FOOD STANDARDS
Australia New Zealand
Te Mana Kounga Kai – Ahitereiria me Aotearoa

6-04
4 August 2004

DRAFT ASSESSMENT REPORT

APPLICATION A493

IODINE AS A PROCESSING AID

DEADLINE FOR PUBLIC SUBMISSIONS to FSANZ in relation to this matter:
15 September 2004
(See 'Invitation for Public Submissions' for details)

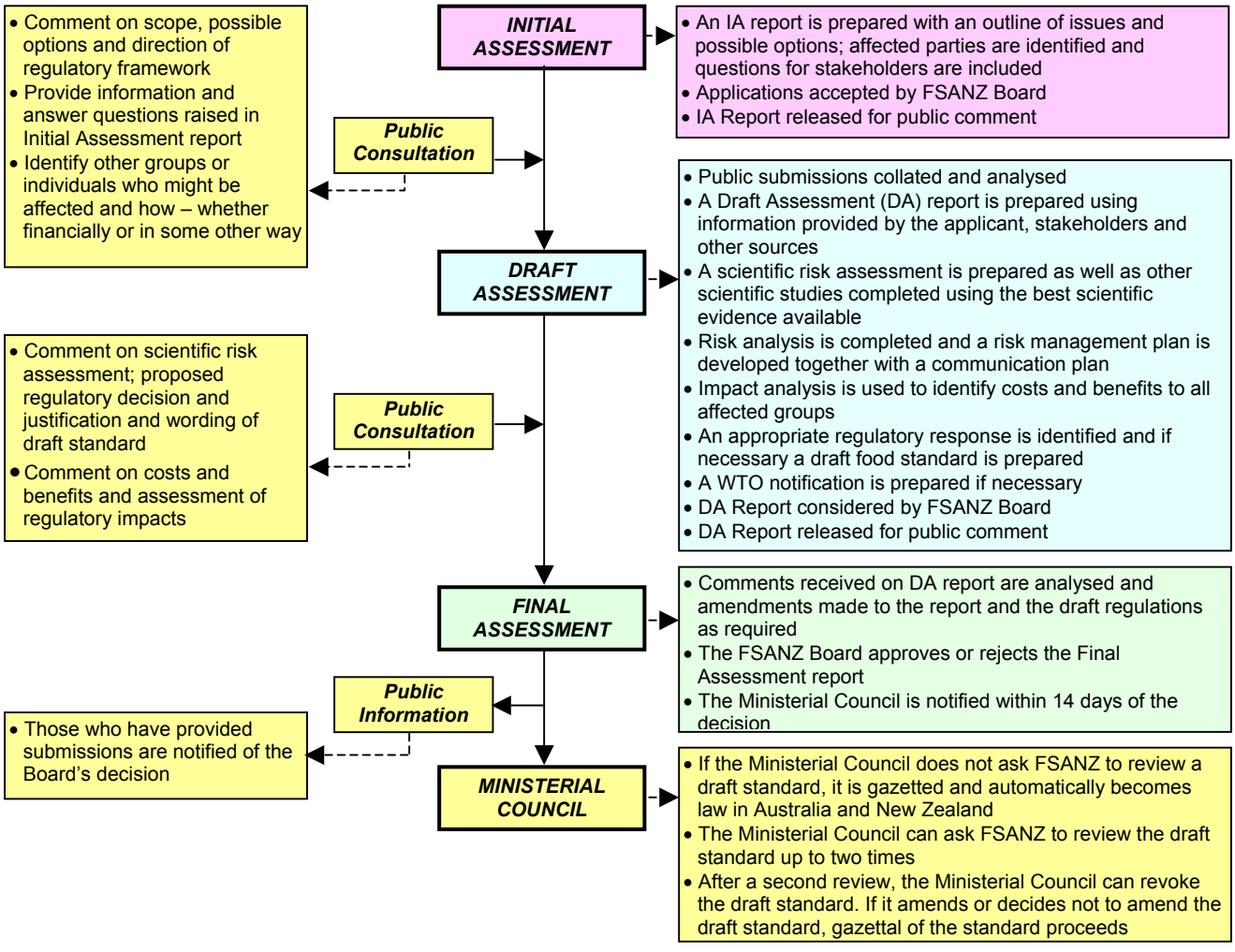
FOOD STANDARDS AUSTRALIA NEW ZEALAND (FSANZ)

FSANZ's role is to protect the health and safety of people in Australia and New Zealand through the maintenance of a safe food supply. FSANZ is a partnership between ten Governments: the Commonwealth; Australian States and Territories; and New Zealand. It is a statutory authority under Commonwealth law and is an independent, expert body.

FSANZ is responsible for developing, varying and reviewing standards and for developing codes of conduct with industry for food available in Australia and New Zealand covering labelling, composition and contaminants. In Australia, FSANZ also develops food standards for food safety, maximum residue limits, primary production and processing and a range of other functions including the coordination of national food surveillance and recall systems, conducting research and assessing policies about imported food.

The FSANZ Board approves new standards or variations to food standards in accordance with policy guidelines set by the Australia and New Zealand Food Regulation Ministerial Council (Ministerial Council) made up of Commonwealth, State and Territory and New Zealand Health Ministers as lead Ministers, with representation from other portfolios. Approved standards are then notified to the Ministerial Council. The Ministerial Council may then request that FSANZ review a proposed or existing standard. If the Ministerial Council does not request that FSANZ review the draft standard, or amends a draft standard, the standard is adopted by reference under the food laws of the Commonwealth, States, Territories and New Zealand. The Ministerial Council can, independently of a notification from FSANZ, request that FSANZ review a standard.

The process for amending the *Australia New Zealand Food Standards Code* is prescribed in the *Food Standards Australia New Zealand Act 1991* (FSANZ Act). The diagram below represents the different stages in the process including when periods of public consultation occur. This process varies for matters that are urgent or minor in significance or complexity.



INVITATION FOR PUBLIC SUBMISSIONS

FSANZ has prepared a Draft Assessment Report of Application A493; and prepared a draft variation to the *Australia New Zealand Food Standards Code* (the Code).

FSANZ invites public comment on this Draft Assessment Report based on regulation impact principles and the draft variation to the Code for the purpose of preparing an amendment to the Code for approval by the FSANZ Board.

Written submissions are invited from interested individuals and organisations to assist FSANZ in preparing the Draft Assessment for this Application. Submissions should, where possible, address the objectives of FSANZ as set out in section 10 of the FSANZ Act. Information providing details of potential costs and benefits of the proposed change to the Code from stakeholders is highly desirable. Claims made in submissions should be supported wherever possible by referencing or including relevant studies, research findings, trials, surveys etc. Technical information should be in sufficient detail to allow independent scientific assessment.

The processes of FSANZ are open to public scrutiny, and any submissions received will ordinarily be placed on the public register of FSANZ and made available for inspection. If you wish any information contained in a submission to remain confidential to FSANZ, you should clearly identify the sensitive information and provide justification for treating it as commercial-in-confidence. Section 39 of the FSANZ Act requires FSANZ to treat in-confidence, trade secrets relating to food and any other information relating to food, the commercial value of which would be, or could reasonably be expected to be, destroyed or diminished by disclosure.

Submissions must be made in writing and should clearly be marked with the word 'Submission' and quote the correct project number and name. Submissions may be sent to one of the following addresses:

Food Standards Australia New Zealand
PO Box 7186
Canberra BC ACT 2610
AUSTRALIA
Tel (02) 6271 2222
www.foodstandards.gov.au

Food Standards Australia New Zealand
PO Box 10559
The Terrace WELLINGTON 6036
NEW ZEALAND
Tel (04) 473 9942
www.foodstandards.govt.nz

Submissions should be received by FSANZ **by 15 September 2004**.

Submissions received after this date may not be considered, unless the Project Manager has given prior agreement for an extension.

While FSANZ accepts submissions in hard copy to our offices, it is more convenient and quicker to receive submissions electronically through the FSANZ website using the Standards Development tab and then through Documents for Public Comment. Questions relating to making submissions or the application process can be directed to the Standards Management Officer at the above address or by emailing slo@foodstandards.gov.au.

Assessment reports are available for viewing and downloading from the FSANZ website. Alternatively, requests for paper copies of reports or other general inquiries can be directed to FSANZ's Information Officer at either of the above addresses or by emailing info@foodstandards.gov.au.

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Executive Summary and Statement of Reasons

Food Standards Australia New Zealand received an application on 21 February 2003 from Ioteq Limited (formerly Iodine Technologies Australia Pty Ltd) to approve the use of iodine as a processing aid under Standard 1.3.3 Processing Aids of the *Australia New Zealand Food Standards Code* (the Code).

Iodine has a long history of use as a water disinfectant, and is also used as a sanitising compound (in iodophors) by the dairy industry. The purpose of this application is to seek approval for the use of iodine for the surface sanitisation of foods, specifically fruit, vegetables, nuts and eggs.

Sanitising agents are used at all levels during food manufacture and processing to reduce the levels of pathogens and spoilage organisms on the surface of foods. Chlorine-based washing systems are by far the most commonly used but are said to possess a number of disadvantages. The Applicant has developed an iodine-based washing system as an alternative to chlorine-based systems.

Under Standard 1.3.3, processing aids are required to undergo pre-market approval in Australia and New Zealand. There is currently no approval for the use of iodine as a processing aid in the Code, although the Australian Pesticides and Veterinary Medicines Authority (APVMA) have registered iodine for use in the Applicant's proprietary system for the post harvest sanitisation of whole fruits and vegetables. This application, if successful, will broaden this use to eggs as well as minimally processed fruits and vegetables, such as fresh cut produce and will also provide clarity in that all proposed uses will be covered by a permission in the Code.

The objective of this assessment is to determine whether it is appropriate to amend the Code to permit the use of iodine as a washing agent for fruit, vegetables, nuts and eggs at good manufacturing practice levels. A range of issues was considered during the assessment, including the technological justification for the use of iodine and the potential impact on public health and safety.

The use of iodine as a washing agent for fruits, vegetables, nuts and eggs is technologically justified. Iodine is superior to chlorine at equivalent concentrations in reducing the number of surface organisms on food and a technological need exists for suitable alternatives to the currently available sanitisers.

The risk assessment indicates that the use of iodine as proposed may result in a small increase in iodine intake but not to a level that would raise safety concerns for the vast majority of the population or pose any adverse nutritional risks. The potential for the safe intake level for iodine to be exceeded is low and any observed increase in iodine intake is unlikely to cause imbalances with other nutrients. In the case of vulnerable individuals, the proposed use of iodine is considered unlikely to pose any additional risks.

Two regulatory options were identified in the assessment – to either approve or not approve the use of iodine as a processing aid. Following an assessment of the potential impact of each of the options on the affected parties (consumers, the food industry and government), the preferred option would be to approve the use of iodine as a processing aid.

This option potentially offers significant benefits to the food industry and consumers with very little associated negative impact. The proposed variation to the Code is therefore considered necessary, cost effective and of net benefit to both the food industry and consumers.

The Initial Assessment Report for this Application was circulated for public comment on 21 May 2003 for a period of six weeks. A total of 12 submissions were received, the majority of which were either supportive of the Application or reserved judgement until the Draft Assessment Report is available. The issues raised in submissions are addressed in this report.

Statement of Reasons

The draft variation to Standard 1.3.3 – Processing Aids of the Code (**Attachment 1**), approving the use of iodine as a processing aid, is recommended for the following reasons:

- the use of iodine as a washing agent for fruit, vegetables, nuts and eggs is technologically justified – the efficacy of iodine as a sanitising agent for foods has been demonstrated and a technological need exists for alternative food sanitisers;

- the use of iodine as proposed may result in a small increase in iodine intake but not to a level that would raise safety concerns for the vast majority of the population or pose any adverse nutritional risks. The proposed use of iodine is also considered unlikely to pose any additional risk for vulnerable individuals;

- the proposed draft variation to the Code is consistent with the section 10 objectives of the FSANZ Act. In particular, FSANZ has addressed the protection of public health and safety by undertaking a risk assessment based on the best available scientific data.

- The regulation impact assessment has concluded that the benefits of permitting use of iodine as a washing agent outweigh any costs associated with its use.

If approved, the variation to the Code will come into effect on the date of gazettal.

1. Introduction

Food Standards Australia New Zealand (FSANZ) received an Application on 21 February 2003 from Ioteq Limited (formerly Iodine Technologies Australia Pty Ltd) to approve the use of iodine as a processing aid under Standard 1.3.3 Processing Aids of the Code. It is proposed to use iodine as a washing/sanitising agent for foods.

A Draft Assessment of the Application has been completed and public comment is now being sought to assist with the Final Assessment.

2. Regulatory Problem

Under Standard 1.3.3, processing aids are required to undergo pre-market approval in Australia and New Zealand. According to Standard 1.3.3, processing aid means:

> a substance listed in clauses 3 to 18, where –
>
> (a) the substance is used in the processing of raw materials, foods or ingredients, to fulfil a technological purpose relating to treatment or processing, but does not perform a technological function in the final food; and
>
> (b) the substance is used in the course of manufacture of a food at the lowest level necessary to achieve a function in the processing of that food, irrespective of any maximum permitted level specified.

There is currently no approval for the use of iodine as a processing aid in the Code, therefore the Applicant has applied to have permission for iodine as a washing agent inserted in the Table to clause 12 of Standard 1.3.3. The substances listed in this Table may be used as bleaching agents, washing and peeling agents in the course of manufacture of the corresponding foods specified in the Table provided the final food contains no more than the corresponding maximum permitted level specified in the Table.

The Applicant originally requested an amendment to Standard 1.3.3 to allow iodine to be used as a washing agent for all foods at good manufacturing practice (GMP)[1] levels. The Applicant has since amended their original application and now seeks approval for iodine as a washing agent for fruit, vegetables (which includes herbs and nuts)[2] and eggs at GMP levels.

3. Objective

The purpose of this assessment is to determine whether it is appropriate to amend Standard 1.3.3 of the Code to permit the use of iodine as a processing aid.

In developing or varying a food standard, FSANZ is required by its legislation to meet three primary objectives, which are set out in section 10 of the FSANZ Act. These are:

- the protection of public health and safety;

[1] Under GMP, the amount of iodine used should be the minimum amount necessary to have the intended effect (i.e. sanitisation).

[2] Standard 2.3.1 Fruit and Vegetables of the Code defines fruit and vegetables as meaning fruit, vegetables, nuts, spices, herbs, fungi, legumes and seeds.

- 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.

4. Background

4.1 Iodine as a Sanitising Agent

Iodine is a member of the halogen family of chemical elements. Like other halogens, such as chlorine and bromine, it has strong anti-microbial activity. Chlorine is the more commonly used halogen and various forms of compounds that deliver chlorine (including hypochlorite) have been used for many years as sanitising agents by the food industry. Elemental iodine has a long history of use as a water disinfectant, and is also used as a sanitising compound (in iodophors) by the dairy industry.

Sanitising agents are used at all levels during food manufacture and processing to reduce the levels of pathogens and spoilage organisms on the surface of foods. The use of sanitising agents is therefore important for improving the safety of food as well as keeping quality and shelf life. Sanitising agents typically do not kill all bacteria (so they are not sterilising agents) but tend to inhibit the growth of the bacteria to acceptable levels.

The Applicant has developed a fully automated and enclosed post harvest sanitising system – the Iodoclean™ System – which uses elemental iodine as the active ingredient to reduce the levels of bacteria and fungi on the surface of food, particularly fresh produce. The sanitising system delivers iodine in treatment water at a controlled concentration, which can be set within the range of 3 to 30 ppm.

The iodine concentration used depends on the contact time and the microbial load on the product to be treated. This system is being promoted as a viable alternative to chlorine for the sanitation of food and is said to offer several advantages over chlorine.

4.2 Iodine Fortification

While not relevant to consideration of this Application, the Ministerial Council has recently agreed to a new policy guideline for the fortification of foods with vitamins and minerals[3]. This guideline recognises particular circumstances in which mandatory fortification to meet public health need is appropriate. FSANZ plans to raise a separate proposal in the near future to investigate the need for increased iodine content in the Australia New Zealand food supply.

4.3 Other Regulatory Approvals

4.3.1 National Approvals

APVMA registered iodine, under the product name of Biomaxa Iodine Granules Post Harvest Sanitiser, on 6 November 2002. The iodine granules are to be used only with the Iodoclean™ System, a fully automated post harvest sanitising system to assist in the control of bacterial and fungi on a range of whole fruits and vegetables.

Under this registration, the APVMA have granted a Table 5 exemption for iodine, which means iodine is permitted as a post harvest sanitiser without the necessity for the setting of a Maximum Residue Limit (MRL). This covers situations where residues do not or should not occur in foods or animal feeds; or where the residues are identical to or indistinguishable from natural food components; or are otherwise of no toxicological significance.

Notwithstanding that, in this instance, the Applicant has an application pending with FSANZ for the use of iodine as a processing aid, this is not inconsistent with the registration and Table 5 exemption provided by the APVMA. Nor does the registration and Table 5 exemption impact on consideration of this application by FSANZ.

The fact there is currently no permission for the use of iodine as a processing aid under the Code is not actually contradictory to the APVMA registration. It is not necessary, in all cases, where there is registration with the APVMA for there to be a corresponding permission in the Code. A number of post harvest sanitizers are in use with APVMA registration for which there is no corresponding permission in the Code. The use of these chemicals pursuant to the APVMA registration would not amount to a breach of the Code. This also applies in the case of the Iodoclean™ system.

In addition to the above, a variety of iodine-based compounds are approved in both Australia and New Zealand for use as teat/udder sanitisers, general equipment sanitisers and for food contact surfaces.

4.3.2 Overseas Approvals

Overseas legislation for the use of iodine as a sanitising agent for foods primarily relates to its use on food contact surfaces. The relevant regulations are:

- United States Code of Federal Regulations Title 21, 178.1010 – Sanitising Solutions (food contact surfaces and utensils only).

[3] http://www.foodsecretariat.health.gov.au/policydocs.htm

- United Kingdom Statutory Instrument 1999 No 919, Schedule 2 – Approved Disinfectants (sanitisation of dairies).

A range of provisions also exists for the short-term use of iodine to treat water supplies in emergency situations.

The Applicant has also advised FSANZ that they are currently seeking registration of iodine, as used in the Iodoclean™ System, as a biochemical pesticide by the United States Environment Protection Agency.

5. Relevant Issues

5.1 Technological Justification

The assessment of technological justification considered the technological function and efficacy of iodine as a sanitising agent and evidence of technological need. The full Report is at **Attachment 2**.

Overall it was concluded that the use of iodine as a sanitising agent for foods is technologically justified. Iodine is superior to equivalent concentrations of chlorine in reducing the number of surface organisms on food and this is also the case in the presence of dirt, which is known to decrease the efficacy of chlorine wash solutions. There is a technological need for safe and effective sanitisers for use on food, and iodine would appear to be a useful alternative to the chlorine-based wash systems currently available.

A number of submissions (Sydney Postharvest Laboratory, Food Science Australia and Dept of Crop Sciences, University of Sydney) also raised the issue of technological need, commenting on the shortage of effective sanitisers. The approval of iodine as a washing agent for foods would be considered by them to be a very useful addition to the available sanitisers.

5.2 Risk Assessment

5.2.1 Safety of Iodine

Iodine is an essential component of the diet, however, as with many other essential nutrients, intakes in excess of physiological requirements may produce adverse effects. In the case of iodine, it is thyroid gland function and the regulation of thyroid hormone production and secretion that may be adversely affected.

FSANZ has undertaken a review of the toxic effects associated with excess dietary iodine, the full report of which is at **Attachment 3.** The findings of this assessment are briefly summarised below.

Excess iodine can produce an enlargement of the thyroid gland (goitre) and/or affect the production of the thyroid hormones. A diminished production of the thyroid hormones is referred to as hypothyroidism (and may be accompanied by goitre) and increased thyroid hormone synthesis and secretion by the thyroid gland is referred to as hyperthyroidism.

The effect on the thyroid depends on the current and previous iodine status of the individual and any current or previous thyroid dysfunction. For example, individuals with a history of iodine deficiency may be prone to the development of iodine-induced hyperthyroidism if iodine exposure increases later in life.

The human response to excess iodine can be quite variable. Some individuals can tolerate quite large intakes (up to 50 µg/kg/day) while others may respond adversely to levels close to recommended intakes (3-7 µg/kg/day). Individuals responding adversely to relatively low intake levels typically have an underlying thyroid disorder or have a long history of iodine deficiency.

For the majority of healthy individuals, the most sensitive endpoint for iodine toxicity is sub-clinical hypothyroidism. Sub-clinical hypothyroidism is defined as an elevation in thyroid stimulating hormone concentration while serum thyroid hormone concentration is maintained within the normal range of values for healthy individuals. While not clinically adverse, such an effect, if persistent, could lead to clinical hypothyroidism. In healthy adults, such an effect has been associated with acute iodine intakes of 1700 µg/day (24 µg/kg body weight/day for a 71 kg person), and for children, has been associated with chronic intakes of 1150 µg/day (29 µg/kg/day for a 40 kg child).

Iodine intakes of approximately 1000 µg/day however appear to be well tolerated by healthy adults. This level has been used by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) to establish a provisional maximum tolerable daily intake (PTDI)[4] for iodine of 17 µg/kg bw from all sources. FSANZ has adopted this level as a safe intake level for the purpose of risk assessment for the general healthy population.

For those individuals with thyroid disorders or a long history of iodine deficiency, this PTDI is not applicable since these individuals may respond adversely at levels of intake below the PTDI. It has been reported that intakes in the range 3-7 µg/kg/day may be sufficient to produce an increase in hyperthyroidism in chronically iodine deficient individuals. The health risk for these individuals needs to be considered separately from the general population.

5.2.2 Dietary Exposure Assessment

A dietary exposure assessment was done to estimate current and potential exposure to iodine from the diet if approval for the use of iodine as a processing aid for fruit, vegetables, nuts and eggs is granted. The full dietary exposure assessment report is at **Attachment 4**. The results of the exposure assessment are briefly summarised below.

Estimated dietary intakes of iodine were calculated for the Australian and New Zealand populations, and for the population sub-group of Australian children aged 2-6 years. This was to ensure that iodine intakes would not exceed the PTDI if approval to use elemental iodine as a washing agent were granted.

[4] PTDIs represent the permissible human exposure to those contaminants unavoidable associated with the consumption of otherwise wholesome and nutritious food, and is a level of intake that is considered safe over a life time.

As iodine is also an essential micronutrient, dietary intakes were also assessed for a range of age-gender categories for the purpose of comparison with the Estimated Average Requirements (EARs)[5] for iodine. The results of these comparisons are discussed in the nutrition risk assessment report at **Attachment 6** and below in section 5.2.3.

Baseline intakes of iodine were calculated using naturally occurring iodine concentrations. Two post-treatment scenarios were examined in each dietary intake assessment: Scenario 1 applied a peeling factor to those fruits and vegetables washed with iodine that may be consumed with the peel either on or off (e.g. apples); and Scenario 2 assumed that fruits and vegetables washed with iodine that may be consumed with the peel on or off were always consumed unpeeled.

Scenario 1 is considered to represent a more accurate estimate of the likely extent to which an elemental iodine wash will impact on dietary iodine intakes for Australian and New Zealand population groups. Scenario 2 is a worst-case scenario.

Both of the above scenarios have assumed that iodine would be used on all fruit and vegetables consumed. Given the widespread availability and use of other sanitising treatments for fruit and vegetables, this is a very conservative assumption.

Estimated mean and 95th percentile dietary intakes of iodine were below the PTDI of 17 µg/kg body weight/day for all population groups and all scenarios examined, with one exception. Although the dietary intakes of iodine for the 2-6 years age group were below the PTDI, analysis of the 2-3 years high consumers (95th percentile) group indicated the PTDI was exceeded when it was assumed that fruits and vegetables that may be consumed with the peel on or off were consumed unpeeled (Scenario 2). Australian children aged 2-3 years had an estimated 95th percentile dietary intake of 17.2 µg/kg bw/day (260 µg/person/day), which is equivalent to 101.2% of the PTDI.

In addition to the above, a second dietary exposure assessment was undertaken to consider the impact of a second application currently being assessed by FSANZ. This application (Application A528) is seeking to increase the maximum permitted quantity of iodine in formulated supplementary foods for young children (FSFYC) from 35µg to 70 µg per serving. FSFYC are defined in the Code as formulated supplementary food for children aged 1 – 3 years. A second exposure assessment was therefore undertaken to look at the effect on iodine intake in 2-3 year olds from using iodine as a washing agent (Scenario 1 and 2 above) in combination with increasing the maximum iodine limit in FSFYC (**Attachment 5**).

Estimated mean and 95th percentile dietary intakes of iodine were below the PTDI of 17 µg/kg body weight/day for the baseline scenario and for Scenario 1. For Scenario 2, the mean dietary intake of iodine was also below the PTDI, however at the 95th percentile, children aged 2-3 years had an estimated intake of 18.7 µg/kg bw/day (289.4 µg/person/day), which is equivalent to 109.7% of the PTDI.

[5] The EAR is defined as the level below which 50 percent of the population may be at risk of having inadequate dietary intake and is used to estimate the prevalence of inadequate intakes in a population.

Given the conservative assumptions built into these dietary exposure assessments, it can be concluded that, under normal conditions of use, it would be unlikely for the use of iodine as a washing agent to result in a dietary exposure above the PTDI for any age group in the general population.

