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Australia New Zealand
Te Mana Kounga Kai – Ahitereiria me Aotearoa

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FINAL ASSESSMENT REPORT

APPLICATION A552

CADMIUM IN PEANUTS

For information on matters relating to this Assessment Report or the assessment process generally, please refer to: http://www.foodstandards.gov.au/standardsdevelopment/

Executive Summary

Food Standards Australia New Zealand (FSANZ) received an unpaid Application from the Confectionery Manufacturers of Australasia Limited (CMA) on 15 November 2004 to amend Standard 1.4.1 – Contaminants and Natural Toxicants of the *Australia New Zealand Food Standards Code* (the Code) with respect to the maximum permitted level of cadmium in peanuts. The CMA originally sought an amendment to the Table to clause 2 of Standard 1.4.1 to remove the existing Maximum Level (ML) of 0.1 mg/kg for cadmium in peanuts. However, the CMA amended the original request and advised FSANZ that it would now prefer to increase the ML from 0.1 to 0.5 mg/kg. The CMA claims that this will enable increased flexibility to source peanuts from a variety of countries to meet changes in supply that may result from crop seasonality and product quality.

Following a review of cadmium levels in food in 1997, the then Australia New Zealand Food Authority (ANZFA) recommended a revised ML for cadmium in peanuts of 0.1 mg/kg on the basis that it would not increase the risk to public health and safety and was less likely to be perceived as a non-tariff barrier to trade (than the previous ML of 0.05 mg/kg). Previous policy principles agreed to by the then Australia New Zealand Food Standards Council in July 1997 indicate that contaminant levels in food should be safe and according to the ALARA principle (**a**s **l**ow **a**s **r**easonably **a**chievable) which is consistent with the Codex Alimentarius approach to contaminants.

In addition, MLs shall be set: (1) only for those contaminants that present both a significant risk to public health and a known or expected problem in international trade; (2) for those foods that are significant for the total exposure of the consumer to the contaminant; and (3) as low as reasonably achievable. Providing it is acceptable from the toxicological point of view, MLs shall be set at a level, which is (slightly) higher than the normal range of variation in levels in foods that are produced with current adequate technological methods, in order to avoid undue disruptions of food production and trade. Therefore, the proposed increased ML of 0.5 mg/kg for cadmium satisfies the above criteria.

In setting an ML, consideration should also be given to Australia and New Zealand's international trade obligations under the World Trade Organization's Sanitary and Phytosanitary (SPS) Agreement and Technical Barriers to Trade (TBT) Agreement.

The risk assessment has concluded that increasing the ML to 0.5 mg/kg will not be a public health and safety issue. The proposed ML is based on the upper range of distribution of cadmium levels in peanuts from the available survey data. Dietary exposure to cadmium in Australia and New Zealand is well below the provisional tolerable weekly intake (PTWI) of 7 μg/kg bw/week even for the highest consumer group (children aged 2-6 years) and is therefore within acceptable safety standards. Peanuts are a minor contributor to overall exposure to cadmium in both Australia and New Zealand. It is recognised that there is a high prevalence of renal disease among Aboriginal and Torres Strait Islander people and concerns have been raised about cadmium exacerbating renal disease. However, the PTWI is also adequately protective of high-risk groups such as Aboriginals and Torres Strait Islanders.

The increase will enable the current shortfall between domestic production and imported peanuts, claimed by the CMA as significant, to be met by imports from a variety of countries, in order to assist specific industry sectors when supplies are short (e.g. during times of drought).

FSANZ received a number of submissions at Draft Assessment that questioned whether or not the current ML is actually a trade barrier to importation of peanuts from other countries. In addition, many of these submissions argued that further justification was needed to quantify the costs to industry if the ML is not increased to 0.5 mg/kg. The CMA has provided further justification to support the increased ML, detailed in Section 7 of the Final Assessment Report.

FSANZ acknowledges that there may be potential costs to Australian industry if the ML is increased. FSANZ also notes that the peanut industry has worked very closely with the Queensland Department of Primary Industries and Fisheries (DPI&F), Commonwealth Scientific Industrial and Research Organisation (CSIRO) and the Grains Research and Development Corporation (GRDC) to develop management guidelines for farmers to ensure Australia's peanut production always meets the current ML for cadmium in peanuts. This considerable investment has delivered safe, high-quality and low cadmium-containing peanuts.

A potential cost that specific Australian industry sectors cited at Draft Assessment was that Australian peanut farmers may choose to use cheaper sources of fertiliser. This would reduce their costs while maintaining high production yields as there would no longer be the requirement to meet the stringent ML of 0.1 mg/kg. It was suggested that these practices may lead to a gradual accumulation of cadmium in the soil and also affect other industries (e.g. fisheries) as a result of agricultural run-off downstream, leading to bioaccumulation in marine life such as prawns, crabs, mussels and other shellfish. However, advice from the Fertilizer Industry Federation of Australia, Inc. (FIFA) to FSANZ is that this is unlikely to occur in practice as the industry has phased out the use of high-cadmium fertilisers under the National Cadmium Minimisation Strategy.

FSANZ considers that issues on fertiliser use are outside of FSANZ's mandate under the *Food Standards Australia New Zealand Act 1991* (FSANZ Act).

Purpose

The Applicant seeks to increase the current Maximum Level (ML) for cadmium in peanuts from 0.1 to 0.5 mg/kg.

Decision

FSANZ has made an assessment and recommends approving the proposed draft variation to Standard 1.4.1 to increase the existing ML for cadmium in peanuts from 0.1 to 0.5 mg/kg.

Reasons for Decision

- the proposed draft variation to the Code is consistent with the section 18 objectives of the FSANZ Act, in particular, it does not raise any public health and safety concerns, it is based on risk analysis using the best available scientific evidence, and helps promote an efficient and internationally competitive food industry;

- FSANZ has conducted an assessment of the safety of cadmium which concludes that the PTWI of 7 μg/kg body weight is adequately protective of consumers. The dietary exposure assessment shows that increasing the current ML from 0.1 to 0.5 mg/kg does not affect public health and safety; and

- the regulatory impact statement concludes that the benefits of the proposed regulatory option outweigh the costs. Adoption of this option will enable the shortfall between domestic peanut production and industry demand to be met by imports, while promoting good agriculture practice, the protection of public health and safety and facilitating trade in peanuts by enabling peanut importers to source peanuts from a wider number of countries.

Consultation

FSANZ has now completed the assessment of Application A552 and held two rounds of public consultation. The draft variations to the Code have been approved by the FSANZ Board and the decision notified to the Australia and New Zealand Food Regulation Ministerial Council (Ministerial Council).

If the Ministerial Council does not request FSANZ review the draft variations to the Code, an amendment to the Code will be published in the *Commonwealth Gazette* and the *New Zealand Gazette* and adopted by reference and without amendment under Australian State and Territory food law.

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INTRODUCTION

FSANZ received an unpaid Application from the Confectionery Manufacturers of Australasia Limited (CMA) on 15 November 2004 to amend Standard 1.4.1 – Contaminants and Natural Toxicants of the *Australia New Zealand Food Standards Code* (the Code) with respect to the maximum permitted level of cadmium in peanuts. The CMA originally sought an amendment to the Table to clause 2 of Standard 1.4.1 to remove the existing Maximum Level (ML) of 0.1 mg/kg for cadmium in peanuts. However, the CMA recently advised FSANZ that it would now prefer to increase the ML from 0.1 to 0.5 mg/kg. The CMA claims that this will enable increased flexibility to source peanuts from a variety of countries to meet changes in supply that may result from crop seasonality and product quality.

The membership of the CMA is in excess of 230 members, including manufacturers of sugar, chocolate and gum confectionery and ranges from multinational organisations such as Cadbury Schweppes, Nestlé Confectionery, MasterFoods Australia New Zealand and the Wrigley Company, through to the small to medium enterprises, niche operators and Australian-based family businesses (e.g. Haigh's and Darrell Lea). Membership also includes suppliers of ingredients, machinery, packaging materials and services to the industry; and wholesaler/distributor firms on both sides of the Tasman. The Peanut Council of Australia (PCA) is an Australian supplier of peanuts and a member of the CMA.

1. Background

1.1 Cadmium

Cadmium is a naturally-occurring heavy metal that is found at low levels throughout the environment. Food represents the major source of cadmium exposure rather than air or water and can be present in food as a result of agricultural practices. Long-term exposure to cadmium may lead to accumulation in the liver and kidneys, particularly the renal cortex, resulting in kidney damage.

1.2 Current Standard

Standard 1.4.1 sets out the MLs of specified metal and non-metal contaminants and natural toxicants in nominated foods. As a general principle, regardless of whether or not a ML exists, the levels of contaminants and natural toxicants in all foods should be kept **a**s **l**ow **a**s **r**easonably **a**chievable (ALARA)[1].

An ML is generally only established where it serves an effective risk management function and only for those foods which provide a significant contribution to the total dietary exposure. MLs are generally not assigned to foods that present a low public health risk.

[1] MLs shall be set: (1) only for those contaminants that present both a significant risk to public health and a known or expected problem in international trade; (2) for those foods that are significant for the total exposure of the consumer to the contaminant; and (3) as low as reasonably achievable. Providing it is acceptable from the toxicological point of view, MLs shall be set at a level, which is (slightly) higher than the normal range of variation in levels in foods that are produced with current adequate technological methods, in order to avoid undue disruptions of food production and trade.

MLs in the Code have been set at levels that are consistent with public health and safety and which are reasonably achievable from sound production and natural resource management practices. Consideration is given to Australia's and New Zealand's international trade obligations under the World Trade Organization's Sanitary and Phytosanitary Agreement and Technical Barrier to Trade Agreement.

The ML for cadmium in peanuts specified in the Table to clause 2 of Standard 1.4.1 is 0.1 mg/kg.

1.2.1 Definition of maximum level

Maximum Level is defined in clause 1 of Standard 1.4.1 as follows:

> ***maximum level (ML)*** *means the maximum level of a specified contaminant, or specified natural toxicant, which is permitted to be present in a nominated food expressed, unless otherwise specified, in milligrams of the contaminant or the natural toxicant per kilogram of the food (mg/kg).*

1.3 Historical Background

1.3.1 General Principles for Regulation of Contaminants in Foods

The regulation of contaminants and natural toxicants in food was comprehensively examined and discussed with the jurisdictions during the review of the former Australian *Food Standards Code* and the New Zealand *Food Regulations 1984*, culminating in the development of Standard 1.4.1. Prior to the development of Proposals to review metal and non-metal contaminants, a policy paper was prepared to obtain general agreement from all jurisdictions regarding the basis and direction of the review. This paper contained policy principles that were agreed to by the Australian and New Zealand Health Ministers then sitting as the Australia New Zealand Food Standards Council in July 1997. The principles outlined are consistent with the Codex Alimentarius approach to contaminants (the ALARA principle).

In practice, MLs are set at a level that is (slightly) higher than the normal range of variation in foods in order to avoid disruption of food production and trade. However, MLs are not safety limits and any assessment of the public health risk associated with exposure to a contaminant needs to consider total dietary exposure rather than exposure from a single food.

During the review of Metal Contaminants in the food supply under Proposal P157, FSANZ proposed MLs for a range of contaminants where the foods containing those contaminants made a major (>5%) contribution to total dietary exposure of that contaminant.

1.3.2 Review of the Maximum Permitted Concentrations of Cadmium in Food

The then Australia New Zealand Food Authority (ANZFA), now FSANZ, prepared Proposal P144 on 24 July 1996 to review the maximum permitted concentrations (MPCs, now referred to as MLs) of cadmium in all foods. The MPCs that were previously in place in Australia for cadmium were established in 1980 by the National Health and Medical Research Council (NHMRC) on the basis of the limited information available at that time.

There were MPCs for a number of food commodities and some food products, including a category referred to as 'foods not containing a food otherwise specified' with an MPC of 0.05 mg/kg which encapsulated cadmium in peanuts. In New Zealand, there was a single MPC of 1 mg/kg for cadmium in all foods, except shellfish.

ANZFA recommended a revised MPC for cadmium in peanuts of 0.1 mg/kg on the basis that it would not increase the risk in relation to public health and safety and was less likely to be perceived as a non-tariff barrier to trade (than the previous MPC of 0.05 mg/kg). A dietary exposure assessment for cadmium in foods derived from the 1995 National Nutrition Survey did not change conclusions reached on the basis of the 1983 data that peanuts were a low contributor to overall cadmium exposure. The MPC for cadmium in peanuts of 0.1 mg/kg was gazetted in 1999.

2. The Issue / Problem

Confectionery manufacturers are seeking an increase to the ML for cadmium in peanuts of 0.1 mg/kg to enable increased flexibility to source peanuts from a variety of countries to manage changes in supply that may result from crop seasonality and product quality. Peanuts are used extensively in a range of confectionery items.

The Applicant states that, at present, confectionery manufacturers are severely restricted in their choice of countries from which they can source peanuts, because some may exceed the current ML for cadmium in peanuts. Some peanut growing areas in Australia are also at increased risk of higher uptake of cadmium from the soil, in particular, sandy coastal areas with light sandy soils that are acidic and have a long history of phosphate fertiliser use.

There are a number of approaches that growers can take to minimise cadmium uptake by peanut crops such as adequate irrigation, applying lime to the soil to raise the pH, and use of low-cadmium sources of fertiliser. These types of approaches have been endorsed by the Australian Government National Cadmium Minimisation Strategy.

2.1 International regulation

Codex has not established a ML for cadmium in peanuts. At the 36th session of the Codex Committee on Food Additives and Contaminants (CCFAC) in March 2004, it was agreed to discontinue the development of a range of proposed MLs for cadmium in commodities, including peanuts, where these foods do not contribute to overall global exposure. Most countries have not established an ML for cadmium in peanuts.

The Applicant asserts that the ML for cadmium in peanuts could be increased without compromising public health and safety on the basis of assessments undertaken by the Joint FAO/WHO Expert Committee on Food Additives (JECFA), and retaining the ML for cadmium in peanuts in the Code could be perceived as a Technical Barrier to Trade.

3. Objectives

In developing or varying a food standard, FSANZ is required by its legislation to meet three primary objectives which are set out in section 18 of the FSANZ Act.

These are:

- the protection of public health and safety;
- the provision of adequate information relating to food to enable consumers to make informed choices; and
- the prevention of misleading or deceptive conduct.

In developing and varying standards, FSANZ must also have regard to:

- the need for standards to be based on risk analysis using the best available scientific evidence;
- the promotion of consistency between domestic and international food standards;
- the desirability of an efficient and internationally competitive food industry;
- the promotion of fair trading in food; and
- any written policy guidelines formulated by the Ministerial Council.

The specific objective of this Application is to determine whether an increase to the ML for cadmium in peanuts is appropriate on public health and safety grounds, and also taking into consideration:

- the level of cadmium in peanuts that would be reasonably achievable from sound production and natural resource management practices; and
- Australia's and New Zealand's international trade obligations under the World Trade Organization's Sanitary and Phytosanitary and Technical Barriers to Trade Agreements.

4. Key Assessment Questions

These are the key assessment questions that FSANZ considered at Draft Assessment:

- Would increasing the ML for cadmium in peanuts pose any risk to public health and safety?
- What would be the potential dietary intake of cadmium for mean and high consumers of peanuts and peanut containing products should the ML be increased?

RISK ASSESSMENT

5. Risk Assessment Summary

5.1 Hazard identification and characterisation

Cadmium is a heavy metal that is found as an environmental contaminant, both through natural occurrence and from industrial and agricultural sources. Cadmium is present in food primarily as a result of its uptake from soil and water.

Cadmium absorbed from food accumulates in the kidney and liver of mammals, including humans, where over time it can cause kidney dysfunction. Perhaps the best-known example of the effects of cadmium contamination of food is the prevalence of Itai Itai disease, characterised by kidney tubular dysfunction, severe bone pain and numerous bone fractures, predominantly in women, in the Jinzu River basin of Japan following contamination of water sources from an upstream mine[2].

The majority of cadmium in food passes through the gastrointestinal tract unabsorbed, with the level of absorption being influenced both by nutritional status (e.g. iron status) and bioavailability (e.g. associated with phytic acid). Absorbed cadmium is redistributed to the liver and kidneys where it is normally sequestered by small proteins called metallothioneins. Toxicity of cadmium is thought to occur when the binding capacity of metallothionein is exceeded.

Cadmium has a long biological half-life, estimated at between 16-33 years. Even low exposure levels may, in time, cause considerable accumulation, especially in the kidneys. The kidney is the critical target organ for toxic effects of long-term dietary exposure to cadmium. Toxicity is manifested only after many years of slow accumulation of cadmium in the renal cortex and then only if a critical concentration is achieved. The chronic effects on the kidney are characterised by tubular dysfunction and cell damage, most often manifested as urinary excretion of low molecular mass proteins. Urinary levels of two proteins in particular, protein HC (also know as α_1-microglobulin) and β2-microglobulin, are commonly used as biomarkers of effect on renal tubule function. A small fraction of cadmium in the kidneys is excreted in the urine, and the level of cadmium in the urine normally correlates with total body burden. However, if damage to the kidney proximal tubules has occurred, cadmium levels in the urine are paradoxically lower.

Numerous animal studies have confirmed the toxicity of cadmium to the kidney and liver. The majority of human data comes from epidemiological studies, from both industrial and environmental exposure to cadmium, including studies of populations exposed to low levels of cadmium through normal dietary exposure. Data from autopsy and epidemiological studies have been used to estimate a critical cadmium level in renal cortex at which signs of renal dysfunction may be present, and levels of lifetime cumulative intake of cadmium that would produce that critical level. The Joint FAO/WHO Expert Committee on Food Additives (JECFA) estimates the critical cadmium level in renal cortex is 200 mg/kg wet weight, resulting from the cumulative intake of about 2000 mg cadmium.

JECFA considered that levels of cadmium in the kidney should not exceed 50 mg/kg, and established a Provisional Tolerable Weekly Intake (PTWI) of 7 μg/kg bw based on an absorption rate of 5% and a daily excretion of 0.005% of body load. This PTWI, established in 1972, has been confirmed by subsequent considerations by JECFA, in 1988, 1993, 2000 and 2003. JECFA has acknowledged that there is only a relatively small safety margin between exposure in the normal diet and exposure that produces deleterious effect. In 2003, JECFA reaffirmed that 'no excess prevalence of renal tubular dysfunction would be predicted to occur at the current PTWI under the most appropriate assumptions about the fractional bioavailability of cadmium and the percentage of the absorbed cadmium that is excreted in urine.'

[2] WHO. (1992) Cadmium (Environmental Health Criteria 134). World Health Organisation, Geneva. http://www.inchem.org/documents/ehc/ehc/ehc134.htm.

The JECFA PTWI remains the internationally-accepted value for the safe level of intake for cadmium over a lifetime of exposure.

The PTWI is based on the most sensitive parameter for kidney damage, namely, an increase in the urinary excretion of low molecular weight protein as a result of reduced re-absorption in the renal tubules. The toxicological significance of this observed change with respect to kidney damage is still not established as it is clear that the excretion of low molecular weight proteins normally increases with age.

The other important factor to consider when interpreting the potential risk of intake levels near or above the PTWI for cadmium is that it has been established on the basis of cumulative intake. Thus, intake above this figure may be tolerated provided it is not sustained for long periods of time and does not produce a significant increase in the integrated life-time dose.

The safety assessment focuses on the risks associated with cadmium in food, and is primarily based on epidemiological data, particularly those conducted on populations that are likely to have a similar diet and nutritional status to Australians and New Zealanders. In particular, epidemiological studies in Belgium and Sweden suggest that even normal dietary exposure to cadmium can result in changes in kidney function, as evidenced by increased urinary excretion of low molecular mass proteins. However, increased excretion of biomarkers of effect does not necessarily indicate an adverse effect on kidney function and the long-term significance of these changes on human health is unclear. Also, several of these studies suggest that cadmium exposure explains only a small proportion of overall kidney effects, and that age is the major component affecting renal tubular function in populations chronically exposed to low levels of cadmium in the diet (**Attachment 2**).

5.2 Dietary exposure assessment

A dietary exposure assessment was deemed necessary for Application A552 in order to estimate the potential dietary exposure to cadmium from peanuts and the overall diet for both the Australian and New Zealand populations should the current ML of 0.1 mg/kg be increased to 0.5 mg/kg as requested by the Applicant (**Attachment 3**).

Dietary exposures to cadmium were calculated for the Australian and New Zealand populations and for the population sub-group Australian children 2-6 years. Scenarios considered for this Application were a *Baseline* dietary exposure scenario and a *Proposed Maximum Level (ML)* scenario. Both scenarios included foods representative of the overall diet as well as peanuts at a baseline level (0.04 mg/kg) and at a proposed ML of 0.5 mg/kg. Dietary exposure from *Peanuts Only* was also estimated to determine the specified population groups' dietary exposure to cadmium from peanuts.

The food consumption data used were from the 1995 Australian National Nutrition Survey (NNS) and the 1997 New Zealand NNS. The cadmium concentration data were from the Revised Dietary Exposure Assessment for Cadmium conducted in 2001 for both Australian and New Zealand populations (Food Standards Australia New Zealand, Unpublished). This revised assessment was conducted following the conclusion of Proposal P144 - Review of the Maximum Permitted Concentrations of Cadmium in Food that had based its risk assessment on 1983 and 1985 National Nutrition survey (NNS) data.

In 2001, the 1995 Australian and 1997 New Zealand NNS data became available and were incorporated into the FSANZ dietary exposure assessment computer program, DIAMOND (DIetAry Modelling Of Nutritional Data), forming the basis of the revised 2001 assessment.

The estimated dietary exposures to cadmium were found to be below the reference health standard, the Provisional Tolerable Weekly Intake (PTWI) of 0.007 mg/kg bw/week, for both the Australian and New Zealand general population groups for all scenarios assessed. A cadmium concentration of peanuts at the proposed ML of 0.5 mg/kg did not have a large impact on the estimated dietary exposures to cadmium for any of the population groups assessed. Estimated dietary exposures as a percent of the PTWI were slightly higher when based on the proposed ML by between 5 to 15% of the PTWI across all population groups assessed. Estimated dietary exposures were highest for children aged 2-6 years where estimated exposures at baseline were 50-60% of the PTWI at the 90th percentile of exposure and 65-75% of the PTWI at the 90th percentile exposure when based on the proposed ML of 0.5 mg/kg.

Major food contributors to total cadmium dietary exposure for *Baseline* included potato for all Australians as well as cacao beans (chocolate) for those 2-6 years, and potato and mussels for New Zealand. These foods, together with peanuts, were also major contributors to total cadmium dietary exposure for the respective population groups for the *Proposed ML* scenario.

Under the FSANZ Science Strategy 2006-2009, FSANZ agreed to review its dietary modelling procedures. As part of this review an international peer review was sought. FSANZ has previously reported chronic dietary exposures for high consumers at the 95th percentile. The recommendation of the peer review by an international dietary exposure assessment expert from the US Food and Drug Administration was that FSANZ should consider aligning its reporting of food chemical dietary exposures with international best practice by reporting at the 90th percentile not the 95th percentile, if only one 24 hour recall record per person was used for the assessment[3]. This is because the 95th percentile results are likely to be an overestimate of dietary exposure on a daily basis over a life-time of exposure. Basing risk management decisions on the 95th percentile will potentially result in an overly conservative risk management approach.

5.3 Risk Characterisation

Tubular dysfunction in the kidneys is the critical effect resulting from chronic exposure to cadmium. The PTWI for cadmium is currently set at 7 μg/kg bw. The PTWI is based on lifetime accumulation of cadmium and is considered to be protective of kidney function following exposure to cadmium.

The dietary exposure assessment indicates that peanuts are currently a minor contributor to overall dietary exposure to cadmium, even for high consumers.

The dietary exposure calculations for whole population groups are reflective of life-time exposure to cadmium in the diet and are consistent with the current PTWI which is based on life-time accumulation of cadmium in the kidneys.

[3] Lambe, J., Kearney, J., Leclercrq, C., Berardi, D., Zunft,H., De Henauw, S., De Volder, M., Lamberg-Allardt, C., Karkkainen, M., Dunne, A. and Gibney, N. (2000) Enhancing the capacity of food consumption surveys of short duration to estimate long term consumer-only intakes by combination with a qualitative food frequency questionnaire. Food Additives and Contaminants, 17(3), pp. 177-187.

Modelling of total dietary exposure to cadmium from all dietary sources, including an increase in the ML for cadmium in peanuts to 0.5 mg/kg, indicated that Australian and New Zealand consumers would have dietary intakes of cadmium within safe limits. The highest consumers (90th percentile) had intakes of 30-35% of the PTWI for the whole Australian population aged 2 years+ and for the New Zealand population aged 15 years+.

Estimated dietary exposures to cadmium from all sources with peanuts assumed to contain 0.5 mg/kg, as a percentage of the PTWI

Country	Population group	Number of consumers of cadmium	Consumers♦ as a % of total respondents#	Mean all respondents	90th percentile consumers
				% PTWI*	
Australia	2 years+	13854/13856	100	15 - 20	30 - 35
	2-6 years	989/989	100	30 - 40	65-75
New Zealand	15 years+	4632/4635	100	15 - 20	30 - 35

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.
♦ Consumers only – This only includes the people who have consumed a food that contains cadmium.
* PTWI = 0.007 mg/kg bw

The exposure assessment indicated that the highest consumption estimated for Australian children aged 2-6 years was at 65-75% of the PTWI. Dietary modelling often identifies children as the sub-population most likely to have the highest exposure to various food components, partly because young children have relatively high food consumption levels when expressed on a body weight basis. At its 33rd meeting, JECFA recognised that exposure to cadmium will not be uniform with age, and that the estimate of the PTWI takes into account the higher cadmium intake on a body weight basis by infants and children[4]. Higher intakes during childhood are unlikely to be maintained over the long-term. The exposure estimates for the whole population include data from all age groups including 2-6 year olds and are a more accurate estimate of lifetime exposure to cadmium.

The exposure estimates for all population groups are likely to be overestimates because the estimate assumes that all peanuts are contaminated with cadmium at 0.5 mg/kg which is extremely unlikely as the available data on cadmium levels in peanuts indicates that most peanuts will have cadmium concentrations well below this level. Also, the data used for modelling is a 24-h record which overestimates food consumption for consumers as they are unlikely to consume the same foods in the same quantities every day (the use of multiple day records tends to significantly reduce predicted high consumer exposure).

In conclusion, because all population groups have estimated dietary intakes well below the PTWI, and due to the conservative assumptions in the dietary exposure calculations, increasing the current ML for cadmium in peanuts from 0.1 to 0.5 mg/kg would not raise public health and safety concerns.

[4] WHO. (1989) Cadmium. In: *Toxicological evaluation of certain food additives and contaminants. Thirty-third meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 24

RISK MANAGEMENT

6. Options

FSANZ is required to consider the impact of various regulatory (and non-regulatory) options on all sectors of the community, which includes consumers, food industries and governments in Australia and New Zealand. The benefits and costs associated with the proposed amendment to the Code will be analysed using regulatory impact principles.

Three regulatory options have been identified for this Application:

Option 1 – Reject the Application, thus retaining the ML for cadmium in peanuts of 0.1 mg/kg (*status quo*).

Option 2 – Harmonise with Codex and remove the ML for cadmium in peanuts.

Option 3 – Establish a higher ML for cadmium in peanuts.

