FOOD STANDARDS
Australia New Zealand

The 22nd Australian Total Diet Study

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Te Mana Kounga Kai – Ahitereiria me Aotearoa

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Foreword

When we eat, we ingest a range of chemicals that make up food. These chemicals can occur naturally in the food as nutrients or, become introduced as contaminants or residues from agricultural and veterinary practices or the environment. They can also be intentionally added to a food, as food additives, to perform a number of technological functions, such as increasing storage time. There are also a number of fortified foods now available, with nutrients intentionally added over and above natural levels.

One of the roles of Food Standards Australia New Zealand (FSANZ) – an independent Australian Government agency – is to assess the risks posed by the presence of such chemicals in food and to set safe limits where necessary. The limits we set are consistent with international standards.

Since 1970, the Australian Government has conducted regular studies of consumer exposure to chemicals in the food supply, the last seven studies by the national food agency now known as FSANZ. The first twenty studies looked at dietary exposure to pesticide residues and contaminants, with each study giving Australia's food a clean bill of health. These studies are the most comprehensive dietary exposure studies carried out in Australia.

We have now expanded the scope and frequency of the studies to consider a wider range of food chemicals, including additives and nutrients, thereby enabling us to focus on chemicals which may present a public health concern or where we have identified gaps of information.

The 22nd Australian Total Diet Study was conducted by FSANZ with the assistance of our regulatory partners in all the States and Territories. This study focused on the dietary exposure of the Australian population to the trace elements iodine, selenium, molybdenum, chromium and nickel.

We selected iodine and, to some extent, selenium, for the 22nd Australian Total Diet Study because there is evidence that some Australians are deficient in these important nutrients. The study has been especially valuable as input to our current work on the proposed mandatory fortification of food with iodine and will fill in gaps in knowledge regarding the composition of Australian foods for the other nutrients.

I extend my thanks to the experts who formally peer-reviewed this study and to the staff of FSANZ and other agencies who have contributed to a successful outcome. I am pleased to commend the 22nd Australian Total Diet Study as a contribution to maintaining the Australian food supply as one of the safest in the world.

Rob Knowles
CHAIRMAN

Abbreviations

AGAL	Australian Government Analytical Laboratory
AI	Adequate Intake
ANZFA	Australia New Zealand Food Authority (now FSANZ)
ATDS	Australian Total Diet Study
DIAMOND	Dietary Modelling of Nutritional Data (FSANZ computer software program)
EAR	Estimated Average Requirement
FAO	Food and Agriculture Organization
FSANZ	Food Standards Australia New Zealand
IDD	Iodine Deficiency Disorder
JECFA	Joint FAO/WHO Expert Committee on Food Additives
LOR	Limit of Reporting
LOAEL	Lowest Observable Adverse Effect Level
mg/kg	milligrams per kilogram
mg/kg bw	milligrams per kilogram of body weight
ND	Not detected
NHMRC	National Health and Medical Research Council
NMI	National Measurement Institute
NNS	National Nutrition Survey
NOAEL	No Observable Adverse Effect Level
NRV	Nutrient Reference Value
RDI	Recommended Dietary Intake
TPN	Total Parenteral Nutrition
UL	Upper Level of Intake
µg/day	micrograms per day
WHO	World Health Organization

Note: Definitions for some of these abbreviations are in Appendix 1.

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Executive Summary

The purpose of the Australian Total Diet Study (ATDS) is to estimate the level of dietary exposure (intake) of the Australian population to a range of chemicals including pesticide residues, contaminants, nutrients, additives and other substances that may be found in the food supply. The 22nd ATDS estimates dietary intake of five trace elements: iodine, selenium, chromium, molybdenum, and nickel. In the ATDS, dietary intake was estimated by determining the level of the substance in foods by laboratory analysis, and then combining this with the amount of food consumed, as determined in a separate consumption study. The dietary intake of these elements was assessed against their respective reference health standard for Australian population groups, where available. In order to achieve more accurate dietary intake estimates, the foods examined in the ATDS were prepared to a 'table ready' state before they were analysed. As a consequence, both raw and cooked foods were examined.

FSANZ funded and coordinated the 22nd ATDS, while the food regulatory agencies in the State and Territory governments obtained the food samples in their region. The Australian Government Analytical Laboratory (now the National Measurement Institute) carried out sample preparation and analyses.

Ninety-six types of foods, sampled during July/August and November/December 2004, were tested for the five trace elements. The food types selected included both foods that might be expected to show regional variation (regional foods), and foods that were available nationwide and were not expected to show regional variation (national foods). Food types were sampled in each of the States and Territories in Australia. For each food, between six and ten composite samples were prepared, each consisting of at least three purchases.

The dietary intake of each trace element was estimated using the 1995 National Nutrition Survey (NNS) food consumption data and the level of the element present in each food. Any contribution that medicines, such as dietary supplements, may make to dietary intake was excluded.

Estimated dietary intakes of iodine, selenium, chromium, molybdenum, and nickel were calculated for a range of age–gender groups: infants aged 9 months; girls and boys aged 2-3 years; girls and boys aged 4-8 years; girls and boys aged 9-13 years; adolescent females and males aged 14-18; adult females and males aged 19-29; adult females and males aged 30-49 years; adult females and males aged 50-69 years; and adult females and males aged 70 years and over.

Diets for each individual in the representative age-gender groups from the 1995 NNS were used for intake estimations. For the infants aged 9 months, a theoretical diet was constructed based on an extrapolation of the 2 year old diet from the NNS. The NNS used a 24-hour food recall methodology. A second 24-hour recall was also conducted on 10% of respondents for a non-consecutive day. Standard methodologies were used to estimate an adjusted nutrient (usual) intake based on nutrient intake from the first 24-hour recall (day one), which were then adjusted using nutrient intake information from the second 24-hour recall (day two).

The estimated dietary intake of each nutrient from the Australian diet was compared to the relevant Australian Estimated Average Requirements (EAR) or Adequate Intake levels (AI) and the Upper Level of Intake (UL) endorsed by the National Health and Medical Research Council (NHMRC) in 2006.

Results

The mean, maximum and minimum concentrations of each element in each food analysed were determined. The 5th percentile, mean and 95th percentile levels of dietary intake were calculated for each nutrient and population group, based on the mean nutrient concentration.

Iodine

In relation to iodine intakes:

- most adult respondents had dietary intakes below the EAR;
- no population groups approached the UL; and
- major contributing foods to dietary iodine intake were similar in adults and children and included milk, yoghurt, ice cream, tap water, iodised salt, soft drink and eggs.

Selenium

In relation to selenium intakes:

- For all population groups, other than females aged 70+ yrs, the majority of respondents had mean dietary intakes approaching or above the EAR;
- less than 1% of males aged 2-18 years had intakes greater than their respective age group ULs, but this finding is not considered to be of concern as these ULs are highly conservative estimates. Other population groups did not exceed their respective ULs; and
- concentrations of selenium in some foods, and total intakes, appear to be lower in this study than previously estimated in the 20th ATDS;
- major contributors to dietary intake of selenium were bread, cereal, chicken, pasta, beef, fish and eggs.

Molybdenum

In relation to molybdenum intakes:

- the majority of respondents had dietary intakes well above the EAR;
- there were no concerns about excessive dietary intake among the Australian population groups assessed; and
- major food contributors to dietary intake of molybdenum were bread, milk, rice, peanut butter, cereal and soy beverage.

Chromium

In relation to chromium intakes:

- this study has generated Australian intake estimates for the first time;
- most population groups had mean dietary intakes approaching or above the AI, which was established using US data; and
- major contributors to dietary intake of chromium were bread, cereal, milk, juice, cake, deli meats, chocolate, tea and beer.

Nickel

In relation to nickel intakes:

- this study has generated Australian intake estimates for the first time;
- due to the absence of nutrient reference values for nickel in Australia, no risk characterisation has been performed; and
- major contributors to dietary intake of nickel were bread, cake, peanut butter, cereal, chocolate and tea.

The ATDS uses internationally accepted methodology for studies of this kind and is well regarded internationally. Nevertheless, there are a number of uncertainties inherent in the dietary exposure assessments for all total diet studies. These are associated with the assumptions that were made in the calculations, limitations of the laboratory test data and sampling, and the age of the food consumption data that were derived from the 1995 NNS.

Despite these uncertainties, the intake assessments presented in this study represent a reliable estimate of dietary intake for the five elements for the Australian population using the available data.

Whilst the majority of Australians have dietary intakes approaching or above the EAR or AI for selenium, molybdenum and chromium, a substantial proportion of the population has iodine intakes below the EAR. FSANZ has subsequently commissioned further analyses of iodine levels in Australian foods and has used this information in its consideration of the need for mandatory fortification of foods with iodine (Proposal P230 (New Zealand) and P1003 (Australia)). Continued monitoring of selenium concentrations and intakes may be warranted given the lower levels found in a range of foods compared to the findings of the 20th ATDS.

There were no concerns about excessive dietary intake of the nutrients assessed against established reference values, where these exist.

Part A Background

The purpose of the Australian Total Diet Study (ATDS) is to estimate the level of dietary exposure (intake) of the Australian population to a range of chemicals, including pesticide residues, contaminants, additives and nutrients that may be found in the food supply. Dietary exposure is estimated by analysing foods to determine the concentration of the chemical and then multiplying the concentration by the amount of the food consumed. Traditionally, the ATDS has estimated dietary exposure of the Australian population to a range of pesticide residues and contaminants. However, more recently the focus of the ATDS has expanded to consider a broader range of food chemicals, including additives and nutrients. This ATDS, the 22nd, examines the dietary intake of selected trace elements: iodine, selenium, molybdenum, chromium and nickel. These elements occur naturally in foods and their intakes have public health significance, particularly iodine and selenium.

Trace elements have a number of biological roles within the body. Although essential in the human diet, they can have potentially toxic effects (Solomans & Ruz, 1998). Adverse effects can therefore result from intakes that are too low as well as too high.

Many trace elements are widely distributed in the food supply, although the levels can vary greatly depending on factors such as the geographical origin of the food, processing practices and food preparation methods. Little information is available on the intake of many trace elements in Australian population groups.

The National Health and Medical Research Council (NHMRC) released new Nutrient Reference Values (NRVs) early in 2006 which revised the recommended intakes for a number of nutrients, as well as including values for some nutrients with no previous existing reference values (NHMRC, 2006). Therefore, the 22nd ATDS focused on trace elements that have previously been identified in Australia either as having undesirably low levels of intake or for which there were little current data.

Origin of the survey

In Australia, the NHMRC recommended in 1969 that a ‘market basket’ survey be carried out to examine the levels of pesticide residues and contaminants in foods that constitute a significant part of the normal Australian diet. This resulted in the first total diet survey in 1970, conducted by the NHMRC. Another 15 surveys were conducted by the NHMRC before responsibility passed to the initial predecessor of FSANZ, the National Food Authority. The 22nd ATDS is the seventh study to be conducted by FSANZ or its predecessors.

Previous ATDS were conducted approximately every two years, with sampling and analysis of foods taking place over 12 months, capturing seasonal variation in the food supply.

Past ATDSs have consistently shown that chronic dietary exposure of Australians to a range of agricultural and veterinary chemicals and contaminants was well below international health standards and did not represent a public health and safety risk. Therefore, in 2003, FSANZ decided, in consultation with the State and Territory government food regulatory agencies, that the scope and format of the ATDS would change to consider a wider range of food chemicals including additives and nutrients, with smaller surveys being conducted more frequently. The change in format of the ATDS has allowed FSANZ greater flexibility in focusing the study on a wider range of food chemicals to gather significant public health information about the Australian diet, particularly where there may be concern that dietary exposures may exceed or not meet the reference health standard for some population groups, or where significant data gaps exist on chemicals in foods.

Other food chemical surveillance activities in Australia

The Australian Government, through the Department of Agriculture, Fisheries and Forestry, conducts two programs that collect information on the levels of pesticide residues, contaminants and other substances in foods:

- the National Residue Survey; and
- the Imported Food Inspection Scheme, conducted by the Australian Quarantine and Inspection Service (AQIS), which undertakes the surveillance of imported foods to ensure that they comply with the Imported Food Control Act 1992 and the Food Standards Code.

The main aim of these programs is to monitor pesticide residues, contaminants and other substances in food commodities in export and import trade respectively.

In addition to these programs, the Food Regulation Standing Committee's Implementation Sub-Committee (ISC) has developed a 'Coordinated Food Survey Plan' (the Plan) to enhance survey activities across Australian jurisdictions and New Zealand. The aim of the Plan is to realise efficiencies and enhance the quality of surveys through greater collaboration in the planning, implementation and consistent management of the outcomes of food surveys. The Australian Total Diet Study is now part of the ISC Co-ordinated Food Survey Plan. The Plan outlines cross-jurisdictional survey activities which may include:

- assessing the composition of foods against standards;
- analysing the level of chemical contaminants in food;
- analysing the level of microbiological agents in food;
- assessing the labelling and advertising of food;
- auditing food processing and handling standards;
- assessing nutrient and food chemical content of foods;
- evaluating the impact of food regulation on public health and safety;
- providing risk-based information as a basis for establishing new regulations or reviewing current regulations; and
- providing contemporary information to underscore the promotion of a safe and healthy food supply.

