The 20th Australian Total Diet Survey
Food Standards Australia New Zealand

The 20th Australian Total Diet Survey

A total diet survey of pesticide residues and contaminants
Foreword

The Australian Total Diet Survey, formerly known as the Australian Market Basket Survey, is Australia’s most comprehensive assessment of consumers’ dietary exposure (intake) to pesticide residues, contaminants and other substances. The survey is conducted approximately every two years, and this is the 20th such survey.

The survey estimates the level of dietary exposure of the Australian population to a range of pesticide residues, contaminants and other substances through the testing of food samples representative of the total diet. These samples were prepared to a ‘table-ready’ form; for example, the potatoes were cooked.

The format and presentation of the survey are similar to the 19th survey, where a short report has been produced with more detailed information provided on the Food Standards Australia New Zealand (FSANZ) web site (www.foodstandards.gov.au). Like the 19th survey, food consumption data derived from the 1995 National Nutrition Survey have been used in the calculation of dietary exposures to pesticides, contaminants and other substances.

The results demonstrate that the levels of pesticide residues, contaminants and other substances in our food are very low, and in all cases they are within acceptable safety limits. The 20th survey has incorporated lower limits of reporting for mercury and antimony in food and this has allowed a more refined dietary exposure assessment to be calculated for these substances than in previous surveys. There were no detections of either aflatoxins or ochratoxin A in nut and cereal products tested. Inhibitory substances, which can indicate the presence of antibiotic residues, were not detected in any of the meat and poultry products, dairy products or eggs tested.

The survey also provides valuable background data that can be used for the development of food regulatory measures. Data from previous surveys were used by the Australia New Zealand Food Authority (ANZFA) during the Review of the Food Standards Code and were integral to the development of standards in Volume 2 of the Australia New Zealand Food Standards Code. The survey is also used by the National Registration Authority for Agricultural and Veterinary Chemicals when considering registration of chemical products.

The results of this survey will be provided to the World Health Organization as a contribution to the Global Environmental Monitoring System (GEMS) that collects data on the levels of pesticide residues and contaminants in the food supply worldwide.
The health authorities and the educational and scientific institutions in the States and the Northern Territory have provided invaluable assistance with this survey and Food Standards Australia New Zealand acknowledges their very important contribution. Expert peer reviewers have also made an important contribution to the preparation of this report.

I am pleased to present the Australian Total Diet Survey as part of Food Standards Australia New Zealand’s commitment to protecting the public health and safety of the Australian food supply.

Rob Knowles
CHAIRMAN
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**Note:** The supplementary information supporting this report is available in five parts. It can be downloaded from the FSANZ website at www.foodstandards.gov.au

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The assistance and advice from the State and Northern Territory health authorities and their staff members have been central to the operation of the 20th Australian Total Diet Survey.

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The Australian Government Analytical Laboratories (AGAL) carried out the analyses and provided advice and comments. Their assistance is greatly appreciated.

The following institutions have either prepared the food or made kitchens available for this survey:

- Edith Cowan University, and the School of Public Health, Curtin University, Western Australia;
- School of Hospitality and Tourism, Palmerston Campus, Northern Territory University, Northern Territory;
- Catering Services, St Johns Park, Department of Health and Human Services, Tasmania;
- Panorama Campus, Douglas Mawson Institute of Technical and Further Education;
- Department of Employment, Training and Further Education, South Australia;
- Queensland Health Scientific Services, Brisbane, Queensland;
- State Chemistry Laboratory, Victoria; and
- Western Sydney Institute of Technical and Further Education, Penrith, New South Wales.

This survey has been peer reviewed and FSANZ would like to thank the following international peer reviewers for their valuable assistance:

- Ms Katie Egan, Centre for Foods Safety and Nutrition, US FDA, United States of America
- Dr Richard Vannoort, Scientist – Food Safety Group, Institute of Environmental Science & Research, New Zealand
# Abbreviations

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<tr>
<td>ADI</td>
<td>Acceptable Daily Intake</td>
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<tr>
<td>AGAL</td>
<td>Australian Government Analytical Laboratories</td>
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<td>AMBS</td>
<td>Australian Market Basket Survey</td>
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<td>ANZFA</td>
<td>Australia New Zealand Food Authority</td>
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<td>ATDS</td>
<td>Australian Total Diet Survey</td>
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<tr>
<td>DIAMOND</td>
<td>Dietary Modelling of Nutritional Data (computer software program)</td>
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<td>FAO</td>
<td>Food and Agriculture Organization</td>
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<td>FSANZ</td>
<td>Food Standards Australia New Zealand</td>
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<td>LOD</td>
<td>Limit of Detection</td>
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<td>LOEL</td>
<td>Lowest Observable Effect Level</td>
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<td>LOR</td>
<td>Limit of Reporting</td>
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<tr>
<td>µg/kg bw</td>
<td>micrograms per kilogram of body weight</td>
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<tr>
<td>mg/kg</td>
<td>milligrams per kilogram</td>
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<td>ML</td>
<td>Maximum level</td>
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<td>MPC</td>
<td>Maximum permitted concentration</td>
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<td>MRL</td>
<td>Maximum Residue Limit</td>
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<tr>
<td>ng/kg bw</td>
<td>nanograms per kilogram of body weight</td>
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<tr>
<td>NHMRC</td>
<td>National Health and Medical Research Council</td>
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<td>NNS</td>
<td>National Nutrition Survey</td>
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<td>NOEL</td>
<td>No Observable Effect Level</td>
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<tr>
<td>NRA</td>
<td>National Registration Authority for Agricultural and Veterinary Chemicals</td>
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<td>NZFSA</td>
<td>New Zealand Food Safety Authority</td>
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<tr>
<td>PTDI</td>
<td>Provisional Tolerable Daily Intake</td>
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<td>PTWI</td>
<td>Provisional Tolerable Weekly Intake</td>
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<td>RDI</td>
<td>Recommended Dietary Intake</td>
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<td>TDI</td>
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<tr>
<td>USEPA</td>
<td>United States Environmental Protection Agency</td>
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<td>WHO</td>
<td>World Health Organization</td>
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**Note:** Definitions for some of these abbreviations can be found in Appendix 5.
Summary

The Australia New Zealand Food Authority (ANZFA) became Food Standards Australia New Zealand (FSANZ) on 1 July, 2002. Food Standards Australia New Zealand is a bi-national statutory authority that develops food standards for composition, labelling and contaminants, including microbiological limits, that apply to all foods produced or imported for sale in Australia and New Zealand.

The primary role of Food Standards Australia New Zealand, in collaboration with others, is to protect the health and safety of Australians and New Zealanders through the maintenance of a safe food supply. Monitoring the food supply for pesticide residues, contaminants and other substances\(^1\) is conducted in both Australia and New Zealand. In Australia, this monitoring was conducted by ANZFA (and now by FSANZ) and in New Zealand, by the Ministry of Health (and from 1 July 2002, by the New Zealand Food Safety Authority (NZFSA)).

FSANZ monitors the food supply to ensure that existing food regulatory measures provide adequate protection of consumer health and safety. The Australian Total Diet Survey (ATDS) is part of that monitoring. It was previously named the Australian Market Basket Survey (AMBS). A total diet survey is also conducted in New Zealand and the New Zealand Ministry of Health have been responsible for administering that survey. Future surveys in New Zealand will be administered by the NZFSA.

The survey

The purpose of the ATDS is to estimate the level of dietary exposure of the Australian population to a range of pesticide residues, contaminants and other substances that can be found in the food supply. Dietary exposure is the intake of pesticide residues, contaminants and other substances from foods consumed. In the ATDS, dietary exposure is estimated by determining the level of the substance in foods by direct analysis, and then multiplying this by the amount of food consumed, as determined in a separate study. In order to achieve more accurate dietary exposure 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.

ANZFA coordinated the survey while the States and Northern Territory purchased and prepared the food samples. The Australian Government Analytical Laboratories (AGAL) carried out all analyses.

Sixty-five types of foods representative of the Australian diet were tested for pesticide residues, contaminants and other substances from foods sampled during July and November 2000 and February and April 2001. These food types incorporate foods central to the Australian diet (core foods), foods that might be expected to show regional variation of residue, contaminant or other substance levels (regional foods), and foods that are available nationwide and are not expected to show regional variation (national foods). These food types were sampled in each of the States and the Northern Territory and some were sampled at four different times throughout the year.

All foods were screened for pesticide residues, including chlorinated organic pesticides, organophosphorus pesticides, synthetic pyrethroids, carbamates and fungicides; as well as antimony, arsenic, cadmium, copper, lead, mercury, selenium, tin and zinc. Breads, biscuits, rice,

\(^1\) The term “other substances” refers to aflatoxins B\(_1\), B\(_2\), G\(_1\) and G\(_2\), and ochratoxin A.
oats, processed wheat bran, breakfast cereals (including infant cereal), instant coffee, peanut butter, almonds and milk chocolate were tested for aflatoxins (B$_1$, B$_2$, G$_1$ and G$_2$) and ochratoxin A. A range of meats, dairy products, eggs, offal meat and infant formula were tested for inhibitory substances (penicillin G, streptomycin and oxytetracycline).

Dietary exposures to pesticide residues, contaminants and other substances were estimated for six age–gender groups. These age-gender groups were infants (9 months), toddlers (2 years), girls (12 years), boys (12 years), adult females (25-34 years), and adult males (25-34 years). Each food in the survey was chemically analysed to measure the levels of pesticide residues, contaminants and other substances. Representative age-gender groups were selected and individual diets for these groups were examined, based on food consumption data from the 1995 National Nutrition Survey (NNS). Dietary exposure to each pesticide residue and metal$^2$ was estimated using the food consumption data and the level of substance present in each food.

The estimated dietary exposure to each chemical from the Australian diet was compared to Australian health standards (Commonwealth Department of Health and Ageing, December 2001). In those cases where there were no Australian health standards, international health standards were used.

Results

The key results from the survey are:

- The estimated dietary exposures to antimony, arsenic, cadmium, lead, mercury, copper, selenium, zinc and tin were within acceptable health standards. Analytical techniques with a lower limit of reporting for antimony and mercury were instituted for this survey and as a result a more refined dietary exposure estimate for these contaminants was achieved than in the 19th survey.

- Aflatoxins (B$_1$, B$_2$, G$_1$ and G$_2$) and ochratoxin A were not found in any food tested, namely: breads, biscuits, rice, oats, processed wheat bran, breakfast cereals (including infant cereal), instant coffee, peanut butter, almonds and milk chocolate.

- A range of meats, dairy products, eggs, offal meat and infant formula were tested for inhibitory substances (penicillin G, streptomycin and oxytetracycline). These substances were not detected in any of these foods.

- The estimated dietary exposures to pesticide residues were all within acceptable health standards.

Both this report and the detailed supplementary information can be downloaded from the Food Standards Australia New Zealand website at www.foodstandards.gov.au.

Conclusion

In conclusion, the 20th ATDS, conducted between July 2000 and April 2001, confirms the overall safety of the Australian food supply and demonstrates that pesticide residues, metals, and selected antibiotics, aflatoxins and ochratoxins are either absent or present in low amounts.

$^2$ The term “metals” has been used to encompass antimony, arsenic, cadmium, lead, mercury, tin, and zinc. Both arsenic and antimony are metalloids and selenium is a non-metal (Bentor 1996-2000) but are grouped with metals for simplicity.
Report recommendations

It is recommended that:

- method development be undertaken to achieve lower LORs for antimony, arsenic, cadmium, lead and mercury. This would allow a more accurate and refined estimate of dietary exposure to be presented in future total diet surveys;
- in future surveys, tin analyses be focussed on canned foods;
- analyses of antimony, arsenic, cadmium, copper, lead, mercury, selenium and zinc, continue to be undertaken in future surveys so that dietary exposure assessments can be undertaken for these substances;
- future surveys should continue to monitor aflatoxins and ochratoxins but this should be targeted to specific foods where these toxins are more likely to be found;
- pesticide residues should continue to be monitored to determine dietary exposure to pesticide residues. Over a number of surveys, a large amount of data relating to pesticide residues has been collected, with the estimated dietary exposures to pesticide residues being well below that of the respective health standards (ADIs). As a consequence, it is recommended that monitoring of pesticide residues be undertaken at a lower frequency in future surveys;
- monitoring of pesticide residues in future total diet surveys should focus on those chemicals for which there are no recent data and should not be limited to those chemicals registered for use in Australia.
Part A  Background

The purpose of the Australian Total Diet Survey (ATDS) is to estimate the level of dietary exposure of the Australian population to a range of pesticide residues, contaminants and other substances that can be found in the food supply. This is estimated by determining the level of the substance in foods by direct analysis of samples collected for the ATDS. The levels are then multiplied by the amount of food consumed to estimate the dietary exposure.

Pesticides are used for agricultural and veterinary purposes for the control of unwanted insects, mites, fungi, rodents, weeds, nematodes and other pests, and for the control of diseases in farm animals and crops.

Pesticides have been used in world agriculture for many years and provide important benefits in agriculture, resulting in a number of benefits to society. Their use provides the community with year-round availability of, and improved quality and variety in, our food supply, and leads to the production of food at a cost to the consumer that would otherwise not be possible.