7 Impact Analysis

7.1 Affected Parties

Parties possibly affected by the regulatory options outlined in Section 6 include:

1. Consumers of peanuts and products containing peanuts.

2. Those sectors of the food industry wishing to market peanuts or processed foods containing peanuts.

3. Importers of peanuts and processed foods containing peanuts.

4. Peanut growers of Australia in Queensland, the Northern Territory and northern New South Wales.

5. Government agencies enforcing the food regulations.

7.2 Benefit Cost Analysis

7.2.1 Background

Peanuts and peanut products are most likely to be imported under Tariff Chapters 8 (Edible Fruit and Nuts; Peel of Citrus Fruit and Melons) and 20 (Preparations of Vegetables, Fruit, Nuts or Other Parts of Plants).

In Australia, all imported peanuts (and food containing greater than 30% peanuts) are initially referred to the Australian Quarantine and Inspection Service (AQIS) by Customs because peanuts are tested in the risk category for aflatoxin under the Imported Food Inspection Scheme (IFIS).

The major countries (contributing greater than 10,000 kg) from which peanuts were imported into Australia in 2004 are shown in **Table 1**. China was clearly the largest exporter of peanuts to Australia with a total of approximately 2,789,611 kg in 2004.

Table 1: Major peanut exporters[5] to Australia in 2004

Country	Quantity (KG)
China	2,789,611
New Zealand	405,152
India	318,353
Thailand	171,441
Indonesia	149,000
UK	134,435
Vietnam	84,367
Malaysia	47,844
Iran	37,225
Philippines	35,701
Hong Kong	14,676
Germany	13,612
Turkey	10,154

Source: Australian Quarantine and Inspection Service data

Other countries contributing peanut imports to Australia in 2004 (up to 2000 kg amounts) included the Netherlands, Denmark, Taiwan, USA, Singapore and Korea.

More recently, major exporters to Australia for the 12-month period to June 2006 and 2007 have been the following countries, with the majority sourced from Argentina followed by China (**Table 2**).

Table 2: Major peanut exporters to Australia in 2006 and 2007

Country	Quantity (KG)	Quantity (KG)
Year	**2006**	**2007**
Argentina	20,660	11,539,858
Brazil	0	138,600
China	2,139,896	7,626,036
Vietnam	127,681	119,816
Thailand	96,523	116,898
Indonesia	81,565	142,763
Malaysia	<10,000 (7,653)	18,036
Singapore	37,887	40,644
Nicaragua	22,000	2,976,810
Hong Kong	16,314	<10,000 (360)
India	15,424	15, 915
New Zealand	14,362	<10,000 (7,777)
Switzerland	12,000	0
USA	<10,000 (647)	521,668

Source: Data from the Peanut Company of Australia (PCA) submitted during the public consultation period obtained from the Australian Bureau of Statistics Report ID: 00786

[5] These estimates also include the situation where another country is a through port for import from the originating country to Australia as the end destination. For example, the Applicant has informed us that New Zealand do not have a domestic peanut growing industry to any extent.

New Zealand does not have a domestic peanut growing industry to any extent, but rather, relies on imports. The import data (**Table 3**) indicates that over the past two years (2005 to 2006) New Zealand has imported peanuts from Australia, China, Fiji, Hong Kong, India, Nicaragua, Singapore and Samoa.

Table 3: Major peanut exporters to New Zealand for 2005 to 2006.

Country	Quantity (KG)
Australia	1,731,814
China	651, 874
Fiji	9, 819
Hong Kong	10, 396
India	751
Nicaragua	300
Singapore	850
Samoa	10

Source: New Zealand Food Safety Authority

To further develop the analysis of the costs and benefits of the regulatory options proposed, FSANZ sought comment at Initial Assessment on what the potential costs or benefits of this Application were for the possibly affected parties in Section 7.1 above.

The following is a summary of the information received and the key issues raised from the public submissions relating to the merits of the Application and the cost and benefits to specific industry sectors.

7.2.2 Current ML as a trade barrier

At Initial Assessment, specific submitters questioned whether the current ML is actually a trade barrier. FSANZ requested additional data from the Applicant in regard to past instances where the CMA has experienced problems in sourcing peanuts and further clarification and evidence to support its arguments that the current ML of 0.1 mg/kg is in effect causing a trade barrier. Other submitters supplied data that FSANZ found useful to understand the broader issues in relation to imports of peanuts.

7.2.2.1 Evaluation

The Australian Peanut Industry

Australia's and New Zealand's combined annual domestic consumption of peanut kernels is approximately 40,000 tonnes (of edible kernels). Australia's average production of edible peanuts for the last five years is estimated at 36,894 tonnes while consumption has been 32,000 tonnes of edible kernels. There is an annual background importation of 5000 to 7000 tonnes of peanuts/year.

Australia produces approximately 0.2% of the world's peanut production. Approximately 80% of the peanut growers are located in Queensland with major growing areas in the Kingaroy/Burnett region, southern and central Queensland, the Atherton Tableland, Katherine (Northern Territory) and Wee Waa (NSW). The majority of the crop is bought and primary-processed by the Peanut Company of Australia (PCA).

The PCA and the Queensland Department of Primary Industries and Fisheries maintain that Australia can produce peanuts that meet the current cadmium ML of 0.1 mg/kg via appropriate agricultural practices and other quality controls (including appropriate testing, with failures leading to price penalties or complete rejection of product). In addition, in their view, there is no restriction on the import of peanuts into Australia if appropriate quality testing is undertaken at the time of purchase (or dispatch) in the exporting country.

Data submitted by industry in the public comment period indicated that in 1996, imports of peanuts were larger (15,000 tonnes) and in addition during the periods 2002-03 and 2003-04 (approximately 27,000 and 16,000 tonnes, respectively).

In the period from July 2006 to March 2007, the PCA will have imported in excess of 10,000 tonnes of peanuts (due to drought in February/March 2006) which they claim have met the current cadmium ML of 0.1 mg/kg.

Table 4 provides an overview of import statistics on amounts of peanuts that were imported across all peanut users from 2002 to 2006. These data also suggest that for the 2002-03 and 2006 periods, an increase in imports occurred to alleviate supply issues to manufacturers that use peanuts in confectionary products.

Table 4: Imports under tariff code 1202.20.00.03 – Peanuts, not roasted or otherwise cooked, shelled from 2002-2006 (includes blanched and non-blanched)

	2002	**2003**	**2004**	**2005**	**2006**
Argentina	15634	11911	413	1293	7145
China	6656	7523	4536	4670	6196
India	0	0	0	0	0
Nicaragua		806	1577	306	216
USA	0	0	0	0	394
TOTAL	**22290**	**20240**	**6526**	**6269**	**13951**

Source: CMA data submitted during the public consultation period obtained from the Australian Bureau of Statistics (tonnes).

Therefore, in most years apart from severe droughts (1996, 2003/2004 and 2006) Australian peanut production was able to meet domestic consumption requirements. Changing agronomy practices from dryland areas (reliant on annual rainfall) to irrigated areas has increased yields and offset the decline in dryland production.

CMA's View

The CMA has maintained that increasing the ML will ensure greater continuity of the peanut supplies where on occasion the current ML has restricted imports. In particular, it would allow greater sourcing of peanuts from such countries as the USA, China, Argentina, Nicaragua and possibly South Africa and India. However, the sourcing from various countries depends on the variety required as some peanuts may not meet the specific product specifications of confectionery manufacturers. Unnecessary limitations in being able to source from a variety of countries may result in costly disruptions to manufacturers' supply chains and potential loss of customers.

Peanut varieties are selected for incorporation into confectionary based on a range of variables (flavour, shape and size) and require a greater flexibility in the sourcing of peanuts to improve reliability for importation. Based on the global variability in cadmium levels, a need for flexibility to respond to the drought and volatility in weather conditions, and a need to look at broader origins for continuity of supply, the CMA recommends that an increase to the ML would provide reasonable flexibility, without unnecessarily restricting import sourcing arrangements, while minimizing the threat of sustainability of supply.

The CMA argues that the current cadmium ML for peanuts presents a trade barrier to manufacturers for the following reasons:

- it is unable to compete against world peanut prices; thereby impacting on the price of finished confectionery products and the ability to remain competitive, both in the domestic market against imports and globally with exports;

- the restrictions placed on local manufacturers by way of the current cadmium ML means that flexibility in sourcing is prevented which has consequences for supply chain interruptions. Failure to source peanuts when required leads to production down time and consequently interrupted supply of finished product. An interruption to supply has many and varied business implications; not the least, diminishes the reputation of the supplier and threatens their competitiveness with an under utilised workforce, meeting supply deadlines for finished products, loss of sales and loss of contracts due to unreliability;

- in order to ascertain flexibility in sourcing arrangements, Australian and New Zealand manufacturers will be forced to enter offshore manufacturing or contract manufacturing, therefore impacting on the local manufacturing industry. This has flow on consequences for the economy in Australia and New Zealand; and

- retaining the *status quo* (0.1 mg/kg) is not consistent with the Australian and New Zealand Governments' commitment to the World Trade Organization. Deviation from Codex is not in the spirit of international regulatory alignment, a key objective of FSANZ.

The CMA has suggested that there have been periods where demand for Australian peanuts outstrips the availability and that price is at a premium compared to imports. In regard to the commodity price of peanuts, the CMA maintains that the issue is not solely the sourcing price of peanuts but in addition other costs emerge (e.g. promotional, packaging, air freight) when sustainability of supply is not consistently achieved leading to supply interruptions. If alternative sources/origins are required to address supply issues and this demand is unable to be met, then a risk emerges in relation to a future loss of business, reputation and future contracts for that manufacturer.

Summary of respective views

The PCA claims that there appears to be no financial benefit or reduced price for products marketed to consumers by obtaining peanuts from countries which contain higher levels of cadmium. No specific benchmark price exists for peanuts, but rather, prices depend on negotiations between the buyer and seller with imported peanuts traded at similar prices to those paid to Australian and New Zealand producers.

In addition, the PCA has worked closely with the Queensland Department of Primary Industries and Fisheries, CSIRO and the Grains Research and Development Corporation to develop management guidelines for farmers to ensure Australia's peanut production always meets the ML for cadmium in peanuts. The CMA claims that an increase to the ML will permit the Australian and New Zealand food industry to compete more effectively in the global marketplace as they would have increased flexibility to source peanuts from a variety of countries.

7.2.2.2 Issues raised at Draft Assessment

Specific submissions re-iterated that there is no evidence of a trade barrier if the current ML of 0.1 mg/kg is retained and required FSANZ to provide further comment on the following issues:

- the major beneficiaries for the increased ML are some importers and manufacturers who would be able to import inferior, presumably cheaper peanuts;
- the cost-benefits analysis is qualitative rather than quantitative and provides little detail on how the benefits outweigh the costs; and
- industry should be asked to provide more evidence, such as examples of incidences where peanuts have been rejected where the cadmium levels has exceeded 0.1 mg/kg.

One submitter also identified the following as potential costs if the ML was increased:

- the viability of local industry may be undermined with increased potential for dumping;
- the increase does not support the concept of As Low As Reasonably Achievable;
- increased use of cheaper fertilisers containing higher levels of cadmium; and
- increased costs to AQIS Imported Food Surveillance Program testing for heavy metals.

7.2.2.3 Evaluation

The focus of this application is about international regulatory alignment, removing regulatory barriers to trade while respecting public health and safety, achieving continuity of supply that meet the unique quality specifications of the confectionery industry and improving the global competitiveness (the risk for the economy is a move to offshore manufacturing).

The CMA advised FSANZ that it has consulted with a range of industry stakeholders in order to ascertain how the current ML of 0.1 mg/kg hinders trade in peanuts. In particular, the International Peanut Forum, the US Peanut Council, the Peanut and Tree Nut Processors Association in the US, the European Nut Association, and the German Peanut Council and in Australia with the Food and Beverages Importers Association (FBIA).

However, due to the current ML of 0.1 mg/kg being in place, there is limited if any evidence of exceedance of the ML from these stakeholders. Therefore, data on cadmium levels in peanuts in the range 0.1 to 0.5 mg/kg is severely limited. In addition, data from enforcement agencies (e.g. on rejections at the import barrier) is also limited as peanuts imported to Australia and New Zealand are targeted to comply with an ML of 0.1 mg/kg.

The CMA would not import inferior quality peanuts if the ML is increased as this would not be in its best interest of maintaining high-quality confectionary products. Peanut varieties are selected for incorporation into confectionary based on a range of variables (flavour, shape and size).

MLs are not determinates or indicators of quality of food commodities but rather are based on levels that are consistent with public health and safety and which are reasonably achievable from sound production and natural resource management practices.

Potential benefits to the CMA and Industry if the ML is increased

Australian Bureau of Statistics (ABS) data provided by the CMA show that on an average during 2002-2006, about 13,850 tonnes of ground-nuts were imported into the country annually. According to submissions in some years (including any drought/shortfall years) more than 10,000 tonnes of peanuts have been imported that meet the current ML for cadmium in peanuts.

The CMA has also cited data from the FBIA indicating that currently, a certain variety of peanut costs approximately $AUD0.50 /kg more in Australia compared to a Chinese equivalent.

Assuming the peanuts are of similar quality and a quarter of the confectioners are using these peanuts, and based on the cost differences submitted by the CMA, FSANZ estimates the potential benefits to confectionery manufacturers could be around $AUD1.25 million in a year where 10,000 tonnes of peanuts are imported at a price differential of $AUD0.50/Kg. Intangible benefits may include stability in supply and less fluctuation in prices for manufacturers.

If the quantity of imports, number of manufactures using this type and the difference in cost of peanuts increased, monetary gains to confectionery manufacturers could be possibly greater than $AUD1.25 million per year in this case.

The CMA presented a case study to reflect the impact due to unavailability or shortages in supply of peanuts. A manufacturer contracted to supply to a major retailer in peak selling season could risk loss of business, reputation and future contacts if they fail to deliver products containing peanuts of a certain shape and size. To prevent this situation, additional resources and arrangements will be needed to meet the scheduled spike in demand e.g. air freight to bring supplies from alternative sources. However, if this situation could be avoided including delayed return on investment, potential cost savings could be in the range of $AUD100,000 to $AUD1 million depending on the individual situation.

An industry source indicated that peanut growers and farmers in Australia could also possibly benefit from increasing the ML. Currently peanut growers may be restricted in terms of the land and resources required for cultivation to produce peanut containing less than 0.1 mg/kg of cadmium. An increase in the ML will give them more flexibility and choice in utilizing land and resources for their produce.

Potential costs to Australian industry if the ML is increased

The level of the cadmium found as a contaminant in some sources of phosphate rock varies widely. Typically, superphosphate has cadmium levels of 45-50 ppm which arises mostly from the phosphate rock. Ammoniate phosphates (monoammonium phosphate (MAP) and diammonium phosphate (DAP)) have less than 10 ppm of cadmium. The source of phosphate rock and the rate of application are the main controllers of cadmium input into soils, and these factors also affect the price.

Rapid increases in the cost of oil and indirect costs of transport, as well as increasing demand for greater crop yields in the face of increasing global pressures on the food supply have resulted in a significant increase in costs for the supply of fertilisers. It is predicted that input prices will continue to climb. Recent reports warn that urea prices are set to soar following a Chinese export tax on all fertilisers at 135%.

Reduction in global availability has seen the international urea price rise more than $US100/tonne to the current price of around $US620/tonne in Australia. DAP and MAP fertiliser prices have quadrupled in price over the past 18 months with DAP prices reaching $US1195/tonne at the end of March, up from $US433/tonne in October 2007.

The peanut farmers suggested that it is possible that they will not be able to maintain current practices, using more expensive low cadmium fertilisers, given the imperative to reduce the input costs while maintaining high production yields. The implications of using fertilisers with higher cadmium levels is that it will not only result in the gradual accumulation of cadmium, further affecting crop production in years to come, but also affects growers down stream through agricultural run-off as well as the potential to affect fisheries and bioaccumulating marine life such as prawns, shrimps, crabs, mussels and other shellfish. Direct cost impacts have not been identified by industry to support these assertions. FSANZ sought additional data from relevant sources to quantify the impacts in regard to these issues. No additional information could be obtained to inform the evaluation.

However, advice from the Fertilizer Industry Federation of Australia Inc. (FIFA) to FSANZ was that this is unlikely to occur in practice as the industry has phased out the use of high-cadmium fertilisers under the National Cadmium Minimisation Strategy.

7.2.3 Public Health Perspectives

At initial assessment, various submitters raised the following key issues in regard to public health and safety of cadmium exposure:

- cadmium is a cumulative toxin and may have adverse impacts on particular segments of the population that are exposed to high levels of cadmium;
- the promotion of trade should not be at the expense of public health and safety;
- high risk groups such as diabetics, iron-deficient women, Torres Strait Islander groups and vegetarians are more susceptible to increased intakes of cadmium and their risk is not adequately addressed by dietary exposure assessments for the average population;
- there is significant variation in individual uptake of cadmium from foods including interactions with iron, zinc, calcium, vitamin C and phytate which needs further consideration; and
- dietary survey data is out-of-date and may not reflect current exposure to cadmium.

At Draft Assessment, a range of public health and safety issues were re-raised, the majority of which had been addressed by FSANZ at Draft Assessment. However, submitters re-raised the following specific additional issues:

- High risk groups such as vegetarians and Torres Strait Islander populations may be more susceptible to increased intakes and this risk is not adequately addressed at Draft Assessment.

- It is still questionable whether the dietary exposure assessment takes into account populations (e.g. vegetarians) that may have higher intakes of peanuts and the subsequent health implications.

- Due to the narrow margin of safety between exposure and the PTWI there would need to be substantial other benefits to be gained to justify an erosion of this safety factor when it is already narrow.

- 1995 National Nutrition Survey data used for the dietary exposure assessment is out-of-date and inappropriate. It would be more prudent to await results of the 2008 Australian Total Diet Survey to ensure that the levels for cadmium used in the current assessment reflect what consumers may be exposed to from the diet.

7.2.3.1 Evaluation

FSANZ acknowledges that cadmium is a cumulative toxin, has a long biological half-life and the kidney is the critical target organ for toxic effects of long-term dietary exposure to cadmium. However, the risk assessment has concluded that dietary exposure to cadmium in Australia and New Zealand is below the internationally accepted value for the safe level of cadmium over a lifetime of exposure. In addition, peanuts are a minor contributor to overall exposure to cadmium in both Australia and New Zealand.

Therefore, the option to raise the maximum limit for cadmium in peanuts to 0.5 mg/kg is sufficient to maintain exposure within acceptable limits for all consumers including high-risk groups, even assuming that all peanuts are contaminated at the maximum limit.

Potential impacts on high risk populations of cadmium exposure

Due to the small number of the Torres Strait Islander population sampled in the 1995 NNS, there was insufficient information on consumption to provide robust results for this specific group. Furthermore, consumption of dugong, turtle or other foods traditionally consumed by this population group that possibly have high cadmium concentration levels were not recorded in the NNS. However, it is noted that there are health education campaigns in northern Queensland and the Northern Territory that specifically advise against the consumption of organ meat from dugong and turtles, since cadmium is known to accumulate in the kidney and liver of long living animals.

A separate assessment for vegetarians was not conducted as behavioural questions on vegetarians were not included in the NNS and it cannot be assumed that the non consumption of meat on the day of the survey is an indication of whether a person was a vegetarian or not at that time. The dietary exposure assessment therefore included people reporting consumption of meat, poultry and dairy products on the day of the survey and those who did not.

Even if vegetarians were high consumers of peanuts at the proposed ML of 0.5 mg/kg, and other cadmium-containing foods, the 90th percentile exposure is still well under the PTWI and they are adequately protected.

FSANZ examined other sources of consumption data to further clarify the following key issues:

- Has consumption of peanuts changed since 1995?
- Are vegetarians likely to consume significantly more peanuts than the general population?
- Are Torres Straight Islanders and Australian Aborigines likely to consume significantly more peanuts than the general population?

A number of data sources were used to address the issue of whether consumption of peanuts has changed since the 1995 NNS. In particular, the Australian Bureau of Statistics (ABS) Apparent Consumption of Food Stuffs provides data on peanut consumption from 1948-1999. There is some variation in the apparent peanut consumption, with the overall trend suggesting a decrease in peanut consumption since the 1940s with slight reversal in this trend in the 1990s. Tree nut consumption apparently has been increasing, but in 1998-99 was still below the peak in 1968-69. These data are based on nuts in shell, and might include uses such as oils; stock feed production and cannot easily be compared to the individual consumption data of the NNS.

FSANZ also looked at the Single Source[6] data for the period from 2001-2007. Single Source does not differentiate between peanuts and tree nuts. It only reports frequency of nut consumption, not amounts consumed. Generally, there is a trend for increasing frequency of consumption of nuts that applies across the whole population.

In regard to vegetarians the Single Source data provided information on self declared vegetarians. The data suggest that self declared vegetarians consume nuts 4% more frequently than other consumers.

Neither the 1995 NNS nor Single Source provides data on consumption of peanuts by TSI or other indigenous Australian Aborigines. Single Source only identifies ethnicity by country of birth. Therefore, FSANZ approached staff at the Tropical Public Health Unit based in Cairns and the Menzies School of Health Research in Darwin to seek their views on whether specific populations such as Aboriginals or Torres Trait Islanders are at higher risk from consumption of cadmium-containing foods and whether or not they had any further data on consumption of cadmium-containing foods such as peanuts by indigenous populations.

Although it is recognised that there is a high prevalence of renal disease among Aboriginal and Torres Strait Islander people and concerns have been raised about cadmium exacerbating this, they were unable to comment on whether or not consumption of peanuts with higher levels of cadmium could place these populations at higher risk of renal disease. Sources of cadmium certainly included marine animals such as turtle and dugong which are favoured foods and widely consumed both by Torres Strait Islander people and coastal Aboriginal people. However, the PTWI is also adequately protective of high-risk groups such as Aboriginals and Torres Strait Islanders.

[6] Data provided by Roy Morgan Research

Margin of safety between dietary exposure to cadmium and the PTWI

JECFA has acknowledged that there is only a relatively small safety margin between exposure in the normal diet and exposure that produces deleterious effect. In 2003, JECFA reaffirmed that 'no excess prevalence of renal tubular dysfunction would be predicted to occur at the current PTWI under the most appropriate assumptions about the fractional bioavailability of cadmium and the percentage of the absorbed cadmium that is excreted in urine.' The JECFA PTWI remains the internationally-accepted value for the safe level of intake for cadmium over a lifetime of exposure.

The PTWI is based on the most sensitive parameter for kidney damage, namely, an increase in the urinary excretion of low molecular weight protein as a result of reduced re-absorption in the renal tubules. The toxicological significance of this observed change with respect to kidney damage is still not established as it is clear that the excretion of low molecular weight proteins normally increases with age.

The exposure assessment indicated that the highest consumption estimated for Australian children aged 2-6 years was at 65-75% of the PTWI. Dietary modelling often identifies children as the sub-population most likely to have the highest exposure to various food components, partly because young children have relatively high food consumption levels when expressed on a body weight basis. At its 33rd meeting, JECFA recognised that exposure to cadmium will not be uniform with age, and that the estimate of the PTWI takes into account the higher cadmium intake on a body weight basis by infants and children[7]. Higher intakes during childhood are unlikely to be maintained over the long-term. The exposure estimates for the whole population include data from all age groups including 2-6 year olds and are a more accurate estimate of lifetime exposure to cadmium.

The exposure estimates for all population groups are likely to be overestimates because the estimate assumes that all peanuts are contaminated with cadmium at 0.5 mg/kg which is extremely unlikely as the available data on cadmium levels in peanuts indicate that most peanuts will have cadmium concentrations well below this level. Also, the data used for modelling are a 24-hour record which overestimates food consumption for high consumers as they are unlikely to consume the same foods in the same quantities every day (the use of multiple day records tends to significantly reduce predicted high consumer exposure).

Variations in the individual uptake of cadmium from foods

Experimental studies have identified various factors that can significantly influence the extent of absorption and retention of cadmium from the diet, including sex, developmental stage, and nutritional status. The gastrointestinal absorption in humans is influenced by the type of diet and the nutritional status with on average, 5% of the total oral intake of cadmium is absorbed, but individual values range from less than 1% to more than 20%. Low dietary concentrations of protein and of essential minerals such as zinc, calcium, copper, and iron have been shown to promote the absorption of cadmium in rats, while, in contrast, high or adequate dietary concentrations reduce absorption and retention.

[7] WHO. (1989) Cadmium. In: *Toxicological evaluation of certain food additives and contaminants. Thirty-third meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 24

Although it has been suggested that low iron status increases the uptake of cadmium from the gastrointestinal tract, not all the studies have confirmed this and a recent study of female farmers exposed to cadmium at levels close to the current PTWI of 7 μg/kg bw did not find any statistically significant difference in cadmium absorption between women with diabetes mellitus and controls, or women with anaemia and a control group. This confirms the large degree of variation in rates of cadmium absorption in iron deficient or anaemic females.

The bioavailability of cadmium may be reduced in some foods, for example where cadmium is bound to phytates, metallothionein or other proteins. Phytic acid, a strong chelator of minerals, is found in many plants and is known to impair absorption of nutrients including iron and zinc, and also of contaminants such as cadmium.

Composition of the diet, including fibre, protein and carbohydrates, may also affect cadmium absorption. In a recent study comparing a vegetarian/high-fibre diet and a mixed-diet group, no differences in blood or urinary cadmium concentrations were found, despite the high-fibre diet having a higher concentration of cadmium, suggesting that fibre had an inhibitory effect on cadmium absorption.

In summary, although FSANZ acknowledges that various factors influence cadmium absorption in humans, the PTWI takes into account these differences and also any differences in respect of groups that may be deemed as high risk to cadmium exposure from the diet.

Validity of the 1995 Australian or 1997 New Zealand NNS data

FSANZ is aware of the lapse of time since the previous National Nutrition Surveys and of the need to generate new survey data that documents the changes that may have occurred in the eating patterns of consumers over the last decade.

FSANZ has access to more recent food consumption data for Australia and New Zealand for selected food groups from Roy Morgan Single Source data. These data are usually assessed to determine whether consumption patterns for key foods applicable to each dietary exposure assessment are consistent with the 1995 Australian or 1997 New Zealand NNS data, and therefore, the dietary exposure assessments based on these NNS data are also reliable. However, the data from Roy Morgan were not specific enough to determine consumption patterns for key foods relating to this assessment, such as peanuts, or the major contributors cocoa and potatoes.

The 1995 NNS indicated that peanuts contributed less than 5% of the total dietary exposure to cadmium. It is not expected that there would have been a significant change in peanut consumption since then to indicate that the data are not representative. Also recent Total Diet Surveys in both Australia and New Zealand has confirmed that cadmium exposures are below the PTWI.

The 2002 Australian Total Diet Survey (ATDS) established that the average Australian dietary exposures to cadmium were below the tolerable limit of 7μg/kg bw/week and ranged from 13-68% of the PTWI. Similarly, in New Zealand the 2003-2004 New Zealand Total Diet Survey estimated the dietary exposures to cadmium to be well below the PTWI for all population groups (see section 7.2.5).

FSANZ considers that an adequate future monitoring arrangement for imported and domestic peanut products should be maintained to periodically monitor cadmium in peanuts and other foods. Therefore, FSANZ has included cadmium in the next Australian Total Diet Survey (ATDS) for 2008 as a means of monitoring cadmium levels in all foods.

7.2.4 Good Agricultural Practice (GAP)

Specific submitters raised the issue of whether the removal of the current ML of 0.1 mg/kg would lead to reductions in Good Agricultural Practices (GAP) and furthermore contribute to practices that would not encourage GAP. In particular, whether removing or permitting an increased cadmium level in one plant product could lead to transference into other foods through stock feeds, agricultural runoff or use of higher cadmium fertilisers. It was also suggested that Australia can, and does, produce peanuts that meet the current cadmium ML and Australian industry invests money and effort to ensure that each batch meets the ML. If the ML is removed it may open up the way for other countries to export to Australia without any restrictions on cadmium levels in peanuts.

