | Male<br>Female | 0<br>0 |
| 9-13 years¤ | Male<br>Female | <1<br>0 |
| 14-16 years¤ | Male<br>Female | <1<br>0 |
| 17-18 years* | Male<br>Female | 2<br>1 |
| 19-29 years* | Male<br>Female | 2<br><1 |
| 30-49 years* | Male<br>Female | <1<br>0 |
| 50-69 years* | Male<br>Female | <1<br><1 |
| 70 years & above* | Male<br>Female | 0<br>0 |

^ does not include the contribution from supplements
¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake)
* derived using the Australian 1995 National Nutrition Survey

*Comparisons with previous studies*

As this is the first ATDS to study intake estimates of calcium, no comparisons can be made with previous studies.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Calcium | • Development and maintenance of bone and teeth structure<br><br>• Neural transmission, muscle contraction and cardiac function | • Milks and cream<br><br>• Cheeses | • UL 2,500 mg/day | • There is little expectation that adverse effects could occur through normal dietary exposure to calcium<br><br>• Exceedance of the UL for calcium only in a small number of respondents |

*^ UL for all age-gender groups, excluding infants aged 7-12 months where no UL has been established.*

## Chromium

*Health effects*

Chromium deficiency is rare and has been reported only in patients who are fed intravenously for a long period, where symptoms included nerve and brain disorders, impaired glucose tolerance, insulin resistance and weight loss (Mertz, 1993; NHMRC 2006).

It has been suggested that tissue levels of chromium are lower in individuals with diabetes than in healthy controls (Gunton *et al*., 2005).

Chromium has low toxicity in foods in part due to its low bioavailability; however, adequate data on excess intakes of trivalent chromium are limited (Institute of Medicine, 2006; NHMRC, 2006). No adverse effects were reported in a review of 19 randomised controlled trials in humans, in which individuals received 175-1000 μg oral trivalent chromium per day for durations of between 6 and 64 weeks. However, not all of the studies reported adverse health effects specifically (Jeejeebhoy, 1999).

The AIs for chromium range from 5.5-35 μg/day based on different age-gender groups. There are currently no established EARs or ULs for chromium in Australia.

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of chromium in the foods analysed are presented in Table A11.2 of Appendix 11. The food with the highest mean chromium content was mango with a concentration of 0.41 mg/kg.

For the majority of foods, a considerable proportion of the analysed samples did not contain quantifiable concentrations of chromium. These included; grapes, nectarines, strawberries and watermelon. Given only one of the four mango samples contained a quantifiable chromium concentration, the results for this commodity should be considered in this context.

The estimated chromium intakes for all age-gender groups assessed are presented in Table A19.2 of Appendix 19. For all age groups, males had higher mean chromium intakes than females. Mean intake was highest for males aged 17-18 and 19-29 years (146 µg/day) and also for females aged 17-18 and 19-29 years (99 µg/day).

*Major food contributors*

Figure 20 illustrates that cereal and grain based foods, dairy products, meats, poultry, offal, eggs, fruits and nuts, non-alcoholic beverages and vegetables were major sources of chromium intake across different age-gender groups. This is with the exception of 9 month olds where, although 'cereal and grain-based foods' were also a significant source, infant formula was the major source of intake.

More specific details regarding the major food group contributors to chromium intake are presented in Table A22.3 and Table A22.4 of Appendix 22. Infant formula contributed to 41% of total chromium intake in 9 month old infants. For those aged 2-3 years, the major source was the food group 'milks and creams', contributing to 14% of intake. This food group also featured as a prominent source of chromium intake across the other age-gender groups. However, for those aged 4 and above, the food groups 'white breads (including high-fibre white)', and 'multigrain, wholemeal and rye breads' were the major sources of chromium intake. For older women, tea contributed to 7% of total chromium intake.

*Figure 20: Major food contributors to chromium dietary intakes*
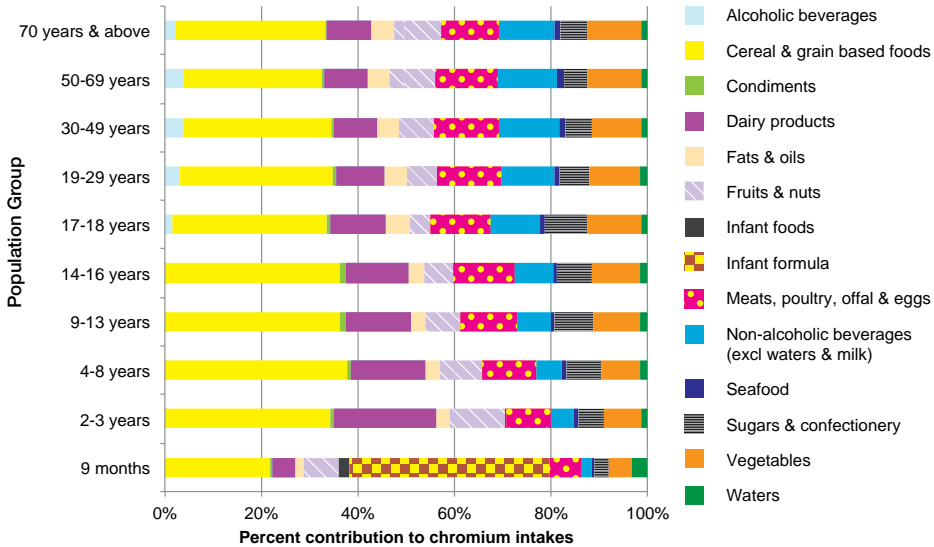


### Risk characterisation

All age-gender population groups had estimated mean dietary intakes of chromium that exceeded their respective AIs (Table A16.1of Appendix 16 and Table A19.2 of Appendix 19).

Given the limitations of the ATDS methodology and those associated with the AI together with the lack of an established EAR for chromium in Australia, conclusions cannot be drawn from this study as to the adequacy of chromium intake in the population.

No ULs for chromium have been established because of insufficient data.

### Comparisons with previous studies

In the present study, mean chromium intakes were noticeably higher than those reported in the 22nd ATDS (FSANZ, 2008). In the former study, the mean estimated chromium intakes for 9 month old infants and children 2-8 years of age were similar to, or higher than, their respective AIs. In contrast, most population groups aged 9 years and above had mean estimated intakes below their respective AIs in the 22nd ATDS. The chromium dietary exposures estimated in this study are higher than those in the 22nd ATDS due to the higher mean concentrations of chromium found in analysed samples such as milk, breads, beef, chicken, apples, bananas, oranges, potatoes, carrots, eggs and rice.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Chromium | • An essential element important for the action of insulin | • Milks and cream<br><br>• White breads (including high-fibre white)<br><br>• Multigrain, wholemeal and rye breads | • AIs 5.5-35 µg/day<br><br>• No EARs or ULs are established for chromium | • Due to the absence of EARs for chromium, no conclusions can be made around the adequacy of dietary intake of chromium<br><br>• In the absence of established ULs, no risk characterisation has been conducted |

^ AIs based on different age-gender groups.

**Cobalt**

*Health effects*

Adverse health effects from cobalt have been observed at very high intakes. Humans ingesting cobalt chloride at 150,000 µg/day for 22 days experienced an increase in red blood cell number and haemoglobin concentration (WHO, 2006a). Case reports suggest that acute intakes of more than 30,000 µg/day of cobalt salts may cause gastrointestinal upset, skin rashes and hot flushes (WHO, 2006a). It has also been observed that chronic cobalt intakes equivalent to 10,000-234,000 µg/day for a 60 kg adult depresses iodine uptake and results in symptoms of hypothyroidism, including goitre (Expert Group on Vitamins and Minerals, 2003).

There are currently no EARs or ULs established in Australia for cobalt.

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of cobalt in the foods analysed are presented in Table A11.3 of Appendix 11. The foods with the highest cobalt concentrations were potato crisps, followed by almonds with concentrations of 0.15 and 0.14 mg/kg, respectively.

For approximately half of the foods surveyed, a considerable proportion of the analysed samples did not contain quantifiable concentrations of cobalt. These foods included; nectarines, mushrooms, grapes, soft drink, canola and olive oil, monounsaturated margarine, mango and beer.

The estimated cobalt intakes for all age-gender groups assessed are presented in Table A19.3 of Appendix 19. Intakes were similar for both males and females up until the age of 8 years, but after this age males had higher mean cobalt intakes than females. Mean intake increased for both genders with age up until 30-49 years, after which intake declined, and was highest for males aged between 19-29 and 30-49 years (33 µg/day) and for females aged 30-49 years (26 µg/day).

*Major food contributors*

Figure 21 indicates that cereal and grain based foods, non-alcoholic beverages (excluding waters and milk), vegetables and dairy products were major sources of cobalt dietary intake; however, the degree to which this was the case varied across different age-gender groups.

More specific details regarding the major food group contributors to cobalt intake are presented in Table A22.5 and Table A22.6 of Appendix 22. Infant formulas and infant cereals contributed to 38% and 9% of total intake in the diets of 9 month old infants, respectively. For those aged 2-3 years, the major source of cobalt intake was the food group 'milks and creams' (12-13% of intake). This food group also featured prominently in the diets of 4-8, 9-13 and 14-16 year olds, but to a lesser extent for those aged 17 years and above. For those aged 17 years and above, the food group coffee (from instant) and cereal-based beverages, contributed up to 28% of total cobalt dietary intake. For adults 70 years and above, tea contributed 7-8% of cobalt intake.

