Australia New Zealand Food Authority

The 19th 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 to pesticide residues and contaminants. The survey is conducted approximately every two years, and this is the 19th such survey.

The survey estimates the level of dietary exposure for Australian consumers to a range of pesticide residues and contaminants through the testing of food samples representative of the total diet. These samples were prepared 'table-ready', for example, the potatoes were cooked.

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

A number of changes have been made by ANZFA in the conduct of the 19th survey. The most obvious change is to the format and presentation of the survey, where a shorter report has been produced with more detailed information provided on the ANZFA web site. There have also been a number of changes to the methods for estimating dietary exposure, and in the use of the latest food consumption data derived from the 1995 National Nutrition Survey.

The results demonstrate that the levels of pesticide residues and contaminants in our food are very low, and in all cases they are within acceptable safety limits where reliable dietary exposure estimates could be calculated. However, the survey has indicated the need to further investigate the potential for obtaining analyses with a lower limit of reporting for mercury and antimony in food, and to develop more refined dietary exposure models for dithiocarbamate fungicides. These issues will be addressed in future surveys.

These results will be provided to the World Health Organization as a contribution to the Global Environmental Monitoring System which collects data on the levels of pesticide residues and contaminants in the food supply worldwide.

I would like to thank the health authorities and the educational and scientific institutions in the States and the Northern Territory without whose valuable assistance this survey would not have been possible. I would also like to pay tribute to the peer reviewers and the ANZFA staff for their important contribution to the preparation of this report.

I am pleased to present the Australian Total Diet Survey as part of ANZFA's commitment to protecting public health and safety.

Michael MacKellar
Chairman

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Acknowledgments

The assistance and advice from the State and Territory health authorities and their staff members have been central to the operation of the 19th Australian Total Diet Survey.

The Australia New Zealand Food Authority (ANZFA) would like to thank the officers from the States and Territories who arranged the collection of the samples during 1998.

The Australian Government Analytical Laboratories carried out the analyses and provided advice and comments. Their assistance was appreciated.

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

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- School of Hospitality and Tourism, Palmerston Campus, Northern Territory University, Northern Territory;
- Royal Hobart Hospital, Tasmania;
- Panorama Campus, Douglas Mawson Institute of Technical and Further Education, and Department of Employment, Training and Further Education, South Australia;
- Southbank Institute of Technical and Further Education, 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 ANZFA would like to thank the following peer reviewers for their valuable assistance:

- Dr Denis Hamilton, Queensland Department of Primary Industries and member of the World Health Organization/Food and Agriculture Organization Joint Meeting on Pesticide Residues; and
- Dr Richard Vannoort, Environmental Science and Research, New Zealand.

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Abbreviations

ADI	Acceptable Daily Intake
AGAL	Australian Government Analytical Laboratories
AMBS	Australian Market Basket Survey
ANZFA	Australia New Zealand Food Authority
ATDS	Australian Total Diet Survey
DBT	Dibutyl tin
DIAMOND	Dietary Modelling on Nutritional Data
FAO	Food and Agriculture Organization
LOEL	Lowest observable effects level
LOR	Limit of reporting
µg/kg bw	micrograms[1] per kilogram of body weight
mg/kg	milligrams[2] per kilogram
MPC	Maximum permitted concentration
MRL	Maximum residue limit
NDS	National Dietary Survey
ng/kg bw	nanograms[3] per kilogram of body weight
NHMRC	National Health and Medical Research Council
NNS	National Nutrition Survey
NRA	National Registration Authority for Agricultural and Veterinary Chemicals
PCBs	Polychlorinated biphenyls
RDI	Recommended Dietary Intake
TBT	Tributyl tin
USEPA	United States Environmental Protection Agency
WHO	World Health Organization

Summary

The role of the Australia New Zealand Food Authority (ANZFA), 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 and contaminants is part of this role.

ANZFA is a statutory authority that develops food standards and other food regulatory measures for Australia and New Zealand. ANZFA does this in partnership with the Australian Commonwealth, State and Territory Governments and the New Zealand Government.

ANZFA monitors the food supply to ensure that existing food regulatory measures give adequate consumer protection. The Australian Total Diet Survey (ATDS) is part of that monitoring. It was previously named the Australian Market Basket Survey. A total diet survey is also conducted in New Zealand and the New Zealand Ministry of Health administers that survey.

The survey

The purpose of the ATDS is to estimate the level of dietary exposure of Australian consumers to a range of pesticide residues and contaminants that can be found in the food supply. Dietary exposure is the intake of pesticide residues and contaminants from food.

ANZFA coordinated the survey and relied on the States and Territories involved to arrange purchase and preparation of food samples. The Australian Government Analytical Laboratories carried out all analyses.

Sixty-nine types of foods were tested for pesticide residues and contaminant content from foods sampled throughout the 1998 calendar year. These food types were sampled in different States and Territories 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 and fungicides as well as the contaminants antimony, arsenic, cadmium, copper, lead, mercury, selenium, tin and zinc. Fruits and vegetables were analysed for dithiocarbamate fungicides. Walnuts, tahina and roasted salted peanuts were tested for aflatoxins and milk samples were examined for the presence of Aflatoxin M1.

Dietary exposures to pesticide residues and contaminants were estimated for six age–gender groups. Each food in the survey was chemically analysed to measure the level of pesticide residues and contaminants. A ‘model’ diet was constructed for each age–gender group based on these foods and food consumption data from the 1995 National Nutrition Survey. The contributions of each pesticide residue and contaminant in every food in a diet were added to give the total dietary exposure.

The estimated dietary exposure from the Australian diet to each chemical was compared to Australian health standards (Commonwealth Department of Health and Aged Care 2000). In the absence of Australian health standards, international (World Health Organization) health standards were used. These health standards were derived from toxicological studies.

Results

The key results from the survey are:

- The estimated dietary exposures to arsenic, cadmium, lead, copper, selenium, zinc and tin were within acceptable health standards. Analytical techniques with a lower limit of reporting are required for mercury and antimony analysis to more accurately define the dietary exposure to these chemicals.
- Aflatoxins were not found in milk, tahina or walnuts, although they were found in one sample of roasted salted peanuts. This sample was found to contain 0.038 mg/kg of Aflatoxin B1 and 0.006 mg/kg of Aflatoxin B2. This amount exceeds the Australian *Food Standards Code* maximum permitted concentration for aflatoxins in these foods of 0.015 mg/kg and has been brought to the attention of the relevant enforcement agency. Any detection of aflatoxin is significant because these substances are of high toxicity. The ATDS will continue to monitor aflatoxins in future surveys.
- Polychlorinated biphenyls were not found in any food.
- The estimated dietary exposures to pesticide residues were all within acceptable health standards.

Survey changes

A number of changes have been made to the survey. One of these changes is the preparation of a shorter printed survey report with more detailed data and results available on the ANZFA website. This approach enables ANZFA to better manage requests for information about the survey. ANZFA's website is www.anzfa.gov.au.

Another major change is that this survey has used the 1995 National Nutrition Survey data as a basis for the dietary exposures estimates. Previous surveys have used consumption data from the 1983 and 1985 National Dietary Surveys. In order to bridge the gap between one database to another:

- the report includes a short assessment on the differences between the 1995 food consumption data and the 1983 and 1985 food consumption data; and
- dietary exposures using the analytical results from the 1996 Australian Market Basket Survey have been recalculated with the 1995 National Nutrition Survey consumption data for comparison with the 19th ATDS results.

Conclusion

In conclusion, the 19th ATDS confirms the overall safety of the Australian food supply regarding the presence of pesticide residues and contaminants.

Part A Background

The purpose of the Australian Total Diet Survey (ATDS) is to estimate the level of dietary exposure of Australian consumers to a range of pesticide residues and contaminants that can be found in the food supply. 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 safety of food which contains residues of pesticides before the pesticide is approved for use in Australia. These agencies must be satisfied that the use of the pesticide will result in no appreciable risk of adverse health effects.

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 19th ATDS is the fourth 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 in one year, and the report writing and planning for the next survey take place in the following year. Publication of the report follows peer review of the survey.

Pesticide and contaminant 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 and contaminants in foods:

- the National Residue Survey; and
- the Imported Food Program, conducted by the Australian Quarantine and Inspection Service, which undertakes the surveillance of imported foods to ensure that they comply with the *Food Standards Code*.

The main aim of these programs is to monitor pesticide residues and contaminants in food commodities in export and import trade respectively. In contrast, the ATDS aims to estimate the level of dietary exposure to pesticide residues and contaminants in the overall Australian diet.

In addition to these programs, State and Territory health and agriculture authorities carry out surveys of specific contaminants or pesticide residues. 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 and pesticide status of foods.

Comparison with other surveys

The ATDS differs from other surveys of pesticide residues and contaminant 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 and contaminants 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.
- 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 or contaminants in the food, an analysis of prepared foods more accurately reflects the levels of residues or contaminants that are likely to be consumed.

Using information from the survey

Data from the ATDS provides background information for developing food regulatory measures. The ATDS data on the dietary exposure to agricultural and veterinary chemicals is used as a check on exposure assessments undertaken during the registration process at the National Registration Authority for Agricultural and Veterinary Chemicals (NRA).

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 (WHO/FAO) Global Environmental Monitoring System, 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

As usual, this survey was coordinated by ANZFA 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 February, April, August and October 1998. 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 to AGAL. Analytical chemists in the laboratories undertook the chemical analyses of the foods in accordance with the quality assurance procedures in Part 5 of the supplementary information available on ANZFA's website. 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 may indicate a breach of the Australian *Food Standards Code*.

Foods included in the survey

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, such as bread, beef, eggs, milk, lettuce, orange juice and potatoes. Lamb's liver is also sampled as a core food although it is not consumed to a great extent in Australia. Liver, as a major organ of detoxification, may contain high levels of contaminants or pesticide residues and therefore it is appropriate to examine it at the same level as core foods.

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 would ordinarily result in 28 composite samples of each core food. However, floods in the Northern Territory during one season meant that core foods were sampled only three times from Darwin. This meant that there were only 27 core food samples for the 19th ATDS.