Although pesticides present the community with significant benefits, there are risks associated with their use. In order to ensure safe pesticide use, a number of Australian government agencies assess the various safety aspects of pesticides before the pesticide is approved for use in Australia. It is FSANZ’s responsibility to assess the safety of pesticide residues in food. Other Australian government agencies assist in this assessment via the provision of information such as Acceptable Daily Intakes (ADIs) and Maximum Residue Limits (MRLs). An ADI is an estimate of the amount of a chemical that can be ingested daily over a lifetime without appreciable risk to health. An MRL is the highest concentration of a chemical residue that is legally permitted or accepted in a food or animal feed and is an indicator of the highest residue that could result from the registered conditions of use. All agencies must be satisfied that the use of the pesticide will result in no appreciable risk of adverse health effects.

Between 1998 and 2000, ANZFA conducted a review of the Australian Food Standards Code (referred to as Volume 1 of the Food Standards Code). In November 2000, the Australia New Zealand Food Standards Code (referred to as Volume 2 of the Food Standards Code) came into effect. Until 20 December 2002 when Volume 2 becomes the sole Food Standards Code, foods must comply with either Volume 1 or Volume 2 of the Food Standards Code, but not a combination of both.

During the review of Volume 1 of the Food Standards Code, the following substances in foods were examined: antimony, arsenic, cadmium, copper, lead, mercury, selenium, tin and zinc (ANZFA 1999). In Volume 2 of the Food Standards Code, maximum levels (MLs) were set only for those contaminants that presented a significant risk to public health and safety and for those foods that significantly contributed to the dietary exposure of the contaminant. The ML is the limit placed on the level of a contaminant, such as a heavy metal, in food. The 20th ATDS examined the metals that were examined during the review of Volume 1 of the Food Standards Code.
Aflatoxins and ochratoxins were included in the 20th ATDS due to the high toxicity of these substances. Additionally, there were detections of aflatoxins in peanuts in the 19th ATDS. Due to recent interest in the presence of antibiotic residues in foods, inhibitory substances were also included in the 20th ATDS.

**Origin of the survey**

In Australia, the National Health and Medical Research Council (NHMRC), at its 68th session held in 1969, recommended 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.

The NHMRC conducted the first total diet survey in 1970. Another 15 surveys were conducted by the NHMRC before responsibility passed to the predecessor of the Australia New Zealand Food Authority (ANZFA), the National Food Authority. The 20th ATDS is the fifth survey to be conducted by ANZFA or its predecessor.

The ATDS is conducted approximately every two years. The sampling and analysis of foods usually take place over 12 months, and the report writing and planning for the next survey take place in the following 12 months. Publication of the report follows peer review of the survey.

**Pesticide, contaminant and other substance surveillance in Australia**

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

- the National Residue Survey; and
- the Imported Food Program, 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 contrast, the ATDS aims to estimate the level of dietary exposure to pesticide residues, contaminants and other substances in the overall Australian diet, including both locally produced and imported foods which are prepared to ‘table ready’ form.

In addition to these programs, State and Territory health and agriculture authorities carry out surveys of specific contaminants, pesticide residues or other substances. These surveys usually investigate specific concerns and determine whether primary producers are complying with the law. They are a valuable source of supplementary information on the contaminant, pesticide residue and other substance status of foods.
Comparison with other surveys

The ATDS differs from other surveys of pesticide residue, contaminant and other substance levels in the following ways:

- The ATDS monitors the level of certain substances in the total diet to determine whether they pose an unacceptable risk to human health. Other surveys examine the level of residues, contaminants and other substances in individual raw agricultural commodities or foods to determine compliance with the law but do not carry out a comprehensive examination of their significance in the diet; and

- The ATDS contrasts with other national surveys in that 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. Food preparation varies 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 food preparation is known to affect the concentration of pesticide residues, contaminants or other substances in the food, an analysis of prepared foods more accurately reflects the levels of residues, contaminants or other substances that are likely to be consumed.

Using information from the survey

Data from the ATDS provide information for developing food regulatory measures. During the review of the Food Standards Code, data from previous Australian Market Basket Surveys were used to supplement dietary modelling information in the risk assessments for metal contaminants (ANZFA 1999). The ATDS data on the dietary exposure to agricultural and veterinary chemicals can be used as a check on exposure assessments undertaken during the registration process at the National Registration Authority for Agricultural and Veterinary Chemicals (NRA).

Caution should be exercised in the direct comparison of the levels of residues, contaminants and other substances found in this total diet survey with food standards since samples are analysed ‘table-ready’ and the sampling protocols used differ from those employed for compliance with food standards.

In addition, the results of the survey are a source of information for Australia’s contribution to the World Health Organization/Food and Agriculture Organization (FAO/WHO) Global Environmental Monitoring System (GEMS), which monitors food contamination internationally; the Codex Committee on Pesticide Residues, the Codex Committee on Food Additives and Contaminants, and independent researchers both inside and outside government agencies.
Conducting the survey

This survey was coordinated by ANZFA and undertaken in cooperation with each of the States’ and the Northern Territory’s departments of health or equivalent. A working group, including liaison officers nominated by each State and the Northern Territory, was formed to advise ANZFA on the food and contaminants to be examined in the survey. Other participants in the working group were representatives of the Australian Government Analytical Laboratories (AGAL) and the National Residue Survey as well as ANZFA staff.

State and Territory officers were responsible for arranging the purchase and preparation of food samples. Food was sampled in each State capital city and Darwin—seven jurisdictions in all. Food was sampled over an entire year in four batches in July and November 2000, and in February and April 2001. This accommodates seasonal variation in foods and allows for the sampling of foods that are available only in certain seasons.

The food was prepared according to strict instructions, frozen and dispatched for analysis. Analytical chemists in the laboratories undertook the chemical analyses of the foods in accordance with quality assurance procedures. Following analysis, the results were sent to ANZFA where the total dietary exposures were estimated and a report prepared. States and Territories were informed of any results that indicated a breach of the Food Standards Code.

Foods included in the survey

The ATDS Working Group chose foods according to the following criteria:

- Representative foods from each major food group\(^3\) and therefore the total foods surveyed must be consistent with a nutritionally acceptable diet;

- The most commonly consumed food in each food group, as shown by the National Nutrition Survey 1995 (NNS). If the food was examined in a recent survey then another representative food from the group may be chosen;

- Foods that may be of particular interest from a pesticide, contaminant or other substance viewpoint may be included in the survey, although their intake may be low. For example, offal and offal products are not a significant component in the Australian diet. However, these products are recognised as typically high in accumulated metal contaminants and pesticide residues and therefore were sampled in the 20\(^{th}\) ATDS;

- Foods may be included if they form a significant part of the diet of a subpopulation of Australians.

The 65 foods surveyed in the 20\(^{th}\) ATDS were chosen according to the above criteria and are shown in Part 1 (Table 1) of the Supplementary Information (FSANZ 2002). All the foods examined in the survey were prepared to a ‘table ready’ state before analysis (refer to Part 5 of the Supplementary Information (FSANZ 2002) for details on food preparation instructions). For example, meats and eggs were cooked, while fruits that are normally consumed without peel were peeled.

\(^3\) The major food groups considered are Breads and Cereals, Fruits and Vegetables, milk and Dairy Products, Meat and Meat Alternatives, and Fats and Oils.
In preparing food as ‘table ready’, local tap water is used rather than distilled water to ensure that pesticide residues, contaminants and other substances that may be present in tap water are taken into account in the overall estimate of dietary exposure.

Foods were sampled according to a schedule that categorises them into core, national or regional foods. This allows a good overview of the Australian diet.

**Core foods** were defined as foods central to the Australian diet. In the 20th ATDS, these foods were bread, beef, eggs, milk, orange juice, margarine, potatoes and tomatoes.

Composite samples of core foods, consisting of four purchases each, were collected in each of Australia’s six States and the Northern Territory in each of the four seasons. This results in 28 composite samples of each core food.

**Regional foods** were defined as those foods that might be expected to show regional variation of residue, contaminant or other substance levels. Regional foods include fruits, vegetables and meats. Three composite samples of these foods, consisting of three purchases each, were collected in each of Australia’s six State capital cities and Darwin, making 21 composite samples for each regional food.

**National foods** were defined as those foods that are available nationwide and are not expected to show regional variation. They are foods, such as sweet biscuits, canned tuna and infant cereal, that are distributed nationwide from a small number of outlets. Three composite samples, of three purchases each, were collected in three capital cities, making nine composite samples for each national food.

**Pesticide residues, contaminants and other substances examined**

All foods were tested for pesticides residues including residues of chlorinated organic pesticides, organophosphorus pesticides, carbamates, synthetic pyrethroids and fungicides (see Part 1 (Table 5) of the Supplementary Information (FSANZ 2002) for a complete list). All foods were tested for antimony, arsenic, cadmium, copper, lead, mercury, selenium and zinc. A selected range of foods was tested for tin. Breads, biscuits, rice, oats, processed wheat bran, breakfast cereals (including infant cereal), instant coffee, peanut butter, almonds and milk chocolate were tested for aflatoxins (B1, B2, G1 and G2) and ochratoxin A. Inhibitory substances (penicillin G, streptomycin and oxytetracycline) were tested for in meats, liver pate, dairy products and eggs.

**Estimating dietary intake of chemical contaminants**

Dietary modelling was used to estimate the exposure to chemical contaminants through the diet for a number of age-gender groups of the Australian population. These age-gender groups were infants (9 months), toddlers (2 years), girls (12 years), boys (12 years), adult females (25-34 years), and adult males (25-34 years).
What is dietary modelling?

Dietary modelling is a scientific method for estimating the levels of pesticide residues, contaminants, or other substances a person or population may be eating. Dietary modelling techniques have been used by food regulators internationally for a number of years to determine if dietary exposure to pesticide residues, contaminants and other substances 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 exposure data for the total diet that can be compared to established reference health standards. The comparison of dietary exposure data to health standards is crucial in identifying whether the estimated dietary exposure to pesticide residues, contaminants or other substances from foods poses an unacceptable health risk to any population group.

A glossary of terms used in determining safe exposures and regulatory limits for pesticide residues, contaminants and other substances is included in Appendix 5.

How is dietary modelling conducted?

DIAMOND (Dietary Modelling of Nutritional Data) is a computer program developed by ANZFA to computerise dietary modelling calculations. The amount of chemical in each food is multiplied by the amount of food consumed and summed over all foods to determine the exposure to the chemical from the whole diet.

Once dietary exposure to the chemical from the total diet has been estimated, this is compared to reference health standards to assess the potential risk to human health. Reference health standards are Acceptable Daily Intakes (ADIs) for pesticide residues and Tolerable Limits for contaminants and other substances. These are the amounts of substances that can be consumed on a daily or weekly basis without appreciable risk.

The chemical levels used in dietary modelling for the ATDS are representative levels taken from the analytical tests on each surveyed food conducted by the AGAL. The data on the amount of foods consumed are taken from the Australian National Nutrition Survey (NNS) that was conducted in 1995 and released in 1998.

A major step in dietary modelling is matching (or mapping) the 65 ATDS foods to the 4053 foods reported as consumed in the food consumption data (the NNS foods). This process assigns the levels of substances detected in the ATDS survey foods to the appropriate food consumption data to estimate dietary exposure to the substance. Given that the ATDS cannot survey all foods in the food supply, a single ATDS food (for example milk) may be assumed to represent a whole group of foods (for example milk, yoghurt and dairy fats) with appropriate adjustment factors for concentration (e.g. the proportion of milk fat in these foods). Recipes are used for mixed foods to assign ingredients to the appropriate ATDS food (e.g. the proportion of milk in vegetables in white sauce). Food mapping is based on traditional nutritional groupings as well as potential or possible pesticide use.
It is recognised that registered pesticide uses may apply only to specific crops (often major crops) in a crop group rather than to the whole group. Therefore, the assumption of a certain residue level, normally measured in the major crop, to the whole group is generally conservative in those cases and may overestimate the amount of potential pesticide exposure.

Use of DIAMOND for dietary modelling brings many benefits. DIAMOND enables the dietary exposure assessments to be conducted more efficiently and accurately. Records from the NNS of actual diets for approximately 13,800 people of all ages (≥ 2 years of age) are used in place of ‘average’ diets that were used in surveys prior to the 19th ATDS. This means that dietary exposure is calculated for each individual in the survey before deriving mean dietary exposure results for the age-gender group. Use of this more up-to-date food consumption data greatly improves the reliability and accuracy of the dietary exposure estimates, and takes account of the different eating patterns of consumers.

**Construction of the infant diet**

As there are no data available from the NNS on children under two years, a diet was constructed to estimate dietary exposure to the food chemicals of interest for infants at 9 months of age. Recommended energy intake for a nine-month-old boy at the 50th percentile weight was used as the basis for the model diet (WHO 1983). Boys’ weights were used because boys tend to be heavier than girls at the same age and therefore have higher energy and food requirements. It was assumed that 50 per cent of the energy intake was derived from milk and 50 per cent from solids (Hitchcock et al. 1986). The patterns of consumption of a two-year-old child from the NNS were scaled down and used to determine the solid portion of the nine-month-old’s diet. Certain foods such as nuts, coffee and alcohol were removed from the infant diet since nuts can be a choking risk (NHMRC 2001a) and coffee and alcohol are unsuitable foods for infants (ACT Community Care 2000). Consumption of breakfast cereals was assumed to be in the form of either infant cereal or single grain breakfast cereals, excluding bran-based cereals. All milk consumption was assumed to be in the form of infant formula.