*Figure 21: Major food contributors to cobalt dietary intakes*
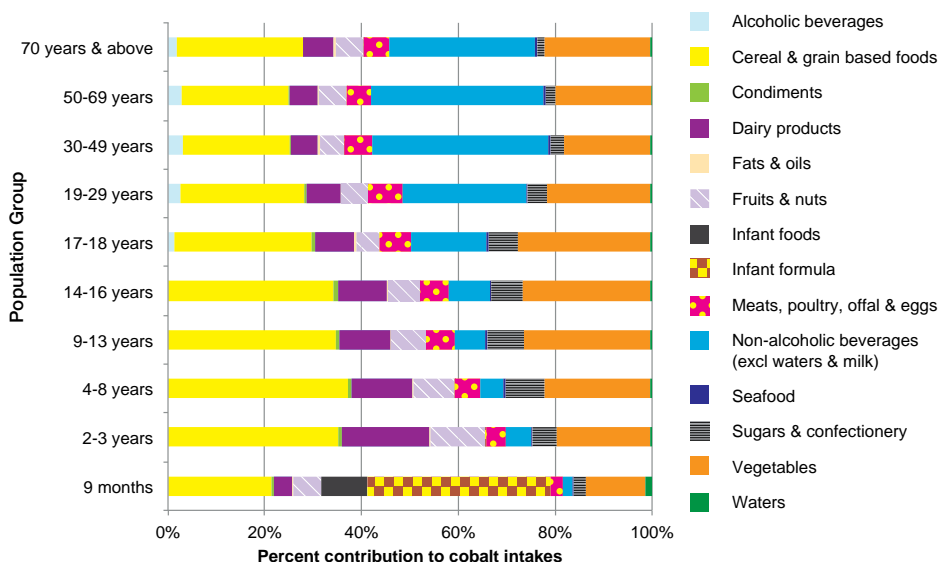


*Risk characterisation*

The estimated dietary intakes of cobalt for adults in the current study are comparable to the mean value of 39.7 µg/day estimated in a study of 10 non-vegetarian Australian adults using diet history records (Hokin *et al*., 2004b). Other studies have estimated the mean daily dietary intakes of cobalt in France (29 µg/day), the USA (5-40 µg/day), Canada (11 µg/day), and the UK (120 µg/day) (WHO, 2006a).

Given the limitations of the ATDS methodology, and those associated with the AI and that no EAR has been established for cobalt in Australia, conclusions cannot be drawn from this study as to whether intakes of cobalt in the population are adequate.

As there is currently no established UL for cobalt in Australia, no conclusions can be made about whether intakes of cobalt by the Australian population are excessive. The Expert Group on Vitamins and Minerals (2003) set a guidance value for cobalt of 0.023 mg/kg bw/day as an intake level that would not be expected to result in any adverse effects. As all mean and 95th percentile intakes are well below this value (when corrected for bodyweight), it is unlikely that current Australian intakes of cobalt are a human health and safety risk.

*Comparisons with previous studies*

As this is the first ATDS to study intake estimates of cobalt, no comparisons can be made with previous total diet studies in Australia.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard | Risk characterisation |
|----------|----------|------------------------|--------------------------|----------------------|
| Cobalt | • A constituent of vitamin $B_{12}$, which is essential for folate and fatty acid metabolism | • White breads (including high-fibre white)<br><br>• Instant coffee and cereal-based beverages<br><br>• Root vegetables | • No EARs or ULs are established for cobalt | • Given there is no UL for cobalt in Australia, no conclusions can be made as to whether intakes of cobalt by the Australian population are excessive |

## Copper

*Health effects*

Overt copper deficiency is rare in humans and results in vascular and skeletal problems and defective iron metabolism. In certain circumstances, deficiency has been observed in individuals receiving total parenteral nutrition, and in infants. Symptoms include various forms of anaemia, a decrease in the levels of specific sub-sets of white blood cells and osteoporosis in infants and growing children (NHMRC, 2006).

Although there are little data, chronic excessive intake of copper appears to be rare in people with normal copper homeostasis. Copper toxicity is uncommon and usually results from consuming contaminated food or beverages. The acute consumption of water and other beverages containing high levels of copper (>30 mg/kg) have resulted in gastrointestinal problems such as abdominal pain, cramps, nausea, vomiting and diarrhoea (Barceloux, 1999b; Institute of Medicine, 2006).

The AIs for copper range from 0.2-1.7 mg/day based on different age-gender groups. There are currently no established EARs for copper in Australia. The ULs for copper range from 1-10 mg/day for different age-gender groups. For adults, the ULs are based on adverse effects on the liver (NHMRC, 2006). The ULs for children and adolescents are derived from the adult ULs (NHMRC, 2006).

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of copper in the foods analysed are presented in Table A11.4 of Appendix 11. The foods with the highest levels of copper were prawns, almonds and desiccated coconut with mean concentrations of 8.8, 8.4 and 8.2 mg/kg, respectively.

For all of the foods analysed in this study, the vast majority of the samples contained quantifiable concentrations of copper, with the exception of white sugar.
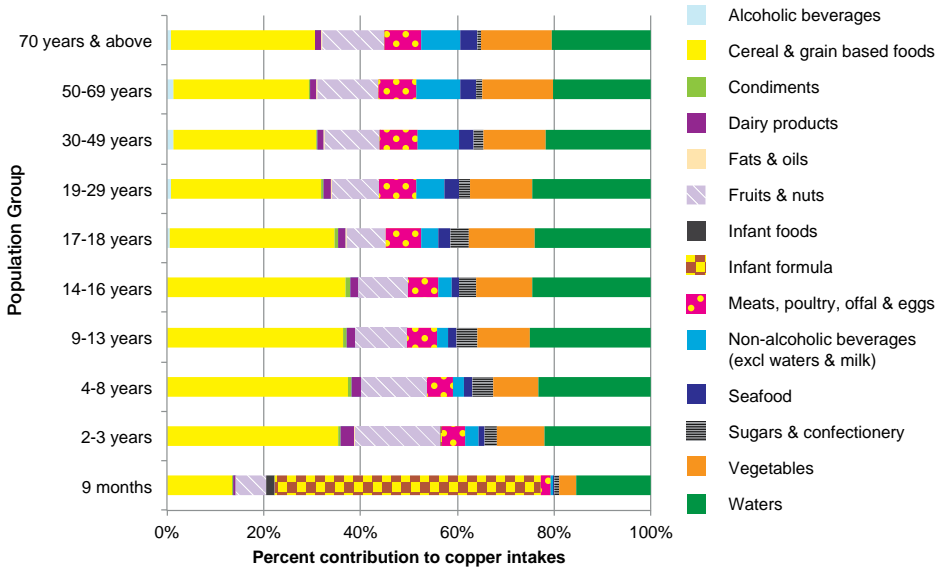
The estimated dietary copper intakes for all age-gender groups assessed are presented in Table A19.4 of Appendix 19. For all age groups, males had higher mean copper intakes than females, with this disparity more apparent after 8 years of age. Among both males and females, dietary intake was highest in those aged 19-29 years (1.89 and 1.42 mg/day, for males and females, respectively). Intake for both genders declined steadily beyond 19-29 years of age.

*Major food contributors*

Figure 22 indicates that cereal and grain-based foods, waters, vegetables, and fruits and nuts were major sources of copper intake. This is with the exception of 9 month olds, where infant formula was the predominant source of copper intake.

More specific details regarding the major food group contributors are presented in Table A22.7 and Table A22.8 of Appendix 22. The food group 'water (non-bottled)' was by far the greatest source of copper intake in the diets of all Australians, excluding infants, contributing to between 17-26% of total intake. Infant formula, followed by water (non-bottled), contributed to between 55% and 15% of total copper intake in the diets of infants, respectively, noting that infant formulas are generally reconstituted with water. The foods within the 'cereal and grain based foods' group that were major contributors included 'white breads (including high-fibre bread)', 'multigrain, wholemeal and rye breads', 'flours and single grains cereals,' and 'pasta, noodles (except rice) and couscous'.

*Figure 22: Major food contributors to copper dietary intakes*



### Risk characterisation

The mean estimated dietary intakes of copper for all age-gender population groups were similar to, or exceeded slightly, their respective AIs (Table A16.1of Appendix 16 and Table A19.4 of Appendix 19). When mean intake of a population group exceeds the AI, the expected prevalence of inadequate intake is likely to be low (Institute of Medicine, 2000).

However, given the limitations around the AI and until an EAR is developed for Australia, conclusions cannot be drawn from this study as to whether dietary intakes of copper by a significant proportion of the Australian population are adequate.

There is no established UL for copper for 9 month old infants in Australia; therefore, no risk characterisation was undertaken for this group.

In 2-3 year olds, 48% of male respondents and 32% of female respondents exceeded the UL of 1 mg/day, with the 95th percentile intake being below 1.5 mg/day (Table 6). It is important to consider that the minimum age covered by the UL (1 year) is lower than the age range of the respondents in the current survey. On this basis, the UL is likely to be a conservative estimate of maximum tolerable intake for 2-3 year olds because it is also designed to cover 1 year olds. Additional conservatism in the UL is introduced because it is extrapolated from the adult UL of 10 mg/day on the basis of relative bodyweight. The adult

UL is itself highly conservative because it is based on a study in only 7 adults where no adverse effects occurred at 10 mg/day. Therefore, there is a high degree of uncertainty as to where the maximum limit for copper resides. On this basis, the exceedance of the UL in 2-3 year olds is highly unlikely to represent an actual human health and safety risk. No other age groups exceeded their respective UL.

It is worth noting that JECFA established PMTDI of 0.5 mg/kg bw/day for copper from all sources, which preceded the establishment of the UL by the NHMRC and NZ MOH (2006). If the intake of copper for all 2-3 year old respondents is expressed on a per body weight basis (i.e., as a dose) by dividing the daily intake (mg/day) by the body weight of each child, the intakes range from 0.03 to 0.16 mg/kg bw/day, which are well below the PMTDI. This comparison re-affirms that there is no human health and safety risk associated with the current dietary intake of copper in 2-3 year olds.

*Table 6: Estimated proportion of population with dietary copper intakes above the UL*

| Age | Gender | Estimated proportion with dietary copper intakes >UL (%)^ |
|---|---|---|
| 2-3 years¤ | Male<br>Female | 48<br>32 |
| 4-8 years¤ | Male<br>Female | 0<br>0 |
| 9-13 years¤ | Male<br>Female | 0<br>0 |
| 14-16 years¤ | Male<br>Female | 0<br>0 |
| 17-18 years* | Male<br>Female | 0<br>0 |
| 19-29 years* | Male<br>Female | 0<br>0 |
| 30-49years* | Male<br>Female | 0<br>0 |
| 50-69years* | Male<br>Female | 0<br>0 |
| 70 years & above* | Male<br>Female | 0<br>0 |

^ does not include the contribution from supplements
¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted
   nutrient intake)
* derived using the Australian 1995 National Nutrition Survey

*Comparisons with previous studies*

Mean dietary copper intake for 9 month old infants, toddlers 2 years of age, males and females 12 years of age, and male and female adults 25-34 years of age were estimated in the 20th ATDS, concluding that the estimates were within acceptable health standards.

