FOODSTANDARDS
Australia New Zealand
Te Mana Kounga Kai – Ahitereiria me Aotearoa

Supporting document 2

Risk and technical assessment report (at Approval) – Application A1100

Maximum Permitted Level of Acesulphame Potassium in Chewing Gum

Executive summary

Acesulphame potassium (Ace K) is currently permitted in the Code as an intense sweetener that can be added to chewing gum at a maximum permitted level (MPL) of 2000 mg/kg. This Application requests an increase in the MPL to 5000 mg/kg, which is the MPL for Ace K in chewing gum in the Codex General Standard for Food Additives.

FSANZ considers the Acceptable Daily Intake (ADI) of 0–15 mg Ace K per kg bodyweight established by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) to be a suitable health-based guidance value to compare with estimated dietary exposure. No subsequent studies have been located which could be considered to affect this ADI.

A dietary exposure assessment has been undertaken using food consumption data for chewing gum from the available Australian and New Zealand national nutrition surveys, and data on Australia/New Zealand consumption of other foods containing intense sweeteners. Using conservative assumptions to account for the additional dietary exposure resulting from the increase in the MPL for chewing gum, estimated dietary exposures for high (90th percentile) consumers were 6–20% of the ADI for the population groups assessed.

It is concluded that the proposed increase in the MPL for Ace K in chewing gum from 2000 mg/kg to 5000 mg/kg would result in estimated dietary exposure to Ace K well below the ADI of 15 mg/kg bw. Therefore, there are no public health and safety concerns associated with the proposed increase in the MPL for Ace K in chewing gum.

All references to *the Australia New Zealand Food Standard* Code (the Code) in this report are to the revised Code which takes effect and replaces the current Code on 1 March 2016. This is because the gazettal of any draft variation is not expected until close to this date (if approved by the FSANZ Board and no review of that decision is requested by Ministers). FSANZ therefore considers it is unnecessary to amend the current Code.

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1   Introduction

FSANZ received an Application from Brooke-Taylor & Co Pty Ltd, on behalf of The Wrigley Company Pty Ltd, seeking approval for an increase in the maximum permitted level (MPL) for acesulphame potassium (Ace K) in chewing gum. The current MPL in the *Australia New Zealand Food Standards Code* (the Code) is 2000 mg/kg and the proposed MPL is 5000 mg/kg which is the MPL for Ace K in chewing gum in the Codex General Standard for Food Additives.[1] The purpose of the requested increase in MPL is to allow the sale of chewing gum products in Australia and New Zealand which have flavour (sweetness) profiles similar to chewing gums available in overseas markets.

Ace K is a food additive with technological purposes as an intense sweetener and as a flavour enhancer. It has been a permitted food additive in the Code for many years in a range of food categories as detailed in the table to section S15—5 (Food Additives). One of these permissions is for item 5 – Confectionery, which includes chewing gum, with an MPL of 2000 mg/kg.

1.1   Objectives of the assessment

The objectives of this risk and technical assessment are to:

- determine whether the proposed increase in the maximum permitted level is justified and that Ace K achieves its technological function in the quantity proposed to be used as a food additive in chewing gum;

- evaluate any potential public health and safety concerns that may arise from an increase in the maximum permitted level of Ace K in chewing gum.

2   Food technology assessment

2.1   Characterisation of acesulphame potassium

2.1.1   Identity of the food additive

Common name:	Acesulphame potassium (Ace K)
Other names:	Acesulfame potassium (Codex spelling); Acesulfame K; potassium salt of 6-methyl-1,2,3-oxathiazine-4(3H)-one-2,2-dioxide; potassium salt of 3,4-dihydro-6-methyl-1,2,3-oxathiazine-4-one-2,2-dioxide
INS Number:	950
Chemical Abstract (CAS) Number:	55589-62-3
Chemical formula:	$C_4H_4KNO_4S$
Molecular weight:	201.24 g/mol