5.2.3 Nutrition Assessment

A nutrition assessment was undertaken to consider the current iodine status of the Australian and New Zealand populations, and to compare this with the results of the dietary exposure assessment (**Attachment 4**) in order to subsequently determine the nutritional risks, if any, to Australian and New Zealand populations from the proposed amendments to the Code. The full nutrition risk assessment report is at **Attachment 6**. A brief summary of the findings is given below.

Several published studies, measuring urinary iodine concentration, have investigated the iodine status of various populations in Australia and New Zealand. Urinary iodine measures are more indicative of population iodine status than measures of dietary iodine intake. The general conclusion from these studies is that a sizeable proportion of Australians and New Zealanders suffer from iodine deficiency to varying extents.

In addition to examining studies of urinary iodine concentration, dietary modelling has been conducted to determine the percentage of Australian and New Zealand populations not meeting the Estimated Average Requirement (EAR) for iodine intake (baseline intake data). Results of the dietary modelling, based on Australian and New Zealand National Nutrition Surveys (NNS), suggest that between 41 and 65 percent of the Australian and New Zealand populations are not meeting the EAR for dietary iodine intake.

Although not directly comparable, the general inference from both types of data is that a considerable proportion of Australians and New Zealanders are mildly iodine deficient. Data on the median urinary iodine levels in Australian and New Zealand populations suggests the baseline levels of iodine intake used in the dietary modelling for this application may be slightly higher than in reality.

In terms of interactions with other nutrients, there is no literature to suggest that iodine competes with, or inhibits the bioavailability of any other nutrient. This suggests that increasing the levels of dietary iodine intake will not have an adverse consequential effect on the nutritional status of consumers.

5.2.4 Risk Characterisation

5.2.4.1 Safety

Healthy population

The data support the safety of iodine as a washing agent for the specified foods for the normal healthy population. Exposure for all population groups, with the exception of 2-3 year olds, is predicted to be below the PTDI, even at the 95th percentile (high consumer) exposure level and applying the worst-case scenario (Scenario 2).

Estimated exposure for 2-3 year olds at the 95th percentile level is estimated to only marginally exceed the PTDI but this is only if the worst-case scenario is applied. If a more realistic scenario (Scenario 1) is applied, the 95th percentile exposure level is below the PTDI.

As the PTDI represents a level of intake that is considered safe over a life time, short-term intakes over the PTDI generally do not raise any safety concerns as the PTDI is not itself a threshold for toxicity. In spite of this however, the potential to exceed the PTDI, even by 2-3 year olds, is considered low, as the dietary exposure estimates tend to be over-estimates as a result of the conservative nature of the assumptions made.

These conservative assumptions include:

- all eligible fruit, vegetable, nut and egg commodities will be washed with iodine – i.e. 100% market share.

  (This is extremely unlikely given the availability of other commercial sanitisers. The market share is likely to be significantly less than 100%. On this basis alone, the estimated dietary exposure is therefore likely to be far less than indicated.)

- all fruits, vegetables, nuts and eggs will not be rinsed after the iodine wash, or prior to preparation and consumption in the home;

  (Fruit and vegetables are often washed prior to consumption and this will most likely reduce the actual exposure to iodine.)

- there are no reductions in iodine concentrations on cooking.

  (In the case of some vegetables, cooking will certainly reduce the concentration of iodine.)

In reality, it is expected that the iodine concentrations on the fruit and vegetables will be considerably less than those concentrations used in the dietary modelling, and that the percentage of fruit and vegetables treated with iodine will be considerably less than 100%.

In addition to these conservative assumptions, a limitation of the model used is that only 24-hour survey data are available, which tends to over-estimate consumption for high consumers. Therefore, predicted high percentile intakes are likely to be higher than actual high percentile intakes over a lifetime.

Another relevant consideration is that according to data on the median urinary iodine concentration in Australian and New Zealand populations (see Section 5.2.3 above and **Attachment 6**) the baseline levels of iodine intake used in the dietary exposure assessment may be slightly overestimated. It's currently not possible to determine the impact this overestimation might have on the exposure estimates, although potentially it could be significant.

Vulnerable individuals

In relation to the vulnerable individuals identified in the safety assessment, further consideration is necessary. Under certain circumstances these individuals may respond to excess iodine in the diet by developing thyrotoxicosis (also referred to as iodine-induce hyperthyroidism) (discussed in detail in **Attachment 3**). Symptoms include rapid heartbeat, nervousness, weakness, heat intolerance, and weight loss. The most vulnerable are those over 40 years of age who have a long history of iodine deficiency, although individuals with underlying thyroid disorders may also be affected.

Comparison of estimated intakes with the PTDI is not appropriate when considering the health risk for these individuals, as typically they respond adversely to levels of intake that fall below the PTDI and, in some cases, at levels that approximate recommended dietary intakes (RDIs). Such individuals may therefore potentially be at risk even from natural fluctuations in the iodine levels in foods.

In the case of individuals with underlying thyroid disease, such as Graves' disease, the risk is considered low. Such individuals will typically be under the care of a medical professional, therefore should there be any exacerbation of the condition, this should be detected quickly and remedial action taken. In the case of individuals with a long history of iodine deficiency, there may be cause for greater concern as such individuals may not be aware of their condition. Typically however, iodine-induced hyperthyroidism is mild and self-limiting and readily treated. In addition, although there is evidence of iodine deficiency in the Australian and New Zealand populations, the deficiency is believed to be relatively mild. For this reason very few individuals would be expected to be vulnerable to the occurrence of iodine-induced hyperthyroidism.

The proposed use of iodine as a sanitising agent is therefore not considered to pose any increased risk to these individuals – it is not expected to increase dietary iodine intake to any significant extent and very few vulnerable individuals would exist in the Australian and New Zealand populations.

Conclusion

For the vast majority of the population, there are no safety concerns associated with the use of iodine as a washing agent for fruit, vegetables, nuts and eggs. Also, because the proposed use of iodine is expected to have only a small impact on dietary iodine intake, it is not considered to pose any additional risks to vulnerable individuals, in particular those with a long history of iodine deficiency.

5.2.4.2 Nutrition Considerations

As with the use of iodophors, the use of iodine as a processing aid may result in adventitious contamination of the food supply. It is very unlikely that the observed increase in iodine intake as a result of this Application will cause imbalances with other nutrients; to the contrary, it may have the beneficial outcome of helping to replete populations with poor iodine status. There are no identified adverse nutritional risks created by the proposed amendment to the *Food Standards Code.* The use of iodine as a processing aid, and its contribution to iodine intake, would need to be taken into account should any iodine fortification programs be contemplated in the future.

5.2.4.3 Overall conclusion

The use of iodine as a washing agent for fruit, vegetables, nuts and eggs is likely to result in a small in increase in iodine intake but not to a level that would raise any safety concerns or pose any nutritional risks for the vast majority of the population, or pose any additional risks for vulnerable groups.

5.3 Issues Raised in Public Submissions

5.3.1 Classification of Iodine as a Processing Aid.

The Western Australian Food Advisory Committee, of the WA Department of Health noted that iodine residues are more likely to remain on food surfaces and reach the final consumer, and that this raises the issue of the appropriateness of classifying iodine as a processing aid. The New Zealand Food Safety Authority (NZFSA) submitted that as part of the assessment of the application, FSANZ should consider the function of iodine when used as a sanitising agent to determine if it is solely acting as a processing aid, or is there also a food additive function.

5.3.1.1 Response

To be classified as a processing aid a substance must fulfil a technological purpose relating to treatment or processing, but should not perform a technological function in the final food. Substances performing a technological function in the final food would generally be classed as a food additive.

It is correct that residues may remain on the surface of foods that have been washed with iodine. However, the iodine remaining on these foods will largely be in the form of iodide, which has virtually no biocidal activity. Any iodine remaining in the final food is therefore unlikely to be performing a technological function related to sanitisation. Sanitisation occurs exclusively during the washing process, which is optimised to ensure the greatest contact with the most biocidally active forms of iodine – elemental iodine (I_2) and hypoiodous acid (which forms from the reaction of I_2 with water).

5.3.2 Iodine Deficiency

Two submissions were made that commented on the issue of iodine deficiency. The Dietitians Association of Australia (DAA) submitted that the reintroduction of iodine as a sanitising agent may be a suitable replacement for chlorine-based sanitising agents, but should not be seen as a way of correcting nutritional inadequacies. The Australian Food and Grocery Council (AFGC) noted recent concerns expressed by others suggesting that iodine consumption in Australia may be below optimum levels, and stated that mandatory iodine fortification should be considered for certain foods. The AFGC mentioned that this outcome was a point in favour of approving this Application.

5.3.2.1 Response

The Dietary Exposure Assessment (**Attachment 4**) has shown that the use of iodine as a washing agent for fruit, vegetables, nuts and eggs may increase the dietary iodine intake of Australian and New Zealand populations to some extent.

The Dietary Exposure Assessment is based on a conservative assumption that food producers will exclusively use iodine for sanitisation, in which case some population groups may experience a significant increase in their iodine intake, although not to a level that would raise safety or nutritional concerns. However, in reality it is highly unlikely the use of iodine will achieve a 100% market share, therefore the increase in iodine intake is likely to be considerably smaller than estimated.

Further clarification of the population iodine status is required, however current evidence indicates that some level of iodine deficiency exists among Australian and New Zealand populations. A positive public health benefit may therefore occur if iodine is approved as a washing agent. Even so, FSANZ agrees that it would not be appropriate to rely on an iodine wash as a means of addressing iodine deficiency, particularly as its only likely to result in small increases in iodine intake. Instead, any strategies to address iodine deficiency should be developed through work on food fortification. FSANZ has received policy guidance on fortification from Australian and New Zealand Health Ministers, and will be undertaking such work on iodine as a matter of priority.

5.3.3 Bioavailability of Iodine

The NZFSA submitted that as part of the assessment of the application, FSANZ should consider the bioavailability of iodine from this source.

5.3.3.1 Response

The majority of the iodine remaining on the surface of the food following treatment will be in the form of iodide, which is nearly 100% bioavailable. Other chemical forms of iodine, such as elemental iodine and iodate, if they were present, would tend to undergo reduction to iodide in the small intestine before absorption.

A conservative approach is taken in the dietary exposure assessment where it is assumed all iodine present in foods is 100% bioavailable, and there are no inhibitors to iodine absorption (such as goitrogens) present in the diet.

6. Regulatory Options

The following two regulatory options have been considered:

Option 1. Maintain the *status quo* and not approve the use of iodine as a food processing aid.

Option 2. Amend the Code and approve the use of iodine as a food processing aid.

7. Impact Analysis

7.1 Affected Parties

The affected parties to this Application include:

- consumers;

- those sectors of the food industry wishing to produce and market food products that have been washed with sanitising agents; and

- Australian Government, State, Territory and New Zealand Government enforcement agencies.

7.2 Impact of Regulatory Options

In the course of developing food regulatory measures suitable for adoption in Australia and New Zealand, FSANZ is required to consider the impact of all options on all sectors of the community, including consumers, the food industry and governments.

7.2.1 Option 1

There are no perceived benefits to the food industry, consumers or government agencies if this option is taken. Parties potentially disadvantaged by not permitting iodine to be used as a washing agent on foods are those sectors of the food industry, including the Applicant, who wish to use iodine as an alternative to existing washing agents and who have invested in the development in the Iodoclean™ System.

7.2.2 Option 2

This option is likely to deliver a benefit to the food industry in that it will provide a cost-effective alternative to the chlorine compounds that are currently been used as washing agents. Consumers may also benefit from the potential increased iodine intake that may result if widespread use of iodine as a washing agent occurs.

There would be little or no direct impact on government.

7.2.3 Conclusion

Option 2 is the preferred option. This option potentially provides benefits to both the food industry and consumers with very little associated negative impact on any sector.

8. Consultation

8.1 Public Consultation

The Initial Assessment Report for this Application was circulated for public comment on 21 May 2003 for a period of six weeks. A total of 12 submissions were received and these are summarised at **Attachment 6** to this report.

Following the first round of public consultation, FSANZ carried out an assessment of the application, taking into account the public comments received. The specific issues raised by these comments are addressed in this report.

8.2 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 are not any relevant international standards and amending the Code to allow the use of iodine as a washing agent for certain foods is unlikely to have a significant impact on international trade as suppliers of food products are not required to take up permissions granted through amendments to the Code therefore this matter will not be notified.

9. Conclusion and Recommendation

It is concluded that approval of iodine as a washing agent for fruit, vegetables and eggs is technologically justified and would not pose a risk to the health and safety of the general population, or any additional risk to vulnerable individuals.

The draft variation to Standard 1.3.3 – Processing Aids of the Code (**Attachment 1**), approving the use of iodine as a processing aid, is recommended for the following reasons:

- the use of iodine as a washing agent for fruit, vegetables and eggs is technologically justified – the efficacy of iodine as a sanitising agent for foods has been demonstrated and a technological need exists for alternative food sanitisers;

- the use of iodine as proposed may result in a small increase in iodine intake but not to a level that would raise safety concerns for the vast majority of the population or pose any adverse nutritional risks. The proposed use of iodine is also considered unlikely to pose any additional risk for vulnerable individuals;

- the proposed draft variation to the Code is consistent with the section 10 objectives of the FSANZ Act. In particular, FSANZ has addressed the protection of public health and safety by undertaking a risk assessment based on the best available scientific data.

- The regulation impact assessment has concluded that the benefits of permitting use of iodine as a washing agent outweigh any costs associated with its use.

10. Implementation and review

If approved it is proposed that the draft variation come into effect on the date of gazettal.

ATTACHMENTS

1. Draft variation to the *Australia New Zealand Food Standards Code*
2. Food Technology Report
3. Toxicology Report
4. Dietary Exposure Assessment Report – A493
5. Dietary Exposure Assessment Report – A493 and A528
6. Nutrition Risk Assessment Report
7. Summary of Public Comments

ATTACHMENT 1

DRAFT VARIATION TO THE *AUSTRALIA NEW ZEALAND FOOD STANDARDS CODE*

To commence: On gazettal

[1] ***Standard 1.3.3*** *of the Australia New Zealand Food Standards Code is varied by inserting in the* Table to clause 12 –

Iodine	Fruits, vegetables and eggs	GMP

ATTACHMENT 2

FOOD TECHNOLOGY REPORT

Introduction

FSANZ received an application from Ioteq Limited (formerly Iodine Technologies Australia Pty. Ltd) to amend Standard 1.3.3 – Processing Aids of the *Australia New Zealand Food Standards Code* (the Code) to allow the use of iodine as a processing aid, specifically as a washing agent for fruits, vegetables, nuts and eggs.

Background

The food industry uses a number of primary sanitising agents, at all levels during food manufacture and processing, to reduce the levels of bacteria and fungi which can cause food safety concerns as well as reduce keeping quality and shelf life of products. Such bacteria include *E. coli*, *Listeria* and *Salmonella* spp and non-pathogenic spoilage organisms including yeasts and moulds. These sanitising agents, also called disinfecting agents, do not kill all bacteria so they are not sterilising agents but tend to inhibit the growth of the bacteria to acceptable levels.

Halogens are a family of elements with a high affinity for electrons. They belong to group VII of the periodic table. The affinity for electrons makes the elements very reactive with biological molecules[1]. The halogen elements chlorine and iodine are sanitising agents, which act as intermediate level disinfectants. Halogens are very reactive towards biological molecules and are strong oxidising agents that can disrupt enzyme activity and membrane structure.

Chlorine is used extensively in the food industry as a sanitising agent. An alternative food sanitiser is iodine, which has a history of use in the dairy industry and offers a number of advantages over chlorine.

Chemical Details

Elemental iodine (I_2) has a molecular weight of 253.809 g/mol and a CAS number of 7553-56-2. Iodine exists as blue violet to black crystals which melt at 114˚C and boil at 184˚C. Iodine is poorly soluble in water but soluble in many organic solvents. Iodine readily sublimes at room temperature to form violet corrosive vapour.

Specification

Iodine is listed in the Merck Index[2], which is one of the secondary sources listed in clause 3 of Standard 1.3.4 – Identity and Purity, for specifications. The applicant has provided a more detailed specification of the moist iodine crystals it uses in their process with lists of various trace impurities.

Technological Function

Halogens such as chlorine, iodine and bromine have strong antimicrobial activities. Chlorine is the more commonly used element and various forms of compounds that deliver chlorine (including hypochlorite) have been used for many years as sanitising agents in the food industry (as well as many other industries).

Iodine disrupts hydrogen bonds and disulphide bonds of proteins structures[3]. Free iodine enters the cells of microorganisms and binds with cellular components, to disrupt protein synthesis by hindering or blocking the formation of hydrogen and disulphide bonds[4].

Iodine is able to act as a sanitising agent or microbiological control agent for treated food by destroying or limiting the numbers of pathogens or food spoilage organisms. Iodine is lethal to all forms of vegetative forms of microorganisms, can inactivate viruses and is fairly effective at higher concentrations against endospores[1]. Specifically, in terms of the application, this means inactivating and limiting the numbers of such bacteria and other microorganisms on the surface of treated foods, such as vegetables and fruit.

Chemical reactions of iodine dissolved in water

The various important chemical reactions of iodine dissolved in water are listed below[5].

$I_2 + H_2O \leftrightarrow HIO + I^- + H^+$ (hydrolysis, formation of HIO (hypoiodous acid))

$HIO \leftrightarrow OI^- + H^+$ (hypoiodous acid dissociation)

$3HIO \leftrightarrow IO_3^- + 2I^- + 3H^+$ (disproportionation of HIO)

$3I_2 + H_2O \leftrightarrow IO_3^- + 5I^- + 6H^+$ (iodate formation)

$I_2 + I^- \leftrightarrow I_3^-$ (triiodide formation)

All the above reactions, except for those that form iodate, are very rapid and reach equilibrium quickly. Iodate has no activity as a sanitiser so it is important to understand the conditions (predominately time and pH) that form it, since this diminishes the sanitising activity of the iodine solution. At neutral pH and up to 30 minutes after adding iodine (1-25 ppm) to water the predominate iodine species are iodine (I_2) and hypoiodous acid (HIO).

For normal levels of iodine used for sanitation (10-25 ppm) it is best to keep the water pH below 8.5 to limit the iodate production. Sanitation with iodine is still effective between pH levels of 4-8.5. It has also been reported that over the use concentrations proposed by the Applicant over 80% of the iodine is present in biocidal active forms, I_2 and HIO, from pH 3.0 to 8.0.

Hypoiodous acid (HIO) is the most effective iodine compound for effectiveness against bacteria. Also removal of the iodide ion (I^-) is important to help maintain the iodine sanitation effectiveness by limiting the formation of the less reactive triiodide species.

Because elemental iodine is rather poorly soluble in water, iodine has been added to other various solvents and carriers or solubilising agents. Such compounds are commonly referred to as iodophors and are widely used as disinfecting agents. Such products have advantages in often enhancing the bactericidal activity of iodine, reducing vapour pressure, odour and staining as well as increasing water solubility. A number of iodophors are approved and used as sanitisers for food contact surfaces and equipment in the food industry.

In the present application the Applicant is intending to use elemental iodine in a closed system where the iodide that is formed is removed from the process stream using resins, and then regenerated to produce iodine which is then reused.

Using iodine as a food surface sanitiser has a number of advantages over various chlorine sources. These are summarised below.

Advantages

- It is highly reactive, meaning less iodine has the same effect as higher concentrations of chlorine.
- It is not as readily inactivated by organic matter, which includes dirt, as chlorine. Chlorine reacts with organic matter to produce unpleasant by-products that are carcinogenic and may cause flavour taints (trihalomethanes, including trichloromethane, trichloroanisole)[3]. Trihalomethanes and excess chlorine solution also have environmental implications for wastewater disposal.
- Iodine is less corrosive than chlorine on metals and other surfaces.

Disadvantages

- Iodine compounds may stain plant including metal and plastic surfaces.
- Iodine has unpleasant and toxic odours and vapours.
- Elemental iodine has limited solubility in water, which is why iodophors have often been used since they have better solubility.

Some of the disadvantages of using iodine are overcome by using an automated, sealed system for iodine delivery and by regenerating iodine from the spent iodide.

Efficacy of washing food with iodine solutions

The Application (as well as other supporting documentation subsequently supplied by the Applicant) provides trial data indicating the efficacy of treating freshly harvested vegetables and fruit with an iodine washing system. These trial results were performed using simple dipping solutions of iodine concentrations ranging from 3-30 ppm compared to the same chlorine concentrations, as well as the Applicant's own enclosed recycled system. Treatment times were 1, 2 and 4 minutes. Results were compared with the reduction in total bacteria and fungi post treatment on the treated produce. Also trials were performed to assess the effect of dirt, which is a problem with freshly harvested produce, and is known to decrease the efficacy of chlorine wash solutions.

The results indicated that iodine was superior to the equivalent concentration of chlorine. This was also the case when dirt was a factor. Some of the fruits and vegetables tested were orange, nectarine, peach, apple, strawberry, rock melon, avocado, potato, tomato, lettuce and bean sprouts. It was generally found that a treatment of 30 ppm or less of iodine caused a 30 fold reduction (1.5 $\log_{10}$) in bacteria and fungi concentrations on the treated produce. It was also reported that increasing the contact time by a factor of four (dip time) was equivalent in effectiveness to doubling the iodine concentration.

Trials were also performed evaluating the efficacy of iodine washing on reducing pathogen concentrations (*Salmonella* spp. and *Listeria monocytogenes*) on three food types, lettuce, fish and meat. The $\log_{10}$ reductions for treatment with 30 ppm iodine for *Listeria* spp. for lettuce, fish and meat are 1.4, 0.8 and 0.9 and for S*almonella* spp. for the same products, 1.1, 0.7 and 0.6 respectively.

Evidence of Technological Need

There is a technological need to improve the keeping quality and safety of fresh food, primarily fresh fruit and vegetables. These are usually contaminated with soil and various microbiological contaminants, which need to be removed or at least controlled for both food safety and shelf life reasons. The food industry does this by various means such as washing and bleaching the foods. Approved washing and bleaching agents and their restrictions on use are listed in the Table to clause 12 of Standard 1.3.3 – Processing Aids. These agents comprise various compounds that produce active chlorine (various hypochlorites, chlorine itself and chlorine dioxide), hydrogen peroxide, peracetic acid and metabisulphite.

The present application is another proposed sanitiser that can be used as an alternative to the other approved processing aids listed in the Table to clause 12 of Standard 1.3.3. If the application is successful the proposed generic approval within this Table will be for 'iodine' which is broader than the applicant's specific technology.

Conclusion

The use of iodine as a sanitising agent for foods is technologically justified.

References

1. Website article, Methods of Microbial Control,
http://us.geocities.com/alliaria/rec12.html

2. The Merck Index, 13th Edition, Merck and Co. Ltd Whitehouse Station, N.J. (2001).

3. Website article, from Franklin College, Control of growth-Physical Methods
http://www.franklincoll.edu/bioweb/micro/control.htm

4. Website article from the University of Virginia, Office of Environmental Health and Safety, Biosafety Program, Summary and Comparison of Liquid Disinfectants,
http://keats.admin.virginia.edu/bio/disinfectant_summary.html.

5. A report supplied by the Applicant, Stephen Morris, Sydney Postharvest Lab, 12 May 2003, A Brief Summary of Iodine Chemistry and Disinfection in Water.

Extra reference of interest

Website article from the Food and Agriculture Organisation of the United Nations (FAO) Rome 1994, Assurance of Seafood Quality, chapter 6 Cleaning and Sanitation in Seafood Processing. http://www.fao.org/DOCREP/003/T1768E/T1768E07.htm

ATTACHMENT 3

TOXICOLOGY REPORT FOR IODINE

Executive Summary

Iodine is an important trace element that is required for the synthesis of the thyroid hormones, thyroxine (T_4) and triiodothyronine (T_3). These hormones have a key role in influencing cellular metabolism and metabolic rate. The recommended daily intake for iodine for different population groups varies. For adults, the RDI ranges from 100-200 μg/day.

Although iodine is an essential component of the diet, intakes in excess of physiological requirements may produce adverse effects, particularly on the thyroid gland and the regulation of thyroid hormone production and secretion.

Diet is the major source of iodine intake for humans. The major food categories contributing to dietary intake include dairy products, seafood, fruits, vegetables and eggs, with meat and cereals being secondary sources. The iodine content of food is reflective of background levels in the environment as well as the use of iodine and its compounds in food production, processing and manufacturing. In addition to dietary sources, various mineral supplements and medical preparations can further add to iodine intake.

Greater than 97% of ingested iodine is absorbed from the gastrointestinal tract, generally as iodide. Absorbed iodide enters the circulation where it is taken up primarily by the thyroid gland. The uptake of iodide by the thyroid gland is controlled by the thyroid-stimulating hormone (TSH), which is highly sensitive to dietary iodine intake. At low intakes representing iodine deficiency, uptake of iodide into the thyroid gland is increased and at very high intakes, iodide uptake into the thyroid gland decreases. Once the physiological requirements for thyroid hormone synthesis have been met, the thyroid does not accumulate more iodide and any excess is excreted, primarily in the urine.

A large number of human experimental, clinical, and epidemiological studies on the effects of excess iodine on human health have been reported and reviewed in detail by both the Joint FAO/WHO Expert Committee on Food Additives (JECFA) and the US Agency for Toxic Substances and Disease Registry (ATSDR). These studies indicate that the primary effect of excess iodine is on the thyroid gland and regulation of thyroid hormone production and secretion, and it is these effects that are the focus of the report.