In the 20th ATDS, the highest mean copper intake was for 9 month old infants (65 µg/kg bw/day, which equates to approximately 0.59 mg/day for an infant 7-11 months of age with a standard body weight of 9 kg), attributed to their high food consumption relative to body weight.

The mean dietary copper intakes in the present study are higher than the mean copper exposures estimated in the 20th ATDS for similar age-gender groups. For example, the mean copper exposure in children 2 years of age (40 µg/kg bw/day, which equates to approximately 0.52 mg/day for children 1-3 years of age with a standard body weight of 13 kg) was approximately half of the intake estimated in the current ATDS. This difference is most likely due to water (tap or bottled) not being sampled in the 20th ATDS, as non-bottled water is the major contributing food group to copper intakes in the 23rd ATDS.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Copper | • Component of several metalloenzymes which act as oxidases in a variety of biological reactions | • Water (non-bottled)<br><br>• White breads (including high-fibre bread)<br><br>• Multigrain, wholemeal and rye breads | • AIs 0.2-1.7 mg/day<br><br>• No established EARs for copper<br><br>• ULs 1-10 mg/day<br><br>• ULs are based on adverse effects on the liver | • No conclusions can be made in relation to adequate dietary intakes of copper due to an absence of EARs<br><br>• A proportion of children aged 2-3 years exceeded the UL for copper<br><br>• Given the conservatism associated with the setting of the UL, this finding is unlikely to pose a human health and safety risk |

^ AIs and ULs based on different age-gender groups. No UL has been established for infants aged 7-12 months.

### Fluoride

*Health effects*

Excessive fluoride intake in young children can result in dental fluorosis, an effect that is characterised by a mottling of the tooth enamel. Chronic excess fluoride intake can also result in skeletal fluorosis, characterised by elevated bone ash fluoride concentrations resulting in potentially debilitating symptoms. The severity of skeletal fluorosis depends on the duration and level of fluoride exposure (Institute of Medicine, 2006).

The AIs for fluoride range from 0.5-4 mg/day and ULs for fluoride range from 0.9-10 mg/day based on different age-gender groups. There are currently no established EARs for fluoride in Australia. The ULs for fluoride in infant and children up to 8 years of age are based on the occurrence of moderate dental fluorosis (NHMRC, 2006). For older children and adults, the ULs are based on the occurrence of skeletal fluorosis (NHMRC, 2006).

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of fluoride in the foods analysed are presented in Table A11.5 of Appendix 11. The foods with the highest levels of fluoride were 'sweet, plain biscuits' and potato crisps, with mean concentrations of 8.9 and 5.2 mg/kg, respectively.

For about three-quarters of foods analysed, at least a portion of the samples did not contain quantifiable concentrations of fluoride. Bacon, honey, infant formula, mango, canola and olive oil did not contain quantifiable fluoride concentrations in any of the samples analysed.

It is important to note that since food samples were prepared in Brisbane prior to the introduction of water fluoridation, the water used in the preparation of certain food samples was unfluoridated. Those foods that were possibly impacted by the use of unfluoridated water were those that were prepared by washing in tap water (i.e. lettuce, mushrooms, and strawberries), cooking in tap water (i.e. cabbage, carrots, pasta, potatoes, pumpkin, rice) and those requiring the addition of water in their preparation (e.g. instant coffee, tea, infant formula, infant cereal, rolled oats). The use of unfluoridated water in the analysis of these samples is likely to lead to lower concentrations of fluoride in some food samples and therefore potentially underestimate dietary exposure to fluoride from these products. Please refer to Table A3.1 in Appendix 3 for further details regarding food preparation methodology prior to analysis.

The estimated fluoride intakes for all age-gender groups assessed are presented in Table A19.5 of Appendix 19. For all age groups, males had higher mean fluoride intakes than females. Among males, intake steadily increased to reach its highest level in those aged 19-29 years (3.36 mg/day), after which intake steadily declined. For females, intake steadily increased with age and was highest in those aged 50-69 years (2.80 mg/day), declining for those over 70 years of age.

*Major food contributors*

Figure 23 indicates that waters, non-alcoholic beverages (excluding waters and milk) and cereal and grain-based foods were major sources of fluoride intake. This is with the exception of 9 month olds, where infant formula was the major source of fluoride intake.

More specific details regarding the major food contributors are presented in Table A22.9 and Table A22.10 of Appendix 22. 'Water (non-bottled)' was by far the greatest source of fluoride intake for all age-gender groups up to and including those aged 30-49 years, contributing between 25-37% of total intake. For 9 month old infants, infant formulas contributed up to 20% of total intake, noting that infant formulas are generally reconstituted with water. For those aged 50 years and above, teas were high contributors to dietary intake of fluoride compared to water, contributing between 26-35% of intake, again noting that these would generally be prepared using water. The food groups, apples and quinces and fruit juice and ciders were sources of fluoride intake for those aged 16 years and under, but not in the diets of those older than 16 years. The food groups, beef, veal and venison and non-alcoholic beverages (except milk, water and juices) were sources of fluoride intake across some but not all adult age-gender groups.

*Figure 23: Major food contributors to fluoride dietary intakes*
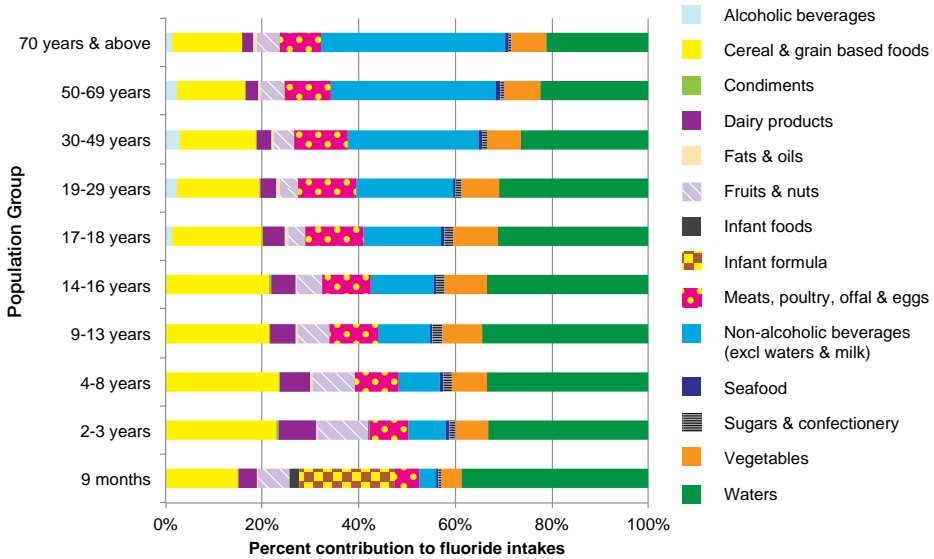


*Risk characterisation*

The mean dietary intakes exceeded the respective AIs for 9 month old infants, males and females 2-3 years and 4-8 years of age, and males aged 9-13 and 17-18 years (Table A16.1 of Appendix 16 and Table A19.5 of Appendix 19). The mean dietary intakes of fluoride for all other population groups were slightly below their respective AIs.

Due to the limitations of the ATDS methodology and those associated with the AI, and given no EAR has been established for fluoride in Australia, conclusions cannot be drawn from this study as to whether intakes of fluoride in the population are adequate.

The UL for fluoride (0.9 mg/day) was exceeded in 9 month old infants at the 95th percentile (150% of the UL) (Table 7). In 2-3 year olds the UL of 1.3 mg/day was exceeded in 77% and 68% of males and females, respectively, while the UL of 2.2 mg/day was exceeded in 15% and 7% of 4-8 year old males and females, respectively (Table 8). The 95th percentile of intake was around 1.6 times the UL for 2-3 year olds and 1.1 times the UL for 4-8 year olds. A very small proportion (<1%) of males aged 30-49 years and females aged 50-69 years exceeded the UL for fluoride intake.

*Table 7: Estimated 95th percentile dietary fluoride intake for 9 month old infants, as a %UL*

| Age | Estimated 95th percentile dietary fluoride intake[x] | |
| --- | --- | --- |
| | mg/day | %UL |
| **9 months** | 1.37 | 150 |

[x] Derived using a model diet

FSANZ recently evaluated the UL for fluoride in Application A588 – Voluntary Addition of Fluoride to Packaged Water (FSANZ, 2009). As part of the dietary exposure assessment, prepared in relation to this Application, a proportion of infants and children up to 8 years of age were found to have fluoride intakes greater than the UL. However, these exceedances were not considered to represent a safety issue on the basis of the following:

- Moderate dental fluorosis (the adverse effect on which the UL is based) is a rare condition in Australia and New Zealand.

- The apparent exceedances were considered to be the result of comparing values based on actual consumption data for children up to 8 years of age to a UL that was originally based on model data for these ages.

FSANZ is of the view that the current UL values provide a theoretical estimate of fluoride intake because they are not based on actual food consumption data. The apparent discordance between the theoretical and actual intakes without an increase in adverse clinical signs of moderate dental fluorosis suggests that the existing UL may need to be revised and increased. Indeed, FSANZ is currently working with the NHMRC to review the current UL for fluoride.

In light of the above considerations, the apparent exceedances of the UL in infants and children in the current survey in the absence of an increase in moderate dental fluorosis are not considered to represent a human health and safety risk.