**Limitations and assumptions in dietary modelling**

Although improvements have been made to the methods of estimating dietary exposure, limitations do exist in the methods as well as in the data itself. For example, we draw conclusions about lifetime eating patterns from food consumption data derived from a single 24-hour diet, leading to conservative dietary exposure estimates. More comprehensive data on multiple-day intakes may provide better estimates of long-term dietary exposure and food consumption.

Assumptions were also made about the value of analytical results below the limit of reporting (LOR). The LOR is the lowest concentration of a chemical that can be detected and quantified, with an acceptable degree of certainty, using a specified laboratory method and/or item of laboratory equipment. In the case of pesticide residues, some results were reported between the limit of detection (LOD) and the LOR. The LOD is the lowest concentration of a chemical that can be qualitatively detected using a specified laboratory method and/or item of laboratory equipment (i.e.
its presence can be detected but not accurately quantified). There is a lower degree of certainty associated with results reported between the LOD and LOR. In the 20th ATDS, reported results below the LOR were used in calculating the mean concentrations of pesticide residues in foods.

In the 20th ATDS, pesticide residue results that were reported as less than LOD were assumed to be zero when calculating the mean concentrations of pesticide residues in foods. Given that pesticides are selectively applied to food crops, it has been assumed that pesticide residues are not present when pesticide residues are less than LOD.

In the case of metal contaminants that occur naturally in the environment, it may not be reasonable to assume that the metal is not present in the food when the analytical results are less than the LOR. For this reason, results below the LOR could be anywhere between zero and the LOR. To allow for this uncertainty, the results for dietary exposure to metals are presented as a range. The lower end of the range was calculated using a median metal concentration, based on the assumption that results below the LOR are equal to zero. The upper end of the range, representing a very conservative ‘worst-case’ estimate, was calculated using a median metal concentration, based on the assumption that results below the LOR are all equal to the LOR.

The DIAMOND program multiplied the mean concentration of each pesticide residue or metal by the amount of food that an individual consumed from that group in order to estimate the intake of a specific pesticide residue/metal from each food. Once this had been completed for all of the foods found to contain a particular chemical, the total amount of the chemical consumed from all foods was summed for each individual. Population statistics (mean intakes) for each age-gender group were then derived from the individuals’ ranked intakes.

**Use of mean concentration levels for pesticide residues**

In choosing a pesticide residue concentration level for use in dietary modelling, FSANZ chose the mean level. Where a high number of results are below the LOD, the mean level is a more conservative indicator of the detected levels of pesticide residues than the median level. This method is also consistent with the approach used in previous surveys.

**Use of median concentration levels for substances other than pesticide residues**

In choosing a concentration level for substances other than pesticide residues for use in dietary modelling, FSANZ used the statistical middle value (median), rather than the mean level (as in surveys prior to the 19th survey), to represent the most likely level in any given commodity. The median level is a more stable central statistic and is not sensitive to skewing by chemical detections above the normally expected range. The median simplifies calculations for surveys containing analytical results below the limit of reporting (LOR) because the position of the median, unlike the mean, is not dependent on the treatment of results below the LOR. Median values were used in the review of metal contaminants in food (ANZFA 1999). Means and medians are generally well correlated where there are few results reported below the LOR. This is demonstrated by the results for copper and zinc, where the means and medians are very similar.
**Previous recommendations**

In the 19th ATDS, it was recommended that the age groups used in calculating dietary exposures be reviewed to ensure that they are in line with changing demographics. The need to consider the age groups was reviewed for this survey. However, changing the age groups would make it difficult to compare the 20th ATDS with previous surveys. For this reason, it was considered appropriate to continue with the existing age groups, particularly since modelling was already being conducted for the group with the highest expected exposure per kilogram of body weight.

The age-gender groups included in this survey were:

- Infants (9 months)
- Toddlers (2 years)
- Girls (12 years)
- Boys (12 years)
- Adult Females (25-34 years)
- Adult Males (25-34 years)
Part B Results

Introduction
The results section of this report has been split into two sections: the first section covers metals\(^4\) and other substances\(^5\) and the second section covers pesticide residues. Within each of these sections there are subsections on each individual substance. The estimated dietary exposures are displayed in the appendices while the background data can be found in Part 1 of the Supplementary Information. The analytical results for metals and pesticide residues in foods are summarised in Parts 2 and 3, respectively, of the Supplementary Information (FSANZ 2002).

All analytical results are expressed in milligrams per kilogram (mg/kg) of the edible portion of food prepared for consumption unless otherwise stated. Dietary exposure estimates for metals are presented as micrograms per kilogram body weight (µg/kg bw) per day. For cadmium, lead, mercury and tin, Provisional Tolerable Weekly Intakes (PTWI) are health references against which dietary exposure is compared since these substances are known to accumulate in animals and humans. To allow comparison to dietary exposure estimates that are presented as a daily estimate, the PTWIs have been converted to a daily intake figure. Dietary exposure estimates for pesticide residues are presented as nanograms per kilogram body weight (ng/kg bw) per day.

Estimated dietary exposures are reported for all individuals surveyed in the 1995 NNS for the respective age-gender group, regardless of whether they consumed the food chemical or not (i.e. “all respondents”). The food consumption and body weight data for each of the age–gender diets are summarised in Part 1 (Tables 3 and 4 respectively) of the Supplementary Information (FSANZ 2002).

Estimated dietary exposures were not reported for high consumers in the 20th ATDS. The 1995 NNS is based on 24-hour food consumption data, and research suggests that such surveys underestimate the food consumption for ‘low consumers’ and overestimate consumption for ‘high consumers’ (Institute of European Food Studies 1998). This is because no one eats the same food in the same amount every day.

Metals
The metals examined in this survey were antimony, arsenic, cadmium, copper, lead, mercury, selenium, tin and zinc. In addition, seafood was analysed for inorganic arsenic and organic mercury.

Copper, selenium and zinc are elements that are essential for health but they can be toxic when exposures exceed certain levels. Recommended Daily Intakes (RDIs) are set for selenium and zinc at levels sufficient to meet the needs of the majority of the healthy population (i.e. to prevent deficiency). There is no RDI for copper. In contrast, tolerable limits are usually set at higher levels than the RDIs and are set at a level below which toxic effects should not occur (i.e. Tolerable Limits are the upper health standard). Consequently both RDIs and Tolerable Limits are discussed for selenium and zinc.

\(^4\) The term “metals” has been used to encompass antimony, arsenic, cadmium, lead, mercury, tin, and zinc. Both arsenic and antimony are metalloids and selenium is a non-metal (Bentor 1996-2000) but are grouped with metals for simplicity.

\(^5\) The term “other substances” refers to aflatoxins B\(_1\), B\(_2\), G\(_1\) and G\(_2\), and ochratoxin A.
Information on the methods of analysis and the levels of metals in the foods analysed is included in Part 4 and Part 2, respectively, of the Supplementary Information (FSANZ 2002). The LORs for each metal are given in Table 1.

Table 1: Limits of reporting for metals

<table>
<thead>
<tr>
<th>Metal</th>
<th>Limit of reporting mg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>0.002</td>
</tr>
<tr>
<td>Arsenic, total</td>
<td>0.01</td>
</tr>
<tr>
<td>Arsenic, inorganic</td>
<td>0.05</td>
</tr>
<tr>
<td>Cadmium</td>
<td>0.005</td>
</tr>
<tr>
<td>Copper</td>
<td>0.01</td>
</tr>
<tr>
<td>Lead</td>
<td>0.01</td>
</tr>
<tr>
<td>Mercury, total</td>
<td>0.002</td>
</tr>
<tr>
<td>Mercury, organic</td>
<td>0.0005</td>
</tr>
<tr>
<td>Selenium</td>
<td>0.01</td>
</tr>
<tr>
<td>Tin</td>
<td>0.01</td>
</tr>
<tr>
<td>Zinc</td>
<td>0.01</td>
</tr>
</tbody>
</table>

All dietary exposure estimates were below the tolerable limit for the metals examined. For metals, the dietary exposure estimates for infants and toddlers were expected to be higher than the other population groups because of their high food consumption relative to body weight and this was apparent in the resulting dietary exposure estimates. The estimated dietary exposures to metals are summarised in both Appendix 1 and Figures 1 to 6.

Figures 1 to 6 represent the dietary exposure to metals as a percentage of the tolerable limit, with each age-gender group represented separately. Information on the tolerable limit of each metal is available in Part 1 (Table 7) of the Supplementary Information (FSANZ 2002).
Figure 1: Range of mean estimated dietary exposure to metal contaminants for adult males (25–34 years) as a percentage of the tolerable limit, based on median analytical results.

Figure 2: Range of mean estimated dietary exposure to metals for adult females (25–34 years) as a percentage of the tolerable limit, based on median analytical results.
Figure 3: Range of mean estimated dietary exposure to metals for boys (12 years) as a percentage of the tolerable limit, based on median analytical results.

Figure 4: Range of mean estimated dietary exposure to metals for girls (12 years) as a percentage of the tolerable limit, based on median analytical results.
Figure 5: Range of mean estimated dietary exposure to metals for toddlers (2 years) as a percentage of the tolerable limit, based on median analytical results.

Figure 6: Range of mean estimated dietary exposure to metals for infants (9 months) as a percentage of the tolerable limit, based on median analytical results.
Antimony

Antimony is found in low-level concentrations in water, soil and air. It is also widely used as an industrial chemical in the manufacture of alloys and in the production of fireproofing chemicals and textiles (ANZFA 1999).

The FAO/WHO Joint Expert Committee on Food Additives has not made any evaluations of antimony and therefore no tolerable limit has been set. However, an oral reference dose for antimony of 0.4 µg/kg bw/day was assigned by the United States Environmental Protection Agency (USEPA 1991). This level has been adopted by FSANZ as a tolerable limit for the purposes of dietary modelling.

The mean, median, maximum and minimum levels of antimony found in foods analysed in the 20th survey are given in Part 2 (Table 8) of the Supplementary Information (FSANZ 2002).

The estimated dietary exposures to antimony for each age–gender category are given in Appendix 1. All estimated dietary exposures were below the tolerable limit for antimony. The highest calculated mean exposure to antimony was for infants because of their high food consumption relative to body weight. The calculated exposure for infants was a wide range (3% to 61% of the tolerable limit). The lower limit was calculated by assuming that foods contained no antimony if they were reported as containing less than the LOR (0.002 mg/kg) and the upper limit was calculated by assuming that foods contained 0.002 mg/kg of antimony if they were reported as containing less than the LOR. The large range results from limitations of the analytical method, which measured antimony levels down to 0.002 mg/kg, and the high proportion of results that were reported as less than the LOR. The actual dietary exposure for antimony lies within this calculated range and it is not possible, with the current method, to be more precise.

In the 19th ATDS, a wider range was reported for antimony dietary exposures and, for some age-gender categories, the range extended above the acceptable health standard. Refinements to the analytical methods and consequent lower limits of reporting for antimony in this survey have enabled a more refined dietary exposure estimate to be made for antimony. The refinements in the 20th ATDS have established that the dietary exposure to antimony for all age-gender categories is within acceptable health standards.

Arsenic

Arsenic occurs naturally in both organic and inorganic forms. Inorganic arsenic is more toxic than organic arsenic. In the past, arsenic compounds were commonly used in drugs, but the more recent major uses are in pesticides, veterinary drugs and industrial applications (WHO 1981). Inorganic arsenic is registered for use in timber preservatives and for control of termites in timber. There are no registered uses in food crops or for animal production. DSMA (disodium methyl arsonate) is registered as a herbicide for turfs and lawns. MSMA (monosodium methyl arsonate) is registered as a herbicide for use in cotton and sugarcane production, on rights-of-way and for non-crop uses.
Generally, most foods contain low levels of arsenic due to its wide distribution in the environment and, to some extent, to its use in agriculture. Dietary arsenic represents the major source of arsenic exposure for most of the population. Some types of seafood contain up to 10 times the arsenic of other foods. People who consume large amounts of seafood may therefore ingest significant amounts of arsenic. The arsenic in seafood is primarily in the organic form.

The 20th ATDS examined total arsenic in all foods and inorganic arsenic in fish portions, fish fillets, prawns, and canned tuna. Inorganic arsenic was only measured in seafood because of the generally higher levels of arsenic that these foods contain and to identify the quantities of the more toxic inorganic arsenic in these foods. The mean, median, maximum and minimum levels of total arsenic and inorganic arsenic found in the foods analysed are given in Part 2 (Tables 9 and 10) of the Supplementary Information (FSANZ 2002). The estimated dietary exposure to total arsenic for each age–gender category is given in Appendix 1.