Regional foods were defined as those foods that might be expected to show regional variation of residue and contaminant 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 cornflakes, canned salmon 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.

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

- The samples in each survey must consist of representative foods from each major food group 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 Dietary Surveys (NDS), should be analysed. If the food was examined in a recent survey and caused no concern, another food from the group may be chosen.
- Foods that may be of particular interest from a pesticide or contaminant viewpoint may be included in the survey, although their intake may be low.
- Foods may be included if they form a significant part of the diet of a subpopulation of Australians. For example, wheat is by far the most popular cereal and is the basis for many foods. However, some individuals cannot consume wheat without ill effect and must substitute other cereals. For this reason, rice, tahina and red kidney beans were also examined.

The foods surveyed in the 19th ATDS are shown in Table 1 of the supplementary information on ANZFA's

website. All the foods examined in the survey were prepared to a 'table ready' state before analysis. For example, meats and eggs were cooked, while fruits that are normally consumed without peel were peeled. In preparing food as 'table ready', local tap water is used rather than distilled water to ensure that pesticide residues and contaminants that may be present in tap water are taken into account in the overall estimate of dietary exposure.

Pesticide residues and contaminants examined

All foods were tested for a range of pesticide residues including residues of chlorinated organic pesticides, organophosphorus pesticides, carbamates, synthetic pyrethroids and fungicides (see Table 6 in the supplementary information for a complete list). Fruits and vegetables were also analysed for dithiocarbamate fungicides. All foods were tested for the contaminants antimony, arsenic, cadmium, copper, lead, mercury, selenium, tin and zinc. Walnuts, tahina and roasted salted peanuts were tested for aflatoxins and milk samples were examined for the presence of Aflatoxin M1. All foods were also tested for polychlorinated biphenyls (PCBs).

Dietary modelling

A glossary of terms used in determining safe exposures and regulatory limits for pesticide residues and contaminants is included in Part 4 of the supplementary information.

What is dietary modelling?

Dietary modelling is a scientific method for estimating the levels of pesticide residues or metal contaminants a person or population may be eating. Dietary modelling techniques have been used by food regulators internationally for a number of years to check that dietary exposure to pesticide residues and metal contaminants is not likely to represent 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 exposure data on the total diet that can be compared to health standards. It is generally the exposure to the chemical from the total diet that is of interest when looking at health outcomes rather than the consumption of specific foods.

Dietary modelling at ANZFA

ANZFA uses dietary modelling in areas of work other than the total diet survey. On receiving an application to vary the *Food Standards Code*, ANZFA must undertake a comprehensive risk assessment. Dietary modelling is an important step in the risk assessment of applications relating to food chemicals, for example pesticide residues, contaminants, food additives or nutrients. Further details on dietary modelling can be found on ANZFA's website and are available upon request from ANZFA.

How is dietary modelling conducted?

DIAMOND (Dietary Modelling on 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 that food we consume and summed over all foods to determine the amount of chemical in 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 metal contaminants. These are the amounts of chemical or contaminant that can be consumed without appreciable risk on a daily or weekly basis.

The chemical levels used in dietary modelling for the ATDS are representative levels taken from the tests on each surveyed food conducted by the AGAL. The data on amount of foods consumed are taken from the recent 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 ATDS foods to the foods reported as consumed in the food consumption data (the NNS foods). This process assigns the pesticide residue and contaminant levels detected in the ATDS survey foods to the appropriate food consumption data to estimate dietary exposure to the chemical. 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, ice cream and dairy fats) with appropriate adjustment factors for concentration. Recipes are used for mixed foods to assign ingredients to the appropriate ATDS food. 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 the crop group rather than to the whole group. Therefore, the assumption of a certain residue level in the whole group is conservative in those cases.

Changes from the previous Australian total diet survey

The methods of dietary modelling have been improved from previous surveys, mainly through the use of the DIAMOND technology and the use of more recent food consumption data from the 1995 NNS instead of the 1983 and 1985 NDS used in previous Australian Market Basket Surveys (AMBS).

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 13,500 people of all ages are used in place of ‘average’ diets that were used in previous surveys. This means that dietary exposure is calculated for each individual in the survey before deriving mean dietary exposure results. Use of this 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.

Use of median levels for contaminant concentration levels

In choosing a metal contaminant concentration level for use in dietary modelling, ANZFA used the statistical middle value (median), rather than the mean level as in previous surveys, to represent the most likely level of contaminant 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 (WHO 1997). The use of medians is consistent with international practice (WHO 1997) and was used in ANZFA’s review of metal contaminants in food (ANZFA 1999).

In choosing a pesticide residue concentration level for use in dietary modelling, ANZFA chose the mean level consistent with previous surveys as it was recognised that given the high number of results below the LOR the mean level better accounted for detected levels of pesticide residues.

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.

Limitations and assumptions

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. 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 LOR. In the case of pesticide residues, the results that were lower than the LOR were assumed to be zero in dietary modelling. Given that pesticides are selectively applied to food crops, it is reasonable to assume that pesticide residues are not present when pesticide residues are less than the LOR. However, in the case of metal contaminants that occur naturally in the environment, it may not be reasonable to assume that the contaminant is not present at all in the food. For this reason, results below the LOR could be anywhere between zero and the LOR. Results for dietary exposure to metals were presented as a range, based on the two concentration levels of zero and the LOR assigned to results below the LOR.

Construction of the infant diet

As there were no data available from the NNS on children under two years, a diet was constructed to estimate dietary exposure for infants. 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 seafood and nuts were removed from the infant diet as it was assumed that infants do not generally consume these products. Consumption of breakfast cereals was assumed to be in the form of infant cereal. All milk consumption was assumed to be in the form of infant formula.

High-consumer diet

In past surveys, ANZFA has calculated dietary exposures for 'high consumers' in the population (those eating more than the average person). These exposures were prepared by multiplying the mean dietary exposures by a factor. This factor was calculated by dividing the 95th percentile energy consumption by the mean energy consumption. This approach does not provide an ideal estimate of dietary exposure for high consumers of chemicals because it is based on the energy content of foods and not on the amount of foods consumed. It assumes that the dietary patterns for a mean and a high consumer are the same. This is unlikely.

In assessing the data for the 19th survey, ANZFA investigated the approach of using DIAMOND and the 1995 NNS to calculate the dietary exposure for 'high consumers'. However, 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. Surveys conducted over longer periods account for variability in the diet and therefore provide more accurate food consumption data

for low and high consumers. However, 24-hour food recall surveys are still appropriate for providing food consumption data for the average person because mean population levels are used. These are generally not affected by the day-to-day variability in our diets.

The lack of reliable food consumption data on high consumers means that it is not possible to calculate realistic dietary exposures for high consumers. ANZFA will be investigating these problems in future surveys to determine if there are other techniques or data that can be used to estimate dietary exposures for habitual high consumers.

Dietary exposure estimates based on the 1983 and 1985 National Dietary Surveys and the 1995 National Nutrition Survey

In addition to the dietary exposures calculated for the 19th ATDS, dietary exposures were also estimated using analytical results from the 1996 AMBS (18th survey) for pesticide residues and contaminants, and food consumption data from the 1995 NNS. The results of the 18th survey previously published were based on food consumption data from the 1983 and 1985 NDS. This enabled comparisons between the 1996 AMBS (18th survey) with the 19th ATDS based on the same food consumption database and using the same dietary modelling techniques (DIAMOND computer program). It also enabled comparisons between the 1996 AMBS dietary exposure estimates based on the 1983 and 1985 NDS and those based on the 1995 NNS data.

A study currently being undertaken by the Australian Food and Nutrition Monitoring Unit, comprising a consortium from The University of Queensland, The University of Sydney and Deakin University, is investigating comparability between the 1983 and 1985 NDS data and the 1995 NNS data. This work aims to determine whether differences in estimates of average food intake between the surveys were due to actual changes in food consumption patterns during the period or due to differences in the sample design or the food intake methodology used to collect the survey data. The Commonwealth Department of Health and Aged Care is funding the work, which is known as the Bridging Study.

Preliminary results reveal that sample design variations, especially changes in the age-range of adult respondents, help to explain observed differences in average food intake estimates between the surveys. Other sample-design-related factors, such as differences in under-coverage and non-response rates between the surveys, are also likely to explain changes in the food intake estimates.

However, these sample design differences were not expected to greatly influence the comparison of food chemical exposure estimates between the 1996 AMBS and the 19th ATDS. In particular, the age-range differences are not relevant to ATDS exposure estimates, which are limited to specific age and sex subgroups. Therefore, it was possible to make direct comparisons between these data sets. Comparisons of the recalculated 1996 AMBS data with the 19th ATDS are detailed in Part B Results.

On comparing the exposures based on the two food consumption databases and 1996 analytical data, exposures for all age groups for both metals and pesticides were very similar. This suggests that general eating patterns and food intake for the identified age groups from 1983 and 1985 to 1995 did not differ significantly enough to influence the estimates of exposure from the total diet. These results also confirm the introduction of DIAMOND technology to estimate dietary exposures for the ATDS.

The 20th survey will, however, review the age groups used in calculating dietary exposures to ensure that they are in line with changing demographics.

Part B Results

Introduction

The purpose of the ATDS is to estimate the dietary exposure of Australian consumers to a range of pesticide residues and contaminants that can be found in the food supply. These exposures are estimated by determining the mean level of residue in each food and multiplying this by the respective amount of food consumed. Total dietary exposures for each pesticide or contaminant are estimated by adding together all contributions from the various foods in the Australian diet.

The results section of this report has been split into two sections: the first section covers contaminants and the second section covers pesticides. Within each of these sections there are subsections on each individual contaminant or pesticide. All the dietary exposure assessments are in the appendixes while the analytical results and background data can be found in the supplementary information on ANZFA's website.

All analytical results are expressed in milligrams per kilogram (mg/kg) of the edible portion of food prepared for consumption. Dietary exposure estimates for metal contaminants are presented as micrograms per kilogram body weight (µg/kg bw) per day. Dietary exposure estimates for pesticide residues are presented as nanograms per kilogram body weight (ng/kg bw) per day.