Excess iodine can produce an enlargement of the gland (goitre) and/or affect the production of the thyroid hormones. A diminished production of the thyroid hormones is referred to as hypothyroidism (and may be accompanied by goitre) and increased thyroid hormone synthesis and secretion by the thyroid gland is referred to as hyperthyroidism.

The effect on the thyroid depends on the current and previous iodine status of the individual and any current or previous thyroid dysfunction. For example, individuals with a history of iodine deficiency may be prone to the development of iodine-induced hyperthyroidism if iodine exposure increases later in life.

The human response to excess iodine can be quite variable. Some individuals can tolerate quite large intakes (up to 50 μg/kg/day) while others may respond adversely to levels close to recommended intakes (3-7 μg/kg/day). Individuals responding adversely to relatively low intake levels typically have an underlying thyroid disorder or have a long history of iodine deficiency.

For the majority of healthy individuals, the most sensitive endpoint for iodine toxicity is sub-clinical hypothyroidism. Sub-clinical hypothyroidism is defined as an elevation in TSH concentration while serum thyroid hormone concentration is maintained within the normal range of values for healthy individuals. While not clinically adverse, such an effect, if persistent, could lead to clinical hypothyroidism. In healthy adults, such an effect has been associated with acute intakes of 1700 μg/day (24 μg/kg body weight/day for a 71 kg person), and for children, has been associated with chronic intakes of 1150 μg/day (29 μg/kg/day for a 40 kg child).

Iodine intakes of approximately 1000 μg/day however appear to be well tolerated by healthy adults. This level has been used by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) to establish a provisional maximum tolerable daily intake (PTDI) for iodine of 17 μg/kg bw from all sources. FSANZ has adopted this level as a safe intake level for the purpose of risk assessment for the general healthy population.

For those individuals with thyroid disorders or a long history of iodine deficiency, this PTDI is not applicable since these individuals may respond adversely at levels of intake below the PTDI. It has been reported that intakes in the range 3-7 μg/kg/day may be sufficient to produce an increase in hyperthyroidism in chronically iodine deficient individuals. The health risk for these individuals needs to be considered separately from the general population

1. Introduction

Iodine is an important trace element that is essential for the maintenance of normal thyroid function where it is required for the synthesis of the thyroid hormones, L-triiodothyronine (T_3) and L-thyroxine (T_4) (also called 3,5,3', 5'- tetraiodothyronine). T_3 and T_4 are responsible for regulating cellular oxidation and hence have a key role in influencing cellular metabolism and metabolic rate.

The recommended daily intake (RDI) for iodine varies for individuals. The RDI for adults ranges from 100-150 μg/day, with intakes of 150-290 μg/day recommended for pregnant and lactating women. Intakes of 70 μg/day are recommended for young children.

Although iodine is an essential component of the diet, intakes in excess of physiological requirements may produce adverse effects, particularly on the thyroid gland and the regulation of thyroid hormone production and secretion. This in turn can have downstream impacts on a wide variety of other organ systems, producing an array of debilitating effects in the affected individual.

The purpose of this review is to examine the toxic effects associated with excess iodine and establish a safe level of exposure.

2. Physical and Chemical Properties

Iodine (I) is a non-metallic element belonging to the halogen family and has a molecular mass of 126.9. Iodine is a bluish-black, lustrous solid, which sublimes at room temperature into a blue-violet gas with a sharp characteristic odour. Iodine dissolves readily in alcohol, benzene, chloroform, carbon tetrachloride, ether or carbon disulfide but is only slightly soluble in water (0.03 g/100 ml at 20˚C).

The chemistry of iodine can be quite complex as it can exist in a number of different valence states, is chemically reactive (although less so than other halogens) and forms various organic and inorganic compounds. The most common compounds formed are the iodides (I^-) and iodates (IO_3^-).

Thirty-six isotopes are recognized with fourteen of these yielding significant radiation. The only naturally occurring isotopes are ^{127}I, which is stable, and ^{129}I, which is radioactive. This report will concentrate on toxic effects associated with stable iodine.

3. Sources

The oceans are considered to be the most importance source of natural iodine. Iodine in seawater enters the air via aerosols or as a gas and from there is deposited onto soil, surface water and vegetation.

Diet is regarded as the major source of iodine intake for the population (WHO 1989). Major food categories contributing to dietary intake in Australia and New Zealand include dairy products, seafood (marine fish, shellfish, algae and seaweed), fruits, vegetables and eggs, with meat and cereals being secondary sources.

Additional sources of intake come from the use of iodine and its compounds in a variety of food-related applications including nutrient fortification (e.g., iodised salt), food additives (e.g., dough conditioning and maturing agents), agricultural chemicals (e.g., herbicides and fungicides), animal drugs (e.g., iodine supplements), and sanitisers (e.g., iodophors).

The iodine content of foods is thus both reflective of background levels in the environment as well as processing technology and manufacturing practices. For example, the high iodine content of milk and dairy products has been attributed to the use of iodine-containing supplements in feed for dairy cattle, iodophore-based medications, teat dips and udder washes as well as iodophors used as sanitising agents in dairy processing establishments. The use of iodophors by the dairy industry has however become less commonplace, resulting in milk becoming a less important source of dietary iodine (Eastman 1999).

In addition to dietary sources, various mineral supplements and medical preparations can further increase iodine intake to a significant extent (WHO 1989).

5. Toxicokinetics

5.1 Absorption

Inorganic iodine is >97% absorbed from the gastrointestinal tract, generally as iodide. Although some absorption occurs in the stomach, the small intestine appears to be the principal site of absorption in both humans and rats (Riggs 1952, Small et al 1961). The mechanism by which iodide is transported across the intestinal epithelium is not known. Gastrointestinal absorption appears to be similar in children, adolescents and adults, although absorption in infants may be lower than in children and adults (ATSDR 2001).

5.2 Distribution

Once absorbed, iodide enters the circulation and is distributed throughout the extracellular fluid where it is taken up by those tissues with specialized transport mechanisms for iodide (Cavalieri 1980). The human body contains about 10 – 15 g iodine in total, the majority of which (>90 %) is stored by the thyroid gland (Cavalieri 1997). The concentration of iodine in serum is about 50 – 100 µg/L under normal circumstances, with about 5% being in the inorganic form as iodide and the remaining 95% consisting of various organic forms of iodine, principally protein complexes of the thyroid hormones.

Other tissues that accumulate iodide include the salivary glands, gastric mucosa, choroid plexus, mammary glands, placenta, and sweat glands. The tissue distribution of iodide and organic iodine are very different and are interrelated by metabolic pathways that lead to the iodination and de-iodination of proteins and thyroid hormones.

The uptake of iodide by the thyroid gland is controlled by the thyroid-stimulating hormone (TSH), which is secreted from the anterior lobe of the pituitary gland. In addition to stimulating iodide transport from the blood into thyroid cells, TSH is also responsible for stimulating the oxidation of iodide to iodine, and iodine binding to tyrosine.

Iodide taken up by the thyroid gland is used for the production of the thyroid hormones, which are stored in the gland. Approximately 90% of the thyroid iodine content is in the organic form and includes iodinated tyrosine residues comprising the thyroid hormones T_4 and T_3, and their various synthesis intermediates and degradation products. Once requirements for thyroid hormone synthesis have been met, the thyroid does not accumulate more iodide and any excess is excreted in the urine (Bender and Bender 1997).

Iodide uptake into the thyroid gland is highly sensitive to iodide intake. At low intakes representing iodine deficiency, uptake of iodide into the thyroid gland is increased (Delange and Ermans 1996). At very high intakes, iodide uptake into the thyroid gland decreases, primarily as a result of decreased iodothyronine synthesis (the Wolff-Chaikoff effect) and iodide transport into the gland (Nagataki and Yokoyama 1996, Saller 1998).

5.3 Metabolism

Once in the thyroid, iodide is oxidised to elemental iodine by the enzyme thyroid peroxidase (Saller 1998). This reaction is the rate-limiting step for protein iodination and hormone synthesis. Once oxidised, iodine enters the biosynthetic pathway for thyroid hormone synthesis.

Initially iodine is incorporated into monoiodotyrosine and diiodotyrosine, which are then coupled together to form the thyroid hormones T_3 (coupling of a monoiodotyrosine and diiodotyrosine residue) and T_4 (coupling of two diiodotyrosine residues). These reactions occur within a large glycoprotein called thyroglobulin, which is synthesized only in the thyroid gland.

TSH regulates every step in the biosynthesis of the thyroid hormones, from the concentration of iodide to the proteolysis of thyroglobulin (Cavalieri 1980). There is a sensitive feedback mechanism between the thyroid and the pituitary gland to maintain the levels of thyroid hormones. This is influenced by the hypothalamus, with thyrotrophin-releasing hormone mediating the secretion of TSH from the pituitary.

Deiodination reactions are carried out by a family of selenoproteins. Iodotyrosine dehalogenase regenerates iodide from monoiodotyrosine and diiodotyrosine for re-use within the thyroid or release into blood, accounting for the iodide leak in the state of chronic iodine excess or certain thyroid conditions (Cavalieri 1997). The liver contains a considerable amount of T_4, some of which is converted into T_3 and some which is excreted into the bile and ultimately reabsorbed or excreted (Cavalieri 1980).

5.4 Excretion

All absorbed iodine is excreted primarily in the urine and faeces, but is also excreted in breast milk, exhaled air, sweat and tears (Cavalieri 1997). Urinary excretion normally accounts for 97% of the elimination of absorbed iodine, while faecal excretion accounts for about 1-2% (Larsen et al 1998). The fraction of the absorbed iodide dose excreted in breast milk varies with functional status of the thyroid gland. A larger fraction of the absorbed dose is excreted in breast milk in the hypothyroid state compared to the hyperthyroid state. In the hypothyroid state, uptake of absorbed iodide into the thyroid gland is depressed, resulting in greater availability of the absorbed iodide for distribution to the mammary gland and breast milk.

6. Toxicity of Iodine

A large number of human experimental, clinical, and epidemiological studies on the effects of excess iodine on human health have been reported. These studies will not be reviewed again in detail as they have already been subject to significant reviews by both the Joint FAO/WHO Expert Committee on Food Additives (JECFA) (WHO 1989) and the Agency for Toxic Substances and Disease Registry (ATSDR 2001).

JECFA concluded there are three potential types of adverse response to excess iodine. The first is disturbance of thyroid activity, which may alter the size of the gland and/or affect the production of thyroid hormones. There is also evidence to indicate that iodine (or the lack of it) may alter the pattern of thyroid malignancy. The second type of response involves sensitivity reactions, and the third type of response results from acute intakes of large quantities of iodine (iodine poisoning).

This review will largely focus on effects on the thyroid gland, which is regarded as the primary and most sensitive indicator of iodine toxicity (ATSDR 2001).

6.1 Disturbance of Thyroid Function

The primary effects of excessive iodine ingestion are on the thyroid gland and regulation of thyroid hormone production and secretion. Adverse effects on the pituitary and adrenal glands are secondary to disorders of the thyroid gland.

Excess iodine can result in goitre, hypothyroidism (with or without goitre), or hyperthyroidism (thyrotoxicosis) (see below). The effect produced depends on the current and previous iodine status of the individual and any current or previous thyroid dysfunction (WHO 1989). For example, individuals exposed to low levels of iodine early in life may be prone to the development of iodine-induced hyperthyroidism if iodine exposure increases later in life. Those with underlying thyroid disease also respond more to increased iodine intake, and it also appears that females are more likely to respond to excess iodine than males.

Definitions

Goitre refers to an enlargement of the thyroid gland that is usually visible as a swelling in the anterior portion of the neck. A number of different types of goitres are known to occur.

Simple or *non-toxic goitre* is an enlargement of the thyroid gland that is not associated with overproduction of thyroid hormone, inflammation or malignancy, whereas *toxic goitre* is one involving excessive production of thyroid hormone. Thyroid enlargement can be uniform (diffuse goitre) or the gland can become enlarged as a result of the occurrence of one or more nodules (nodular goitre).

The two most common causes of simple or non-toxic goitre are iodine deficiency (referred to as endemic goitre) or the ingestion of large quantities of goitrogenic foods or drugs. In these cases, the thyroid gland is unable to meet the demands of the body (i.e., because of an inadequate supply of iodine) and enlarges to compensate. Enlargement of the gland is usually sufficient to overcome the mild impairment to hormone production.

Goitre can also be associated with both hypothyroidism and hyperthyroidism. *Hypothyroidism* refers to the diminished production of thyroid hormone leading to clinical manifestations of thyroid insufficiency and can occur with or without goitre. Typical biomarkers of hypothyroidism are a depression in the circulating levels of T_4 and/or T_3 below their normal ranges. This is usually, but not always, accompanied by an elevation of TSH above the normal range. The most common cause of hypothyroidism is Hashimoto's disease (or lymphocytic thyroiditis). Hashimoto's disease is an autoimmune disease in which abnormal antibodies are produced that impair the ability of the thyroid to produce thyroid hormone. The pituitary gland responds by producing TSH and the additional TSH may cause the thyroid gland to enlarge.

Hyperthyroidism is where accelerated thyroid hormone biosynthesis and secretion by the thyroid gland produce thyrotoxicosis. The term *thyrotoxicosis* refers to the hypermetabolic clinical syndrome resulting from serum elevations in thyroid hormone levels, specifically free thyroxine (T_4), triiodothyronine (T_3), or both. The terms hyperthyroidism and thyrotoxicosis are often used interchangeably but are not synonymous. That is, while many patients have thyrotoxicosis caused by hyperthyroidism, other patients may have thyrotoxicosis caused by inflammation of the thyroid gland, which causes release of stored thyroid hormone but not accelerated synthesis, or thyrotoxicosis, which is caused by ingestion of exogenous thyroid hormone.

The most common cause of hyperthyroidism is Graves' disease (diffuse toxic goitre), an autoimmune disease where the immune system produces antibodies that stimulate the TSH receptors of the thyroid gland resulting in the non-suppressible overproduction of thyroid hormone. This causes the thyroid gland to become enlarged. In the elderly, a condition called toxic nodular goitre may cause hyperthyroidism. Toxic nodular goitre occurs when one or more small benign tumours in the thyroid gland produce excess thyroid hormones.

6.1.1 Iodine-Induced Hypothyroidism

The human body has a number of adaptive mechanisms for dealing with excess iodine. These mechanisms tend to be inhibitory in nature and generally do not significantly affect thyroid function.

The most well known of these is the *Wolff-Chaikoff effect* (Wolff et al 1949), where large dietary or therapeutic intakes of iodine can inhibit organic iodine formation (the binding of iodine to tyrosine in the thyroid), producing a decrease in the circulating thyroid hormone levels, and a subsequent increase in TSH. The effect is typically transient, even if the excess intake continues, with most people being able to escape from the inhibition without a clinically significant change to circulating hormone levels. Most individuals are therefore able to adapt to excess iodine.

Some individuals who fail to escape from the Wolff-Chaikoff effect typically develop goitre and may also become hypothyroid. Susceptible individuals include: foetuses and newborn infants; patients who have autoimmune thyroiditis; patients with Grave's disease previously treated with iodine; women who have post-partum thyroiditis; or those who have subacute thyroiditis. Iodine-induced hypothyroidism is also reported to be more common in women.

Excessive intake of iodine by pregnant women is of particular concern as the foetal thyroid is less able to escape the inhibitory effects of iodine on thyroid hormone formation. Iodine-induced goitres and/or hypothyroidism have occurred in newborn infants of mothers who have taken iodine during pregnancy. Infant goitres may regress spontaneously after several months, but deaths due to compression of the trachea have occurred (WHO 1989).

A number of studies have examined the acute effects of increased intakes of iodine on the thyroid hormone status of adults (Chow et al 1991, Gardner et al 1988, Georgitis et al 1993, Namba et al 1993, Paul et al 1988, Robison et al 1998). These studies suggest that acute (14 days) iodine exposures of 1500 µg/day (21 µg/kg/day) above the pre-existing dietary intake can be tolerated without producing a clinically adverse change in thyroid hormone levels, although such doses may produce a reversible depression in serum T_4 concentration and an elevation in serum TSH concentration, both within the normal range of values for healthy individuals. Changes in thyroid hormone levels within normal ranges are not considered to be clinically adverse; however, they are indicative of a suppressing effect on thyroid hormone production that, if persistent, could result in thyroid gland enlargement and other clinically significant complications. In the case of elderly adults, subclinical hypothyroidism has been induced by an acute increase of 500 µg/day (7 µg/kg/day) (Chow et al 1991), possibly suggesting that the elderly may be less tolerant of excess iodide than younger adults. Based on estimates of the background dietary intakes of the subjects in these studies, in most cases estimated from measurements of urinary iodide excretion, the total iodide intakes producing subclinical hypothyroidism in healthy adults were approximately 1700 µg/day (24 µg/kg/day) (Gardner et al 1988, Paul et al 1988).

Acute intakes of approximately 700 μg/day (10 μg/kg/day) had no detectable effect on thyroid hormone status in healthy individuals. One study also found no evidence of disturbances in thyroid hormone status in 6 healthy euthyroid males who received doses of 20 mg/day (0.3 mg/kg/day) (Robison et al 1998), suggesting that, at least under certain conditions, exposure levels >10-24 μg/kg/day may be tolerated by some individuals.

The level of 1700 μg/day for subclinical hypothyroidism has been used by the Institute of Medicine as a lowest-observable-adverse-effect level (LOAEL) to which an uncertainty factor of 1.5 was applied to derive a Tolerable Upper Intake Level (UL) for iodine in adults of 1100 μg/day (Institute of Medicine 2001). By adjusting this level on the basis of bodyweight, the ULs for other age groups were derived. Thus, a UL of 900 μg/day was established for 14-18 year olds, 600 μg/day for 9-13 year olds, 300 μg/day for 4-8 year olds, and 200 μg/day for 1-3 year olds.

Two studies have been conducted in prison populations exposed to iodine through iodination of the water supply. In a study by Freund et al (1966), the health and thyroid function of representative subjects of a prison population were assessed before and during usage of iodinated water for nine months. Water containing 1000 μg/L iodine induced a marked decrease in the uptake of radioactive iodine but protein bound iodine levels did not increase significantly until the iodine concentration was increased to 5000 μg/L. No information on actual intake is provided but it has been assumed that water consumption would have been about 1-2 litres/day (WHO 1989). In another study, iodination of a prison water supply at a concentration of 500 to 750 μg/L (estimated intake 1000-2000 μg/day) for up to 15 years did not result in any change to serum T_4 levels (Thomas et al 1978). During the same period, 177 women in the prison gave birth to 181 full term infants without any enlargement of the thyroid being noted in the infants (Stockton & Thomas 1978). On the basis of these studies, which indicate that 1000 μg iodine/day is safe for the majority of the population, JECFA set a provisional maximum tolerable daily intake (PTDI) of 17 μg/kg bodyweight for iodine from all sources (WHO 1989).

Results from a number of epidemiological studies (Li et al 1987; Laurberg et al 1998) suggest that chronic exposure to excess iodine can result in or contribute to subclinical hypothyroidism in children (1150 μg/day, 29 μg/kg/day) and elderly adults (160-800 μg/day, 4-12 μg/kg/day). The study in children compared thyroid status in groups of children, aged 7-15 years, who resided in two areas of China with different drinking water iodine concentrations, providing estimated iodine intakes of 29 and 10 μg/kg/day. Both groups were all euthyroid[1] with normal values for serum thyroid hormones and TSH concentrations, although TSH concentrations were significantly higher in the high iodine group. This study was used by the ATSDR to establish a chronic-duration minimal risk level (MRL) for iodine of 10 μg/kg/day based on a no-observed-adverse-effect level (NOAEL) of 10 μg/kg/day and a LOAEL of 29 μg/kg/day for subclinical hypothyroidism in healthy human children (ATSDR 2001).

[1] Where TSH levels are in the normal range and the thyroid is neither hypothyroid nor hyperthyroid and considered “normal”.

Populations that are iodine deficient and, in particular, those that include people exhibiting goitre, appear to be particularly sensitive to an increase in their iodine intake. For example, iodine supplementation (200-400 µg/day, 3-6 µg/kg/day) for treatment of endemic goitre has been associated with thyroid dysfunction, including thyroid autoimmunity (Kahaly et al 1997, Kahaly et al 1998).

Very high doses of iodine exceeding 200 mg/day (2.8 mg/kg/day) given during pregnancy have been shown to result in congenital goitre and hypothyroidism in the newborn infant (Iancu et al 1974). Such doses, however, are atypical and clinical experience with lower doses of iodine supplementation given during pregnancy for the purpose of correcting or preventing iodine deficiency and for the management of Grave's disease indicates that oral doses of 4-5 µg/kg/day can be tolerated without any indication of thyroid dysfunction in the newborn (Momotani et al 1992, Pedersen et al 1993, Liesenkötter et al 1996).

6.1.3 Iodine-Induced Hyperthyroidism (Thyrotoxicosis)

Oral exposure to excess iodine can, under certain circumstances, lead to hyperthyroidism. This condition is referred to as "jodbasedow" although it is not thought to be a single aetiological entity (Fradkin and Wolff 1983). The occurrence of iodine-induced hyperthyroidism is most common in iodine deficient populations following the introduction of iodine supplementation programs. The most vulnerable are those over 40 years of age who have been iodine deficient since birth. Other vulnerable groups include those with thyroid diseases such as Graves' disease or postpartum thyroiditis.

The clinical features of iodine-induced hyperthyroidism are said to be similar to that of Graves' disease, however, in contrast to the diffuse goitres associated with Grave's disease, iodine-induced hyperthyroidism is generally associated with nodular goitres. Nodular goitres are fairly common in elderly subjects and are the result of longstanding iodine deficiency. Many of these nodules are autonomous, meaning they are independent of regulation by TSH and produce thyroid hormone in direct response to dietary iodine. Thus excess iodine may precipitate or aggravate hyperthyroidism in these subjects.

Frequently, iodine-induced hyperthyroidism is mild and follows a self-limited course, but in some cases it is more severe and can sometimes be lethal. Iodine-induced hyperthyroidism can be totally prevented in the next and subsequent generations by correction of iodine deficiency.

A number of epidemiological studies have been conducted in Europe and Africa to monitor the incidence of iodine-induced hyperthyroidism in iodine deficient populations following the introduction of iodine supplementation programs (DeLange et al 1999, Mostbeck et al 1998, Lind et al 1998, Stanbury et al 1998). These studies confirm that iodine supplementation of iodine deficient diets does result in a detectable increase in the incidence of hyperthyroidism. A well-documented case also occurred in Tasmania, Australia, following the introduction of iodised bread in 1966 and the addition of iodophors to milk by the dairy industry (Connolly et al 1970). Milk iodine (from the seasonal use of feed supplements) has also been a factor in Europe (Barker and Phillips 1984, Phillips 1983). A review of these studies indicates that iodine intakes in the range of 3-7 µg/kg/day may be sufficient to produce an increase in hyperthyroidism in iodine deficient populations (ATSDR 2001).

In the Tasmanian case, a 2- to 4-fold increase in hyperthyroidism occurred within a few months after diets were supplemented with iodide for the prevention of endemic goitre from iodine deficiency (Connolly et al 1970). The supplemental dose was 80-200 μg/day from the addition of potassium iodate to bread, but mean urinary iodide excretion rates suggested a total post-supplementation iodide intake of about 230 μg/day (range 94-398), equivalent to 3.3 μg/kg/day, some of which came from other sources such as milk (Connolly 1971a, 1971b). The highest incidence of hyperthyroidism after the iodine supplementation began occurred in people over 40 years of age (Stewart 1975, Stewart and Vidor 1976). Stewart (1975) noted that the small increase in the incidence of hyperthyroidism that occurred in people under 40 years of age was largely due to Graves' disease.

Cases of iodine-induced hyperthyroidism in people who were euthyroid and without apparent thyroid disease have been reported (Rajatanavin et al 1984, Savoie et al 1975, Shilo and Hirsch 1986), however only a few have provided dose information. In these cases, effects were observed following doses in the range 0.05 – 23 mg/kg/day.

6.1.3 Thyroid malignancy

Several large-scale epidemiological studies have examined the relationship between iodine intake and thyroid cancer. The results of these studies suggest that an increased iodine intake may be a risk factor for thyroid cancer in certain populations, namely, populations residing in iodine deficient, endemic goitre regions (Franceschi 1998, Franceschi and Dal Maso 1999). Not all of these studies have found an increased risk of cancer, however, a recurrent observation is an apparent shift in the histopathology towards a higher prevalence of papillary cancers, relative to follicular cancers, after increased iodine intake in otherwise iodine-deficient populations (Bakiri et al 1998, Belfiore et al 1987, Kolonel et al 1990, Petterson et al 1991, 1996). Two studies in particular found a significant excess of thyroid gland cancer in populations from endemic goitre regions whose diets had been supplemented to achieve approximate iodine intakes of 3.5 μg/kg bw/day (Bacher-Stier et al 1997, Harach and Williams 1995).

6.2 Sensitivity Reactions

Oral exposure to excess iodine can produce allergic or sensitivity reactions in certain individuals. The reactions include urticaria (hives), acneiform skin lesions (ioderma), and fevers. Cases of more serious reactions involve angioedema (localised oedema), vasculitis, peritonitis and pneumonitis, and complement activation. Both humoral and cell-mediated immune responses are thought to be involved (Curd et al 1979, Rosenburg et al 1972, Stone 1985). In general, reactions to iodide have occurred in association with repeated oral doses of iodide exceeding 300 mg/day.