In addition to surveillance activities undertaken as part of the Plan, State and Territory health and agriculture authorities carry out surveys of specific contaminants, pesticide residues, food additives and other food chemicals. These surveys are usually targeted to investigate specific concerns and determine whether primary producers and food manufacturers are complying with relevant food regulations. The data generated through these surveys are a valuable source of supplementary information on the chemical status of foods. Results of surveys are shared with other jurisdictions through the Food Surveillance Network. The Network is chaired by FSANZ and provides a valuable technical forum for collaboration on food surveillance issues.

Comparison with other studies

The ATDS differs from other Government surveys of food chemicals in several ways. It monitors the level of certain substances in the total diet to determine whether there are any associated risks to human health. Other surveys examine the level of chemicals in individual agricultural commodities or foods to determine compliance with food legislation but do not carry out a comprehensive examination of their significance in the overall Australian diet. In contrast to most other surveys, all ATDS food samples are prepared to a 'table-ready' state before they are analysed, that is, they are subjected to prescribed preparation or processing steps. These preparation steps vary with the type of food. For example, fruits may be peeled if they are usually eaten without their skins, while beef is dry fried because this food is nearly always consumed after cooking. As storage and preparation is known to affect the

concentration of some chemicals in food, an analysis of foods prepared 'as eaten' will result in more accurate estimations of dietary intake.

Using information from the Australian Total Diet Study

The revised format of the ATDS has allowed greater flexibility to target the studies to inform the food standard setting process. In conjunction with information from other sources, data from the ATDS provides information to be considered when reviewing, developing or amending food regulatory measures.

In addition, the results of the survey provide valuable domestic public health information and are a source of information for Australia's international contribution to: the World Health Organisation/Food and Agriculture Organisation (FAO/WHO) Global Environmental Monitoring System (GEMS), which monitors food contamination internationally; the Joint Expert Committee on Food Additives (JECFA): the Codex Committees on Food Additives, Contaminants in Food, Residues of Veterinary Drugs in Food, and Pesticide Residues: and independent researchers in both government and non-government agencies.

Part B Conducting the study

The 22nd ATDS aimed to assess the status of certain trace elements in the Australian food supply and compare estimates of these intakes to the NHMRC nutrient reference values or international reference standards.

The 22nd ATDS was coordinated by FSANZ and undertaken in cooperation with each of the Australian State and Territory government food regulatory agencies. Every State and Territory nominated liaison officers to provide advice about the study and to co-ordinate food sample collection, packaging and shipment to the laboratory for analysis.

Foods included in the survey

The 22nd ATDS surveyed 96 foods in total, 740 composite samples prepared for analysis (a complete list of foods can be found in Appendix 2). Each composite food was comprised of three primary purchased samples. The following criteria were used to select foods to be surveyed:

- provided optimal information for the analytes of interest;
- current consumption patterns represented;
- met resource capabilities of the State and Territories who collect the samples; and
- purchasing and analysis costs met budget considerations.

Foods were sampled according to a schedule that categorised them into national or regional foods. This ensured more samples were collected where there may be regional variation in composition and allowed a better overview of the Australian diet.

Regional foods were defined as those foods that might be expected to show regional variation in production or manufacture. These foods included meat and meat products, wine, bread and other bakery goods, dips and some cheese. Two composite samples of these foods, consisting of three purchases each, were collected in five capital cities, making 10 composite samples for each regional food.

National foods were defined as those foods that were distributed nationwide from a small number of manufacturers and thus were not expected to show regional variation, such as table spreads, soft drinks and potato crisps. Two composite samples, of three purchases each, were collected in three capital cities, making six composite samples for each national food.

Foods were sampled in each capital city during July/August 2004, and November/December 2004. Due to the large number of samples required, provision was made for purchasing to occur over several days within a reasonable time period. The collection period varied slightly for each State or Territory in order to stagger the arrival of samples at the analytical laboratory, as soon as practicable after purchase. All perishable samples were frozen prior to forwarding to the laboratory. The analytical laboratory prepared foods in accordance with detailed instructions. Perishable foods were prepared within 48 hours of arrival at the laboratory. However, where necessary, the preparation of frozen or shelf-stable foods was delayed, but carried out within a week of purchase.

Preparation of foods

All the foods examined in the study were prepared to a 'table ready' state before analysis (refer to Appendix 3 for details on food preparation instructions). For example, chicken breast and beef steak were grilled prior to analysis. Many foods surveyed were already in their table ready form and did not require additional preparation.

Analysis of samples

The Australian Government Analytical Laboratory (AGAL - now National Measurement Institute [NMI]) analysed the food samples in accordance with accredited quality assurance procedures and the results were provided to FSANZ, after which, trace element intakes were estimated and the report prepared.

All samples were analysed using Inductively Coupled Plasma Mass Spectrometry (ICPMS). Details of analytical methods are outlined in Appendix 4. The Limit of Reporting for each element, which is the lowest concentration level at which the laboratory is confident in the quantitative results reported, is shown in Table 1.

Table 1: Limits of reporting (LOR) for each analyte

Analyte	Substrate	Limit of Reporting
Iodine	Solid & Liquid matrix	0.01 mg/kg
Selenium, Molybdenum, Chromium and Nickel	Solid matrix	0.01 mg/kg

In keeping with good practice, FSANZ conducted inter-laboratory checks by arranging for a second suitably competent laboratory (Queensland Health and Scientific Services-QHSS) to confirm the validity of the results provided by the contracted laboratory AGAL (NMI).

Part C Estimating dietary intake of nutrients

Dietary modelling is a tool used to estimate exposures to (or intake of) food chemicals from the diet. Food regulators have used dietary modelling techniques internationally for a number of years to determine if dietary exposure to specific food chemicals represents an unacceptable risk to public health and safety. Dietary modelling is an important part of the ATDS as it translates analytical results for individual foods into dietary intake estimates that can be compared to established reference health standards.

DIAMOND (Dietary Modelling of Nutritional Data) is a computer program developed by FSANZ to automate dietary intake calculations. DIAMOND combines food consumption data with trace element concentration data (see Appendix 5) to estimate the dietary intake for that element for a range of population groups.

While dietary modelling is a scientific systematic method for estimating the amounts of trace elements a person or population may be eating, the accuracy of these intake estimates depend on the quality of the data used in the dietary models.

Detailed information on the dietary modelling technique used, including assumptions and limitations, is provided in Appendix 6. Food translations (mapping) used in the dietary exposure estimates are presented in Appendix 7.

Food consumption data

Dietary modelling used food consumption data from the 1995 NNS that surveyed 13,858 Australians aged 2 years and above using a 24-hour food recall. DIAMOND multiplied the trace element concentration of each food consumed in the survey against the amount of that food that every survey respondent consumed to estimate each individual's intake of that trace element. Mean food consumption data for each of the foods analysed in this study are provided in Appendix 8.

An individual's trace element intake, and intake of other nutrients, may vary substantially from day to day through consumption of a varied diet. More representative estimates of an individual's usual trace element intake can be developed if more than one day of food consumption data are available. Estimated trace element intakes were adjusted based on information for a second (non-consecutive) day of food consumption that was collected from approximately 10% of NNS respondents. The same statistical technique was used for this purpose as was used in the 1995 NNS for estimating intake of other nutrients. The adjustment calculation is described in Appendix 6.

Population statistics such as low, mean and high percentile intakes for each age-gender group assessed were derived from the adjusted individual intakes.

The 1995 NNS did not include children under two years of age. A theoretical diet was therefore constructed for infants at 9 months of age in order to allow mean dietary intake of the trace elements to be calculated for infants. The theoretical infant diet was based on an extrapolation from the diet of a child at two years for solid foods, with an adjustment for the proportion of the total diet made up of milk. Because the theoretical diet represents a mean intake with no variance, the proportion of the population group with dietary intakes below the EAR or above the UL could not be calculated. As an alternative, the 95th percentile dietary intake was estimated as 2.5 times the estimated mean intake (WHO, 1985). Appendix 6 provides details about how the theoretical infant diet was developed.

Population groups evaluated

The population groups assessed in the 22nd ATDS were infants aged 9 months and males and females aged:

- 2-3 years
- 4-8 years
- 9-13 years
- 14-18 years
- 19-29 years
- 30-49 years
- 50-69 years and
- 70 years and over.

Appendix 11 shows the number of individuals in each age-gender group assessed.

Nutrient levels

The trace element levels used in dietary modelling for the ATDS were the mean from the analytical results of the composite samples of each surveyed food. Where values were reported as being below the limit of reporting (LOR), the mean mineral level has been estimated assuming that the true mineral concentration was equivalent to half the LOR (the 'middle bound' estimate). 'Lower bound' (true element level assumed to be zero) and 'upper bound' (true element level assumed to be the LOR) estimates of intake were also generated and are presented in Appendix 9.

Only 96 different foods were analysed in this study, but there were approximately 4,500 individual foods reported as consumed in the NNS. Therefore the concentrations in the analysed foods were applied to all other consumed foods in a 'best fit' data mapping process outlined in Appendix 7.

Food contribution calculations

The percentage contribution each food group makes to total estimated intakes was calculated by dividing the sum of all individuals' intakes from one food group by the sum of all individuals' intakes from all foods containing the nutrients assessed, and multiplying this by 100.

Assumptions and limitations in dietary modelling

The aim of the dietary intake assessment is to make as realistic an estimate of dietary intake of the trace element of interest as possible. However, where significant uncertainties in the data exist, conservative assumptions are generally used to ensure that the dietary intake assessment does not under- or over-estimate mineral intake. Although improvements have been made to the methods of estimating dietary intake, limitations exist in the methods as well as in the data itself.

Assumptions made in the dietary modelling for the 22nd ATDS include:

- the trace element concentration in the analysed foods is an appropriate representation of the concentration of that element in all foods to which it is mapped, as shown in Appendix 7;
- there is no contribution to intake of the trace elements examined from medicines (including complementary medicines such as vitamin and mineral supplements);
- all of the mineral present in food is absorbed by the body; and
- where a food has a specified trace element concentration, this concentration is carried over to mixed foods where the food has been used as an ingredient.

Particular mention should be made of the assumptions relating to salt in this study. Iodised salt contains, as the name indicates, high levels of added iodine and therefore assumptions about the type of salt consumed has the potential to have a significant impact on estimates of iodine intake. The NNS did not specifically collect accurate data from all respondents on the amount of salt added at the table or in cooking. Therefore, the consumption of salt and, potentially, of iodine from iodised salt, is underestimated. In the small number of cases where NNS respondents reported consumption of salt, or where salt is included as a recipe

ingredient in the DIAMOND program, it was assumed that all this salt was iodised, although industry estimates indicate that only about 15% of household table and cooking salt is iodised (FSANZ, 2006a). The limitations of the NNS data for salt consumption increases uncertainty about the accuracy of estimated iodine intakes.

Use of the 1995 NNS food consumption data provides the best available estimate of actual consumption of a broad range of foods across a nationally representative sample of the Australian population. However, any significant changes to food consumption patterns since 1995 will not be taken into account in this study. Generally, 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 (Cook *et al*., 2001a; Cook *et al*., 2001b).

Comparison of the estimated dietary intakes with reference health standards

Revised Nutrient Reference Values (NRVs) for Australians and New Zealanders were adopted in 2006. They are a collection of reference values for both lower and upper recommended intakes of a range of nutrients, including some trace elements, and therefore can be used to assess both nutrient adequacy and nutrient excess (NHMRC, 2006).

Criteria for estimating the prevalence of inadequate intake within the population

Recommended Dietary Intake's (RDIs) have been established as the level of intake sufficient to meet the needs of 97 - 98% of healthy individuals in the population group to which they refer. Therefore, intakes lying below the RDI do not necessarily indicate an inadequate intake. For this reason, the RDI is not recommended as the criterion for estimating the prevalence of inadequacy in a population. Therefore, the RDIs were not used as a reference health standard in the ATDS.

In this report, the Estimated Average Requirement (EAR) Cut Point Method (NRC, 1986) has been used to estimate the prevalence of inadequate intake. The proportion of the population below the EAR can be used for this purpose if the distribution of nutrient requirements is symmetrical around the EAR and the variance of the intake distribution is greater than the variance of the requirement distribution. For most minerals, this is the case (Health Canada, 2006; Food and Nutrition Board: Institute of Medicine (FNB:IOM), 2000a).