A level of 0.003 mg/kg bw/day was determined to be the tolerable limit for inorganic arsenic, based on a review of available epidemiological data (ANZFA 1999). Inorganic arsenic analyses are more expensive than total arsenic analyses. To make the best use of the available funds for analytical testing, total arsenic, rather than inorganic arsenic, is determined in most cases. There is no accepted ratio that can be used for all foods to convert the total arsenic content to inorganic arsenic. For this reason and to enable comparison of the results with the tolerable limit for inorganic arsenic, it was assumed that all arsenic detected in each food was in the form of the more toxic inorganic arsenic. This is a significant overestimate because not all arsenic is present as inorganic arsenic. This is demonstrated by the presence of total arsenic at levels above the LOR in all of the seafood samples while inorganic arsenic was not present above the LOR in any of the seafood samples. In the 20th ATDS, the seafood samples surveyed (fish fillets, fish portions, prawns, and canned tuna) contained the highest levels of total arsenic in comparison to the other foods surveyed. The mean level of total arsenic present in seafood was between 4 and 68 times higher than the highest mean level found in non-seafoods. In the 20th ATDS, seafoods contributed to approximately 35% of total arsenic intake for infants, toddlers and girls aged 12 years, approximately 50% for boys aged 12 years, 65% for adult females, and 70% for adult males.

Even with the overestimation for inorganic arsenic content, all estimated dietary exposures to total arsenic were below the tolerable limit for inorganic arsenic. The highest mean exposure to arsenic was for infants because of their high food consumption relative to body weight. This exposure ranged from 12% of the tolerable limit up to 48%. The wide range results from limitations of the analytical method, which measured arsenic down to 0.01 mg/kg, and the significant proportion of results reported as ‘less than the LOR’. Dietary exposures to arsenic are within acceptable health standards.

**Cadmium**

Cadmium is a metallic element that occurs naturally at low levels in the environment. Food, rather than air or water, represents the major source of cadmium exposure, although tobacco smoking adds significantly to the body’s burden. Long-term exposure to high levels of cadmium may lead to
considerable accumulation in the liver and kidneys, particularly the renal cortex, resulting in kidney damage (WHO 1989b).

Additional cadmium has been added to the environment through industrial processes such as cadmium metal production. Further cadmium has been added to agricultural soils through the use of phosphate fertilisers (WHO 1989b) and certain organic fertilisers based on manures.

The tolerable limit for cadmium, which was set at the 33rd meeting, was maintained at the 55th meeting of the FAO/WHO Joint Expert Committee on Food Additives at 7 µg/kg bw/week (WHO 2001b).

The mean, median, maximum and minimum levels of cadmium found in the foods analysed are given in Part 2 (Table 11) of the Supplementary Information (FSANZ 2002). The estimated dietary exposures to cadmium for each age–gender category are given in Appendix 1.

All estimated dietary exposures to cadmium were below the tolerable limit of 7 µg/kg bw/week, and are consequently within acceptable safety standards. The highest mean exposure to cadmium was for infants because of their high food consumption relative to body weight. This exposure ranged from 13% to 68% of the tolerable limit. This range results from limitations of the analytical method, which measured cadmium levels down to 0.005 mg/kg, and the significant proportion of results reported as ‘less than the LOR’.

**Copper**

Copper is widely distributed in nature. Copper can be released into the soil via mining, agriculture and waste from treatment works (WHO 1998). Copper and its compounds have many industrial, urban and agricultural uses. Copper salts, in the form of Bordeaux mixture, have been used since the 19th century as a fungicide for grapes and other crops. Organic growers’ associations consider Bordeaux acceptable for use in organic food production. For non-occupationally exposed humans, oral intake is the major source of copper exposure (WHO 1998).

Copper is an essential element. Enzymes containing copper are important for the body to transport and use iron (WHO 1996). Anaemia is therefore one of the first symptoms of copper deficiency. Copper deficiency, however, is not common (WHO 1998), as copper is widely distributed in food, particularly in meat, liver, kidney, heart and other forms of offal, fish and green vegetables.

Copper is stored in the liver, heart, brain, kidneys and muscles.

In 1996, a joint FAO/International Atomic Energy Agency/WHO expert consultation set an upper limit for the safe range of population mean exposures for adults of 0.2 mg/kg bw/day (WHO 1996). This value has been used as the tolerable limit for the purposes of dietary modelling and was also used during the review of the Food Standards Code (ANZFA 1999).

The mean, median, maximum and minimum levels of copper in foods are given in Part 2 (Table 12) of the Supplementary Information (FSANZ 2002). The estimated dietary exposures to copper for each age–gender category are given in Appendix 1.
All estimated mean dietary exposures to copper are within acceptable health standards. Because of their high food consumption relative to body weight, the highest mean exposure to copper was for infants, calculated at 32% of the tolerable limit. A range has not been presented for copper because a specific amount of copper was reported for almost all samples and so minimal allowance had to be made for results reported as containing ‘less than the LOR’.

**Lead**

Lead is a widely distributed metal, although lead concentrations are low in environments where there has been little human activity. Lead has been used for centuries because it is easily extracted from its ores. Lead is used for a number of industrial, domestic and rural purposes—for example, in lead batteries and in leaded petrol (WHO 2000b).

A significant source of exposure to lead is via the diet (Friberg et al. 1979, WHO 2000b). Lead can be unintentionally added to food during processing. Canned foods can be a source of lead, if lead solder has been used in the can seam. However, most cans now in use in Australia have welded seams. In addition, the level of lead in food has been falling due to technological improvements in food manufacturing.

Lead is a cumulative toxin that can primarily affect the blood, nervous system and kidneys. In the blood at high concentrations, lead inhibits red blood cell formation and eventually results in anaemia (WHO 2000b). The effects of high concentrations of lead on the nervous system can vary from hyperactive behaviour and mental retardation to seizures and cerebral palsy. As the kidneys are the primary route for lead excretion, lead tends to accumulate in these organs, causing irreversible damage.

Infants and children are considered particularly vulnerable to lead exposure. This is due to their higher energy requirements, their higher fluid, air and food intake per unit of body weight, and the immaturity of their kidneys, liver, nervous and immune systems. In addition, their rapid body growth, their different body composition and the development of their organs and tissues, in particular the brain, may increase their lead absorption. Behavioural characteristics of infants and children, such as the sucking of hands and other objects and the ingestion of non-food items (pica) may also result in a higher exposure to lead compared with adults. Dietary lead is not the only source of lead exposure. In particular, other important sources of exposure for infants and children to lead are from lead paint, soil and dust (Friberg et al. 1979).

The tolerable limit for lead, maintained at the 53rd meeting of the Joint FAO/WHO Expert Committee on Food Additives, is 25 µg/kg bw/week (WHO 2000a).

The mean, median, maximum and minimum levels of lead in foods are given in Part 2 (Table 13) of the Supplementary Information (FSANZ 2002). Estimated dietary exposures to lead for each age–gender category are given in Appendix 1.

All estimated mean dietary exposures to lead were below the tolerable limit of 25 µg/kg bw/week and therefore are within acceptable safety standards. The highest mean exposure to lead was for infants because of their high food consumption relative to body weight. The estimated infant
exposure to lead ranged from 1% to 33% of the tolerable limit. This range results from limitations of the analytical method, which measured lead down to 0.01 mg/kg, and the significant proportion of results reported as ‘less than the LOR’.

**Mercury**

Mercury is found naturally in the environment. It is usually found concentrated only in certain areas, geographically known as mercuriferous belts. Apart from industrial activities, mercury is also released into the environment during earthquakes and volcanic activity (WHO 1989a).

Mercury is found in various forms (elemental, inorganic and organic), all of which have different toxicological properties. The most toxic to humans is the organic form, with the most common organic form being methyl mercury. Methyl mercury is largely produced from the methylation of inorganic mercury by microbial activity (WHO 1989b). This is most likely to occur in marine and freshwater sediments. Methyl mercury is rapidly taken up and concentrated by filter-feeding organisms upon which fish feed.

In general, the diet is the major source of exposure to mercury, with seafood containing much higher levels of mercury than most other foods.

The tolerable limit for total mercury, set at the 16th meeting of the Joint FAO/WHO Expert Committee on Food Additives and maintained after reconsideration at the 22nd meeting of the same committee, is 0.3 mg per person per week, equivalent to 5 µg/kg bw/week (WHO 1989b).

In this survey, total mercury, which included both organic and inorganic mercury, was measured in all foods. Mercury (total) was detected in all of the seafood samples. Low levels of organic mercury were found in canned tuna and fish portions. No organic mercury was detected in fish fillets and prawns. The mean, median, maximum, and minimum levels of total mercury and organic mercury in foods are given in Part 2 (Tables 14 and 15 respectively) of the Supplementary Information (FSANZ 2002). Seafood was shown to be the greatest source of mercury in all the diets for all age–gender categories. Of the foods analysed, fish portions had the highest level of mercury. Estimated dietary exposures to mercury for all age–gender categories are given in Appendix 1.

In the 20th ATDS, the estimated mean dietary exposures to mercury for all age–gender groups were below the tolerable limit. Because of their high food consumption relative to body weight, the highest mean exposure to mercury was for infants, where the exposure ranged from 1% up to 35% of the tolerable limit. This range results from limitations of the analytical method, which measured mercury down to 0.002 mg/kg, and the high proportion of samples reported as containing ‘less than the LOR’.

In the 19th ATDS, a wider range was reported for dietary exposures to mercury and, for some age–gender categories, this range extended above the acceptable health standard. Refinements to the analytical methods and consequent lower limits of reporting for mercury in the 20th survey have meant that a more refined dietary exposure estimate for mercury has been achieved.
The refinements have established that the dietary exposures to mercury for all age-gender categories are within acceptable health standards.

In the ANZFA review of Volume 1 of the Food Standards Code (1998-2000), more comprehensive data on mercury levels in food were available than in the 19th ATDS. Estimated dietary exposures to mercury were lower than reference health standards for the general population. There was, however, cause for concern about the potential exposure to mercury for pregnant women consuming large amounts of fish with high mercury levels, because of the sensitivity of the foetus to mercury. As a result of the review, ANZFA developed an advisory statement for pregnant women on mercury in fish, in consultation with health professionals and the fishing industry. This advisory statement (Mercury In Fish: Advisory Statement for Pregnant Women) is available on the Food Standards Australia New Zealand website (FSANZ 2002).

**Selenium**

Selenium is essential to humans at low levels but potentially toxic at high levels of exposure. Selenium is widely distributed in rocks and soils; however, its distribution is uneven.

Selenium was known as a toxicant before being recognised as a nutrient. At high levels of exposure, it may produce symptoms associated with changes in nail pathology and hair loss. Selenium is also essential to humans, in that it helps maintain cell membrane integrity and has an antioxidant role in the body. Selenium deficiency can lead to diseases such as Keshan disease and Kaschin-Beck disease. Both diseases have been reported in selenium-deficient areas, such as parts of China (ANZFA 1999).

The Australian Recommended Dietary Intake (RDI) for selenium for different sub-populations was set by the NHMRC in 1987. The RDIs are 85 µg/day (1.04 µg/kg bw/day) for adult males; 70 µg/day (1.06 µg/kg bw/day) for adult females; 85 µg/day (1.73 µg/kg bw/day) for boys; 70 µg/day (1.35 µg/kg bw/day) for girls; 25 µg/day (1.79 µg/kg bw/day) for toddlers; and 15 µg/day (1.63 µg/kg bw/day) for infants (NHMRC 2001b), based on the body weights for these age groups given in Part 1 (Table 4) of the supplementary section (FSANZ 2002).

As yet, the WHO has made no recommendation regarding tolerable limits of selenium (WHO 1987a). Based on limited human data, the biochemical changes (reduction in the ratio of plasma selenium levels to erythrocyte selenium) linked with exposure of humans to selenium at 750 µg/day is interpreted to represent the first indicator of chronic selenium toxicity and therefore is a Lowest Observable Effect Level (LOEL). A No Observable Effect Level (NOEL) could not be set from human data but is assumed to lie close to the LOEL. Traditionally, the exposure limit for toxicity is determined by dividing the NOEL by a series of uncertainty factors, depending on the level of uncertainty in the information used to determine the NOEL. However, since selenium is an essential element, it requires a different approach for the estimation of maximum tolerable intake levels. This is because there are two ranges of intakes associated with adverse health effects: excessive intake and inadequate levels, both of which may result in illness. It is therefore not usual to use uncertainty
factors in the determination of the tolerable intake levels for essential elements because division of the NOEL (in this case a LOEL) could produce a recommended level which, if followed, could result in deficiency of that element. Chronic selenium intake of 750 µg/day is proposed as the tolerable limit for selenium. This corresponds to an intake of 12.5 µg/kg bw/day for adults, assuming a 60 kg adult body weight. This level was used in the ANZFA review of the Food Standards Code and in the 19th ATDS for dietary modelling purposes (ANZFA 1999). This tolerable limit has also been used for dietary modelling purposes in the 20th ATDS.

The mean, median, maximum and minimum levels of selenium in foods are given in Part 2 (Table 16) of the Supplementary Information (FSANZ 2002). Estimated dietary exposure to selenium for all age–gender categories are given in Appendix 1.