Ioderma is thought to be a form of cell-mediated hypersensitivity (Rosenburg et al 1972, Stone 1985) and its occurrence appears to be unrelated to thyroid gland function. Characteristic symptoms include acneiform pustules, which can coalesce to form vegetative nodular lesions on the face, extremities, trunk, and mucous membranes. The lesions regress and heal when the excess iodide intake is discontinued. The literature reports cases of ioderma occurring following oral doses of iodide 300-1000 mg/day. However, in many of these cases, pre-existing disease and related drug therapy may have contributed to the reaction to iodide; thus the dose-response relationship for ioderma in healthy people remains highly uncertain.

Oral exposures to iodide > 1000 mg/day have been associated with the occurrence of fevers, which cease once exposure to the excessive iodide intake is discontinued (Kurtz and Aber 1982, Horn and Kabins 1972). The fevers are thought to have an immunological basis and do not appear to be related to thyroid gland function. Reported clinical cases have almost always involved a pre-existing disease, usually pneumonia or obstructive lung disease in which potassium iodide was administered along with other drugs, such as antibiotics, barbiturates and methylxanthines.

6.3 Iodine Poisoning

The effects from acute exposure to high iodine concentrations are largely due to the strong oxidising effect of iodine on the gastrointestinal tract and resultant shock. It is these properties of iodine that make it effective as a topical antiseptic and antimicrobial disinfectant.

Cases of iodine poisoning are rare however and are typically associated with intakes of many grams. Symptoms observed in lethal or near-lethal poisonings have included abdominal cramps, bloody diarrhoea and gastrointestinal ulcerations, oedema of the face and neck, pneumonitis, haemolytic anaemia, metabolic acidosis, fatty degeneration of the liver, and renal failure (Clark 1981, Dyck et al 1979, Finkelstein and Jacobi 1937, Tresch et al 1974). Death has occurred from 30 minutes to 52 days after ingestion, although death generally occurs within 48 hours. Where the dose was known, it ranged from 1.1 to 9 g iodine (18-150 mg/kg for a 60 kg adult), although there is a single case report of a 54-year-old male surviving the accidental ingestion of 15 g iodine (Tresch et al 1974).

7. Safe Limits for Oral Intake

A number of safe intake levels have been recommended as a result of reviews on the toxicity of excess iodine. The highest level of intake that has been found to be safe for the majority of the population is about 1000 µg iodine/day. This level was used by JECFA to establish a PTDI for iodine of 17 µg/kg bw from all sources (WHO 1989). It is recommended this level be adopted for the purpose of risk assessment for the general health population.

For those individuals with thyroid disorders or a long history of iodine deficiency, this PTDI is not applicable since these individuals may respond adversely at levels of intake below the PTDI. It has been reported that intakes in the range 3-7 µg/kg/day may be sufficient to produce an increase in hyperthyroidism in chronically iodine deficient individuals. The health risk for these individuals needs to be considered separately from the general population.

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ATTACHMENT 4

DIETARY EXPOSURE ASSESSMENT

An application was received by FSANZ requesting amendment of Standard 1.3.3 'Processing Aids' – Clause 12 'Permitted bleaching agents, washing and peeling agents' to allow the use of elemental iodine as a washing agent for fruits, vegetables (including herbs), nuts and eggs at good manufacturing practice (GMP) levels.

A dietary intake assessment was deemed necessary in order to determine the potential impact of granting permission for the use of elemental iodine as a washing agent for fruits, vegetables (including herbs), nuts and eggs on the iodine intake of the population. Iodine intakes, based on residues of iodine on treated foods, were assessed to determine if iodine intakes exceeded health standards.

Summary

Estimated dietary intakes of iodine were calculated for the Australian and New Zealand populations, and for the population sub-group of Australian children aged 2-6 years. This was to ensure that iodine intakes would not exceed the Provisional Tolerable Daily Intake (PTDI) if approval to use elemental iodine as a washing agent were granted. Provisional Tolerable Daily Intakes (PTDI) are upper limits that represent the permissible human exposure to those contaminants unavoidable associated with the consumption of otherwise wholesome and nutritious food.

While an upper intake level has been set for iodine, iodine is also an essential micronutrient. Consequently, dietary intakes were also assessed for a range of age-gender categories (as detailed in Table 1) for the purpose of comparison with the Estimated Average Requirements (EARs) for iodine. The EAR is defined as the level below which 50 percent of the population may be at risk of having inadequate intake. Further details regarding the results of the comparison of dietary intake with EARs can be found in the Nutrition Report at Attachment 6.

Baseline intakes of iodine were calculated using naturally occurring concentrations. Two post-treatment scenarios were examined in each dietary intake assessment: Scenario 1 applied a peeling factor to those fruits and vegetables washed with iodine that may be consumed with the peel either on or off (e.g. apples); and Scenario 2 assumed that fruits and vegetables washed with iodine that may be consumed with the peel on or off were always consumed unpeeled, in order to assume a worst-case scenario.

Estimated mean and 95th percentile dietary intakes of iodine were below the PTDI of 17 µg/kg body weight/day (WHO 1989) for all population groups and all scenarios examined, with one exception. Although the dietary intakes of iodine for the 2-6 years age group were below the PTDI, it was noted that analysis of the 2-3 years high consumers (95th percentile) group indicated that the PTDI was exceeded when it was assumed that fruits and vegetables that may be consumed with the peel on or off were consumed unpeeled (Scenario 2). Australian children aged 2-3 years had an estimated 95th percentile dietary intake of 17.2 µg/kg bw/day (260 µg/person/day) which is equivalent to 101.2% of the PTDI.

However, due to the conservative assumptions made in this calculation and taking into account that the use of 24-hour dietary survey data tends to over-estimate habitual food consumption amounts for high consumers, it is likely that the 95th percentile dietary intake is an over-estimate.

Background

Iodine is a substance that is found naturally in the environment, particularly in seawater, igneous rocks and soils (UK FSA 2002). Iodine is an essential micronutrient. Foods rich in iodine include seafood, milk, eggs and iodised salt. In the Code, salt is permitted to be iodised at a level no less than 25 mg/kg and no more than 65 mg/kg of iodine. This permission is voluntary.

The applicant has requested amendment of Standard 1.3.3 'Processing Aids' – Clause 12 'Permitted bleaching agents, washing and peeling agents' to allow the use of elemental iodine as a washing agent for fruits, vegetables (including herbs), nuts and eggs at GMP levels. The applicant has developed a system, the Iodoclean™ system, for delivering active iodine in treatment water at a concentration of 3-30 ppm[1]. This treatment is performed for sanitising purposes

Dietary Intake Assessment provided by the applicant

The applicant stated that fruits and vegetables contribute to approximately 5% of the average iodine intake and that a slight increase in iodine intake from treated fruits and vegetables would be beneficial since the typical Western diet and also the typical Australian diet is substantially deficient in iodine. It was also stated that the Iodoclean™ System increases the existing iodine levels in fruit and vegetables twofold. The data for fruit and vegetable contribution to total iodine intakes were obtained from a paper by Lee et al (1994) and related to iodine intake in the British diet. The dietary intake assessment submitted by the applicant was not detailed enough to allow FSANZ to determine a conclusion about potential dietary intake of iodine if permission to use iodine as a washing agent is granted. Therefore FSANZ conducted its own dietary intake assessment.

Dietary Modelling

Dietary modelling was conducted by FSANZ to estimate potential dietary intakes of iodine for Australia and New Zealand when fruits, vegetables (including herbs), nuts and eggs are washed with elemental iodine in water. The dietary intake assessments include iodine from other food sources in the diet, but not from supplements or iodised salt. Information on iodine intake from supplements was not available therefore was not included in the dietary intake assessment. Discretionary salt use was not measured in the 1995 Australian National Nutrition Survey (1995 NNS) nor the 1997 New Zealand National Nutrition Survey (1997 NNS), therefore intake of iodine from discretionary salt use could not be accurately determined.

[1] 1 part per million (ppm) is equal to 1 mg/kg

The dietary intake assessment was conducted using dietary modelling techniques that combine food consumption data, derived from the 1995 Australian National Nutrition Survey and 1997 New Zealand National Nutrition Survey, with food chemical concentration data to estimate the intake of the food chemical from the diet. The dietary intake assessment was conducted using FSANZ's dietary modelling computer program, DIAMOND.

Dietary intake = food chemical concentration x food consumption

The potential dietary intake of iodine was estimated by combining:

- usual patterns of food consumption, as derived from national nutrition survey (NNS) data;
- the naturally occurring levels of iodine in foods; and
- the iodine residues in fruits, vegetables (including herbs), nuts and eggs resulting from an elemental iodine wash, as indicated by the applicant.

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 these surveys used a 24-hour food recall methodology to collect food consumption data.

The dietary intake assessment was conducted for both Australian and New Zealand populations. Dietary intakes were assessed for a range of age-gender categories for the purpose of comparison with the PTDI, RDIs and EARs. Further details regarding RDIs and EARs can be found in the Nutrition Report at Attachment 5. RDIs and EARs are given for slightly different age groups. The population groups assessed against each reference health standard are listed in Table 1 below:

Table 1: Population Groups Assessed Against Each Reference Health Standard

Reference Health Standard	Country	Population Group Assessed
PTDI	Australia	2-6 years 2 years and above
	New Zealand	15 years and above
RDI	Australia	2-3 years 4-7 years 8-11 years Boys 12-18 years Girls 12-18 years
	New Zealand	Boys 15-18 years Girls 15-18 years
EAR	Australia	2-3 years 4-8 years 9-13 years 14-18 years
	New Zealand	15-18 years

A dietary intake assessment could not be conducted for New Zealand children aged below 15 years since there are no food consumption data available in DIAMOND for this population group at the current time. A dietary intake assessment for comparison with the PTDI was conducted on Australian children, particularly those aged between 2 and 6 years, because children generally have higher intakes due to their smaller body weight, and they consume more food per kilogram of body weight compared to adults.

Scenarios for dietary modelling

A baseline intake and two post-treatment scenarios were examined in the dietary intake assessment: Scenario 1 applied a peeling factor to those fruits and vegetables that may be consumed with the peel either on or off (e.g. apples); and Scenario 2 assumed that fruits and vegetables that may be consumed with the peel on or off are always consumed unpeeled.

Baseline

A baseline iodine dietary intake model was conducted to estimate iodine dietary intake before permission to use iodine as a washing agent was considered.

Scenario 1

The applicant stated that all of the iodine stays on the surface of the produce, essentially remaining on the surface or within a few millimetres of the surface of the produce. Therefore, removal of the peel from fruits and vegetables and the shell from nuts results in the removal of additional iodine residues from the elemental iodine wash. Given this information, the first scenario (Scenario 1) applies a peeling factor to the iodine concentrations of those fruits and vegetables that may be consumed either peeled or unpeeled (e.g. apples). These peeling data were derived from the 1995 NNS for Australia and the 1997 NNS for New Zealand from the proportion of each commodity reported as consumed peeled, unpeeled, juiced and canned. These data are listed in Table 2. This scenario reflects a more accurate estimate of the likely extent to which an elemental iodine wash will impact on the iodine dietary intakes for Australian and New Zealand population groups.

Scenario 2

The second scenario (Scenario 2) assumes that fruits and vegetables that may be consumed either peeled or unpeeled (e.g. apples) are always eaten unpeeled. Scenario 2 is a worst-case scenario. It is likely that actual intakes post-iodine wash treatment will fall somewhere between Scenario 1 and Scenario 2.

Table 2: Peeling factors applied to fruits and vegetables that may be consumed either peeled or unpeeled (Scenario 1)

Commodity	Percentage Consumed Unpeeled (%)	
	Australia	New Zealand
Apples	61	68
Apricots	95	94
Nectarines	91	92
Peaches	44	29
Pears	66	69
Peas, green	7	1
Potatoes	22	16
Tomatoes	81	68

Iodine Concentration Levels

The levels of iodine in foods that were used to establish the baseline level of estimated dietary intake of iodine were derived from a number of sources including Australian, New Zealand, British, and German food composition data, the 1997/8 and 2003/4 New Zealand Total Diet Surveys, the Australian Dairy Corporation and the applicant. The Applicant provided data on the increases in iodine concentrations in foods in parts per billion[2] (ppb). These were converted to mg/kg concentrations for use in the DIAMOND program.

Data on the increase in iodine levels for a number of specific fruit and vegetable commodities were provided by the applicant, with the increase in iodine levels being related to the surface area to volume ratio of the produce. For medium sized smooth skinned produce dipped in 30 mg/kg iodine, iodine increases by 0.100-0.150 mg/kg; for rougher skinned produce, iodine increases by 0.200-0.300 mg/kg; and for very high surface area produce, iodine increases by approximately 3.000 mg/kg. The increase in iodine residues in raw liquid egg following a 30 mg/kg iodine wash is 0.074 mg/kg.

The foods and their iodine levels for Australia and New Zealand, for baseline, Scenario 1 and Scenario 2, are shown below in Tables 3 and 4, respectively.

Table 3: Iodine levels in foods available in Australia before and after washing fruits, vegetables (including herbs), nuts and eggs with elemental iodine

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
AP0001	Honey	3.4	3.4	3.4	2,3
DM, GS	Sugars	6.7	6.7	6.7	2,3
CF, GC	Cereal foods	16.8	16.8	16.8	2,3,4
CF0600	Bran, processed and unprocessed	10.0	10.0	10.0	2,3
CF1210	Germ	20.0	20.0	20.0	2
CM	Bran, unprocessed	8.9	8.9	8.9	2,3
CM1205	Rice	5.1	5.1	5.1	2,3
DF	Dried fruits	8.4	8.4	8.4	2
DF0295	Dried dates	14.8	14.8	14.8	2,3

[2] One part per billion (ppb) is equal to 0.001 mg/kg (1 µg/kg)

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
DT	Teas	15.0	15.0	15.0	2,3
DV	Dried vegetables	236.3	236.3	236.3	8
FB	Berries and other small fruits	1.8	151.8	151.8	4
FB0269	Grapes	2.0	2.0	2.0	4
FB02691	Wine	23.3	23.3	23.3	2
FB0275	Strawberries	1.5	301.5	301.5	4
FC	Citrus fruits	73.4	73.4	73.4	7
FC0004	Oranges	73.4	73.4	73.4	7
FI	Tropical fruits – inedible peel (smooth skinned)	0.1	0.1	0.1	2
FI0326	Avocado	1.0	1.0	1.0	4
FI0327	Banana	0.1	0.1	0.1	2
FI0341	Kiwifruit	5.5	5.5	5.5	2,4
FI0353	Pineapple	10.0	10.0	10.0	2
FI0331, FI0332, FI0334, FI0338, FI0342, FI0343, FI0356, FI0358	Tropical fruits – inedible peel (rough skinned)	5.5	5.5	5.5	2,4
FP	Pome fruits	9.6	159.6	159.6	2,4
FP0226	Apples	6.3	97.8	156.3	2,4
FP0230	Pears	13.0	112.0	163.0	2,4
FS	Stone fruits (smooth skinned)	29.9	179.9	179.9	7
FS0240	Apricots	50.5	335.5	350.5	7
FS0245	Nectarines	29.9	166.4	179.9	7
FS0247	Peaches	50.5	182.5	350.5	7
FT, DM0305	Tropical fruit – edible peel	15.0	165.0	165.0	5
HH	Herbs	75.8	3075.8	3075.8	8
HS	Spices	75.8	75.8	75.8	8
IM	Molluscs	1050.0	1050.0	1050.0	1
MF	Other mammalian fats (not cattle, pig or sheep)	27.7	27.7	27.7	1,2
MF0812	Cattle fat	50.0	50.0	50.0	1
MF0818	Pig fat	22.9	22.9	22.9	2
MF0822	Sheep fat	0.2	0.2	0.2	2
ML	Dairy products	82.7	82.7	82.7	6
MM	Other mammalian meats (not cattle, pig or sheep)	19.8	19.8	19.8	2,3,4
MM0812	Cattle meat	17.8	17.8	17.8	2,3,4
MM0818	Pig meat	25.3	25.3	25.3	2,3,4
MM0822	Sheep meat	13.9	13.9	13.9	2,3,4
MO	Mammalian offal	65.6	65.6	65.6	2,3,4
OC, OR	Fats and oils	1.6	1.6	1.6	2,3
PE	Eggs	501.0	575.0	575.0	1,7
PF, PM, PO	Chicken meat and offal	53.0	53.0	53.0	2,3
SB	Coffee, cocoa, cola	5.3	5.3	5.3	2,4
SO, CO0691, TN	Oilseeds and nuts	42.1	42.1	42.1	1
SO0697	Peanuts	32.5	32.5	32.5	1
TN0663	Cashews	100.0	100.0	100.0	1

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
VA	Bulb vegetables	12.5	162.5	162.5	2,4
VA0384	Leeks	12.5	162.5	162.5	2,4
VA0386	Onions	12.5	63.5	63.5	2,4
VB	Brassica vegetables	9.5	407.5	407.5	2,4
VB0041	Cabbage	13.0	13.0	13.0	2,4
VB0400	Broccoli	1.5	540.0	540.0	4
VB0404	Cauliflower	5.8	403.8	403.8	2,4
VC	Cucurbit vegetables	9.7	159.7	159.7	2,4,7
VC0046	Melons, except watermelon	27.0	27.0	27.0	7
VC0424	Cucumber	1.0	151.0	151.0	4
VC0429	Pumpkin	5.5	5.5	5.5	2,4
VC0431	Zucchini	1.3	151.3	151.3	4
VC0432	Watermelon	1.0	1.0	1.0	4
VD	Pulses	69.3	69.3	69.3	2,3
VL	Leafy vegetables	75.8	744.8	744.8	7
VL0482	Lettuce	75.8	735.8	735.8	7
VO	Other fruiting vegetables (smooth skinned)	24.8	174.8	174.8	2,4,7
VO0051	Capsicum	1.0	151.0	151.0	4
VO0448	Tomatoes	36.0	157.5	186.0	7
VO0442, VO0446	Other fruiting vegetables (rough skinned)	21.4	321.4	321.4	2,4
VO0447	Sweetcorn	40.0	340.0	340.0	2
VO449, VO0450	Mushrooms	2.8	214.8	214.8	4
VP	Legume vegetables	125.0	595.0	595.0	1
VP00611	Beans, green	200.0	670.0	670.0	1
VP0529	Peas, garden	50.0	67.8	304.0	1
VR	Root and tuber vegetables	20.2	20.2	20.2	1,2,4,7
VR0508	Sweet potatoes	2.0	2.0	2.0	4
VR0574	Beetroot	50.0	50.0	50.0	1
VR0577	Carrots	8.3	293.5	293.5	2,4
VR0589	Potatoes	32.7	98.7	332.7	7
VS	Stalk and stem vegetables	5.0	426.0	426.0	4
VS0621	Asparagus	5.0	513.0	513.0	4
VS0624	Celery	5.0	338.0	338.0	4
WC	Crustacea	300.0	300.0	300.0	1
WD	Diadromous fish	600.0	600.0	600.0	1
WF	Other freshwater fish	625.0	625.0	625.0	1
WF0864, WF0866, WF0870, WF0897	Morwong	950.0	950.0	950.0	1
WR, WS	Other marine fish	254.2	254.2	254.2	1
WS0003	Fish portions	31.2	31.2	31.2	2,3
WS0004	Gemfish	250.0	250.0	250.0	1
WS0008	Flathead	75.0	75.0	75.0	1
WS0010	Snapper	400.0	400.0	400.0	1
WS0130	Sardine	100.0	100.0	100.0	1
WS0131	Flake	100.0	100.0	100.0	1

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
WS0858, WF0858	Bream	300.0	300.0	300.0	1
WS0927	Cod	250.0	250.0	250.0	1
WS0943	Mullet	100.0	100.0	100.0	1
WS0952	Tuna	150.0	150.0	150.0	1
WS0953	Whiting	50.0	50.0	50.0	1
WW	Water	0.8	0.8	0.8	2,4
XX0001	Seaweed	14,700.0	14,700.0	14,700.0	9
XX0002	Dry soup mixes	120.0	120.0	120.0	1

(1) unpublished Australian food composition data; (2) unpublished New Zealand food composition data; (3) 1997/8 New Zealand Total Diet Survey (Ministry of Health 2000); (4) 2003/4 New Zealand Total Diet Survey (Vannoort 2003); (5) German Food Composition tables (Souci et al 1994); (6) Australian Dairy Corporation (Australian Dairy Corporation 1999); (7) A493 applicant; (8) derived data; (9) British food composition data (Holland et al 1991).

Table 4: Iodine levels in foods available in New Zealand before and after washing fruits, vegetables (including herbs), nuts and eggs with elemental iodine

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
AP0001	Honey	3.4	3.4	3.4	2,3
DM, GS	Sugars	6.7	6.7	6.7	2,3
CF, GC	Cereal foods	16.8	16.8	16.8	2,3,4
CF0081, CF0600, CF0654, CM0001, CM0081, CM0654	Bran, processed and unprocessed	10.0	10.0	10.0	2,3
CF1210	Germ	20.0	20.0	20.0	2
CM	Bran, unprocessed	8.9	8.9	8.9	2,3
CM1205	Rice	5.1	5.1	5.1	2,3
DF	Dried fruits	8.4	8.4	8.4	2
DF0295	Dried dates	14.8	14.8	14.8	2,3
DT	Teas	15.0	15.0	15.0	2,3
DV	Dried vegetables	236.3	236.3	236.3	8
FB	Berries and other small fruits	1.8	151.8	151.8	4
FB0269	Grapes	2.0	2.0	2.0	4
FB02691	Wine	23.3	23.3	23.3	2
FB0275	Strawberries	1.5	301.5	301.5	4
FC	Citrus fruits	1.0	1.0	1.0	4
FC0004	Oranges	1.0	1.0	1.0	4
FI	Tropical fruits – inedible peel (smooth skinned)	0.1	0.1	0.1	2
FI0326	Avocado	1.0	1.0	1.0	4
FI0327	Banana	0.1	0.1	0.1	2
FI0341	Kiwifruit	5.5	5.5	5.5	2,4
FI0353	Pineapple	10.0	10.0	10.0	2
FI0331, FI0332, FI0334, FI0338, FI0342, FI0343, FI0356, FI0358	Tropical fruits – inedible peel (rough skinned)	5.5	5.5	5.5	2,4
FP	Pome fruits	9.6	159.6	159.6	2,4
FP0226	Apples	6.3	108.3	156.3	2,4
FP0230	Pears	13.0	116.5	163.0	2,4

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
FS	Stone fruits (smooth skinned)	1.0	151.0	151.0	4
FS0240	Apricots	85.0	367.0	385.0	2
FS0245	Nectarines	1.0	139.0	151.0	4
FS0247	Peaches	10.0	97.0	310.0	2
FT, DM0305	Tropical fruit – edible peel	15.0	165.0	165.0	5
HH	Herbs	13.4	3013.4	3013.4	8
HS	Spices	13.4	13.4	13.4	8
IM	Molluscs	1204.2	1204.2	1204.2	2,3,4
IM1003	Mussels	1602.5	1602.5	1602.5	3,4
IM1004	Oysters	1044.9	1044.9	1044.9	2,3,4
MF	Other mammalian fats (not cattle, pig or sheep)	16.5	16.5	16.5	2
MF0812	Cattle fat	20.0	20.0	20.0	2
MF0818	Pig fat	22.9	22.9	22.9	2
MF0822	Sheep fat	0.2	0.2	0.2	2
ML	Dairy products	85.7	85.7	85.7	2,3,4
MM	Other mammalian meats (not cattle, pig or sheep)	19.8	19.8	19.8	2,3,4
MM0812	Cattle meat	17.8	17.8	17.8	2,3,4
MM0818	Pig meat	25.3	25.3	25.3	2,3,4
MM0822	Sheep meat	13.9	13.9	13.9	2,3,4
MO	Mammalian offal	65.6	65.6	65.6	2,3,4
OC, OR	Fats and oils	1.6	1.6	1.6	2,3
PE	Eggs	444.2	518.2	518.2	2,3,4
PF, PM, PO	Chicken meat and offal	53.0	53.0	53.0	2,3
SB	Coffee, cocoa, cola	5.3	5.3	5.3	2,4
SO, CO0691, TN	Oilseeds and nuts	17.5	17.5	17.5	2,3
SO0697	Peanuts	17.5	17.5	17.5	2,3
VA	Bulb vegetables	12.5	162.5	162.5	2,4
VA0384	Leeks	12.5	162.5	162.5	2,4
VA0386	Onions	12.5	63.5	63.5	2,4
VB	Brassica vegetables	9.5	407.5	407.5	2,4
VB0041	Cabbage	13.0	13.0	13.0	2,4
VB0400	Broccoli	1.5	540.0	540.0	4
VB0404	Cauliflower	5.8	403.8	403.8	2,4
VC	Cucurbit vegetables	2.9	152.9	152.9	2,4
VC0046	Melons, except watermelon	1.0	1.0	1.0	4
VC0424	Cucumber	1.0	151.0	151.0	4
VC0429	Pumpkin	5.5	5.5	5.5	2,4
VC0431	Zucchini	1.3	151.3	151.3	4
VC0432	Watermelon	1.0	1.0	1.0	4
VD	Pulses	69.3	69.3	69.3	2,3
VD0541	Soya bean	85.5	85.5	85.5	3
VD05411	Tofu	5.1	5.1	5.1	2,3
VL	Leafy vegetables	13.4	682.4	682.4	4
VL0464	Silverbeet	24.0	693.0	693.0	4
VL0482	Lettuce	2.8	662.8	662.8	4

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
VO	Other fruiting vegetables (smooth skinned)	8.1	158.1	158.1	2,4
VO0051	Capsicum	1.0	151.0	151.0	4
VO0448	Tomatoes	10.4	112.4	160.4	2,4
VO0442, VO0446	Other fruiting vegetables (rough skinned)	21.4	321.4	321.4	2,4
VO0447	Sweetcorn	40.0	340.0	340.0	2
VO449, VO0450	Mushrooms	2.8	214.8	214.8	4
VP	Legume vegetables	6.7	476.7	476.7	2,3
VP00611	Beans, green	10.0	480.0	480.0	2
VP0529	Peas, garden	5.1	7.6	259.1	2
VR	Root and tuber vegetables	8.1	8.1	8.1	2,4
VR0508	Sweet potatoes	2.0	302.0	302.0	4
VR0505	Taro	9.0	9.0	9.0	4
VR0574	Beetroot	10.0	10.0	10.0	2
VR0577	Carrots	8.3	293.5	293.5	2,4
VR0589	Potatoes	9.6	57.6	309.6	4
VS	Stalk and stem vegetables	5.0	426.0	426.0	4
VS0621	Asparagus	5.0	513.0	513.0	4
VS0624	Celery	5.0	338.0	338.0	4
WC	Crustacea	300.0	300.0	300.0	1
WD	Diadromous fish	349.0	349.0	349.0	2,3
WR, WS	Other marine fish	202.3	202.3	202.3	2,3,4
WS0003	Fish portions	31.2	31.2	31.2	2,3
WS0006	Orange Roughy	10.0	10.0	10.0	2
WS0014	Hoki	110.8	110.8	110.8	2,4
WS0952	Tuna	230.0	230.0	230.0	2
WW	Water	0.8	0.8	0.8	2,4
XX0001	Seaweed	14,700.0	14,700.0	14,700.0	9
XX0002	Dry soup mixes	120.0	120.0	120.0	1

(1) unpublished Australian food composition data; (2) unpublished New Zealand food composition data; (3) 1997/8 New Zealand Total Diet Survey (Ministry of Health 2000); (4) 2003/4 New Zealand Total Diet Survey (Vannoort 2003); (5) German Food Composition tables (Souci et al 1994); (6) Australian Dairy Corporation (Australian Dairy Corporation 1999); (7) A493 applicant; (8) derived data; (9) British food composition data (Holland et al 1991).