A small percentage of the population (i.e. 3% or less) with intakes below the EAR may be a reflection of the inaccuracies that are inherent in population nutrient intake datasets. Therefore, if less than 3% of a population group has an intake below the EAR, FSANZ considers that the population group as a whole has an adequate intake of the relevant nutrient. When assessing population intakes, two or more subgroups with greater than 3% of intakes below the EAR spread across a broad range of ages has been considered indicative of an inadequate population-wide intake of a nutrient.

When there is inadequate information on which to base an EAR, an Adequate Intake (AI) is used. The AI is based on experimental data or on the median intake in the Australian and New Zealand population from the 1995 Australian and 1997 New Zealand NNS (NHMRC, 2006). When a population group has a mean nutrient intake at or above the AI, this suggests there is a low prevalence of inadequate intake. However, caution must be applied to the interpretation of population intakes in comparison with the AI because it is not known how far above the unknown EAR the AI is located (NHMRC, 2006).

For infants aged less than 1 year, all nutrients have an AI rather than an EAR. With respect to the nutrients examined in the 22nd ATDS, iodine, selenium and molybdenum have EARs whereas chromium has AIs for age groups 2 years and older. Nickel has neither value because it is not regarded as an essential nutrient in Australia and New Zealand.

Criteria for establishing an excessive intake within the population

The Upper Level (UL) of Intake is 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). A UL may also be used to estimate the percentage of the population at potential risk of adverse effects from excessive nutrient intake (NHMRC, 2006). 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.

Not all nutrients have defined ULs owing to lack of data. ULs are not available for nickel or chromium.

Part D Results and Risk Characterisation

Introduction

This section contains information on the estimated middle bound intakes of iodine, selenium, molybdenum, chromium and nickel and on the foods that are major contributors to intake of these nutrients (i.e. contribute more than 5% of nutrient intake). Details of foods that were contributors to iodine, selenium, molybdenum, chromium and nickel estimated dietary intakes are included in Appendix 10.

For risk characterisation of nutrients, both essentiality and toxicity need to be considered. Therefore, both the 5th and 95th percentile of intake are presented, as well as mean intakes.

To reflect the uncertainties associated with the food consumption data, analytical data and dietary modelling methodology, estimated dietary intakes, as a percentage of the reference health standards, have been rounded. Values greater than 100% have been rounded to the nearest 10%, values between 10% and 100% have been rounded to the nearest 5%, and values less than 10% rounded to the nearest 1.

Iodine

Iodine is an essential nutrient required for normal thyroid function, growth and development. Historically, parts of Australia have experienced iodine deficiency due to domestic food supplies being grown in the naturally low iodine soils. Between the 1960s and 1980s, as a result of additional sources of dietary iodine being available, the population was thought to be replete and iodine deficiency was no longer considered a problem. However, recent studies have indicated that mild iodine deficiency has re-emerged over the last 10-15 years (Li *et al*, 2006). As a result, iodine is recognised as being a substance that is of public health significance for Australia (Eastman, 1999; Thomson, 2002; Guttikonda, *et al*., 2002).

About 70-80% of iodine is concentrated in the thyroid gland where it is used to produce the thyroid hormones, thyroxine (T4) and triiodothyronine (T3). These hormones have a key role in influencing cellular metabolism and metabolic rate, regulating body temperature and affecting growth and maturation. These hormones are important for neural development and thus a deficiency in iodine may have particular effects on the brain development of the foetus and young child (Jones, 2002; Gibson, 2005).

Greater than 97% of all iodine consumed is absorbed from the gastrointestinal tract, generally as iodide (Gibson, 2005). The uptake of iodide by the thyroid gland is regulated by

thyroid-stimulating hormone (TSH), which is sensitive to dietary iodine intake. At low intakes representative of iodine deficiency, uptake of iodide is enhanced whereas at very high intakes, iodide uptake into the thyroid gland decreases. Once the physiological requirements for thyroid hormone synthesis have been met, the thyroid does not accumulate more iodide and any excess is excreted, primarily in the urine.

Health effects

Iodine insufficiency and excess both have negative effects on the body through effects on the thyroid gland. The effect on the thyroid depends on the current and previous iodine status of the individual, any current or previous thyroid dysfunction, and the stage in life at which the deficiency occurs.

Nutrient adequacy

A diet deficient in iodine is associated with a range of adverse health effects collectively referred to as iodine deficiency disorders (IDD) (Hetzel, 2000) and has been identified as the single most important cause of preventable brain damage and mental retardation in the world (De Benoist *et al*., 2004).

The most well known consequence of iodine deficiency is goitre, an enlargement of the thyroid gland. Prolonged years of iodine deficiency can lead to adverse changes in the thyroid, which can predispose individuals to thyroid disease (such as iodine-induced hyperthyroidism and thyroid cancer) later in life or following increases in iodine intake (Hetzel and Clugston, 1998; Delange and Hetzel, 2005).

Iodine deficiency can also result in problems ranging from mild intellectual impairment to severe mental retardation. The foetus, neonate, young children, preadolescents and women of child-bearing age are at greatest risk of IDD (Riano Galan *et al*., 2005; Haddow *et al*., 1999; Choudhury and Gorman, 2003; Zimmermann *et al*., 2006; van den Briel, 2000). Impairment occurring during early brain and nervous system development, before the age of two to three years, is irreversible (Hetzel, 1994; Hetzel, 2000). In contrast, impairment resulting from iodine deficiency in later childhood can be largely reversed with adequate iodine intake in childhood or early adolescence (van den Briel *et al*., 2000; Zimmermann *et al*., 2006).

Nutrient excess

Iodine is an essential component of the diet, however, as with many other nutrients, intakes in excess of physiological requirements may produce adverse effects. The human response to excess iodine can be quite variable. Some individuals can tolerate quite large intakes (up to 50 µg/kg/day) while others may respond adversely to levels close to recommended intakes (3-7 µg/kg/day). Individuals responding adversely to relatively low intake levels typically have an underlying thyroid disorder or a long history of iodine deficiency.

There are biological mechanisms to protect against iodine toxicity, including excretion of excess iodide in the urine and preferential production of the more heavily iodinated thyroid hormones. Further iodine excess may disrupt thyroid function, resulting in hyperthyroidism, hypothyroidism and changes in the incidence of thyroid malignancies.

Iodine-induced hypothyroidism is an underproduction of thyroid hormones in response to recently substantially increased or chronically very high iodine intakes (FAO/WHO, 1989a and b); ATSDR, 2004; Delange and Hetzel 2005). The condition, which may or may not be accompanied by goitre, has generally been observed only in populations with either long-term very high iodine intakes or a recent increase in iodine intake from deficient to above adequate or excessive. The most vulnerable are those over 40 years of age who have a long history of iodine deficiency, although individuals with underlying thyroid disorders may also be affected (DeBenoist *et al*., 2004, Delange and Hetzel 2005, Teng *et al*., 2006). Sub-clinical hypothyroidism is defined as an elevation in TSH concentration while serum thyroid hormone concentration is maintained within the normal range of values for healthy individuals. In healthy adults, such an effect has been associated with acute iodine intakes of 1700 µg/day (24 µg/kg body weight/day for a 71 kg person), and for children, has been associated with chronic intakes of 1150 µg/day (29 µg/kg/day for a 40 kg child) (FSANZ, 2005; NHMRC, 2006). Individuals who are particularly susceptible include those with Grave's disease previously treated with iodine; women who have post-partum thyroiditis; or those who have subacute thyroiditis. Globally, the more common cause of hypothyroidism is however not excess iodine, but iodine deficiency (Delange and Hetzel, 2005).

In some areas, such as Japan where high levels of seafood and seaweed are consumed, inhabitants consume as much as 50 to 80 mg/day of iodine. Some of these persons develop goitres, but most maintain normal thyroid function (Beers and Berkow, 2005).

Reference Health Standards

Australian reference health standards for iodine in different population groups have been established by the NHMRC (2006) and are summarised in Table 4. These standards have been used in the risk characterisation for iodine.

EARs are based on studies indicating that iodine balance is achieved at intakes over 100 µg/day but not below 40 µg/day in adults. For children, EARs were also based on balance studies in addition to some extrapolations from studies conducted in adults. AIs for 0-6 and 12 months infants (90 and 110 µg/day, respectively) and EARs for pregnancy and lactation (160 and 190 µg/day, respectively) have also been established by the NHMRC (2006). The AIs for infants are based on the average intake of iodine in breast milk (FAO/WHO, 2001).

ULs are based on elevated TSH concentrations, which are the first sign of iodine excess (NHMRC, 2006). The UL in adults is based on the lowest observed adverse affect level (LOAEL) of 1700 µg/day in adults following supplementation with iodine. ULs for adolescents and children were extrapolated from this finding based on metabolic bodyweight.

Sources

Diet is the major source of iodine intake for humans, although medicines may also contribute to iodine intake (FAO/WHO, 1989b). The oceans are the most important source of natural iodine. Iodine in seawater enters the air and is then deposited onto soil, surface water and vegetation. Some areas in Australia have soils with very low levels of iodine resulting from leaching caused by snow, rain, irrigation and fertilisers (NHMRC, 2006). The iodine content of food reflects background levels in the environment (e.g. soil), which means that fish and seaweed have relatively high concentrations while vegetables, fruit and cereals grown in soils of low iodine content will be poor dietary sources unless treated with iodine containing fertilisers.

Food processing and cooking may produce varying levels of iodine loss in the food (Wang *et al*., 1999, reviewed in Thomson, 2004a). A number of vegetables such as cabbage, broccoli, cassava and cauliflower contain substances known as goitrogens which interfere with iodine uptake in the thyroid gland (Vannort & Thomson, 2005; Jones, 2002).

From the 1960s, milk became an important source of dietary iodine in Australia due to its contamination from iodine-based disinfectants used by the dairy industry (Thomson, 2002). These have gradually been replaced by more effective non-iodine containing disinfectants. Iodine is still used in agricultural practice in some parts of Australia and milk is still considered an important source of iodine where these practices exist (Seal, 2004). Iodised household salt has been available in Australia since the 1920s (Thomson, 2002), however the use of added salt in Australian diets has gradually decreased (Guttikonda *et al*., 2002). Most salt in Australian diets is now consumed through processed food however the food industry does not generally use iodised salt in food preparation and production (Eastman, 1999; Li, *et al*., 2006). These changes in the iodine content of the food supply and consumption of salt appear to have led to falling levels of iodine intake by the Australian population and precipitated the re-emergence of iodine-deficiency in some areas of Australia (Eastman, 1999).

Study findings

Iodine content of food

The foods with the highest iodine concentrations were iodised salt, nori sheets (seaweed), seafoods (including prawns, fish fillets and canned salmon), boiled eggs and cheese. The mean, maximum and minimum levels of iodine in foods are shown in Appendix 5 (Table A5.1).

A considerable proportion of the foods analysed in this study did not contain quantifiable levels of iodine. For example, one quarter of foods, particularly fresh fruits and vegetables and some fresh meats, did not contain quantifiable iodine levels in any of the composite samples analysed.

Although large variations in soil and water iodine contents have previously been found, the analytical results of this study did not find large regional differences in iodine levels in food in Australia. The most significant variations in iodine levels, in terms of potential impact on intake, were found in water and iodised salt.

Summary of estimated intakes

Tables 2-3 and Figure 1 show the estimated iodine intakes for all population groups assessed.

For all age groups assessed, males had higher mean iodine intakes than females, reflecting higher overall food consumption by males. Among males, mean consumption was highest in those aged 9 – 29 years. Intake was relatively constant with age for females, declining slightly in the over 70 years group.

Table 2: Estimated dietary iodine intakes (µg/day) for infants and age gender groups 2-18 yrs.

Iodine	Infant	2 – 3 Male	2 - 3 Female	4 – 8 Male	4 - 8 Female	9 – 13 Male	9 - 13 Female	14 – 18 Male	14 – 18 Female
5th Percentile	-	62	58	62	55	67	58	79	51
Mean	58	101	88	105	84	117	91	139	94
95th Percentile	145	150	130	162	126	212	146	231	156

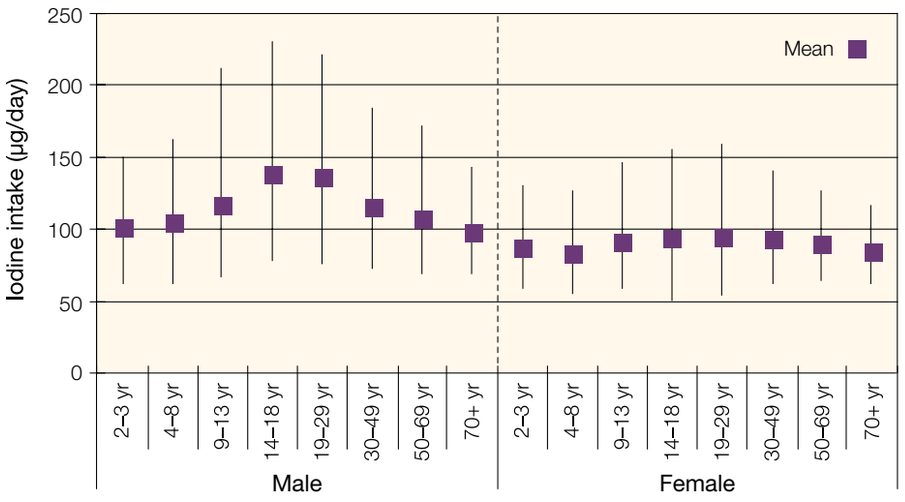
Note: Numbers have been rounded to the nearest whole number; infant estimated intakes calculated differently

Table 3: Estimated dietary iodine intakes (µg/day) for age gender groups 19-70+ yrs.