7.2.4.1 Evaluation

FSANZ acknowledges the commitment that Australian industry invests in meeting the ML. By maintaining an ML this will still serve as a mechanism of restricting cadmium levels in food and of encouraging GAP from countries other than Australia from which peanuts are sourced.

Cadmium is naturally present in soil and is taken up by plants through their root system and peanuts can absorb cadmium from the soil directly into pods. A range of soil factors (pH, salinity, amount of cadmium concentration in the soil, metal sorption, micronutrient and macronutrient status, temperature, crop cultivar and aeration) play a major role in controlling cadmium accumulation in food crops[8]. Manipulation of these factors by Australian farmers has been shown to be successful in mitigating cadmium concentrations in soils. In regard to the accumulation of cadmium in peanuts, a previous study suggested that neither addition of lime or zinc fertilisers had a major impact on the peanut kernel cadmium concentration; but rather, soil type, site selection and fertiliser history remains the most effective practice to minimise cadmium concentrations in peanut kernels[9]. Furthermore, it was suggested that agronomic techniques to manage cadmium uptake by peanuts in the field may need to consider modification of the soil to the full depth of the roots, rather than just the surface strata where pods develop[10]. Therefore, it is clear that cadmium levels in soils involve a number of variables and minimising these levels requires a broad approach.

In light of all these variables that affect cadmium uptake by plants, Australia put in place a National Cadmium Minimisation Strategy (NCMS) managed and implemented by the National Cadmium Management Committee (NCMC). This was established from July 2000 to November 2006 and comprised representatives of all States, the Commonwealth (FSANZ), CSIRO, the National Farmers' Federation and FIFA.

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[8] Information about cadmium in Australia. Prepared by the Australian Government for the United Nations Environment Programme. December 2005.

[9] Bell MJ, Wright GC, McLaughlin MJ et al (1998) Impact of agronomic practices on cadmium uptake by peanuts. Proceedings Australian Agronomy Conference, Wagga Wagga, Australia. pp 371-374.

[10] McLaughlin MJ, Bell MJ, Wright GC and Cozens GD (2000) Uptake and partitioning of cadmium by cultivars of peanut (Arachis hypogaea L.) Plant and Soil, 222, 51-58.

A dedicated web-site is located at www.cadmium-management.org.au for stakeholders seeking further detailed information on the NCMS.

The NCMC reported regularly to the Primary Industries Standing Committee (PISC) on the success of the strategy. The Strategy was funded for five years by FIFA, Horticulture Australia Ltd and the Grains Research and Development Corporation.

The following key strategies were implemented in Australia:

- development of Best Management Practices for the production and processing of agricultural produce for those industries and/or areas which have an existing or potential problem with cadmium levels in their produce;

- development of a Code of Practice by the fertiliser industry to target low cadmium fertiliser to those areas/industries which have an existing or potential cadmium problem;

- encouragement of all State departments to re-affirm their previous commitment to reduce the regulated level of cadmium in phosphatic fertilisers to 300 mg cadmium/kg phosphorous by 2000;

- all States were to consider the labelling of fertilisers and soil ameliorants, to alert growers to their cadmium content; and

- to increase the awareness by the Standing Committee on Agriculture and Resource Management (SCARM) and all State departments of the risks of the use of biosolids and other soil ameliorants in adding cadmium to the soil, and to take this risk into account when setting standards for their use.

In summary, it is evident that Australia has taken significant actions to reduce the impact of cadmium in the environment and in effect to contribute to reduced exposure to cadmium for humans. There are now appropriate controls and guidelines in place to encourage good agricultural practices by Australian farmers.

7.2.5 Surveillance of cadmium levels in peanuts

Specific submitters sought information on past dietary exposure estimates of cadmium in the food supply and whether FSANZ is proposing to monitor cadmium levels in peanuts and other foods in the future.

7.2.5.1 Evaluation

FSANZ considers that an adequate future monitoring arrangement for imported and domestic peanut products should be maintained to periodically monitor cadmium in peanuts and other foods. Therefore, FSANZ has included cadmium in the next ATDS for 2008 as a means of monitoring cadmium levels in all foods.

FSANZ is one agency responsible for monitoring the levels of contaminants in the Australian and New Zealand food supply via the Australian Total Diet Survey (ATDS).

In 2002, the ATDS established that average Australian dietary exposures to cadmium were below the tolerable limit of 7µg/kg bw/week and ranged from 13 to 68% of the PTWI.

Similarly, cadmium dietary exposures were assessed for the New Zealand population in the 2003-2004 New Zealand Total Diet Survey with estimated dietary exposures to cadmium well below the PTWI for all population groups. Children six years and below showed the highest dietary exposure for cadmium at 30-37% of the PTWI[11].

In 2001, FSANZ conducted a revised dietary exposure assessment for cadmium for both the Australian and New Zealand populations. This report's findings (FSANZ, unpublished) indicated that dietary exposures to cadmium were well below the PTWI. More specifically, the Australian population's estimated dietary exposure to cadmium ranged from 13-16% of the PTWI, while New Zealand's estimated dietary exposures were 14-17% of the PTWI, these ranges relating to lower and upper bound estimates[12].

Further detailed information on current and possible exposure to cadmium from peanuts and other foods has been detailed in the Dietary Exposure Assessment Report (**Attachment 3**) and has been detailed in Section 5 above.

7.2.6 Discussion of costs and benefits of options

7.2.6.1 Option 1 – Reject the Application and thus retain the ML for cadmium in peanuts of 0.1 mg/kg (*Status quo*)

AFFECTED PARTY	BENEFITS	COSTS
Government	May assist in contributing to the National Cadmium Management Strategy's objective of reducing cadmium exposure for Australian and New Zealand consumers by a variety of measures.	Retaining the ML may not be consistent with the current government policy of setting MLs for foods that only provide a significant contribution to total dietary exposure or an effective risk management strategy. May potentially be viewed as being inconsistent international trade obligations under the World Trade Organization's Sanitary and Phytosanitary Agreement and Technical Barriers to Trade Agreement as Codex does not set an ML for cadmium in peanuts.

[11] New Zealand Food Safety Authority. (2005) 2003/04 New Zealand Total Diet Survey. Rutishauser I. (2000) Getting it right: How to use the data from the 1995 National Nutrition Survey. Commonwealth of Australia: Canberra.

[12] Where analysis indicates a result that is below the level of reporting (LOR), the lower bound model assigns a value of zero to these non-detect results; the upper bound model assigns a value of LOR to the non-detect result.

AFFECTED PARTY	BENEFITS	COSTS
Industry	The Australian Peanut Industry's image of being clean and green is maintained as the Peanut Company of Australia[13] have maintained that they can produce adequate peanuts in Australia and source a range of peanuts from other countries that meet the current ML of 0.1 mg/kg.	Manufacturers of peanut-containing products may be disadvantaged if the supply of peanuts is decreased from the lack of availability in sourcing peanuts. This may affect the flow of imports into Australia which impacts on the timely and cost-effective supply of imported peanuts. This may mean that some confectionery manufactures may move to manufacture offshore from Australia and import finished product to the local markets which may impact negatively on the Australian and New Zealand economies.
Consumers	Consumers would not be exposed to the possibility of increased levels of cadmium in the diet.	If the availability of peanuts was limited there may be an increase in price of peanuts leading to a disadvantage from a price perspective to consumers.

7.2.6.2 Option 2 – Harmonise with Codex and remove the ML for cadmium in peanuts.

AFFECTED PARTY	BENEFITS	COSTS
Government	This would support the policy of setting MLs for foods that provide a significant contribution to total dietary exposure. This option would allow alignment with Codex leading to removal of barriers to trade and the Government would be viewed as facilitating international trade. Therefore, removal of the current ML may be perceived as removing a potential trade barrier.	The Government may be viewed as favouring industry free trade over an ability to reduce overall cadmium exposure to all consumers by containing levels of cadmium in foods at as low as reasonable achievable levels without undue disruption to that food in the market.
Industry	Manufactures of peanut-containing foods and importers of peanuts would have increased flexibility to source peanuts from a variety of countries such as USA, Argentina and China (e.g. when supply is threatened) and compete more effectively in the global marketplace If the Australian peanut crop was insufficient to satisfy the market demand this option would allow manufactures and importers access to as wide a range of sources of peanuts as possible. This option would allow increased flow of imports into Australia in a timely and cost-effective manner	Particular sectors of the Australian peanut industry may be disadvantaged; as they will be required to compete with imported peanuts that may be less costly to source from specific countries. May be viewed as counterproductive to the various programs that Australian peanut farmers employ to keep cadmium levels to a minimum in Australian soils.
Consumers	The cost of peanuts and peanut-containing products may be lower if a plentiful supply of imports is maintained during times of need.	There may be an increased risk for consumers of higher cadmium exposure.

[13] The PCA is the largest supplier of peanuts to the peanut butter, confectionary and snack foods markets in Australia and one of the major suppliers to New Zealand.

7.2.6.3 Option 3 – Establish a higher ML for cadmium in peanuts.

AFFECTED PARTY	BENEFITS	COSTS
Government	Australia has adopted a national strategy to maintain safe levels of cadmium in its agricultural soils and produce and is an important move in ensuring safe food for Australians and a competitive edge for agricultural exports. The National Cadmium Minimisation Strategy, working under the Primary Industries Standing Committee provided a consistent and coordinated program to address issues related to the control of cadmium in soils and crops. Therefore, this option would continue to serve as an additional incentive for industry to contribute to the overall cadmium management strategy of reducing cadmium exposure for Australian and New Zealand consumers and thus not undermine the work done in Australia to date to reduce overall cadmium levels in foods.	The Government may be viewed as favouring industry free trade over an ability to reduce overall cadmium exposure to all consumers by containing levels of cadmium in foods at as low as reasonable achievable levels without undue disruption to that food in the market.
Industry	Some importers and manufactures would be advantaged by this option as they could potentially source cheaper peanuts with higher cadmium levels and these savings may be passed onto the consumer. Would continue to serve as an incentive for industry to contribute to the overall cadmium management strategy of reducing cadmium exposure for Australian and New Zealand consumers.	Particular sectors of the Australian peanut industry may be disadvantaged; as they will be required to compete with imported peanuts that may be less costly to source from specific countries. May be viewed as counterproductive to the various programs that Australian peanut farmers employ to keep cadmium levels to a minimum in Australian soils.
Consumers	The cost of peanuts and peanut-containing products may be lower if a plentiful supply of imports is maintained during times of need.	

7.3 Comparison of Options

For naturally-occurring contaminants such as cadmium, there will be a range of levels in any sample of peanuts sourced from a range of countries, with high outlier values from time to time. To reflect this variability either a deletion of the ML or an increase could be considered to ensure that consignments of peanuts could continue to be traded, provided that this would not lead to public health and safety concerns.

Option 1 would restrict the sourcing of peanuts from a variety of overseas countries and would serve to limit any further exposure to cadmium in the diet and would alleviate perception from particular stakeholders that the Government is placing trade aspects ahead of consumers' health. Although concerns have been expressed in regard to negative effects on public health and safety for Australian and New Zealand populations, based on the risk assessment FSANZ has undertaken, there is no evidence that there would be an increased risk to public health and safety. To maintain the current ML may still represent a trade barrier and may possibly lead to retaliatory action against Australia under the rules of the WTO.

Option 2 would allow sourcing from a variety of countries with any cadmium levels. This would be fully consistent with Australia's and New Zealand's obligations under the WTO in that Codex does not have an ML for peanuts as they are deemed as low contributors to cadmium exposure in the diet of consumers. This approach is consistent with the Codex Alimentarius approach to contaminants and is referred to as the ALARA principle (**a**s **l**ow **a**s **r**easonably **a**chievable) and with the policy principles that were agreed to by the then Australia New Zealand Food Standards Council in July 1997.

Option 3 would serve as a compromise from the open ended approach of no ML (option 2) by maintaining an ML to ensure that the overall cadmium levels in food are no higher than necessary, yet will alleviate the current restrictions in trade of peanuts experienced by the CMA.

Both Option 2 and 3 may potentially lead to a greater availability of peanuts on the Australian and New Zealand market. Whilst this potential for increased exposure to peanuts may increase the probability of specific consumers being at greater risk of more allergies developing if there was a greater supply of peanuts on the market, mandatory labelling of peanuts in food is required under Standard 1.2.3-Mandatory Labelling and Advisory Statements and Declarations to alert consumers to the presence of peanuts in foods.

7.3.1 Preferred option

The preferred option is **Option 3** as it is preferable to increase the ML for cadmium in peanuts from 0.1 to 0.5 mg/kg, rather than delete the current ML. The proposed ML of 0.5 mg/kg is safe and based on the upper range of distribution of cadmium levels in peanuts from the available survey data. It will also enable the current shortfall between domestic production and imported peanuts claimed by the CMA as significant to be met by imports from a variety of countries, in order to assist specific industry sectors when supplies are in short (e.g. during times of drought).

COMMUNICATION

8. Communication and Consultation Strategy

When the ML for cadmium in peanuts in the Code was raised from 0.05 mg/kg to 0.1 mg/kg during the review of cadmium (Proposal P144), there was strong opposition from key stakeholders in Australia. Peanut growers appear to favour the maintenance of an ML in the Code as a means of controlling imported peanuts, which they claim have higher cadmium levels.

Codex has not established an ML for cadmium in peanuts because peanuts do not contribute significantly to overall global cadmium exposure. Therefore, the current ML for cadmium in peanuts in the Code of 0.1 mg/kg may be considered as a technical barrier to trade.

Consumers may become concerned by the Application as it seeks a loosening of regulations. Any subsequent amendments to the Code may be interpreted as preference being given to trade considerations over public health and safety.

FSANZ held discussions with the Australian State/Territory and New Zealand Health Departments to ascertain their views on this Application on 4 February 2007, 31 October 2007 and 12 February 2008 via the Jurisdictional Forum[14]. The discussions covered the proposed options and, in particular, the implications of removing the ML for cadmium in peanuts. Other issues included the effect, if any, on good agricultural practice (GAP), managing the enforcement of occasional incidents of high levels of cadmium and whether there would be a need to specifically monitor public health if the ML is removed. Specific jurisdictions also indicated that should an ML be continued it may have to be defended with the WTO, as Codex has no level set for cadmium in peanuts.

FSANZ maintains that the proposed measures protect public health and safety while permitting the appropriate and practical sourcing of peanuts from a variety of countries. The measures are also based upon the best available scientific evidence, which indicates there is no risk to public health and safety if the ML is increased to a level of 0.5 mg/kg.

9. Consultation

The Initial Assessment was advertised for public comment between 4 October 2006 and 15 November 2006.

Many of the submissions providing data to the questions posed in the Initial Assessment Report were valuable in understanding stakeholder views in regard to options 2 or 3 (delete or increase the current ML for cadmium in peanuts) as well as giving an insight into consumer and jurisdictional attitudes towards the proposed risk management options.

The following key issues were identified:

- the public health and safety risk of deleting or increasing the current ML of 0.1 mg/kg;
- the issue of removal of the ML leading to reductions in Good Agricultural Practices (GAP) and an increase in overall cadmium exposure for consumers; and
- whether the ML is currently a trade barrier as some submissions questioned whether the CMA can justify this claim.

Overall, the CMA supports the removal of the ML as it considers peanuts are a low contributor to cadmium exposure and this would ensure continuity of supply from USA, Argentina and China and allow the CMA to compete more effectively in the global marketplace.

However, the PCA does not believe that maintaining the ML is a trade barrier and that CMA should provide further details on where the trade barrier exists and that the CMA is not restricted in the choice of countries to source peanuts that meet the current ML in the Code.

[14] The Jurisdictions Forum provides an additional means by which jurisdictions can participate in the FSANZ standards setting process and engage in informed discussion around specific issues related to standards development.

9.1 Issues raised at Draft Assessment

Seventeen submissions were received in response to the DAR (**Attachment 4**). The additional issues raised have been addressed in section 7.2 above and two items that were considered general are evaluated below.

FSANZ was requested to supply evidence to support how an increase to the ML would encourage adherence to good agricultural practice (GAP) in Australia and furthermore how it would be consistent with the established cadmium minimization strategy implemented from 2000 to 2006.

FSANZ did not mean to imply that an ML is the principle means of ensuring GAP for any contaminants in the food supply, but rather that the NCMS is a long-term commitment to cadmium reduction. Therefore, maintenance of an ML in peanuts would broadly continue to encourage adherence to the NCMS key strategies identified in Section 7.2.4.

Although FSANZ considers that there are no public health and safety concerns with an increased ML, FSANZ suggested that overall an ML albeit increased would contribute to keeping cadmium levels in soils to as low as reasonably achievable, in comparison to no ML. However, this was not meant to imply that in peanut growing parts of the world that GAP should be completely disregarded.

FSANZ has taken an inconsistent approach on safety in this Application when compared to the assumptions made on the Proposal on cassava chips.

The Proposal for ready-to-eat cassava chips uses a margin of safety (MOS) approach, i.e., by comparing the likely exposure of children to cyanogenic glycoside (primarily linamarin) to the safety factor corrected value in animals (hamsters) which led to an MOS of 10-fold. However, this was derived based from a single oral dose study in animals to account for possible acute toxicity in humans. However, for Application A552 a comparison was made between the anticipated (or known) level of exposure from all cadmium-containing foods compared to the PTWI for each population group. This takes into account chronic or lifetime exposure for populations of all ages. Therefore, as the two toxicological endpoints (acute versus chronic) used to estimate risk are completely different, the approaches cannot be directly compared.

9.2 World Trade Organization (WTO)

As members of the World Trade Organization (WTO), Australia and New Zealand are obligated to notify WTO member nations where proposed mandatory regulatory measures are inconsistent with any existing or imminent international standards and the proposed measure may have a significant effect on trade.

There is no ML for cadmium in peanuts set by Codex. Amending the Code to increase the ML as proposed at Draft Assessment may have a significant effect on international trade as regulations in Australia and New Zealand would be less restrictive. Therefore, due to the lower restrictions on trade in peanuts that may result from this measure, notification was recommended to the agencies responsible in accordance with Australia's and New Zealand's obligations under the WTO Sanitary and Phytosanitary Measures (SPS) Agreement.

This is because the primary objective of the measure is to protect human, animal and plant health and the environment.

Comment was sought from key stakeholders on whether a level of 0.5 mg/kg is appropriate to reduce the likelihood of rejections of peanuts from other key countries. No comments were received from WTO Members.

CONCLUSION

10. Conclusion and Preferred Approach

It is proposed to increase the current ML for cadmium in peanuts. This will enable the current shortfall between domestic production and imported peanuts claimed by the CMA as significant to be met by imports from a variety of countries to assist specific industry sectors where supplies are in short supply (e.g. during times of drought).

Preferred Approach

Amend the Table to clause 2 of Standard 1.4.1 to increase the current ML for cadmium in peanuts from 0.1 to 0.5 mg/kg.

10.1 Reasons for Preferred Approach

FSANZ recommends the proposed draft variation to Standard 1.4.1-Contaminants and Natural Toxicants for the following reasons:

- the proposed draft variation to the Code is consistent with the section 18 objectives of the FSANZ Act, in particular, it does not raise any public health and safety concerns, it is based on risk analysis using the best available scientific evidence, and helps promote an efficient and internationally competitive food industry;

- FSANZ has conducted an assessment of the safety of cadmium which concludes that the PTWI of 7 μ/kg body weight is adequately protective of consumers. The dietary exposure assessment shows that increasing the current ML from 0.1 to 0.5 mg/kg does not affect public health and safety; and

- the regulatory impact statement concludes that the benefits of the proposed regulatory option outweigh the costs. Adoption of this option will enable the shortfall between domestic peanut production and industry demand to be met by imports, while promoting good agriculture practice, the protection of public health and safety and to facilitate trade in peanuts by enabling peanut importers to source peanuts from a wider number of countries.

11. Implementation and Review

It is proposed that the draft variation comes into effect on the date of gazettal.

ATTACHMENTS

1. Draft variation to the *Australia New Zealand Food Standards Code*
2. Hazard Assessment Report
3. Dietary Exposure Assessment Report
4. Summary of issues raised in public submissions

Attachment 1

Draft variation to the *Australia New Zealand Food Standards Code*

Standards or variations to standards are considered to be legislative instruments for the purposes of the Legislative Instruments Act (2003) and are not subject to disallowance or sunsetting.

To commence: on gazettal

[1] ***Standard 1.4.1*** *of the Australia New Zealand Food Standards Code is varied by omitting from the* Table to clause 2, *under the heading* Cadmium, *the entry for* Peanuts, *substituting –*

Peanuts	0.5

Attachment 2

Hazard Assessment Report

1. Executive Summary

Cadmium is a heavy metal that is found as an environmental contaminant, both through natural occurrence and from industrial and agricultural sources. Cadmium is present in food primarily as a result of its uptake from soil and water. Cadmium absorbed from food accumulates in the kidney and liver of mammals, including humans, where over time it can cause kidney dysfunction. Perhaps the best known example of the effects of cadmium contamination of food is the prevalence of Itai Itai disease, characterised by kidney tubular dysfunction, severe bone pain and numerous bone fractures, predominantly in women, in the Jinzu river basin of Japan following contamination of water sources from an upstream mine (WHO, 1992).

The majority of cadmium in food passes through the gastrointestinal tract unabsorbed, with the level of absorption being influenced both by nutritional status (e.g. iron status) and bioavailability (e.g. associated with phytic acid). Absorbed cadmium is redistributed to the liver and kidneys where it is normally sequestered by small proteins called metallothioneins. Toxicity of cadmium is thought to occur when the binding capacity of metallothionein is exceeded.

Cadmium has a long biological half-life, estimated at between 16-33 years. Even low exposure levels may, in time, cause considerable accumulation, especially in the kidneys. The kidney is the critical target organ for toxic effects of long-term dietary exposure to cadmium. Toxicity is manifested only after many years of slow accumulation of cadmium in the renal cortex and then only if a critical concentration is achieved. The chronic effects on the kidney are characterised by tubular dysfunction and cell damage, most often manifested as urinary excretion of low molecular mass proteins. Urinary levels of two proteins in particular, protein HC (also know as α_1-microglobulin) and β2-microglobulin, are commonly used as biomarkers of effect on renal tubule function. A small fraction of cadmium in the kidneys is excreted in the urine, and the level of cadmium in the urine normally correlates with total body burden. However, if damage to the kidney proximal tubules has occurred, cadmium levels in the urine are paradoxically lower.

Numerous animal studies have confirmed the toxicity of cadmium to the kidney and liver. The majority of human data comes from epidemiological studies, from both industrial and environmental exposure to cadmium, including studies of populations exposed to low levels of cadmium through normal dietary exposure. Data from autopsy and epidemiological studies have been used to estimate a critical cadmium level in renal cortex at which signs of renal dysfunction may be present, and levels of lifetime cumulative intake of cadmium that would produce that critical level. The Joint FAO/WHO Expert Committee on Food Additives (JECFA) estimates the critical cadmium level in renal cortex is 200 mg/kg wet weight, resulting from the cumulative intake of about 2000 mg cadmium.

JECFA considered that levels of cadmium in the kidney should not exceed 50 mg/kg, and established a PTWI of 7 μg/kg bw based on an absorption rate of 5% and a daily excretion of 0.005% of body load.

This PTWI, established in 1972, has been confirmed by subsequent considerations by JECFA, in 1988, 1993, 2000 and 2003. JECFA has acknowledged that there is only a relatively small safety margin between exposure in the normal diet and exposure that produces deleterious effect. In 2003, JECFA reaffirmed that 'no excess prevalence of renal tubular dysfunction would be predicted to occur at the current PTWI under the most appropriate assumptions about the fractional bioavailability of cadmium and the percentage of the absorbed cadmium that is excreted in urine.' The JECFA PTWI remains the internationally accepted value for the safe level of intake for cadmium over a lifetime of exposure.

The PTWI is based on the most sensitive parameter for kidney damage, namely, an increase in the urinary excretion of low molecular weight protein as a result of reduced re-absorption in the renal tubules. The toxicological significance of this observed change with respect to kidney damage is still not established as it is clear that the excretion of low molecular weight proteins normally increases with age.

The other important factor to consider when interpreting the potential risk of intake levels near or above the PTWI for cadmium is that it has been established on the basis of cumulative intake. Thus, intake above this figure may be tolerated provided it is not sustained for long periods of time and does not produce a significant increase in the integrated life-time dose.

This safety assessment focuses on the risks associated with cadmium in food, and is primarily based on epidemiological data, particularly those conducted on populations that are likely to have a similar diet and nutritional status to Australians and New Zealanders. In particular, epidemiological studies in Belgium and Sweden suggest that even normal dietary exposure to cadmium can result in changes in kidney function, as evidenced by increased urinary excretion of low molecular mass proteins. However, increased excretion of biomarkers of effect does not necessarily indicate an adverse effect on kidney function and the long-term significance of these changes on human health is unclear. Also, several of these studies suggest that cadmium exposure explains only a small proportion of overall kidney effects, and that age is the major component affecting renal tubular function in populations chronically exposed to low levels of cadmium in the diet.

Dietary exposure to cadmium in Australia and New Zealand is below the provisional tolerable intake of 7 μg/kg bw/week and is within acceptable safety standards. Peanuts are a minor contributor to overall exposure to cadmium in both Australia and New Zealand. The proposal to raise the maximum limit for cadmium in peanuts to 0.5 mg/kg is sufficient to maintain exposure within acceptable limits for all consumers, even assuming that all peanuts are contaminated at the maximum limit.

2. Introduction

Cadmium is a ubiquitous environmental contaminant, arising from both natural and anthropogenic sources. Cadmium present in the soil and water is taken up by plants and filter-feeding marine animals such as oysters. Food-borne cadmium is the major source of exposure for most people. A varied, healthy diet will unavoidably contain some cadmium. The total dietary intake in non-polluted areas of most countries is estimated to be approximately 10-40 μg/day.

Cadmium is a cumulative toxin, with a relatively long biological half-life of 10-20 years. Cadmium accumulates in kidney and liver and is known to cause kidney damage and bone damage. The kidney is considered the critical target organ. Low level chronic exposure increases the risk of renal dysfunction and may increase the risk of osteoporosis and bone fractures, particularly in post-menopausal women. High levels of chronic exposure due to cadmium pollution are known to have caused severe toxicity, particularly among female farmers in parts of Japan. Itai Itai disease is characterised by renal tubular dysfunction, multiple bone fractures due to osteomalacia and renal anaemia, and is named for the most characteristic symptom, severe bone pain, as Itai Itai translates as ouch-ouch.

Other reviews of cadmium

The toxicity of cadmium has been extensively reviewed in recent years by the International Programme on Chemical Safety (IPCS) (WHO, 1992), by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) (WHO, 1972; WHO, 1989; WHO, 2001; WHO, 2004) and by the US Agency for Toxic Substances and Disease Registry (ATSDR, 1999). JECFA also considered the impact of different maximum limits for cadmium (WHO, 2006). This review draws on the IPCS, JECFA and ATSDR reports, particularly focussing on human data and food exposure.

The reports from a small number of epidemiological studies, including benchmark dose approaches to assessing cadmium risk, have become available since the most recent JECFA consideration. The most relevant of these studies have been assessed and are presented here.

3 Review of Safety

3.1 Absorption, distribution and excretion

Cadmium is a relatively rare metallic element that is not naturally found in a pure state. Cadmium can form a number of inorganic salts, some of which, such as sulphide, carbonate and oxide, are practically insoluble in water. In nature, under the influence of oxygen and acids, these can be converted to water-soluble salts such as the sulphate, nitrate and halogenates. There is no evidence for naturally occurring organic cadmium salts. Cadmium is still regarded as inorganic when bound to proteins and other organic molecules, or in salts with organic acids. Most cadmium found in mammals, birds and fish is probably bound to protein molecules, particularly metallothionein (WHO, 1992).