How were the estimated dietary intakes calculated?

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

Where estimated dietary intakes are expressed per kilogram of body weight, each individuals' total dietary intake is divided by their own body weight, the results ranked, and population statistics derived.

Where estimated intakes are expressed as a percentage of the reference health standard, each individual's total intake is calculated as a percentage of the reference health standard (either using the total intakes in units per day or units per kilogram of body weight per day, depending on the units of the reference health standard), the results are then ranked, and population statistics derived.

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

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, beef in beef pie.

Assumptions in the dietary modelling

Assumptions made in the dietary modelling include:

- where a permission for an iodine wash is given to a food classification, all foods in that group contain iodine;
- all the foods within the group contain iodine at the levels specified in Table 2 for Australia and Table 3 for New Zealand;
- consumption of foods as recorded in the NNS represent current food consumption patterns;
- the mean iodine concentration values determined from the listed data sources are representative of the levels found in foods throughout Australia and New Zealand;
- all iodine present in foods is 100% bioavailable, therefore there are no inhibitors to iodine absorption (such as goitrogens) present in the diet;
- where the concentration of iodine in a food was reported as being less than the Limit of Detection (LOD), then the iodine concentration of the food was equal to half of the LOD value;
- where there were no Australian iodine data for specific food groups, it was assumed that New Zealand data were representative of these food groups, and vice versa for New Zealand;
- where there were no Australian or New Zealand data on iodine concentrations of food groups, it was assumed that overseas data (British and German) were representative of these food groups;
- where a food or food group has a zero concentration of iodine, it was not included in the intake assessment;
- there is a 100% market share for the elemental iodine wash system for those fruit, vegetable, herb, nut and egg commodities able to be washed with an elemental iodine wash system;
- where a food has a specified iodine concentration, this concentration is carried over to mixed foods where the food has been used as an ingredient e.g. apples in apple pie;
- one or more fruits or vegetables from a classification can be deemed to be representative of the entire classification (e.g. asparagus and celery representative of all stalk and stem vegetables);
- there is no consumption of iodine through salt (since NNS did not measure

discretionary salt use) or supplements;

- all fruits, vegetables (including herbs), nuts and eggs will be washed with the maximum concentration of iodine (i.e. 30 mg/kg iodine) and will not be rinsed after the iodine wash;
- fruits and vegetables are not washed with water prior to preparation and consumption in the home;
- there are no reductions in iodine concentrations on cooking;
- where a range of increases in iodine concentrations after washing with Iodoclean™ was specified for a fruit, vegetable or herb in the application, the upper end of the range was used for the intake assessment as a 'worst-case' scenario;
- all herbs are fresh herbs;
- the Australian and New Zealand populations remove the outer leaves from head lettuce and cabbages prior to use, thereby removing the additional iodine residues resulting from the elemental iodine wash;
- there are no increases in iodine residues for nuts, beetroot, sweet potatoes (Australia only), parsnips, citrus fruits, bananas, kiwifruit, pineapple, other fruits with inedible peel, and onions since these products are assumed to be always eaten peeled; and
- food manufacturers do not use iodised salt in their products. In a study by Gunton et al (1999), three major Australian food manufacturers of processed food were contacted and reported using only non-iodised salt.

These assumptions are likely to lead to a conservative estimate for iodine dietary intake.

Other information used in the dietary modelling

The other information used in conducting the dietary intake assessment includes:

-
- grapes are never washed prior to use for technological reasons;
- an iodine wash system will never be used on fruits and vegetables that are dried; and
- all of the iodine stays on the surface of the produce, essentially remaining on the surface or within a few millimetres of the surface. Therefore, removal of the peel from fruits and vegetables and the shell from nuts results in the removal of additional iodine residues from the elemental iodine wash system.

Limitations of the dietary modelling

A limitation of estimating dietary intake 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 intakes are likely to be higher than actual high percentile intakes over a lifetime.

Both the Australian and New Zealand NNSs did not measure discretionary salt use, therefore salt could not be included in the dietary intake assessments. Additionally, iodine intake from supplements was not included in the assessment since no data were available on their consumption or the iodine concentrations in these products.

Results

Estimated dietary intakes of iodine

The estimated dietary intakes of iodine for Australian and New Zealand population groups are shown in Table 5 and Figures 1 and 2. The results presented are for all population groups investigated for the purpose of comparison with the PTDI, RDIs and EARs. There is some overlap between different population groups since the RDIs and EARs are for slightly different age groups.

Baseline

Mean iodine dietary intakes were estimated as 1.3 µg/kg bw/day (94 µg/person/day) for the New Zealand population aged 15 years and above, 1.7 µg/kg bw/day (94 µg/person/day) for the Australian population 2 years and above, and 4.2 µg/kg bw/day (77 µg/person/day) for Australians aged 2-6 years.

At the 95th percentile, baseline dietary intakes of iodine were estimated as being 3.1 µg/kg bw/day (214 µg/person/day) for the New Zealand population aged 15 years and above, 4.6 µg/kg bw/day (212 µg/person/day)for the Australian population 2 years and above, and 8.4 µg/kg bw/day (147 µg/person/day) for Australians aged 2-6 years.

Scenario 1

Estimated mean dietary intakes of iodine were 2.2 µg/kg bw/day (156 µg/person/day) for the New Zealand population aged 15 years and above, 2.7 µg/kg bw/day (155 µg/person/day) for the Australian population 2 years and above, and 6.5 µg/kg bw/day (118 µg/person/day) for Australians aged 2-6 years.

Scenario 1 estimated 95th percentile dietary intakes of iodine were 4.7 µg/kg bw/day (327 µg/person/day) for the New Zealand population aged 15 years and above, 6.6 µg/kg bw/day (324 µg/person/day) for the Australian population 2 years and above, and 13.1 µg/kg bw/day (225 µg/person/day) for Australians aged 2-6 years.

Scenario 2

Scenario 2 mean dietary intakes of iodine were estimated at 2.7 µg/kg bw/day (196 µg/person/day) for the New Zealand population aged 15 years and above, 3.1 µg/kg bw/day (178 µg/person/day) for the Australian population 2 years and above, and 7.4 µg/kg bw/day (134 µg/person/day) for Australians aged 2-6 years.

The estimated 95th percentile dietary intakes of iodine were 5.6 µg/kg bw/day (398 µg/person/day) for the New Zealand population aged 15 years and above, 7.6 µg/kg bw/day (370 µg/person/day for the Australian population 2 years and above, and 14.8 µg/kg bw/day (254 µg/person/day) for Australians aged 2-6 years.

Table 5: Estimated dietary intakes of iodine for Australian and New Zealand population groups

Country	Population group	Average body weight (kg)	Number of consumers of iodine[♦]	Consumers as a % of total respondents[#]	Mean all consumers µg/kg bw/day (µg/person/day)			95th percentile consumers µg/kg bw/day (µg/person/day)		
					Baseline	Scenario 1	Scenario 2	Baseline	Scenario 1	Scenario 2
Australia	Whole population (2 years+)	67	13857	100	1.7 (94.1)	2.7 (154.5)	3.1 (178.4)	4.6 (212.2)	6.6 (324.1)	7.6 (369.8)
	2-6 years	19	989	100	4.2 (76.8)	6.5 (117.7)	7.4 (133.6)	8.4 (146.8)	13.1 (224.6)	14.8 (254.3)
	2-3 years	16	383	100	5.0 (76.7)	7.7 (118.8)	8.8 (135.1)	9.2 (153.7)	14.9 (234.7)	17.2 (259.5)
	4-7 years	22	799	100	3.6 (78.3)	5.5 (118.8)	6.3 (135.5)	7.2 (146.8)	10.7 (222.9)	11.9 (254.5)
	4-8 years	24	977	100	3.6 (81.6)	5.4 (122.9)	6.1 (140.3)	7.1 (159.4)	10.5 (243.1)	11.6 (266.2)
	8-11 years	36	739	100	2.8 (94.1)	4.1 (140.5)	4.8 (163.5)	5.8 (192.7)	8.6 (294.1)	9.6 (331.2)
	9-13 years	43	913	100	2.4 (97.5)	3.6 (146.0)	4.2 (171.2)	5.2 (208.0)	7.6 (302.3)	8.9 (340.6)
	Boys 12-18 years	63	564	100	2.1 (124.1)	3.0 (179.8)	3.6 (214.0)	4.8 (270.1)	6.2 (364.9)	7.5 (433.6)
	Girls 12-18 years	58	522	100	1.6 (91.3)	2.5 (141.3)	3.0 (164.0)	3.9 (211.5)	5.3 (292.4)	6.1 (333.8)
	14-18 years	64	734	100	1.8 (110.7)	2.7 (164.5)	3.1 (193.7)	4.2 (256.2)	5.7 (354.6)	6.5 (398.9)

New Zealand	Whole population (15 years+)	71	4636	100	1.3 (93.9)	2.2 (156.4)	2.7 (196.2)	3.1 (213.5)	4.7 (326.7)	5.6 (398.4)
	Boys 15-18 years	69	109	100	1.5 (103.2)	2.3 (162.1)	3.1 (213.0)	4.4 (297.3)	5.6 (363.1)	7.1 (430.6)
	Girls 15-18 years	61	137	100	1.4 (84.6)	2.4 (140.5)	2.9 (174.1)	3.4 (183.3)	5.4 (266.5)	6.0 (327.8)
	15-18 years	65	246	100	1.5 (93.1)	2.3 (150.3)	3.0 (191.6)	3.7 (218.6)	5.3 (345.1)	6.3 (382.3)

♦ Consumers only – This only includes the people who have consumed a food that contains iodine, in this case, all respondents are consumers.
Respondents – This includes all members of the survey population.

Figure 1. Estimated mean dietary iodine intakes before and after approval of iodine as a washing agent for fruits, vegetables (including herbs), nuts and eggs for Australian and New Zealand population groups.

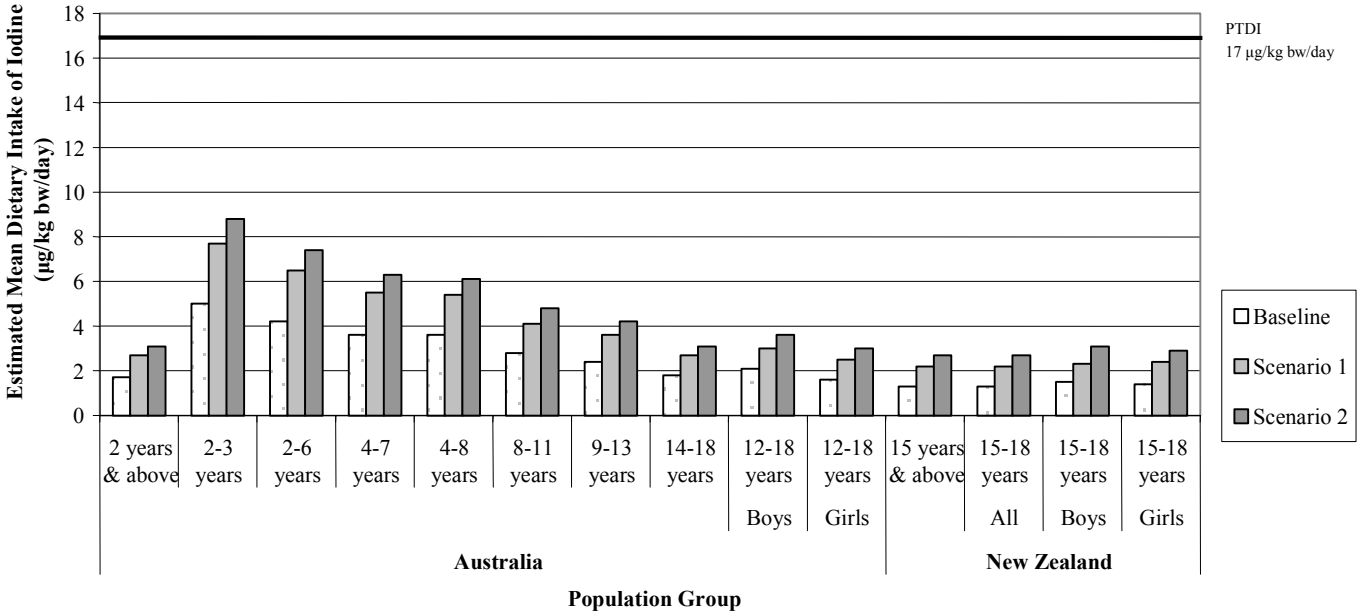
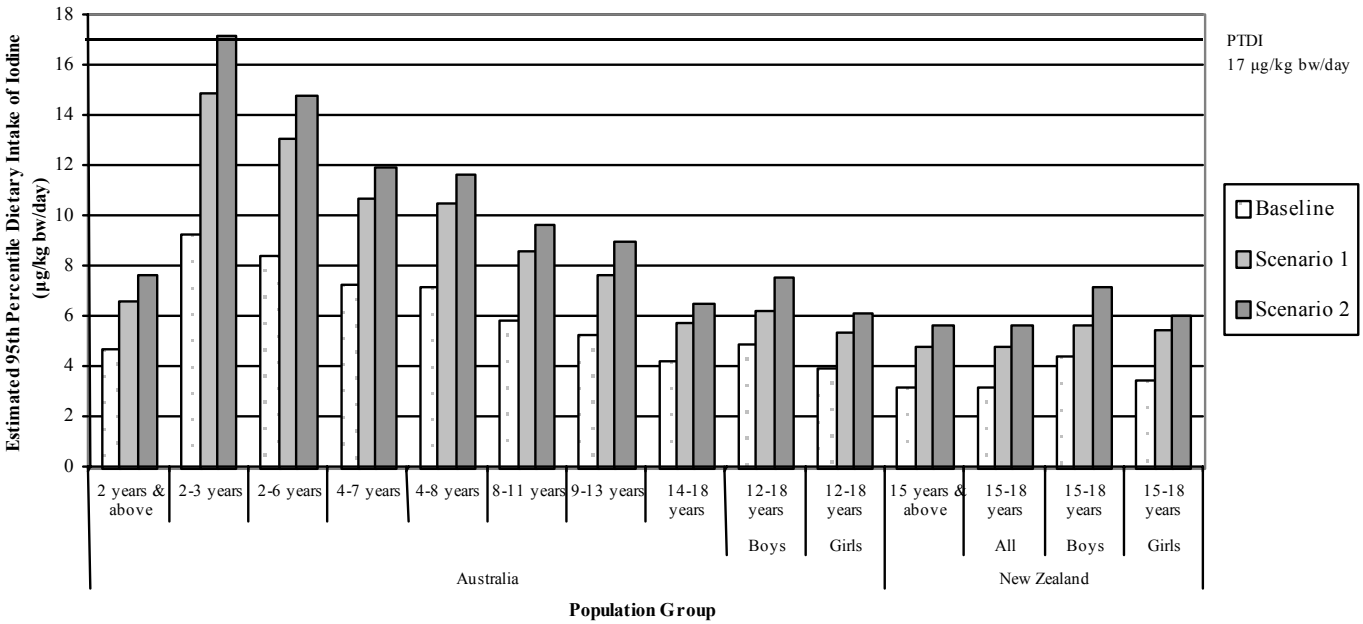


Figure 2. Estimated 95th percentile dietary iodine intakes before and after approval of iodine as a washing agent for fruits, vegetables (including herbs), nuts and eggs for Australian and New Zealand population groups.



Foods contributing to total estimated dietary intakes of iodine

The foods that contributed to the total estimated intakes of iodine are shown in Table 6 and Figures 3-5. These are displayed for the total population models as well as for the younger age group of 2-6 year old Australians.

Baseline

The major contributors (>5%) to iodine dietary intake for all Australians aged 2 years and above at baseline were dairy products (53.1%), all fruits (13.7%), citrus fruits (10.7%), all vegetables (including herbs) (9.4%), eggs (7.9%) and seafood (including seaweed) (6.5%). The major contributors for Australian children aged 2-6 years were dairy products (64.6%), all fruits (16.2%), citrus fruits (13.4%), eggs (5.8%), and all vegetables (including herbs) (5.3%). For New Zealanders aged 15 years and above, the major contributors were dairy products (63.7%), eggs (11.0%), seafood (including seaweed) (9.9%), and meat and poultry (5.0%).

Scenario 1

The Scenario 1 major contributors for all Australians aged 2 years and above were all vegetables (including herbs) (35.7%), dairy products (32.4%), all fruits (16.8%), other fruiting vegetables (8.2%), root and tuber vegetables (7.7%), citrus fruits (6.5%), leafy vegetables (6.3%), pome fruits (6.1%), eggs (5.6%), and Brassica vegetables (5.3%). The major contributors for Australian children aged 2-6 years were dairy products (42.2%), all fruits (28.9%), all vegetables (including herbs) (19.3%), pome fruits (16.3%), citrus fruits (8.7%), root and tuber vegetables (6.1%), and other fruiting vegetables (5.4%). For New Zealanders aged 15 years and above, the major contributors were dairy products (38.2%), all vegetables (including herbs) (32.3%), all fruits (10.1%), root and tuber vegetables (9.6%), eggs (7.7%), leafy vegetables (6.6%), other fruiting vegetables (6.1%), seafood (including seaweed) (5.9%), and pome fruits (5.0%).

Scenario 2

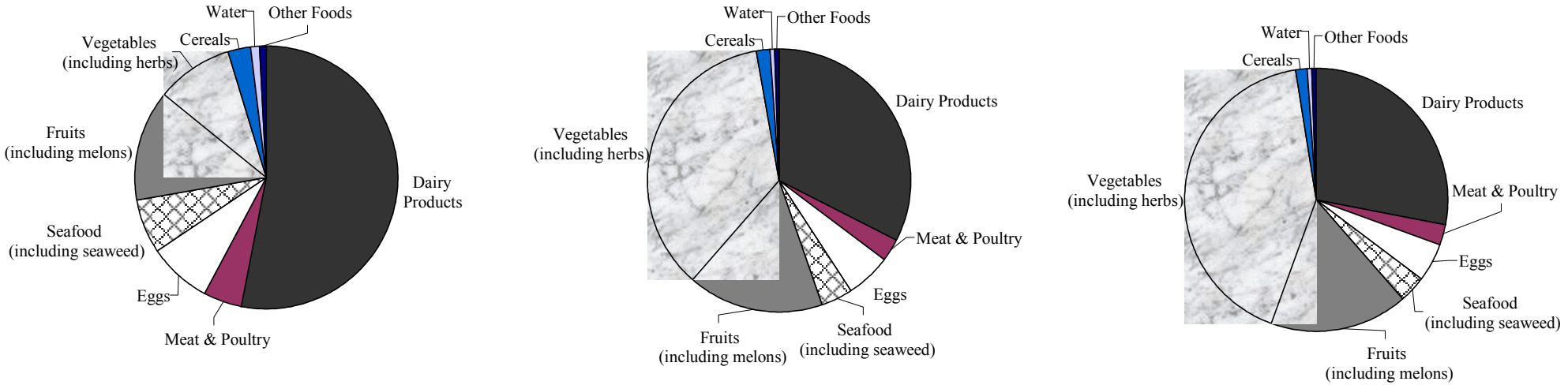
For all Australians aged 2 years and above, the major contributors to iodine dietary intake were all vegetables (including herbs) (41.9%), dairy products (28.0%), all fruits (17.0%), root and tuber vegetables (15.3%), other fruiting vegetables (8.0%), pome fruits (6.9%), citrus fruits (5.7%), and leafy vegetables (5.5%). The major contributors for Australian children aged 2-6 years were dairy products (37.1%), all fruits (28.7%), all vegetables (including herbs) (25.7%), pome fruits (16.9%), root and tuber vegetables (12.7%), citrus fruits (7.7%), and other fruiting vegetables (5.2%). For New Zealanders aged 15 years and above, the major contributors were all vegetables (including herbs) (43.5%), dairy products (30.5%), root and tuber vegetables (22.8%), all fruits (10.7%), eggs (6.1%), other fruiting vegetables (6.0%), pome fruits (5.7%), and leafy vegetables (5.2%).