Iodine	19 - 29 Male	19 - 29 Female	30 - 49 Male	30 - 49 Female	50 - 69 Male	50 - 69 Female	70+ Male	70+ Female
5th Percentile	76	54	72	62	69	64	69	62
Mean	134	95	116	93	108	90	98	85
95th Percentile	221	159	184	141	171	126	143	117

Note: Numbers have been rounded to the nearest whole number

Figure 1: Mean and range (5th to 95th percentiles) of iodine intake by age and sex, Australia



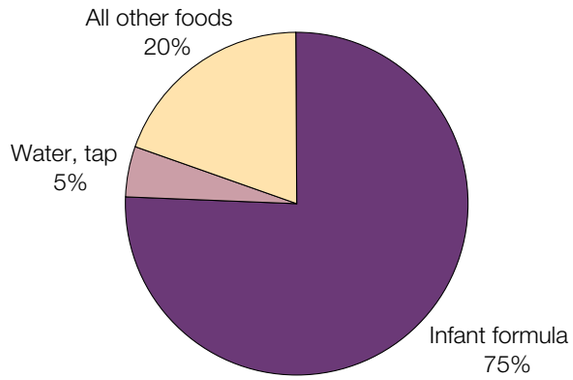
Major contributing foods

Dairy products were the major source of iodine intake for all age and gender groups assessed owing to their widespread consumption in relatively large quantities. Within the dairy category, milk was the dominant iodine source and was more important in the diets of children than adults. Other food categories that made important contributions to dietary iodine intake were eggs, tap water and iodised salt. However, as noted earlier, incomplete reporting of salt consumption in the NNS results in considerable uncertainty about the actual contribution of iodised salt to iodine intake.

Although a good source of iodine, seafood is not widely consumed by the majority of the population on a frequent basis and therefore does not make a major contribution to iodine intakes. The major foods contributing to iodine intake are detailed in Table A10.1 and A10.2 of Appendix 10 and are summarised below in Figures 2, 3 and 4.

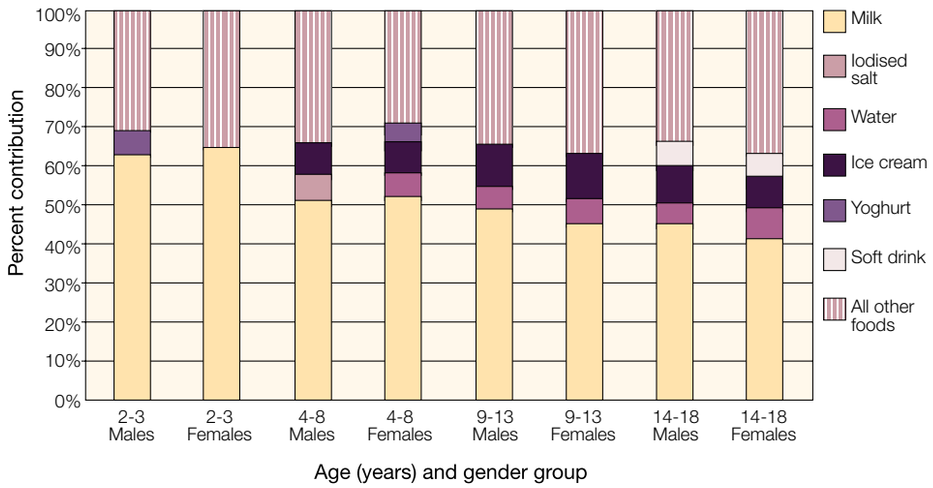
For infants, infant formula provided 75% of the estimated iodine intake (see Figure 2).

Figure 2: Major contributing foods to iodine intakes for infants aged 9 months.



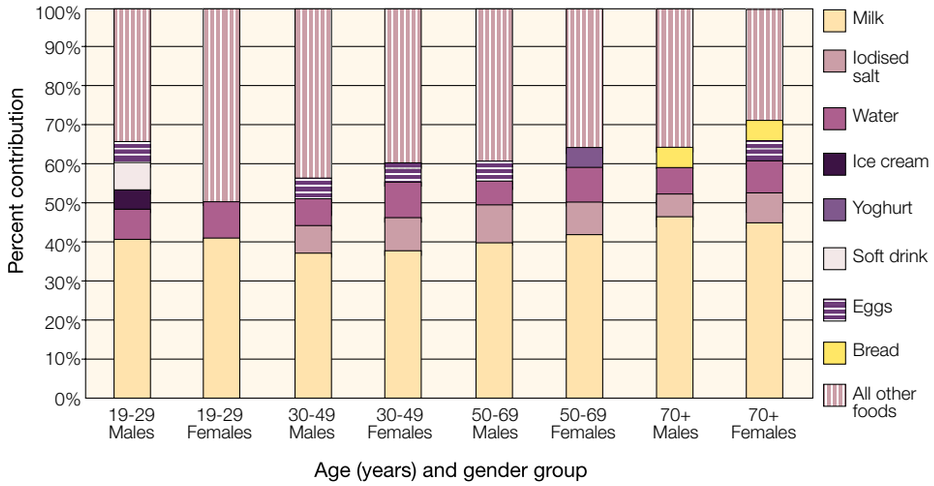
Note: Foods contributing <5% to total intake are included in “other foods”

Figure 3: Percentage contribution of the major contributing foods to iodine intake for children aged 2-18 years



Note: The total iodine intake differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Figure 4: Percentage contribution of the major contributing foods to iodine intake in adults aged 19 years and above.



Note: The total iodine intake differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Risk characterisation

Nutrient adequacy

Due to the absence of an EAR for iodine in Australia or New Zealand for infants, no risk characterisation has been performed. The mean iodine intake for 9 month infants was 58 µg/day, and the AI for this age group (7-12 months) is 110 µg/day. While it is understood that a mean intake at or above the AI suggests low prevalence of inadequate intakes, no accurate conclusions can be drawn regarding values below the AI, as the position of the AI in relation to the EAR is unknown (NHMRC, 2006).

Table 4 shows that between 7 and 84% of the various population groups had inadequate dietary intakes of iodine. There was a trend for this proportion to increase with age such that the prevalence of inadequate intakes was greater than 50% among women aged 19 and older and among men aged 50 years and older.

These data suggest that inadequate iodine intakes are widespread in Australia. As the iodine requirements of pregnant and lactating women are higher than other population groups, these data further suggest that a substantial proportion of women of child-bearing age enter pregnancy with inadequate iodine status.

Nutrient excess

Given that there appears to be inadequate dietary iodine intake in Australia, it is unremarkable that there is no evidence of excessive iodine intake in terms of the exceedance of the UL. A very small (0.8%) proportion of males aged 4-8 years exceeded the UL of 300 µg/day for this age/gender group. However, as this UL is a highly conservative estimate based on an extrapolation from adults using metabolic bodyweight, it is not considered to be of concern.

On this basis, there is no evidence to suggest that intake of iodine by the Australian population exceeds safe levels.

Further consideration of iodine status of Australians

Since commissioning this study, FSANZ has undertaken a detailed assessment of the iodine status of Australians and of iodine intakes (FSANZ, 2006a).

Table 4: Estimated proportion of Australian population groups with inadequate or excessive iodine intakes, assessed by comparison with the relevant EAR and UL respectively

Population group	EAR (µg/day)	Proportion of population with iodine intakes ≤ EAR (%)	UL (µg/day)	Proportion of respondents with iodine intakes ≥ UL (%)
Males 2-3 yrs	65	9	200	0
Females 2-3 yrs	65	12	200	0
Males 4-8 yrs	65	7	300	0.8
Females 4-8 yrs	65	20	300	0
Males 9-13 yrs	75	10	600	0
Females 9-13 yrs	75	34	600	0
Males 14-18 yrs	95	20	900	0
Females 14-18 yrs	95	39	900	0
Males 19-29 yrs	100	27	1100	0
Females 19-29 yrs	100	66	1100	0
Males 30-49 yrs	100	41	1100	0
Females 30-49 yrs	100	73	1100	0
Males 50-69 yrs	100	51	1100	0
Females 50-69 yrs	100	78	1100	0
Males 70+ yrs	100	64	1100	0
Females 70+ yrs	100	84	1100	0

Selenium

Selenium is a naturally occurring trace element and essential nutrient. Selenium is used in the body in the synthesis of several proteins which include the glutathione peroxidases, the iodothyronine deiodinases, selenoprotein P and the thioredoxin reductases (Thomson, 2004b; Janghorbani *et al*., 1999). It is an active component in various enzymes (de Jong *et al*., 2001), and has key roles in redox reactions, energy metabolism and in protection from oxidative DNA damage (de Jong *et al*., 2001; Daniels, 2004). The selenoproteins also maintain cell membrane integrity and support normal thyroid hormone homeostasis, immunity, and fertility (FAO/WHO, 2001; Thomson, 2004b).

The absorption of dietary selenium in the body depends to an extent on its chemical form (Hunt, 2003) but is likely to be around 55 – 70% from foods for the major dietary forms of the element (NHMRC, 2006).

Health effects

Nutrient adequacy

There is little direct evidence linking selenium inadequacy and health problems however, selenium depletion has been associated with viral infections, impaired reproduction, mood, thyroid function, cardiovascular disease, and inflammatory conditions (Combs, 2001; Daniels, 2004). In China, selenium deficiency has been reported to occur in humans as Keshan disease which is an endemic fatal cardiomyopathy (Daniels, 2004). Incidence of Keshan disease has been reported in regions of China where children consume around 7 µg/day of selenium.

Nutrient excess

Selenium's toxic effects were recognised before its essentiality (Whanger, 2004). Indications of chronic selenium poisoning (selenosis) include brittleness and loss of hair and nails, skin lesions, gastrointestinal disturbances and effects on the nervous system (FNB:IOM, 2000b). The estimated selenium intake associated with selenosis in adults is 0.91 mg/day (0.02 mg/kg bw/day). This figure is based on studies of people living in areas of the US and China with selenium-rich soil, as selenium content of food plants is directly related to levels of selenium in the soil. Supplementation trials suggest that 0.2 mg/day for 10 years, or doses of up to 0.4 mg/day for shorter times, do not produce signs of selenosis. Acute selenium poisoning appears to occur at doses higher than 0.5 mg/kg bw or a single dose of 250 mg (Combs, 2001). Daily intakes below 400 µg (0.4 mg) are considered safe for almost all individuals (NHMRC, 2006).

Reference Health Standards

Australian reference health standards for selenium in different population groups have been established by the NHMRC (2006) and are summarised in Table 7. These standards have been used in the risk characterisation for selenium.

The concentration of selenium in the blood is associated with the activity of glutathione peroxidase in various tissues, which is used as an index of selenium status (IPCS, 1987). The EARs for adults are based on the effects of selenium supplements on the activity of a number of glutathione peroxidises. EARs for children and adolescents were then extrapolated from adult data based on metabolic bodyweight. AIs for 0-6 and 12 months infants (12 and 15 µg/day, respectively) have also been established based on the average intake of selenium in breast milk (FAO/WHO, 2001; NHMRC, 2006).

The UL for adults of 400 µg/day is based on the No Observable Adverse Effect Level (NOAEL) of 800 µg/day for brittleness and loss of hair and nails, gastrointestinal disturbances, skin rash, fatigue and effects on the nervous system, and using an uncertainty factor of two to protect sensitive individuals and because of data gaps. The ULs for infants are based on a NOAEL of 7 µg/kg bw from studies showing that human milk concentrations of 60 µg/L are not associated with adverse effects. ULs for children and adolescents are extrapolated from the infant UL on a body weight basis.

Sources

Concentrations of selenium in plant foods reflect environmental levels, particularly soil concentrations. There are significant geographical variations in the selenium content of soil and food crops in many countries including Australia. There has been speculation that environmental changes and agricultural practices in Australia may be reducing selenium concentrations in soil, which is diminishing selenium intake through the food chain (Daniels, 2004).

In Australia, the main dietary sources of selenium are reported to be cereals (i.e. bread), seafood, poultry and eggs (Daniels, 2004; Thomson, 2004a). Animal products such as fish, organ meats and muscle tissues have high levels of selenium. Vegetables and fruit typically have fairly low levels, however mushrooms and brazil nuts are good plant sources of selenium (Daniels, 2004). Other foods that are sources of selenium include milk, eggs, baked beans, lentils, red kidney beans, almonds and peanuts.