Contaminants

The metals examined in this survey for all foods were antimony, total arsenic, cadmium, copper, lead, mercury, selenium, tin and zinc. In addition, seafood was analysed for inorganic arsenic. The LORs for each metal are given overleaf.

Table 1: Limits of reporting for metal contaminants

Metal	Limit of reporting mg/kg
Antimony	0.01
Arsenic, total	0.01
Arsenic, inorganic	0.05
Cadmium	0.005
Copper	0.01
Lead	0.01
Mercury	0.01
Selenium	0.02
Tin	0.02
Zinc	0.01
Dibutyl tin/tributyl tin	0.001

Information on the methods of analysis for the metal contaminants is included in Part 5 of the supplementary information available on ANZFA's website.

Consistent with previous surveys, total dietary exposures were estimated for the following age–gender categories:

- adult males aged 25–34 years;
- adult females aged 25–34 years;
- boys aged 12 years;
- girls aged 12 years;
- toddlers aged two years; and
- infants aged nine months.

The food consumption and body weights data for each of the age–gender diets are included in Tables 3 and 5, respectively, of the supplementary information.

Dietary exposure estimates for toddlers were expected to be higher than the other population groups because of their high food consumption relative to body weight. This was apparent in the resulting dietary exposure estimates for metal contaminants.

The estimated dietary exposures to contaminants for these age–gender categories are given in Appendix 1. Comparisons between dietary exposures from the 19th ATDS and the revised 1996 AMBS could only be made for arsenic, cadmium, lead and mercury as these were the only metals included in the 1996 AMBS.

The following figures represent the dietary exposure to metal contaminants as a percentage of the tolerable limit. Information on the tolerable limit of each contaminant is available in Table 8 of the supplementary information on ANZFA's website.

Figure 1: Range of estimated dietary exposure to metal contaminants for adult males (25–34 years) as a percentage of the tolerable limit

Figure 2: Range of estimated dietary exposure to metal contaminants for adult females (25–34 years) as a percentage of the tolerable limit

Figure 3: Range of estimated dietary exposure to metal contaminants for boys (12 years) as a percentage of the tolerable limit

Figure 4: Range of estimated dietary exposure to metal contaminants for girls (12 years) as a percentage of the tolerable limit

Figure 5: Range of estimated dietary exposure to metal contaminants for toddlers (2 years) as a percentage of the tolerable limit

Figure 6: Range of estimated dietary exposure to metal contaminants for infants (9 months) as a percentage of the tolerable limit

Antimony

Antimony is found in low-level concentrations in water, soil and air. However, it is widely used as an industrial chemical in the manufacture of alloys and in the production of fireproofing chemicals and textiles.

The WHO/FAO Joint Expert Committee on Food Additives and Contaminants has not made any evaluation 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 ANZFA 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 19th survey are given in Table 9 in the supplementary information on ANZFA's website. The estimated dietary exposures to antimony for each age–gender category are given in Appendix 1.

The highest calculated mean exposure to antimony was for toddlers because of their high food consumption relative to body weight. This calculated exposure for toddlers gave a wide range (4% to 240% 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 (0.01 mg/kg) and the upper limit was calculated by assuming that foods contained 0.01 mg/kg of antimony if they were reported as containing less than the LOR. The wide range results from limitations of the current analytical method, which can measure antimony levels down to 0.01 mg/kg but no lower, and the high proportion of results that were reported as less than the LOR. The actual exposure for antimony lies within this calculated range and it is not possible with the current method to be more precise.

In the recent ANZFA review of the *Food Standards Code*, more comprehensive data on antimony levels in food were available than in the 19th ATDS. Estimated dietary exposure to antimony was lower than reference health standards. The review concluded that there was no cause for concern for public health and safety. The review recommended, however, that the ATDS continue to monitor dietary exposures to antimony. For future surveys, ANZFA will request antimony analyses with a lower LOR to enable more specific exposures to be calculated.

Recommendation

It is recommended that analyses with a lower LOR for antimony be undertaken in future surveys so that more accurate dietary exposure assessments can be calculated. ANZFA has been informed that methods are now available that can detect antimony to a LOR of 0.002 mg/kg and ANZFA will be seeking to use these methods in future surveys.

Arsenic

Arsenic occurs naturally in both organic and inorganic forms. In the past, arsenic compounds were commonly used in drugs, but the main uses today are in pesticides, veterinary drugs and industrial applications. 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.

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 (primarily in organic form). However, inorganic arsenic is more toxic than organic arsenic (WHO 1981).

This survey examined total arsenic in all foods and inorganic arsenic in crocodile, fish fillets, mussels, canned red salmon and canned crab. Inorganic arsenic was only measured in seafood and crocodile because of the generally higher levels of arsenic that these foods contain.

A level of approximately 0.0029 mg/kg bw/day is the lowest observable effects level (LOEL) based on a review of available epidemiological data conducted by ANZFA. This level was rounded off to 0.003 mg/kg bw/day to be the tolerable limit for inorganic arsenic for the purposes of dietary modelling.

The mean, median, maximum and minimum levels of total arsenic and inorganic arsenic found in the foods analysed are given in Tables 10 and 11 in the supplementary information on ANZFA's website. The estimated dietary exposure to total arsenic and inorganic arsenic for each age–gender category are given in Appendix 1.

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 an overestimate because not all arsenic is present as inorganic arsenic.

Even with the overestimation for inorganic arsenic content, all estimated dietary exposures were below the tolerable limit for inorganic arsenic. The highest mean exposure to arsenic was for two-year-olds because of their high food consumption relative to body weight. This exposure ranged from 36% of the tolerable limit up to 57%. The wide range results from limitations of the analytical method, which can measure arsenic down to 0.01 mg/kg but no lower, and the high proportion of results reported as 'less than the LOR'. Dietary exposures to arsenic are within acceptable health standards.

Seafood makes the greatest contribution to the dietary intake of arsenic. Although total arsenic levels were

higher in seafood than in other foods, the more toxic inorganic arsenic levels were found to be low in mussels and were less than the LOR in crocodile, fish fillets, canned red salmon and canned crab.

Recommendation

It is recommended that analyses with a lower LOR for arsenic be undertaken in future surveys so that more accurate dietary exposure assessments can be calculated.

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.

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 and certain organic fertilisers based on manures.

The tolerable limit for cadmium, set at the 33rd meeting of the WHO/FAO Joint Expert Committee on Food Additives, is 7 µg/kg bw/week (WHO 1989).

The mean, median, maximum and minimum levels of cadmium found in the foods analysed are given in Table 12 in the supplementary information on ANZFA's website. The estimated dietary exposures to cadmium for each age–gender category are given in Appendix 1.

The highest mean exposure to cadmium was for two-year-olds because of their high food consumption relative to body weight. This exposure ranged from 17% to 58% of the tolerable limit. This range results from limitations of the analytical method, which can measure cadmium levels down to 0.01 mg/kg but no lower, and the high proportion of results reported as 'less than the LOR'. All estimated dietary exposures were below the tolerable limit of 7 µg/kg bw/week. Dietary exposures to cadmium are within acceptable safety standards.

Recommendation

It is recommended that analyses with a lower LOR for cadmium be undertaken in future surveys so that more accurate dietary exposure assessments can be calculated.

Copper

Copper is widely distributed in nature. 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.

Copper is an essential element. Enzymes containing copper are important for the body to transport and use iron. Anaemia is therefore one of the first symptoms of copper deficiency. Copper deficiency, however, is not common, 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. Copper toxicity is rare, except in those suffering Wilson's disease (a hereditary disease resulting in excessive uptake and accumulation of copper by the body, especially in the liver and brain).

The mean, median, maximum and minimum levels of copper in foods are given in Table 13 in the supplementary information on ANZFA's website. The estimated dietary exposures to copper for each age–gender category are given in Appendix 1.

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. This value has been used as the tolerable limit for the purposes of dietary modelling (WHO 1996).

All estimated mean intakes are below the tolerable limit. Because of their high food consumption relative to body weight, the highest mean exposure to copper was for two-year-olds, calculated at 21% of the tolerable limit. No range has been presented for copper because a specific amount of copper was reported for all samples and no allowance had to be made for results reported as containing 'less than the LOR'. Dietary exposures to copper are within acceptable health standards.

Lead

Lead is found almost everywhere, 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—the largest use is in lead batteries.

A significant source of exposure to lead is via food. This is due to lead-contaminated soil and dust finding its way into the food and water supply. Lead can also 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. 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, set at the 30th meeting of the WHO/FAO Joint Expert Committee on Food Additives, is 25 µg/kg bw/week (WHO 1987b).

The mean, median, maximum and minimum levels of lead in foods are given in Table 14 in the supplementary information on ANZFA's website. Estimated dietary exposures to lead for each age–gender category are given in Appendix 1.

The highest mean exposure to lead was for two-year-olds because of their high food consumption relative to body weight. This exposure ranged from 33% to 53% of the tolerable limit. This range results from limitations of the analytical method, which can measure lead down to 0.01 mg/kg but no lower, and the high proportion of results reported as 'less than the LOR'. All estimated intakes of lead were below the tolerable limit of 25 µg/kg bw/week. Dietary exposures to lead are within acceptable safety standards.

Recommendation

It is recommended that analyses with a lower LOR for lead be undertaken in future surveys so that more accurate dietary exposure assessments can be calculated.

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.

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, the most common organic form being methyl mercury. Methyl mercury is largely produced from the methylation of inorganic mercury by microbial activity. 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. Seafoods specifically contain much higher levels of mercury, largely in the toxic methyl mercury form, whereas most other foods contain very low levels of mercury, almost entirely in the inorganic form. In this survey, total mercury, which included both organic and inorganic mercury, was measured.

Methyl mercury accumulates in the brain. The developing nervous system in the foetus is at particular risk. Effects include retarded psychomotor development, mental retardation and seizures.

The tolerable limit for 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 1978).

The mean, median, maximum, and minimum levels of mercury in foods are given in Table 15 in the supplementary information on ANZFA's website. Seafood was shown to be the greatest source of mercury in all the diets for all age–gender categories. Of the foods analysed, fish fillets had the highest level of mercury. Estimated dietary exposures to mercury for all age–gender categories are given in Appendix 1.