Table 6: Contributors to total iodine dietary intakes for Australia and New Zealand, for different population groups

Country	Age group	Major contributing foods	Percentage of total iodine intakes (%)		
			Baseline	Scenario 1	Scenario 2
Australia	Whole population (2 years and above)	Dairy products	53.1	32.4	28.0
		Fruits:	13.7	16.8	17.0
		citrus fruits	10.7	6.5	5.7
		pome fruits	0.7	6.1	6.9
		Vegetables (including herbs):	9.4	35.7	41.9
		Brassica vegetables	0.2	5.3	4.6
		leafy vegetables	1.1	6.3	5.5
		other fruiting vegetables	2.5	8.2	8.0
		root & tuber vegetables	2.6	7.7	15.3
		Eggs	7.9	5.6	4.8
		Seafood (including seaweed)	6.5	3.9	3.4
		Meat & poultry	4.6	2.8	2.4
		Cereals	2.8	1.7	1.5
		Water	1.0	0.6	0.5
		Other foods	1.0	0.6	0.5
	2-6 years	Dairy products	64.6	42.2	37.1
		Fruits:	16.2	28.9	28.7
		citrus fruits	13.4	8.7	7.7
		pome fruits	1.6	16.3	16.9
		Eggs	5.8	4.3	3.8
		Vegetables (including herbs):	5.3	19.3	25.7
		other fruiting vegetables	1.4	5.4	5.2
		root & tuber vegetables	2.0	6.1	12.7
		Meat & poultry	2.6	1.7	1.5
		Cereals	2.2	1.4	1.3
		Seafood (including seaweed)	1.9	1.2	1.1
		Water	0.7	0.5	0.4
		Other foods	0.7	0.5	0.4
New Zealand	Whole population (15 years & above)	Dairy Products	63.7	38.2	30.5
		Eggs	11.0	7.7	6.1
		Seafood (including seaweed)	9.9	5.9	4.7

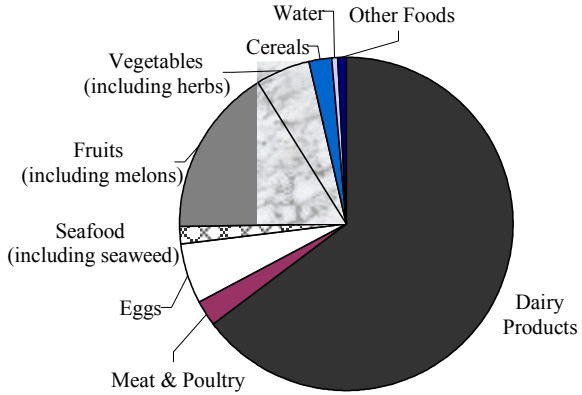
Country	Age group	Major contributing foods	Percentage of total iodine intakes (%)		
			Baseline	Scenario 1	Scenario 2
		Meat & Poultry	5.0	3.0	2.4
		Vegetables (including herbs):	3.9	32.3	43.5
		leafy vegetables	0.2	6.6	5.3
		other fruiting vegetables	0.9	6.1	6.0
		root and tuber vegetables	1.6	9.6	22.8
		Fruits:	2.1	10.1	10.7
		pome fruits	0.5	5.0	5.7
		Cereals	2.9	1.7	1.4
		Water	1.0	0.6	0.5
		Other foods	0.7	0.4	0.3

Figure 3: Contributors to total iodine intakes for the Australian population aged 2 years and above



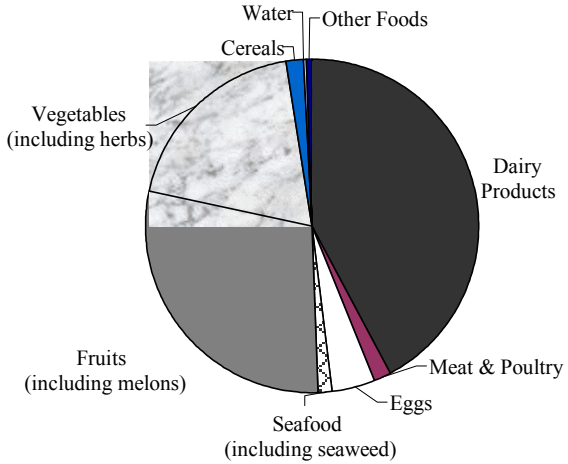
Baseline (94 µg/day)	Scenario 1 (155 µg/day)	Scenario 2 (178 µg/day)

Figure 4 Contributors to total iodine intakes for the Australian population aged 2-6 years



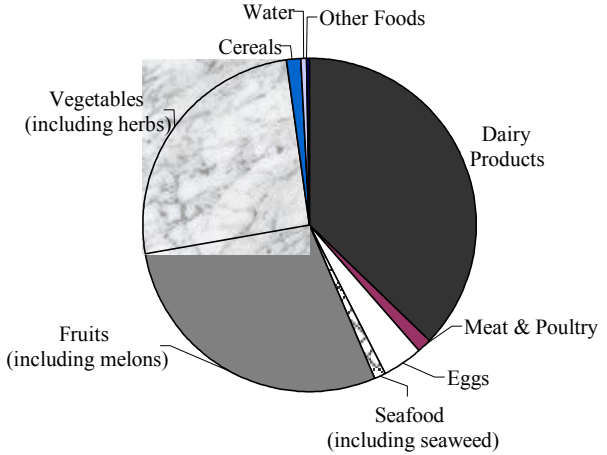
Baseline

(77 µg/day)



Scenario 1

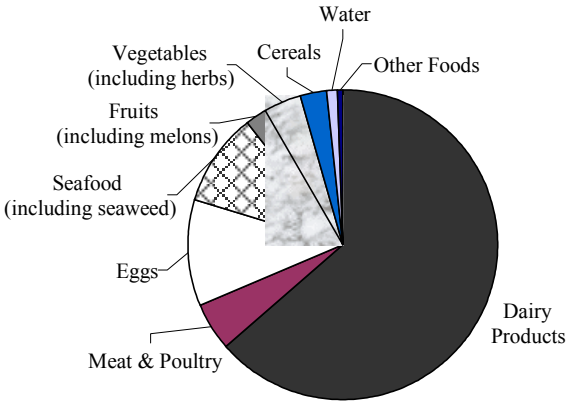
(118 µg/day)



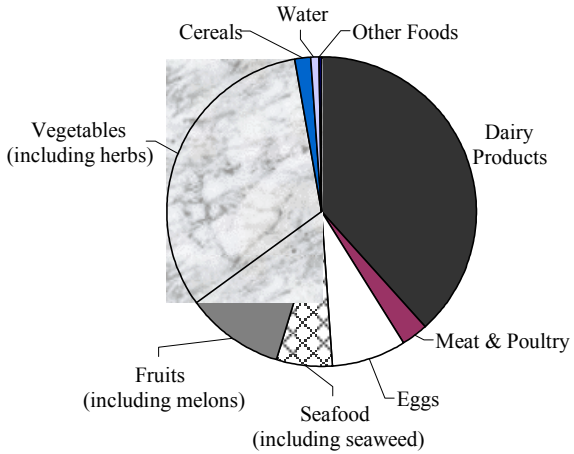
Scenario 2

(134 µg/day)

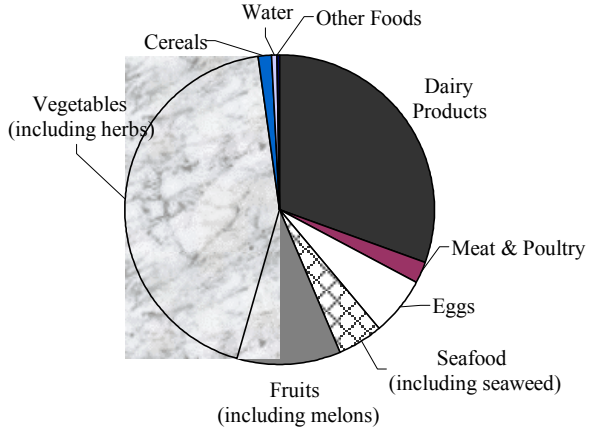
Figure 5: Contributors to total iodine intakes for the New Zealand population aged 15 years and above



Baseline
(94 µg/day)



Scenario 1
(156 µg/day)



Scenario 2
(196 µg/day)

Risk Characterisation

Comparison of the estimated dietary intakes with the PTDI

In order to determine if the level of dietary intake of iodine will be a public health and safety concern if an iodine wash is applied to fruits, vegetables (including herbs), eggs and nuts, the estimated dietary intakes were compared to a Provisional Tolerable Daily Intake (PTDI) of 17 μg/kg body weight/day that was set by the FAO/WHO Joint Expert Committee on Food Additives (JECFA) (WHO 1989).

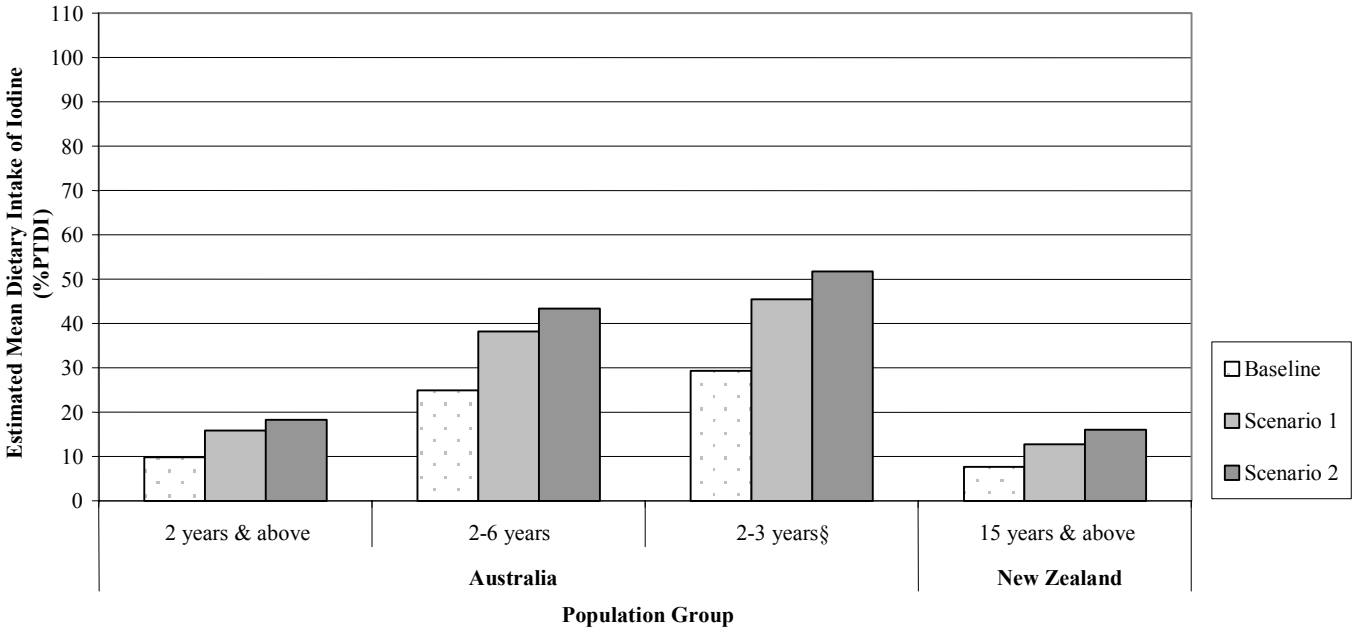
The results of the comparison between estimated dietary intake and the PTDI are given in Table 7 and Figures 6 and 7. Estimated mean and 95th percentile dietary intakes of iodine were below the PTDI of 17 μg/kg body weight/day (WHO 1989) for all population groups and all scenarios examined, with one exception. Although the dietary intakes of iodine for the 2-6 years age group were below the PTDI, it was noted that analysis of the 2-3 years age group high consumers (95th percentile) indicated that the PTDI was exceeded when it was assumed that fruits and vegetables that may be consumed with the peel on or off were consumed unpeeled (Scenario 2). Australian children aged 2-3 years had an estimated 95th percentile dietary intake of 17.2 μg/kg bw/day (260 μg/person/day) which is equivalent to 101.2% of the PTDI. However, due to the conservative assumptions made in this calculation and that the use of 24 hour dietary survey data tends to over-estimate habitual food consumption amounts for high consumers, it is likely that the 95th percentile dietary intake is an over-estimate. Additionally, the PTDI is set for a lifetime of exposure and the 'all population' models are a good indicator of the likely dietary exposures for the population over a lifetime.

Table 7: Estimated dietary intakes of iodine compared to the PTDI

Country	Age group	Number of consumers of iodine♦	Consumers as a % of total respondents#	Mean all consumers (%PTDI*)			95th percentile consumers (%PTDI*)		
				Baseline	Scenario 1	Scenario 2	Baseline	Scenario 1	Scenario 2
Australia	Whole population (2 years+)	13857	100	9.8	15.8	18.3	26.9	39.1	44.4
	2-6 years	989	100	24.9	38.2	43.4	49.3	77.3	86.8
	2-3 years§	383	100	29.4	45.5	51.7	54.3	87.9	101.2
New Zealand	Whole population (15 years+)	4636	100	7.7	12.8	16.0	18.0	27.5	32.9

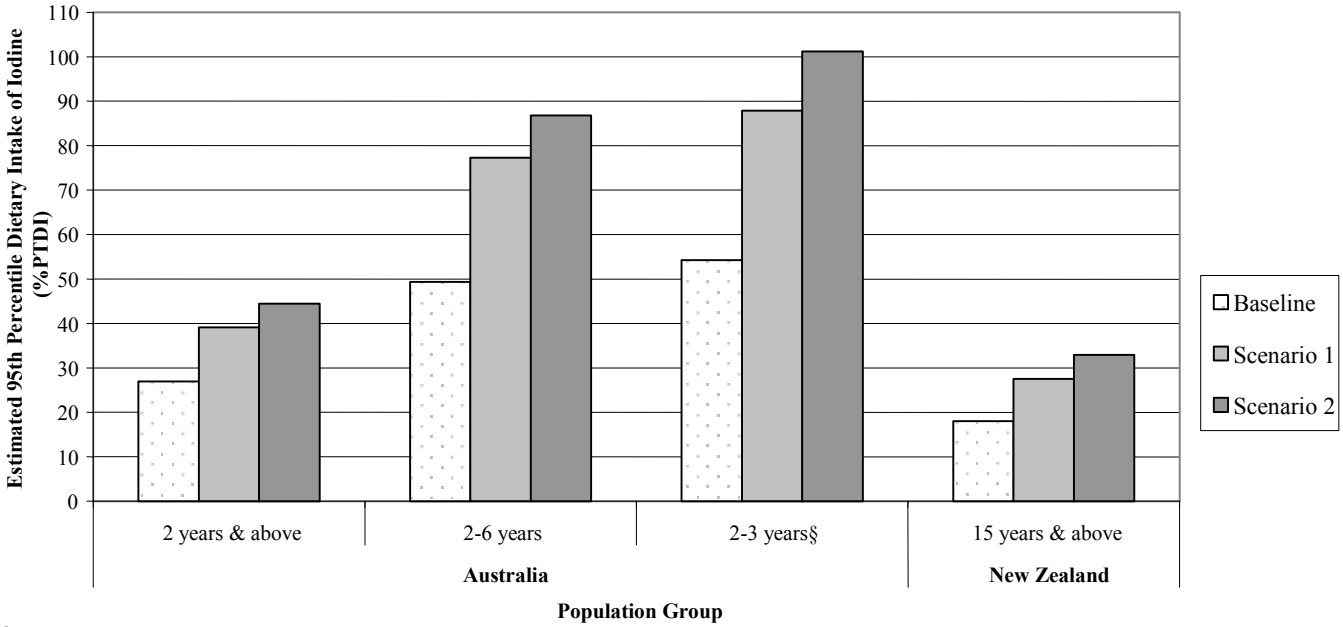
♦ Consumers only – This only includes the people who have consumed a food that contains iodine, in this case, all respondents are consumers.
Respondents – This includes all members of the survey population.
* PTDI = 17 µg/kg bw/day
§ A dietary intake assessment was conducted for the 2-3 years age group to allow a comparison to the RDI and EAR. In conducting this assessment, the dietary intake was noted to exceed the PTDI and, for this reason, the data for the 2-3 years age group are presented here.

Figure 6. Estimated mean dietary iodine intakes, as a percentage of the PTDI, before and after approval of iodine as a washing agent for fruits, vegetables (including herbs), nuts and eggs for Australian and New Zealand population groups.



§ A dietary intake assessment was conducted for the 2-3 years age group to allow a comparison to the RDI and EAR. In conducting this assessment, the dietary intake was noted to exceed the PTDI and, for this reason, the data for the 2-3 years age group are presented here.

Figure 7. Estimated 95th percentile dietary iodine intakes, as a percentage of the PTDI, before and after approval of iodine as a washing agent for fruits, vegetables (including herbs), nuts and eggs for Australian and New Zealand population groups.



§ A dietary intake assessment was conducted for the 2-3 years age group to allow a comparison to the RDI and EAR. In conducting this assessment, the dietary intake was noted to exceed the PTDI and, for this reason, the data for the 2-3 years age group are presented here.

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ATTACHMENT 5

COMBINED DIETARY EXPOSURE ASSESSMENT FOR A493 AND A528

Summary of results of iodine intake from combining A493 and A528

A dietary intake assessment was undertaken to determine the impact on dietary iodine intake if the requested changes to the Code from both applications A493 and A528 were approved. The assessment of the combination of requested permissions from A493 and A528 is referred to as 'both applications (A493 & A528)' hereafter. The assessment for 'both applications (A493 & A528)' was only conducted for Australian children 2-3 years of age.

Results for each separate Application can be found as an attachment to the relevant Draft Assessment report for each Application.

For the purpose of the 'both applications (A493 & A528)' dietary intake assessment, three different scenarios were examined:

- Baseline: naturally occurring levels of iodine in addition to currently permitted maximum quantities of iodine, with adjustments for variations in preparation method, in FSFYC were considered;
- Scenario 1: this scenario applies a peeling factor to the iodine concentrations of those fruits and vegetables that may be consumed either peeled or unpeeled (e.g. apples) and that may be washed with an elemental iodine wash. This scenario reflects a more accurate estimate of the likely extent to which an elemental iodine wash (A493) will impact on the iodine dietary intakes for Australian and New Zealand population groups. Scenario 1 also takes into account the increase in the maximum iodine level in FSFYC from 35 µg/serve to 70 µg/serve, with adjustments for variations in preparation method, by using an adjusted 'full fat milk equivalents' iodine concentration (A528);
- Scenario 2: this scenario assumes that fruits and vegetables that may be consumed either peeled or unpeeled (e.g. apples) are always eaten unpeeled after being treated with an elemental iodine wash (A493). Scenario 2 also takes into account the increase in the maximum iodine level in FSFYC from 35 µg/serve to 70 µg/serve, with adjustments for variations in preparation method, by using an adjusted 'full fat milk equivalents' iodine concentration (A528). Scenario 2 is a worst-case scenario.

At baseline, the estimated mean dietary intake of iodine for children aged 2-3 years for 'both applications (A493 & A528)' was 5.4 µg/kg bw/day (83 µg/person/day), with the 95th percentile intake being 9.9 µg/kg bw/day (165 µg/person/day). The baseline for 'both applications (A493 & A528)' is the same as that for the A528 baseline. For Scenario 1 for 'both applications (A493 & A528)', the estimated dietary intake of iodine was 8.7 µg/kg bw/day (133.9 µg/person/day) at the mean and 16.1 µg/kg bw/day (258.3 µg/person/day) at the 95th percentile. For Scenario 2 for 'both applications (A493 & A528)', the estimated dietary intake of iodine was 9.8 µg/kg bw/day (150.2 µg/person/day) at the mean and 18.7 µg/kg bw/day (289.4 µg/person/day) at the 95th percentile.

Estimated mean and 95th percentile dietary intakes of iodine were below the PTDI of 17 µg/kg body weight/day (WHO 1989) for the baseline scenario and for Scenario 1. Provisional Tolerable Daily Intakes (PTDI) are upper limits that are set for substances that do not accumulate in animals and humans (WHO 2001) and are estimates of the amount of a chemical that can be ingested daily over a lifetime without appreciable risk to health. For Scenario 2, mean dietary intake of iodine was below the PTDI, with 95th percentile intake exceeding the PTDI (110% PTDI). However, due to the conservative assumptions made in this calculation and taking into account that the use of 24-hour dietary survey data tends to over-estimate habitual food consumption amounts for high consumers, it is likely that the 95th percentile dietary intake is an over-estimate.

At baseline, the major contributors to iodine dietary intake were dairy products (70%) and fruits (15%). For Scenario 1, the major contributors to iodine dietary intake were dairy products (50%), fruits (28%) and vegetables (including herbs) (16%). For Scenario 2, the major contributors to iodine dietary intake were dairy products (44%), fruits (28%) and vegetables (including herbs) (22%).

Background

For the detailed background to each separate application for A493 and A528, please refer to the reports for each individual application, as only a summary from each is provided below.

Two separate applications were received by FSANZ requesting amendment of the Food Standards Code (the Code) in relation to iodine: (1) to Standard 1.3.3 'Processing Aids' – Clause 12 'Permitted bleaching agents, washing and peeling agents' to allow the use of iodine as a processing aid for fruits, vegetables, nuts and eggs (A493); and (2) to Standard 2.9.3 – Formulated Meal Replacements and Formulated Supplementary Foods to increase the maximum iodine limit in formulated supplementary foods for young children (FSFYC) from 35 µg per serve to 70 µg per serve (A528). One serve of a FSFYC is equivalent to 200 ml.

Formulated supplementary foods for young children (FSFYC) are for children aged 1-3 years. Since there are no Australian food consumption data for children aged below 2 years of age and there are no New Zealand food consumption data for children aged 1-3 years, dietary iodine intakes were only calculated for the population group of Australian children aged 2-3 years for 'both applications (A493 & A528)'.

In A493, baseline intakes of iodine were calculated using naturally occurring iodine concentrations. For children aged 2-3 years, estimated mean dietary intake of iodine was 5.0 µg/kg bw/day (76.7 µg/person/day) and 95th percentile dietary intake was estimated as 9.2 µg/kg bw/day (153.7 µg/person/day).

The baseline intakes of iodine for A528 differ from those in A493. This is because no FSFYC were consumed in the 1995 Australian National Nutrition Survey and, as a consequence, assumptions were made about the consumption of FSFYC. The baseline dietary intake of iodine for A528 assumed that 2-3 year old children in the NNS would replace 20% of full fat and unspecified fat content fluid cow's milk, including that used in cooking, with FSFYC. The dietary model used to estimate iodine intake converted all milk and milk products consumed to 'full fat milk equivalents', calculated on the basis of the fat content of the milk. 'Full fat milk equivalents' intake therefore includes fluid milks, cheese, cream, yoghurt etc.

Approximately 65% of 'full fat milk equivalents' intake for 2-3 year old children in the NNS was consumed as full fat or unspecified fat content fluid cow's milk. The A528 Applicant indicated that approximately 70% of consumers make up the product using milk with customers probably using less scoops per serve than those who use water to make up the product (more likely using the full 5 scoops per serve). The iodine concentration of 'full fat milk equivalents' was adjusted to take into account the differences in iodine concentration for the different preparation methods for FSFYC.

The A528 application baseline dietary iodine intake has been used as the baseline for the dietary intake assessment for 'both applications (A493 & A528)'. Details on how the iodine concentration of 'full fat milk equivalents' was adjusted to take into account the consumption of FSFYC can be found in Figure 1.

Dietary Intake Assessment provided by the Applicants

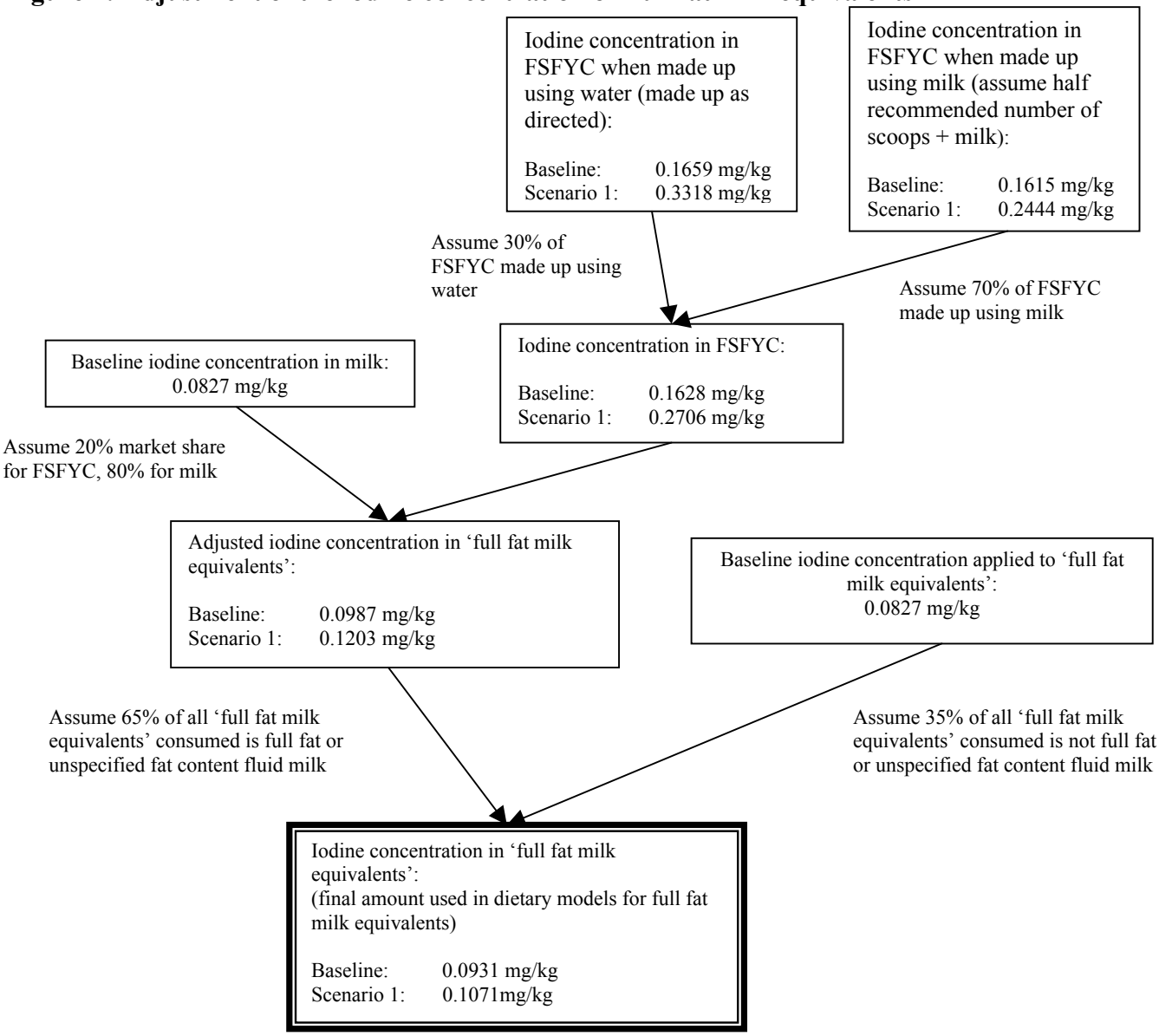
Please refer to the dietary intake assessment in Attachment 4 for A493 and the Draft Assessment Report for A528 for details on the dietary intake assessments submitted by each of the applicants.

FSANZ conducted its own dietary intake assessment for 'both applications (A493 & A528)' in order to take into account the potential impact of approval of iodine permissions from both A493 and A528.

Dietary Modelling

Refer to the reports for the individual applications for details regarding the dietary modelling techniques, dietary survey data, population groups, additional food consumption data used and assumptions made in dietary modelling.

Figure 1: Adjustment of the iodine concentration of 'full fat milk equivalents'



Iodine concentration levels

The levels of iodine in foods that were used to establish the baseline level of estimated dietary intake of iodine were derived from a number of sources including Australian, New Zealand, British, and German food composition data, the 1997/8 and 2003/4 New Zealand Total Diet Surveys, the Australian Dairy Corporation, the A493 Applicant, and the A528 Applicant. The foods and proposed levels of use for 'both applications (A493 & A528)' are shown below in Table 1.