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[1] http://www.codexalimentarius.net/gsfaonline/additives/details.html?id=104

Structural formula:
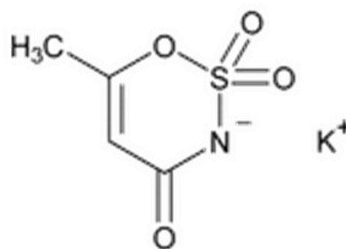
2.1.2 Physical and chemical properties

Ace K is an odourless free-flowing white crystalline powder. It is freely soluble in water and only slightly soluble in ethanol. Ace K does not have a definitive melting point but it decomposes well above 200°C. Decomposition appears to be dependent on the heating rate. It does not decompose during normal food processing conditions. Aqueous solutions of Ace K are approximately neutral in pH. Solid Ace K has a very long shelf life at room temperature and it does not need to be protected from sunlight. The specific gravity of the powder is 1.81 g/cm^3.

2.2 Production of acesulphame potassium

Ace K is produced by chemical synthesis, not by extraction or reactions of various sugars. There are a variety of different production routes using different raw materials (von Rymon Lipinski and Hanger 2001).

2.3 Specifications

Both the Combined Compendium of Food Additive specifications of the Joint FAO/WHO Expert Committee on Food Additives (JECFA) and the Food Chemicals Codex have specifications for Ace K. Both these references are primary sources of specification in section S3–-2 in the revised Code).

2.4 Analytical methods

Ace K can be identified and quantified in foods and beverages using reverse-phase High Performance Liquid Chromatography (HPLC) using UV detection at 227 nm. Products containing protein or carbohydrate should be clarified by adding zinc sulphate and potassium hexacyanoferrate prior to analysis. Analytical methods have been developed that can separate and quantify other intense sweeteners used in combination with Ace K.

2.5 Technological function

Ace K has the sweetness intensity of about 200 times that of sucrose (3% w/v solution). The relative sweetness intensity reduces in comparison to sucrose as the sucrose concentration increases. Ace K is about as sweet as aspartame, half as sweet as saccharin and four to five times as sweet as cyclamate (von Rymon Lipinski and Hanger 2001).

Ace K is noted to have early onset sweetness compared to other intense sweeteners. That is why it is often used in sweetener blends, such as for the current Application in chewing gum where it is used in combination with aspartame which provides the later sweetness onset. The sweet taste does not linger beyond that of the intrinsic taste of the food. There is also a strong synergistic taste enhancement noted when Ace K is blended with aspartame (and separately also with sucralose and neotame). Also the lingering sweetness of aspartame and sucralose is substantially reduced when blended with Ace K.

Sweetener blends therefore help the final product taste more similar to sucrose, which is important for sugar-free products.

Ace K also has synergistic effects when used with sugar alcohols (e.g. sorbitol, xylitol) where the sugar alcohols provide the bulk and texture but have a lower sweetness intensity compared to sucrose. Such blends can provide a good sweetness and taste profile. The low sweetness intensity of the sugar alcohol is enhanced by the addition of Ace K, which also provides earlier sweetness onset. The blend ratio for using Ace K with sugar alcohols is at least 1:100 where the sugar alcohols are acting as bulk sweeteners. Ace K also has flavour enhancing function when used with flavourings added to food.

2.5.1   Use in chewing gum

Intense sweeteners and sugar alcohols (bulk sweeteners) added to chewing gum need to be water soluble so the sweetness is released by dissolving in the saliva in the mouth. Since Ace K is readily water soluble it provides early onset of sweetness to provide the initial sweet taste. However, the Ace K sweetness does not linger for long so it is augmented by using other intense sweeteners in the blend such as aspartame. These intense sweetener blends enhance the low sweetness of the sugar alcohols often used in sugarless chewing gum. Ace K also has flavour enhancing properties when used with flavoured chewing gum, especially mint flavoured chewing gum or fruit flavoured bubble gum.