Study findings

Selenium content of food

The foods with the highest selenium concentrations were sheep liver, seafood (including fish fillets, prawns and canned salmon), pork and bacon and eggs. Fruits and vegetables tended to have concentrations at or below the LOR. The mean, maximum and minimum levels of selenium in foods are shown in Appendix 5 (Table A5.2).

Summary of estimated intakes

Estimated middle bound dietary intakes of selenium for all age-gender groups assessed are given in Tables 5-6 and Figure 5.

Among young children (under 9 years), boys and girls had similar mean selenium intakes but after this age, males had higher intakes than females for all age groups assessed, reflecting higher total food consumption by males. Intakes were highest in males aged 19 – 29 years, at a mean of 90 µg/day. Adult female mean intakes were in the range 52 – 57 µg/day.

Table 5: Estimated dietary selenium intakes (µg/day) for infants and age gender groups between 2-18 years

Selenium	Infant	2 - 3 Male	2 - 3 Female	4 - 8 Male	4 - 8 Female	9 - 13 Male	9 - 13 Female	14 - 18 Male	14 - 18 Female
5th Percentile	-	18	32	23	35	27	36	58	37
Mean	14	37	41	48	44	63	48	84	56
95th Percentile	36	70	52	84	57	120	70	124	88

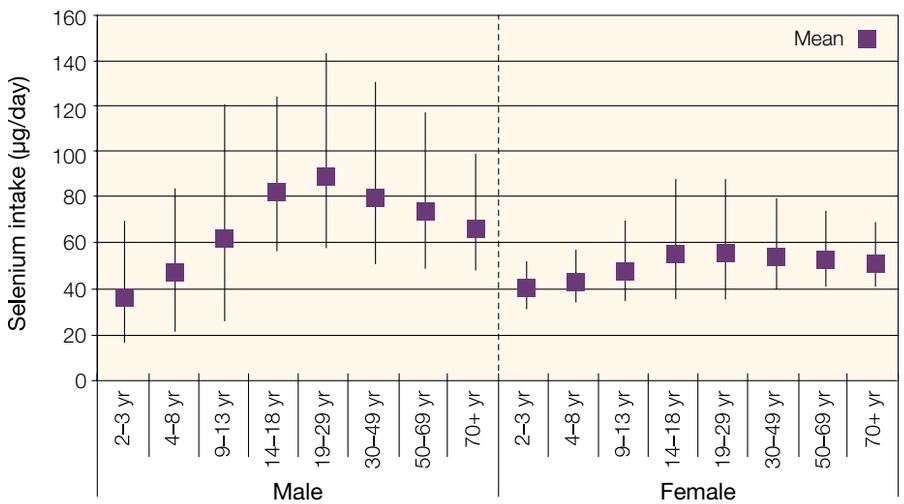
Note: Numbers have been rounded to the nearest whole number; infant estimated intakes calculated differently

Table 6: Estimated dietary selenium intakes (µg/day) for age gender groups 19 years and above

Selenium	19 – 29 Male	19 – 29 Female	30 - 49 Male	30 - 49 Female	50 – 69 Male	50 – 69 Female	70+ Male	70+ Female
5th Percentile	59	37	52	41	50	42	49	42
Mean	90	57	81	55	75	54	67	52
95th Percentile	143	88	130	80	117	74	99	69

Note: Numbers have been rounded to the nearest whole number

Figure 5: Mean and range (5th to 95th percentiles) of selenium intake by age and sex, Australia

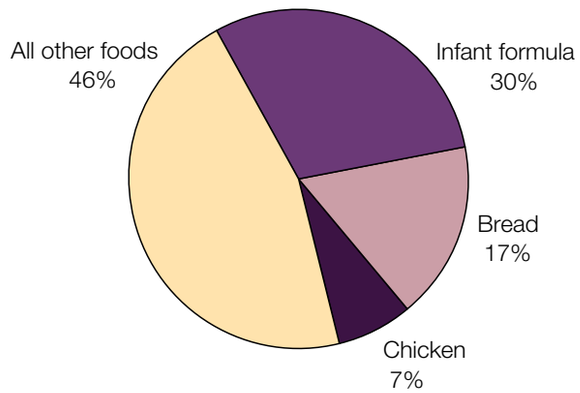


Major contributing foods

Bread was the major contributor to selenium intake for all age and gender groups assessed, other than for infants where infant formula was the most important dietary source of selenium. For all age groups, bread contributed around one quarter of selenium intakes, with other wheat-based foods (e.g. pasta) also contributing selenium. Some animal foods, particularly chicken, beef and eggs, were also important contributors to selenium intake.

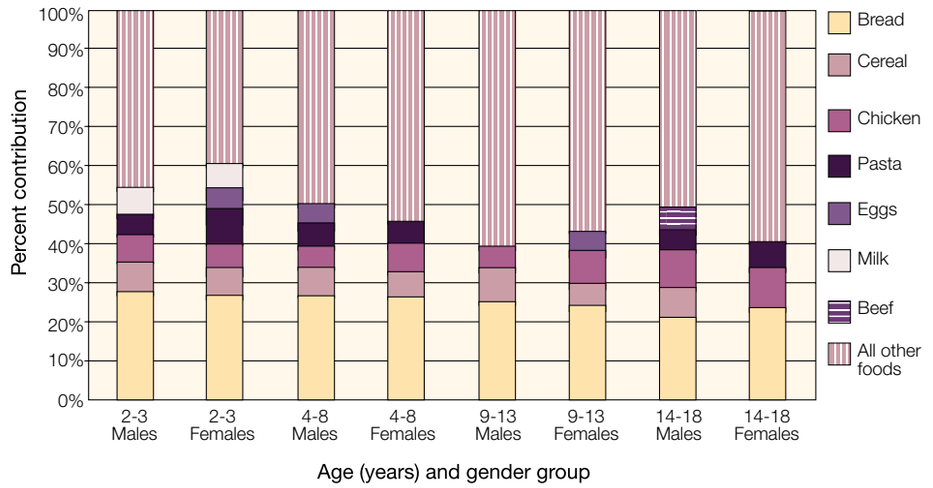
The major foods contributing to selenium intake are detailed in Table A10.3-A10.4 of Appendix 10 and summarised below in Figures 6 through to 8.

Figure 6: Major contributing foods to mean selenium intake in infants aged 9 months



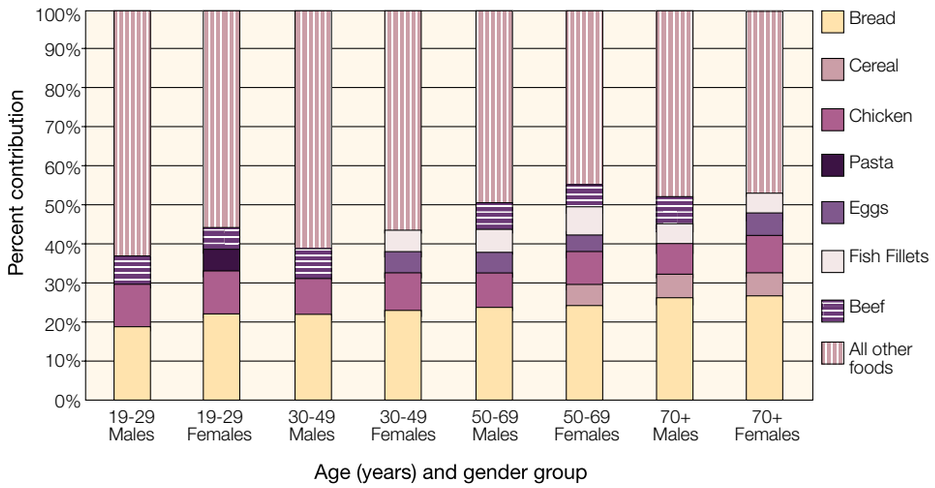
Note: Foods contributing <5% to total intake are included in "other foods"

Figure 7: Percentage contribution of the major contributing foods to selenium intake in children 2-18 years



Note: The total selenium intake differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Figure 8: Percentage contribution of the major contributing foods to mean selenium intake in adults 19 years and above



Note: The total selenium intake differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Risk Characterisation

Nutrient adequacy

Due to the absence of EAR values for selenium in Australia or New Zealand for infants, no risk characterisation has been performed. The mean selenium intake for 9 month infants was 14 µg/day, however the AI for this age group (7-12 months) is 15 µg/day. While it is understood that a mean intake at or above the AI suggests low prevalence of inadequate intakes, no accurate conclusions can be drawn regarding values below the AI, as the position of the AI in relation to the EAR is unknown (NHMRC, 2006).

Table 7 indicates that the prevalence of inadequate intakes ranged from 0% to 56% across the various population groups, with higher proportions of females having intakes below the EAR than males. However, mean intakes for all population groups were above their respective EARs. It is worth noting that the EARs are not based on signs of overt deficiency but rather on the activity of various glutathione peroxidises. So, while the current results suggest that a proportion of the population may have lower than optimal enzyme activity, they do not necessarily indicate widespread deficiency.

Nutrient excess

A very small proportion (≤0.6%) of males aged 2-3, 4-8, 9-13 and 14-18 years had intakes greater than their respective ULs. However, as the ULs are highly conservative estimates extrapolated from infant ULs, which were derived using an uncertainty factor of one, this finding is not considered to be of concern. All other groups had selenium intakes below their respective ULs.

On this basis, there is no evidence to suggest that intake of selenium by the Australian population exceeds safe levels.

Comparison with earlier estimates of selenium intake

In the 20th ATDS (FSANZ, 2003), FSANZ reported intakes of selenium, based on analysis of a smaller range of foods than in this study, although including many foods in common with this study. Intakes estimated in the 20th ATDS were generally around 20% above those estimated in this study. While some of this difference is likely to represent methodological issues such as assessment of different age groups and use of different data mapping techniques, a comparison of common foods suggests that higher selenium levels were found in a range of foods analysed in the 20th ATDS than in the same foods in this study. Appendix 13 compares selenium levels found in the 20th and 22nd ATDS, for foods common to both studies.

Table 7: Estimated proportion of Australian population groups with inadequate or excessive selenium intakes, assessed by comparison with the relevant EAR and UL, respectively

Population group	EAR (µg/day)	Proportion of respondents with selenium intakes ≤ EAR (%)	UL (µg/day)	Proportion of respondents with selenium intakes of ≥ UL (%)
Males 2-3 yrs	20	10.0	90	0.6
Females 2-3 yrs	20	0.0	90	0.0
Males 4-8 yrs	25	7.2	150	0.6
Females 4-8 yrs	25	0.0	150	0.0
Males 9-13 yrs	40	23.0	280	0.2
Females 9-13 yrs	40	20.0	280	0.0
Males 14-18 yrs	60	7.9	400	0.3
Females 14-18 yrs	50	42.4	400	0.0
Males 19-29 yrs	60	6.0	400	0.0

Population group	EAR (µg/day)	Proportion of respondents with selenium intakes ≤ EAR (%)	UL (µg/day)	Proportion of respondents with selenium intakes of ≥ UL (%)
Females 19-29 yrs	50	39.8	400	0.0
Males 30-49 yrs	60	16.1	400	0.0
Females 30-49 yrs	50	37.9	400	0.0
Males 50-69 yrs	60	23.2	400	0.0
Females 50-69 yrs	50	44.6	400	0.0
Males 70+ yrs	60	37.1	400	0.0
Females 70+ yrs	50	55.6	400	0.0

Molybdenum

Molybdenum is an essential nutrient involved in a number of important bodily processes, such as energy metabolism and the formation of blood, bone and cartilage. Molybdenum acts as a cofactor for certain enzymes (sulphite oxidase, xanthine oxidase and aldehyde oxidase) in humans and animals, which are involved in the breakdown of amino acids, purines and pyridines (EVM, 2003; NHMRC, 2006).

Molybdenum is found in food and water as soluble molybdates and molybdenum containing enzymes are found in many plants and animal organisms.

The human body efficiently absorbs molybdenum in foods. For example, water soluble molybdenum compounds and molybdenum in leafy greens are absorbed at a rate of 40-50% (WHO, 1996). Molybdenum intake from drinking water may possibly be absorbed at a similar rate as food.

A portion of absorbed molybdenum (25%) is rapidly detected in the blood and is found closely associated with erythrocytes. The kidney, liver and bone have been found to have the highest levels. Increased molybdenum exposure does not result in bioaccumulation in the tissues, but increases levels in urine until the excess is removed from the body (Tsongas, *et al*., 1980; Scientific Committee on Food (SCF), 2000).