Table 1: Iodine levels in foods available in Australia at baseline and with the proposed levels of use for 'both applications (A493 & A528)'

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
AP0001	Honey	3.4	3.4	3.4	2,3
DM, GS	Sugars	6.7	6.7	6.7	2,3
CF, GC	Cereal foods	16.8	16.8	16.8	2,3,4
CF0600	Bran, processed and unprocessed	10.0	10.0	10.0	2,3
CF1210	Germ	20.0	20.0	20.0	2
CM	Bran, unprocessed	8.9	8.9	8.9	2,3
CM1205	Rice	5.1	5.1	5.1	2,3
DF	Dried fruits	8.4	8.4	8.4	2
DF0295	Dried dates	14.8	14.8	14.8	2,3
DT	Teas	15.0	15.0	15.0	2,3
DV	Dried vegetables	236.3	236.3	236.3	8
FB	Berries and other small fruits	1.8	151.8	151.8	4
FB0269	Grapes	2.0	2.0	2.0	4
FB02691	Wine	23.3	23.3	23.3	2
FB0275	Strawberries	1.5	301.5	301.5	4
FC	Citrus fruits	73.4	73.4	73.4	7
FC0004	Oranges	73.4	73.4	73.4	7
FI	Tropical fruits – inedible peel (smooth skinned)	0.1	0.1	0.1	2
FI0326	Avocado	1.0	1.0	1.0	4
FI0327	Banana	0.1	0.1	0.1	2
FI0341	Kiwifruit	5.5	5.5	5.5	2,4
FI0353	Pineapple	10.0	10.0	10.0	2
FI0331, FI0332, FI0334, FI0338, FI0342, FI0343, FI0356, FI0358	Tropical fruits – inedible peel (rough skinned)	5.5	5.5	5.5	2,4
FP	Pome fruits	9.6	159.6	159.6	2,4
FP0226	Apples	6.3	97.8	156.3	2,4
FP0230	Pears	13.0	112.0	163.0	2,4
FS	Stone fruits (smooth skinned)	29.9	179.9	179.9	7
FS0240	Apricots	50.5	335.5	350.5	7
FS0245	Nectarines	29.9	166.4	179.9	7
FS0247	Peaches	50.5	182.5	350.5	7
FT, DM0305	Tropical fruit – edible peel	15.0	165.0	165.0	5
HH	Herbs	75.8	3075.8	3075.8	8

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
HS	Spices	75.8	75.8	75.8	8
IM	Molluscs	1050.0	1050.0	1050.0	1
MF	Other mammalian fats (not cattle, pig or sheep)	27.7	27.7	27.7	1,2
MF0812	Cattle fat	50.0	50.0	50.0	1
MF0818	Pig fat	22.9	22.9	22.9	2
MF0822	Sheep fat	0.2	0.2	0.2	2
ML	Milk ('full fat milk equivalents')	93.1	107.1	107.1	6,8,10
MM	Other mammalian meats (not cattle, pig or sheep)	19.8	19.8	19.8	2,3,4
MM0812	Cattle meat	17.8	17.8	17.8	2,3,4
MM0818	Pig meat	25.3	25.3	25.3	2,3,4
MM0822	Sheep meat	13.9	13.9	13.9	2,3,4
MO	Mammalian offal	65.6	65.6	65.6	2,3,4
OC, OR	Fats and oils	1.6	1.6	1.6	2,3
PE	Eggs	501.0	575.0	575.0	1,7
PF, PM, PO	Chicken meat and offal	53.0	53.0	53.0	2,3
SB	Coffee, cocoa, cola	5.3	5.3	5.3	2,4
SO, CO0691, TN	Oilseeds and nuts	42.1	42.1	42.1	1
SO0697	Peanuts	32.5	32.5	32.5	1
TN0663	Cashews	100.0	100.0	100.0	1
VA	Bulb vegetables	12.5	162.5	162.5	2,4
VA0384	Leeks	12.5	162.5	162.5	2,4
VA0386	Onions	12.5	63.5	63.5	2,4
VB	Brassica vegetables	9.5	407.5	407.5	2,4
VB0041	Cabbage	13.0	13.0	13.0	2,4
VB0400	Broccoli	1.5	540.0	540.0	4
VB0404	Cauliflower	5.8	403.8	403.8	2,4
VC	Cucurbit vegetables	9.7	159.7	159.7	2,4,7
VC0046	Melons, except watermelon	27.0	27.0	27.0	7
VC0424	Cucumber	1.0	151.0	151.0	4
VC0429	Pumpkin	5.5	5.5	5.5	2,4
VC0431	Zucchini	1.3	151.3	151.3	4
VC0432	Watermelon	1.0	1.0	1.0	4
VD	Pulses	69.3	69.3	69.3	2,3
VL	Leafy vegetables	75.8	744.8	744.8	7
VL0482	Lettuce	75.8	735.8	735.8	7
VO	Other fruiting vegetables (smooth skinned)	24.8	174.8	174.8	2,4,7
VO0051	Capsicum	1.0	151.0	151.0	4
VO0448	Tomatoes	36.0	157.5	186.0	7
VO0442, VO0446	Other fruiting vegetables (rough skinned)	21.4	321.4	321.4	2,4
VO0447	Sweetcorn	40.0	340.0	340.0	2
VO449, VO0450	Mushrooms	2.8	214.8	214.8	4
VP	Legume vegetables	125.0	595.0	595.0	1
VP00611	Beans, green	200.0	670.0	670.0	1
VP0529	Peas, garden	50.0	67.8	304.0	1

Food Code	Food Name	Concentration Level (µg/kg)			Source of Baseline Data
		Baseline	Scenario 1	Scenario 2	
VR	Root and tuber vegetables	20.2	20.2	20.2	1,2,4,7
VR0508	Sweet potatoes	2.0	2.0	2.0	4
VR0574	Beetroot	50.0	50.0	50.0	1
VR0577	Carrots	8.3	293.5	293.5	2,4
VR0589	Potatoes	32.7	98.7	332.7	7
VS	Stalk and stem vegetables	5.0	426.0	426.0	4
VS0621	Asparagus	5.0	513.0	513.0	4
VS0624	Celery	5.0	338.0	338.0	4
WC	Crustacea	300.0	300.0	300.0	1
WD	Diadromous fish	600.0	600.0	600.0	1
WF	Other freshwater fish	625.0	625.0	625.0	1
WF0864, WF0866, WF0870, WF0897	Morwong	950.0	950.0	950.0	1
WR, WS	Other marine fish	254.2	254.2	254.2	1
WS0003	Fish portions	31.2	31.2	31.2	2,3
WS0004	Gemfish	250.0	250.0	250.0	1
WS0008	Flathead	75.0	75.0	75.0	1
WS0010	Snapper	400.0	400.0	400.0	1
WS0130	Sardine	100.0	100.0	100.0	1
WS0131	Flake	100.0	100.0	100.0	1
WS0858, WF0858	Bream	300.0	300.0	300.0	1
WS0927	Cod	250.0	250.0	250.0	1
WS0943	Mullet	100.0	100.0	100.0	1
WS0952	Tuna	150.0	150.0	150.0	1
WS0953	Whiting	50.0	50.0	50.0	1
WW	Water	0.8	0.8	0.8	2,4
XX0001	Seaweed	14,700.0	14,700.0	14,700.0	9
XX0002	Dry soup mixes	120.0	120.0	120.0	1

(1) unpublished Australian food composition data; (2) unpublished New Zealand food composition data; (3) 1997/8 New Zealand Total Diet Survey (Ministry of Health 2000); (4) 2003/4 New Zealand Total Diet Survey (Vannoort 2003); (5) German Food Composition tables (Souci et al 1994); (6) Australian Dairy Corporation (Australian Dairy Corporation 1999); (7) A493 applicant; (8) derived data; (9) British food composition data (Holland et al 1991); (10) A528 Applicant.

Concentrations of iodine were assigned to food groups using DIAMOND food classification codes. Where the A493 Applicant provided a range of possible concentrations, the highest level in the range was used for calculating the estimated intakes in order to assume a worst-case scenario.

Scenarios for dietary modelling

Baseline

A baseline iodine dietary intake assessment was conducted for 'both applications' to estimate iodine dietary intake before permission to use iodine as a washing agent was considered and before permission to increase the maximum permitted iodine level in FSFYC from 35 µg/serve to 70 µg/serve, with adjustments for variations in preparation method, was considered. The baseline assessment incorporated naturally occurring levels of iodine in addition to currently permitted maximum quantities of iodine in FSFYC.

This was done by using an adjusted 'full fat milk equivalents' iodine concentration level to account for the consumption of FSFYC replacing 20% of full fat and unspecified milk consumption of 2-3 year olds.

Scenario 1

The first scenario (Scenario 1) applies a peeling factor to the iodine concentrations of those fruits and vegetables that may be consumed either peeled or unpeeled (e.g. apples). This scenario reflects a more accurate estimate of the likely extent to which an elemental iodine wash (A493) will impact on the iodine dietary intakes for Australian and New Zealand population groups. Scenario 1 also takes into account the increase in the maximum iodine level in FSFYC from 35 µg/serve to 70 µg/serve, with adjustments for variations in preparation method, by using an adjusted 'full fat milk equivalents' iodine concentration (A528).

Scenario 2

The second scenario (Scenario 2) assumes that fruits and vegetables that may be consumed either peeled or unpeeled (e.g. apples) are always eaten unpeeled after being treated with an elemental iodine wash (A493). Scenario 2 also takes into account the increase in the maximum iodine level in FSFYC from 35 µg/serve to 70 µg/serve, with adjustments for variations in preparation method, by using an adjusted 'full fat milk equivalents' iodine concentration (A528). Scenario 2 is a worst-case scenario.

How were the estimated dietary intakes calculated?

Please refer to the reports for A493 and for A528 for further details on the calculation of dietary intakes.

Assumptions in the dietary modelling

Please refer to the reports for A493 and for A528 for details on the assumptions used in the estimation of dietary intakes of iodine.

Other information used in the dietary modelling

The other information used in conducting the dietary intake assessment includes:

- grapes are never washed prior to use for technological reasons;
- an iodine wash system will never be used on fruits and vegetables that are dried; and
- all of the iodine stays on the surface of the produce, essentially remaining on the surface or within a few millimetres of the surface. Therefore, removal of the peel from fruits and vegetables and the shell from nuts results in the removal of additional iodine residues from the elemental iodine wash system.

Limitations of the dietary modelling

Please refer to the reports for A493 and for A528 for further details on the limitations of the dietary modelling.

Results

Estimated dietary intakes of iodine

The estimated dietary intakes of iodine are shown in Figure 1 (full results in Table A1.1 in Appendix 1). The results are presented for all survey respondents in the 2-3 year age group (n=383) because all respondents had an iodine intake due to the nutrient being ubiquitous in the food supply.

The estimated mean dietary intake of iodine for consumers was estimated at 5.4 µg/kg bw/day (83 µg/day) at baseline, 8.7 µg/kg bw/day (134 µg/day) for Scenario 1, and 9.8 µg/kg bw/day (150 µg/day) for Scenario 2. The estimated 95th percentile dietary intake of iodine was estimated at 9.9 µg/kg bw/day (165 µg/day) at baseline, 16.1 µg/kg bw/day (258 µg/day) for Scenario 1, and 18.7 µg/kg bw/day (289 µg/day) for Scenario 2.

Major contributing food groups to total estimated dietary intakes

The food group contributors to total iodine dietary intakes are shown in Figure 2. At baseline, the major contributors (<5%) to iodine dietary intake were dairy products (70%) and fruits (15%). For Scenario 1, the major contributors to iodine dietary intake were dairy products (50%), fruits (28%) and vegetables (including herbs) (16%). For Scenario 2, the major contributors to iodine dietary intake were dairy products (44%), fruits (28%) and vegetables (including herbs) (22%). A full list of all the food groups and their contributions can be found in Table A1.2 in Appendix 1.

Figure 1: Estimated dietary intakes of iodine from 'both applications' (A493 & A528) for Australian children aged 2-3 years

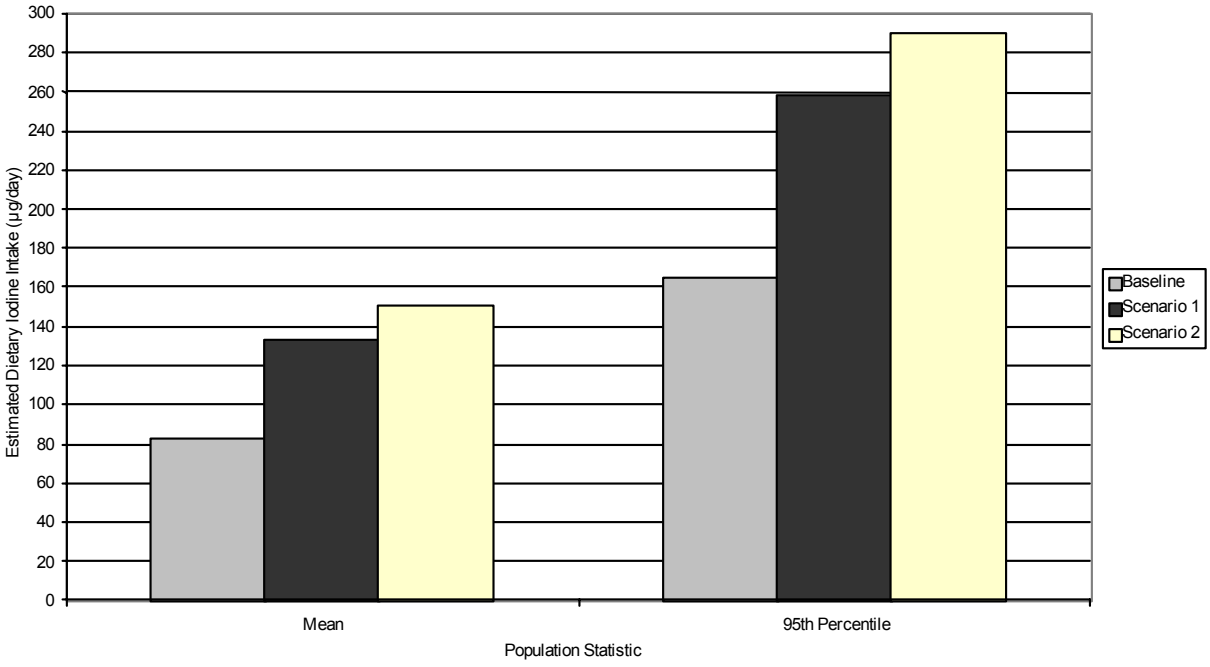
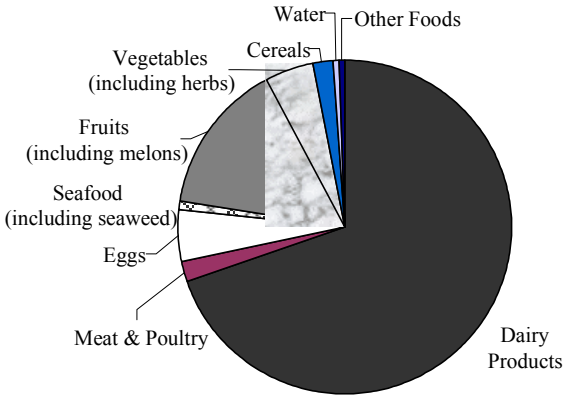
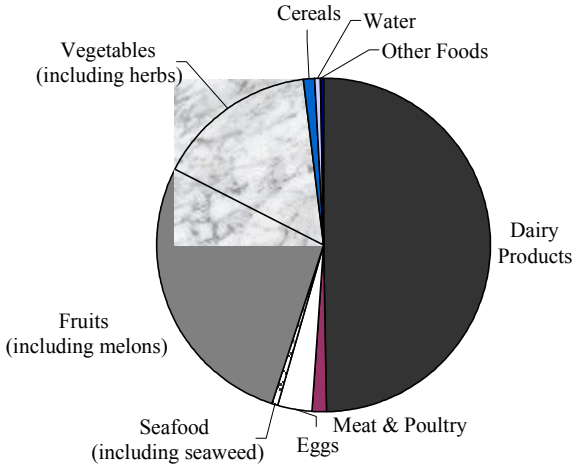


Figure 2: Contributors to total iodine intakes for the Australian population aged 2-3 years



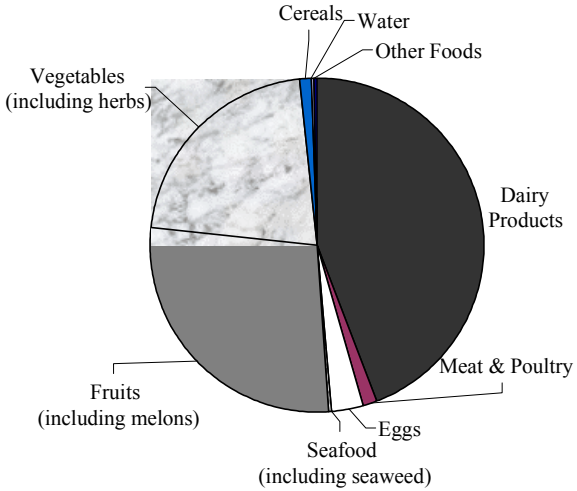
Baseline

(83 µg/day)



Scenario 1

(134 µg/day)



Scenario 2

(150 µg/day)

Risk Characterisation

Comparison of the estimated dietary intakes with the reference health standard

In order to determine if the level of dietary intake of iodine will be a public health and safety concern if an iodine wash is applied to fruits, vegetables (including herbs), eggs and nuts and if the maximum iodine limit in FSFYC is increased from 35 µg/serve to 70 µg/serve, the estimated dietary intakes were compared to a Provisional Tolerable Daily Intake (PTDI) of 17 µg/kg body weight/day. The PTDI was set by the FAO/WHO Joint Expert Committee on Food Additives (JECFA) (WHO 1989).

The estimated dietary intakes of iodine, as compared to the PTDI are shown in Figure 3 (full results in Table A2.1 in Appendix 2).

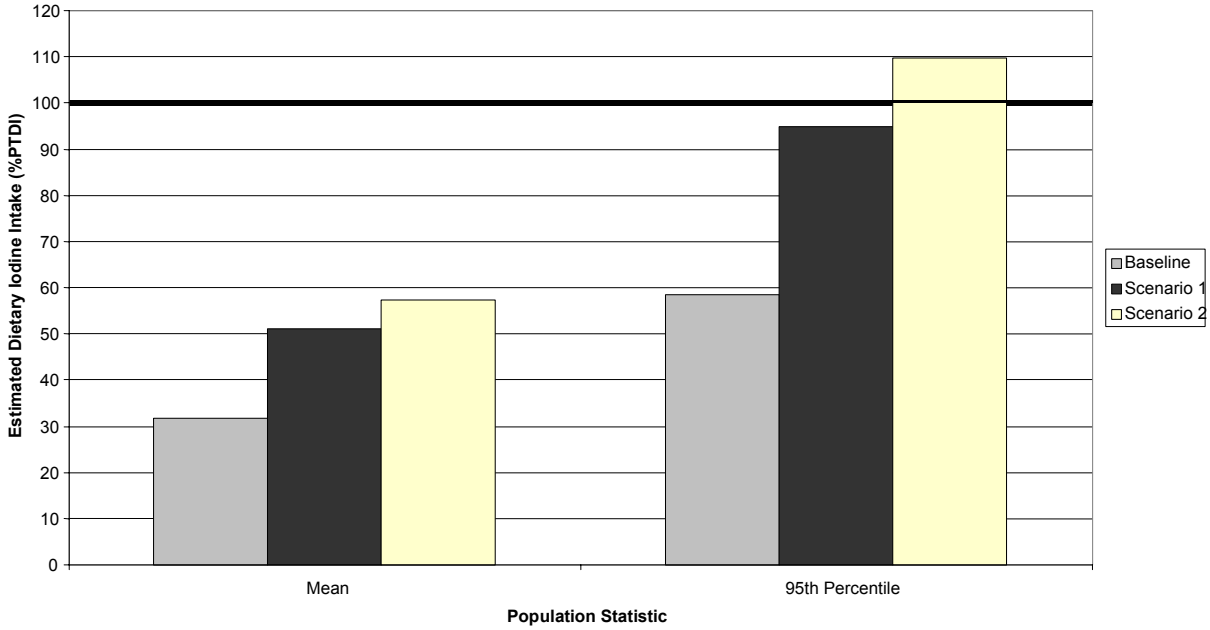
For 'both applications (A493 & A528)', estimated mean and 95th percentile dietary intakes of iodine were below the PTDI for the baseline scenario and for Scenario 1. For Scenario 2, the mean dietary intake of iodine was below the PTDI, with 95th percentile intake exceeding the PTDI (110% PTDI). Scenario 2 assumes that fruits and vegetables that may be consumed with the peel on or off were consumed unpeeled and that all FSFYC contain the proposed maximum iodine level of 70 µg/serve. However, due to the conservative assumptions made in this calculation and taking into account that the use of 24-hour dietary survey data tends to over-estimate habitual nutrient intakes for high consumers, it is likely that the 95th percentile dietary intake is an over-estimate of what this age group would be consuming on a daily basis over a lifetime.

For A493, estimated mean dietary intakes of iodine were below the PTDI for Australian children aged 2-3 years for all scenarios examined. Baseline and Scenario 1 estimated 95th percentile dietary intakes of iodine were also below the PTDI, with Scenario 2 estimated 95th percentile dietary iodine intakes exceeding the PTDI (101% PTDI).

For A528, mean and 95th percentile estimated dietary intakes of iodine were below the PTDI in all of the scenarios examined.

The estimated dietary intakes of iodine, as compared to the PTDI, for A493, A528, and 'both applications (A493 & A528)' are shown in Table A2.2 in Appendix 2. From these data, it would appear that the requested permissions from A493 have a greater impact on iodine dietary intakes for Australian children aged 2-3 years than do the requested permissions from A528.

Figure 3: Estimated dietary intakes of iodine, as a percentage of the PTDI



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APPENDIX 1: COMPLETE INFORMATION ON DIETARY INTAKE ASSESSMENT RESULTS FOR 'BOTH APPLICATIONS A493 & A528)'

Table A1.1: Estimated dietary intakes of iodine for Australian children aged 2-3 years

Number of consumers of iodine	Consumers♦ as a % of total respondents#	Mean consumers µg/day (µg/kg bw/day)			95th percentile consumers µg/day (µg/kg bw/day)		
		Baseline	Scenario 1	Scenario 2	Baseline	Scenario 1	Scenario 2
383	100	83.0 (5.4)	133.9 (8.7)	150.2 (9.8)	165.1 (9.9)	258.3 (16.1)	289.4 (18.7)

Total number of respondents for Australia: 2-3 years = 383. Respondents include all members of the survey population whether or not they consumed a food that contains iodine.
♦ Consumers only – This only includes the people who have consumed a food that contain iodine.

Table A1.2: Contribution of each food group to total iodine dietary intake for Australian children aged 2-3 years

Food Name	% Contribution to iodine dietary intake		
	Baseline	Scenario 1	Scenario 2
Dairy products	69.6	49.7	44.3
Fruits:	14.8	27.5	1.2
citrus fruits	11.7	7.3	6.5
pome fruits	1.7	15.9	16.5
Eggs	4.7	3.4	3.0
Vegetables (including herbs):	4.7	15.6	21.6
root & tuber vegetables	1.8	5.1	11.1
Meat & poultry	2.2	1.4	1.2
Cereals	1.8	1.1	1.0
Seafood (including seaweed)	1.0	0.6	0.5
Water	0.6	0.4	0.3
Other foods	0.6	0.4	0.4

APPENDIX 1: COMPLETE INFORMATION ON RISK CHARACTERISATION FOR 'BOTH APPLICATIONS (A493 & A528)'

Table A2.1: Estimated dietary intakes of iodine for Australian children aged 2-3 years, as a percentage of the PTDI

Number of consumers of iodine	Consumers♦ as a % of total respondents#	Mean consumers (%PTDI)			95th percentile consumers (%PTDI)		
		Baseline	Scenario 1	Scenario 2	Baseline	Scenario 1	Scenario 2
383	100	31.8	51.2	57.5	58.4	94.8	109.7

Total number of respondents for Australia: 2-3 years = 383. Respondents include all members of the survey population whether or not they consumed a food that contains iodine.
♦ Consumers only – This only includes the people who have consumed a food that contains iodine.
* PTDI = 17 µg/kg bw/day

Table A2.2: Estimated dietary intakes of iodine for Australian children aged 2-3 years for consumers, as a percentage of the PTDI for A493, A528, and 'both applications (A493 & A528)'

Application No. –Scenario	Mean dietary intake of iodine for consumers (% PTDI)	95th percentile dietary intake of iodine for consumers (% PTDI)
A493 – Baseline	29.4	54.3
A493 – Scenario 1	45.5	87.9
A493 – Scenario 2	51.7	101.2
A528 – Baseline	31.8	58.4
A528 – Scenario 1	35.1	64.5
'Both applications (A493 & A528)' –- Baseline	31.8	58.4
'Both applications (A493 & A528)' – Scenario 1	51.2	94.8
'Both applications (A493 & A528)' – Scenario 2	57.5	109.7

Total number of respondents for Australia: 2-3 years = 383. In this case, all respondents are consumers. Respondents include all members of the survey population whether or not they consumed a food that contains iodine. Consumers only include the people who have consumed a food that contains iodine.
* PTDI = 17 µg/kg bw/day

ATTACHMENT 6

NUTRITION RISK ASSESSMENT

The aim of this Nutrition Risk Assessment Report is to consider the current iodine status of the Australian and New Zealand populations, and to compare this with the results of dietary modelling (Attachment 4) in order to subsequently determine the nutritional risks, if any, to Australian and New Zealand populations from the proposed amendments to the Food Standards Code.