Because of their high food consumption relative to body weight, the highest mean exposure to mercury was for two-year-olds and infants, where this exposure ranged from 5% up to 140% of the tolerable limit for two-year-olds, and from 2% up to 150% of the tolerable limit for infants. This range results from limitations of the analytical method, which can measure mercury down to 0.01 mg/kg but no lower, and the high proportion of samples reported as containing 'less than the LOR'.

The upper limits of these ranges indicate that the exposure to mercury could be above the acceptable health standard. However, the upper limit of the range is an overestimate because it assumes that foods contain 0.01 mg/kg of mercury if these foods are reported as containing less than the LOR (0.01 mg/kg). Similarly, the lower limit of the range is an underestimate because it assumes that foods contain no mercury if these foods are reported as containing less than the LOR.

In the recent ANZFA review of the *Food Standards Code*, 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 has developed an advisory statement for pregnant women on mercury in fish, in consultation with health professionals and the fishing industry.

Recommendation

It is recommended that analyses with a lower LOR for mercury be undertaken in future surveys so that more accurate dietary exposure assessments can be calculated. ANZFA has been informed that methods are now available that can detect mercury to a LOR of 0.002 mg/kg and ANZFA will be seeking to use these methods in future surveys.

Selenium

Selenium is a metalloid, both essential and toxic to humans. 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. It may produce symptoms associated with changes in nail pathology, hair loss and dental decay. 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.

The Australian Recommended Dietary Intake (RDI) of selenium was set by the NHMRC in 1987. The RDIs are 85 µg/day (1.13 µg/kg bw) for adult males; 70 µg/day (1.18 µg/kg bw) for adult females; 85 µg/day (2.14 µg/kg bw) for boys; 70 µg/day (1.68 µg/kg bw) for girls; 25 µg/day (2.03 µg/kg bw) for toddlers; and 15 µg/day (1.65 µg/kg bw) for infants (NHMRC 1991).

As yet, the WHO has made no recommendation regarding tolerable limits of selenium (WHO 1987a). However, the US National Research Council has suggested that toxicity will occur after prolonged ingestion of upwards of 3 000 µg/day (Reilly 1980) which is equivalent to 50µg/kg bw/day (based on the WHO reference weight of 60 kg for an adult).

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 LOEL. 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.

The mean, median, maximum and minimum levels of selenium in foods are given in Table 16 in the supplementary information on ANZFA's website. Estimated dietary exposure to selenium for all age–gender categories are given in Appendix 1.

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 20% to 29% of the tolerable limit of 750 µg/day (12.5 µg/kg bw/day). This range results from limitations of the analytical method, which can measure selenium down to 0.01 mg/kg but no lower, and the proportion of samples reported as containing 'less than the LOR'.

All estimated mean intakes of selenium for all age–gender categories are below the suggested tolerable limit of 3 000 µg/day (50 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 below). The lower dietary exposure estimates (based on zero values for non-detect results) were lower than the RDI for female adults, boys, girls and infants but exceeded the RDI for male adults and toddlers. The higher dietary exposure estimate (based on 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: Estimated dietary exposures to selenium

	Adult males 25–34 years µg/kg bw/day	**Adult females 25–34 years µg/kg bw/day**	**Boys 12 years µg/kg bw/day**	**Girls 12 years µg/kg bw/day**	**Toddlers 2 years µg/kg bw/day**	**Infants 9 months µg/kg bw/day**
RDI*	1.13	1.18	2.14	1.68	2.03	1.65
Dietary exposure	1.2–1.7	0.97–1.4	1.5–2	1.1–1.5	2.5–3.6	1.0–2.9

* RDI expressed per kilogram body weight for each age–gender group (NHMRC 1991).

Recommendation

It is recommended that analyses with a lower LOR for selenium be undertaken in future surveys so that more accurate dietary exposure assessments can be calculated.

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. Tin is also used extensively for food containers and food-processing equipment as it is generally resistant to corrosion and easy to solder.

The main route of exposure to tin is through food, although levels are generally low. Higher levels are found in canned foods as a result of the coating or plate breaking down. Exposure to tin contamination is greatly reduced when the cans are lacquered.

Toxicity from tin exposure is low. However, high levels of tin 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.

The WHO/FAO Joint Expert Committee on Food Additives, at its 33rd meeting, set a tolerable limit of 14 mg/kg bw/week for inorganic tin (WHO 1989), 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.

The mean, median, maximum and minimum levels of tin in canned foods are given in Table 17 in the supplementary information on ANZFA's website. Estimated dietary exposures to tin for all age–gender categories are given in Appendix 1.

Because of their high food consumption relative to body weight, the highest mean exposure to tin was for two-year-olds, where this exposure ranged from 1.5% to 1.6% of the tolerable limit. All estimated exposures to tin for all age–gender categories are well below the tolerable limit for tin of 14 mg/kg bw/week.

All results were below the current maximum permitted concentrations (MPCs) listed in Standard A12 of the *Food Standards Code* for tin in canned foods. Canned peas and canned pineapple contained the highest concentrations of tin.

Dibutyl and tributyl tin

Tributyl tin compounds are used as antifouling agents on boats as well as for fungal control on timber. Occupational exposure represents the most significant hazard to humans with respect to exposure to organotin compounds.

Mussels were analysed for dibutyl tin (DBT) and tributyl tin (TBT) which were detected in approximately half the samples. The concentrations ranged from less than the LOR (<0.001 mg/kg) to 0.021 mg/kg for dibutyl tin and from less than the LOR (<0.001 mg/kg) to 0.033 mg/kg for tributyl tin. From this data, the median level for both DBT and TBT was 0.005 mg/kg.

The margin of safety for this amount of TBT/DBT can be determined by comparing the intake level of TBT in humans with the intake level known to cause toxic effects in experimental animals. The lowest level shown to have marginal toxic effects in animals is 0.25 mg/kg bw/day (WHO 1990b).

The median concentration for TBT in mussels is 0.005 mg TBT /kg and the median concentration for DBT in mussels was 0.005 mg DBT/kg. If it were assumed that DBT was as toxic as TBT then the total median TBT equivalent concentration would be 0.010 mg/kg.

A high consumer of molluscs (95th percentile for males aged 25–34) is estimated to consume 402 grams per day (1995 National Nutrition Survey). If a person were to consume 402 grams of mussels containing 0.010 mg/kg of TBT every day, then it could be calculated that these high consumers would be exposed to 0.004 mg per day of TBT per person or 0.00006 mg TBT/kg bw/day. This level of consumption of TBT is less than one thousandth of that which resulted in toxic effects in animals (0.25 mg/kg bw/day).

It should be recognised that this calculation overestimates the exposure because it assumes that 402 grams of mussels would be consumed every day (which would be a gross overestimate). On this basis, it can be concluded that consumption by the public of mussels containing the levels of TBT and DBT detected in this survey is safe.

Zinc

Zinc has been mixed with copper to produce brass for more than 2000 years. Major uses of zinc today are in the manufacture of non-corrosive alloys and brass, and in galvanising steel and iron.

Zinc is an essential metal which is extremely important to nutritional 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.

Zinc is widely distributed in food. However, concentrations are low. Seafood, meat and nuts are good sources of zinc.

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). For the purposes of dietary modelling, ANZFA used a tolerable limit of 1 mg/kg bw/day.

The mean, median, maximum and minimum levels of zinc in foods are given in Table 18 in the supplementary information on ANZFA's website. Estimated dietary exposures to zinc for all age–gender

categories are given in Appendix 1.

The highest mean exposure to zinc was for two-year-olds and infants where the exposure was 66% of the tolerable limit. No range has been presented because a specific amount of zinc was reported for all samples and no allowance had to be made for samples reported as containing 'less than the LOR'.

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.

Estimated dietary exposures to zinc exceeded the RDI for each age–gender group by a considerable margin, except for female adults where the estimated dietary exposure was slightly lower than the RDI for that group (see table below). 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 this age group.

Table 3: Estimated dietary exposures to zinc

	Adult males 25–34 years µg/kg bw/day	**Adult females 25–34 years µg/kg bw/day**	**Boys 12 years µg/kg bw/day**	**Girls 12 years µg/kg bw/day**	**Toddlers 2 years µg/kg bw/day**	**Infants 9 months µg/kg bw/day**
RDI*	160	203	302	289	365	494
Dietary exposure	260	190	400	310	660	660

* RDI expressed per kilogram body weight for each age–gender group (NHMRC 1991).

Aflatoxins

Aflatoxins are a group of extremely toxic metabolites produced by the common fungi *Aspergillus flavus* and *Aspergillus parasiticus*, which primarily affect the liver. The WHO considers aflatoxins to be potential carcinogens (WHO 1987c).

Aflatoxins have the potential to contaminate foodstuffs and animal feeds on which mould has been allowed to grow. They can contaminate maize, peanuts, grain 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. Aflatoxin contamination of milk is common in Europe where intensive supplementary feeding of dairy herds is practised. In Australia, dairy herds predominantly graze and aflatoxin contamination has not been reported.

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 acutely toxic of the aflatoxins. Aflatoxins M1 and M2, commonly known as milk toxins, are metabolic by-products found in cow's milk after the animal has ingested aflatoxins in feed. Milk aflatoxins retain the toxic properties of the parent compound.

The best way to control the presence of aflatoxins in animal feeds and food is through good agricultural and manufacturing practices that prevent fungal growth. Aflatoxins 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 some 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).

The 19th survey examined walnuts, tahina and roasted salted peanuts for aflatoxins. In addition, milk samples were examined for the presence of Aflatoxin M1. The results for aflatoxins in food are provided in Tables 23 and 24 in the supplementary information on ANZFA's website.

No aflatoxins were detected in milk, tahina or walnuts. Aflatoxins were found in one of the nine analytical samples of roasted salted peanuts. This sample was found to contain 0.038 mg/kg of Aflatoxin B1 and 0.006 mg/kg of Aflatoxin B2. This level of Aflatoxin B1 exceeds the Australian *Food Standards Code* MPC for aflatoxins in nuts of 0.015 mg/kg and was brought to the attention of the relevant enforcement agency.