Cadmium is virtually absent at birth but accumulates with time, particularly rapidly during the early years of life. Cadmium absorbed by the gastrointestinal tract is mainly stored in the liver and kidneys, predominantly in the renal cortex. It is estimated that, in a non-smoking adult, about a third of the body burden is in the kidney. If exposure to cadmium remains constant and low throughout life, the concentration of cadmium in the kidneys may be 10-20 times higher than those in the liver. Paradoxically, people with high exposure to cadmium may have low levels of kidney cadmium and high levels of liver cadmium, as a result of severe cadmium-induced renal dysfunction.

After oral exposure, up to 90% of cadmium passes through the gastrointestinal tract unabsorbed (WHO, 1972). Only a small proportion of the daily absorbed dose is excreted.

The urinary excretion of cadmium is dependent on a number of factors including body burden, recent exposure and renal damage and is estimated to be approximately 0.01% of the total body burden (WHO, 1992). In exposed people with renal damage, urinary excretion of cadmium increases sharply so that the whole body half-life is shortened. Gastrointestinal excretion is thought to be of a similar magnitude to urinary excretion, but it cannot be easily measured as it is not possible to distinguish excretion from unabsorbed cadmium in the faeces.

The slow excretion results in cadmium concentrations in most organs increasing with age and an extremely long biological half-time in the kidneys, liver and total body. Estimates for whole body biological half-life range from 16 years to 33 years based on estimates of daily excretion rates of 0.005% to 0.01% of total body burden.

In the animal species tested, the oral bioavailability of cadmium ranged from 0.5 to 3.0%, on average. Experimental studies also identified various factors that can significantly influence the extent of absorption and retention of cadmium from the diet, including sex, developmental stage, and nutritional status. Low dietary concentrations of protein and of essential minerals such as zinc, calcium, copper, and iron have been shown to promote the absorption of cadmium in rats, while, in contrast, high or adequate dietary concentrations reduce absorption and retention. In particular, low iron status increases the uptake of cadmium from the gastrointestinal tract (WHO, 2004). Metallothionein-bound cadmium in food does not appear to be absorbed and/or distributed in the same way as inorganic cadmium compounds.

Gastrointestinal absorption in humans is also influenced by the type of diet and the nutritional status. On average, 5% of the total oral intake of cadmium is absorbed, but individual values range from less than 1% to more than 20% (WHO, 1992). In women with low body iron stores, as evidenced by low serum ferritin levels, gastrointestinal absorption of radiolabelled cadmium (cadmium chloride) was higher than for a control group of women (around 9% compared to 3%, (Flanagan et al., 1978). In another study, women with reduced body iron stores had low serum ferritin concentrations which were highly correlated with blood cadmium concentrations, suggesting that cadmium absorption is enhanced when the body iron stores are suboptimal (Berglund et al., 1994).However, several recent studies indicate that women with iron deficiency or anaemia do not have significant increases in cadmium body burden, suggesting a large degree of variation in rates of cadmium absorption (WHO, 2004). A study of female farmers exposed to cadmium at levels close to the current PTWI of 7 □g/kg bw did not find any statistically significant difference in cadmium absorption between women with diabetes mellitus and controls, or women with anaemia and a control group. Age was the only independent factor that was a significant predictor of the rate of absorption of cadmium (Horiguchi et al., 2004a) summarised in (WHO, 2004). In another study of the effect of iron deficiency on cadmium uptake, a total of 1482 women living in six prefectures of Japan were classified into groups of anaemic, iron deficient and normal iron levels, based on ferritin and haemoglobin levels. No significant increases in levels of urinary cadmium or low-molecular-mass protein (protein HC and □2-microglobulin) were found in the groups of anaemic and iron-deficient women compared with the matched control population (Tsukahara et al., 2003) summarised in (WHO, 2004).

The bioavailability of cadmium may be reduced in some foods, for example where cadmium is bound to phytates, metallothionein or other proteins. Phytic acid, a strong chelator of minerals, is found in many plants and is known to impair absorption of nutrients including iron and zinc, and also of contaminants such as cadmium.

There is evidence suggesting that cadmium is less efficiently absorbed from shellfish, including oysters, where most cadmium is metallothionein bound (WHO, 1992; Vahter et al., 1996). Composition of the diet, including fibre, protein and carbohydrates, may also affect cadmium absorption. In a study comparing a vegetarian/high-fibre diet and a mixed-diet group, no differences in blood or urinary cadmium concentrations were found, despite the high-fibre diet having a higher concentration of cadmium, suggesting that fibre had an inhibitory effect on cadmium absorption (Berglund et al., 1994).

After absorption by rats, cadmium is distributed mainly to the liver, with subsequent redistribution to the kidney in conjugated forms such as cadmium–metallothionein and cadmium–albumin (WHO, 2004). The distribution of cadmium is also influenced by the route of exposure, with dietary cadmium being almost equally distributed between the liver and kidney, while subcutaneous exposure results in liver levels 11 times higher than in kidneys (WHO, 1992).

Following absorption from the human gastrointestinal tract and slower absorption into the systemic circulation, cadmium is redistributed primarily to the liver and subsequently to the kidney (WHO, 2004). Cadmium can induce metallothionein synthesis in many organs including the liver and kidney. Metallothioneins are small, cysteine-rich proteins that bind a variety of divalent and trivalent cations with high affinity and are important transport and storage proteins for cadmium. Cadmium also induces metallothionein in the placenta and the fully developed placenta retains cadmium while transporting metallothionein bound zinc and copper, essential metals, to the foetus (WHO, 2001). Cadmium in the liver and kidneys is mainly bound to metallothionein and the binding of intracellular cadmium to metallothionein is thought to protect against the toxicity of cadmium. Hepatic damage is more severe in mice that do not express metallothionein (WHO, 2004). Cadmium not bound to metallothionein may therefore play a role in the pathogenesis of cadmium-related tissue injury.

Metallothionein bound cadmium is slowly released into the plasma, filtered through the glomeruli and reabsorbed in the proximal tubules, leading to selective accumulation of cadmium in the renal cortex (WHO, 2001). Accumulation of cadmium in the kidneys declines at 50-60 years of age, possibly due to age-related changes in kidney function (WHO, 2001). When cadmium taken up by the kidney proximal tubules exceeds the binding capacity of metallothionein, cadmium induces cytotoxicity, and eventually causes structural and functional damage (WHO, 2004).

3.1.1 Biological indices of cadmium burden (biomarkers of exposure)

As there is no easy way to directly measure whole body burden of cadmium, levels of cadmium in urine, blood and hair have been used to estimate exposure and accumulation of cadmium. For long-term exposure to low levels of cadmium, levels of excreted in urine are significantly correlated with kidney concentrations of cadmium. However, this is not the case for episodes of high level exposure or when cadmium levels have already resulted in nephropathy. Urinary excretion of cadmium is commonly adjusted for dilution by adjustment to grams of creatinine excreted, but can also be adjusted to mean urinary density.

Cadmium levels in blood mainly reflect recent exposure over weeks or months, although workers with relatively long durations of exposure may have elevated blood cadmium for several years. Smokers are known to have higher blood cadmium levels than non-smokers.

Cadmium in hair is not a reliable indicator of exposure or body burden, primarily because external contamination of the hair cannot be distinguished from endogenous cadmium. Most faecal cadmium represents unabsorbed material, and faecal cadmium can indicate the daily amount of cadmium ingested via food and water.

3.2 Toxicity

3.2.1 Acute studies

As the focus of this report is on the chronic effects on human health following long-term dietary exposure to cadmium, data on the effects of a single exposure in animals will only be briefly covered.

Oral LD_{50} values for experimental animals, primarily rodents, range from approximately 100 to 300 mg/kg bw, depending on the form of cadmium administered. The LD_{50} for cadmium salts with very low solubility, such as cadmium sulphide (CdS, LD_{50} 910 mg/kg bw for cadmium ion) is approximately an order of magnitude greater than the LD_{50} for very soluble forms of cadmium, such as cadmium chloride ($CdCl_2$, 57 mg/kg bw, IPCS, 1992; WHO, FAS, 2004). At cadmium doses slightly below the LD_{50}, histopathological evidence of liver toxicity (fibrosis, necrosis) is commonly observed, as well as testicular atrophy and necrosis in male rats and mice (WHO, 2004). Large oral doses also damage the gastric and intestinal mucosa (WHO, 1992).

The LD_{50} after parenteral administration is about 10-20 times lower than for oral administration (WHO, 1992). Following injection of large doses, severe endothelial damage is seen in the small vessels of the peripheral nervous system and in the testis. The liver appears to be the major target organ for acute cadmium toxicity in rats, and liver damage is probably the lethal effect of a single high parenteral exposure (WHO, 1992). Single injections of cadmium salts cause pronounced effects in the gonads: testicular haemorrhagic necrosis resulting from endothelial damage, and haemorrhages and necroses in the ovaries of prepubertal rats and adult rats in persistent oestrus (WHO, 1992).

3.2.2 Long term studies

Long-term oral or parenteral administration of cadmium produces effects primarily on the kidneys, but also on the liver as well as the haematopoietic, immune, skeletal and cardiovascular systems (WHO, 1992). The chronic effects on the kidney, characterised by tubular dysfunction and cell damage, are of greatest relevance to human exposure to cadmium through food. Cadmium exposure also results in a disturbance of calcium and vitamin D metabolism. Some, but not all, studies report that this leads to osteomalacia and/or osteoporosis.

The kidney effects of long-term oral exposure to cadmium have been demonstrated in a range of laboratory animals, including rats, rabbits, cats, pigs and monkeys (summarised in(WHO, 1992; WHO, 2004). Long-term exposure to cadmium leads to renal tubular lesions with proteinuria, glucosuria and aminoaciduria, and to histopathological changes including damage to proximal renal tubules (WHO, 1992).

Cadmium produces low-molecular-mass proteinuria and renal tubular dysfunction in experimental animals that is analogous to the effect in humans (WHO, 1992).

The results of animal studies have provided valuable information about the mechanisms of cadmium-induced kidney damage and the significance of various biological indicators of exposure and effect. However, many animal studies have administered cadmium parenterally rather than in food or water, or have used inorganic forms of cadmium, such as cadmium chloride in drinking water. The different forms of cadmium and different routes of administration lead to different patterns of cadmium absorption and distribution, for example, cadmium chloride distributes preferentially to the liver rather than the kidney (WHO, 1992). Thus, while the animal data support the findings in humans and some provide similar threshold levels of cadmium in the kidney cortex, the data are not suitable for estimating safe intake levels. In addition, the efficiency of the rat liver in detoxifying many chemicals often results in much higher NOEL values than found in human studies. For example, a number of animal studies reviewed by JECFA at their 55th meeting (WHO, 2001) suggest a LOEL of 13-15 mg/kg bw/day and a NOEL of 0.8 mg/kg bw/day, values well above the PTWI, equivalent to 0.001 mg/kg bw/day. Therefore, available human data are better suited to estimating safe levels for human dietary intake.

3.2.3 Effects in humans

Cadmium is capable of causing renal effects, bone effects, pulmonary effects, cardiovascular effects and cancer. In relation to cadmium exposure via food, the only relevant effects are renal and bone effects. The pulmonary effect which has been reported in humans is chronic obstructive airway disease in a number of cadmium workers. Cardiovascular effects such as high blood pressure and effects on the myocardium have been observed in animal studies only. Cadmium chloride, sulphate and oxide fumes and dust have been shown to cause cancer in animals in inhalation studies and there is some evidence that occupational exposure to cadmium may contribute to the development of cancer of the lung in humans.

Of greatest relevance to human health following long-term dietary exposure are the effects on the kidney, characterised by tubular dysfunction and tubular cell dysfunction, although glomerular dysfunction may also occur. Daily cadmium intakes in food of 140-260 □g/day for more than 50 years have produced an increase in renal tubular dysfunction in some exposed populations (WHO, 1992). Cadmium-induced renal tubular dysfunction can lead to impaired re-absorption of proteins, glucose and amino acids.

A characteristic sign of tubular dysfunction is an increased excretion of low molecular weight proteins in urine (tubular proteinuria) thought to be due mainly to a decreased tubular re-absorption capacity. In the absence of acute exposure to cadmium, this may serve as an indicator of a renal effect. A number of biomarkers of effect are used in studies of renal toxicity, including urinary levels of human complex-forming glycoprotein (protein HC, also called α_1-microglobulin), β_2-microglobulin, alanine aminopeptidase, albumin and NAG. In the assessment of tubular proteinuria, protein HC is regarded as preferable to β2-microglobulin as it is more stable at the typical pH of urine (WHO, 2004). However, these biomarkers of effect are not necessarily specific for cadmium exposure (ATSDR, 1999).

A consequence of renal tubular dysfunction is a disturbance of calcium and vitamin D metabolism. This may lead to osteomalacia and/or osteoporosis, but a direct effect of cadmium on bone mineralisation has not been excluded.

Cadmium complexed with metallothionein is non-toxic when stored within cells. Cadmium toxicity occurs when levels in the kidney exceed a critical concentration and surpass the ability of metallothionein to sequester cadmium. Nephrotoxicity is thought to be related to non-metallothionein-bound cadmium. It is also possible that cadmium-metallothionein excreted by the kidney nephron may injure renal cells. Or that excretion of hepatic cadmium and its presence in blood can result in preferential accumulation of cadmium in the kidneys.

In industry, exposure to cadmium, primarily through inhalation, has resulted in renal dysfunction, proteinuria, renal glucosuria and aminoaciduria. In advanced cases there is a combination of tubular and glomerular effects. In early cases, only proteinuria is evident. Proteinuria is sometimes reversible, but may persist for years after cessation of exposure. High exposure levels can lead to hypercalciuria, phosphaturia and polyuria. Hypercalciuria may cause disturbances in bone and calcium metabolism and leads to renal stone formation in some workers. Occupational exposure to cadmium has also been linked to cases of osteomalacia (WHO, 1992). Workers exposed to cadmium oxide dust displaying proteinuria and morphological kidney changes had kidney concentrations of cadmium ranging from 20-120 μg/g wet weight at autopsy. Workers without morphological changes and no or slight proteinuria had kidney cadmium concentrations of 180-450 μg/g wet weight. This result is consistent with increased excretion of cadmium from the more severely damaged kidneys, also found in animal studies (WHO, 1992).

This report concentrates on the effects on humans of long term dietary exposure to cadmium on kidney and bone.

3.2.4 Long term effects on humans of cadmium in the environment

A number of epidemiological studies, particularly in Japan, Belgium and Sweden, have investigated the prevalence of kidney toxicity caused by cadmium and found elevated incidences of tubular proteinuria in cadmium-polluted areas. Studies in non-polluted areas have also reported a correlation between cadmium body burden and increased incidence of both kidney and bone effects. In particular, recent studies in Belgium and Sweden investigating the health effects of environmental, predominantly dietary, exposure to cadmium are likely to be particularly relevant to this assessment, as diet and nutritional status of the population are likely to be similar to the populations of Australia and New Zealand.

3.2.4.1 Japan health surveys

Excessive ingestion of cadmium in the Jinzu river basin (Toyama Prefecture) of Japan resulted in Itai Itai disease, characterised by severe osteomalacia (inadequate mineralisation of the bone matrix) and osteoporosis leading to multiple fractures, as well as renal tubular dysfunction. Cadmium levels in rice were elevated as water polluted by a zinc mine was used for irrigation of rice fields. Polluted water was also used as drinking water. This sensitive population was noted to have a diet low in both protein and calcium. Most reported cases occurred in women over 40 years of age who had borne multiple children, so are likely to have been iron deficient.

In Japanese cadmium-polluted areas, signs of renal dysfunction have been found, with between 30-80% of exposed people in one area showing proteinuria and glucosuria. While some data indicates that mild cases of renal dysfunction among young individuals are reversible, other studies have not demonstrated reduced proteinuria following cessation of exposure.

In Japan, environmental exposure to cadmium has also been associated with bone disease and abnormalities of calcium metabolism. It has been proposed that cadmium accumulation in the proximal tubular cells depresses production of 1,25-dihydroxy-vitamin D3 in the mitochondria of these cells, leading to decreased calcium absorption and decreased mineralisation of the bone, in turn resulting in osteomalacia (WHO, 1992).

A number of health surveys in contaminated areas of Japan were conducted following the recognition of the association of cadmium exposure to Itai Itai disease. The studies were originally undertaken to identify cases of Itai Itai disease, but also made estimates of the prevalence of proteinuria and glucosuria and cadmium exposure. It was estimated that an average of about 50%, and up to 85%, of daily cadmium intake came from rice.

From 1976 to 1984, epidemiological health surveys of residents in areas with environmental cadmium pollution included more than 13,000 inhabitants of polluted areas and more than 7000 inhabitants of non-polluted areas, aged 50 years or more. The emphasis of the later surveys was on detection of low molecular weight proteinuria as early evidence of tubular dysfunction. Following an initial semi-quantitative screen of urine for proteinuria and/or glucosuria, urine was further analysed for β2-microglobulin, RBP, lysozyme, total amino acid nitrogen and cadmium. Participants whose test levels exceeded a defined level in any one item for tested for renal function by urine and blood analysis. Participants with a tubular re-absorption of phosphate (TRP) less than 80% underwent a detailed health examination, including skeletal radiography.

In seven of eight surveyed prefectures (Akita, Fukushisuma, Gunma, Toyama, Ishikawa, Hyogo and Nagasaki) the number of individuals with or suspected of having proximal renal tubular dysfunction or related findings tended to be greater in the polluted areas than the non-polluted areas, and was often significantly related to the degree of pollution and the levels of cadmium in rice. The 1992 IPCS monograph (WHO, 1992) reported that ‘this suggests that environmental cadmium pollution is associated with the occurrence of proximal renal tubular dysfunction.’ Five of the areas in which significantly increased prevalence of β2-microglobulinuria was found were reviewed in detail (WHO, 1992) and are summarised in Table 1.

Table 1: Age-adjusted prevalence rate (%) of renal tubular dysfunction and related conditions[a] (taken from WHO, 1992)

Prefecture studied[b]	Year of Investi-gation	Polluted (P) or non-polluted (NP) area[c]	No. of examinees		ß2-Micro-globulinuria (> 10 mg/litre)		Decreased TRP (< 80%)		Tubular dysfunction		Cadmium concentration in rice (mg/kg fresh weight) [f]	Daily cadmium intake (µg/day)
			male	female	male	female	male	female	male	female		
Toyama (Fuchu area)	1979-1984	P NP	3432 944	4099 1205	6.5 0.4[d]	10.8 0.5[d]	4.6 0.6[d]	5.5 0.2[d]	1.4 0.0[d]	3.3 0.0[d]	0.6-2.0	600
Hyogo (Ikuno area)	1977	P NP	230 212	280 251	12.8 2.7[d]	16.8 1.9[d]	4.4 0.9[e]	6.8 0.4[d]	2.0 0.0	3.5 0.0[e]	0.2-1.0	
Ishikawa (Kakehashi area)	1976	P NP	260 200	306 275	7.6 1.1[d]	10.9 1.5[d]	6.9 0.5[d]	5.7 0.0[d]	2.4 0.0	3.2 0.0[d]	0.2-0.8	160
Akita (Kosaka area)	1976	P NP	179 168	247 234	6.4 0.0[d]	5.0 0.0[d]	2.3 0.0	0.7 0.0	0.0 0.0	0.0 0.0	0.2-0.6	185
Nagasaki (Tsushima area)	1976	P NP	143 210	191 291	3.4 1.9	10.6 0.3[d]	4.6 1.5	9.1 0.0[d]	1.7 0.0	6.2 0.0[d]	0.5-0.8	213-255

[a] From: Japan Cadmium Research Committee (1989). The response rates for these studies were greater than 90% of the target population. The age composition (50-59, 60-69, 70-79, and 80+) in each prefecture was adjusted to the Japanese population in 1980. The criteria for renal tubular dysfunction were: one out of three signs (low molecular weight proteinuria, glucosuria, and generalized aminoaciduria), %TRP < 80% and acidosis (blood hydrogen carbonate below 23 mEq/litre).

[b] The total number of people living in polluted areas in each prefecture is shown in Table 7.

[c] Total number of people examined was 5657 males and 6902 females in polluted areas and 2782 males and 3653 females in non-polluted areas.

[d] Significant difference ($P < 0.01$)

[e] Significant difference ($P < 0.05$)

[f] Derived from separate studies, as cited in WHO (1992)

3.2.4.2 Japan - recent studies in previously contaminated areas

The recent Japanese Multicentered Environmental Toxicant Study (JMETS) of female farmers from five districts in Japan with varying cadmium exposure levels concluded that women with dietary exposure to cadmium at a level close to the current PTWI did not show any excess development of renal tubular dysfunction (Horiguchi *et al*., 2004b). Four districts (B-D) were selected as cadmium polluted, based on government monitoring data showing relatively high cadmium contamination of rice. One district (A) where no highly contaminated rice had been detected was selected as a control. The study population comprised 1381 female farmers who had consumed rice and vegetables grown in the own fields and locally produced foods from birth. Participants provided information about their health status and completed a self-administered diet history questionnaire to determine each participant's usual intake levels of rice and miso (a bean paste made from rice and soybeans). The cadmium content of the foods was determined from small amounts of polished rice and miso provided by the participants. Blood and urine samples were taken for determination of blood and urinary cadmium levels, and urinary protein HC, β2-microglobulin and creatinine levels. Exclusion criteria included past or current smoking, chronic renal failure, history of collagen disease or evidence of inflammatory disease. Participants included diabetic individuals and anaemics.

The cadmium levels in rice in the five districts were used to establish a gradient of cadmium pollution (A<B=C<D<E). Individual daily intake of cadmium from rice and miso was calculated and used to estimate total daily cadmium intake based on the average Japanese intake of cadmium cited in the total diet study (conducted by the National Institute of Health Sciences of Japan in 2001). The regional sequence of dietary cadmium exposure was the same as that established for regional cadmium pollution (A<B=C<D<E). Cadmium intake in reference group A was as low as that of the general Japanese population. The average weekly cadmium intake in district E was 5.7-6.7 μg/kg, a value very close to the PTWI of 7 μg/kg. Analysis of urinary cadmium levels revealed the pattern of area-dependent increase as cadmium pollution of rice and dietary exposure (A<B=C<D<E). However, there were no significant differences in urinary protein HC or β2-microglobulin excretion among the five districts nor area-dependent increases corresponding to cadmium exposure or cadmium body burden. The proportion of subjects with renal dysfunction did not show any regional or sequential difference. The authors conclude that the current PTWI is sufficient to prevent cadmium-induced renal dysfunction in the general population (Horiguchi *et al*., 2004b).

When group populations were subdivided by age (40-49, 50-59 and 69-69), age dependent increases were observed in both the levels of renal protein excretion and the proportion of renal dysfunction in almost all districts. Multiple regression models of the relationship between levels of urinary protein HC and β2-microglobulin and age or cadmium exposure showed much bigger regression coefficients for age than for blood cadmium or urinary cadmium, indicating that renal tubular function is more affected by age than by cadmium exposure at the levels observed in this study.

A further study of the same population investigated effects of cadmium exposure on forearm bone mineral density. Data was grouped by urinary cadmium concentration and age-related menstrual status.

The results suggest that cadmium accelerates the increase in urinary calcium excretion around the time of menopause and subsequent decreases in bone density. However, multivariate analyses found no significant contribution of cadmium to bone density or urinary calcium excretion, suggesting confounding factors affected the results. The authors conclude that exposure to cadmium at a level that is insufficient to induce renal dysfunction does not increase the risk of osteoporosis (Horiguchi *et al*., 2005).

3.2.4.3 Belgium

The CadmiBel (Cadmium in Belgium) study was a cross-sectional, population based epidemiological study conducted between 1985 and 1989 on the effects of long-term exposure to low doses of cadmium on human health (Buchet *et al*., 1990; Lauwerys *et al*., 1990). The study provides data on a variety of health end-points and was described in detail in the report of the 55th meeting of JECFA (WHO, 2001).

The 2,327 participants constituted a random sample of the population of four Belgian districts, chosen in order to provide a wide range of environmental exposure to cadmium. Study subjects within each area were chosen to include equal numbers of men and women and equal numbers of individuals in the age groups 20-39, 40-59 and 60-79 years. Participants completed a questionnaire covering medical and occupational histories, smoking, alcohol and diet. A 24-hour urine sample was collected, as well as a spot urine sample and a venous blood sample. A series of biomarkers were analysed. Blood was tested for cadmium, lead, zinc, protoporphyrin and selenium. Serum was analysed for creatinine, alkaline phosphatase, gamma-glutamyltranspeptidase, cholesterol, calcium, ferritin and β2-microglobulin. The 24-hour urine sample was tested for creatinine, cadmium, calcium, total amino acids, total protein, β2-microglobulin, retinol-binding protein, albumin and N-acetyl-β-glucosaminidase. The spot urine sample was tested for creatinine, cadmium and β2-microglobulin.

The urinary concentration of cadmium was significantly associated with five biomarkers of renal function (β2-microglobulin, retinol-binding protein, N-acetyl-β-glucosaminidase, amino acids and calcium) in 1699 persons. In multivariate models, all variables except β2-microglobulin were also associated with the cadmium concentration in the 24 hour urine sample, after adjustment for age, sex, smoking, diuresis, diabetes and other factors (WHO, 2001). Dose-response relationships were found. For two biomarkers (N-acetyl-β-glucosaminidase and β2-microglobulin) the interaction between a diagnosis of diabetes and urinary cadmium excretion was statistically significant, suggesting that diabetics may be particularly vulnerable to the adverse effects of cadmium on renal function.

The findings suggest that an increased body burden of cadmium is associated with changes in proximal tubular function. The dose-response analyses suggest that these changes largely occurred when urinary excretion of cadmium exceeded 2 μg/24 hours (or 0.5-2 μg/g of creatinine). This rate is considered to reflect a renal cortical concentration of 10-40 mg/kg. It was estimated that 10% of the general population of Belgium has a body burden of cadmium associated with an excretion rate of 2 μg/24 hours, putting them at increased risk for renal changes, compared with a background prevalence of tubular proteinuria of 5% (WHO, 2001).

A 5-year follow up study of 593 Cadmibel participants found no evidence of progressive renal damage. The investigators concluded that the renal effects due to cadmium were weak, stable or even reversible after measures were taken to reduce exposure.

The Cadmibel Study also investigated the effect of environmental cadmium exposure on calcium metabolism by analysing three biomarkers of calcium metabolism in 1987 study participants (Staessen *et al*., 1991) summarised in (WHO, 2001).

Serum alkaline phosphatise activity and urinary calcium excretion correlated significantly and positively with urinary cadmium excretion in both men and women, and serum total calcium concentration correlated negatively with urinary cadmium excretion, but was significant in men only. The study authors conclude the results suggest that calcium metabolism is gradually affected as cadmium accumulates in the body, even at environmental exposure levels. However, in evaluating the study, JECFA report that, while statistically significant, cadmium excretion accounts for only a small amount of variation in the biomarkers of calcium metabolism (1-2%) and that the clinical significance of the changes is uncertain (WHO, 2001).