All estimated mean dietary exposures to selenium were below the tolerable limit of 12.5 µg/kg bw/day. Because of their high food consumption relative to body weight, the highest mean exposure to selenium was for two-year-olds, where this exposure ranged from 21% to 24% of the tolerable limit.

All estimated mean intakes of selenium for all age–gender categories are below the suggested tolerable limit of 12.5 µg/kg bw/day. Dietary exposures to selenium are within acceptable health standards.

Estimated dietary exposures to selenium were in the same range as the RDI for each age–gender group (see Table 2). The lower dietary exposure estimates (based on zero values for non-detect results) were lower than the RDI for female adults, boys and girls but exceeded the RDI for male adults, infants and toddlers. The higher dietary exposure estimate (based on LOR numerical values for non-detect results) exceeded the RDI in all cases, except for boys and girls aged 12 years. However, since RDIs are established so that the nutrient requirements of virtually all the population are met, it is likely that actual requirements for selenium will be met for most people in these age groups.

Table 2: Mean estimated dietary exposures to selenium compared with the Recommended Dietary Intake (RDI)

<table>
<thead>
<tr>
<th></th>
<th>Adult males</th>
<th>Adult females</th>
<th>Boys</th>
<th>Girls</th>
<th>Toddlers</th>
<th>Infants</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25–34 years</td>
<td>25–34 years</td>
<td>12 years</td>
<td>12 years</td>
<td>2 years</td>
<td>9 months</td>
</tr>
<tr>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
</tr>
<tr>
<td>RDI*</td>
<td>1.04</td>
<td>1.06</td>
<td>1.73</td>
<td>1.35</td>
<td>1.79</td>
<td>1.63</td>
</tr>
<tr>
<td>Mean Dietary exposure</td>
<td>1.17–1.41</td>
<td>0.96–1.18</td>
<td>1.48–1.66</td>
<td>1.14–1.31</td>
<td>2.61–3.0</td>
<td>2.14–2.41</td>
</tr>
</tbody>
</table>

*RDI expressed per kilogram body weight for each age–gender group (NHMRC 2001b).
**Tin**

Tin is a metal that has been used since ancient times as an alloy in combination with copper to produce bronze. Today tin is used in plating, solders and alloys.

The main route of exposure to tin is through food, although levels are generally low. The main dietary source of tin is food that has been in contact with tin-plate coated cans. Steel cans used in the food industry are coated in either a thin layer of tin (tin-plate) and/or a lacquer. The tin-plate or lacquer acts to prevent corrosion of the steel. Higher levels of tin may be found in canned foods as a result of the tin coating breaking down. The concentration of tin in foods from lacquered cans is generally lower than the concentration of tin in foods from unlacquered cans (WHO 1989c, WHO 2001a).

Toxicity from tin exposure is low. However, high levels of tin exposure may produce acute gastrointestinal disturbances such as nausea, vomiting and diarrhoea. Small children and infants are also more likely to consume high levels of tin from a single source, on a body weight basis (WHO 1989b).

The FAO/WHO Joint Expert Committee on Food Additives, at its 33rd meeting, set a tolerable limit of 14 mg/kg bw/week for inorganic tin (WHO 1989b), and recommended that efforts be made to keep tin levels in canned foods as low as practical, consistent with the application of good manufacturing practice.

Since most foods, with the exception of canned foods, contain low levels of tin, canned foods were the primary focus for sample selection for tin analysis. However, within one sampling period all samples were analysed for tin. The mean, median, maximum and minimum levels of tin in canned foods are given in Part 2 (Table 17) of the Supplementary Information (FSANZ 2002). Estimated dietary exposures to tin for all age–gender categories, based primarily on tin exposure from canned foods, are given in Appendix 1.

All estimated exposures to tin for all age–gender categories are well below the tolerable limit for tin of 14 mg/kg bw/week. Because of their high food consumption relative to body weight, the highest mean exposure to tin was for two-year-olds, where this exposure was approximately 0.6% of the tolerable limit. Dietary exposure to tin is within acceptable health standards.

For canned foods, all results were below the maximum permitted concentrations/maximum limits (MPC/ML) in Standard 1.4.1 of the *Food Standards Code* for tin in canned foods. The MPC/ML is the limit placed on the level of a contaminant in food.

**Zinc**

The major uses of zinc are in the manufacture of non-corrosive alloys and brass, and in galvanising steel and iron.

Zinc is also an essential nutrient that is extremely important to long term health. Zinc is necessary for the function of various enzymes and plays an essential role in DNA, RNA and protein synthesis. The major symptoms of zinc deficiency are delayed growth and slow maturation (WHO 1996).
Zinc is widely distributed in food, with seafood, meat and nuts being good sources of zinc (WHO 1982).

In 1996, the WHO Expert Consultation Committee on Trace Elements recommended that the adult population mean intake of zinc should not exceed 45 mg/day in order to avoid zinc-related interactions (WHO 1996). Since this figure was set to “ensure that very few individuals in a population have an intake of zinc of 60 mg/day or higher”, ANZFA used a tolerable limit of 1 mg/kg bw/day for the purposes of dietary modelling, based on a 60 kg body weight (ANZFA 1999).

The mean, maximum and minimum levels of zinc in foods are given in Part 2 (Table 18) of the Supplementary Information (FSANZ 2002). Estimated dietary exposures to zinc for all age–gender categories are given in Appendix 1.

All estimated mean intakes of zinc for all age–gender categories are below the tolerable limit for zinc. Therefore dietary exposures to zinc are within acceptable health standards. The highest mean exposure to zinc was for infants where the exposure was 63% of the tolerable limit. No range has been presented for zinc because a specific amount of zinc was reported for most samples and minimal allowance had to be made for results reported as containing ‘less than the LOR’.

Estimated dietary exposures to zinc exceeded the Recommended Daily Intake (RDI) for adult males, boys, toddlers and infants. The estimated dietary exposure was lower than the RDI for adult females and girls (refer to Table 3). However, since RDIs are established so that the nutrient requirements of virtually all the population are met, it is likely that actual requirements for zinc will be met for most females in these age groups.

Table 3: Mean estimated dietary exposures to zinc compared with the Recommended Dietary Intake (RDI)

<table>
<thead>
<tr>
<th></th>
<th>Adult males 25–34 years µg/kg bw/day</th>
<th>Adult females 25–34 years µg/kg bw/day</th>
<th>Boys 12 years µg/kg bw/day</th>
<th>Girls 12 years µg/kg bw/day</th>
<th>Toddlers 2 years µg/kg bw/day</th>
<th>Infants 9 months µg/kg bw/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>RDI*</td>
<td>146</td>
<td>182</td>
<td>245</td>
<td>231</td>
<td>321</td>
<td>489</td>
</tr>
<tr>
<td>Mean Dietary exposure</td>
<td>170</td>
<td>127</td>
<td>247</td>
<td>168</td>
<td>368</td>
<td>627</td>
</tr>
</tbody>
</table>

*RDI expressed per kilogram body weight for each age–gender group (NHMRC 2001b).

Aflatoxins and ochratoxins

Aflatoxins and ochratoxins are two classes of mycotoxins. Mycotoxins are substances produced by fungi and they may have detrimental effects in higher organisms.
**Aflatoxins**

Aflatoxins are a group of toxic compounds produced by the fungi *Aspergillus flavus*, *Aspergillus parasiticus* and *Aspergillus nomius*. Aflatoxins exert their toxicity primarily in the liver and are considered to be carcinogens (WHO 1999).

Aflatoxins have the potential to contaminate foodstuffs and animal feeds on which mould has been allowed to grow. They can contaminate maize, peanuts, sorghum, cottonseed, brazil nuts, almonds, walnuts, pecans, filberts, copra, rice, legumes, peppers, potatoes, dried fruits and dairy products. The most pronounced contamination is generally in peanuts, maize, and oilseed including cottonseed. Milk and milk products can also be contaminated by aflatoxins if the dairy herd has been fed contaminated feedstuffs.

Seventeen aflatoxins have been isolated, but only six are significant contaminants of food. These are called B1, B2, G1, G2, M1 and M2. Aflatoxin B1 is usually found in the greatest concentration in foods and is the most toxic of the aflatoxins. Aflatoxins M1 and M2, commonly known as milk aflatoxins, may be found in cow’s milk after the animal has ingested feed containing aflatoxins B1 and B2 (WHO 1999). Milk aflatoxins retain the toxic properties of the parent compound but do not have the same potency.

**Ochratoxins**

Ochratoxins are naturally produced by several species of fungi from the genera *Aspergillus* and by *Penicillium verrucosum*. Both are widely distributed and may be found in grain, legumes and other commodities. Ochratoxin formation by the specific species of *Aspergillus* appears to be limited to conditions of high humidity. *Penicillium verrucosum* grows at temperatures below 30°C, whereas *A. ochraceus* and *A. carbonarius* grow at moderate and high temperatures, respectively (WHO 2001b).

There are two types of ochratoxins: ochratoxin A and ochratoxin B. Ochratoxin A is the most toxic and the highest incidences have been found in cereals and legumes, as well as other commodities. Ochratoxin B contamination occurs very rarely. Both ochratoxin A and ochratoxin B can cause toxicity in the kidneys. Ochratoxin A can also have a carcinogenic effect in the kidneys (WHO 2001b).

**Controlling aflatoxin and ochratoxin contamination**

The best means of controlling the presence of aflatoxins and ochratoxins in animal feeds and food is through good agricultural and manufacturing practices that prevent fungal growth. Mycotoxins are relatively stable compounds and, once formed, can persist in animal feeds and foods. The usual methods of processing peanuts to make peanut butter and processing nuts for confectionery may appreciably reduce aflatoxin contamination. Effective means of reducing contamination include removing undersized nuts, removing nuts that resist splitting and blanching, and removing discoloured nuts by hand or electronic sorting (Cole 1989).
Aflatoxin and ochratoxin results

The following foods were analysed for aflatoxins B1, B2, G1 and G2 and ochratoxin A: breads, biscuits, rice, oats, processed wheat bran, breakfast cereals (including infant cereal), instant coffee, peanut butter, almonds and milk chocolate. These foods were chosen for aflatoxin and ochratoxin analyses because they are more likely to contain these substances than other foods.

Aflatoxins B1, B2, G1 and G2 and ochratoxin A were not detected in any of the foods analysed for these contaminants. The LOR for both aflatoxin and ochratoxin A analyses was 0.001 mg/kg.

No Tolerable Intake has been set for aflatoxins. The WHO suggests that intake of aflatoxins be kept as low as possible (WHO 1987b). A Provisional Tolerable Weekly Intake (PTWI) was first set for Ochratoxin A at the 37th Meeting (1990) of the Joint FAO/WHO Expert Committee on Food Additives, with the PTWI being rounded to 100 ng/kg bw/week at the 44th meeting (1995) and retained at the 56th meeting (WHO 2001b).

Inhibitory substances

Inhibitory substances are those substances that inhibit the growth of bacteria. The presence of inhibitory substances in a food indicates that residues of antibiotics may be present. In the 20th ATDS, the inhibitory substances that were investigated were penicillin G, streptomycin and oxytetracycline. The limits of detection for these substances are 0.025 units/ml organism, 0.2 units/ml organism, and 0.2 µg/ml organism, respectively.

A range of foods from animal sources were tested for inhibitory substances: bacon, thick sausages, minced beef, cheddar cheese, chicken breasts, eggs, infant formula, lamb chops, leg ham, liver pate, milk and ice cream. The listed inhibitory substances were not detected in any of these foods.

Comparison between the 19th ATDS and the 20th ATDS results for metals and other substances

In general terms it is possible to note some differences between the dietary exposures to metals and other substances in the 19th survey and the 20th survey. These differences have not been emphasised because the small number of samples in comparison to the large range of foods in Australia’s food supply mean that it is difficult to draw many definite conclusions.

The key items to note from the exposures in the 20th survey were that:

- the lower limits of reporting for antimony and mercury substantially reduced the upper end of the dietary exposure range for these metals in comparison to the dietary exposure range reported in the 19th ATDS;
- dietary exposure to arsenic, copper, cadmium, lead, selenium, tin and zinc were within acceptable safety standards and were consistent with those determined in the 19th survey;
- dietary exposure to lead was lower than that determined for the 19th survey. In the 19th ATDS, many sugar-containing foods were assigned the lead concentration in honey (0.080 mg/kg) as...
part of the dietary exposure calculation. This was done because honey was assumed to be the food that best represented sugar-containing foods out of all of the foods sampled. In the 20th ATDS, sugar was sampled and no detections of lead were reported (LOR = 0.01 mg/kg). Honey contains higher lead levels (0.080 mg/kg) than sugar (<0.01 mg/kg). Therefore assigning the higher lead concentration reported in honey to all sugar-containing foods (for example soft drinks) resulted in a higher calculated dietary exposure to lead in the 19th ATDS and, based on the information from the 20th ATDS, this was an over-estimate of lead dietary exposure.

- there were no detections of aflatoxins (B1, B2, G1, G2) in the 20th ATDS. In the 19th ATDS, Aflatoxin B1 and Aflatoxin B2 were found in one of the nine analytical samples of roasted salted peanuts.