No ADI has been set for aflatoxins. The WHO suggests that intake of aflatoxins be kept as low as possible (WHO 1987c) and the Codex Committee on Food Additives and Contaminants has established an MPC of 0.015 mg/kg for aflatoxins in peanuts. This limit is the same as that included in the Australian *Food Standards Code*. Limits placed on the level of aflatoxins in nuts and other foods are the most appropriate way to control aflatoxin intake.

Recommendation

It is recommended that future surveys continue to monitor aflatoxins in peanut products.

Polychlorinated biphenyls (PCBs)

PCBs are industrial pollutants that are extremely stable compounds. Before the 1970s they were used extensively in a wide range of industrial applications such as in heat transfer and hydraulic systems, and as insulators in electrical components.

As PCBs degrade slowly, widespread environmental contamination has occurred. When environmental contamination was linked to the toxic properties of PCBs, Australia banned their further importation. As well, policies to retrieve all components containing PCBs were implemented.

At very high concentrations, PCBs are reputed to produce a variety of effects including skin discolouration, skin eruptions and respiratory problems. All PCBs are fat-soluble and therefore accumulate in the fat tissue. They can also concentrate up the food chain.

The Joint FAO/WHO Expert Committee on Food Additives has not prescribed a tolerable limit for PCBs. However, it has concluded that, on the basis of studies with monkeys, 40 µg/kg bw/day is a no-effect level (WHO 1990a).

In the 19th survey, no PCBs were found in any food (LOR 0.01 mg/kg).

Comparison between the 19th ATDS and the recalculated 1996 AMBS (18th ATDS) results for contaminants

In the 18th survey (sampled in 1996), foods were only analysed for the contaminants arsenic, cadmium, mercury, lead, PCBs and aflatoxins. Dietary exposures were recalculated using analytical results from the 18th survey and the more recent food consumption data from the 1995 NNS. These recalculations are included in Tables A2 and A4 of Appendix 1.

In general terms it is possible to note some differences between the dietary exposures to contaminants in the 18th survey and the 19th survey. These differences have not been emphasised in great detail because

the small number of samples and the large range of foods in Australia's food supply mean that it is not possible to draw many definite conclusions.

The key items to note from the recalculated exposures for the 18th survey were that:

- dietary exposure to arsenic and cadmium were within acceptable safety standards and were consistent with those determined in the 19th survey;
- dietary exposure to lead was within acceptable safety standards and was three to five times less than that determined for the 19th survey. This is because many sugar-containing foods (soft drinks, jam, jelly cordials) were assigned the lead concentration in honey (0.080 mg/kg) as part of the dietary exposure calculation in the 19th ATDS. These foods were assigned the concentration for honey because honey, of all foods sampled in the 19th ATDS, was assumed to best represent sugar-containing food. In the 18th ATDS raw sugar was sampled and no detections of lead were reported in raw sugar (<0.01 mg/kg). 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. In future surveys, a more representative food will be sampled for sugar-containing foods to avoid overestimating the exposure to lead; and
- the method of analysis and the high proportion of results reported as 'less than the LOR' meant that a definitive dietary exposure calculation was not possible for mercury. However, the range of exposure calculated from the 18th survey data was similar to that determined for the 19th survey.

Pesticides

Pesticides benefit agriculture and the community, and assist in food production by controlling pests and diseases.

Pesticide residues may remain in crops and animals following treatment. Good agricultural practices and food processing, including preparation in the home, can reduce the levels of these residues. In some cases, residues may still be present in the food we eat.

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 Table 6 in the supplementary information on ANZFA's website. The range of pesticide residues tested were:

- chlorinated organic pesticides (organochlorines);
- organophosphorus pesticides;
- synthetic pyrethroid pesticides;
- fungicides including chlorothalonil, dicloran, diphenylamine, dithiocarbamates, procymidone and vinclozolin;
- some carbamates; and
- piperonyl butoxide (a synergist).

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, non-biodegradable compounds that persist in soil and concentrate in the food chain.

Due to their fat solubility they are stored in the fatty tissue of humans and animals. The use of persistent

organochlorines in developed countries has been heavily restricted since it was shown that some of these compounds were becoming an environmental hazard and an impediment to trade in food commodities.

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.

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 the case for organochlorine pesticides.

Organophosphorus pesticides act on the central nervous system of insects and animals, and in high doses they are highly toxic. They have the potential to prevent the break-up of the chemical acetylcholine, which transmits signals between nerve 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.

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.

Carbamate pesticides act on the central nervous system of insects and animals and in high doses are highly toxic. They prevent the break-up of the chemical acetylcholine, which transmits signals between nerve cells, and thus interfere with nervous system function. Carbamate pesticides tend not to accumulate in the human body.

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.

Fungicides

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

The fungicides examined in the 19th ATDS were all protectant fungicides and included chlorothalonil, dicloran, diphenylamine, dithiocarbamates, iprodione, procymidone and vinclozolin.

Dithiocarbamates are a group of chemically similar fungicides. They include the chemically related compounds mancozeb, maneb, metiram, propineb, thiram, zineb and ziram. The chemistry of the dithiocarbamates means that they require a different method of analysis from that used for other fungicides. Dithiocarbamate residues are analysed by measuring the amount of carbon disulphide given off when the food is chemically digested with hot acid. Analysts can not differentiate between most dithiocarbamates because the required pesticide-specific methods of analysis are not available.

Results and dietary exposures to pesticides

Unlike contaminants, 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 the LOR’ for pesticide residues were assumed to contain no pesticide residues for the purposes of dietary exposure assessments. Aldrin, dieldrin, endrin, heptachlor, hexachlorobenzene, chlordane and DDT are not registered for use in Australia. However, for simplicity, these chemicals have been treated as pesticides for the purposes of estimating dietary exposure.

Figure 7: Estimated dietary exposure to pesticide residues for adult males (25–34 years) as a percentage of the ADI (19th ATDS)

Figure 8: Estimated dietary exposure to pesticide residues for adult females (25–34 years) as a percentage of the ADI (19th ATDS)

Figure 9: Estimated dietary exposure to pesticide residues for boys (12 years) as a percentage of the ADI (19th ATDS)

Figure 10: Estimated dietary exposure to pesticide residues for girls (12 years) as a percentage of the ADI (19th ATDS)

The concentrations of pesticide residues reported in the surveyed foods are included in Part 3 of the supplementary information on ANZFA's website, sorted by food (Table 23) and by pesticide (Table 24). The LOR for all pesticide residues was 0.01 mg/kg, except for dithiocarbamates (0.1 mg/kg) and benomyl/carbendazim (0.2 mg/kg). Part 3 of the supplementary information includes data on the concentrations of pesticide residues reported in both the 18th and 19th surveys.

Dietary exposures were estimated only when a pesticide was detected in a food. The estimated dietary exposures to the pesticide residues reported in the surveyed foods (that is, for those pesticides that were detected) are in Table A5 in Appendix 2. Dietary exposures to dithiocarbamates were calculated differently from other pesticides and are discussed in a separate section below.

Some pesticides were not detected in any food and consequently their estimated intakes were zero. These pesticides are tabulated in Appendix 4.

The dietary exposures for each pesticide are also expressed as percentages of the respective ADIs (Tables A7 and A8 in Appendix 2). All exposures are less than the applicable health standard.

Figure 11: Estimated dietary exposure to pesticide residues for toddlers (2 years) as a percentage of the ADI (19th ATDS)

The estimated dietary exposures to pesticide residues, except dithiocarbamates, for different age–gender groups are given in Figures 7 to 12. To simplify the figures, only dietary exposures greater than 0.1% of the ADI have been included. The dietary exposures for all detected pesticides except dithiocarbamates are included in Appendix 2.

Figure 12: Estimated dietary exposure to pesticide residues for infants (9 months) as a percentage of the ADI (19th ATDS)

Dietary exposures to dithiocarbamates

Some crops, particularly vegetables such as brassicas and onions, naturally produce carbon disulphide under the acid digestion conditions employed in analysis. In calculating the dietary exposure for dithiocarbamates, it was assumed that all carbon disulphide generated by acid digestion was from dithiocarbamates. This is likely to lead to an overestimate of the exposure.

The level of dithiocarbamates in food is indicated by the concentration of carbon disulphide that is measured by the analytical method. Maximum residue limits (MRLs) for dithiocarbamates are also based on the carbon disulphide released by acid digestion of a sample. The ADI, however, is not based on carbon disulphide but on the whole parent molecule. Therefore, to calculate the intake of dithiocarbamates for comparison with the ADI, a molecular weight adjustment is necessary to convert the carbon disulphide values to parent dithiocarbamate values. The conversion factors are different for each dithiocarbamate.

To obtain the most conservative estimate of dietary exposure, it was assumed that all dithiocarbamate residues were thiram residues. Thiram has the lowest ADI of all the dithiocarbamates (0.004 mg/kg bw/day). The actual dietary exposure expressed as a percentage of the ADI will be lower than this conservative estimate because some of the measured carbon disulphide will have come from other dithiocarbamates and natural compounds in onions and brassicas.

The analyses for dithiocarbamates were only performed on fruit and vegetable products as only these foods are treated with these fungicides. Dithiocarbamates are not used on animals.

The estimated dietary exposures to thiram are presented in Appendix 3. The levels of the dithiocarbamates found in foods are given in Tables 23 and 24 in the supplementary information on ANZFA's website. The ADIs for dithiocarbamates are given in Table 7 of the supplementary information.

ANZFA investigated the option of refining the dietary exposure by considering the existing registrations and MRLs for dithiocarbamates, and by investigating whether data were available on the specific quantities of dithiocarbamates used. However, this proved to be problematic because:

- data are not available on the specific quantities of dithiocarbamates that are used;
- an existing registration or MRL does not provide information on the relative frequency of each dithiocarbamate's use and does not account for misuse; and
- it is questionable whether the MRLs and usage data can be extrapolated to the residues of individual dithiocarbamates in food.