In a follow-up to the Cadmibel study, the Public Health and Environmental Exposure to Cadmium (PheeCad) project undertook a prospective population study of whether moderate environmental exposure to cadmium is associated with low bone density and high risk of fractures (Staessen *et al*., 1999) summarised in (WHO, 2001). Chronic low-level exposure to cadmium may promote calcium loss via urinary excretion. Urinary cadmium excretion, a biomarker of lifetime exposure, was measured in people from ten districts of Belgium, of which six districts bordered on three zinc smelters. Cadmium in soil and in vegetables from the districts was also measured. Data on incidence of fractures was recorded and verified from medical records. Bone density was measured at the forearm just above the wrist by single photon absorptiometry, and calculated as the mean of six proximal and four distal scans. Median follow-up was 6.6 years. In post-menopausal women, a two-fold increase in urinary cadmium excretion correlated with 0.01 g/cm^2 decrease in bone density ($p<0.02$) and a 73% increase in the risk for fractures in women. This corresponds to a population attributable risk of fracture of 28.9% for women with above-median, age-adjusted internal cadmium exposure. The study authors concluded that, even environmental exposure to cadmium may promote skeletal demineralisation, which may lead to increased bone fragility and raised risk of fractures (Staessen *et al*., 1999).

In its evaluation, JECFA conclude that these findings suggest that environmental exposure to cadmium is associated with decreased bone density and an increased risk for fractures in women, possibly due to increasing calcium loss and bone resorption. Increased gastrointestinal absorption of cadmium, due to depleted iron stores, may be related to the apparent increased vulnerability of women (WHO, 2001).

3.2.4.4 Sweden

The OSCAR (OSteoporosis - CAdmium as a Risk factor) study in the south of Sweden involved people living near a nickel-cadmium battery plant and a nearby group of ‘environmentally-exposed’ people.

A positive, linear relationship was found between both urinary and blood concentrations of cadmium and protein HC in urine (p,0.0001 in men and p=0.0033 in women). Tubular proteinuria was defined on the basis of the 95th percentile of the concentration of protein HC in urine for a Swedish reference population (0.8 mg/g creatinine in men and 0.6 mg/g creatinine in women). The concentrations of urinary protein HC exceeded the cut-offs for defining tubular proteinuria for 171 people, and the prevalence of tubular proteinuria showed a clear dose-response relationship with urinary concentrations of cadmium (Jarup *et al*., 2000) summarised in(WHO, 2004). Table 2 summarises the prevalence and odds ratios (OR) for tubular proteinuria associated with different ranges of concentration of urinary cadmium for the whole cohort (n=1021) and for the environmentally exposed subgroup (Jarup *et al*., 2000).

Table 2: Tubular proteinuria (defined as excreting protein HC >0.8 and 0.6 mg/mmol creatinine, for men and women, respectively) in people exposed to cadmium. From Järup et al, 2000

Urinary Cd (nmol/mmol creatinine)	Mean urinary Cd (nmol/mmol creatinine)	Environmentally exposed people cases/total (prevalence %) (missing, n=17)	Occupationally exposed people cases/total (prevalence %) (missing, n=3)	Total population cases/total (prevalence %)	OR, total (95% CI) (test for trend, p<0.001)	OR, environmentally exposed people (95% CI) (test for trend, p=0.001)
<0.3	0.21	12/252 (4.8)	1/13 (7.7)	13/265 (4.9)	1.0	1.0
0.3–<0.5	0.38	33/243 (14)	4/30 (13)	37/273 (14)	1.8 (0.9 to 3.5)	2.5 (1.1 to 5.5)
0.5–<1	0.69	60/232 (26)	8/66 (12)	68/298 (23)	2.7 (1.4 to 5.3)	4.3 (1.9 to 11)
1–<2	1.4	22/53 (42)	10/55 (18)	32/108 (30)	3.6 (1.7 to 7.6)	7.5 (3.5 to 44)
2–<3	2.5	1/1 (100)	7/23 (30)	8/24 (33)	4.0 (1.4 to 12)	—
3–<5	3.8	0/1 (0)	7/20 (35)	7/21 (33)	4.7 (1.6 to 14)	—
>5	6.8	— (—)	6/12 (50)	6/12 (50)	6.0 (1.6 to 22)	—

A criticism of the study was that a higher concentration of cadmium in urine may reflect existing renal damage, complicating interpretation. This was addressed by analysis of blood cadmium levels, which may be a better estimate of dose when tubular proteinuria is already present (Alfven *et al*., 2002) summarised in (WHO, 2004). Again, a significant positive relationship was found between cadmium in blood and protein HC in both men and women, after adjusting for age, smoking and lead in blood. Individual results were stratified into five groups based on the concentration of cadmium in blood. This revealed a dose-response relationship between cadmium in blood and tubular proteinuria, with elevated risks for tubular proteinuria for the three groups with the higher concentrations of cadmium in blood (Alfven *et al*., 2002) summarised in (WHO, 2004).

The OSCAR study also measure bone mineral density at a distal site on the non-dominant forearm in study participants over 60 years old (Alfven *et al*., 2002) summarised in (WHO, 2004). After adjustment for age, body weight, concentration of blood lead and smoking, bone mineral density was negatively correlated with concentration of blood cadmium. The association was statistically significant for women, but not for men, and showed a dose response, as shown in Table 3.

Table 3: Logistic regression model for low BMD (z-score[a] < –1) including blood cadmium and smoking as categoric variables and weight as a continuous variable, for the subgroup older than 60 years.

Variable	OR	95% CI
Blood Cd < 5 nmol/L (mean 2.5)	1	—
Blood Cd ≥ 5 nmol/L and <10 nmol/L (mean 7.2)	2.0	1.1–3.9
Blood Cd ≥ 10 nmol/L (mean 21)	2.9	1.4–5.8
Smoking	0.82	0.46–1.5
Weight	0.96	0.94–0.98

[a] z-score is used as a measure of osteoporosis. A common definition of low bone mineral density is z-score < –1, which indicates one standard deviation below a sex- and age-standardized mean.

3.2.5 Studies of long term effects on humans available since last JECFA evaluation

3.2.5.1 Sweden

The Women's Health in the Lund Area (WHILA) population based study in Sweden was extended to include an assessment of the possible effects of cadmium in populations residing in areas with no known historical cadmium contamination. The aim of the first study was to assess the association between cadmium concentrations in blood and urine and a series of markers of tubular and glomerular function (Akesson *et al*., 2005). This study included 820 women between the ages of 54 and 63. Participants provided morning first-voided urine (n=813) and blood (n=742) samples in 1999-2000. Levels of cadmium in blood were determined as a measure of ongoing exposure, and levels in urine as a measure of body burden. The effect markers measured were cystatin C in serum (to calculate glomerular filtration rate (GFR)), creatinine clearance (marker of glomerular function), protein HC, α1-microglobulin, NAG and calcium in urine (markers of tubular damage). Data on smoking and various health and physical characteristics were collected. The group of 'ever smokers' had 90% higher cadmium concentrations in blood and 40% higher in urine compared with 'never smokers'.

In univariate analysis, cadmium in both blood and urine was associated with all kidney effect markers except serum creatinine and urinary calcium. After controlling for confounders and adjusting for other co-variables, cadmium in urine remained significantly associated with GFR, creatinine clearance, protein HC and NAG. Similar results were obtained for blood cadmium. In a separate analysis of 'never smokers', cadmium remained associated with protein HC and NAG, and, for blood cadmium only, creatinine clearance. Protein HC, NAG and creatinine clearance (after adjustment for blood lead) differed significantly between the group with the lowest exposure level (urinary cadmium,<0.5 μg/L; mean, 0.36 μg cadmium/L = 0.48 μg cadmium/g creatinine) and the next lowest exposure level (0.50-0.75 μg/L; mean, 0.61 μg cadmium/L = 0.79 μg cadmium/g creatinine). This allowed calculation of a lowest observed effect level of 0.6 μg cadmium/L (0.8 μg/g creatinine), corresponding to approximately 20 μg cadmium/g kidney cortex. This is lower than the LOEL in previous studies. The study authors postulate this may be due to the homogeneity of the study population, the absence of 'healthy worker' effects and good analytical precision for exposure and effect markers.

The authors note that only a few percent of the variances in nephrotoxic effects were explained by cadmium levels, but that they concern a large segment of the population worldwide so are of public health concern (Akesson *et al*., 2005).

The WHILA study also included a study to assess associations between cadmium retention and bone effects in the same population of upper middle age women, considered the most susceptible part of the population for both cadmium retention and osteoporosis (Akesson *et al*., 2006). Study participants were aged between 54 and 63. Data was collected on lifetime smoking, alcohol consumption, physical activity and reproductive factors including hormone replacement therapy (HRT). Exposure assessment was based on cadmium in blood (n=727) as a measure of ongoing exposure, and cadmium in morning spot urine (n=797) as a measure of body burden. Indicators of short-term effects on bone status included markers of bone metabolism; parathyroid hormone (PTH) osteocalcin, bone alkaline phosphatise (bALP in serum, and deoxypyridinoline (U-DPD) and calcium in urine. Bone mineral density (BMD) of the non-dominant wrist was measured as a marker of long term effects on bone status. Kidney-effect markers were also measured, as described above.

All urinary markers were adjusted to the group mean urinary density (1.015 g/ml) rather than urinary creatinine, as creatinine excretion is dependent on muscle mass, which may in turn predict BMD. Consistent with results of the earlier study, current smokers had average blood cadmium concentrations three times higher than never-smokers, and urine cadmium levels 1.5 times higher than never-smokers (Akesson *et al*., 2006).

Associations were evaluated in multiple linear regression analysis. Possible confounders or effect modifiers considered were weight, menopausal status, use of hormone replacement therapy, age at menarche, alcohol consumption, smoking history, and physical activity. After multivariate adjustment, BMD, parathyroid hormone, and U-DPD were adversely associated with concentrations of urinary cadmium ($p < 0.05$) in all subjects. These associations persisted in the group of never-smokers, which had the lowest cadmium exposure (mainly dietary). For U-DPD, there was a significant interaction between cadmium and menopause ($p = 0.022$). The results suggest negative effects of low-level cadmium exposure on bone, possibly exerted via increased bone resorption, which seemed to be intensified after menopause. Based on the prevalence of osteoporosis and the low level of exposure, the authors consider that the observed effects, although slight, should be considered as early signals of potentially more adverse health effects. The authors conclude that 'the overall role of cadmium in the aetiology of osteoporosis is limited. The observed difference in BMD between high and low-exposed individuals corresponded to that of a 6-year increase in age or an 11-kg lower body weight' (Akesson *et al*., 2006).

3.3 Dose Response Assessment

The critical organ in long-term exposure to low concentrations of cadmium is the kidney (WHO, 1992). The critical effect is renal tubular dysfunction, most often manifested as low molecular mass proteinuria. In assessing dose-response relationships, the cadmium concentration in the kidney cortex is of prime importance (WHO, 1992).

3.3.1 Threshold Approaches

Appropriate human data have been difficult to obtain since data from biopsies or autopsies have only provided cross-sectional information i.e. renal cadmium concentrations and effects were measured simultaneously. Since cadmium is lost from the kidney when damage progresses, information on the critical concentration of cadmium has been difficult to obtain. *In vivo* neutron activation analysis offers a new method of obtaining longitudinal information but studies to date are limited. In exposed workers, the critical kidney concentration for renal tubular dysfunction has been estimated by a graphical extrapolation method to be 319 mg/kg tissue, but there is considerable individual variation and the 95% tolerance (corresponding to a confidence interval) is in the range +/- 90 mg/kg from the mean. Another study, using similar assumptions, has reported a critical value of 332 mg/kg, with 10% of workers having a peak cadmium level of 216 mg/kg tissue.

Two models were employed by the International Programme on Chemical Safety to estimate dose-response relationships to (WHO, 1992). One begins with the critical concentration in the kidney and employs a metabolic model to calculate the exposure that is required to reach a critical concentration. The other employs epidemiological evidence from industry and the general environment to study associations between exposure and response.

The average critical renal concentration in groups of exposed workers varied from 200 to 320 mg/kg wet weight. A renal cortex level of cadmium of about 200 mg/kg wet weight is regarded as a 'critical level' where it can be expected that sensitive members of a population may have signs of renal dysfunction (WHO, 1972). Using the metabolic model method, cadmium in the kidney is assumed to accumulate according to a one-compartment model, and a third or quarter of the body burden of cadmium is assumed to be in the kidney. Using this model, the average daily intake that would give rise to an average concentration of 200 mg/kg wet weight in the kidney cortex at age 50 would be 260-480 μg/day, assuming 5% gastrointestinal absorption. Assuming 10% absorption, the intake needed would be 140-260 μg/day (WHO, 2001).

With regard to using epidemiological evidence, the first method, using industrial data are relevant only to inhalation exposure. The epidemiological data based on environmental exposure are generally based on studies from Japan where high cadmium exposure was found in certain areas as a result of consumption of contaminated rice and possibly drinking water. Using the most sensitive method for diagnosis of low molecular weight proteinuria (β2-microglobulin excretion), there appears to be an association between cadmium exposure and increased excretion of low molecular weight proteins among some people over 50 years of age at a daily intake of about 140-260 μg cadmium or a cumulative cadmium intake of about 2000 mg or more (WHO, 1992). The IPCS reached this conclusion after taking into account epidemiological data from Japanese health surveys in five prefectures (including (Nogawa *et al*., 1989) and three European polluted areas.

The values derived from calculations based on a metabolic model and critical concentrations are in close agreement with the levels at which effects have been observed in epidemiological studies.

In a meta-analysis, Jarup et al (Jarup et al., 1998) reviewed the literature on exposure to cadmium and renal dysfunction and concluded that the concentration of cadmium in the kidneys should be maintained below 50 mg/kg and cadmium in the urine below 2.5 μg/g creatinine in order to prevent renal tubular damage that can proceed to clinical disease (cited in WHO, 2001).

3.3.2 Benchmark dose approaches

A number of recent publications have applied a benchmark dose approach to the epidemiological data obtained in Japan, Belgium and Sweden. The benchmark dose (BMD) corresponds to the exposure that results in a certain change in response compared to background. The lower confidence limit (usually 95% or 90%) of the BMD (BMDL) that corresponds to that predetermined increase has been suggested to replace the NOAEL. An advantage of the BMD approach as an alternative to a NOAEL is in taking account of information from the entire dose response curve.

The 1981-1982 results from the health survey conducted among the entire population over 50 years of age in the cadmium polluted region of the Kakehashi River basin of Japan were used to estimate a BMDL (Shimizu *et al*., 2006). The study population comprised 3103 participants in cadmium polluted areas (1397 men and 1706 women) and 289 participants inhabiting non-polluted areas (130 men and 159 women). A control group, comprising 424 men and 1611 women non smokers 50 years old or older inhabiting non-polluted areas, were used to calculate the cut-off values for β2-microglobulinuria corresponding to the 84% and 95% upper limits.

Using these cut-off values, the BMDL10 for urinary cadmium at which the excess risk is 0.05 was determined to be 2.9-4.0 μg/g creatinine for males and 1.5-3.6 μg/g creatinine for females.

Two other studies have estimated BMDL for urinary cadmium in non-polluted regions in Japan. One study (Uno *et al*., 2005) used β2-microglobulin and NAG as indicators of renal dysfunction and urinary cadmium as an indicator of cadmium exposure in urine samples from 828 participants (410 men and 418 women aged 40-59 years) living in areas without known environmental cadmium pollution. The lower 95% confidence limit of the dose corresponding to a 5% (BMDL5) or 10% (BMDL10) level of each indicator of renal dysfunction above background level was calculated. The BMDL10 was 0.6-1.2 μg/g creatinine for men and 1.2-3.6 μg/g creatinine for the women. [According to Suwazono et al, 2006, the BMDL5 for men was 0.3-0.6 μg/g creatinine and BMDL5 for women was 0.6-1.8 μg/g creatinine.]

In another study, (Kobayashi *et al*., 2006) estimated the threshold levels of urinary Cd using the benchmark dose approach using data from 2778 inhabitants (1114 men and 1664 women aged 50 years, living in non-polluted areas of Japan). Urinary cadmium excretion was divided into 10 categories and an abnormality rate was calculated for each. Cut-off values for indicators of renal dysfunction (β2-microglobulin and NAG) were defined as corresponding to the 84% and 95% upper limit values of the target population who have not smoked. The BMD and BMDL were calculated using a log-logistic model. The BMDL5 for the 84% cut-off value of β2-microglobulin was 2.4 μg/g creatinine in men and 3.3 μg/g creatinine in women.

A study of urinary cadmium, NAG and protein HC in 790 Swedish women 53-64 years of age was used to calculate BMDs for renal effects of cadmium. BMDs corresponding to an additional risk (benchmark response) of 5% or 10%, and their 95% lower confidence bounds (BMDLs) were calculated (the background risk at zero exposure was set to 5%). For both NAG and protein HC, the BMDs (BMDLs) for urinary cadmium were 0.6-1.1 (0.5-0.8) μg/g creatinine (Suwazono *et al*., 2006).

4 Evaluation

4.1 JECFA

JECFA first considered cadmium in 1972 (WHO, 1972). JECFA set a PTWI as 400-500 μg/person. Available data on human renal concentrations and daily intake was used as the basis for estimating a critical cadmium level in renal cortex of 200 mg/kg wet weight. JECFA considered that 'in order that levels of cadmium in the kidney will not exceed 50 mg/kg, and assuming an absorption rate of 5% and a daily excretion of only 0.005% of the body load… total intake should not exceed about 1 □g/kg body weight per day.'

The evaluation conducted by JECFA (WHO, 1989) identified the kidney as the critical organ in relation to chronic exposure to relatively low levels of cadmium. JECFA recognised that the critical concentration of cadmium at which renal injury occurs is subject to individual variation and the Population Critical Concentration (PCC) has been applied in relation to a specific response rate. An increase in low molecular weight proteinuria occurs in the elderly at intakes in the range 140-255 μg/day. While the pathological significance of this change is unclear, it has been used as an indicator of the threshold of a possible toxic effect and it was considered appropriate to set the PTWI on the basis of the dose-response data for this end-point.

It has been estimated on the basis of the limited data available that a PCC_{10} for the critical concentration in the kidney would be 200 mg/kg. Thus, a 10% prevalence rate for LMW proteinuria would be estimated to occur after a dietary intake of 175 μg cadmium per day for 50 years.

JECFA considered that for the levels of cadmium in the renal cortex to not exceed 50 mg/kg, and that, assuming an absorption rate of 5%, and a daily excretion of 0.005% of the body burden, total intake should not exceed about 1 μg/kg bw /day continuously for 50 years. The provisional tolerable weekly intake for cadmium was therefore set at 7 μg/kg bw (equivalent to 1 μg/kg bw/day, or 70 μg/day for a 70 kg person)(WHO, 1989). JECFA noted that excursions above this figure may be tolerated provided they are not sustained for long periods of time and do not produce a significant increase in the integrated lifetime dose, and that the recommended PTWI does, in fact, take into account the higher cadmium intake on a bodyweight basis by infants and children.

At its 41st meeting in 1993 (WHO, 1993), JECFA maintained the PTWI for cadmium at 7 μg/kg bw. The IPCS monograph, produced in 1992, provides a detailed description of the models on which the PTWI is based and the various assumptions used in their construction (WHO, 1992).

Specifically, a model based on the critical concentration of cadmium in the kidney and a metabolic model to calculate the exposure that is required to reach that critical concentration, in combination with epidemiological evidence from health surveys in polluted environments including five Japanese prefectures and three European areas to study associations between exposure and response.

JECFA reconsidered cadmium at its 55th meeting in 2000 (WHO, 2001) and 61st meeting in 2003 (WHO, 2004). Much of the information that had become available since the IPCS publication (WHO, 1992) was based on epidemiological studies in Belgium and Japan examining association of cadmium exposure with effects on kidney function and bone/calcium metabolism (described in the preceding section). At its 61st meeting, the Committee reaffirmed its conclusion that the critical health outcome is renal tubular dysfunction. While acknowledging that some results indicated that changes in renal function and calcium metabolism are observed at urinary cadmium concentration of <2.5 μg/g creatinine, the Committee considered that appreciable uncertainty remained about the long term significance of those changes. The Committee concluded that the new data do not provide a sufficient basis for revising the PTWI. The Committee reaffirmed its conclusion that an excess prevalence of renal tubular dysfunction would not be expected to occur at the current PTWI / if urinary cadmium concentration remained <2.5 μg/g creatinine (WHO, 2004).

The safe intake level derived by JECFA is based on biomarkers of absorption and effect, and a model comprising a cadmium absorption rate of 5%, and a daily excretion of 0.005% of the body burden. Recognising that the absorption rate of cadmium is influenced by the food matrix and the nutritional status of the individual, JECFA have considered the effect on the model of different gastrointestinal absorption rates (WHO, 1992). Assuming a 10% absorption rate, the intake needed to reach the critical concentration in the kidney cortex of 200 mg/kg at age 50 would be 140-260 μg per day. For populations where it is reasonable to assume that 10% of dietary cadmium is available, the model predicts that the current PTWI would not result in an increased prevalence of renal tubular dysfunction (WHO 2001).

JECFA will revisit the evaluation of cadmium on completion of current projects by the Joint FAO/WHO Project to Update the Principles and Methods for the Risk Assessment of Chemicals in Food on the dose-response assessment of biomarkers of effect and their relationship to disease outcome, and the possible specification of longer tolerable intake periods (e.g. PTMI) for contaminants with longer biological half lives.

4.2 ATSDR

The ATSDR (1999) derived a ‘minimal risk level’ (MRL) for chronic oral exposure to cadmium. The ATSDR oral MRL is based on a threshold associated with an increased incidence of proteinuria identified in residents of cadmium polluted areas of Japan (Nogawa *et al*., 1989). Subjects were 1850 (878 male and 972 female) cadmium exposed and 294 (133 male and 161 female) non-exposed inhabitants of the Kakchashi River basin in Ishikawa Prefecture. A regression equation relating total cadmium intake to prevalence of β 2-microglobulinuria was derived. For both sexes, the regression equation gave a prevalence of β2-microglobulinuria equal to controls at a lifetime cadmium intake of 2000 mg.

The ATSDR assessment incorporated an uncertainty factor of 10 for variability in the human population. This uncertainty factor was considered appropriate to account for possible increased sensitivity demonstrated by the Cadmibel study (Buchet *et al*., 1990), which suggested that, for the general population, a urinary cadmium excretion of <2 μg/24 hours would result in a low risk of renal effects. The Cadmibel authors estimated that this cadmium excretion rate would correspond to a mean renal cortex concentration of about 50 mg/kg. The ATSDR acknowledge that this may result from the use of a more sensitive cutoff for β2-microglobulinuria in the Cadmibel study than other studies. Incorporating an uncertainty factor of 10 resulted in an MRL of 0.0002 mg/kg/day, derived from a NOAEL of 0.0021 mg/kg/day.' This is equivalent to a NOAEL of 14 μg/kg bw/week and a weekly MRL of 1.4 μg/kg bw.

4.3 Comparison of safe intake levels

The safety limits for dietary exposure to cadmium set by JECFA and the ATSDR are both based on a lifetime accumulation of cadmium set at a threshold level of 2,000 mg of cadmium from dietary sources. However, the MRL set by the ATSDR (0.0002 mg/kg bw/day equivalent to 1.4 μg/kg bw/week) is one-fifth of the PTWI set by JECFA (7 μg/kg bw/day). The JECFA limit effectively uses a safety factor of four, as the critical concentration in the kidney cortex was established as 200 mg/kg, and the JECFA PTWI is set to keep the kidney cortex concentration below 50 mg/kg. This safety factor seems reasonable since the PTWI is set based on human data incorporating a large numbers of subjects. In addition, JECFA have reviewed and confirmed the PTWI on several occasions after considering the results from numerous epidemiological studies including data from susceptible subpopulations (i.e. post-menopausal women with a diet low in iron, calcium and total protein, and diabetics). The ATSDR uncertainty factor of ten may be unduly conservative.

4.4 Limitations of the assessment

The PTWI is based on human epidemiological data from areas where there has been known high cadmium dietary intake using a conservative dose-response analysis and supported by experimental data from animal models. Such epidemiological evidence is difficult to interpret particularly when the prevalence of kidney disease increases with age in both polluted and non-polluted areas.

There are also limitations in estimating actual exposure to cadmium, often relying on retrospective information and diet questionnaires to estimate cadmium intake. It is also not possible to have a non-exposed group as a control in the strict sense, as all populations are exposed to dietary cadmium.

The PTWI is based on the most sensitive parameter for kidney damage, namely, an increase in the urinary excretion of low molecular weight protein as a result of reduced re-absorption in the renal tubules. The toxicological significance of this observed change with respect to kidney damage is still not established as it is clear that the excretion of low molecular weight proteins normally increases with age. It is also well recognised that increased levels of urinary biomarkers are not specific for cadmium induced damage, but are generic indicators of tubular dysfunction.

The critical effect level has been calculated to be 200 mg/kg in the kidney cortex, which JECFA estimated would not be reached if urinary cadmium levels did not exceed 2.5 μg/g creatinine. However, recent research suggests that effects on urinary biomarkers of exposure are seen at lower levels of cadmium exposure although the long term health implications of the increased levels remain unclear.

5 Risk Characterisation

Tubular dysfunction in the kidneys is the critical effect resulting from chronic exposure to cadmium. The PTWI for cadmium is currently set at 7 μg/kg bw. The PTWI is based on lifetime accumulation of cadmium and is considered to be protective of kidney function following exposure to cadmium.

The dietary exposure assessment indicates that peanuts are currently a minor contributor to overall dietary exposure to cadmium, even for high consumers.

The dietary exposure calculations for whole population groups are reflective of life-time exposure to cadmium in the diet and are consistent with the current PTWI which is based on life-time accumulation of cadmium in the kidneys. Modelling of total dietary exposure to cadmium from all dietary sources, including an increase in the ML for cadmium in peanuts to 0.5 mg/kg, indicated that Australian and New Zealand consumers would have dietary intakes of cadmium within safe limits (Table 4). The highest consumers (90th percentile) had intakes of 30-35% of the PTWI for the whole Australian population aged 2 years+ and for the New Zealand population aged 15 years+.

Table 4: Estimated dietary exposures to cadmium from all sources with peanuts assumed to contain 0.5 mg/kg, as a percentage of the PTWI

Country	Population group	Number of consumers of cadmium	Consumers♦ as a % of total respondents#	Mean all respondents	90th percentile consumers
				% PTWI*	
Australia	2 years+	13854/13856	100	15 - 20	30 - 35
	2-6 years	989/989	100	30 - 40	65 - 75
New Zealand	15 years+	4632/4635	100	15 - 20	30 - 35

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.
♦ Consumers only – This only includes the people who have consumed a food that contains cadmium.
* PTWI = 0.007 mg/kg bw

The exposure assessment indicated that the highest consumption estimated for Australian children aged 2-6 years was at 65-75% of the PTWI. Dietary modelling often identifies children as the sub-population most likely to have the highest exposure to various food components, partly because young children have relatively high food consumption levels when expressed on a body weight basis.

At its 33rd meeting, JECFA recognised that exposure to cadmium will not be uniform with age, and that the estimate of the PTWI takes into account the higher cadmium intake on a body weight basis by infants and children[15]. Higher intakes during childhood are unlikely to be maintained over the long-term. The exposure estimates for the whole population include data from all age groups including 2-6 year olds and are a more accurate estimate of lifetime exposure to cadmium.

The exposure estimates for all population groups are likely to be overestimates because the estimate assumes that all peanuts are contaminated with cadmium at 0.5 mg/kg which is extremely unlikely as the available data on cadmium levels in peanuts indicates that most peanuts will have cadmium concentrations well below this level. Also, the data used for modelling is a 24-h record which overestimates food consumption for consumers as they are unlikely to consume the same foods in the same quantities every day (the use of multiple day records tends to significantly reduce predicted high consumer exposure).

In conclusion, because all population groups have estimated dietary intakes well below the PTWI, and due to the conservative assumptions in the dietary exposure calculations, increasing the current ML for cadmium in peanuts from 0.1 to 0.5 mg/kg would not raise public health and safety concerns.