*Table 8: Estimated proportion of population with dietary fluoride intakes above the UL*

| Age | Gender | Estimated proportion with dietary fluoride intakes >UL (%)^ |
|---|---|---|
| **2-3 years□** | Male<br>Female | 77<br>68 |
| **4-8 years□** | Male<br>Female | 15<br>7 |
| **9-13 years□** | Male<br>Female | 0<br>0 |
| **14-16 years□** | Male<br>Female | 0<br>0 |
| **17-18 years*** | Male<br>Female | 0<br>0 |
| **19-29 years*** | Male<br>Female | 0<br>0 |
| **30-49 years*** | Male<br>Female | <1<br>0 |
| **50-69 years*** | Male<br>Female | 0<br><1 |
| **70 years & above*** | Male<br>Female | 0<br>0 |

^ does not include the use of supplements

□ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake)

* derived using the Australian 1995 National Nutrition Survey

*Comparisons with previous studies*

As this is the first ATDS to study intake estimates of fluoride, no comparisons can be made with previous studies.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Fluoride | • Mineralisation of teeth and bones<br><br>• Stimulation of new bone formation | • Water (non-bottled)<br><br>• Tea | • AIs 0.5-4 mg/day<br><br>• No established EARs for fluoride<br><br>• ULs 0.9-10 mg/day<br><br>• ULs are based on the occurrence of moderate enamel fluorosis and skeletal fluorosis | • No conclusions can be made in relation to adequate dietary intakes of fluoride due to an absence of EARs<br><br>• A proportion of 9 month old infants, children aged 2-3 years and 4-8 years exceeded respective ULs for fluoride<br><br>• Given the basis of the current UL for fluoride, these exceedances are not considered to be a human health and safety risk |

^ AIs and ULs based on different age-gender groups.

**Iron**

*Health effects*

In the general population, the risk of adverse health effects from excessive dietary intake of iron appears to be low. Depending on the amount of iron absorbed, adverse health effects can range from gastrointestinal upset to systemic toxicity. Some individuals with certain conditions (such as haemochromatosis) are particularly susceptible to the adverse effects of excess iron intake (NHMRC, 2006).

The ULs for iron range from 20-45 mg/day for different age-gender groups. For adults, the UL is based on adverse gastrointestinal effects (NHMRC, 2006). For infants and children, the ULs are derived from the adult ULs (NHMRC, 2006).

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of iron in the foods analysed are presented in Table A11.6 of Appendix 11.  The foods with the highest levels of iron were breakfast cereals (both mixed grain and single grain types), with mean concentrations of 120 and 98 mg/kg, respectively. It should be noted that these foods typically contain added iron.

All foods contained quantifiable concentrations of iron. This is with the exception of bottled water, where quantifiable concentrations of iron were not found in any of the composite samples analysed.

The estimated iron intakes for all age-gender groups assessed are presented in Table A19.6 of Appendix 19. For all age groups, males had considerably higher mean iron intakes than females, with this disparity more apparent after 8 years of age. Among males, mean intake was highest in those aged 17-18 and 19-29 years (15.3 and 14.8 mg/day, respectively). Among females, intake was highest in those aged 19-29 years (9.7 mg/day). Intake declined slightly from 30 years of age for both genders.

*Major food contributors*

Figure 24 illustrates that major sources of iron dietary intake were 'cereal and grain-based foods', 'meats, poultry, offal and eggs', and vegetables; infant formula was the main contributor for 9 month old infants.

More specific details regarding the major food contributors to iron dietary intake are presented in Table A22.11 and Table A22.12 of Appendix 22. For 'cereal and grain- based foods', the food group 'flours and single grain cereals' contributed to between 13-30% of total dietary iron intake to the diets of all Australians apart from 9 month old infants. Other major contributors were 'breakfast cereals with mixed grains/fruits/nuts', 'white breads (including high-fibre white)', and 'multigrain, wholemeal and rye breads'. For 9 month old infants, infant formula and infant cereals contributed to 68% and 9% of total iron intake, respectively.

Although meat is generally considered to be a good source of readily absorbed iron, the only major food group contributor derived from an animal source was 'beef, veal and venison', contributing between 6-14% of total iron intake for all age-gender groups.

*Figure 24: Major food contributors to iron dietary intakes*
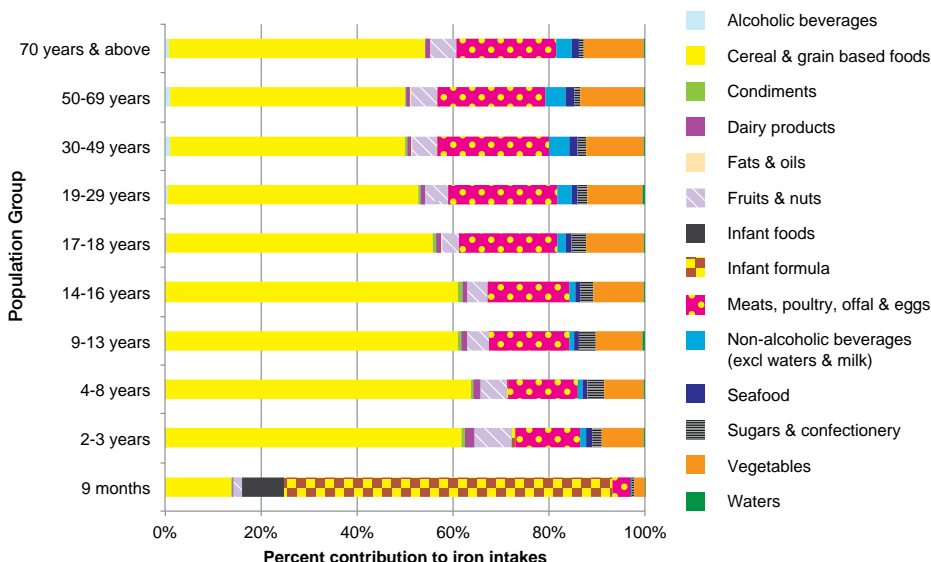


*Risk characterisation*

The ULs for all population groups were generally not exceeded at the mean and 95th percentile of intake. The UL for iron at the 95th percentile for 9 month old infants was 85% (Table 9). A very small proportion (<1%) of both males and females aged 2-3 years and males aged 19-29 years exceeded their respective ULs for iron intake (Table 10). On this basis, there is no evidence to suggest that the dietary iron intake of the Australian population is excessive.

*Table 9: Estimated 95th percentile dietary iron intake for 9 month old infants, as a %UL*

| Age | Estimated 95th percentile dietary iron intake | |
|---|---|---|
| | mg/day | %UL |
| **9 months** | 16.6 | 85 |

[x] Derived using a model diet

*Table 10: Estimated proportion of the population with dietary iron intakes above the UL*

| Age | Gender | Estimated proportion with dietary iron intakes >UL (%)^ |
|---|---|---|
| 2-3 years¤ | Male | <1 |
| | Female | <1 |
| 4-8 years¤ | Male | 0 |
| | Female | 0 |
| 9-13 years¤ | Male | 0 |
| | Female | 0 |
| 14-16 years¤ | Male | 0 |
| | Female | 0 |
| 17-18 years* | Male | 0 |
| | Female | 0 |
| 19-29 years* | Male | <1 |
| | Female | 0 |
| 30-49 years* | Male | 0 |
| | Female | 0 |
| 50-69 years* | Male | 0 |
| | Female | 0 |
| 70 years & above* | Male | 0 |
| | Female | 0 |

^ does not include the use of supplements.
¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake).
* derived using the Australian 1995 National Nutrition Survey.

*Comparisons with previous studies*

As this is the first ATDS to study intake estimates of iron, no comparisons can be made with previous studies.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Iron | • Component of several proteins, including haemoglobin, and several enzymes | • Flours and single grain cereals<br><br>• Breakfast cereals with mixed grains/ fruits/nuts<br><br>• Beef, veal and venison | • ULs 20-45 mg/day<br><br>• ULs are based on adverse gastrointestinal effects | • A very small proportion (<1%) of respondents exceeded the respective ULs for dietary iron intake<br><br>• This finding suggests that there is no human health and safety risk around excessive intake of dietary iron |

^ ULs based on different age-gender groups.

**Manganese**

*Health effects*

In otherwise healthy individuals, manganese deficiency has not been attributed to inadequate dietary intake (NHMRC, 2006). In a small number of studies where males consumed diets deficient in manganese, physical effects included dermatitis, hypocholesterolaemia, and a slight reddening of the hair, which was resolved by re-establishing manganese in the diet (Finley and Davis, 1999).

In excess, manganese can interfere with iron absorption (Finley and Davis, 1999; Rossander-Hulten *et al*., 1991). Symptoms of accidental manganese overdose include scaly dermatitis, lowering of cholesterol, loss of hair pigmentation, and reduced vitamin K-dependent clotting factors. Neurological effects, such as muscle pain, tremor, fatigue, memory problems and impaired reflexes, have been reported in population groups exposed to high levels of manganese in drinking water (NHMRC, 2006).

The AIs for manganese range from 600-5,500 mg/day based on different age-gender groups. There are currently no established EARs and ULs in Australia for manganese.

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of manganese in the foods analysed are presented in Table A11.7 of Appendix 11. The foods with the highest manganese levels were notably 'mixed grain breakfast cereal' and almonds, with mean concentrations of 35 and 32 mg/kg, respectively. 'Single grain breakfast cereals' and desiccated coconut also had relatively high manganese concentrations of 23 and 21 mg/kg respectively, compared with the other analysed foods.

Composite food samples reported quantifiable concentrations of manganese, with the exception of eight food types which had some composite samples for which there were no detections. The types of food included; butter, fruit juice, margarine, canola and olive oil, soft drink and sugar.

The estimated manganese intakes for all age-gender groups assessed are presented in Table A19.7 of Appendix 19. For all age groups, males had higher mean manganese intakes than females. For both males and females, mean intake increased with age, peaking for those aged 50-69 years (5.38 mg/day and 4.55 mg/day for males and females, respectively) and subsequently declining slightly for those in the over 70 year age groupgroup.

*Major food contributors*

Figure 25 indicates that cereal and grain based foods were a major source of manganese intake across all age-gender groups. Non-alcoholic beverages (excluding waters and milk), fruits and nuts, and vegetables were also major sources; however the contribution of these foods varied across different age-gender groups.

More specific details regarding the major contributors are presented in Table A22.13 and Table A22.14 of Appendix 22. The food group 'flours and single grain cereals' contributed to between 14-19% of total intake of those aged 16 years and under. This food group also featured prominently as a source of intake in the older age groups. Other major cereal based food group contributors (for some but not all age-gender groups) included 'multigrain, wholemeal and rye breads', 'white breads (including high-fibre white)' and 'breakfast cereals with mixed grains', and 'fruits and nuts'. Other major contributors were teas, coffee (from instant) and cereal-based beverages. In particular, teas contributed to 29% of the total manganese intake in females over 70 years of age.