To extend the sweetness of chewing gum the intense sweeteners can be encapsulated so they provide a slower, more progressive release of sweetness as the chewing gum is chewed. This also limits the initial high sweetness spike.

The fine crystals of Ace K can be blended directly into the chewing gum mass. If sorbitol syrup is used as the sugar alcohol base then Ace K can be dissolved initially into the syrup.

Ace K is quite stable in chewing gum. There have been no reactions observed between Ace K and flavours added to chewing gum. As noted earlier Ace K has synergistic flavour effects when used with other intense sweeteners such as aspartame, as well as with sugar alcohols, and it does not have any negative reactions with these other sweeteners.

Ace K can also have positive flavour enhancing effects when added in relatively small amounts (a few hundred mg/kg) to flavoured sugar-based chewing gum to prolong the flavour perception.

2.6   Food technology conclusion

Ace K is currently permitted as an intense sweetener that can be added to chewing gum. There are specifications for Ace K in the Code and analytical methods are available to detect and quantify its presence in food. Ace K is often used in sweetener blends used in chewing gum to provide early sweetness onset, as well as having a synergistic effect with other intense sweeteners and sugar alcohols used in chewing gum to produce a sweetness profile more like sucrose. An increase in the MPL for Ace K in chewing gum will allow the sale of chewing gum in Australia and New Zealand with desired flavour (sweetness) profiles.

3 Hazard assessment

3.1 Background

3.1.1 Assessments by JECFA

The toxicological database for Ace K has been evaluated by JECFA on three occasions: at the 25th, 27th and 37th meetings (WHO 1981a; WHO 1983a; WHO 1991). At its 25th meeting, JECFA concluded that there were insufficient data to establish an ADI. Additional data were available for the 27th meeting and JECFA concluded that there was no evidence that Ace K was mutagenic or carcinogenic. In long-term (2-year) feeding studies in rats and dogs, the no observed effect level (NOEL) for Ace K was 1500 mg/kg bw/day in rats and 900 mg/kg bw/day in dogs (highest tested dose in each study). An ADI of 0–9 mg/kg bw was established based on the NOEL observed in the dog study and application of a safety factor of 100 (WHO 1983a, 1983b). At its 37th meeting, JECFA reviewed additional data including pharmacokinetic data in rats and humans. From these data JECFA concluded that the rat was an appropriate surrogate model for humans and withdrew the existing ADI. The Committee then established an ADI of 0–15 mg/kg bw based on the above rat NOEL of 1500 mg/kg bw/day and a safety factor of 100 (WHO 1991).

3.2 Evaluation of submitted data

The Applicant identified two relevant studies published since the last JECFA evaluation of Ace K. The studies, which were conducted in two genetically altered mouse strains by the US National Toxicology Program, were published in a single report (NTP 2005).

Tg.AC hemizygous and p53 haploinsufficient mice were exposed to Ace K (purity at least 99%) in feed for 9 months. The two mouse strains have been genetically altered with either a loss of heterozygosity in a critical cancer gene (Trp53) or a gain of oncogene function (Ha-ras) to provide mouse models that are susceptible to the rapid development of cancer.

Groups of Tg.AC hemizygous mice (n=15/sex/group) were fed diets containing 0%, 0.3%, 1%, or 3% Ace K (equivalent to average daily doses of approximately 420, 1400, or 4500 mg/kg bw for males and 520, 1700, or 5400 mg/kg bw for females) for 40 weeks. Dietary exposure to Ace K had no effect on survival or mean body weights. Feed consumption by the exposed groups was similar to that by the control groups throughout the study. There were no neoplasms or non-neoplastic lesions that were attributed to exposure to Ace K. There were no statistically significant changes in the frequency of micronucleated erythrocytes in any of the treated groups compared to controls.