References

Akesson, A., Bjellerup, P., Lundh, T., Lidfeldt, J., Nerbrand, C., Samsioe, G., Skerfving, S. and Vahter, M. (2006) Cadmium-induced effects on bone in a population-based study of women. *Environ.Health Perspect.* 114(6):830-834.

Akesson, A., Lundh, T., Vahter, M., Bjellerup, P., Lidfeldt, J., Nerbrand, C., Samsioe, G., Stromberg, U. and Skerfving, S. (2005) Tubular and glomerular kidney effects in Swedish women with low environmental cadmium exposure. *Environ.Health Perspect.* 113(11):1627-1631.

Alfven, T., Jarup, L. and Elinder, C.G. (2002) Cadmium and lead in blood in relation to low bone mineral density and tubular proteinuria. *Environ.Health Perspect.* 110(7):699-702.

ATSDR (1999) *Toxicological Profile for Cadmium*. Agency for Toxic Substances and Disease Registry. U.S. Department of Health and Human Services, Public Health Service. http://www.atsdr.cdc.gov/toxprofiles/tp5.html.

Berglund, M., Akesson, A., Nermell, B. and Vahter, M. (1994) Intestinal absorption of dietary cadmium in women depends on body iron stores and fiber intake. *Environ.Health Perspect.* 102(12):1058-1066.

Buchet, J.P., Lauwerys, R., Roels, H., Bernard, A., Bruaux, P., Claeys, F., Ducoffre, G., De, P.P., Staessen, J., Amery, A. and . (1990) Renal effects of cadmium body burden of the general population. *Lancet* 336(8717):699-702.

[15] WHO. (1989) Cadmium. In: *Toxicological evaluation of certain food additives and contaminants. Thirty-third meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 24

Flanagan, P.R., McLellan, J.S., Haist, J., Cherian, G., Chamberlain, M.J. and Valberg, L.S. (1978) Increased dietary cadmium absorption in mice and human subjects with iron deficiency. *Gastroenterology* 74(5 Pt 1):841-846.

Horiguchi, H., Oguma, E., Sasaki, S., Miyamoto, K., Ikeda, Y., Machida, M. and Kayama, F. (2004a) Comprehensive study of the effects of age, iron deficiency, diabetes mellitus, and cadmium burden on dietary cadmium absorption in cadmium-exposed female Japanese farmers. *Toxicol.Appl Pharmacol.* 196(1):114-123.

Horiguchi, H., Oguma, E., Sasaki, S., Miyamoto, K., Ikeda, Y., Machida, M. and Kayama, F. (2004b) Dietary exposure to cadmium at close to the current provisional tolerable weekly intake does not affect renal function among female Japanese farmers. *Environ.Res.* 95(1):20-31.

Horiguchi, H., Oguma, E., Sasaki, S., Miyamoto, K., Ikeda, Y., Machida, M. and Kayama, F. (2005) Environmental exposure to cadmium at a level insufficient to induce renal tubular dysfunction does not affect bone density among female Japanese farmers. *Environ.Res.* 97(1):83-92.

Jarup, L., Berglund, M., Elinder, C.G., Nordberg, G. and Vahter, M. (1998) Health effects of cadmium exposure--a review of the literature and a risk estimate. *Scand.J Work Environ.Health* 24 Suppl 1:1-51.

Jarup, L., Hellstrom, L., Alfven, T., Carlsson, M.D., Grubb, A., Persson, B., Pettersson, C., Spang, G., Schutz, A. and Elinder, C.G. (2000) Low level exposure to cadmium and early kidney damage: the OSCAR study. *Occup.Environ.Med* 57(10):668-672.

Kobayashi, E., Suwazono, Y., Uetani, M., Inaba, T., Oishi, M., Kido, T., Nishijo, M., Nakagawa, H. and Nogawa, K. (2006) Estimation of benchmark dose as the threshold levels of urinary cadmium, based on excretion of total protein, beta2-microglobulin, and N-acetyl-beta-D-glucosaminidase in cadmium nonpolluted regions in Japan. *Environ.Res.* 101(3):401-406.

Lauwerys, R., Amery, A., Bernard, A., Bruaux, P., Buchet, J.P., Claeys, F., De, P.P., Ducoffre, G., Fagard, R., Lijnen, P., Nick, L., Roels, H., Rondia, D., Saint-Remy, A., Sartor, F. and Staessen, J. (1990) Health effects of environmental exposure to cadmium: objectives, design and organization of the Cadmibel Study: a cross-sectional morbidity study carried out in Belgium from 1985 to 1989. *Environ.Health Perspect.* 87:283-289.

Nogawa, K., Honda, R., Kido, T., Tsuritani, I., Yamada, Y., Ishizaki, M. and Yamaya, H. (1989) A dose-response analysis of cadmium in the general environment with special reference to total cadmium intake limit. *Environ.Res.* 48(1):7-16.

Shimizu, A., Kobayashi, E., Suwazono, Y., Uetani, M., Oishi, M., Inaba, T., Kido, T. and Nogawa, K. (2006) Estimation of benchmark doses for urinary cadmium based on beta2-microglobulin excretion in cadmium-polluted regions of the Kakehashi River basin, Japan. *Int J Environ.Health Res.* 16(5):329-337.

Staessen, J., Amery, A., Bernard, A., Bruaux, P., Buchet, J.P., Claeys, F., De, P.P., Ducoffre, G., Fagard, R., Lauwerys, R.R. and . (1991) Effects of exposure to cadmium on calcium metabolism: a population study. *Br.J Ind.Med* 48(10):710-714.

Staessen, J.A., Roels, H.A., Emelianov, D., Kuznetsova, T., Thijs, L., Vangronsveld, J. and Fagard, R. (1999) Environmental exposure to cadmium, forearm bone density, and risk of fractures: prospective population study. Public Health and Environmental Exposure to Cadmium (PheeCad) Study Group. *Lancet* 353(9159):1140-1144.

Suwazono, Y., Sand, S., Vahter, M., Filipsson, A.F., Skerfving, S., Lidfeldt, J. and Akesson, A. (2006) Benchmark dose for cadmium-induced renal effects in humans. *Environ.Health Perspect.* 114(7):1072-1076.

Tsukahara, T., Ezaki, T., Moriguchi, J., Furuki, K., Fukui, Y., Ukai, H., Okamoto, S., Sakurai, H. and Ikeda, M. (2003) No significant effect of iron deficiency on cadmium body burden or kidney dysfunction among women in the general population in Japan. *Int Arch.Occup.Environ.Health* 76(4):275-281.

Uno, T., Kobayashi, E., Suwazono, Y., Okubo, Y., Miura, K., Sakata, K., Okayama, A., Ueshima, H., Nakagawa, H. and Nogawa, K. (2005) Health effects of cadmium exposure in the general environment in Japan with special reference to the lower limit of the benchmark dose as the threshold level of urinary cadmium. *Scand.J Work Environ.Health* 31(4):307-315.

Vahter, M., Berglund, M., Nermell, B. and Akesson, A. (1996) Bioavailability of cadmium from shellfish and mixed diet in women. *Toxicol.Appl Pharmacol.* 136(2):332-341.

WHO. (1972) Cadmium. In: *Evaluation of mercury, lead, cadmium and the food additives amaranth, diethylpyrocarbonate and octyl gallate. Sixteenth meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 4.

WHO. (1989) Cadmium. In: *Toxicological evaluation of certain food additives and contaminants. Fifty-fifth meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 24.

WHO. (1992) Cadmium (Environmental Health Criteria 134). World Health Organisation, Geneva. http://www.inchem.org/documents/ehc/ehc/ehc134.htm.

WHO. (1993) Cadmium. In: *Evaluation of certain food additives and contaminants. Forty-first report of JECFA.* World Health Organization, Geneva. WHO Technical Report Series 837.

WHO. (2001) Cadmium. In: *Safety evaluation of certain food additives and contaminants. Fifty-fifth meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 46.

WHO. (2004) Cadmium. In: *Safety evaluation of certain food additives and contaminants. Sixty-first meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 52.

WHO. (2006) Cadmium - Impact assessment of different maximum limits. In: *Safety evaluation of certain contaminants in food. Sixty-fourth meeting of JECFA.* World Health Organization, Geneva. WHO Food Additive Series 55.

Attachment 3

Dietary Exposure Assessment Report

Executive Summary

A dietary exposure assessment was deemed necessary for Application A552 in order to estimate the potential dietary exposure to cadmium from peanuts and the overall diet for both the Australian and New Zealand populations should the current ML of 0.1 mg/kg be increased to 0.5 mg/kg as requested by the Applicant.

Dietary exposures to cadmium were calculated for the Australian and New Zealand populations and for the population sub-group Australian children 2-6 years. Scenarios considered for this Application were a *Baseline* dietary exposure scenario and a *Proposed Maximum Level (ML)* scenario. Both scenarios included foods representative of the overall diet as well as peanuts at a baseline level (0.04 mg/kg) and at a proposed ML of 0.5 mg/kg. Dietary exposure from *Peanuts Only* was also estimated to determine the specified population groups' dietary exposure to cadmium from peanuts.

The food consumption data used were from the 1995 Australian National Nutrition Survey (NNS) and the 1997 New Zealand NNS. The cadmium concentration data were from the Revised Dietary Exposure Assessment for Cadmium conducted in 2001 for both Australian and New Zealand populations (Food Standards Australia New Zealand, Unpublished). This revised assessment was conducted following the conclusion of Proposal P144 - Review of the Maximum Permitted Concentrations of Cadmium in Food that had based its risk assessment on 1983 and 1985 National Nutrition survey (NNS) data. In 2001, the 1995 Australian and 1997 New Zealand NNS data became available and were incorporated into the FSANZ dietary exposure assessment computer program, DIAMOND (DIetAry Modelling Of Nutritional Data), forming the basis of the revised 2001 assessment.

The estimated dietary exposures to cadmium were found to be below the reference health standard, the Provisional Tolerable Weekly Intake (PTWI) of 0.007 mg/kg bw/week, for both the Australian and New Zealand general population groups for all scenarios assessed. A cadmium concentration of peanuts at the proposed ML of 0.5 mg/kg did not have a large impact on the estimated dietary exposures to cadmium for any of the population groups assessed. Estimated dietary exposures as a percent of the PTWI were slightly higher when based on the proposed ML by between 5 to 15% of the PTWI across all population groups assessed. Estimated dietary exposures were highest for children aged 2-6 years where estimated dietary exposures at baseline were 50-60% of the PTWI at the 90th percentile dietary exposure and 65-75% of the PTWI at the 90th percentile dietary exposure when based on the proposed ML of 0.5 mg/kg.

Major food contributors to total cadmium dietary exposure for *Baseline* included potato for all Australians as well as cacao beans for those 2-6 years, and potato and mussels for New Zealand. These foods, together with peanuts, were also major contributors to total cadmium dietary exposure for the respective population groups for the *Proposed ML* scenario.

Under the FSANZ Science Strategy 2006-2009, FSANZ agreed to review its dietary modelling procedures. As part of this review an international peer review was sought. FSANZ has previously reported chronic dietary exposures for high consumers at the 95th percentile. The recommendation of the peer review by an international dietary exposure assessment expert from the US Food and Drug Administration was that FSANZ should consider aligning its reporting of food chemical dietary exposures with international best practice by reporting at the 90th percentile not the 95th percentile, if only one 24 hour recall record per person was used for the assessment[16]. This is because the 95th percentile results are likely to be an overestimate of dietary exposure on a daily basis over a life-time of exposure. Basing risk management decisions on the 95th percentile will potentially result in an overly conservative risk management approach.

1. Background

The Applicant is seeking to increase the ML for cadmium in peanuts from 0.1 mg/kg to 0.5 mg/kg to enable increased flexibility to source peanuts from a variety of countries for use in confectionery to meet demand that may result from crop seasonality and product quality. The Applicant states that at present, confectionery manufacturers are severely restricted in their choice of countries from which they can source peanuts because some may exceed the current ML for cadmium in the Code. The Applicant suggests that retaining the current ML for cadmium in peanuts could be perceived as a Technical Barrier to Trade because Codex and many other countries do not set a ML for cadmium in peanuts.

A dietary exposure assessment was deemed necessary in order to estimate the potential dietary exposure to cadmium from peanuts and the overall diet for both the Australian and New Zealand populations should the current ML of 0.1 mg/kg be increased to 0.5 mg/kg.

2. Dietary exposure assessments

2.1 What is dietary modelling?

Dietary modelling is a tool used to estimate exposures to food chemicals from the diet as part of the risk assessment process. To estimate dietary exposure to food chemicals records of what foods people have eaten are required as well as information on how much of the food chemical is in each food. The accuracy of these exposure estimates depend on the quality of the data used in the dietary models. Sometimes not all of the data required are available or there is uncertainty about the accuracy so assumptions are made, either about the foods eaten or about chemical levels, based on previous knowledge and experience. The models are generally set up according to international conventions for food chemical exposure estimates, however, each modelling process requires decisions to be made about how to set the model up and what assumptions to make; a different decision may result in a different answer. Therefore, FSANZ documents clearly all such decisions and model assumptions to enable the results to be understood in the context of the data available and so that risk managers can make informed decisions.

[16] Lambe, J., Kearney, J., Leclercrq, C., Berardi, D., Zunft,H., De Henauw, S., De Volder, M., Lamberg-Allardt, C., Karkkainen, M., Dunne, A. and Gibney, N. (2000) Enhancing the capacity of food consumption surveys of short duration to estimate long term consumer-only intakes by combination with a qualitative food frequency questionnaire. Food Additives and Contaminants, 17(3), pp. 177-187.

2.2 Previous dietary exposure estimates for cadmium

In 2001, FSANZ conducted a revised dietary exposure assessment for cadmium for both the Australian and New Zealand populations. This revised assessment was conducted following the conclusion of Proposal P144 - Review Of The Maximum Permitted Concentrations Of Cadmium In Food that had based its risk assessment on 1983 and 1985 National Dietary Survey (NDS) data. By 2001, the 1995 Australian and 1997 New Zealand NNS data had been incorporated into the FSANZ dietary exposure assessment computer program, DIAMOND, forming the basis of the 'Revised' 2001 assessment. The new data selection criteria introduced during the review of all other heavy metals in 1999-2000 (Proposal P157 - Review of heavy metals) were also applied in the 2001 revised assessment. This report's findings (Food Standards Australia New Zealand, Unpublished) indicated that dietary exposures to cadmium were well below the PTWI. More specifically, the Australian population's mean estimated dietary exposure to cadmium ranged between 13-16% of the PTWI, while New Zealand's mean estimated dietary exposures were 14-17% of the PTWI, these ranges relating to lower and upper bound estimates[17].

The exposure to cadmium from the Australian diet was also assessed in the 20th Australian Total Diet Survey (ATDS). This survey found estimated dietary exposures to cadmium to be below the tolerable limit of 0.007 mg/kg bw/week, with children 9 months to 2 years predicted to have the highest mean dietary exposure ranging between 13-68% of the PTWI due to their high food consumption relative to their body weight (Food Standards Australia New Zealand, 2002).

Similarly, cadmium dietary exposures were assessed for the New Zealand population in the 2003-2004 New Zealand Total Diet Survey with estimated dietary exposures to cadmium well below the PTWI for all population groups assessed. Children aged 1-6 years were predicted to have the highest dietary exposure for cadmium at 37% of the PTWI, while for adult age groups aged 19 years and over the estimated dietary exposures ranged between 22% and 29% of the PTWI (New Zealand Food Safety Authority, 2005).

2.3 Dietary exposure assessments for A552

While the Australian and New Zealand populations' estimated dietary exposure to cadmium appears to be well below the PTWI for cadmium based on previous estimates, the potential for any increased health risk were the ML for cadmium in peanuts to be increased from 0.1 to 0.5 mg/kg should nevertheless be assessed.

Dietary exposures for this application were estimated by combining usual patterns of food consumption, as derived from the 1995 and 1997 NNS data, with concentrations of cadmium in food, in addition to the proposed ML of 0.5 mg/kg of cadmium in peanuts. See Figure 1 for an overview of the dietary modelling approach.

The dietary exposure assessment to cadmium was conducted using dietary modelling techniques that combine food consumption data with food chemical concentration data to estimate the exposure to the food chemical from the diet, using FSANZ's dietary modelling computer program, DIAMOND.

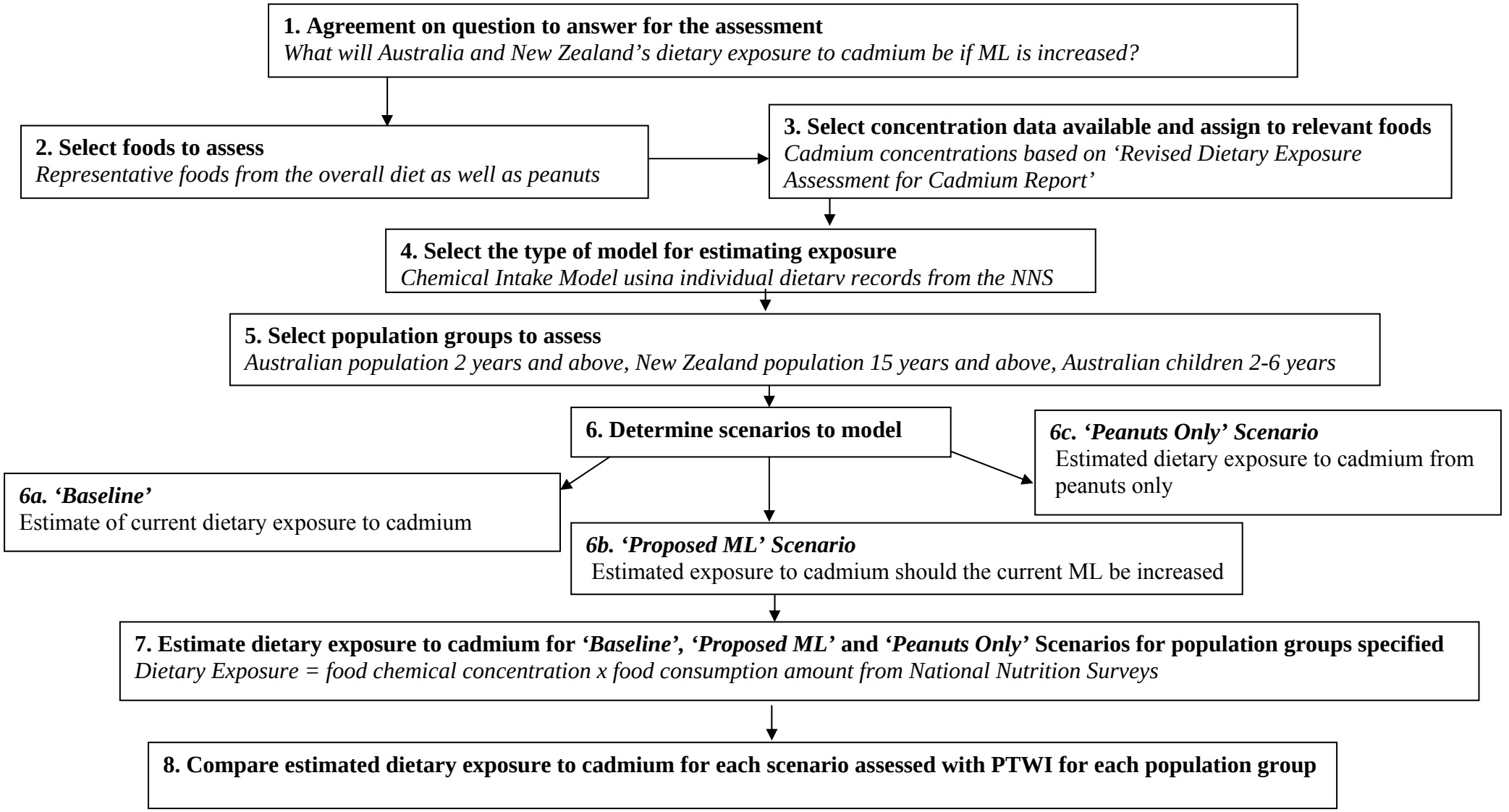
[17] Where analysis indicates a result that is below the level of reporting (LOR), the lower bound model assigns a value of zero to these non-detect results; the upper bound model assigns a value of LOR to the non-detect result.

Dietary exposure = food chemical concentration x food consumption

2.4 Dietary survey data

DIAMOND contains dietary survey data for both Australia and New Zealand; the 1995 NNS from Australia that surveyed 13 858 people aged 2 years and above, and the 1997 New Zealand NNS that surveyed 4 636 people aged 15 years and above. Both of the NNSs used a 24-hour food recall methodology. It is recognised that these survey data have several limitations. For a complete list of limitations see Section 8.

Figure 1: Dietary modelling approach used to determine Australia and New Zealand's dietary cadmium exposures from the whole diet and peanuts only



2.5 Population groups assessed

The dietary exposure assessment was conducted for both Australian and New Zealand populations for the whole population, as well as for Australian children 2-6 years. Dietary exposure assessments were conducted for the whole population as a proxy for lifetime exposure. A dietary exposure assessment was conducted on children because children generally have higher exposures due to their smaller body weight, and they consume more food per kilogram of body weight compared to adults. It is important to note that, while Australian children aged 2-6 years have been assessed as a separate group, this group has also been assessed in the whole population's dietary exposure assessment.

Due to the New Zealand 1997 NNS collecting data for individuals 15 years and above, an exposure assessment for New Zealand children was not possible. Data from the 2002 New Zealand Children's Nutrition Survey are held by FSANZ, however were not in the correct format at the time of this assessment to enable dietary exposure to be estimated. However, it is assumed that Australian children are representative of New Zealand children of the same age (2-6 years for this dietary exposure assessment). This can be justified by the similar dietary exposures obtained for children in the respective total diet studies.

To address issues raised in submissions received from the Initial Assessment Report, adverse impacts for particular segments of the population possibly exposed to high cadmium levels were considered, for example Torres Strait Islanders and vegetarians.

Due to the small number of the Torres Strait Islander population sampled in the 1995 NNS, there was insufficient information on consumption to provide robust dietary exposure assessment results for this specific group. Furthermore, consumption of dugong, turtle or other foods traditionally consumed by this population group that possibly have high cadmium concentration levels were not recorded in the NNS. However, it is noted that there are health education campaigns in northern Queensland and the Northern Territory that specifically advise against the consumption of organ meat from dugong and turtles, since cadmium is known to accumulate in the kidney and liver of long living animals.

A separate assessment for vegetarians could not be conducted as behavioural questions on vegetarians were not included in the NNS and it cannot be assumed that the non consumption of meat on the day of the survey is an indication of whether a person was a vegetarian or not at that time. The dietary exposure assessment therefore included people reporting consumption of meat, poultry and dairy products on the day of the survey and those who did not.

3. Cadmium concentration levels

The levels of cadmium in foods that were used in the dietary exposure assessment were derived from the 2001 FSANZ Revised Dietary Exposure Assessment for Cadmium (FSANZ, Unpublished) which sourced cadmium concentrations both from within FSANZ and external sources. No updated concentration data on cadmium have been collected by FSANZ since that time.

For *Baseline*, the median cadmium concentration of 0.04 mg/kg for peanuts was used for the dietary exposure assessment. For the *Proposed ML* scenario the cadmium concentration for peanuts was assumed to be the maximum of 0.5 mg/kg.

This value was not only the proposed ML requested by the Applicant, but was also the upper end of the range of concentration data for cadmium in peanuts reported both internationally as well as from Australian data received by FSANZ in the Application. Based on peanut cadmium concentrations available, 0.41 mg/kg was the highest reported concentration. Mean concentrations were found to lie within the range of 0.06-0.09 mg/kg (see Appendix 1 for further details)).

For the dietary exposure assessments that included foods from the whole diet, the concentrations of cadmium were assigned to food groups using DIAMOND food classification codes, which for raw commodities are based on the Codex raw commodity classification system. Foods containing cadmium were matched to the most appropriate raw commodity classification codes for dietary modelling purposes (See Appendix 1).

Due to the nature of this Application from the CMA, FSANZ ensured confectionery recipes stored within DIAMOND included peanuts where appropriate. For example, for the recipe recorded as 'Bar, caramel nougat with nut, milk chocolate coated', nuts in this instance were assumed to be peanuts. This ensured that the potential cadmium content of foods relevant to this Application were included in the assessment.

Where foods included in this dietary exposure assessment were found to have cadmium concentrations at or below the limit of reporting (LOR), upper bound and lower bound cadmium concentrations were assigned to the food. The LOR is the lowest concentration of a chemical that can be detected and quantified, with an acceptable degree of certainty, using a specified laboratory method and/or item of laboratory equipment. The lower bound is where foods with a 'not detected' concentration for cadmium were assigned a zero concentration and the upper bound where they were assigned a concentration equal to the LOR. This enabled a range of possible dietary exposures to be estimated.

4. Scenarios for dietary modelling

The scenarios assessed for this Application to determine the possible public health risk from dietary exposure to cadmium were as follows:

Baseline – estimate of current dietary exposure to cadmium from the whole diet (including peanuts) based on cadmium concentrations from the FSANZ Revised Dietary Exposure Assessment to Cadmium report (FSANZ Unpublished);

Proposed Maximum Level **(ML)** – estimate of cadmium exposure should the current ML of 0.1 mg/kg be increased to 0.5 mg/kg. This scenario included all foods assessed at *'Baseline'*, but with cadmium in peanuts at 0.5 mg/kg. This was therefore a 'worst case scenario' in relation to peanuts since even if the ML was to be established at 0.5 mg/kg, the actual concentration of cadmium in peanuts will be distributed such that the majority of values lie below 0.1 mg/kg; and

Peanuts Only – estimate of dietary cadmium exposure from peanuts only, assuming a concentration at the proposed ML of 0.5 mg/kg, again a 'worst case' scenario for peanuts.

5. How were the estimated dietary exposures calculated?

A detailed explanation of how the estimated dietary exposures were calculated can be found in Appendix 2.

5.1 Respondents versus Consumers

Dietary exposure assessment results are often provided in terms of 'respondents' and 'consumers only'. Respondents refers to all people included in the NNS survey regardless of whether they were exposed to the food chemical or not; consumers refers to only those people who reported consuming the food/s containing the chemical being assessed.

For this dietary exposure assessment (excluding the *Peanuts Only* scenario) all respondents were consumers of cadmium as cadmium is ubiquitous in the food supply and found in almost all foods.

6. Assumptions in the dietary modelling

The aim of the dietary exposure assessment was to make as realistic an estimate of dietary exposure as possible. However, where significant uncertainties in the data existed, conservative assumptions were generally used to ensure that the dietary exposure assessment did not underestimate exposure.

The assumptions made in the dietary modelling are listed below, broken down into several categories.

6.1 Concentration data

- Where a permission is given to a food classification code, all foods in that group contain cadmium (for example, SO697 – Peanuts);
- all the foods within the group contain cadmium at the levels specified in Appendix 1;
- where the concentration of cadmium in a food was reported as being less than the LOR, then the cadmium concentration of the food was equal to zero (lower bound) and the LOR value (upper bound);
- as DIAMOND provides dietary exposures for one day, results were multiplied by seven to obtain weekly exposures for direct comparison to the reference health standard, the Provisional Tolerable Weekly Intake (PTWI);
- where a food was not included in the exposure assessment, it was assumed to contain a zero concentration of cadmium; and
- where a food has a specified cadmium concentration, this concentration was carried over to mixed foods where the food has been used as an ingredient e.g. peanuts in peanut butter or peanuts in confectionery.

6.2 Consumption data

- Foods selected to be representative of the overall diet were included in this assessment and were assigned cadmium concentrations (a list of these foods can be found in Appendix 1);

- Australian children 2-6 years are representative of New Zealand children of the same age in terms of food consumption and dietary exposure to cadmium;
- consumption of foods as recorded in the NNS represent current food consumption patterns; and
- consumers always select products containing cadmium.