*Figure 25: Major food contributors to manganese dietary intakes*
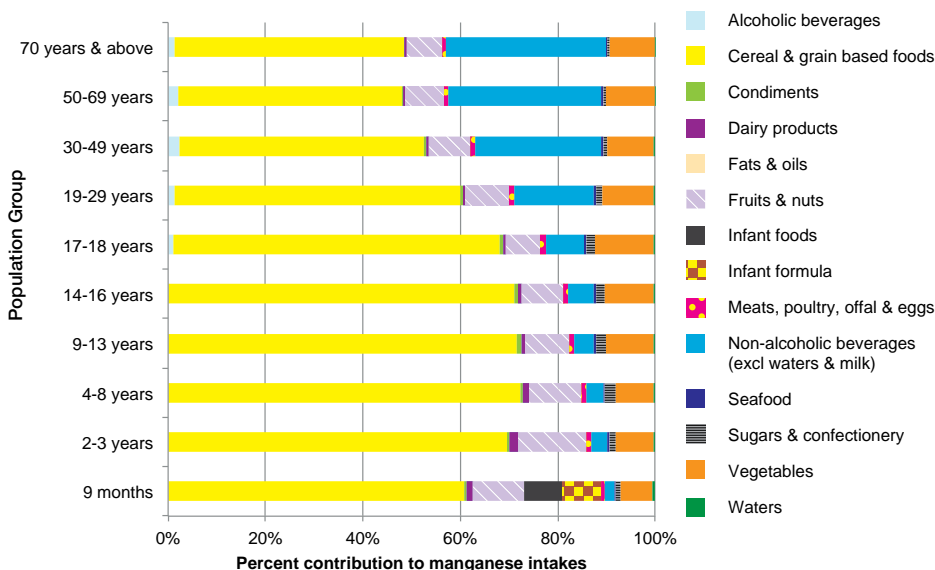


*Risk characterisation*

The mean estimated dietary manganese intake of 9 month old infants (0.88 mg/day) exceeded the AI of 0.60 mg/day (Table A19.7 Appendix 19).

For children and adolescents 2-18 years of age, the mean estimated dietary manganese intakes for each age-gender group were similar to, but exceeded slightly, their respective AIs (Table A16.1 Appendix 16 and Table A19.7 Appendix 19).

The mean estimated dietary intake of manganese for males in age groups 19 years and older were similar to but slightly lower than their AI of 5.5 mg/day, with intakes ranging from 5.0-5.4 mg/day (Table A19.7 of Appendix 19). Similarly for women aged 19 years and older, the mean intakes for each age-gender group were slightly lower than their AI of 5 mg/day, with intakes ranging from 3.9-4.5 mg/day.

Due to the limitations of the ATDS methodology and those associated with the AI, and the lack of an established EAR for manganese in Australia, conclusions cannot be drawn from this study as to whether intakes of manganese in the population are adequate.

Currently there are no established ULs for manganese due to a lack of suitable data. The NHMRC (2006) concluded that intakes of manganese higher than that normally present in food and beverages "could represent a risk of adverse health effects without evidence of any health benefit". On this basis, the current dietary intakes of manganese are unlikely to be considered excessive.

*Comparisons with previous studies*

As this is the first ATDS to study intake estimates of manganese, no comparisons can be made with previous total diet studies in Australia.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Manganese | • Carbohydrate, cholesterol and amino acid metabolism<br><br>• Bone formation | • Flours and single grain cereals<br><br>• Multigrain, wholemeal and rye breads<br><br>• Tea and instant coffee | • AIs 600 – 5,500 mg/day<br><br>• No established EARs and ULs in Australia | • No conclusions can be made in relation to adequate dietary intakes of manganese due to an absence of EARs<br><br>• Current intakes are considered unlikely to be excessive |

^AIs based on different age-gender groups.

## Molybdenum

*Health effects*

Molybdenum deficiency has not been observed in otherwise healthy humans (NHMRC, 2006). Deficiency has been reported in people with a rare metabolic disorder (Johnson *et al*., 1980) and in those receiving long-term total parenteral nutrition deficient in molybdenum (Sardesai, 1993)..

Molybdenum appears to have low toxicity in humans. The risk of deficiency and toxicity may be reduced by the body's capacity to eliminate excess molybdenum in the urine at high intakes and increase uptake into tissues at low intakes (Novotny and Turnlund, 2007).

It has been suggested that the more soluble forms of molybdenum have greater toxicity than less soluble or insoluble forms (FSANZ, 2008). Limited data are available on excess molybdenum intake; however consumption of food and water which contain more than 100 mg/kg body weight can result in signs of toxicity including diarrhoea, anaemia and elevated uric acid in the blood (Expert Group on Vitamins and Minerals, 2003).

The AI for molybdenum is 3 µg/day and is only set for 7-12 month olds. The EARs range from 13-34 µg/day and ULs from 300-2,000 µg/day based on different age-gender groups. The ULs for molybdenum are based on adverse effects on reproduction and development in rodents (NHMRC, 2006).

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of molybdenum in the foods analysed are presented in Table A11.8 of Appendix 11. The food with the highest molybdenum level was peanut butter, with a mean concentration of 1.8 mg/kg.

For the majority of foods, a good proportion of the analysed samples contained quantifiable concentrations of molybdenum with the exception of mango, canola and olive oil and soft drinks which did not contain quantifiable molybdenum concentrations.

The estimated molybdenum intakes for all age-gender groups assessed are presented in Table A19.8 of Appendix 19. Intakes were similar for males and females up to the age of 8, after which males had higher mean molybdenum intakes than females. Among both males and females, mean intake was highest in those aged 19-29 years (120 and 85 µg/day respectively). Intake declined steadily from 30 years of age.

*Major food contributors*

Figure 26 illustrates that 'cereal and grain- based foods' were a major source of molybdenum intake across all age-gender groups, together with dairy products and vegetables. This is with the exception of 9 month old infants, where infant formula was a more significant source of molybdenum intake.

More specific details regarding the major food contributors are presented in Table A22.15 and Table A22.16 of Appendix 22. Of the cereal and grain based foods, the food groups, 'flours and single grains cereals', 'white breads (including high-fibre white)', 'multigrain, wholemeal and rye breads', and 'rice and rice products' were major contributors. However this varied amongst the age-gender groups assessed. Of the dairy products, the food group 'milks and cream' was a major contributor. In addition, the food group 'soy beverages, soy beans and tofu' was a major contributor to the diets of those aged 2-3 years and females aged 4-8 years. Infant formulas and cereals contributed to 33% and 9% of molybdenum intake in the diet of 9 month old infants, respectively.

*Figure 26: Major food contributors to molybdenum dietary intakes*
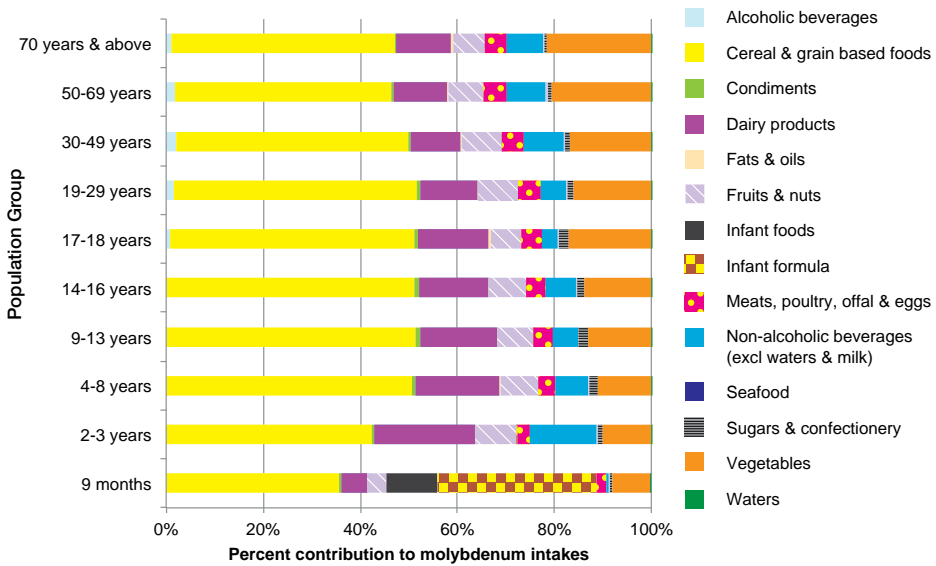


*Risk characterisation*

The mean estimated molybdenum intake for 9 month old infants (28 µg/day) exceeded the AI for infants 7-12 months of age (3 µg/day) (Table A19.8 of Appendix 19).

Only a very small proportion (<1%) of females aged 19-29 and 30-49 years and males aged 50-69 years had dietary intakes below the EAR (Table 11). Therefore, molybdenum intakes are considered to be adequate in the Australian population.

There is no established UL for molybdenum intake of infants in Australia; therefore no risk characterisation was undertaken for this group.

In the absence of human data, current ULs for molybdenum are derived from the NOAEL of 0.9 mg/kg bw/day in an animal reproduction and developmental toxicity study and using a 30-fold uncertainty factor. This same study was used as the basis for the European TDI, which is a lower value because of the application of a 100-fold uncertainty factor to the NOAEL. The UL for molybdenum was not exceeded for any population group in the current study, with the exception of a very small proportion of 2-3 year old males (2%). Given this very small exceedance and the basis of the UL, current intakes of molybdenum are not considered to be a human health and safety risk.

*Table 11: Estimated proportion of population with dietary molybdenum intakes below the EAR and above the UL*

| Age | Gender | Estimated proportion with dietary molybdenum intakes^ | |
|---|---|---|---|
| | | <EAR (%) | >UL (%) |
| **2-3 years¤** | Male | 0 | 2 |
| | Female | 0 | <1 |
| **4-8 years¤** | Male | 0 | 0 |
| | Female | 0 | 0 |
| **9-13 years¤** | Male | 0 | 0 |
| | Female | 0 | 0 |
| **14-16 years¤** | Male | 0 | 0 |
| | Female | 0 | 0 |
| **17-18 years*** | Male | 0 | 0 |
| | Female | 0 | 0 |
| **19-29 years*** | Male | 0 | 0 |
| | Female | <1 | 0 |
| **30-49 years*** | Male | 0 | 0 |
| | Female | <1 | 0 |
| **50-69 years*** | Male | <1 | 0 |
| | Female | 0 | 0 |
| **70 years & above*** | Male | 0 | 0 |
| | Female | 0 | 0 |

^ does not include the use of supplements.
¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake).
* derived using the Australian 1995 National Nutrition Survey.