Health effects

Nutrient adequacy

Molybdenum deficiency is extremely rare and usually the result of a genetic disorder that prevents the synthesis of sulphite oxidase, which leads to an inability to tolerate and break down dietary amino acids (EVM, 2003). Deficiency has also been reported in patients receiving prolonged total parenteral nutrition (TPN), resulting in irritability, headaches, tachycardia, mental disturbances and biochemical abnormalities such as abnormal excretion of sulphur metabolites (Sardesai, 1993). These symptoms were reversed following supplementation. Sulphite toxicity due to molybdenum deficiency was noted in a patient on long-term TPN (Beers & Berkow, 2006).

Nutrient excess

Current evidence suggests that molybdenum has low toxicity in humans, it does not bioaccumulate and is rapidly excreted in urine, with increased intake being balanced by increased urinary excretion. Research also suggests that the more soluble forms of molybdenum have greater toxicity than insoluble or less soluble forms (FSANZ, 2005). Molybdenum toxicity has been observed in animal studies and is common in cattle. In humans, consumption of food or water containing over 100 mg/kg of molybdenum may result in toxicity signs such as diarrhoea, anaemia and elevated uric acid levels (EVM, 2003). Elevated uric acid levels, which are associated with the onset of gout, are believed to result from stimulation of xanthine oxidase by high molybdenum intake. A high incidence of gout has been observed in populations with high dietary molybdenum intakes (Turnland *et al*., 1995). There is also some evidence in animals that high molybdenum intake can impair copper utilisation by preventing the gut from absorbing dietary copper, leading to copper deficiency (NHMRC, 2006).

Reference Health Standards

Australian reference health standards for molybdenum in different population groups have been established by the NHMRC (2006) and are summarised in Table 10. These standards have been used in the risk characterisation for molybdenum.

The EARs for adults are based on the results of balance studies in young men and using a mean bioavailability of 75%. EARs for children and adolescents were then extrapolated from adult data based on metabolic bodyweight. AIs for 0-6 and 12 months infants (2 and 3 µg/day, respectively) have also been established based on the average intake of molybdenum in breast milk (FNB:IOM, 2001).

In the absence of suitable human data, ULs for adults are based on the NOAEL of 0.9 mg/kg bw/day for reproductive effects in rodents and using an uncertainty factor of 30 (to cover intra- and inter-species variation) (NHMRC, 2006).

Sources

Molybdenum is found in most foods with legumes, dairy products and meats being the richest sources (NHMRC, 2006; Sardesai, 1993). It is also present in plant foods such as spinach, strawberries and grains (cereals, wheat germ). However, some 40% of molybdenum content of cereals is lost on milling. In plant foods, levels vary depending on soil content and type; foods grown on neutral or alkaline soil are rich in molybdenum, while those grown on leached acid soil are molybdenum deficient (EVM, 2003). Levels are generally higher in foods grown above ground than those grown under ground (EVM, 2003).

Study findings

Molybdenum content of foods

Molybdenum levels were highest, among the foods analysed, in sheep liver, peanut butter, nori, and soy beverage. The mean, maximum and minimum levels of molybdenum in foods are shown in Appendix 5 (Table A5.3).

Summary of estimated intakes

Estimated middle bound dietary intakes of molybdenum for all age-gender groups assessed are given in Tables 8-9 and Figure 9.

Intakes were similar for boys and girls up to the age of 8 years, but after this age, males consumed more molybdenum than females, consistent with their greater food consumption. For males, intake peaked at 14 – 29 years (117 µg/day), with a pronounced decline after this age. By contrast, intakes of molybdenum by females 14 years and above remained relatively constant, around 75 µg/day.

Table 8: Estimated dietary molybdenum intakes (µg/day) for infants and age gender groups between 2-18 years

Molybdenum	Infant	2 – 3 Male	2 - 3 Female	4 – 8 Male	4 - 8 Female	9 – 13 Male	9 - 13 Female	14 – 18 Male	14 – 18 Female
5th Percentile	-	30	36	35	42	41	44	84	55
Mean	22	79	67	79	68	101	75	115	79
95th Percentile	55	196	119	162	114	207	122	167	126

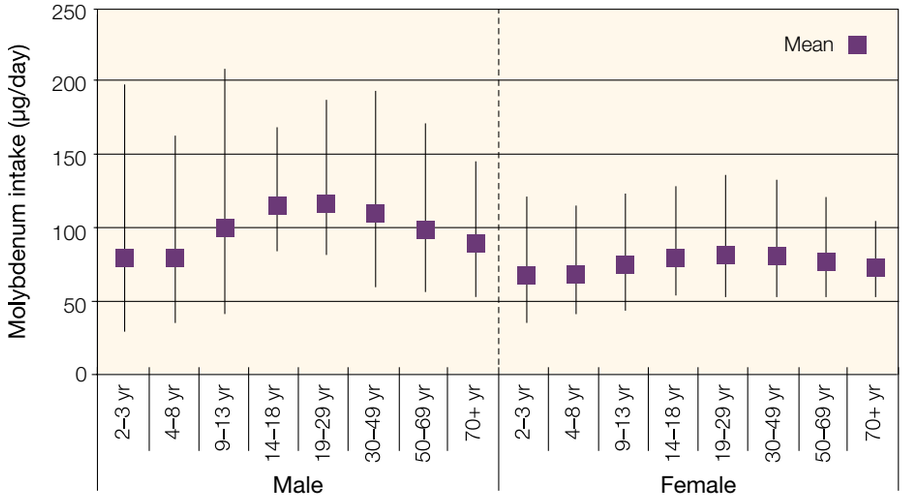
Note: Numbers have been rounded to the nearest whole number; infant estimated intakes calculated differently

Table 9: Estimated dietary molybdenum intakes (µg/day) for age gender groups 19 years and over

Molybdenum	19 - 29 Male	19 - 29 Female	30 - 49 Male	30 - 49 Female	50 - 69 Male	50 - 69 Female	70+ Male	70+ Female
5th Percentile	82	53	61	53	57	54	54	54
Mean	117	82	110	81	100	77	90	73
95th Percentile	186	135	191	131	170	119	144	104

Note: Numbers have been rounded to the nearest whole number

Figure 9: Mean and range (5th to 95th percentiles) of molybdenum intake by age and sex, Australia

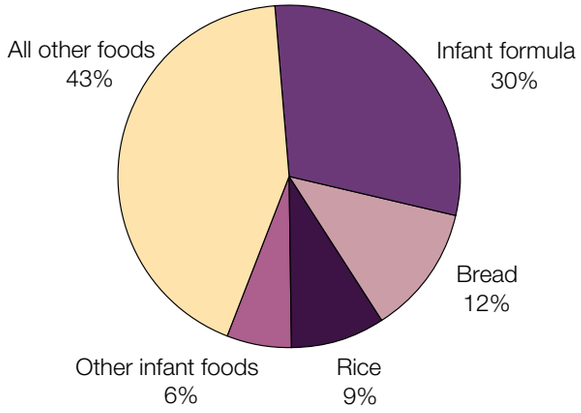


Major contributing foods

Bread, rice and milk (or infant formula in the case of infants) were major contributors to molybdenum intake for all population groups assessed. Those foods identified as being the richest sources of molybdenum tend to be consumed by only a small proportion of the population or in small quantities. However, soy beverages contributed important amounts of molybdenum for children aged 2 – 3 years and for older adults (women above 30 years, men above 50 years).

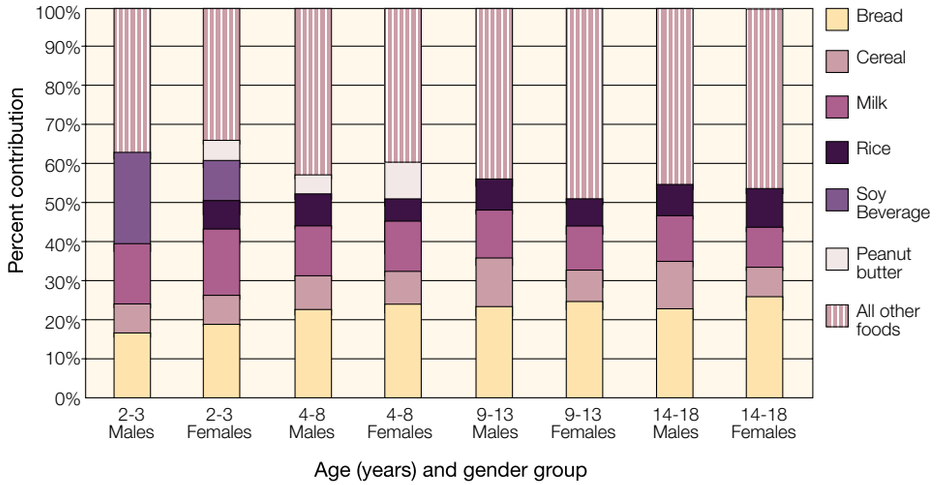
Major foods contributing to molybdenum dietary intake for each age group assessed are summarised in Figures 10 through to 12 (full results provided in Table A10.5-A10.6 of Appendix 10).

Figure 10: Major contributing foods to mean molybdenum dietary intake in infants 9 months of age (%)



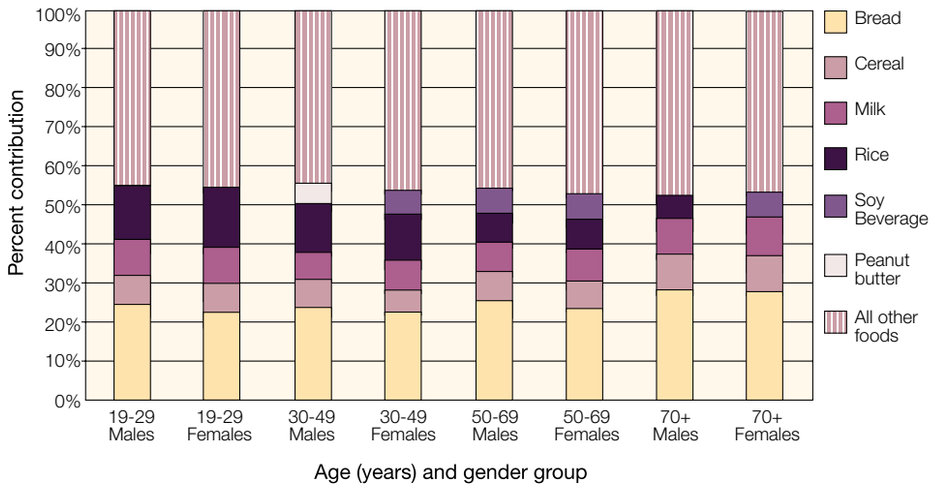
Note: Foods contributing <5% to total intake are included in "other foods"

Figure 11: Percentage contribution of the major contributing foods to molybdenum intake for children aged 2-18 years



Note: The total intake of molybdenum differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Figure 12: Percentage contribution of the major contributing foods to molybdenum intake for adults aged 19 years and above



Note: The total intake of molybdenum differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Risk Characterisation

Nutrient adequacy

Table 10 shows that molybdenum intakes are adequate in the majority of the Australian population when compared to the EAR. Given that the estimated mean and 95th percentile intakes of molybdenum were well above the EARs for all population groups, and only a small proportions of intakes were below the EAR (0.2 – 1.1%), there is not considered to be inadequacy of this nutrient in the Australian population.

Nutrient excess

There is no UL set for infants aged 0-12 months. Table 10 shows that, except for boys aged 2-3 years, no population groups had intakes above the respective ULs. Among boys aged 2-3 years, the prevalence of excess intake was 3.5 %, but as the UL is conservative (based on the most sensitive endpoint in rodent studies and using a large uncertainty factor), such intakes are not considered to indicate a health concern.

Overall, the findings indicate that dietary intakes of molybdenum are not excessive.

Table 10: Estimated proportion of Australian population groups with inadequate or excessive molybdenum intakes, assessed by comparison with the relevant EAR and UL, respectively

Population group	EAR (µg/day)	Proportion of population with intakes ≤ EAR (%)	UL (µg/day)	Proportion of population with intakes ≥ UL(%)
Males 2-3 yrs	13	0.0	300	3.5
Females 2-3 yrs	13	0.0	300	0.0
Males 4-8 yrs	17	0.4	600	0.0
Females 4-8 yrs	17	0.0	600	0.0
Males 9-13 yrs	26	1.1	1100	0.0
Females 9-13 yrs	26	0.2	1100	0.0
Males 14-18 yrs	33	0.0	1700	0.0
Females 14-18 yrs	33	0.0	1700	0.0
Males 19-29 yrs	34	0.0	2000	0.0
Females 19-29 yrs	34	0.0	2000	0.0
Males 30-49 yrs	34	0.0	2000	0.0
Females 30-49 yrs	34	0.0	2000	0.0
Males 50-69 yrs	34	0.1	2000	0.0
Females 50-69 yrs	34	0.0	2000	0.0
Males 70+ yrs	34	0.0	2000	0.0
Females 70+ yrs	34	0.0	2000	0.0

Chromium

Chromium is an essential element involved in carbohydrate and lipid metabolism (Garcia *et al*., 2001). Chromium is abundant in the environment in different oxidation states; trivalent chromium or hexavalent chromium. Trivalent chromium occurs naturally and is the form found in foods and nutrient supplements (Cefalu & Hu, 2004). As chromium in food is always in the trivalent state, this evaluation is limited to this form.