6.3 General

- There are no changes in cadmium concentrations from food preparation or due to cooking;
- for the purpose of this assessment, it is assumed that 1 millilitre is equal to 1 gram for all liquid and semi-liquid foods (e.g. milk, yoghurt); and
- there is no contribution to cadmium exposure through the use of complementary medicines (Australia) or dietary supplements (New Zealand).

These assumptions are likely to lead to a conservative estimate for cadmium dietary exposure.

7. Dietary exposure assessment results

7.1 Estimated dietary exposures to cadmium

The estimated mean dietary exposures to cadmium for *Baseline* and *Proposed ML* scenarios are shown in Figure 2. Estimated 90th percentile dietary exposures to cadmium for *Baseline* and *Proposed ML* scenarios are shown in Figure 3 (full results for both scenarios can be found in Table A3.1 and Table A3.2 in Appendix 3). The estimated dietary exposures are presented as a range (lower bound to upper bound estimates).

7.1.1 Baseline Scenario

Estimated mean dietary exposure to cadmium for all Australian respondents two years and above was 0.0009–0.0011 mg/kg bw/week. For Australian children 2-6 years, estimated *Baseline* dietary exposure was 0.0018-0.0024 mg/kg bw/week. Estimated 90th percentile dietary exposure was 0.0018-0.0021 mg/kg bw/week for the group two years and above and 0.0034-0.0042 mg/kg bw/week for 2-6 years.

For the New Zealand population 15 years and above, estimated dietary exposure to cadmium for *'Baseline'* was 0.0011-0.0013 mg/kg bw/week. Estimated 90th percentile exposure was 0.0019-0.0022 mg/kg bw/week.

7.1.2 Proposed ML Scenario

Estimated mean dietary exposure to cadmium for all Australian respondents two years and above was 0.0011-0.0013 mg/kg bw/week. For Australian children 2-6 years, estimated *Proposed ML* dietary exposure was 0.0022-0.0029 mg/kg bw/week. Estimated 90th percentile dietary exposure was 0.0022-0.0026 mg/kg bw/week for two years and above and 0.0045-0.0053 mg/kg bw/week for 2-6 years.

For the New Zealand population 15 years and above, estimated mean dietary exposure to cadmium for the *Proposed ML* scenario was 0.0012-0.0014 mg/kg bw/week. Estimated 90th percentile exposure was 0.0022-0.0025 mg/kg bw/week.

7.1.3 Peanuts Only Scenario

The estimated dietary exposures to cadmium for the *Peanuts Only* scenario are shown in Table A3.3 in Appendix 3 and assumed a cadmium concentration of 0.5 mg/kg for peanuts (*Proposed ML scenario*), hence there was no range for the estimated dietary exposures.

Estimated mean dietary exposures to cadmium for all Australian respondents two years and above from peanuts only were 0.0002 mg/kg bw/week; for peanut consumers only, the dietary exposure was 0.0009 mg/kg bw/week. For Australian children 2-6 years, estimated maximum mean dietary exposure for the *Peanuts Only* scenario was 0.0005 mg/kg bw/week for respondents and 0.0016 mg/kg bw/week for consumers of peanuts. Estimated 90th percentile exposures for peanut consumers were 0.0022 mg/kg bw/week for the group two years and above and 0.0041 mg/kg bw/week for children aged 2-6 years.

For the New Zealand population 15 years and above, estimated mean dietary exposure to cadmium for the *Peanuts Only* scenario was 0.0001 mg/kg bw/week for all respondents and 0.0006 mg/kg bw/week for peanut consumers. Estimated 90th percentile exposure for peanut consumers was 0.0013 mg/kg.

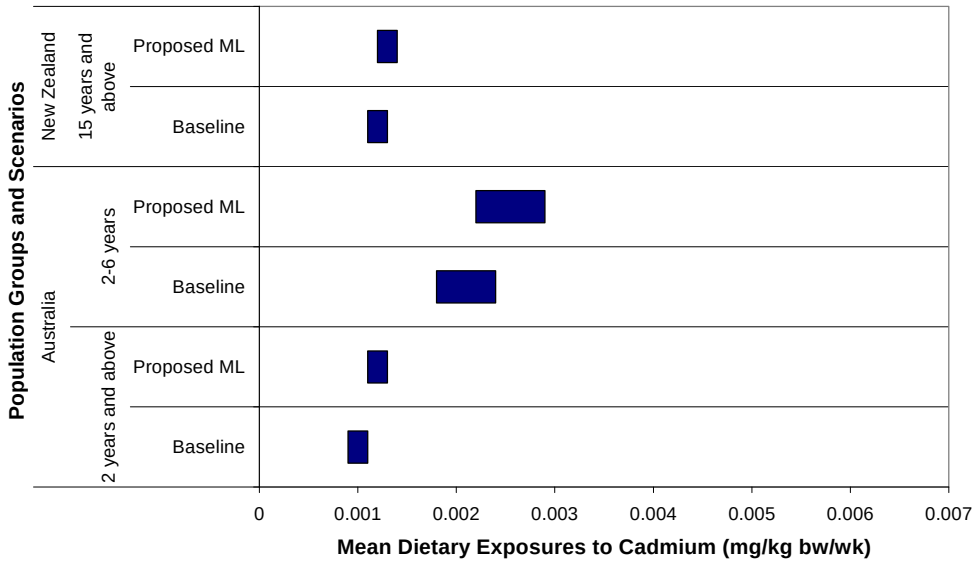


Figure 2: Estimated mean dietary exposures to cadmium for Baseline *and* Proposed ML *scenarios for all population groups assessed*

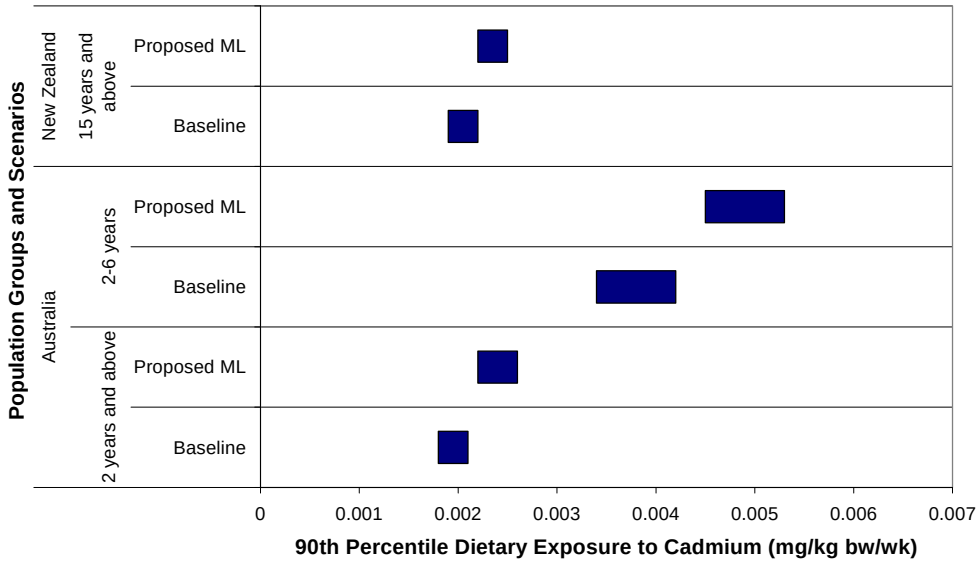


Figure 3: Estimated 90th percentile dietary exposures to cadmium for Baseline *and* Proposed ML *scenarios for all population groups assessed*

7.2 Major contributing foods to total cadmium exposure

Lower bound results were used to calculate the percentage contribution each food group makes to total estimated exposures, as this provides the best indication of the food groups most likely to contribute to dietary exposure as it only includes foods containing levels of cadmium at or above the LOR. It should be noted that the percent contribution of each food group is based on total dietary cadmium exposure for all consumers of cadmium in the population groups assessed. Therefore the total cadmium exposures differ for each population group. A complete list of contributions from all foods is shown in Appendix 4.

7.2.1 Baseline scenario

The major contributors to total cadmium dietary exposures for the *Baseline* scenario are shown below in Table 1. These are displayed for the total population assessments as well as for Australian children 2-6 years. The major contributor was potato for Australians two years (28%) and above and for children 2-6 years (30%). Another high contributor for children was cacao beans (10%), as well as orange juice and wheat flour. The contribution of peanuts to cadmium dietary exposure was low for all Australian groups assessed (2%).

For New Zealanders 15 years and above, the major contributors for the *Baseline* scenario were potato (41%) and mussels (6%). Peanuts were minor contributors (1%).

Table 1: Major contributors (>5%) to total cadmium dietary exposures at *Baseline* for Australia and New Zealand population groups assessed

Food Name	% Contribution to Cadmium Dietary Exposure		
	Australians 2 years and above	Australians 2-6 years	New Zealand 15 years and above
Cacao Beans	5	10	<5
Carrot	5	5	<5
Cattle Meat	5	<5	<5
Mussels	<5	0	6
Orange Juice	<5	7	<5
Potato	28	30	41
Sugar	<5	5	<5
Wheat Flour	5	7	<5

Note: The per cent contribution of each food group is based on total cadmium exposure for all consumers in the population groups assessed. Therefore the total cadmium exposures differ for each population group and each scenario.

7.2.2 Proposed ML scenario

The major contributors to total cadmium dietary exposures for the *Proposed ML* scenario are shown below. These are displayed for the total population assessments as well as for Australian children 2-6 years. The major contributor was potato (24%) for Australians two years and above and children 2-6 years. Other high contributors for children were cacao beans (8%) and wheat flour (6%). The contribution of peanuts to cadmium dietary exposure was 16% for all Australians and 22% for children.

For New Zealanders 15 years and above, the major contributors were potato (37%), mussels (6%) and peanuts (11%).

Table 2: Major contributors (>5%) to total cadmium dietary exposures for the *Proposed ML* scenario for Australia and New Zealand, and for different population groups

Food Name	% Contribution to Cadmium Dietary Exposure		
	Australians 2 years and above	Australians 2-6 years	New Zealand 15 years and above
Cacao Beans	<5	8	<5
Mussels	<5	0	6
Orange Juice	<5	5	<5
Potato	24	24	37
Wheat Flour	5	6	<5
Peanut	**16**	**22**	**11**

Note: The per cent contribution of each food group is based on total cadmium exposure for all consumers in the population groups assessed. Therefore the total cadmium exposures differ for each population group and each scenario.

While this scenario shows that peanuts would be a major contributor to dietary cadmium exposure, the actual contribution from peanuts is expected to be lower for reasons discussed above (Section 4) in that for this scenario the concentrations for all foods other than peanuts were median concentrations based on analytical data while the peanut concentration was the equivalent of the *Proposed ML* (0.5 mg/kg) which would not be the case in reality.

8. Limitations of the dietary modelling

Dietary modelling based on 1995 or 1997 NNS food consumption data provides the best estimate of actual consumption of a food and the resulting estimated dietary intake of a nutrient for the population. However, it should be noted that the NNS data have limitations. These limitations relate to the age of the data and the changes in eating patterns that may have occurred since the data were collected. Generally, consumption of staple foods such as fruit, vegetables, meat, dairy products and cereal products, which make up the majority of most people's diet, is unlikely to have changed markedly since 1995/1997 (Cook, Rutishauser and Allsop, 2001 and Cook, Rutishauser and Seelig, 2001). However, there is uncertainty associated with the consumption of foods that may have changed in consumption since 1995/1997, or that have been introduced to the market since 1995/1997.

FSANZ has access to more recent food consumption data for Australia and New Zealand for selected food groups from Roy Morgan Single Source data. These data are usually assessed to determine whether consumption patterns for key foods applicable to each dietary exposure assessment are consistent with the 1995 Australian or 1997 New Zealand NNS data, and therefore, the dietary exposure assessments based on these NNS data are also reliable. However, the data from Roy Morgan were not specific enough to determine consumption patterns for key foods relating to this assessment, such as peanuts, or the major contributors potatoes and cocoa.

A limitation of estimating dietary exposure over a period of time associated with the dietary modelling is that only 24-hour dietary survey data were available, and these tend to over-estimate habitual food consumption amounts for high consumers. Therefore, predicted high percentile exposures are likely to be higher than actual high percentile exposures over a lifetime, in particular when the food chemical is in occasionally consumed foods (Lambe *et al.*, 2000 and WHO, 1985). FSANZ presents the 90th percentile as the exposure for a 'high' consumer as it a better reflection, and not as much of an overestimate, of exposures for high consumers over a long period of time compared to higher percentiles.

While the results of NNSs can be used to describe the usual intake of groups of people, they cannot be used to describe the usual intake of an individual (Rutishauser, 2000). In particular, they cannot be used to predict how consumers will change their eating patterns as a result of an external influence such as the availability of a new type of food.

Over time, there may be changes to the ways in which manufacturers and retailers make and present foods for sale. Since the data were collected for the Australian and New Zealand NNSs, there have been significant changes to the Code to allow more innovation in the food industry. As a consequence, another limitation of the dietary modelling is that some of the foods that are currently available in the food supply were either not available or were not as commonly available in 1995/1997.

Assumptions were also made in these assessments about the value of analytical results below the LOR (non-detects). The LOR is the lowest concentration of a chemical that can be detected and quantified, with an acceptable degree of certainty, using a specified laboratory method and/or item of laboratory equipment. In the case of metal contaminants that occur naturally in the environment, it may not be reasonable to assume that the metal is not present in the food when the analytical results are less than the LOR. For this reason, results below the LOR could be anywhere between zero and the LOR. For cadmium, dietary exposure estimates are presented as a range of possible cadmium dietary exposures across the population, from the lower bound estimate to the upper bound estimate to allow for this uncertainty.

Where the NNSs collected data on the use of complementary medicines (Australia) or dietary supplements (New Zealand), it was either not in a robust enough format to include in DIAMOND or has simply not been included in the DIAMOND program. Consequently, exposure to substances consumed via complementary medicines or dietary supplements could not be included in the dietary exposure assessment.

FSANZ does not apply statistical population weights to each individual in the NNSs in order to make the data representative of the population. This prevents distortion of actual food consumption amounts that may result in an unrealistic exposure estimate. Maori and Pacific Islanders were over-sampled in the 1997 New Zealand National Nutrition Survey so that statistically valid assessments could be made for these population groups. As a result, there may be bias towards these population groups in the dietary exposure assessment because population weights were not used.

9. Comparison of estimated dietary exposures to cadmium with the reference health standard

In order to determine if the level of exposure to cadmium will be of concern to public health and safety, the estimated dietary exposures were compared to the PTWI of 0.007 mg/kg bw set by JECFA. PTWIs are upper limits that are set for substances such as heavy metals that are known to accumulate in animals and humans, and estimate the amount of a chemical that can be ingested weekly over a lifetime without appreciable risk to health (WHO, 2001).

The estimated dietary exposures to cadmium for Australia and New Zealand, as assessed against the PTWI are shown below in Figure 4 and Figure 5 with full details in Appendix 5.

9.1 Baseline Scenario

For the Australian population two years and above at *Baseline*, the estimated mean dietary exposure was 15-15% of the PTWI and 25-30% of the PTWI for 90th percentile exposure. Australian children 2-6 years had a higher mean exposure at 25-35% of the PTWI and 50-60% of the PTWI for 90th percentile exposure.

For the New Zealand population 15 years and above at *Baseline*, estimated mean dietary exposure to cadmium from the total diet was 15-20% of the PTWI and 25-30% of the PTWI for 90th percentile dietary exposure.

The estimated baseline dietary exposures for Australians were in a similar range to those estimated previously by FSANZ in the revised cadmium dietary exposure assessment in 2001 (FSANZ, Unpublished) and from the 20th ATDS (FSANZ, 2002).

The estimated baseline dietary exposures for New Zealanders were slightly lower than those estimated in the New Zealand Total Diet Survey (New Zealand Food Safety Authority, 2005): 15-20% PTWI compared to 22-29% PTWI. This could be attributed to the different age groups assessed and different methodologies used.

9.2 *Proposed ML* Scenario

When the concentration of cadmium in peanuts was at the proposed ML of 0.5 mg/kg (*Proposed ML scenario)*, the mean estimated dietary exposure expressed as a proportion of the PTWI was slightly higher than baseline. For the Australian population aged 2 years and above it was 15-20% of the PTWI and for children 2-6 years it was 30-40% of the PTWI. The 90th percentile dietary exposure to cadmium was 30-35% of the PTWI for Australians aged 2 years and above and 65-75% of the PTWI for 90th percentile exposure.

For New Zealanders aged 15 years and above for the *Proposed ML* scenario, the estimated dietary exposure was 15-20% PTWI at the mean and 30-35% of the PTWI for 90th percentile consumers of cadmium.

9.3 Peanuts Only Scenario

When assessing dietary exposure to cadmium from peanuts only, Australians' dietary exposure for all respondents 2 years and above was 3% of the PTWI with 2-6 year olds at 7% of the PTWI. Mean exposures for consumers only were 10% and 20% of the PTWI for 2 years and above and 2-6 years respectively. Dietary exposures for consumers at 90th percentiles were 30% of the PTWI for 2 years and above and 60% of the PTWI for 2-6 years.

For the New Zealand population 15 years and above, estimated dietary exposures to cadmium from peanuts were 2% of the PTWI at the mean for all respondents, 8% of the PTWI at the mean for consumers of peanuts only, and 20% of the PTWI for 90th percentile consumers.

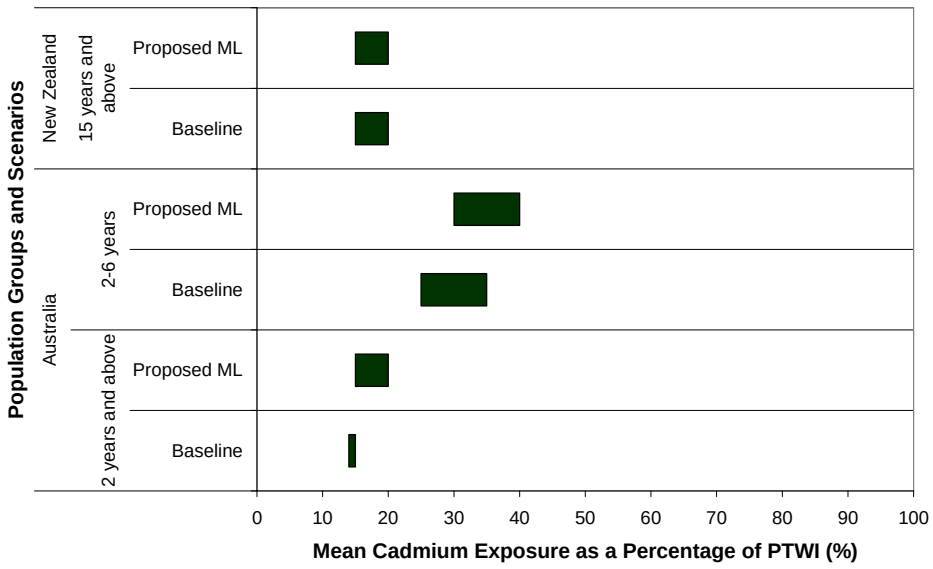


Figure 4: Estimated mean dietary exposures to cadmium for Baseline *and* Proposed ML *scenarios for all population groups assessed as a percent of the Provisional Tolerable Weekly Intake (PTWI)*

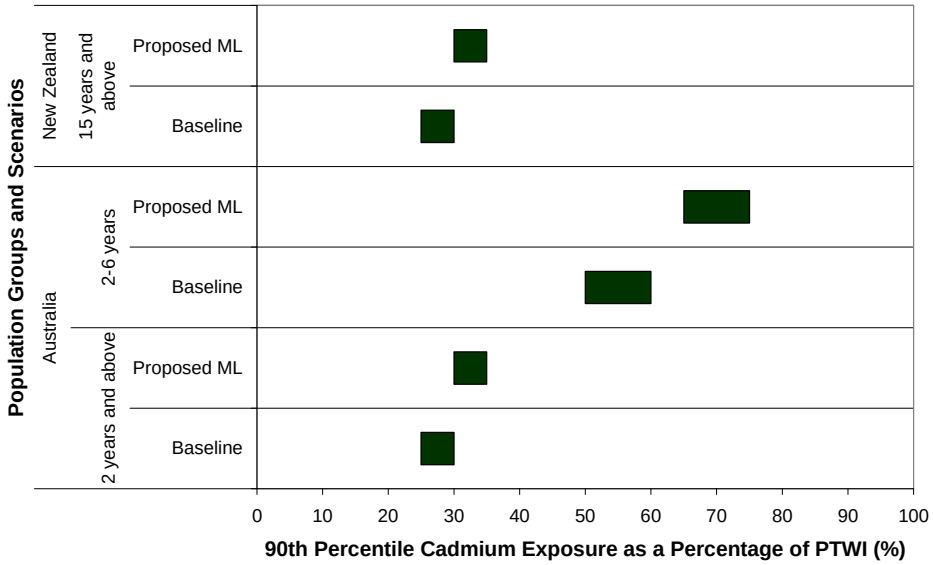


Figure 5: Estimated 90th percentile dietary exposures to cadmium for Baseline *and* Proposed ML *scenarios for all population groups assessed as a percent of the Provisional Tolerable Weekly Intake (PTWI)*

References

Cook, T. I. Rutishauser and Seelig, M. (2001) Comparable Data on Food and Nutrient Intake and Physical Measurements from the 1983, 1985 and 1995 National Nutrition Surveys. Commonwealth Department of Health and Aged Care: Canberra.

Cook, T. I. Rutishauser, and Allsopp, R. (2001) The Bridging Study: Comparing results from the 1983, 1985 and 1995 Australian National Nutrition Surveys. Commonwealth Department of Health and Aged Care: Canberra.

Food Standards Australia New Zealand. (2001) Revised Dietary Exposure Assessment for Cadmium. Unpublished: Canberra.

Food Standards Australia New Zealand. (2002) The 20th Australian Total Diet Survey. FSANZ: Canberra.

Lambe, J., Kearney, J., Leclercrq, C., Berardi, D., Zunft,H., De Henauw, S., De Volder, M., Lamberg-Allardt, C., Karkkainen, M., Dunne, A. and Gibney, N. (2000) Enhancing the capacity of food consumption surveys of short duration to estimate long term consumer-only intakes by combination with a qualitative food frequency questionnaire. Food Additives and Contaminants, 17(3), pp. 177-187.

New Zealand Food Safety Authority. (2005) 2003/04 New Zealand Total Diet Survey.

Rutishauser I. (2000) Getting it right: How to use the data from the 1995 National Nutrition Survey. Commonwealth of Australia: Canberra.

United States Food And Drug Administration (USFDA). (2006) Total Diet Study Statistics on Element Results. Revision 4, Market Baskets 1991-3 through 2004-4. August 21, 2006. http://www.cfsan.fda.gov/~acrobat/tds1byel.pdf

World Health Organisation. (1985) Guidelines for the Study of Dietary Intakes of Chemical Contaminants. WHO, Geneva.

World Health Organization. (2001) Summary of Evaluations Performed by the Joint FAO/WHO Expert Committee on Food Additives (JECFA 1956-2001) (First through fifty-seventh meetings) Internet Edition. ILSI Press International Life Sciences Institute: Washington DC.

Appendix 1

Data sources of peanut cadmium concentrations

International concentration data for cadmium in peanut and peanut products for the current study were sourced from both within Australia and from overseas agencies. These sources were as follows:

- Australian Quarantine and Inspection Service (AQIS) test results for imported foods analysed for cadmium content between October 2001 to October 2006 (Personal communication, 2007);
- the United States Food and Drug Administration (USFDA) Total Diet Study – Market Baskets 1991 – 1993 through 2004 (USFDA, 2006);
- the Chinese Total Diet Study – 2000 (Personal communication, 2007); and
- the New Zealand Total Diet Survey – 2003/04 (New Zealand Food Safety Authority, 2005).

Table A1.1: Summary of cadmium concentrations for peanuts available from Australia and internationally since the 2001 assessment

Country	Source	Year	Cadmium Concentration (mg/kg)		
			Mean	Lowest Concentration	Highest Concentration
China	Chinese Total Diet Study	2000	0.087	0.020	0.239
United States	United States Total Diet Survey	1991-2004	0.057	0.020	0.117
Australia	Australian Quarantine and Inspection Service	2001-2006	0.090	0.010	0.410
New Zealand	Total Diet Survey	2003-2004	0.075	0.028	0.157

Cadmium concentrations used for this dietary exposure assessment for all other foods were from the FSANZ Revised Dietary Exposure Assessment for Cadmium (2001). Foods included in this assessment are outlined below in Table A1.2 and were obtained from a number of sources.