*Comparisons with previous studies*

In the 22nd ATDS, a larger proportion of 2-3 year old males (3.5%) than that determined in this study exceeded the UL for molybdenum. The 22nd ATDS concluded that there were no concerns about the excessive dietary intake of molybdenum among the Australian population groups assessed. Consistent with the current study, no other population groups in the 22nd ATDS had estimated intakes above their respective ULs.

Similar to the present study, in the 22nd ATDS a very small proportion (0.1%) of males aged 50-69 years had intakes below the EAR (FSANZ, 2008). Other population groups with inadequate intakes in the 22nd ATDS were 0.4% of males 4-8 years old and 1.1% of males and 0.2% of females 9-13 years old.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Molybdenum | • Energy metabolism<br>• Formation of blood, bone and cartilage | • Flours and single grains cereals<br>• White breads (including high-fibre white<br>• Milks and cream | • AI 3 µg /day*<br>• EARs 13-34 µg / day<br>• ULs 300-2,000<br>• µg /day<br>• ULs are based on adverse effects on reproduction and development in rodents | • A very small proportion of the population reported intakes below respective EARs<br>• Dietary intakes for all age-gender groups were below respective ULs except for males aged 2-3 years<br>• Given the basis for current ULs, this very small exceedance is not considered to be a human health and safety risk |

* AI established for 7-12 month olds only.
^ EARs and ULs based on different age-gender groups. No UL has been established for infants aged 7-12 months.

### Potassium

*Health effects*

Severe potassium deficiency is very rare in the general population, but results in hypokalaemia, characterised by abnormalities in heartbeat, muscle weakness and glucose intolerance (Institute of Medicine, 2006). A low potassium, high protein diet may induce bone demineralisation, osteoporosis and kidney stones (NHMRC, 2006).

There is little evidence to suggest that a high potassium intake from food has any adverse health effects in otherwise healthy individuals. However, very high potassium intakes can cause gastrointestinal discomfort and stress (NHMRC, 2006).

AIs for potassium range from 700-3800 mg/day based on different age-gender groups. There are currently no established EARs and ULs in Australia for potassium.

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of potassium in the foods analysed are presented in Table A11.9 of Appendix 11. The foods with the highest potassium levels were potato crisps and dried apricots, with mean concentrations of 16,000 and 15,000 mg/kg, respectively.

For all of the foods analysed in this study, all samples contained quantifiable concentrations of potassium. This is with the exception of canola and olive oil which did not contain quantifiable concentrations in all four analysed samples.

The estimated potassium intakes for all age-gender groups assessed are presented in Table A19.9 of Appendix 19. Intakes were similar for both males and females up until the age of 8, but after this age males had higher mean potassium intake than females. For both males and females, mean intakes increased with age up to 19-29 years (4,440 mg/day) for males, and 30-49 years (3,330 mg/day) for females, after which intake declined.

*Major food contributors*

Figure 27 indicates that sources of potassium dietary intake were evenly spread across a range of food groups including 'cereal and grain- based foods', 'dairy products', 'fruits and nuts', 'meats, poultry, offal and eggs', 'non-alcoholic beverages (excluding waters and milk)' and vegetables.  For 9 month old infants, infant formula was a significant contributor. The proportion that each food group contributed to dietary potassium intake varied across different age-gender groups.

More specific details regarding the major food contributors are presented in Table A22.17 and Table A22.18 of Appendix 22. For males and females in most age groups the food group, 'milks and creams', was a major contributor to intake, contributing to between 10-25% of potassium intake. This was more apparent in those under 16 years of age, excluding infants, where infant formula was the major source of intake, contributing to 49% of total intake.

*Figure 27: Major food contributors to potassium dietary intakes*
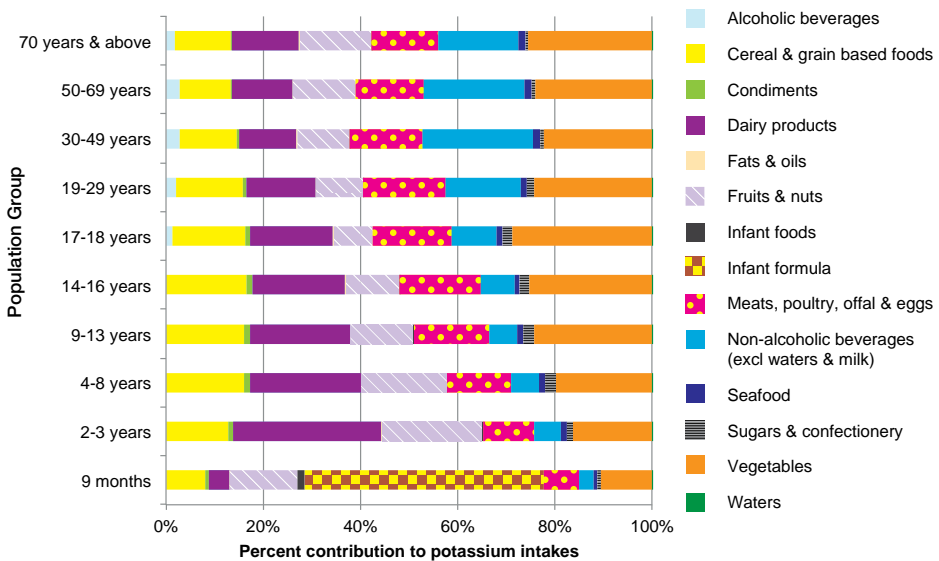


*Risk characterisation*

The mean potassium intakes were very close to, or exceeded, the respective AIs for most age-gender groups, except in males aged 70 years and above where dietary intake (3,610 mg/day) was slightly lower than the AI (3,800 mg/day) (Table A19.9 of Appendix 19). When mean intake of a population group exceeds the AI the expected prevalence of inadequate intake is likely to be low, however no assumptions can be made about the prevalence of inadequate intakes when the mean intake of the group falls below the AI (Institute of Medicine, 2000).

Due to the limitations of the ATDS methodology and those associated with the AI and the lack of an established EAR for potassium in Australia, conclusions cannot be drawn from this study as to whether intakes of potassium in the population are adequate.

No ULs have been established for potassium from dietary sources due to a lack of reports of adverse effects (e.g. hyperkalaemia) from ingestion of potassium occurring naturally in food (NHMRC, 2006). On this basis, the current estimated intakes of potassium from food are not considered excessive.

*Comparisons with previous studies*

As this is the first ATDS to study intake estimates of potassium no comparisons can be made with previous total diet studies in Australia.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| Potassium | • Main cation of intracellular fluid<br><br>• Nerve transmission and muscle contractions | • Milks and cream<br><br>• Root vegetables (starchy) | • AIs 700 – 3,800 mg/day<br><br>• No established EARs and ULs in Australia | • No conclusions can be made in relation to adequate potassium intakes due to absence of EARs<br><br>• Given the absence of ULs due to limited evidence of adverse effects from dietary sources, current intakes are not considered to be excessive |

^ AIs based on different age-gender groups.

### Selenium

*Health effects*

Data is limited on selenium toxicity in humans; however the most common symptoms of chronic selenium poisoning include brittleness and loss of hair and nails, gastrointestinal disturbance, skin rash, irritability and nervous system abnormalities (NHMRC, 2006).

The AI for selenium is 15 µg/day and is only set for 7-12 month olds. The EARs for zinc range from 20-60 µg/day and the ULs range from 60-400 mg/day depending on age-gender group. The ULs for selenium are based on a range of adverse effects observed in humans as mentioned above (e.g. brittleness and loss of hair and nails and gastrointestinal disturbances) (NHMRC, 2006).

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of selenium in the foods analysed are presented in Table A11.10 of Appendix 11. The food with the highest selenium concentration was 'tuna, canned in brine', with a mean concentration of 0.87 mg/kg. Certain fresh fruits and vegetables, including apples, nectarine, watermelon, tomatoes and lettuce contained quantifiable selenium concentrations in less than half of the eight samples analysed for each.

The estimated selenium intakes for all age-gender groups assessed are presented in Table A19.10 of Appendix 19. Intakes were similar for both males and females up until the age of 8, but after this age males had higher mean selenium intake than females. For both males and females, mean intake increased with age up until the ages of 19-29 years (171 and 111 µg/day for males and females, respectively), after which intake declined.

*Major food contributors*

Figure 28 indicates that significant sources of selenium intake were spread across a range of food groups, namely, 'cereal and grain based foods', dairy products, 'meats, poultry, offal and eggs', and seafood. For 9 month old infants, infant formula was the major source of dietary selenium.

More specific details regarding the major food contributors are presented in Table A22.19 and Table A22.20 of Appendix 22. Of the 'cereal and grain- based foods', major food group contributors were 'white breads (including high-fibre white)', and 'multigrain, wholemeal and rye breads'. Of the dairy products, the food group 'milks and cream' was a major contributor for females of all ages, and for males of most ages. This was more apparent in

those under 16 years of age. This is with the exception of 9 month old infants, where infant formulas were the major source of intake, contributing to 51% to total intake.

*Figure 28: Major food contributors to selenium dietary intakes*
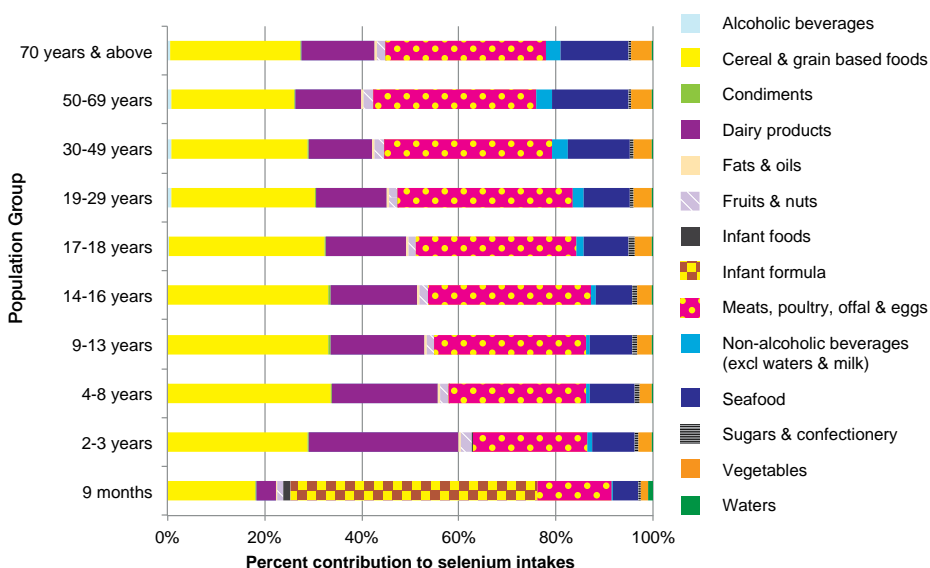


*Risk characterisation*

The mean estimated dietary intake of selenium for 9 month old infants (38 µg/day) exceeds the AI of 15 µg/day for this age group (Table A19.10 of Appendix 19).