**Recommendations for metals and other substances**

- Method development should be undertaken to achieve lower LORs for antimony, arsenic, cadmium, lead and mercury. This would allow a more accurate and refined estimate of dietary exposure to be presented in future total diet surveys.
- Analyses of copper, selenium and zinc should be undertaken in future surveys so that dietary exposure can continue to be monitored.
- In future surveys, it is recommended that tin analyses be focussed on canned foods.
- Future surveys should continue to monitor aflatoxins and ochratoxins but this should be targeted to specific foods where these toxins are more likely to be found.

**Pesticides**

Pesticides assist in food production by controlling pests and diseases. Even following good agricultural practices (GAP), residues of pesticides may still end up in the food we eat. However, food handling, storage and food processing, including preparation in the home, can reduce the levels of these residues.

The survey tested for the residues of a number of pesticides in a number of foods. A complete list of the pesticide residues for which foods in the survey were analysed can be found in Part 1 (Table 5) of the Supplementary Information (FSANZ 2002). The range of pesticide residues tested were:

- chlorinated organic pesticides (organochlorines);
- organophosphorus pesticides;
- synthetic pyrethroid pesticides;
- fungicides including chlorothalonil, dichloran, diphenylamine, procymidone and vinclozolin;
- some carbamates; and
- a range of other pesticides.
**Chlorinated organic pesticides**

Chlorinated organic pesticides (organochlorines) were among the first of the modern pesticides to be used in the 1940s. In general, they are highly stable compounds that persist in the environment, especially in soil and can concentrate in the food chain.

Due to their fat solubility, they are stored in the fatty tissue of humans and animals. The use of these organochlorines in developed countries has been heavily restricted since it was shown that some of these compounds were becoming an environmental hazard.

The ATDS pesticide tests examined food for a number of the organochlorine compounds and their metabolites. Metabolites include DDE and DDD, which are the metabolic products of DDT, and heptachlor epoxide, which is the metabolic product of heptachlor. The organochlorine pesticides aldrin, dieldrin, endrin, heptachlor, hexachlorobenzene, chlordane and DDT are no longer registered for use in Australia.

**Organophosphorus pesticides**

Organophosphorus pesticides are widely used insecticides with an array of chemical structures, properties and agricultural uses. Organophosphorus pesticides are mostly biodegradable and therefore do not concentrate in the food chain as is generally the case for organochlorine pesticides.

Organophosphorus pesticides act on the central nervous system of insects and animals, and in high doses are highly toxic. They inhibit the enzyme responsible for the metabolism of acetylcholine, which transmits signals between nerve cells and between nerve and muscle cells, and thus interfere with nervous system function. The hydrolysis of organophosphorus pesticides in biological systems generally yields less toxic substances, which are more readily excreted and tend not to accumulate in the human body. A list of the organophosphorus pesticides examined in the 20th ATDS can be found in Part 1 (Table 5) of the Supplementary Information (FSANZ 2002).

**Carbamate pesticides**

Like organophosphorus pesticides, carbamate pesticides are mostly biodegradable, and therefore do not concentrate in the food chain as is generally the case for organochlorine pesticides. They also act on the central nervous system of insects and animals, are highly acutely toxic, and tend not to accumulate in the human body.

The carbamate pesticides that were examined in the 20th ATDS are: aldicarb, carbaryl, fenoxycarb and pirimicarb.

**Synthetic pyrethroid pesticides**

Synthetic pyrethroid pesticides are man-made insecticides which have a similar chemical structure to natural pyrethrins found in chrysanthemums. Synthetic pyrethroids are fast-acting on the nervous system of insects. They are generally biodegradable and therefore tend not to persist in the environment.
The synthetic pyrethroid pesticides that were examined in the 20th ATDS are: bifenthrin, bioresmethrin, cyfluthrin, cyhalothrin, cypermethrin, deltamethrin, fenvalerate and esfenvalerate, flumethrin and permethrin.

**Fungicides**

Fungicides are used to control plant diseases caused by fungi. Fungicides can either be protectant or eradicant. Protectant fungicides protect plants from fungal infections and retard fungal growth before the fungi causes damage to the plants. Eradicant fungicides are used on plants that have already been invaded and damaged by fungal organisms.

The fungicides examined in the 20th ATDS were all protectant fungicides and included chlorothalonil, dicloran, diphenylamine, iprodione, procymidine and vinclozolin.

**Results and dietary exposures to pesticides**

Unlike metals and other substances, registered pesticides are either intentionally applied to crops to achieve a purpose or are not used, and therefore should not be present in food. For this reason, foods reported as containing ‘less than LOD’ for pesticide residues were assumed to contain no pesticide residues for the purposes of dietary exposure assessments. Even though certain organochlorine pesticides are no longer registered for use in Australia, for simplicity, their dietary exposures have been assessed in a similar manner to that used for other pesticide residues. There were no detections of the following organochlorine pesticide residues: aldrin, dieldrin, endrin, heptachlor, hexachlorobenzene and chlordane. DDT and/or its metabolites were detected in some foods at very low levels. Further details can be found in Table 20 of Part 3 of the Supplementary Information (FSANZ 2002).

The concentrations of pesticide residues reported in the surveyed foods are included in Part 3 of the Supplementary Information (FSANZ 2002), sorted by food (Table 19) and by pesticide (Table 20). Table 21 lists details of the LODs and LORs for the pesticide residues analysed.

Dietary exposures were estimated only when a pesticide was detected in a food. Some pesticides were not detected in any food and consequently their estimated dietary exposures were zero. These pesticides are tabulated in Appendix 3. Additionally, the foods that had no pesticide residue detections are listed in Appendix 4.

The estimated dietary exposures to pesticide residues for different age–gender groups are given in Figures 7 to 12 and in Appendix 2. All estimated dietary exposures to pesticide residues were below 16% of the respective ADIs and therefore all exposures are less than the applicable health standards. To simplify the figures, only dietary exposures greater than or equal to 0.2% of the ADI have been included. The detected pesticide residues for which dietary exposure for all age groups was less than 0.2% of the ADI were: acephate, azinphos methyl, bifenthrin, captan, chlorfenvinphos, chlorothalonil, total DDT$^6$, dimethoate, endosulfan, fenoxycarb, fenthion, maldison, metalaxyl, methidathion, methoprene, o-phenylphenol, permethrin, pirimicarb, pirimiphos-methyl, propiconazole, pyrimethanil and tetradifon.

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$^6$ Total DDT is the sum of p,p’ & o,p’ DDD, p,p’ & o,p’ DDE, and p,p’ & o,p’ DDT
Figure 7: Mean estimated dietary exposure to pesticide residues for adult males (25–34 years) as a percentage of the ADI, based on mean analytical results

(Note: Only pesticide residues present at > 0.2% of the ADI are shown in the figure above)

Figure 8: Mean estimated dietary exposure to pesticide residues for adult females (25–34 years) as a percentage of the ADI, based on mean analytical results

(Note: Only pesticide residues present at > 0.2% of the ADI are shown in the figure above)
Figure 9: Mean estimated dietary exposure to pesticide residues for boys (12 years) as a percentage of the ADI, based on mean analytical results

(Note: Only pesticide residues present at > 0.2% of the ADI are shown in the figure above)

Figure 10: Mean estimated dietary exposure to pesticide residues for girls (12 years) as a percentage of the ADI, based on mean analytical results

(Note: Only pesticide residues present at > 0.2% of the ADI are shown in the figure above)
Figure 11: Mean estimated dietary exposure to pesticide residues for toddlers (2 years) as a percentage of the ADI, based on mean analytical results

(Note: Only pesticide residues present at > 0.2% of the ADI are shown in the figure above)

Figure 12: Mean estimated dietary exposure to pesticide residues for infants (9 months) as a percentage of the ADI, based on mean analytical results

(Note: Only pesticide residues present at > 0.2% of the ADI are shown in the figure above)
Approximately 9% of all pesticides investigated had dietary exposures of greater than or equal to 0.2% of the ADI for all age-gender groups. For pesticides with estimated dietary exposures of less than 0.2% of the ADI for all age-gender categories, 65% had no detections in any food surveyed.

In general, the dietary exposure to pesticide residues was highest for the toddler age group. This is due to the high food consumption relative to body weight.

For adults (male and female), boys (aged 12 years) and girls (12 years), the highest dietary exposure to a pesticide residue, as compared to the ADI, was for methamidophos. Methamidophos is an organophosphorus insecticide. Methamidophos residues were detected only in tomatoes (refer to Tables 19 and 20 in the Supplementary Information for further details (FSANZ 2002)).

**Vinclozolin**

Residues of vinclozolin were detected in kiwifruit in the 20th ATDS. Vinclozolin is not registered for use on kiwifruit in Australia and the Food Standards Code does not include an MRL (the highest concentration of a chemical residue that is legally permitted or accepted in a food) for vinclozolin in kiwifruit. However, vinclozolin is permitted to be used on kiwifruit in New Zealand and it is possible that the kiwifruit containing vinclozolin residues were imported from New Zealand. The estimated dietary exposures to the residues of vinclozolin were below the reference health standard for vinclozolin.

If the kiwifruit containing the residues of vinclozolin were in fact imported from New Zealand, then it is legal for these kiwifruit to be imported and sold in Australia even though the residue concentrations were above the MRLs in the Food Standards Code. This is because, following the commencement of the Trans Tasman Mutual Recognition Arrangement on 1 May 1998:

- food produced in Australia that complies with the MRLs in the Food Standards Code can be legally sold in New Zealand; and
- food produced in New Zealand that complies with the New Zealand (Maximum Residue Limits of Agricultural Compounds) Mandatory Food Standard 1999 can be legally sold in Australia.

The agreement between the Commonwealth of Australia and the Government of New Zealand to establish a system for the development of joint food standards (the Treaty) excluded MRLs for agricultural and veterinary chemicals in food. Australia and New Zealand independently and separately develop MRLs for agricultural and veterinary chemicals in food.

**Comparison between the 19th ATDS and 20th ATDS results for pesticide residues**

The differences between the dietary exposures to pesticide residues in the 20th ATDS and the 19th ATDS are not discussed because the small number of samples and the large range of foods in the food supply mean that it is difficult to draw definite conclusions.
Persistent organochlorine pesticide residues

DDT and/or its metabolites were detected in the 18th AMBS (0.11-0.19% of the ADI), 19th ATDS (0.04-0.50% of the ADI) and 20th ATDS (0.02-0.05% of the ADI).

Aldrin, chlordane and hexachlorobenzene were not detected in any sample in either the 18th, 19th or 20th surveys. Dieldrin, endrin and heptachlor were not detected in any sample in the 19th or 20th surveys. However, residues of both dieldrin and heptachlor were detected in the 18th ATDS at levels of 1.28-3.23% of the ADI and 0.02-0.05% of the ADI respectively. Endrin was not examined in the 18th ATDS.

Benzenehexachloride (BHC) – total residues were detected in both the 18th AMBS and the 19th ATDS but not in the 20th ATDS.

These figures indicate that the dietary exposure of Australians to persistent organochlorine pesticide residues is declining.

Recommendations for pesticide residue analyses

- Pesticide residues should continue to be monitored to determine dietary exposure to pesticide residues. Over a number of surveys, a large amount of data relating to pesticide residues has been collected, with the estimated dietary exposures to pesticide residues being well below that of the respective health standards (ADIs). As a consequence, it is recommended that monitoring of pesticide residues be undertaken at a lower frequency in future surveys;

- Monitoring of pesticide residues in future total diet surveys should focus on those for which there are no recent data and should not be limited to those chemicals registered for use in Australia.

Pesticide residues and metals in the infant diet

The infant-specific foods that were analysed in the 20th ATDS were mixed infant cereal, infant dessert, strained infant dinner and infant formula. These foods account for approximately 53% of energy intake in the constructed infant diet. No pesticide residues were detected in infant dessert, strained infant dinner and infant formula for the pesticides analysed. Residues of pirimiphos-methyl (an organophosphorus grain protectant) at levels close to the LOD were found in two of the nine analytical samples of mixed infant cereal. These results confirm that although infant foods contain pesticide residues, these are at very low levels.

The estimated dietary exposures to pesticides were generally higher in the toddler age category than in the infant age category. The contributing factors to this are: the solid portion of the infant diet is constructed by scaling down the patterns of consumption of a two-year-old child from the NNS; and pesticide residues were not detected in most of the infant specific foods.

In general, infants had a higher dietary exposure to metals (except for selenium and tin) than toddlers. Differences in body weight, levels of consumption of foods, or concentrations of metals may have contributed to the higher infant dietary exposures.
Part C Appendices

The detailed supplementary information for the 20th ATDS can be downloaded from the Food Standards Australia New Zealand website at www.foodstandards.gov.au.
Appendix 1
Estimated dietary exposures to metals

Notes on the tables:

1. For most substances, a range of exposures is presented in these tables. Exposure = mean food consumption x lower or upper bound median concentrations (i.e. analytical results). The lower end of the range (the first result) assumes that results less than the limit of reporting are equal to zero, and the upper end of the range (the second result) assumes that results less than the limit of reporting are the same as the limit of reporting. The limits of reporting are provided in Table 1 of the Report.