Despite the overestimation, the estimated dietary exposure to dithiocarbamates was below the ADI for all age–gender categories. The NRA has scheduled a review of the use of some dithiocarbamates as part of their Existing Chemicals Review Program. ANZFA has recommended to the NRA that the dietary exposure to dithiocarbamates should be considered during this review, particularly the need to determine more definitive models for estimating the exposure to individual dithiocarbamates. Once this NRA review is complete, ANZFA will again include dithiocarbamates in the ATDS to reassess the exposure to dithiocarbamates.

Recommendation

It is recommended that:

- more definitive models for estimating the exposure to individual dithiocarbamates be developed so that more accurate dietary exposure assessments can be calculated; and
- dithiocarbamates be included in future surveys once these models have been developed.

Comparison between the 19th ATDS and the recalculated 1996 AMBS (18th ATDS) results for pesticide residues

Dietary exposures for pesticide residues were recalculated using analytical results from the 18th survey (sampled in 1996) and the more recent food consumption data from the 1995 NNS.

In general terms it is possible to note some differences between the dietary exposures to pesticide residues in the 18th survey and the 19th survey (samples taken in 1998). These differences have not been emphasised in great detail because the small number of samples and the large range of foods in the food supply mean that it is not possible to draw many definite conclusions. The key differences were:

- ***The exposure to dicofol appeared to be higher in the 19th survey than in the 18th survey for all age–gender groups except infants.***

  Dicofol is an acaricide used to control mites on fruit and vegetables. The calculated exposure to dicofol is greater in the 19th survey than in the 18th survey for all diets except the infant diet. This has occurred because of the way the ATDS foods were 'mapped' to NNS foods in each survey. In the 19th survey all berries, grapes and strawberries were assigned the dicofol concentration that was detected in grapes (0.2 mg/kg). However, in the 18th survey, strawberries were sampled separately and only grapes were assigned the higher dicofol concentration that was detected in grapes (0.2 mg/kg), with strawberries and other berries being assigned the much lower dicofol concentration (0.001 mg/kg) that was detected in strawberries. The higher calculated exposure to dicofol in the 19th survey is not likely to be a result of higher or greater residues of dicofol or major changes in food consumption patterns.

  For infants, an additional change between the 18th and 19th surveys was a change in the data sources used to derive the infant diet. The diets used in the 19th survey were derived directly from the 1995 data for two-year-old children, and those for the 18th survey from a longitudinal survey of young children in Perth (1979–1984) (Hitchcock et al. 1986). In both cases, it was assumed that the infant diet would meet the same energy requirements for a 9-month-old boy of median body weight and that infant formula would provide half of the total energy requirement. The difference between the infant diets for the two surveys was that less food was required to meet the energy requirement for the solid food component of the 19th survey diet than that of the 18th survey diet because the total energy content of the foods chosen was higher. Therefore the amount of grapes and berries in the infant diet for the 19th survey was lower than that for the 18th survey and the estimated dietary exposure to dicofol did not appear to increase as for the other age groups.

  Recommendation

  It is recommended that dicofol continue to be monitored in future surveys and that representative foods be included in future surveys to enable more accurate calculation of the dietary exposure to dicofol.

- ***The dietary exposures to organophosphorus pesticide residues are mostly in the range of 0–5% of the ADI.***

  All of these chemicals are used as insecticides to protect our grain, fruit and vegetables from insects.

  Recommendation

  It is recommended that organophosphorus pesticide residues continue to be monitored to determine both chronic and acute dietary exposure to these pesticides.

- ***The carbamates carbaryl and pirimicarb were included in the 19th survey but were not included in the 18th survey.***

  Although carbaryl and pirimicarb are not organophosphorus insecticides, they are insecticides with similar toxicological effects to organophosphorus compounds. Pirimicarb and carbaryl were included in the 19th survey and the exposures to these chemicals were within acceptable health standards. Pirimicarb, carbaryl and other carbamates will be included in future surveys to monitor the exposure to these compounds.

  Recommendation

  It is recommended that carbamates continue to be monitored to determine both chronic and acute dietary exposure to these pesticides.

Table of recommendations

It is recommended that:

- in future surveys, analyses with lower LORs for antimony, arsenic, cadmium, mercury, lead and selenium be undertaken so that more accurate dietary exposure assessments can be calculated;
- future surveys continue to monitor aflatoxins in peanut products;
- more definitive models for estimating the exposure to individual dithiocarbamates be developed so that more accurate dietary exposure assessments can be calculated;
- dithiocarbamates be included in future surveys once appropriate dietary exposure models have been developed;
- dicofol, organophosphorus and carbamate pesticide residues continue to be monitored in future surveys to determine both chronic and acute dietary exposure to these pesticides;
- the 20th ATDS consider the acute dietary exposure to organophosphorus and carbamates pesticide residues;
- that representative foods be included in future surveys to ensure that representative and realistic dietary exposure assessments can continue to be calculated;
- the 20th survey review the age groups used in calculating exposures to ensure that they are in line with changing demographics;
- the possibility of greater collaboration with the New Zealand Total Diet Survey be investigated; and
- method development for lower limits of reporting for antimony and mercury be undertaken as a matter of urgency.

Part C Appendixes

Supplementary information to the 19th ATDS can be found on ANZFA's website www.anzfa.gov.au.

Appendix 1

Notes on the tables:

1 A range of exposures is presented in these tables. The lower end of the range (the first result) assumes that results less than the limit of reporting = 0 and the upper end of the range (the second result) assumes that results less than the limit of reporting = limit of reporting. The limits of reporting are provided on page 12 of this report.

2 There were no results reported as less than the limit of reporting for copper and zinc, so only one dietary exposure estimate is provided.

3 1 µg = one millionth of 1 g.

4 Estimated dietary exposures are based on food consumption data from the 1995 National Nutrition Survey.

5 Tolerable limits for metal contaminants are listed in Table 8 in the supplementary information on ANZFA's website.

Table A1: Mean estimated daily dietary exposure to metals in µg/kg bw based on median analytical results (19th ATDS)

	Adult males	**Adult females**	**Boys**	**Girls**	**Toddlers**	**Infants**
	25–34 years	**25–34 years**	**12 years**	**12 years**	**2 years**	**9 months**
Metal	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**
Antimony	0.01 – 0.39	0.01 – 0.34	0.01 – 0.45	0.01 – 0.35	0.02 – 0.95	0.00 – 0.53
Arsenic, total	0.76 – 0.98	0.59 – 0.82	0.91 – 1.17	0.48 – 0.68	1.1 – 1.7	0.57 – 1.4
Cadmium	0.10 – 0.26	0.09 – 0.23	0.12 – 0.30	0.09 – 0.23	0.17 – 0.58	0.08 – 0.56
Copper	17	15	26	18	42	74
Lead	0.42 – 0.73	0.27 – 0.56	0.70 – 1.01	0.59 – 0.84	1.19 – 1.92	0.57 – 1.50
Mercury	0.03 – 0.42	0.02 – 0.36	0.03 – 0.48	0.02 – 0.37	0.03 – 0.99	0.02 – 1.1
Selenium	1.2 – 1.7	0.97 – 1.4	1.5 – 2.0	1.1 – 1.5	2.5 – 3.6	1.0 – 2.9
Tin	8.8 – 9.4	9.0 – 9.6	11	9.1 – 9.6	31 – 32	13 – 15
Zinc	260	190	400	310	660	660

*Table A2: Mean estimated daily dietary exposure to metals in **µ**g/kg bw based on median analytical results (18th ATDS)*

	Adult males	**Adult females**	**Boys**	**Girls**	**Toddlers**	**Infants**
	25–34 years	**25–34 years**	**12 years**	**12 years**	**2 years**	**9 months**
Metal	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**	**µg/kg bw/day**
Arsenic	0.91 – 1.04	0.66 – 0.78	1.3 – 1.5	0.63 – 0.81	1.2 – 1.9	0.90 – 1.9

Cadmium	0.09 – 0.16	0.08 – 0.15	0.13 – 0.25	0.10 – 0.19	0.17 – 0.49	0.12 – 0.64
Lead	0.10 – 0.26	0.10 – 0.25	0.13 – 0.41	0.10 – 0.31	0.23 – 0.92	0.20 – 1.3
Mercury	0.05 – 0.24	0.04 – 0.22	0.06 – 0.39	0.04 – 0.28	0.06 – 0.82	0.04 – 1.2

Table A3: Mean estimated daily dietary exposure to metals as a percentage of the tolerable limit based on median analytical results (19th ATDS)

	Adult males 25–34 years	**Adult females 25–34 years**	**Boys 12 years**	**Girls 12 years**	**Toddlers 2 years**	**Infants 9 months**
Metal	**%**	**%**	**%**	**%**	**%**	**%**
Antimony	1.5 – 97	1.3 – 85	3.4 – 110	1.7 – 87	4.3 – 240	0.93 – 130
Arsenic	25 – 33	20 – 27	30 – 39	16 – 23	36 – 57	19 – 48
Cadmium	9.9 – 26	8.6 – 23	12 – 30	8.6 – 23	17 – 58	7.9 – 56
Copper	8.4	7.5	13	9.1	21	37
Lead	12 – 20	7.6 – 15	19 – 28	16 – 23	33– 53	16 – 42
Mercury	4.4 – 60	3.4 – 52	4.7 – 69	2.2 – 52	4.8 – 140	2.2 – 150
Selenium	9.5 – 14	7.8 – 11	12 – 16	9.0 – 12	20 – 29	8.2 – 23
Tin	0.44 – 0.47	0.45 – 0.48	0.54 – 0.57	0.45 – 0.48	1.5 – 1.6	0.67 – 0.76
Zinc	26	19	40	31	66	66

Table A4: Mean estimated daily dietary exposure to metals as a percentage of the tolerable limit based on median analytical results (18th ATDS)

	Adult males 25–34 years	**Adult females 25–34 years**	**Boys 12 years**	**Girls 12 years**	**Toddlers 2 years**	**Infants 9 months**
Metal	**%**	**%**	**%**	**%**	**%**	**%**
Arsenic	30 – 35	22 – 26	43 – 50	21 – 27	41 – 62	30 – 63
Cadmium	9.1 – 16	7.9 – 15	13 – 25	9.5 – 19	17 – 49	12 – 64
Lead	2.9 – 7.1	2.9 – 6.9	3.6 – 11	2.8 – 8.7	6.6 – 26	5.4 – 35
Mercury	6.8 – 34	5.5 – 31	8.8 – 56	5.1 – 49	8.9 – 120	5.9 – 170

Appendix 2 Dietary exposure to pesticides

Notes on the tables:

1 1 ng = one millionth of 1 mg.