Only a very small proportion (<1%) of females aged 17-18, 19-29, and 30-49 years had dietary intakes that were below the EAR (Table 12).

The selenium intake appeared to be adequate for all age-gender population groups as no or only a very small proportion of these groups had intakes below their respective EARs.

From these data it is considered that the selenium intake of the Australian population is adequate.

The UL for selenium was exceeded at the 95th percentile in 9 month-old infants (130%) and in a proportion of 2-3 and 4-8 year olds. The ULs for these groups are conservative because they were derived from the UL established in young infants (i.e. <6 months old) of 47 µg/day based on bodyweight extrapolation to these higher age groups. The actual UL for 6 month old infants is based on a study on the concentration of selenium in human milk in the USA

where no adverse effects occurred at a concentration of 60 μg/L. Therefore, there is a high degree of uncertainty as to where the maximum limit for adverse effects of selenium resides. On this basis, the exceedance of the UL in these population groups is highly unlikely to represent an actual human health and safety risk.

*Table 12: Estimated proportion of population with dietary selenium intakes below the EAR and above the UL*

| Age | Gender | Estimated proportion with dietary selenium intakes^ | |
| --- | --- | --- | --- |
| | | <EAR (%) | >UL (%) |
| 2-3 years¤ | Male | 0 | 35 |
| | Female | 0 | 23 |
| 4-8 years¤ | Male | 0 | 4 |
| | Female | 0 | 2 |
| 9-13 years¤ | Male | 0 | <1 |
| | Female | 0 | 0 |
| 14-16 years¤ | Male | 0 | <1 |
| | Female | 0 | 0 |
| 17-18 years* | Male | 0 | 2 |
| | Female | <1 | 0 |
| 19-29 years* | Male | 0 | <1 |
| | Female | <1 | 0 |
| 30-49 years* | Male | 0 | <1 |
| | Female | <1 | <1 |
| 50-69 years* | Male | 0 | <1 |
| | Female | 0 | 0 |
| 70 years & above* | Male | 0 | 0 |
| | Female | 0 | 0 |

^ does not include the use of supplements.
¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake).
* derived using the Australian 1995 National Nutrition Survey.

In the 19th and 20th ATDS, intakes of selenium were compared to a tolerable limit of 12.5 µg/kg bw/day as these surveys pre-dated the establishment of ULs by the NHMRC. Converting intakes from the current survey (µg/day) to a dose (µg/kg bw/day) by dividing all individual intakes by their respective bodyweight allows current intakes to be compared to this previous tolerable limit. The 95th percentile selenium intakes for 9 month old infants and children aged 2-16 years were below the tolerable limit (Table 13).

Based on the above considerations, the apparent exceedances of the UL for selenium in some population groups are not considered to represent a public health and safety issue.

*Table 13: Estimated dietary intakes of selenium at the 95th percentile, derived using mean analytical concentrations, for children aged 9 months to 16 years*

| Age | Gender | Estimated 95th Percentile Consumer Dietary Selenium Intake^ | |
|-----|--------|-------------------|--------|
| | | µg/kg bw/day | %PTDI |
| 9 months[x] | All | 4.3 – 8.6 | 35 – 70 |
| 2-3 years[¤] | Male | 8.1 | 65 |
| | Female | 8.2 | 65 |
| 4-8 years[¤] | Male | 6.8 | 55 |
| | Female | 5.7 | 45 |
| 9-13 years[¤] | Male | 4.4 | 35 |
| | Female | 3.7 | 30 |
| 14-16 years[¤] | Male | 3.7 | 30 |
| | Female | 2.5 | 20 |

^ does not include the use of supplements

¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake)

x derived using a model diet. The lower end of the range represents dietary selenium intakes derived using 99th percentile body weight infants; the upper end of the range is where 50th percentile body weight infant's mean selenium intake is multiplied by a factor of 2.

*Comparisons with previous studies*

Mean selenium intakes were considerably higher in the current study compared to the 22nd ATDS (FSANZ, 2008). This may be due to the higher mean concentrations of selenium in a wide variety of foods together with variation in foods sampled and food mapping techniques. For example, coffee is higher in selenium than tea and was surveyed in the 23rd ATDS although not in the 22nd ATDS. In the 22nd ATDS, the much lower concentration of selenium present in tea was mapped to include coffee. Hence the contribution of selenium intakes from coffee was underestimated in the 22nd ATDS. Conversely, the contribution of selenium intakes from tea in the 23rd ATDS is overestimated.

In the 22nd ATDS, the prevalence of inadequate intakes was up to 56% in some age-gender population groups, with higher proportions of females having intakes below the EAR than males.

The proportion of consumers with selenium intakes greater than the UL was higher in the present study compared to the 22nd ATDS, particularly for children 2-3 years of age.

In the 19th ATDS, mean estimated dietary intakes of selenium as a percentage of the TDI ranged from 0.44 -1.6%. In contrast, mean estimated dietary intakes of selenium in the 20th ATDS ranged from 7.7-24% of the TDI. Mean estimated selenium intakes were considerably higher in the 20th ATDS compared to the 19th ATDS. This is likely to be due to enhanced method sensitivity, differences in the types of foods surveyed, particularly seafood, and food mapping techniques.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|---|---|---|---|---|
| **Selenium** | • Thyroid hormone regulation, energy metabolism and immune response | • White breads (including high-fibre white)<br><br>• Milks and cream<br><br>• Beef, veal and venison | • EARs 20-60 μg/day<br><br>• ULs 60-400 μg/day<br><br>• ULs are based on the occurrence of brittleness and loss of hair and nails, and gastrointestinal disturbances | • Dietary intakes for all age-gender groups were below respective ULs except for 9 month old infants, children aged 2-3 years and 4-8 years<br><br>• Given the conservatism around the ULs for selenium, these exceedances are not considered to be a human health and safety risk |

^ EARs and ULs based on different age-gender groups.

**Zinc**

*Health effects*

There is no evidence of adverse health effects from high intakes of zinc naturally occurring in food (NHMRC, 2006).The chronic consumption of high levels of zinc in supplemental form can result in impaired immune function, decreased HDL cholesterol and induced copper deficiency (Fosmire, 1990).

The ULs for zinc range from 5-40 mg/day depending on age-gender group. The ULs for zinc are based on adverse effects on copper homeostasis (NHMRC, 2006).

*Analytical and dietary exposure results*

The mean, maximum and minimum concentrations of zinc in the foods analysed are presented in Table A11.11 of Appendix 11.  The food with the highest zinc levels was lean minced beef, with a mean concentration of 49 mg/kg.

The majority of the foods in this study contained quantifiable concentrations of zinc in all of the analysed foods, in at least half of the analysed composite samples.

The estimated zinc intakes for all age-gender groups assessed are presented in Table A19.11 of Appendix 19. Intakes were similar for both males and females up until the age of 13 years, but after this age males exhibited higher mean zinc intake than females. For both males and females, mean intakes increased with age up until the ages of 17-18 and 19-29 years (14.8 and 9.4 mg/day for males and females, respectively), after which intakes declined.

*Major food contributors*

Figure 29 indicates that major sources of zinc dietary intake were 'cereal and grain–based foods', dairy products, 'meats, poultry, offal and eggs', vegetables and, for infants, infant formula.

More specific details regarding the major food contributors are presented in Table A22.21 and Table A22.22 of Appendix 22. For males and females over the age of 3 years (and particularly for those over 16 years of age) 'beef, veal and venison' was a major food group, contributing to between 13-29% of total zinc intake. For infants, infant formulas contributed to 67% of total intake. For those aged 2-3 years, 'milks and cream' contributed to 18% of total intake. Other contributors for some but not all sectors of the Australian population included 'flours and single grains cereals', and cheeses. The food group 'lamb, mutton, kangaroo and rabbit' emerged as a major contributor to zinc intake in the diets of over 50 year olds.

*Figure 29: Major food contributors to zinc dietary intakes*
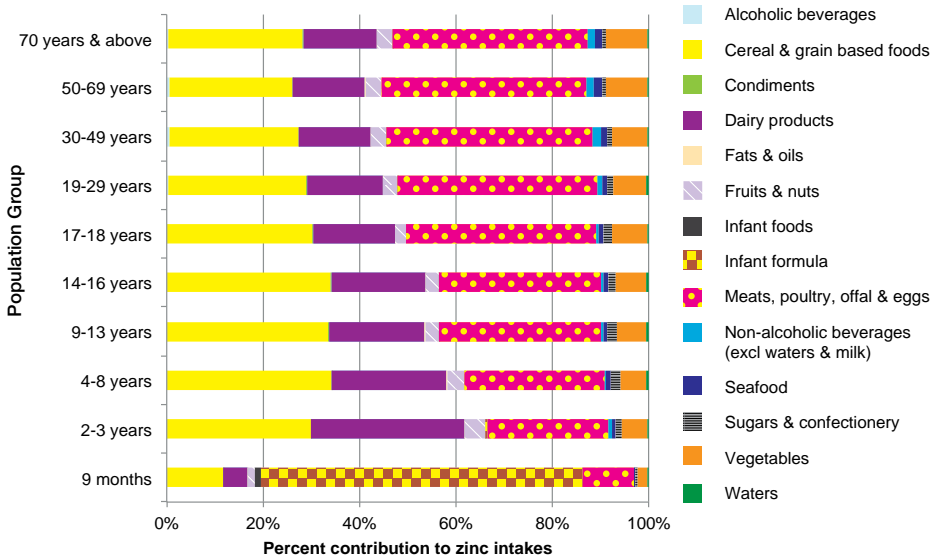


*Risk characterisation*

The estimated 95th percentile zinc intake for 9 month old infants was 210% of the UL of 5 mg/day covering 7-12 month-old infants (Table 14). In 2-3 year old children, 62% of male respondents and 38% of female respondents exceeded the UL of 7 mg/day covering 1-3 year olds (Table 15). In 4-8 year olds, a small proportion of respondents (5% of males and 1% of females) exceeded the UL of 12 mg/day.