2. Since almost all of results for copper and zinc were above the LOR, it was not necessary to calculate an upper and lower mean concentration. Therefore only one dietary exposure estimate is provided.

3. 1 µg = one millionth of 1 gram.

4. Estimated dietary exposures are based on food consumption data from the 1995 National Nutrition Survey. For infants, estimated dietary exposures are based on a constructed infant diet.

5. Tolerable limits for metal contaminants are listed in Part 1 (Table 7) of the Supplementary Information (FSANZ 2002).

6. All results have been rounded to two significant figures.

Table A1: Range of mean estimated daily dietary exposures to metals in µg/kg bw/day based on median analytical results

<table>
<thead>
<tr>
<th>Metal</th>
<th>Adult males (25-34 years) µg/kg bw/day</th>
<th>Adult females (25-34 years) µg/kg bw/day</th>
<th>Boys (12 years) µg/kg bw/day</th>
<th>Girls (12 years) µg/kg bw/day</th>
<th>Toddler (2 years) µg/kg bw/day</th>
<th>Infant (9 months) µg/kg bw/day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antimony</td>
<td>0.01-0.08</td>
<td>&lt;0.01-0.07</td>
<td>&lt;0.01-0.09</td>
<td>&lt;0.01-0.07</td>
<td>0.01-0.19</td>
<td>0.01-0.25</td>
</tr>
<tr>
<td>Arsenic, total</td>
<td>0.56-0.88</td>
<td>0.49-0.78</td>
<td>0.50-0.83</td>
<td>0.28-0.54</td>
<td>0.55-1.3</td>
<td>0.37-1.4</td>
</tr>
<tr>
<td>Cadmium</td>
<td>0.08-0.24</td>
<td>0.07-0.22</td>
<td>0.11-0.29</td>
<td>0.09-0.22</td>
<td>0.18-0.57</td>
<td>0.13-0.68</td>
</tr>
<tr>
<td>Copper</td>
<td>16</td>
<td>14</td>
<td>21</td>
<td>16</td>
<td>40</td>
<td>65</td>
</tr>
<tr>
<td>Lead</td>
<td>0.06-0.40</td>
<td>0.02-0.35</td>
<td>0.02-0.43</td>
<td>0.01-0.34</td>
<td>0.03-0.93</td>
<td>0.01-1.2</td>
</tr>
<tr>
<td>Mercury</td>
<td>0.01-0.09</td>
<td>0.01-0.08</td>
<td>0.01-0.10</td>
<td>0.01-0.08</td>
<td>0.01-0.20</td>
<td>0.01-0.25</td>
</tr>
<tr>
<td>Tin</td>
<td>1.3-1.6</td>
<td>1.5-1.8</td>
<td>1.7-2.0</td>
<td>0.64-0.93</td>
<td>12-13</td>
<td>8.7-9.0</td>
</tr>
<tr>
<td>Zinc</td>
<td>170</td>
<td>130</td>
<td>250</td>
<td>170</td>
<td>370</td>
<td>630</td>
</tr>
</tbody>
</table>
Table A2: Range of mean estimated daily dietary exposures to metals as a percentage of the tolerable limit based on median analytical results

<table>
<thead>
<tr>
<th>Metal</th>
<th>Adult males (25-34 years)</th>
<th>Adult females (25-34 years)</th>
<th>Boys (12 years)</th>
<th>Girls (12 years)</th>
<th>Toddler (2 years)</th>
<th>Infant (9 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%TDI</td>
<td>%TDI</td>
<td>%TDI</td>
<td>%TDI</td>
<td>%TDI</td>
<td>%TDI</td>
</tr>
<tr>
<td>Antimony</td>
<td>3.1-21</td>
<td>1.8-18</td>
<td>2.3-23</td>
<td>1.9-18</td>
<td>3.6-48</td>
<td>2.7-61</td>
</tr>
<tr>
<td>Arsenic</td>
<td>18.6-29.2</td>
<td>16.2-25.9</td>
<td>16.6-27.5</td>
<td>9.4-18.0</td>
<td>18-44</td>
<td>12-48</td>
</tr>
<tr>
<td>Cadmium</td>
<td>7.6-24</td>
<td>6.9-22</td>
<td>11-29</td>
<td>8.9-22</td>
<td>18-57</td>
<td>13-68</td>
</tr>
<tr>
<td>Copper</td>
<td>8.0</td>
<td>7.2</td>
<td>11</td>
<td>8.2</td>
<td>20</td>
<td>32</td>
</tr>
<tr>
<td>Lead</td>
<td>1.8-11</td>
<td>0.67-9.9</td>
<td>0.45-12</td>
<td>0.27-9.5</td>
<td>0.72-26.0</td>
<td>0.35-33.3</td>
</tr>
<tr>
<td>Mercury</td>
<td>1.9-13</td>
<td>2.1-12</td>
<td>1.9-14</td>
<td>1.4-11</td>
<td>1.9-28</td>
<td>1.4-35</td>
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<tr>
<td>Selenium</td>
<td>9.3-11</td>
<td>7.7-9.5</td>
<td>12-13</td>
<td>9.1-10</td>
<td>21-24</td>
<td>17-19</td>
</tr>
<tr>
<td>Tin</td>
<td>0.06-0.08</td>
<td>0.08-0.09</td>
<td>0.08-0.10</td>
<td>0.03-0.05</td>
<td>0.59-0.63</td>
<td>0.43-0.45</td>
</tr>
<tr>
<td>Zinc</td>
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<td>13</td>
<td>25</td>
<td>17</td>
<td>37</td>
<td>63</td>
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</tbody>
</table>
Appendix 2
Estimated Dietary exposure to pesticides

Notes on the tables:

1. Exposure = mean food consumption x mean concentrations of pesticide residues. Mean concentrations are calculated assuming residues less than LOD are equal to ‘0’.

2. 1 µg = one millionth of 1 gram.

3. Estimated dietary exposures are based on food consumption data from the 1995 National Nutrition Survey. For infants, estimated dietary exposures are based on a constructed infant diet.

4. Pesticides screened for but not detected are included in Appendix 3.
Table A3: Mean estimated daily dietary exposure to detected pesticide residues in ng/kg bw/day based on mean analytical results

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Adult males (25-34 years)</th>
<th>Adult females (25-34 years)</th>
<th>Boys (12 years)</th>
<th>Girls (12 years)</th>
<th>Toddler (2 years)</th>
<th>Infant (9 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
<td>µg/kg bw/day</td>
</tr>
<tr>
<td>Acephate</td>
<td>0.0028</td>
<td>0.0027</td>
<td>0.0034</td>
<td>0.0030</td>
<td>0.0034</td>
<td>0.0025</td>
</tr>
<tr>
<td>Azinphos-methyl</td>
<td>0.0004</td>
<td>0.0006</td>
<td>0.0004</td>
<td>0.0005</td>
<td>0.0020</td>
<td>0.0015</td>
</tr>
<tr>
<td>Bifenthrin</td>
<td>0.0030</td>
<td>0.0032</td>
<td>0.0033</td>
<td>0.0031</td>
<td>0.0026</td>
<td>0.0038</td>
</tr>
<tr>
<td>Bioresmethrin</td>
<td>0.0017</td>
<td>0.0014</td>
<td>0.0033</td>
<td>0.0026</td>
<td>0.0026</td>
<td>0.0027</td>
</tr>
<tr>
<td>Captan</td>
<td>0.0074</td>
<td>0.0110</td>
<td>0.0026</td>
<td>0.0115</td>
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<td>0.0403</td>
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<td>Carbaryl</td>
<td>0.0341</td>
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<td>0.0539</td>
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<tr>
<td>Chlorfenvinphos</td>
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<td>0.0003</td>
<td>0.0003</td>
<td>0.0003</td>
<td>0.0003</td>
<td>0.0003</td>
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<tr>
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<td>0.0034</td>
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<tr>
<td>Chlorpyrifos</td>
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<td>0.0060</td>
<td>0.0111</td>
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<tr>
<td>Chlorpyrifos-methyl</td>
<td>0.0723</td>
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<td>0.1816</td>
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<tr>
<td>DDT (total)²</td>
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<td>0.0005</td>
<td>0.0005</td>
<td>0.0004</td>
<td>0.0010</td>
<td>0.0007</td>
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<tr>
<td>Dimethoate</td>
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<td>0.0010</td>
<td>0.0006</td>
<td>0.0008</td>
<td>0.0033</td>
<td>0.0024</td>
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<td>Diphenylamine</td>
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<tr>
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<td>0.0022</td>
<td>0.0025</td>
<td>0.0023</td>
<td>0.0033</td>
<td>0.0025</td>
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<td>Maldison</td>
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<td>0.0010</td>
<td>0.0011</td>
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<td>0.0035</td>
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<tr>
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<td>0.0005</td>
<td>0.0007</td>
<td>0.0019</td>
<td>0.0013</td>
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<tr>
<td>Methamidophos</td>
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<td>0.0737</td>
<td>0.0930</td>
<td>0.0826</td>
<td>0.0941</td>
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<tr>
<td>Methidathion</td>
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<td>0.0005</td>
<td>0.0003</td>
<td>0.0003</td>
<td>0.0004</td>
<td>0.0003</td>
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<tr>
<td>Methoprene</td>
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<td>0.0035</td>
<td>0.0197</td>
<td>0.0042</td>
<td>0.0196</td>
<td>0.0142</td>
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<tr>
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<td>0.0018</td>
<td>0.0031</td>
<td>0.0030</td>
<td>0.0091</td>
<td>0.0069</td>
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<td>Parathion-methyl</td>
<td>0.0026</td>
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<td>0.0065</td>
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<td>0.0149</td>
<td>0.0109</td>
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<td>Permethrin</td>
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<td>0.0150</td>
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<td>Piperonyl butoxide</td>
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<td>0.1489</td>
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<tr>
<td>Pirimicarb</td>
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<td>0.0003</td>
<td>0.0009</td>
<td>0.0006</td>
<td>0.0019</td>
<td>0.0014</td>
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<tr>
<td>Pirimiphos-methyl</td>
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<tr>
<td>Procymidone</td>
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<td>Propargite</td>
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<td>0.0010</td>
<td>0.0013</td>
<td>0.0012</td>
<td>0.0013</td>
<td>0.0009</td>
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</tr>
</tbody>
</table>

² Total DDT is the sum of p,p' & o,p' DDD, p,p' & o,p' DDE, and p,p' & o,p' DDT
Table A4: Mean estimated daily dietary exposure to pesticide residues as a percentage of the ADI based on mean analytical results