Table A1. 2: Cadmium concentrations used in this dietary exposure assessment as derived from the Revised Dietary Exposure Assessment for Cadmium (2001) by FSANZ

Australia			New Zealand		
Food Commodity	ML* (mg/kg)	Survey Level (mg/kg)	Food Commodity	ML* (mg/kg)	Survey Level (mg/kg)
Honey		0.000-0.0025	Honey		0.000-0.0025
Wheat bran, processed	0.1	0.015	Wheat bran, processed	0.1	0.015
Wheat germ	0.1	0.004	Wheat germ	0.1	0.004
Wheat flour	0.1	0.004	Wheat flour	0.1	0.004

Australia			New Zealand		
Food Commodity	**ML* (mg/kg)**	**Survey Level (mg/kg)**	**Food Commodity**	**ML* (mg/kg)**	**Survey Level (mg/kg)**
Wheat wholemeal	0.1	0.004	Wheat wholemeal	0.1	0.004
Maize flour		0.003	Maize flour		0.003
Wheat bran, unprocessed	0.1	0.018	Rice, husked	0.1	0.005
Rice, polished	0.1	0.004	Wheat bran, unprocessed	0.1	0.018
Rice bran, unprocessed	0.1	0.004	Rice, polished	0.1	0.004
Dried prunes		0.004	Dried prunes		0.004
Dried grapes		0.003	Dried grapes		0.003
Dried apricots		0.01	Dried apricots		0.01
Sugar cane, molasses		0.007	Sugar cane, molasses		0.007
Tea, green and black		0.001	Tea, green and black		0.001
Blueberries		0.003	Blueberries		0.003
Grapes		0.003	Grapes		0.003
Wine		0.001	Wine		0.001
Strawberry		0.021	Strawberry		0.021
Oranges, sweet and sour		0.003	Oranges, sweet and sour		0.003
Orange juice		0.003	Orange juice		0.003
Mandarins		0.003	Mandarins		0.003
Tropical fruit, inedible peel		0.003	Tropical fruit, inedible peel		0.003
Avocado		0.014	Avocado		0.014
Banana		0.003	Banana		0.003
Mango		0.003	Mango		0.003
Papaya		0.003	Papaya		0.003
Pineapple		0.000-0.0025	Pineapple		0.000-0.0025
Pineapple juice		0.003	Pineapple juice		0.003
Apple		0.000-0.0025	Apple		0.000-0.0025
Apple juice		0.003	Apple juice		0.003
Pear		0.003	Pear		0.003
Peach		0.003	Peach		0.003
Plums		0.003	Plums		0.003
Barley		0.004	Barley		0.004
Barley, beer		0.003	Barley, beer		0.003
Oats		0.01	Maize		0.001
Rice	0.1	0.004	Oats		0.01
Wild rice	0.1	0.004	Rice	0.1	0.004

Australia			New Zealand		
Food Commodity	**ML* (mg/kg)**	**Survey Level (mg/kg)**	**Food Commodity**	**ML* (mg/kg)**	**Survey Level (mg/kg)**
Molluscs	2	0.08	Wheat	0.1	0.011
Mussels	2	0.48	Wild rice	0.1	0.004
Oysters	2	0.49	Molluscs	2	0.08
Scallops	2	0.3	Mussels	2	0.48
Squids	2	0.058	Oysters	2	0.49
Cattle milk		0.000 – 0.0025	Scallops	2	0.3
Cattle meat	0.05	0.005	Squids	2	0.058
Deer meat		0.003	Cattle milk		0.000 – 0.0025
Kangaroo meat		0.003	Cattle meat	0.05	0.005
Pig meat	0.05	0.003	Deer meat		0.003
Sheep meat	0.05	0.005	Pig meat	0.05	0.003
Sheep, edible offal of		0.005	Sheep meat	0.05	0.005
Sheep kidney	2.5	0.24	Sheep, edible offal of		0.005
Sheep liver	1.25	0.163	Cattle kidney	2.5	0.32
Vegetable oil, edible		0.003	Cattle liver	1.25	0.075
Olive oil, refined		0.003	Pig liver	1.25	0.06
Rape seed oil, edible		0.003	Sheep kidney	2.5	0.24
Peanut oil, edible		0.04	Sheep liver	1.25	0.163
Chicken eggs		0.003	Vegetable oil, edible		0.003
Chicken meat		0.003	Olive oil, refined		0.003
Turkey meat		0.003	Rape seed oil, edible		0.003
Chicken, edible offal of		0.008	Peanut oil, edible		0.04
Cacao beans	0.5	0.425	Chicken eggs		0.003
Coffee beans		0.014	Chicken meat		0.003
Mustard seed		0.036	Turkey meat		0.003
Peanut	0.1	0.04	Chicken, edible offal of		0.008
Sesame seed		0.04	Cacao beans	0.5	0.425
Sunflower seed		0.06	Coffee beans		0.014
Almonds		0.003	Mustard seed		0.036
Cashew nut		0.003	Peanut	0.1	0.04

Australia			New Zealand		
Food Commodity	**ML* (mg/kg)**	**Survey Level (mg/kg)**	**Food Commodity**	**ML* (mg/kg)**	**Survey Level (mg/kg)**
Macadamia nut		0.004	Sesame seed		0.04
Walnuts		0.000-0.0025	Sunflower seed		0.06
Garlic		0.02	Almonds		0.003
Onion bulb		0.014	Cashew nut		0.003
Cabbages head		0.004	Macadamia nut		0.004
Broccoli		0.000-0.0025	Walnuts		0.000-0.0025
Cauliflower		0.000-0.0025	Garlic		0.02
Melons, except watermelons		0.003	Onion bulb		0.014
Cucumber		0.003	Cabbages head		0.004
Pumpkins		0.004	Broccoli		0.000-0.0025
Watermelon		0.003	Cauliflower		0.000-0.0025
Soya bean (dry)		0.003	Melons, except watermelons		0.003
Tofu		0.011	Cucumber		0.003
Leafy vegetables	0.1	0.01	Pumpkins		0.004
Pak-choi	0.1	0.005	Watermelon		0.003
Lettuce head	0.1	0.01	Soya bean (dry)		0.003
Egg plant		0.013	Leafy vegetables	0.1	0.01
Peppers sweet		0.01	Lettuce head	0.1	0.01
Sweet corn		0.003	Egg plant		0.013
Tomato		0.006	Peppers sweet		0.01
Tomato canned		0.015	Sweet corn		0.003
Fungi edible		0.03	Tomato		0.006
Mushrooms		0.000-0.0025	Tomato canned		0.015
Beans, except broad and soya		0.003	Fungi edible		0.03
Garden peas shelled		0.003	Mushrooms		0.000-0.0025
Root and tuber vegetable	0.1	0.01	Beans, except broad and soya		0.003
Beetroot	0.1	0.018	Garden peas shelled		0.003
Carrot	0.1	0.022	Lentil		0.004
Potato	0.1	0.033	Soya bean		0.007
Celery		0.011	Root and tuber vegetable	0.1	0.01
Crabs		0.05	Beetroot	0.1	0.018

Australia			New Zealand		
Food Commodity	**ML* (mg/kg)**	**Survey Level (mg/kg)**	**Food Commodity**	**ML* (mg/kg)**	**Survey Level (mg/kg)**
Lobsters		0.02	Carrot	0.1	0.022
Prawns		0.13	Potato	0.1	0.033
Diadromous fish		0.009	Potato baked/fried	0.1	0.098
Marine fish of all species		0.009	Celery		0.011
Shark		0.004	Crabs		0.05
Tuna		0.009	Crayfish		0.009
			Prawns		0.13
			Diadromous fish		0.009
			Marine fish of all species		0.009
			Shark		0.004
			Tuna		0.009

* Where there is no Maximum Level (ML) listed, no ML is specified for this commodity in the Australia New Zealand Food Standards Code.

Appendix 2

How were the estimated dietary exposures calculated?

The DIAMOND program allows cadmium concentrations to be assigned to food groups. Exposure to the contaminant was calculated for each individual in the NNSs using his or her individual food records from the dietary survey. The DIAMOND program multiplies the specified concentration of cadmium by the amount of food that an individual consumed from that group in order to estimate the exposure to cadmium from each food. Once this has been completed for all of the foods specified to contain cadmium, the total amount of cadmium consumed from all foods is summed for each individual. Population statistics (mean and high percentile exposures) are then derived from the individuals' ranked exposures.

Where estimated dietary exposures are expressed per kilogram of body weight, each individual's total dietary exposure is divided by their own body weight, the results ranked, and population statistics derived. A small number of NNS respondents did not provide a body weight. These respondents are not included in calculations of estimated dietary intakes that are expressed per kilogram of body weight.

Where estimated exposures are expressed as a percentage of the reference health standard, each individual's total exposure is calculated as a percentage of the reference health standard (by using the total exposures in mg per kilogram of body weight per week), the results are then ranked, and population statistics derived.

Food consumption amounts for each individual take into account where each food in a classification code is consumed alone and as an ingredient in mixed foods. For example, peanuts added to confectionery and peanuts in peanut butter are all included in the consumption of peanuts. Where a higher level food classification code (for example Processed fruits and vegetables) is given a cadmium concentration, as well as a sub-category (e.g. 4.3.1.3 Nuts and seeds), the consumption of the foods in the sub-classification is not included in the higher level classification code.

In DIAMOND, all mixed foods have a recipe. Recipes are used to break down mixed foods into their raw commodity components (for example bread will be broken down to wheat flour, yeast, water etc). The data for consumption of the raw commodities are then used in models that assign cadmium permissions to raw commodity food codes.

When a food is classified in two food groups (for example, mixed fruit juice may be entered in the apple and pear groups), and these food groups are assigned different cadmium permissions, DIAMOND will assume the food is in the food group with the highest assigned cadmium level to assume a worst case scenario. If the food groups have the same permitted cadmium level, DIAMOND will assume the food is in the food group that appears first, based alpha-numerically on the DIAMOND food code.

In DIAMOND, hydration and raw equivalence factors are applied to some foods to convert the amount of food consumed in the dietary survey to the equivalent amount of the food in the form to which a food chemical concentration is assigned. Factors are only applied to individual foods, and not major food group codes.

For example, consumption figures for instant coffee powder are converted into the equivalent quantities of coffee beans; consumption figures for tomato paste are converted into the equivalent quantities of raw tomatoes.

A2.1 How were percentage contributions calculated?

Percentage contributions of each food group to total estimated exposures are calculated by summing the exposures for a food group from each individual in the population group who consumed a food from that group. This is the dividing by the sum of the exposures of all individuals from all food groups containing cadmium and multiplying this by 100.

A2.2 Reporting of dietary exposure assessment results for high consumers

Under the FSANZ Science Strategy 2006-2009, FSANZ agreed to review its dietary modelling procedures. As part of this review an international peer review was sought. FSANZ has previously reported chronic dietary exposures for high consumers at the 95th percentile. The recommendation of the peer review by an international dietary exposure assessment expert from the US Food and Drug Administration was that FSANZ should consider aligning its reporting of food chemical dietary exposures with international best practice by reporting at the 90th percentile not the 95th percentile, if only one 24 hour recall record per person was used for the assessment (Lambe *et al*, 2000). This is because the 95th percentile results are likely to be an overestimate of dietary exposure on a daily basis over a life-time of exposure. Basing risk management decisions on the 95th percentile will potentially result in an overly conservative risk management approach.

Appendix 3

Complete information on cadmium dietary exposure assessment results

Table A3.1: Estimated dietary exposures to cadmium at *Baseline*

Country	Population group	Number of cadmium consumers LB/UB	Consumers♦ as a % of respondents #	Mean all respondents# (mg/kg bw/wk)	90th percentile consumers♦ (mg/kg bw/wk)
Australia	2 years +	13854/13856	100	0.0009 - 0.0011	0.0018 - 0.0021
	2-6 years	989/989	100	0.0018 - 0.0024	0.0034 - 0.0042
New Zealand	15 years +	4632/4635	100	0.0011 - 0.0013	0.0019 - 0.0022

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.

♦ Consumers only – This only includes the people who have consumed a food that contains cadmium.

Table A3.2: Estimated dietary exposures to cadmium for the *Proposed ML* scenario

Country	Population group	Number of cadmium consumers LB/UB	Consumers♦ as a % of respondents #	Mean all respondents# (mg/kg bw/wk)	90th percentile consumers♦ (mg/kg bw/wk)
Australia	2 years +	13854/13856	100	0.0011 - 0.0013	0.0022 - 0.0026
	2-6 years	989/989	100	0.0022 - 0.0029	0.0045 - 0.0053
New Zealand	15 years +	4632/4635	100	0.0012 - 0.0014	0.0022 - 0.0025

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.

♦ Consumers only – This only includes the people who have consumed a food that contains cadmium

Table A3.3: Estimated dietary exposures to cadmium for the '*Peanuts Only*' scenario

Country	Population group	Number of cadmium consumers	Consumers♦ as a % of respondents#	Mean all respondents#	Mean consumers♦	90th percentile consumers♦
				(mg/kg bw/wk)		
Australia	2 years +	2986	20	0.0002	0.0009	0.0022
	2-6 years	308	30	0.0005	0.0016	0.0041
New Zealand	15 years +	1046	25	0.0001	0.0006	0.0013

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.

♦ Consumers only – This only includes the people who have consumed a food that contains cadmium.

Appendix 4

Contributions from all foods to cadmium dietary exposure

Table A4.1: Percentage contribution of all foods used in the dietary modelling to cadmium dietary exposure for both Australian population groups

Food Name	Australia			
	2 years and above		2-6 years	
	Baseline	Proposed ML	Baseline	Proposed ML
Honey	0	0	0	0
Wheat bran, processed	<1	<1	<1	<1
Wheat germ	<1	<1	<1	<1
Wheat flour	**5**	**5**	**7**	**6**
Wheat wholemeal	<1	<1	<1	<1
Maize flour	<1	<1	<1	<1
Wheat bran, unprocessed	<1	<1	<1	<1
Rice, polished	<1	<1	1	<1
Rice bran, unprocessed	<1	<1	0	0
Dried prunes	<1	<1	<1	<1
Dried grapes	<1	<1	<1	<1
Dried apricots	<1	<1	<1	<1
Sugar cane molasses	4	3	**5**	4
Tea, green, black	<1	<1	<1	<1
Blueberries	<1	<1	0	0
Grapes	<1	<1	<1	<1
Wine	<1	<1	<1	<1
Strawberry	<1	<1	<1	<1
Oranges, sweet, sour	<1	<1	<1	<1
Orange juice	4	4	**7**	**5**
Mandarins	<1	<1	<1	<1
Tropical fruit-inedible peel	<1	<1	<1	<1
Avocado	<1	<1	<1	<1
Banana (includes banana dwarf)	<1	<1	2	1
Mango	<1	<1	<1	<1
Papaya (Pawpaw)	<1	<1	<1	<1
Pineapple	0	0	0	0
Pineapple, juice	<1	<1	1	<1
Apple	0	0	0	0
Apple, juice	<1	<1	3	3
Pear	<1	<1	<1	<1
Peach	<1	<1	<1	<1
Plums	<1	<1	<1	<1

Food Name	Australia			
	2 years and above		2-6 years	
	Baseline	Proposed ML	Baseline	Proposed ML
Barley	<1	<1	<1	<1
Barley, beer	<1	<1	<1	<1
Oats	<1	<1	<1	<1
Rice	<1	<1	<1	<1
Wild rice	<1	<1	0	0
Molluscs	<1	<1	<1	<1
Mussels	1	<1	0	0
Oysters	1	1	0	0
Scallops	1	<1	<1	<1
Squids	<1	<1	<1	<1
Cattle milk	0	0	0	0
Cattle meat	**5**	4	4	3
Deer meat	<1	<1	0	0
Kangaroo meat	<1	<1	0	0
Pig meat	1	<1	<1	<1
Sheep meat	<1	<1	<1	<1
Sheep, edible offal of	<1	<1	0	0
Sheep, kidney	<1	<1	<1	<1
Sheep, liver	<1	<1	0	0
Vegetable oil, edible	<1	<1	1	<1
Olive oil, refined	<1	<1	<1	<1
Rape seed oil, edible	<1	<1	<1	<1
Peanut oil, edible	<1	<1	<1	<1
Chicken eggs	<1	<1	<1	<1
Chicken meat	1	1	1	<1
Turkey meat	<1	<1	<1	<1
Chicken, edible offal of	<1	<1	<1	<1
Cacao beans	**5**	4	**10**	**8**
Coffee beans	<1	<1	<1	<1
Mustard seed	<1	<1	<1	<1
Peanut	2	**16**	2	**22**
Sesame seed	<1	<1	<1	<1
Sunflower seed	<1	<1	<1	<1
Almonds	<1	<1	<1	<1
Cashew nut	<1	<1	<1	<1
Macadamia nuts	<1	<1	0	0
Walnuts	0	0	0	0
Garlic	<1	<1	<1	<1
Onion, bulb	3	3	2	2
Cabbages, head	<1	<1	<1	<1

Food Name	Australia			
	2 years and above		2-6 years	
	Baseline	Proposed ML	Baseline	Proposed ML
Broccoli	0	0	0	0
Cauliflower	0	0	0	0
Melons, except Watermelon	<1	<1	<1	<1
Cucumber	<1	<1	<1	<1
Pumpkins	<1	<1	<1	<1
Watermelon	<1	<1	<1	<1
Soya bean (dry)	<1	<1	<1	<1
Tofu	<1	<1	<1	<1
Leafy vegetables	<1	<1	<1	<1
Pak-choi	<1	<1	<1	<1
Lettuce, Head	1	<1	<1	<1
Egg plant	<1	<1	0	0
Peppers, sweet	<1	<1	<1	<1
Sweet corn	<1	<1	<1	<1
Tomato	4	4	3	2
Tomato, canned	<1	<1	<1	<1
Fungi, edible (not including cultivated	<1	<1	1	<1
Mushrooms	0	0	0	0
Beans, except broad bean and soya bean	<1	<1	<1	<1
Garden pea, shelled	<1	<1	<1	<1
Root and tuber vegetables	<1	<1	<1	<1
Beetroot	<1	<1	<1	<1
Carrot	5	4	**5**	4
Potato	**28**	**24**	**30**	**24**
Celery	<1	<1	<1	<1
Crabs	<1	<1	<1	<1
Lobsters	<1	<1	0	0
Prawns	4	3	<1	<1
Diadromous fish	<1	<1	<1	<1
Marine fish of all species	<1	<1	<1	<1
Shark	<1	<1	<1	<1
Tuna	<1	<1	<1	<1

Table A4.2: Percentage contribution of all foods used in the dietary modelling to cadmium dietary exposure for New Zealand

Food Name	New Zealand 15 years and above	
	Baseline	Proposed ML
Honey	0	0
Wheat bran, processed	<1	<1
Wheat germ	<1	<1
Wheat flour	4	3
Wheat wholemeal	<1	<1
Maize flour	<1	<1
Rice, husked	0	0
Wheat bran, unprocessed	<1	<1
Rice, polished	<1	<1
Dried prunes	<1	<1
Dried grapes	<1	<1
Dried apricots	<1	<1
Sugar cane molasses	3	3
Tea, green, black	<1	<1
Blueberries	<1	<1
Grapes	<1	<1
Wine	<1	<1
Strawberry	<1	<1
Oranges, sweet, sour	<1	<1
Orange juice	2	1
Mandarins	<1	<1
Tropical fruit-inedible peel	<1	<1
Avocado	<1	<1
Banana (includes banana dwarf)	<1	<1
Mango	<1	<1
Papaya (Pawpaw)	<1	<1
Pineapple	0	0
Pineapple, juice	<1	<1
Apple	0	0
Apple, juice	<1	<1
Pear	<1	<1
Peach	<1	<1
Plums	<1	<1
Barley	<1	<1
Barley, beer	<1	<1
Oats	<1	<1
Rice	<1	<1
Wheat	<1	<1
Wild rice	<1	<1
Molluscs	<1	<1
Mussels	**6**	**6**

Food Name	New Zealand 15 years and above	
	Baseline	Proposed ML
Oysters	3	2
Scallops	<1	<1
Squids	<1	<1
Cattle milk	0	0
Cattle meat	4	4
Deer meat	<1	<1
Pig meat	<1	<1
Sheep meat	<1	<1
Sheep, edible offal of	<1	<1
Cattle, kidney	2	1
Cattle, liver	<1	<1
Pig, liver	<1	<1
Sheep, kidney	<1	<1
Sheep, liver	<1	<1
Vegetable oil, edible	1	1
Olive oil, refined	<1	<1
Rape seed oil, edible	<1	<1
Peanut oil, edible	<1	<1
Chicken eggs	<1	<1
Chicken meat	<1	<1
Turkey meat	<1	<1
Chicken, edible offal of	<1	<1
Cacao beans	3	3
Coffee beans	<1	<1
Mustard seed	<1	<1
Peanut	1	**11**
Sesame seed	<1	<1
Sunflower seed	<1	<1
Almonds	<1	<1
Cashew nut	<1	<1
Macadamia nuts	<1	<1
Walnuts	0	0
Garlic	<1	<1
Onion, bulb	2	2
Cabbages, head	<1	<1
Broccoli	0	0
Cauliflower	0	0
Melons, except Watermelon	<1	<1
Cucumber	<1	<1
Pumpkins	<1	<1
Watermelon	<1	<1
Lentil (dry)	<1	<1
Soya bean (dry)	<1	<1
Soya bean	0	0

Food Name	New Zealand 15 years and above	
	Baseline	Proposed ML
Leafy vegetables	<1	<1
Lettuce, Head	<1	<1
Egg plant	<1	<1
Peppers, sweet	<1	<1
Sweet corn	<1	<1
Tomato	2	2
Tomato, canned	<1	<1
Fungi, edible (not including cultivated	<1	<1
Mushrooms	0	0
Beans, except broad bean and soya bean	<1	<1
Garden pea, shelled	<1	<1
Root and tuber vegetables	2	1
Beetroot	<1	<1
Carrot	4	4
Potato*	**41**	**37**
Celery	<1	<1
Crabs	<1	<1
Crayfish	<1	<1
Prawns	<1	<1
Diadromous fish	<1	<1
Marine fish of all species	1	1
Shark	<1	<1
Tuna	<1	<1

* Percentage contribution for potato and potato, baked/fried were consolidated for reporting.

Appendix 5

Complete information on comparison of estimated dietary exposures to cadmium with the PTWI

Table A5.1: Estimated dietary exposures to cadmium for *Baseline,* as a percentage of the PTWI

Country	Population group	Number of consumers of cadmium	Consumers♦ as a % of total respondents#	Mean all respondents#	90th percentile consumers♦
		LB/UB		% PTWI*	
Australia	2 years+	13854/13856	100	15 - 15	25 - 30
	2-6 years	989/989	100	25 - 35	50 - 60
New Zealand	15 years+	4632/4635	100	15 - 20	25 - 30

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.
♦ Consumers only – This only includes the people who have consumed a food that contains cadmium.
* PTWI = 0.007 mg/kg bw

Table A5.2: Estimated dietary exposures to cadmium for *Proposed ML* scenario, as a percentage of the PTWI

Country	Population group	Number of consumers of cadmium	Consumers♦ as a % of total respondents#	Mean all respondents#	90th percentile consumers♦
		LB/UB		% PTWI*	
Australia	2 years+	13854/13856	100	15 - 20	30 - 35
	2-6 years	989/989	100	30 - 40	65 - 75
New Zealand	15 years+	4632/4635	100	15 - 20	30 - 35

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.
♦ Consumers only – This only includes the people who have consumed a food that contains cadmium.
* PTWI = 0.007 mg/kg bw

Table A5.3: Estimated dietary exposures to cadmium for *Peanuts Only* scenario, as a percentage of the PTWI

Country	Population group	Number of cadmium consumers	Consumers♦ as a % of total respondents#	Mean all respondents#	Mean consumers♦	90th percentile consumers♦
				% PTWI*		
Australia	2 years+	2986	20	3	10	30
	2-6 years	308	30	7	20	60
New Zealand	15 years+	1046	25	2	8	20

Total number of respondents for Australia: 2 years and above = 13 858, 2-6 years = 989; New Zealand: 15 years and above = 4 636. Respondents include all members of the survey population whether or not they consumed a food that contains cadmium.

♦ Consumers only – This only includes the people who have consumed a food that contains cadmium.

* PTWI = 0.007 mg/kg bw

Attachment 4

Summary of issues raised in the second round of consultation

Submitter	Comment
Cadbury Schweppes	**Supports** the recommendation to increase the ML for cadmium in peanuts from 0.1 to 0.5 mg/kg
Food Technology Association of Australia	**Supports** the recommendation; however, they did not necessarily agree that the ML of 0.5 mg/kg represented the absolute maximum limit for cadmium in peanuts
Queensland Health	**Does not support** the recommendation to increase the ML from 0.1 to 0.5 mg/kg. Queensland Health does not believe that an adequate case has been presented to increase the ML. In particular the following aspects need to be addressed: 1. FSANZ needs to supply evidence to support how an increase to the ML would encourage adherence to good agricultural practice (GAP) in Australia and furthermore how it would be consistent with the established cadmium minimization strategy implemented from 2000 to 2006; 2. The major beneficiaries for the increased ML are some importers and manufacturers who would be able to import inferior, presumably cheaper peanuts; 3. High risk groups such as vegetarians and Torres Strait Islander populations may be more susceptible to increased intakes and this risk is not adequately addressed; 4. 1995 National Nutrition Survey data used for the dietary exposure assessment is out-of-date and inappropriate; 5. Due to the narrow margin of safety between exposure and the PTWI there would need to be substantial other benefits to be gained to justify an erosion of this safety factor when it is already narrow; 6. FSANZ has taken an inconsistent approach on safety in this Application when compared to the assumptions made on the Proposal on cassava chips; 7. The cost-benefits analysis is qualitative rather than quantitative and provides little detail on how the benefits outweigh the costs.
Prolife Foods	Although, Prolife Foods preferred option is removal of the ML, they **support** the proposal to increase the ML to provide more flexibility in sourcing peanuts from a range of countries.
Australian Food and Grocery Council	Supports the risk-based principles by which FSANZ recommended the increase to the ML and that to retain the level of 0.1 mg/kg may be unnecessarily trade restrictive which could be challenged by the WTO. However, notes that there are significant potential costs associated with this recommendation and the benefits for Australian industry and consumers are not clearly quantified. In particular the AFGC identify the following as potential costs: • May undermine the viability of local industry with increased potential for dumping;

Submitter	Comment
	• The increase does not support the concept of As Low As Reasonably Achievable; • Increased use of cheaper fertilisers containing higher levels of cadmium; and • Increased costs to AQIS Imported Food Surveillance Program testing for heavy metals.
Confectionery Manufacturers of Australasia Limited (CMA)	**Supports** the recommendation to increase the ML for cadmium in peanuts from 0.1 to 0.5 mg/kg as FSANZ has determined that there are no public health and safety (PH&S) issues, it will alleviate the current non-tariff trade barrier, promote an efficient and competitive food industry, promote GAP and is not contrary to Australia's international obligations.
SA Department of Health	Industry should be asked to provide more evidence, such as examples of incidences where peanuts have been rejected where the cadmium levels has exceeded 0.1 mg/kg. Therefore, FSANZ should be provided with evidence of insufficient supplies of peanut availability and the subsequent price variation that results for importers. SA also questions whether it would be more prudent to await results of the 2008 Australian Total Diet Survey to ensure that the levels for cadmium used in the current assessment reflect what consumers may be exposed to from the diet
Department of Human Services Victoria	**Supports** the recommendation to increase the ML for cadmium in peanuts from 0.1 to 0.5 mg/kg
Food and Beverage Importers Association	**Supports** the recommendation as there are no public health and safety concerns and it would ensure a timely and cost-effective supply of peanuts.
MWT Foods Victoria	**Supports** the recommendation to increase the ML for cadmium in peanuts from 0.1 to 0.5 mg/kg as FSANZ has determined that there are no public health and safety issues, it will alleviate the current non-tariff trade barrier, promote an efficient and competitive food industry, promote GAP and is not contrary to Australia's international obligations.
NSW Food Authority	Does not object to further consideration of this Application. The Authority notes that there are many measures in place in Australia to minimize cadmium levels in soils and agrees that there are no PH&S issues as peanuts are a minor contributor to total dietary exposure. However, the NSW Food Authority would urge FSANZ to seek quantitative information from the CMA to support the view that the current ML of 0.1 mg/kg represents a trade barrier.
New Zealand Food Safety Authority (NZFSA)	**Supports** the recommendation to increase the ML for cadmium in peanuts from 0.1 to 0.5 mg/kg as FSANZ has determined that there are no public health and safety issues, it will alleviate the current non-tariff trade barrier, promote an efficient and competitive food industry, promote GAP and is not contrary to Australia's international obligations.
Peanut Company of Australia (PCA)	The PCA **does not support** the increase to the ML and believes that no submissions have demonstrated a health or economic benefit, there is no problem in sourcing peanuts with levels that meet the current ML and that the increase will be anti to the cadmium minimisation strategy that Australia has implemented.

Submitter	Comment
	To ensure a steady supply of peanuts in future years, the PCA has commenced purchasing and developing farming areas specifically for peanuts production. These properties are strategically located and represent a very significant investment. The PCA still questions whether the dietary exposure assessment takes into account populations (e.g. vegetarians) that may have higher intakes of peanuts and the subsequent health implications.
Michelle Gibbs (consumer)	**Does not support** the recommendation to increase the ML from 0.1 to 0.5 mg/kg due to the following reasons: • It does not maintain cadmium at a level commensurate with the principle of ALARA; • Increasing the ML will place females at greater risk of fractures from osteoporosis as cadmium has been implicated in being a contributor to osteoporosis; • The CMA has not provided sufficient evidence that there is a trade barrier. Further evidence should be sought from independent groups regarding cadmium contents of peanuts required from confectionary purposes, costs of peanuts and trade barriers for the CMA claims to be substantiated.
Rose Skerten (consumer)	Did not offer a specific position but raised a number of points in regard to public health aspects of cadmium, the limited safety factors inherent in the PTWI, the lack of currency on the dietary survey data, increasing the ML is counterproductive to Australia's National Cadmium minimization Strategy, the CMA should reveal evidence that economic aspects are substantial if the ML is not increased, the environmental effects of cadmium have not been considered in the report.
Clara Madden (consumer)	Gave provisional support pending that an increase in the ML did not cause PH&S issues and that the CMA could prove their claim that the current cadmium ML for peanuts is causing a trade barrier. Proposed lower MLs of either 0.2 or 0.3 mg/kg supported by the data that already existed in the DAR.
Box Hill TAFE (Food Teachers)	**Does not support** the recommendation to increase the ML due to the cumulative properties of cadmium and that it would decrease the current high standard for peanuts in Australia.