New policy guidelines on fortification have been established which recognise particular circumstances in which mandatory fortification to meet public health need is appropriate. Although this is not the focus of this Application, FSANZ plans to raise a separate proposal in the near future to investigate the need for increased iodine content in the Australia New Zealand food supply.

1. Current iodine status of the population

The International Council for the Control of Iodine Deficiency Disorders (ICCIDD) and the World Health Organization (WHO) have determined criteria for assessing population iodine status based on median urinary iodine concentrations. Many researchers have chosen to use these criteria in assessing their research population. Table 1 below describes the criteria for assessing iodine nutrition the population, and Table 2 illustrates the results of several studies conducted to examine the iodine status of study populations in Australia and New Zealand. Urinary iodine measures are more indicative of population iodine status than measures of dietary iodine intake.

Table 1: Epidemiological criteria for assessing iodine nutrition, based on median urinary iodine concentrations in school-aged children (ICCIDD)

Median urinary iodine (μg/L)	Iodine intake	Iodine nutrition
< 20	Insufficient	Severe iodine deficiency
20 – 49	Insufficient	Moderate iodine deficiency
50 – 99	Insufficient	Mild iodine deficiency
100 – 199	Adequate	Optimal

The ICCIDD suggest that, in adults, a urinary iodine concentration of 100 μg/L corresponds roughly to a daily iodine intake of about 150 μg under steady state conditions (ICCIDD 2001). A median of 100 μg/L or greater is recommended by WHO as being indicative of iodine sufficiency in a population.

Table 2: Results from studies[1] investigating iodine status of Australian and New Zealand populations.

Author	Subjects	n	% < 50 μg/L	% <100 μg/L	Median urinary iodine concentration[2]
AUSTRALIA					
Gunton (1999)	Pregnant women	81	19.8	49.6	
	Postpartum women	28	19.2	53.9	
	Patients with diabetes	135	34.1	71.9	
	Volunteers	19	26.3	73.7	
Guttikonda (2003)	Children 5 -13 years	301	14	69	82 μg/L
Li (2001)	Children 6 -13 years	94	13.8		84 μg/L
	Pregnant women from antenatal class	101	20.6		88 μg/L
	Adult volunteers, medical staff	86	18		88 μg/L
	Diabetes patients	85	23		69 μg/L
McDonnell (2003)	Children 11-18 years, Male	167	17	69	
	Female	410	31	79	
	Total	577	27	76	
NEW ZEALAND					
Thomson (1997)	Blood Donors	333	57	92	Male 51 μg/L Female 42 μg/L
Skeaff (2002)	Children 8 - 10 years	282	31.4	79.7	66 μg/L
Thomson (2001)	Men and women 18 - 49 years	233			59 μg/L ±33
New Zealand National Children's Survey	Children 5 -14 years		28		66 μg/L 68 μg/L males 62 μg/L females

[1]Further details of these studies can be found at Appendix 1.

[2]The WHO recommends that the median urinary iodine concentration for populations as a whole should be more than 100 μg/L, and that less than 20% of the populations should have a urinary iodine concentration below 50 μg/L as a measure of nutritional adequacy.

In the early 1990s it was reported that there was no evidence of iodine deficiency anywhere in Australia (Stanbury 1996). In more recent years however, a downward trend in iodine status has been noted in both Australian and New Zealand populations (Thomson 2002).

Studies shown in Table 2, indicate that iodine deficiency exists to various extents in both Australian and New Zealand population groups. In Australia, no national surveys have been undertaken to assess the iodine status of Australians, although national data collection in a National Iodine Nutrition study is currently in progress. New Zealand has regularly monitored national iodine status because of the low iodine content of its soils. Monitoring of iodine status also occurs in Tasmania where iodised salt is now used in the majority of Tasmanian bread manufacture, however the data are currently unpublished.

Both the WHO and the ICCIDD (ICCIDD 2001) suggest that no more than 20 percent of a population should have a urinary iodine level less than 50 μg/L, and that a median urinary iodine of 100μg/L or greater is indicative of iodine sufficiency. The general conclusion from the studies of urinary iodine levels in Table 2 is that a sizable proportion of Australians and New Zealanders suffer from iodine deficiency to varying extents.

2. Population intake of iodine compared to Estimated Average Requirements (EAR)

The EAR is defined as the level below which 50 percent of the population may be at risk of having inadequate dietary intake and is used to estimate the prevalence of inadequate intakes in a population. Dietary modelling has been conducted to determine the percentage of Australian and New Zealand populations not meeting the EAR for iodine intake (baseline intake data). The food consumption data used in the dietary iodine intake assessment were as measured in the 1995 National Nutrition Surveys (NNS) and reflects the food consumption patterns prevailing at that time. Table 3 illustrates these results.

Table 3: Estimated percentage of respondents for Australian and New Zealand population groups consuming less than the EAR for iodine.

Country	Population group	EAR[1] μg/day	Percentage of Respondents with Dietary Intakes of Iodine < EAR (%)
Australia	2-3 years	65	43
	4-8 years	65	41
	9-13 years	80	45
	14-18 years	100	52
	19 years and above	100	65
New Zealand	15-18 years	100	64
	19 years and above	100	65

[1]The Estimated Average Requirement (EAR) for iodine intake of Australians and New Zealanders as proposed by Thomson 2002.

The data in Table 3 are only suggestive of iodine intake due to the difficulties in measuring iodine in the food supply. Also, discretionary salt use was not measured in the National Nutrition Surveys. Depending on the level of discretionary iodised salt use, the extent of dietary inadequacy shown by the data in Table 3 might be overestimated. The levels of iodine in foods that were used to establish the level of estimated dietary intake of iodine were derived from a number of sources including Australian, New Zealand, British, and German food composition data, the 1997-8 and 2003-4 New Zealand Total Diet Surveys, the Australian Dairy Corporation and the Applicant. Iodine composition varies from country to country depending on soil levels and use of iodophors. The iodine content of plants and animals reflects the environment in which they grow.

Although both types of data are not directly comparable, the general inference can be drawn that a considerable proportion of Australians and New Zealanders are mildly iodine deficient.

3. Nutrient interactions

Some nutrients are known to compete with others for absorption and bioavailability, for example, dietary calcium and iron compete for absorption in the body when consumed at the same meal. There is no literature to suggest that iodine competes with, or inhibits the bioavailability of any other nutrient. This suggests that increasing the levels of dietary iodine intake will not have an adverse consequential effect on the nutritional status of consumers.

4. Conclusion

Research in both Australia and New Zealand indicates that the prevalence of iodine deficiency disorders is likely to be increasing in some populations in Australia and New Zealand. Data on the median urinary iodine levels in Australian and New Zealand populations suggests the baseline levels of iodine intake used in the dietary modelling may be slightly higher than in reality. As with the use of iodophors, the use of iodine as a processing aid may result in adventitious contamination of the food supply. It is very unlikely that the observed increase in iodine intake as a result of this Application will cause imbalances with other nutrients; to the contrary, it may have the beneficial outcome of helping to replete populations with poor iodine status. There are no identified adverse nutritional risks created by the proposed amendment to the *Food Standards Code.* The use of iodine as a processing aid, and its contribution to iodine intake, would need to be taken into account should any iodine fortification programs be contemplated in the future.

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Appendix 1

STUDIES OF IODINE STATUS IN AUSTRALIA AND NEW ZEALAND DISCUSSED IN MAIN BODY OF REPORT.

Gunton J, Hams G, Fiegert M, McElduff A. (1999). Iodine Deficiency in ambulatory patients at as Sydney Teaching Hospital; Is Australia Truly Iodine Replete? *Med J Aust.* 171: 467-470.

Study participants:

Study conducted at a tertiary referral hospital Sydney, Australia.

- 81 pregnant women attending obstetric clinic with 26 of the same women being retested at three months postpartum.
- 135 diabetes patients attending diabetes clinic for annual screening.
- 19 volunteers.

Methodology:
Spot urine samples were collected and urinary iodine measured by mass spectrometry. Iodine status based on urinary iodine concentration.

Results:
Table 1 – Results of iodine deficiency in Sydney participants. Gunton et al

	Pregnant women	Postpartum women	Patients with diabetes	Volunteers
Number of participants	81	26	135	19
Mean age	32.9 ± 9.8	35.3 ± 11.3	50.1 ± 35.3	49.5 ± 17.4
% participants with severe to mod deficiency	19.8	19.2	34.1	26.3
% participants with mild deficiency	29.6	34.6	37.8	47.4
% participants with normal iodine status	50.6	46.1	28.1	26.3

A weakness of this study is that subjects were recruited from hospital rather than the community. The small number of community volunteers did show a similar pattern of iodine status as seen in those subjects with diabetes. All patients were out patients and generally well.

Guttikonda K, Travers C, Lewis P, Boyages S. (2003). Iodine deficiency in urban primary school children: a cross-sectional analysis. *Med J Aust* 179: 346-348.

Study Participants:

- 324 children 5-13 years from a public school on the central coast of New South Wales.

Methods:
Thyroid ultra-sonography was used to determine thyroid volume.
First morning urine samples were collected.

Results:
Table 2 – Urinary iodine concentration in children. Guttikonda et al.

	% Urinary iodine concentration < 50 μg/L	% Urinary iodine concentration 50- 100 μg/L
n = 301	14	55

The median urinary iodide concentration was 81 μg/L for boys and 79 μg/L for girls with the over all median being 82 μg/L - indicative of mild iodine deficiency according to ICCIDD guidelines.

3% of the 144 girls had thyroid volumes above the WHO/ICCIDD median by age and 1% had thyroid volumes above the WHO/ICCIDD by body surface area. None of the boys had thyroid volumes above the WHO/ICCIDD medians. The results are indicative of long-term iodine deficiency in a small number of the population.

Li M, Ma G, Guttikonda K, Boyages S, Eastman C. (2001). Re-emergence of iodine deficiency in Australia. ***Asia Pacific J Clin Nutr.*** **10: 200-203.**

Study participants :

Study was undertaken in Sydney late 1998 and early 1999.

- 94 Healthy children aged 6-13 years randomly selected from a Western Suburb – upper middle income homes.
- 101 full term pregnant women attending antenatal classes at Westmead Hospital.
- 86 healthy Institute of Clinical Pathology and Medical Research Staff aged 21-60 years – subjects had not knowingly taken iodine medications or supplements in the previous 6 months.
- 85 people with diabetes.

Methods:
Urine samples were analysed for urinary iodine concentration.

Results:

Table 3 - Urinary iodine concentration in Sydney populations 1998/99. Li et al

	% urinary iodine concentration <50 μg/L	Median Urinary Iodine excretion
94 children 6- 13 years - Sydney	13.8	84 μg/L
101 Pregnant women from antenatal class, Sydney	20.6	88 μg/L
86 Adult volunteers, medical staff, Sydney	18	88 μg/L
85 Diabetes patients	23	69 μg/L

Approximately 60% of pregnant women in study displayed urinary iodine concentrations consistent with mild to moderate iodine deficiency.

McDonnell C, Harris M, Zacharin M. (2003). Iodine Deficiency and Goitre in School Children in Melbourne, 2001. *Med J Aust.* 178: 159-162.

Study participants:

- 607 children aged 11 –18 years from private schools, suggesting that they were not socio economically disadvantaged.

Methods:
577 children provided urine samples two hours after getting out of bed.
Urinary iodine was measured by Sandell-Koltoff reaction:

Results:

Table 4 – Urinary iodine excretion Melbourne school children. McDonnell et al.

	< 50 μg/L	50 – 99 μg/L	> 100 μg/L
Male (n=167)	17%	51%	32%
Female (n=410)	31%	48%	21%
Total (n=577)	27%	49%	24%

Median urinary iodine excretion for the total population was 70μg/L, indicative of mild iodine deficiency.

Thomson C, Colls A, Conaglen J, MacCormack M, Stiles M, Mann J. (1997). Iodine status of New Zealander residents as assessed by urinary iodide excretion and thyroid hormones. *British Journal of Nutrition* 78: 901-912.

Study participants:

Subjects were recruited between November 1993 and June 1994

- 189 subjects (102 males, 87 females) from Dunedin Blood Transfusion Centre
- 144 (67 males, 77 females) from the Waikato Blood Transfusion Centre

Methods:
Blood was collected and assayed for serum free T^3, free T^4 and TSH to determine circulating levels.

The following urine samples were also collected - fasting over night, and complete 24-hour specimen – urine collections were started two days after the blood donation to allow for dehydration of body fluids.

93 percent of subjects reported using iodised salt and 1.7 percent reported using non-iodised salt. 48 per cent reported never adding salt to food at the table; 23 percent always and 29 percent sometimes; 30 percent never used salt in cooking; 50 percent always and 19 percent sometimes. There was no difference in salt usage between the two geographical regions. Subjects also provided information regarding iodine supplement use.

Results:

Table 5 –Urinary iodide concentrations in Dunedin and Waikato subjects (median values with ranges in parentheses). Thompson et al

	Male		Female	
	All Subjects	Non-supplementers*	All Subjects	Non-supplementers*
n	169	156	164	155
24 hour iodide (μg/day)	73 (13-323)	70 (13-193)	62 (15-421)	59 (15-165)
Iodide concentration (μg/L) 24 hour urine	49 (12-281)	45 (12-152)	44 (6-350)	42 (6-123)
Iodide concentration (μg/L) Fasting morning urine	51 (9-240)	49 (9-200)	42 (8-384)	40 (8-130)

* Subjects who did not report taking regular supplements or medicines

Table 6 - Proportion of subjects at risk from iodine deficiency disorders (IDD) according to Urinary iodine concentration and 24h iodide excretion. Thompson et al

	Urinary Iodide concentration		24h Iodide excretion	
Risk of IDD	Criteria	% of study population	Criteria	% of study population
Severe	< 20 μg/L	7%	< 25 μg/day	5%
Moderate	20 – 49 μg/L	50%	25-49 μg/day	26%
Mild	50 – 100 μg/L	35%	50-100 μg/day	50%
None	> 100 μg/L	8%	>100 μg/day	19%

The authors concluded that although 24-hour urine samples are difficult to collect on a population level, they suggest that for research purposes that they are the most suitable and accurate measure of iodine status.

Ministry of Health 2003. ***NZ food NZ children: key results of the 2002 National Children's Nutrition Survey*****. Wellington: Ministry of Health.**

Subjects:

- 24-hour dietary recall from 3275 participants from urban and rural children around New Zealand. Urine and blood samples were collected from those participants who live in urban areas (number unknown). Children were aged between 5 and 14 years.

Results:
Median urinary iodine concentration of 66 μg/L (males 68 μg/L and females 62 μg/L).
Twenty eight percent of children had a urinary iodine concentration of less than 50μg/L, indicative of IDD.

Urinary iodine concentration did not differ across the three age groups (5-6 years, 7-10 years, 11-14 years). Females had a lower mean urinary iodine concentration than males.

Maori children had a lower mean urinary iodine concentration than New Zealand European and Pacific children.

Children from the lowest socio economic quartile had lower mean urinary iodine levels than those in the highest socio economic quartile.

Skeaff S, Thomson C, Gibson R. (2002). Mild Iodine Deficiency in a Sample of New Zealand School Children. ***Eur J Clin Nutr.*** **56: 1169-1175.**

Study participants:

- 282 children aged 8-10 years randomly selected from 30 schools in Dunedin and Wellington, New Zealand.

Methods:
Casual urine sample taken and frozen within 24 hrs before being analysed by single technician.

Thyroid volume determined by ultrasonography as an average of both thyroid lobes.

Results:

Table 7 - Urinary iodine levels and percentage of children below cut offs (WHO and ICCIDD) for severe, moderate and milk iodine deficiency. Skeaff et al

Age (y)	n	median (inter quartile range) μg/L	% <20 μg/L	percentage <50 μg/L	percentage < 100μg/L
Girls 8 years	34	67 (41-93)	3.1	42.8	76.5
Girls 9 years	57	67 (46-84)	1.9	26.2	85.2
Girls 10 years	48	61 (41-82)	6.4	35.8	87.2
Boys 8 years	54	56 (47-93)	5.4	34.0	77.4
Boys 9 years	57	71 (46-96)	3.7	27.6	78.8
Boys 10 years	32	75 (60-102)	0.0	24.0	68.1
TOTAL	282	66 (45-91)	3.6	31.4	79.7

The mean urinary iodine level for this population was 66 μg/L and clearly indicative of mild IDD. Thirty percent of the children in the study had iodine levels below 50 μg/L.

Thomson C, Woodruff S, Colls A, Joseph J, Doyle T. (2001). Urinary iodine and thyroid status of New Zealand residents. *Eur J Clin Nutr*. 55: 387-392.

Study participants:

- 350 Otago residents aged 18-49 years, initially selected randomly from electoral roll then later non-randomly from blood donors. 233 participants completed the research.

Methods:
350 participants collected complete 24 hour urine samples on two occasions.
233 then gave blood for assessment of thyroid hormone status and had their thyroid volumes measured by ultrasonography.

Results

Table 8 – Urinary iodine status and thyroid status of all subjects (n = 233) in Otago study 1997/1998. Thomson et al

	Mean ± s.d.	Median	CI (95th)
Age (y)	32±7	33	31-33
Weight (kg)	77±16	75	75-79
24h creatinine (g/day)	1.60±0.46	1.55	1.54-1.66
Iodide excretion 24 hour urinary iodide(μg/day)	86±49	75	80-93
Iodide/creatinine ratio (μg/g Cr)	57±35	47	53-62
Urinary iodide concentration (μg/L)	59±33	54	55-64
Thyroid status TSH (μIU/mL)	1.63±0.78	1.55	1.53-1.74
T4 (μg/dL)	7.3±1.8	7.2	7.0-7.6
Thyroglobin (ng/mL)	6.9±6.1	5.1	6.1-7.7
Thyroid volume (mL)	14.8±6.0	14.2	13.9-15.6

The authors comment that the median measures of urinary iodide excretion were lower than mean values due to very high excretions, 60 participants had median excretions higher than 100 μg/day. All of these participants reported consuming kelp, iodine containing supplements or iodine containing medicines. When subjects with excretion >140 μg/day were excluded, the mean and median values for urinary iodine excretion were 75±25 and 76 μg/day for males and 71±28 and 67 μg/day for females.

Significant inverse correlations were found for relationships between two measures of urinary iodide excretion (total 24h excretion and iodide/creatinine ratio) and thyroid volume and thyroglobin. Inverse correlations for urinary iodide concentration were significant for thyroglobin but not for thyroid volume.

Comments:
The overall aim of the study was to ascertain the correlation between the urinary iodide excretion and measures of thyroid status. For this reason there was no breakdown of numbers of participants into groups that may be disposed to various levels of IDD according to international cut offs.

ATTACHMENT 7

SUMMARY OF PUBLIC COMMENTS

1. Queensland Health

- Tentatively supports Option 2 – to amend the Code and approve the use of iodine as a food processing aid.

- Acknowledges the previous use of iodine for cleaning and sanitising in the dairy industry.

- The safety and efficacy of iodine as a washing agent for foods will need to be more fully considered.

Question why the permission should involve all foods.

2. Mr Keith Richardson, Food Science Australia

- Supports the progression of the Application.

- There is ample evidence that the washing agents already approved have limited effectiveness in the decontamination of fresh cut fruit and vegetables.

- If the material in the application is substantiated, iodine would appear to be a useful addition to those washing agents already approved.

3. Western Australia Department of Health

- Has considered various issues including the comparison of iodine with chlorine agents, food tainting, the use of iodine in various industries and the cessation of iodine sanitiser use in the dairy industry and the concerns of allergic reaction by sensitive individuals to iodine products.

- Notes that iodine intake in Australia is relatively low but not significant enough to warrant intervention, and that incidental intake of iodine through foods subject to water treatment agents may not present a public health and safety concern.

- Recognises that iodine is comparatively a more stable surface sanitiser than chlorine and has greater potential effects against a wide range of microorganisms.

- Notes that iodine residues are more likely to remain on food surfaces and reach the final consumer, which raises the issue of the appropriateness of classifying iodine as a Processing Aid.

- Seeks further clarification on the use and methods of Application and the proposed control measure for iodine.

- Prefers Option 1 – maintain the status quo and not approve the use of iodine as a food processing aid.

- Will reconsider the Application as additional information becomes available.

4. Professor Joe Montecalvo, California Polytechnic State University

- Supports the Application.

- Has been conducting a detailed review of direct contact sanitisers for use within the fruit and vegetable segment of the industry, especially the whole fruit and fresh cut convenience products and has also studied the Applicant's Iodoclean™ System.

- Has found the Iodoclean™ System to not only be effective in the reduction of the risk of microbial food infections and food borne disease but also to be the most significant advancement in sanitation technology in the past twenty years. His assessment is based on rapid microbial kill rates with longer shelf life possibilities. The system also offers very significant advantages especially in environmental, equipment maintenances and occupational health and safety.

5. Dr Stephen Morris, Sydney Postharvest Laboratory

- There is a shortage of effective sanitisers that can be used directly on foodstuffs and which do not have major problems with undesirable breakdown products, waste disposal and maintaining accurate sanitiser levels. The Iodoclean™ System is a useful addition to the available sanitisers.

- In a range of tests undertaken at the laboratory, iodine was found to be more effective than chlorine against a considerable number of bacteria and fungi. It was also more effective than chlorine when the dip became contaminated with dirt or small particles of organic material.

- The treatment of food does result in an increase of iodide in the food. For fruits and vegetables, the increase can be as little as 10%, however over a range of fruits and vegetables and a range of concentrations the increase was found to be about 100%. Fruits and vegetables only account for about 5% of dietary iodine, and the typical Australian diet has been reported as significantly deficient in iodine.

6. Dr Kerry McDonalds, Department of Crop Sciences, University of Sydney

- It has long been demanded that an alternative to chlorine washes be found.

- Chlorine is used widely as a washing agent for fresh produce but is not very safe for consumers and is also corrosive for the processing plants.

- Approval for the use of iodine as a washing agent would provide an additional option for the food processing industry.

- The levels of iodine used would not be harmful for human consumption and will add dietary iodine to the food supply.

7. Australian Quarantine and Inspection Service (AQIS)

- AQIS will assess the regulatory impact on AQIS operations of any proposed amendment to the Code after the draft assessment stage has been completed.

8. New Zealand Food Safety Authority

- Cannot comment on the safety aspects or the nutritional implications as the Application is still at Initial Assessment.

- Trusts that when preparing the Draft Assessment, FSANZ will consider the following:

  - the bioavailability of iodine from this source;
  - the function of iodine when used as a sanitising agent to determine if it is solely acting as a processing aid, or is there also a food additive function;
  - whether iodine is approved for this use in any other jurisdiction.

9. Ms Jenny Jobling

- Saw the Iodoclean™ System on television and was impressed with the invention and considers it to be an excellent new technology.

- Believes iodine would be a useful addition to the food sanitisers available to the Australian food industry.

10. Australian Food and Grocery Council

- Supports the Application, subject to an appropriate safety assessment by FSANZ.

- The use of iodine in the washing of fruits and vegetables could be useful in reducing the bacterial load, and studies submitted by the applicant in support of this.

- Some years ago, iodine was used as a sanitising agent in the dairy industry and, when high residual levels were found in dairy products, a maximum level of 500 µg/L was imposed for milk and liquid milk products. This restriction, however, has not been carried over into the new Code, as iodophors are no longer used in the dairy industry.

- Concerns have been expressed that iodine consumption in Australia may be below optimum levels and even some suggestion that mandatory fortification of certain foods with iodine should be considered. This would seem to be a point in favour of approving this application.

- The Applicant has stated increased iodine intake would be relatively low as fruit and vegetables contribute only about 5% of the iodine in an average diet. This clearly ignores other potential sources of increased iodine intake if the application were to be approved for foods generally. The application therefore requires a thorough assessment.

- Should FSANZ decide that approving the use of iodine as a processing aid for foods generally with a residual subject only to GMP, poses a potential risk to public health, FSANZ has three options other than rejecting the application outright. These are: approve for foods generally but with a prescribed maximum residue, approve for fruits and vegetables with a residue subject to GMP; and approve for fruits and vegetables with a prescribed maximum level.

11. Food Technology Association of Victoria Inc

- Agree with Option 2 – to amend the Code and approve the use of iodine as a food processing aid in the applications nominated by the applicant.

12. Dietitians Association of Australia

- Although there is some evidence that dietary intakes of iodine may be below the recommended levels for prevention of iodine deficiency disorders in selected populations in Sydney, Melbourne and Tasmania, there is no evidence to support an increased incidence of IDD in conjunction with the lower urinary iodine excretion values.

- A national survey to more fully document iodine nutritional status in Australia as well as the prevalence of IDD would provide data to develop an appropriate public health policy if necessary.

- The DAA understands that such a survey is currently underway but the results will not be available until mid 2004.

- The DAA believes that the reintroduction of iodine as a sanitising agent may be a suitable replacement for chlorine-based sanitising agents, but should not be seen as a way of correcting nutritional inadequacies.

- The DAA recommends strongly that modelling be done to estimate the maximum exposure to iodine, if it were used in a wide range of foods, to ensure that maximum dietary intakes would not exceed 1000 μg/day.