*Table 14: Estimated 95th percentile dietary zinc intake for 9 month old infants, as a %UL*

| Age | Estimated 95th percentile dietary zinc intake | |
| --- | --- | --- |
| | mg/day | %UL |
| 9 months | 10.3 | 210 |

ˣ Derived using a model diet

The ULs for the above age-gender groups were derived from the UL established for 6 month-old infants via a bodyweight adjustment. The UL in 6 month old infants is based on three published studies. In two of these studies, consumption of infant formula containing 5.8 mg/L, or a daily intake of 10 mg/day, was without any adverse effect such as perturbation of plasma copper levels. However, in the third study, administration of zinc to 6-12 month old infants at 10 mg/day reduced plasma copper levels. An examination of this study reveals a number of limitations, which indicate an overly conservative basis for the UL. In particular, the study was designed to examine the effect of daily zinc supplementation on the incidence and severity of diarrhoea in infants in Indian villages; that is, it was conducted in an unhealthy population not representative of the general population. Additionally, the study cohort had elevated plasma copper levels in both test and placebo groups prior to the commencement of the study. At the conclusion of the study, a relatively modest difference in plasma copper levels occurred between the test and placebo groups (~15 µg/mL) with copper levels still elevated.

Surveys in the US have indicated that young children have a high proportion of intakes of some nutrients (including zinc) exceeding the UL; however there is a lack of data on the effects of such intakes (Health Canada, 2006).

In the 20th ATDS, FSANZ used a tolerable limit for zinc of 1 mg/kg bw/day, which pre-dated the establishment of a UL. For the purpose of consistency and given the current ULs for zinc are considered overly conservative by FSANZ, it is appropriate that  the same comparator be used as a basis to further characterise the risk of excessive zinc exposure in 9 month old infants, 2-3 and 4-8 year old children.

If the high percentile estimated intakes (mg/day) of zinc for 9 month old infants are calculated using the 50th and 99th percentile bodyweights (Appendix 8), intakes are 60-120% of the tolerable limit of 1 mg/kg bw/day. Given intake for 9 month old infants is based on a model diet which is inherently conservative, this more refined estimate indicates no concern with the current intake of zinc by this age group. Using the same approach, the 95th percentile zinc intakes for 2-3 and 4-8 year old children were below the tolerable limit (Table 16).

On the basis of the above considerations, current intakes of zinc across the Australian population are not considered to be excessive.

*Table 15: Estimated proportion of population with dietary zinc intakes above the UL*

| Age | Gender | Estimated proportion with dietary zinc intakes >UL (%)^ |
|-----|--------|---------------------------------------------------------|
| 2-3 years¤ | Male<br>Female | 62<br>38 |
| 4-8 years¤ | Male<br>Female | 4<br>1 |
| 9-13 years¤ | Male<br>Female | 0<br>0 |
| 14-16 years¤ | Male<br>Female | 0<br>0 |
| 17-18 years* | Male<br>Female | 0<br>0 |
| 19-29 years* | Male<br>Female | <1<br>0 |
| 30-49 years* | Male<br>Female | <1<br>0 |
| 50-69 years* | Male<br>Female | 0<br>0 |
| 70 years & above* | Male<br>Female | 0<br>0 |

^ does not include the use of supplements.
¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake).
* derived using the Australian 1995 National Nutrition Survey.

*Table 16: Estimated dietary intakes of zinc at the 95th percentile, derived using mean analytical concentrations, for children aged 9 months to 16 years*

| Age | Gender | Estimated 95th Percentile Consumer Dietary Zinc Intake^ | |
| --- | --- | --- | --- |
| | | mg/kg bw/day | %PTWI |
| 9 months^x | All | 0.58 – 1.2 | 60 – 120 |
| 2-3 years^¤ | Male | 0.66 | 65 |
| | Female | 0.69 | 70 |
| 4-8 years^¤ | Male | 0.54 | 55 |
| | Female | 0.49 | 50 |
| 9-13 years^¤ | Male | 0.37 | 35 |
| | Female | 0.31 | 30 |
| 14-16 years^¤ | Male | 0.33 | 35 |
| | Female | 0.21 | 20 |

^ does not include the use of supplements
¤ derived using the Australian 2007 Children's National Nutrition and Physical Activity Survey (Day 1 adjusted nutrient intake)
x derived using a model diet. The lower end of the range represents dietary zinc intakes derived using 99th percentile body weight infants; the upper end of the range is where 50th percentile body weight infant's mean zinc intake is multiplied by a factor of 2.

*Comparisons with previous studies*

In the 20th ATDS, estimated mean dietary zinc intake exceeded the Recommended Daily Intake (RDI) for adult males, boys, toddlers and infants but was lower than the RDI for adult females and girls. The report concluded that actual requirements for zinc would likely be met for most females in these age groups since RDIs are established so that the nutrient requirements of virtually all the population are met (FSANZ, 2003).

It was also concluded in the 20th ATDS that the estimates met acceptable health standards as all were below the tolerable limit for the five age-gender groups analysed. However, it is important to note that the reference health standards used in the 20th ATDS are different to those used in the current study.

In the 20th ATDS, mean dietary intakes of zinc were estimated to be 630 µg/kg bw/day for 9 month old infants, 170 µg/kg bw/day for adult males aged 25-34 years and 130 µg/kg bw/day for adult females aged 25-34 years. In the 23rd ATDS, mean dietary intakes of zinc were estimated to be 579 µg/kg bw/day for 9 month old infants, 181 µg/kg bw/day for adult males aged 25-34 years and 145 µg/kg bw/day for adult females aged 25-34 years.

*Summary table*

| Nutrient | Function | Major food contributors | Reference health standard^ | Risk characterisation |
|----------|----------|-------------------------|----------------------------|------------------------|
| **Zinc** | • Required for growth and development<br><br>• Component of several enzymes involved in the metabolism of protein, carbohydrate and fat | • Beef, veal and venison<br><br>• Flours and single grains cereals<br><br>• Milks and cream | • ULs 5-40 mg/day<br><br>• ULs are based on adverse effects on copper homeostasis. | • Dietary intakes for all age-gender groups were below respective ULs except for 9 month old infants, children aged 2-3 years and 4-8 years<br><br>• Given the ULs for zinc are highly conservative, current zinc intakes are not considered to be excessive |

^ ULs based on different age-gender groups.

# Part E – Conclusions and recommendations

The 23rd ATDS confirms the current safety of the Australian food supply in terms of the levels of agricultural and veterinary chemicals, contaminants, selected mycotoxins and nutrients.

Estimated dietary exposures for all agricultural and veterinary chemicals were below relevant reference health standards for the population. There were no detections of the mycotoxins in any of the foods analysed.

In relation to contaminants, estimated dietary exposures for all age-gender groups were below the relevant reference health standard. For lead, the risk assessment was based on a MOE approach as there is currently no reference health standard. Estimated mean dietary exposures to lead were below the level considered by JECFA to have a low risk of reducing the population IQ for children or increasing the systolic blood pressure in adults.

Estimated dietary exposures to total arsenic or organic arsenic were similar to levels found in previous studies. No detections of inorganic arsenic were found in any of the seafood samples analysed. This finding suggests that there is unlikely to be any human health and safety risks in relation to inorganic arsenic on the basis of the seafood sampled in this study.

Comparisons of the estimated dietary intakes of nutrients to the relevant AIs, EARs (excluding calcium, iron and zinc) and ULs, where established, provide a useful indication of the nutritional adequacy/excess for different age-gender groups. For nutrients with AIs, the majority of mean estimated dietary intakes for all age-gender groups were similar to, or exceeded their respective AIs.

For nutrients, where EARs have been assessed, the prevalence of intakes below the EAR was low. Given these are only estimates of the prevalence of inadequate nutrient intakes, the prevalence of impaired nutritional status in the population would require further investigation using biological measures.

For nutrients with established ULs, some age-gender groups exceeded these values at varying magnitudes, specifically for fluoride, selenium and zinc. In each of these cases, the exceedances were considered unlikely to pose a human health and safety risk.

The recommendations from this study are to:

- Continue the ATDS as a national collaborative effort to estimate the level of dietary exposure of the Australian population to a range of food chemicals in order to assess public health and safety.

- Continue to monitor agricultural and veterinary chemicals to ensure that existing regulatory measures for these chemicals in food provide adequate protection of the Australian population. Given the large body of data relating to dietary exposure to these chemicals in Australia, it is recommended that the current program of low frequency monitoring in food through the ATDS is appropriate.

- Monitor agricultural and veterinary chemicals in future ATDS with a focus on chemicals for which there are no recent or limited data on levels in the food supply. Particularly where the possibility exists that, based on a theoretical calculation of exposure assuming the chemical is present in all relevant foods at the MRL, the reference health standard could be approached.

- For foods which contributed to total dietary exposure of a contaminant at 10% or more of the reference health standard for one age-gender group or 5% or more of the reference health standard for two or more age-gender groups, should be included in future ATDS. This recommendation is based on criteria used by the CCCF for assessing the significance that a food or food group contributes to total dietary exposure of a contaminant.

- Continue to monitor contaminants in foods for which maximum levels have been established and that are likely to be major contributors to dietary exposure.

- Continue to include nutrients for which fortification and addition to food and water has been mandated or permitted (e.g. iron, calcium and fluoride) to monitor their potential variability.

- Monitor international developments in reference health standards to inform the selection of food chemicals and foods for inclusion in future ATDS.

- Investigate the possibility of greater collaboration with the New Zealand Total Diet Study.

- Continue collaboration with and provision of data to the WHO/Global Environment Monitoring System-Food Contamination Monitoring and Assessment Programme (GEMS/Food).

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