2 Estimated dietary exposures are based on food consumption data from the 1995 National Nutrition Survey.

3 Pesticides screened for but not detected are included in Appendix 4.

4 Deltamethrin, hexaconazole and parathion were detected but they have not been included in either Table A5 or Table A7 because their calculated exposure in all diets was less than 0.01 ng/kg body weight and less than 0.01% of their respective ADIs.

Table A5: Mean estimated daily dietary exposure to detected pesticide residues in ng/kg bw based on mean analytical results (19th ATDS)

Chemical	Adult males 25–34 years ng/kg bw/day	Adult females 25–34 years ng/kg bw/day	Boys 12 years ng/kg bw/day	Girls 12 years ng/kg bw/day	Toddlers 2 years ng/kg bw/day	Infants 9 months ng/kg bw/day
Azinphos-methyl	9.32	11.27	30.41	21.18	63.23	16.08
Benomyl/ carbendazim	45.03	47.80	101.96	73.41	179.63	34.74
Bitertanol	0.58	0.69	0.02	0.36	9.88	4.79
Bromophos-ethyl	0.36	0.28	0.52	0.41	0.79	0.37
Carbaryl	127.44	195.37	129.77	142.55	594.15	382.34
Chlordane	0.61	0.45	0.89	0.71	1.28	0.61
Chlorothalonil	0.95	0.99	0.93	0.81	2.13	0.62
Chlorpyrifos	15.27	15.96	40.24	28.63	76.47	20.27
Chlorpyrifos-methyl	95.55	83.03	157.85	110.77	230.27	107.04
Cyfluthrin	0.82	0.69	1.10	1.01	0.97	0.47
Cyhalothrin	0.42	0.39	0.47	0.39	0.45	0.21
Cypermethrin	7.00	6.75	9.94	8.70	11.49	5.12
pp-DDE	2.96	2.66	6.50	3.48	10.05	4.76
Demeton-S-methyl	0.07	0.08	0.05	0.06	0.40	3.22
Dicofol	76.75	99.20	78.22	111.48	295.10	93.47

Continued

Table A5: Mean estimated daily dietary exposure to detected pesticide residues in ng/kg bw based on mean analytical results (19th ATDS) (continued)

Chemical	Adult males 25–34 years ng/kg bw/day	Adult females 25–34 years ng/kg bw/day	Boys 12 years ng/kg bw/day	Girls 12 years ng/kg bw/day	Toddlers 2 years ng/kg bw/day	Infants 9 months ng/kg bw/day
Dimethoate	0.87	0.94	0.86	0.69	1.59	0.75
Diphenylamine	122.68	143.44	349.99	252.22	806.33	193.15
Endosulfan	58.83	60.45	67.87	51.47	66.69	62.10
Fenitrothion	14.22	11.74	24.62	16.89	33.35	19.82
Fenoxycarb	1.23	1.39	2.01	1.80	9.06	8.03
Fenthion	17.09	18.09	17.99	15.70	29.08	13.51
Fenvalerate	2.31	2.19	2.59	2.12	2.45	1.17
Flumethrin	2.74	0.81	<0.01	<0.01	<0.01	<0.01
Heptachlor	1.34	1.38	1.18	1.45	1.90	0.90
Imazalil	23.66	26.90	46.72	48.49	185.90	97.79
Iprodione	101.04	134.55	148.83	128.04	436.87	204.00
Methamidophos	15.29	16.28	18.24	12.88	15.32	7.57
Mevinphos	0.53	0.57	0.64	0.45	0.54	0.26
Omethoate	1.26	1.18	1.40	1.16	1.34	0.64
Parathion-methyl	2.94	3.77	5.25	4.47	18.57	6.84
Permethrin	47.64	43.41	87.99	68.82	139.74	261.84
Piperonyl butoxide	123.30	94.95	188.68	131.84	230.69	98.03
Pirimicarb	6.51	5.65	4.81	4.65	13.15	4.88
Pirimiphos-methyl	10.17	10.83	13.86	9.15	17.10	7.48
Procymidone	59.61	73.04	66.43	57.94	149.52	72.35
Tetradifon	2.22	2.89	2.29	2.42	7.36	3.36
Vinclozolin	0.59	0.63	0.70	0.50	0.59	0.29

Table A6: Mean estimated daily dietary exposure to detected pesticide residues in ng/kg bw based on mean analytical results (18th ATDS)

Chemical	Adult males 25–34 years ng/kg bw/day	Adult females 25–34 years ng/kg bw/day	Boys 12 years ng/kg bw/day	Girls 12 years ng/kg bw/day	Toddlers 2 years ng/kg bw/day	Infants 9 months ng/kg bw/day
Azinphos-methyl	21.96	25.43	79.50	50.85	139.78	93.60
BHC total	0.10	0.11	0.09	0.05	0.18	0.12
Chlorothalonil	16.53	18.33	19.69	14.06	17.33	12.18
Chlorpropham	9.35	7.14	13.41	10.45	20.20	13.46
Chlorpyrifos	16.05	23.33	29.45	21.34	42.56	28.40
Chlorpyrifos-methyl	232.19	207.18	370.85	249.07	527.20	375.06
Cyfluthrin	0.18	0.19	0.20	0.16	0.20	0.14
Cypermethrin	0.99	1.10	1.79	1.43	2.53	1.72
DDT (total)	3.09	2.18	2.13	3.71	3.76	2.47
Deltamethrin	4.76	4.23	6.80	5.75	10.31	7.07
Diazinon	1.53	1.77	6.06	3.86	9.57	6.39
Dichlorvos	0.04	0.06	0.07	<0.01	<0.01	<0.01
Dicloran	0.21	0.26	0.27	0.33	0.46	0.33
Dicofol	36.82	48.36	46.83	63.01	143.35	95.19
Dieldrin	1.65	1.86	1.46	1.28	3.23	2.17
Dimethoate	40.29	44.62	56.38	41.76	99.45	68.28
Diphenylamine	246.98	285.74	904.57	577.76	1568.37	1050.05
Endosulfan	97.71	112.36	132.40	96.38	163.33	111.26
Ethion	0.56	0.66	0.69	0.71	1.23	0.85
Fenitrothion	140.72	129.02	187.35	139.79	310.72	221.76
Fenthion	0.17	0.20	0.67	0.43	1.06	0.72
Fenvalerate	1.95	1.90	1.87	1.78	2.27	1.55
Heptachlor epoxide	0.14	0.15	0.12	0.10	0.27	0.18
Iprodione	161.51	185.69	463.05	307.88	905.75	639.04
Lindane	<0.01	0.01	0.01	<0.01	<0.01	<0.01
Maldison	4.38	7.66	4.96	4.10	5.72	3.74
Methamidophos	22.91	26.60	23.63	18.43	37.61	25.41
Methidathion	0.06	0.11	0.10	0.19	0.49	0.32
Mevinphos	0.83	0.88	0.98	0.69	0.83	0.58

Continued

Table A6: Mean estimated daily dietary exposure to detected pesticide residues in ng/kg bw based on mean analytical results (18th ATDS) (continued)

Chemical	Adult males 25–34 years ng/kg bw/day	Adult females 25–34 years ng/kg bw/day	Boys 12 years ng/kg bw/day	Girls 12 years ng/kg bw/day	Toddlers 2 years ng/kg bw/day	Infants 9 months ng/kg bw/day
Monocrotophos	0.10	0.14	<0.01	0.06	<0.01	<0.01
Parathion	0.34	0.47	0.92	0.84	2.35	1.57
Parathion-methyl	0.83	0.98	1.03	0.69	6.03	4.07
Permethrin	17.89	26.66	21.18	16.24	22.31	14.96
Pirimiphos-methyl	24.48	20.73	52.28	25.09	63.64	42.30
Procymidone	69.89	80.09	101.17	57.26	101.65	70.46
Prothiophos	0.57	1.03	0.89	1.69	4.42	2.91
Tetradifon	0.20	0.23	0.77	0.47	1.13	0.76
Vinclozolin	38.57	47.15	97.79	63.26	166.81	111.80

Table A7: Mean estimated daily dietary exposure to pesticide residues as a percentage of the ADI based on mean analytical results (19th ATDS)

Chemical	Adult males 25–34 years %ADI	Adult females 25–34 years %ADI	Boys 12 years %ADI	Girls 12 years %ADI	Toddlers 2 years %ADI	Infants 9 months %ADI
Azinphos-methyl	0.93	1.13	3.04	2.12	6.32	1.61
Benomyl/carbendazim	0.15	0.16	0.34	0.24	0.60	0.58
Bitertanol	0.01	0.01	<0.01	<0.01	0.10	0.05
Bromophos-ethyl	0.01	0.01	0.01	0.01	0.02	0.01
Carbaryl	3.19	4.88	3.24	3.56	14.85	9.56
Chlordane	0.12	0.09	0.18	0.14	0.26	0.12
Chlorothalonil	0.01	0.01	0.01	0.01	0.02	0.01
Chlorpyrifos	0.51	0.53	1.34	0.95	2.55	0.68
Chlorpyrifos-methyl	0.96	0.83	1.58	1.11	2.30	1.07
Cyfluthrin	<0.01	<0.01	0.01	0.01	<0.01	<0.01
Cyhalothrin	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Cypermethrin	0.01	0.01	0.02	0.02	0.02	<0.01
PP-DDE	0.15	0.13	0.32	0.17	0.50	0.04
Demeton-S-methyl	0.02	0.03	0.02	0.02	0.13	0.03