Previously, there has been no national food composition data available for chromium to enable a comprehensive intake assessment to be conducted (NHMRC, 2006). Whilst there are small amounts of data available, these data were either not from Australian sources, were not sufficiently extensive across the whole diet or had not been assessed for accuracy.

The physiological role of chromium is not fully understood, however, evidence suggests that it is involved in lipid and glucose metabolism, the latter through the potentiation of insulin (IPCS, 1988; Lukaski, 2000).

Intestinal absorption of trivalent chromium is low (0.4-2.5%) (FNB:IOM, 2001; NHMRC, 2006). The mechanism of absorption has not been clearly defined, but may involve processes other than passive diffusion (EVM, 2003). Ingested trivalent chromium remains largely unabsorbed and is excreted via the faeces. Absorbed trivalent chromium does not enter blood cells, but binds to plasma proteins such as transferrin and is transported to the liver. Absorbed chromium is mainly excreted via urine, with only small amounts being eliminated in perspiration and bile. Chromium is widely distributed in the body with the highest levels found in bones and organs such as the kidney and liver (Cefalu & Hu, 2004).

Health effects

Nutrient adequacy

Chromium deficiency has been linked to elevated blood glucose, insulin, cholesterol and triglycerides and decreased High Density Lipoprotein (HDL) in humans (Anderson, 1998). More severe symptoms have been seen in patients on Total Parenteral Nutrition (TPN) and can include nerve and brain disorders. Patients receiving TPN nutrition are the only documented cases of chromium deficiency. Epidemiological studies suggest that tissue levels of chromium are reduced among diabetic individuals, especially in those with existing cardiovascular disease, compared with healthy control subjects (Gunton *et al*.,2005).

Nutrient excess

Trivalent chromium has low toxicity, in part because of its low bioavailability (Cefalu & Hu, 2004). Toxicity has been observed in laboratory animals but only following parenteral administration (IPCS, 1988). In fact, large oral doses of trivalent chromium given in food or drinking water (up to 750 mg/kg bw/day) did not cause any adverse effects (IPCS, 1988). Limited data from human supplementation studies have indicated that doses up to 1 mg/day of trivalent chromium compounds in general are not associated with adverse effects (EVM, 2003).

Reference Health Standard

Adequate Intake (AI) levels have been set by the NHMRC (2006) as there are insufficient data available to set EARs. In the absence of Australian or New Zealand intake data or food composition data, these AIs are based on data from the FNB:IOM review (2001). Table 13 lists the AIs used in the risk characterisation for chromium.

The basis of the AI are the results from dietary studies in adults, which were then extrapolated to derive AIs for other population groups. AIs for 0-6 and 12 months infants (0.2 and 5.5 µg/day, respectively) have also been established based on the average intake of chromium from breast milk (FNB:IOM, 2001).

In the absence of suitable data, ULs for the Australian population have not been set.

Sources

Chromium is abundant in the environment and is therefore widely distributed in the food supply. Dietary sources of chromium include meat, fish, legumes, wholegrain cereals, vegetables and yeast (Porter *et al*., 1999). Other sources include egg yolks, spices, cheese, fruits (e.g. apple, orange and pineapple), and peanuts (Cefalu & Hu, 2004).

Study findings

Chromium content of foods

The highest levels of chromium were found in the following foods: chocolate and chocolate cake, ham, parsley and salt. The mean, maximum and minimum levels of chromium in foods are shown in Appendix 5 (Table A5.4).

Summary of estimated intakes

Estimated middle bound dietary intakes of chromium for all age-gender groups assessed are given in Tables 11-12 and Figure 13.

Intakes were similar for boys and girls up to the age of 8 years. After this age, boys consumed larger amounts than girls. There was a large increase with age in males up to 29 years of age and then a pronounced decline with age after 30 years of age. By contrast, there was only a slight decline in mean intake with age among women.

Table 11: Estimated dietary chromium intakes (µg/day) for infants and age gender groups between 2-18 years

Chromium	Infant	2 – 3 Male	2 - 3 Female	4 – 8 Male	4 - 8 Female	9 – 13 Male	9 - 13 Female	14 – 18 Male	14 – 18 Female
5th Percentile	-	14	13	15	13	16	14	23	16
Mean	18	20	18	22	19	26	21	34	22
95th Percentile	43	28	26	33	28	41	30	50	31

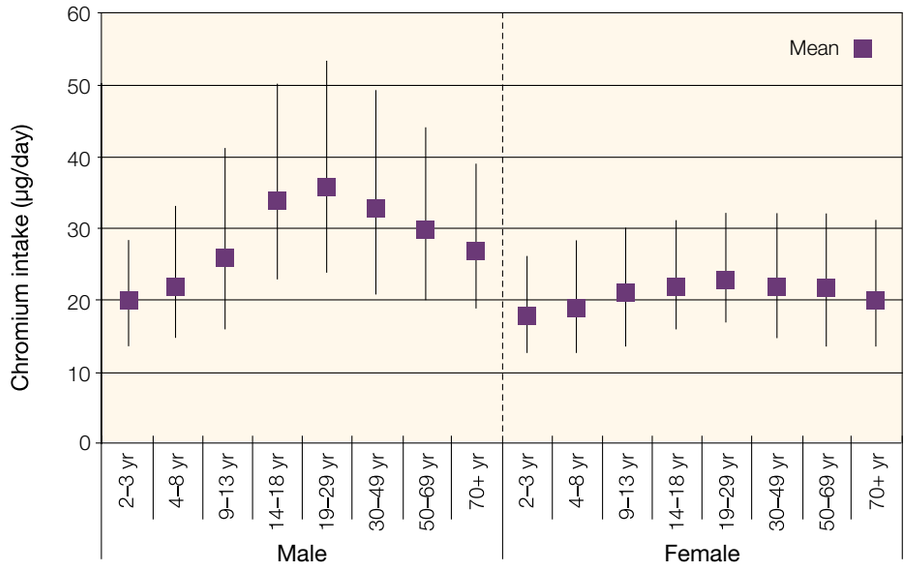
Note: Numbers have been rounded to the nearest whole number; infant estimated intakes calculated differently

Table 12: Estimated dietary chromium intakes (µg/day) for age gender groups 19 years and over

Chromium	19 - 29 Male	19 - 29 Female	30 - 49 Male	30 - 49 Female	50 - 69 Male	50 - 69 Female	70+ Male	70+ Female
5th Percentile	24	17	21	15	20	14	19	14
Mean	36	23	33	22	30	22	27	20
95th Percentile	53	32	49	32	44	32	39	31

Note: Numbers have been rounded to the nearest whole number

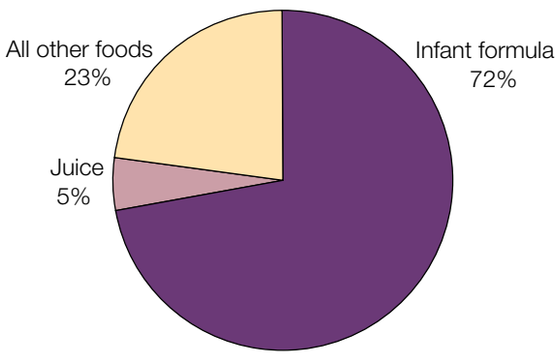
Figure 13: Mean and range (5th to 95th percentiles) of chromium intake by age and sex, Australia



Major contributing foods

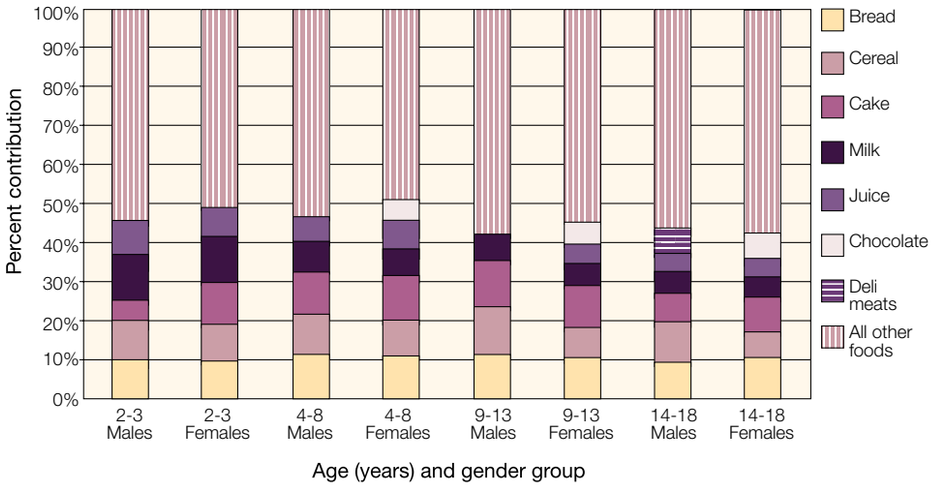
Bread, other cereals, milk and juice were major contributors to chromium intakes for all ages from 2 years, with milk contributing greater proportions of the chromium intake of children than of adults. Infant formula was the major source of chromium in infant diets. For older women, tea contributed approximately 7-13% and for adult males, beer, hamburger meat and delicatessen meats also made important contributions to chromium intake. The major foods contributing to chromium intake are shown in Table A10.7-A10.8 of Appendix 10 and summarised in Figures 14 through to 16.

Figure 14: Major contributing foods to mean chromium intake for infants aged 9 months (%)



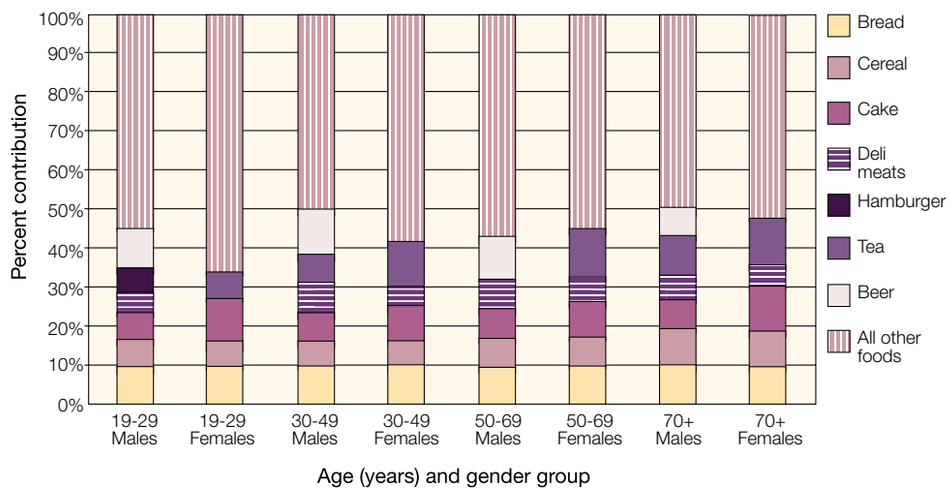
Note: Foods contributing <5% to total intake are included in "other foods"

Figure 15: Percentage contribution of the major contributing foods to chromium intakes for children aged 2-18 years



Note: The total chromium intake differs for each age and gender group; percent contribution was calculated only from day 1 nutrient intakes; Foods contributing <5% to total intake are included in "other foods"

Figure 16: Percentage contribution of the major contributing foods to chromium intake for adults 19 years and above



Note: The total chromium intake differs for each age and gender group; percent contribution was calculated only from day 1 nutrient intakes; Foods contributing <5% to total intake are included in "other foods"

Risk Characterisation

Nutrient adequacy

Due to the absence of an EAR for chromium in Australia or New Zealand, no risk characterisation has been performed.

The mean infant intake of 18 µg/day was above the AI of 5.5 µg/day and only a small proportion of children aged 2 to 8 years had intakes below the respective AIs. While most population groups from 9 years and above had mean intakes below the AI, no accurate conclusions can be drawn regarding values below the AI, as the position of the AI in relation to the EAR is unknown (NHMRC, 2006).

Table 13: Estimated proportion of Australian population groups consuming less than or equal to the AI for chromium

Population group	AI (µg/day)	Proportion of respondents with intakes ≤ AI (%)
Males 2-3 yrs	11	0
Females 2-3 yrs	11	0
Males 4-8 yrs	15	5
Females 4-8 yrs	15	13
Males 9-13 yrs	25	53
Females 9-13 yrs	21	54
Males 14-18 yrs	35	63
Females 14-18 yrs	24	71
Males 19-29 yrs	35	57
Females 19-29 yrs	25	74
Males 30-49 yrs	35	68
Females 30-49 yrs	25	76
Males 50-69 yrs	35	79
Females 50-69 yrs	25	79
Males 70+ yrs	35	90
Females 70+ yrs	25	85

Nutrient excess

In the absence of an Australian UL for chromium, it is unclear whether the current dietary intakes are excessive. However, given that all mean and 95th percentile intakes were well below an intake of 0.15 mg trivalent chromium/kg bw/day (equivalent to 10.5 mg/day for a 70 kg person) concluded by the UK Expert Group on Vitamins and Minerals (2003) to be unlikely to have adverse health effects, it is unlikely that Australian chromium intakes are excessive.