<table>
<thead>
<tr>
<th>Chemical</th>
<th>Adult males (25-34 years) %TDI</th>
<th>Adult females (25-34 years) %TDI</th>
<th>Boys (12 years) %TDI</th>
<th>Girls (12 years) %TDI</th>
<th>Toddler (2 years) %TDI</th>
<th>Infant (9 months) %TDI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acephate</td>
<td>0.09</td>
<td>0.09</td>
<td>0.11</td>
<td>0.10</td>
<td>0.11</td>
<td>0.08</td>
</tr>
<tr>
<td>Azinphos-methyl</td>
<td>0.04</td>
<td>0.06</td>
<td>0.04</td>
<td>0.05</td>
<td>0.20</td>
<td>0.15</td>
</tr>
<tr>
<td>Bifenthrin</td>
<td>0.03</td>
<td>0.03</td>
<td>0.03</td>
<td>0.03</td>
<td>0.06</td>
<td>0.03</td>
</tr>
<tr>
<td>Bioresmethrin</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Captan</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>0.06</td>
<td>0.04</td>
</tr>
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<td>Carbaryl</td>
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<td>1.24</td>
<td>1.35</td>
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<tr>
<td>Chlorfenvinphos</td>
<td>0.05</td>
<td>0.05</td>
<td>0.06</td>
<td>0.06</td>
<td>0.07</td>
<td>0.05</td>
</tr>
<tr>
<td>Chlorothalonil</td>
<td>0.03</td>
<td>0.05</td>
<td>0.04</td>
<td>0.03</td>
<td>0.07</td>
<td>0.05</td>
</tr>
<tr>
<td>Chlorpyrinos</td>
<td>0.17</td>
<td>0.20</td>
<td>0.37</td>
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</tr>
<tr>
<td>Chlorpyrifos-methyl</td>
<td>0.72</td>
<td>0.63</td>
<td>1.29</td>
<td>0.89</td>
<td>1.82</td>
<td>1.29</td>
</tr>
<tr>
<td>DDT (total)</td>
<td>0.03</td>
<td>0.02</td>
<td>0.02</td>
<td>0.02</td>
<td>0.05</td>
<td>0.04</td>
</tr>
<tr>
<td>Dimethoate</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.02</td>
<td>0.01</td>
</tr>
<tr>
<td>Diphenylamine</td>
<td>0.15</td>
<td>0.17</td>
<td>0.52</td>
<td>0.35</td>
<td>1.01</td>
<td>0.72</td>
</tr>
<tr>
<td>Endosulfan</td>
<td>0.04</td>
<td>0.04</td>
<td>0.07</td>
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<td>0.05</td>
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</tr>
<tr>
<td>Fenitrothion</td>
<td>0.54</td>
<td>0.48</td>
<td>0.87</td>
<td>0.63</td>
<td>1.30</td>
<td>0.91</td>
</tr>
<tr>
<td>Fenoxycarb</td>
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<td>&lt;0.01</td>
<td>0.02</td>
<td>0.01</td>
<td>0.03</td>
<td>0.02</td>
</tr>
<tr>
<td>Fenthion</td>
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<td>0.11</td>
<td>0.13</td>
<td>0.12</td>
<td>0.17</td>
<td>0.12</td>
</tr>
<tr>
<td>Iprodione</td>
<td>1.19</td>
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<td>0.39</td>
<td>0.34</td>
<td>1.12</td>
<td>0.82</td>
</tr>
<tr>
<td>Malathion</td>
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<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Metalaxyl</td>
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<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Methamidophos</td>
<td>13.11</td>
<td>12.28</td>
<td>15.49</td>
<td>13.76</td>
<td>15.68</td>
<td>5.72</td>
</tr>
<tr>
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<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
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</tr>
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<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>o-phenylphenol</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.02</td>
<td>0.01</td>
<td>0.05</td>
<td>0.03</td>
</tr>
<tr>
<td>Parathion-methyl</td>
<td>1.30</td>
<td>1.68</td>
<td>3.25</td>
<td>2.37</td>
<td>7.44</td>
<td>5.44</td>
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<td>Permethrin</td>
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<td>0.04</td>
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<td>0.03</td>
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<td>Piperonyl butoxide</td>
<td>0.07</td>
<td>0.07</td>
<td>0.13</td>
<td>0.09</td>
<td>0.22</td>
<td>0.15</td>
</tr>
<tr>
<td>Pirimicarb</td>
<td>0.01</td>
<td>0.02</td>
<td>0.05</td>
<td>0.03</td>
<td>0.09</td>
<td>0.07</td>
</tr>
<tr>
<td>Pirimiphos-methyl</td>
<td>0.06</td>
<td>0.05</td>
<td>0.11</td>
<td>0.06</td>
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</tr>
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<td>Procymidone</td>
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<td>0.19</td>
<td>0.22</td>
<td>0.14</td>
<td>0.40</td>
<td>0.30</td>
</tr>
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<td>Propargite</td>
<td>2.47</td>
<td>3.10</td>
<td>6.61</td>
<td>4.75</td>
<td>14.90</td>
<td>10.78</td>
</tr>
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<td>Propiconazole</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Pyrimethanil</td>
<td>&lt;0.01</td>
<td>0.01</td>
<td>0.01</td>
<td>0.02</td>
<td>0.06</td>
<td>0.04</td>
</tr>
<tr>
<td>Tebufenpyrad</td>
<td>0.18</td>
<td>0.23</td>
<td>0.44</td>
<td>0.33</td>
<td>1.06</td>
<td>0.77</td>
</tr>
<tr>
<td>Tetrachlorvinphos</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Vinlozolin</td>
<td>2.75</td>
<td>2.73</td>
<td>2.31</td>
<td>2.17</td>
<td>9.73</td>
<td>7.06</td>
</tr>
</tbody>
</table>

8 Total DDT is the sum of p,p’ & o,p’ DDD, p,p’ & o,p’ DDE, and p,p’ & o,p’ DDT
Appendix 3  Pesticide residues and other substances not detected in the 20th ATDS

The dietary exposures for these substances were estimated to be zero, as the concentration of these substances in surveyed foods is less than the limit of detection.

<table>
<thead>
<tr>
<th><strong>Carbamates</strong></th>
<th><strong>Organophosphorus Pesticides</strong></th>
<th><strong>Synthetic pyrethroids</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Aldicarb</td>
<td>Azinphos ethyl</td>
<td>Cyfluthrin</td>
</tr>
<tr>
<td><strong>Fungicides</strong></td>
<td>Bromophos-ethyl</td>
<td>Cyhalothrin</td>
</tr>
<tr>
<td>Bupirimate</td>
<td>Carbophenothonthion</td>
<td>Cypermethrin</td>
</tr>
<tr>
<td>Dicloran</td>
<td>Coumaphos</td>
<td>Deltamethrin</td>
</tr>
<tr>
<td>Difenconazole</td>
<td>Demeton-S-methyl</td>
<td>Fenvalerate (&amp; Esfenvalerate)</td>
</tr>
<tr>
<td>Dimethomorph</td>
<td>Diazinon</td>
<td>Flumethrin</td>
</tr>
<tr>
<td>Flusilazole</td>
<td>Dichlorvos</td>
<td></td>
</tr>
<tr>
<td>Hexaconazole</td>
<td>Dioxathion</td>
<td></td>
</tr>
<tr>
<td>Imazalil</td>
<td>Ethion</td>
<td></td>
</tr>
<tr>
<td>Myclobutanil</td>
<td>Fenamiphos</td>
<td></td>
</tr>
<tr>
<td>Tebuconazole</td>
<td>Fenchlorphos</td>
<td></td>
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<tr>
<td>Triadimefon</td>
<td>Formothion</td>
<td></td>
</tr>
<tr>
<td>Triadimethol</td>
<td>Methaclofos</td>
<td></td>
</tr>
<tr>
<td><strong>Chlorinated Organic Pesticides</strong></td>
<td>Monocrotophos</td>
<td></td>
</tr>
<tr>
<td>Aldrin</td>
<td>Omethoate</td>
<td></td>
</tr>
<tr>
<td>BHC (total: α,β,γ[Lindane],δ)</td>
<td>Parathion</td>
<td></td>
</tr>
<tr>
<td>Chlordane</td>
<td>Phorate</td>
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</tr>
<tr>
<td>Dicofol</td>
<td>Phosalone</td>
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<tr>
<td>Dieldrin</td>
<td>Phosmet</td>
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<tr>
<td>Endrin</td>
<td>Thiometon</td>
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<tr>
<td>Heptachlor</td>
<td>Trichlorfon</td>
<td></td>
</tr>
<tr>
<td>Hexachlorobenzene</td>
<td>Vanidothion</td>
<td></td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td><strong>Aflatoxins</strong></td>
<td><strong>Ochratoxins</strong></td>
</tr>
<tr>
<td>Coumatetralyl</td>
<td>Aflatoxin B1</td>
<td>Ochratoxin A</td>
</tr>
<tr>
<td>Oxyfluorfen</td>
<td>Aflatoxin B2</td>
<td></td>
</tr>
<tr>
<td>Pendimethalin</td>
<td>Aflatoxin G1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Aflatoxin G2</td>
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</tr>
<tr>
<td><strong>Inhibitory Substances</strong></td>
<td>Oxytetracycline</td>
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</tr>
<tr>
<td>Penicillin G</td>
<td>Penicillin G</td>
<td></td>
</tr>
<tr>
<td>Streptomycin</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix 4
Foods that had no detections of pesticide residues

Almonds
Bacon
Baked beans
Bananas
Beef, minced
Broccoli
Cheese, cheddar
Chicken breasts
Coffee, instant
Dim sim
Fish portions
Hamburger
Infant dessert
Infant dinner, strained
Infant formula
Lamb chops
Liver pate, chicken
Margarine, table spread
Milk chocolate
Milk, whole
Onions
Orange juice
Pasta, mixed
Peanut butter
Peas, frozen
Potato
Rice, white
Soft drink
Sugar, white
Tomato sauce
Vanilla ice cream
Watermelon
Appendix 5
Definitions and glossary of terms

In the process of estimating safe intakes and regulatory limits for pesticide residues and contaminants, the following terms are most commonly used.

**Acceptable Daily Intake (ADI)**

The Acceptable Daily Intake (ADI) for humans is defined as an estimate of the amount of a chemical that can be ingested daily over a lifetime without appreciable risk to health (WHO 2001c).

ADIs are set using information obtained from toxicological studies, including data from studies on various laboratory animals. From these studies, a No Observable Effect Level (NOEL) is established. The NOEL is the highest dose level that produces no observable toxic effect in the most sensitive test species and is expressed in milligrams per kilogram of body weight per day (mg/kg bw/day).

The ADI is derived by applying a safety factor to the NOEL. The safety factor takes into consideration the nature of the effect, differences between laboratory test animals and humans, and genetic variation in the human population. If any information on exposure in humans is available, usually from short to mid-term, this will be used to set the ADI. The unit for the ADI is milligrams per kilogram of body weight per day.

The ADIs for pesticides, as recommended by the Therapeutic Goods Administration (TGA), were used in this survey. These ADIs can also be found on the TGA website at www.health.gov.au/tga/chem/chem.htm. The ADIs used in this report are listed in Table 6 in Part 1 of the supplementary section. The body masses used for each age–gender category to compare the ADIs with the estimated daily intakes are listed in Table 4 in Part 1 of the supplementary section.

**Limit of Detection (LOD)**

The LOD is the lowest concentration of a chemical that can be qualitatively detected using a specified laboratory method and/or item of laboratory equipment (i.e. its presence can be detected but not quantified).

**Limit of Reporting (LOR)**

The LOR is the lowest concentration of a chemical that can be detected and quantified, with an acceptable degree of certainty, using a specified laboratory method and/or item of laboratory equipment.
Maximum Permitted Concentration (MPC) or Maximum Level (ML)

The Maximum Level (ML) (previously referred to as Maximum Permitted Concentration (MPC)), expressed as milligram per kilogram, is the limit placed on the level of a contaminant, such as a heavy metal, in food. An ML is set at the lowest level that is achievable with good practices, while taking into account likely intakes of the contaminant in comparison to the PTDI or PTWI.

MPCs are listed in Volume 1 of the Food Standards Code and MLs are listed in Volume 2 of the Food Standards Code.

Maximum Residue Limit (MRL)

The Maximum Residue Limit (MRL) is the highest concentration of a chemical residue that is legally permitted or accepted in a food or animal feed. The MRL does not indicate the amount of chemical that is always present in a treated food but it does indicate the highest residue that could result from the registered conditions of use. The concentration is expressed in milligrams per kilogram (mg/kg) of the food or animal feed. If the MRL is exceeded then this indicates a likely misuse of the chemical. MRLs are not direct public health and safety limits but are indicators of whether an agricultural or veterinary chemical product has been used according to its registered use.

Although MRLs are not direct health measures, the toxicology of the chemical is taken into consideration when setting the MRL. An MRL is not recommended and the use of a pesticide not approved where the resulting residues from all foods may lead to exposures exceeding the ADI.

Generally, specific MRLs are not set for processed foods. For foods such as vegetables, fruits, meat and fish that undergo little processing before consumption, the MRL for the commodity can suffice for the food as consumed. In the case of a mixed food, the applicable MRL is calculated from the combined proportionate quantities of the ingredients of the mixed food and the MRLs that apply to these ingredients.

The MRLs allowed for residues in foods sold in Australia are listed in the Food Standards Code.

Provisional Tolerable Daily Intakes (PTDI) and Provisional Tolerable Weekly Intakes (PTWI)

Provisional Tolerable Daily Intakes (PTDI) are upper limits that are set for substances that do not accumulate in animals and humans (WHO 2001c), PTDIs have been set for arsenic, copper, selenium, and zinc. Antimony has an oral reference dose (USEPA 1991).

Provisional Tolerable Weekly Intakes (PTWI) are set for substances, such as heavy metals, that are contaminants in food and are known to accumulate in animals and humans. PTWIs have been set for cadmium, lead, mercury and tin.

The unit of time for PTWIs is different to that used for ADIs and PTDIs. PTWIs use a one week time unit while ADIs and PTDIs use a one day time unit. Another difference is the term ‘provisional’, which is used in PTWIs and PTDIs in order to emphasize the paucity of safety data on contaminants. The levels of PTDIs and PTWIs are continually being re-evaluated.
The method for calculating a PTDI/PTWI for a contaminant is similar to that used for calculating an ADI for a pesticide. A NOEL is set and the PTDI/PTWI is derived from the NOEL using a safety factor. In the case of metals, there is often human epidemiological information, generally occupational exposure, on which NOELs can be based. Heavy metals have been recognised as poisons for centuries and many accidental poisonings have been recorded. Because heavy metal NOELs are generally based on known effects on humans, a lower safety factor than commonly used for food additives is therefore normally applied.

Tolerable levels are recommended by the Joint FAO/WHO Expert Committee on Food Additives. The PTDIs and PTWIs used in this report are listed in Table 7 in Part 1 of the supplementary section.

**Recommended Dietary Intakes (RDI)**

Recommended dietary intakes are “the levels of intake of essential nutrients considered...to be adequate to meet the known nutritional needs of practically all healthy people...” (NHMRC 2001b). RDIs are established based on the available scientific knowledge and take into account the variability in absorption and metabolism of the nutrients between individuals.

**Tolerable Daily Intake**

Refer to definition of PTDI/PTWI.
References

ACT Community Care (Child, Youth and Women’s Health Program), 2000, From Milk to More… Introducing foods to your baby, Publishing Services, Canberra.


National Health and Medical Research Council, 2001a, Dietary Guidelines for Children and Adolescents In Australia Incorporating Infant Feeding Guidelines For Health Workers (Draft).

National Health and Medical Research Council, 2001b, National Health and Medical Research Council Website (www.health.gov.au/nhmrc/publications/diet)