Continued

Table A7: Mean estimated daily dietary exposure to pesticide residues as a percentage of the ADI based on mean analytical results (19th ATDS) (continued)

	Adult males 25–34 years	Adult females 25–34 years	Boys 12 years	Girls 12 years	Toddlers 2 years	Infants 9 months
Chemical	**%ADI**	**%ADI**	**%ADI**	**%ADI**	**%ADI**	**%ADI**
Dicofol	7.67	9.92	7.82	11.15	29.51	9.35
Dimethoate	<0.01	<0.01	<0.01	<0.01	0.01	<0.01
Diphenylamine	0.61	0.72	1.75	1.26	4.03	0.97
Endosulfan	0.98	1.01	1.13	0.86	1.11	1.04
Fenitrothion	0.71	0.59	1.23	0.84	1.67	0.99
Fenoxycarb	<0.01	<0.01	<0.01	<0.01	0.02	0.02
Fenthion	0.85	0.90	0.90	0.79	1.45	0.68
Fenvalerate	0.01	0.01	0.01	0.01	0.01	0.01
Flumethrin	0.05	0.02	<0.01	<0.01	<0.01	<0.01
Heptachlor	0.27	0.28	0.24	0.29	0.38	0.18
Imazalil	0.08	0.09	0.16	0.16	0.62	0.33
Iprodione	0.25	0.34	0.37	0.32	1.09	0.51
Methamidophos	2.55	2.71	3.04	2.15	2.55	1.26
Mevinphos	0.03	0.03	0.03	0.02	0.03	0.01
Omethoate	0.42	0.39	0.47	0.39	0.45	0.21
Parathion-methyl	1.47	1.89	2.62	2.24	9.28	3.42
Permethrin	0.10	0.09	0.18	0.14	0.28	0.52
Piperonyl butoxide	0.12	0.09	0.19	0.13	0.23	0.10
Pirimicarb	0.33	0.28	0.24	0.23	0.66	0.24
Pirimiphos-methyl	0.05	0.05	0.07	0.05	0.09	0.04
Procymidone	0.12	0.15	0.13	0.12	0.30	0.14
Tetradifon	0.01	0.01	0.01	0.01	0.04	0.02
Vinclozolin	0.01	0.01	0.01	<0.01	0.01	<0.01

Table A8: Mean estimated daily dietary exposure to pesticide residues as a percentage of the ADI based on mean analytical results (18th ATDS)

	Adult males 25–34 years	Adult females 25–34 years	Boys 12 years	Girls 12 years	Toddlers 2 years	Infants 9 months
Chemical	**%ADI**	**%ADI**	**%ADI**	**%ADI**	**%ADI**	**%ADI**
Azinphos-methyl	2.20	2.54	7.95	5.09	13.98	9.36
Chlorothalonil	0.17	0.18	0.20	0.14	0.17	0.12
Chlorpropham	0.02	0.01	0.03	0.02	0.04	0.03
Chlorpyrifos	0.53	0.78	0.98	0.71	1.42	0.95
Chlorpyrifos-methyl	2.32	2.07	3.71	2.49	5.27	3.75
Cyfluthrin	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Cypermethrin	<0.01	<0.01	<0.01	<0.01	0.01	<0.01
DDT (total)	0.15	0.11	0.11	0.19	0.19	0.12
Deltamethrin	0.05	0.04	0.07	0.06	0.10	0.07
Diazinon	0.15	0.18	0.61	0.39	0.96	0.64
Dichlorvos	0.01	0.01	0.01	<0.01	<0.01	<0.01
Dicloran	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Dicofol	3.68	4.84	4.68	6.30	14.33	9.52
Dieldrin	1.65	1.86	1.46	1.28	3.23	2.17
Dimethoate	0.20	0.22	0.28	0.21	0.50	0.34
Diphenylamine	1.23	1.43	4.52	2.89	7.84	5.25
Endosulfan	1.63	1.87	2.21	1.61	2.72	1.85
Ethion	0.06	0.07	0.07	0.07	0.12	0.09
Fenitrothion	7.04	6.45	9.37	6.99	15.54	11.09
Fenthion	0.01	0.01	0.03	0.02	0.05	0.04
Fenvalerate	0.01	0.01	0.01	0.01	0.01	0.01
Heptachlor epoxide	0.03	0.03	0.02	0.02	0.05	0.04
Iprodione	0.40	0.46	1.16	0.77	2.26	1.60
Lindane	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Maldison	0.02	0.04	0.02	0.02	0.03	0.02
Methamidophos	3.82	4.43	3.94	3.07	6.27	4.23
Methidathion	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
Mevinphos	0.04	0.04	0.05	0.03	0.04	0.03

Continued

Table A8: Mean estimated daily dietary exposure to pesticide residues as a percentage of the ADI based on mean analytical results (18th ATDS) (continued)

	Adult males 25–34 years	Adult females 25–34 years	Boys 12 years	Girls 12 years	Toddlers 2 years	Infants 9 months
Chemical	**%ADI**	**%ADI**	**%ADI**	**%ADI**	**%ADI**	**%ADI**
Monocrotophos	0.03	0.05	<0.01	0.02	<0.01	<0.01
Parathion	0.01	0.01	0.02	0.02	0.05	0.03
Parathion-methyl	0.41	0.49	0.52	0.34	3.02	2.03
Permethrin	0.04	0.05	0.04	0.03	0.04	0.03
Pirimiphos-methyl	0.12	0.10	0.26	0.13	0.32	0.21
Procymidone	0.14	0.16	0.20	0.11	0.20	0.14
Prothiophos	0.06	0.10	0.09	0.17	0.44	2.91
Tetradifon	<0.01	<0.01	<0.01	<0.01	0.01	<0.01
Vinclozolin	0.39	0.47	0.98	0.63	1.67	1.12

Appendix 3 Dietary exposure to thiram

Notes on the table:

1 1 µg = one millionth of 1 g.

2 ADIs for dithiocarbamates are in Table 7 in the supplementary information on ANZFA's website.

3 Estimated dietary exposures are based on food consumption data from the 1995 National Nutrition Survey.

Table A9: Mean estimated daily dietary exposure to thiram in µg/kg bw and as a percentage of ADI based on mean analytical results (19th ATDS)

Thiram intake	Adult males 25–34 years	Adult females 25–34 years	Boys 12 years	Girls 12 years	Toddlers 2 years	Infants 9 months
µg/kg bw/day	0.818	0.901	1.0698	1.0296	2.5342	1.1684
% ADI	20	23	27	26	63	29

Appendix 4 P

The dietary exposure for these substances has not been calculated, as the concentration of these substances in surveyed foods is less than the limit of reporting.

Carbamate

Methomyl

Fungicides

Bupirimate
Dicloran
Penconazole
Triadimenol
Captan/captafol decomposition products
Myclobutanil
Thiabendazole

Chlorinated organic pesticides

Aldrin
p,p'-DDD
Dieldrin
Heptachlor epoxide
Methoxychlor
Chlordane, cis
p,p'-DDT
Endrin
Hexachlorobenzene
Oxychlordane

Organophosphorus pesticides

Azinphos ethyl
Chlorfenvinphos
Dichlorvos
Ethion
Malathion/maldison
Monocrotophos
Phosmet
Vamidothion
Carbophenothion (trithion)
Diazinon
Dioxathion
Fenamiphos
Methidathion
Phosalone
Temephos

Synthetic pyrethroid

Bioresmethrin

References

Australia New Zealand Food Authority, 1999, *Contaminants in foods—metals, full assessment report*, Proposal P157, ANZFA, Canberra.

Cole, R. J., 1989, *Technology of aflatoxin decontamination*, United States Department of Agriculture, Agricultural Research Service, National Peanut Research Laboratory, Georgia, USA.

Commonwealth Department of Health and Aged Care, Therapeutic Goods Administration, 2000, *ADI list—acceptable daily intakes for agricultural and veterinary chemicals*, Commonwealth Department of Health and Aged Care, Canberra.

Friberg, L., Nordberg, G. F., and Vouk, V. B., (eds), 1979, *Handbook on the toxicology of metals*, Elsevier/North Holland Biomedical Press, Amsterdam.

Hitchcock, N.E., Gracey, M., Gilmour, A.I., Owler, E.N., 1986 'Nutrition and growth in infancy in infancy and early childhood: a longitudinal study from birth to five years', *Monographs in Paediatrics*, 19, pp 1–92.

National Health and Medical Research Council, 1991, *Recommended dietary intakes for use in Australia*, Australian Government Publishing Service, Canberra.

Institute of European Food Studies, 1998, *The effect of survey duration on the estimation of food chemical intakes*, Institute of European Food Studies, Dublin.

Reilly, C., 1980, *Metal contamination of food*, Applied Science Publishers Ltd, London.

United States Environmental Protection Agency, 1991, *Antimony—IRIS (Integrated Risk Information System)*. US Environmental Protection Agency, Washington.

World Health Organization, 1978, *Report of the 22nd meeting, Joint FAO/WHO Expert Committee on Food Additives*, Technical Report Series 631, Geneva.

—1981, *Environmental Health Criteria 18, Arsenic*, WHO, Geneva.

—1983, *Measuring Change in Nutritional Status*, WHO, Geneva.

—1987a, *Environmental Health Criteria 58, Selenium*, WHO, Geneva.

—1987b, *Toxicological evaluation of certain food additives and contaminants No. 21*, International Programme on Chemical Safety, WHO, Geneva.

—1987c, *Report of the 31st meeting, Joint FAO/WHO Expert Committee on Food Additives*, Technical Report Series 759, WHO, Geneva.

—1989, *Toxicological evaluation of certain food additives and contaminants No. 24*, International Programme on Chemical Safety, WHO, Geneva.

—1990a, *Report of the 35th meeting, Joint FAO/WHO Expert Committee on Food Additives*, Technical Report Series 789, WHO, Geneva.

—1990b, *Environmental health criteria 116, tributyltin compounds*, WHO, Geneva

—1996, *Trace elements in human nutrition*, WHO, Geneva.

—1997, *Guidelines for predicting dietary intake of pesticide residues (revised)*, WHO, Geneva.