Nickel

Nickel is a trace metal, which is widespread in soil, water and air due to both natural and human processes (IPCS, 1991). Small quantities of nickel are present in food by virtue of its absorption and metabolism by plants and microorganisms. While nickel can be absorbed via inhalation and through the skin, food is the dominant source of exposure for the general population, with water generally being a minor contributor to total intake (FSANZ, 2006b).

Nickel is an essential nutrient for certain microorganisms and is incorporated into several nickel-containing enzymes. To date, several enzymes have been identified including cytochrome-c3 hydrogenase, carbon monoxide dehydrogenase and acetyl–CoA synthase (ExPASy: Bairoch, 2000).

A specific biological function for nickel has not yet been established in humans and, therefore, there is uncertainty about the "essentiality" of nickel as a nutrient (NHMRC, 2006). Metabolism and nutrition studies indicate that nickel deficiency causes a variety of adverse effects in animals (IPCS, 1991; Barceloux, 1999). However, a proportion of the effects seen in nickel-deficient animals are likely to be attributable to effects on gut microorganisms and their nickel-dependent enzymes (eg. decreased urease activity), which are not relevant findings for humans.

In humans, nickel is poorly absorbed from the gastrointestinal tract. Sunderman *et al*., (1989) determined that absorbed nickel was less-than 1% of the dose ingested in food. In contrast, an approximately 30-fold higher absorption occurred when the same dose of nickel was ingested via drinking water. Similar findings have been reported in laboratory animals (IPCS 1991). It is known that nickel absorption can be affected by intake of other foods such as milk, tea, coffee and orange juice (FNB:IOM, 2001).

Health effects

Nutrient adequacy

There is currently no evidence to indicate that nickel inadequacy causes adverse health effects in humans (IPCS, 1991).

Nutrient excess

The toxicity of nickel has been reviewed by a number of agencies around the world including the International Program on Chemical Safety (IPCS) (1991), US EPA, UK Expert Group on Vitamins and Minerals (2003) and the European Agency for the Evaluation of Medicinal Products (EMEA, 1998). Most of the information on the toxicity of nickel to humans comes from occupational exposure studies via the inhalational route, which is not relevant for dietary risk assessment purposes. Oral exposure to nickel has been shown to cause hypersensitivity (e.g. in the form of hand eczema) in people previously sensitised via the dermal route to metallic nickel or nickel salts (Flyvholm, 1984; EMEA 1998), with oral doses as low as 500 µg/day causing a positive response in sensitive individuals (EMEA 1998).

Reference Health Standard

In the absence of evidence for the essentiality of nickel in humans, no reference health standards have been set by the NHMRC.

Sources

There is little information on nickel levels in Australian foods. Rich sources of nickel are chocolate, nuts, legumes and grains (Neilsen, 1991). Plant tissue contains about four times more nickel than animal tissue; therefore, total dietary intake of nickel per day varies depending on the amount of plant and animal food consumed (FNB:IOM, 2001).

Study findings

While the essentiality of nickel in the diet has not been established, the current analysis of nickel in food was undertaken to generate estimates of dietary intakes to assist in any future research in this area.

Nickel content of foods

Nickel concentration levels were highest in peanut butter, desiccated coconut and avocado. The mean, median, maximum and minimum levels of nickel in foods are shown in Appendix 5 (Table A5.5).

Summary of estimated intakes

Tables 14-15 and Figure 17 show the 5th, mean and 95th percentile intakes of nickel for each population group assessed.

Up to the age of 7-8, boys and girls had similar intakes. A divergence appeared during adolescence and led to different levels in adulthood, with men consuming an average of around 150 µg/day and women only around 115 µg/day. Adult intakes in Australia seem to be higher than in the US where adult intakes have been estimated to fall in the range 79-105 µg/day, but lower than Canada where studies report that adults consume between 207-406 µg/day (FNB:IOM, 2001).

Table 14: Estimated dietary nickel intakes (µg/day) for infants and age gender groups between 2-18 years

Nickel	Infant	2 – 3 Male	2 - 3 Female	4 – 8 Male	4 - 8 Female	9 – 13 Male	9 - 13 Female	14 – 18 Male	14 – 18 Female
5th Percentile	-	53	63	62	68	68	69	114	73
Mean	0.3	91	83	100	89	117	93	152	107
95th Percentile	0.7	170	114	168	128	192	129	210	175

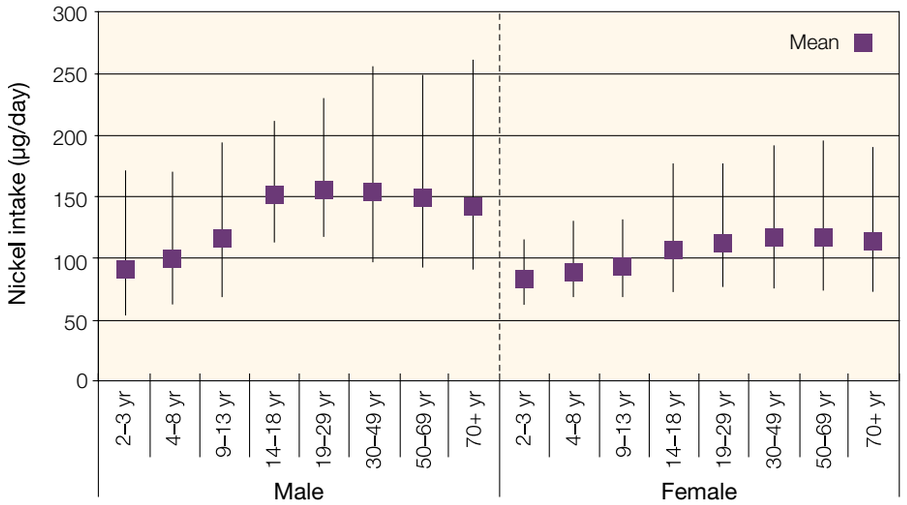
Note: Numbers have been rounded to the nearest whole number; infant estimated intakes calculated differently

Table 15: Estimated dietary nickel intakes (µg/day) for age gender groups 19 years and over

Nickel	19 - 29 Male	19 - 29 Female	30 - 49 Male	30 - 49 Female	50 - 69 Male	50 - 69 Female	70+ Male	70+ Female
5th Percentile	117	77	97	75	92	74	90	73
Mean	156	112	154	117	149	117	143	113
95th Percentile	228	174	254	190	247	193	259	188

Note: Numbers have been rounded to the nearest whole number.

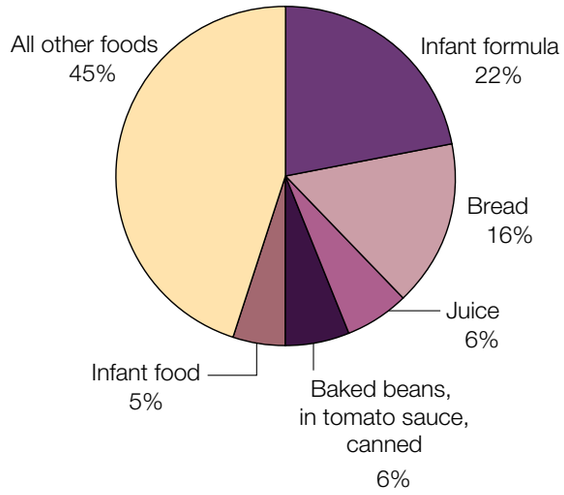
Figure 17: Mean and range (5th to 95th percentiles) of nickel intake by age and sex, Australia



Major contributing foods

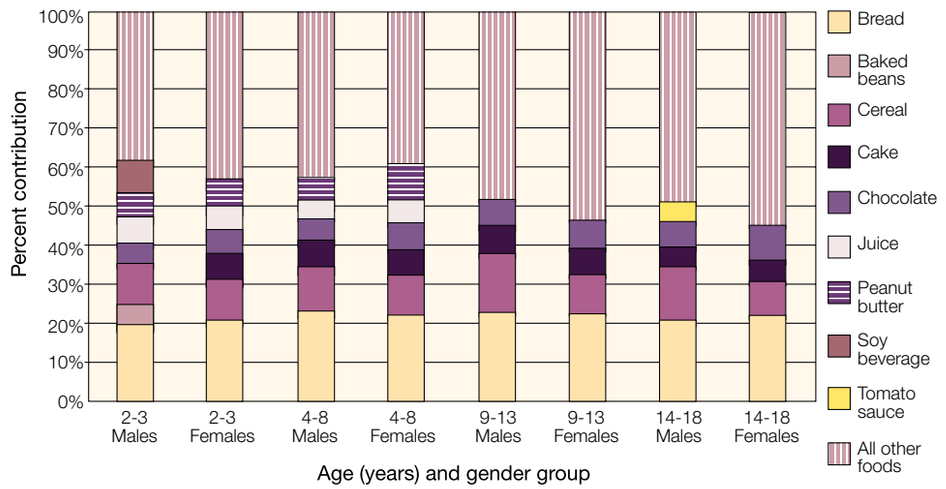
Major foods contributing to nickel dietary intake for each age group assessed are summarised in Figures 18 through to 20 (full results provided in Table A10.9-A10.10 of Appendix 10). The food that was the major contributor to dietary intake to nickel for all population groups aged 2 years and above was bread. Other foods that contributed more than 5% to total dietary intake of nickel, for one or more population groups assessed, were cake, peanut butter, baked beans, orange juice and breakfast cereals. For adults, particularly women, tea was a major contributor to nickel intake. For infants aged 9 months, infant formula was the major source of nickel, but bread was also a major contributor.

Figure 18: Major contributing foods to dietary nickel intake for infants aged 9 months (%)



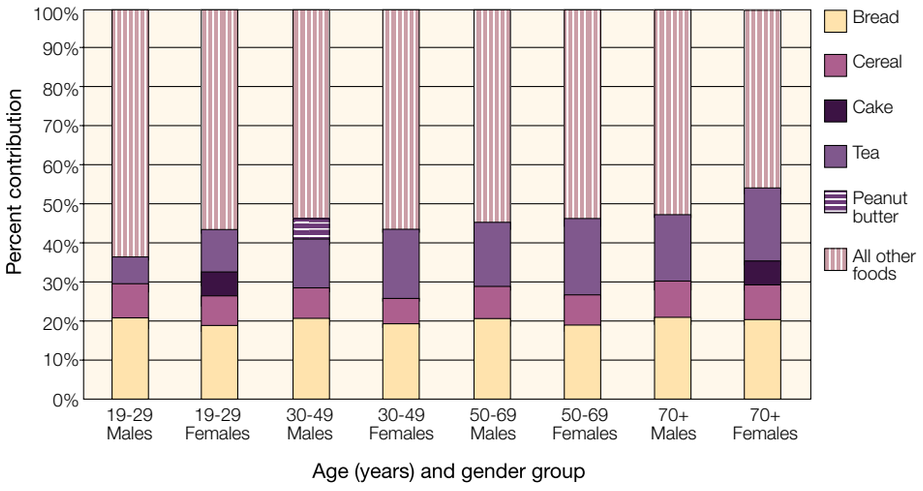
Note: Foods contributing <5% to total intake are included in "other foods"

Figure 19: Major contributing foods to nickel intake for children 2-18 years



Note: The total nickel intake differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Figure 20: Major contributing foods to nickel intake for adults 19 and above



Note: The total nickel intake differs for each age and gender group; Foods contributing <5% to total intake are included in "other foods"

Risk Characterisation

Due to the absence of nutrient reference values for nickel in Australia or New Zealand, no risk characterisation has been performed.

Part E Conclusion and recommendations

The Total Diet Study format was found to be an effective approach for studying the dietary intake of nutrients among the Australian population. Comparing the estimated dietary intakes to the EAR and UL, where these have been established, provides useful indications of the nutritional adequacy/excess for different age gender populations. The contribution of specific foods to the dietary intake of nutrients for different age gender groups were usefully quantified through this study. From a technical perspective, this study is robust and repeatable and it lends itself to international comparisons of nutrient intakes or studying intake trends in the same national population.

In relation to the three minerals investigated for which EARs have been established, for iodine and selenium there is evidence of inadequate intakes in a range of population groups. Intakes of molybdenum appear to be adequate for most Australians. There were no concerns with any of the minerals investigated in relation to excessive intake, to the extent this can be assessed in the absence of reference health standards.

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