Groups of p53 haploinsufficient mice (n=15/sex/group) were fed diets containing 0%, 0.3%, 1%, or 3% Ace K (equivalent to average daily doses of approximately 475, 1500, or 4700 mg/kg bw for males and 570, 1800, or 5700 mg/kg bw for females) for 40 weeks. Dietary exposure to Ace K had no effect on survival or mean body weights. Feed consumption by the exposed groups was similar to that by the control groups throughout the study. There were no neoplasms or non-neoplastic lesions that were attributed to exposure to Ace K. There was a statistically significant increase in the frequency of micronucleated erythrocytes at the end of the exposure period in males that received 1% or 3% Ace K ($p = 0.004$ and 0.001, respectively); however the magnitude of the increase was only approximately 1 micronuclei/1000 cells. Moreover, no increase was observed in females. This observation is therefore not considered to be toxicologically relevant. Furthermore, a micronucleus assay in mice orally administered Ace K, previously evaluated by JECFA, was negative (WHO 1981b).

3.3　Additional published data

FSANZ did not locate any additional studies considered to be relevant for the hazard assessment of Ace K.

3.4　Hazard assessment conclusion

The results of studies in mice susceptible to the rapid development of cancer are consistent with previous animal studies showing that Ace K is well tolerated at high doses and does not show potential for carcinogenicity. No other studies were identified which could potentially affect the ADI of 0–15 mg/kg bw established by JECFA.

4　Dietary exposure assessment

4.1　Approach to estimating dietary exposure to Ace K

Dietary exposure assessments require data on the concentrations of the chemical of interest in the foods requested and consumption data for the foods that have been collected through a national nutrition survey.

The dietary exposure for Ace K for this Application was estimated using:

- the existing MPL for Ace K in intensely sweetened chewing gums and the increased MPL requested in this Application
- food consumption data from the available Australian and New Zealand national nutrition surveys
- the previously conducted survey of intense sweeteners by FSANZ (FSANZ 2004).

4.1.1　Previous estimates of dietary exposure to Ace K

Estimated dietary exposure to Ace K for both the Australian and New Zealand populations aged 12 years and above has been previously assessed as part of sweetener surveys in both 1995 and 2004 [National Food Authority (1995) and FSANZ (2004), respectively]. The sweetener surveys consisted of a 7-day food diary of intensely sweetened foods by brand and flavour from a population of high consumers of intensely sweetened foods, as identified by a screener survey. Food product groups containing intense sweeteners included in the surveys were carbonated soft drink, cordials, fruit drinks, table-top sweeteners, confectioneries (including chewing gum, lollies, chocolate etc), flavoured yoghurts and mousses, jellies and milk based puddings, jams or conserves, flavoured milks, canned fruits, toppings and ice creams. Detailed data on the types and concentrations of intense sweeteners used in intensely sweetened food products were provided by industry to allow a refined dietary exposure assessment to be undertaken, which is considered to be the best baseline estimate available for Ace K.

The 2004 sweetener survey concluded that the mean and 90th percentile dietary exposure to Ace K for Australian and New Zealand populations aged 12 years and above, from the range of all possible foods containing the sweetener to be 3% and 6% of the ADI (0–15 mg/kg bw), respectively. (See Table A2.1 in Appendix 2) for Australia and New Zealand). The 2004 sweetener survey indicated a small increase in estimated dietary exposure to Ace K in comparison to the 1995 sweetener survey which concluded that estimated dietary exposure to Ace K was 1% of the ADI at the mean and 3% of the ADI at the 90th percentile. Further details of the 2004 sweetener survey report are available at http://www.foodstandards.gov.au/publications/Pages/evaluationreportseries/intensesweetenerssurveymarch2004/Default.aspx.

Following these assessments, there has been no change to the Ace K permissions in the Code, while permissions for new sweeteners have been included (e.g. neotame, steviol glycosides, advantame). The food industry use of Ace K in their products might have changed since the time of the 2004 sweetener survey due to reformulation of existing products and formulation of new products. The availability of other intense sweeteners provides the opportunity to use different sweetener combinations in different food products, which may affect the use of individual sweeteners.

4.1.2 Assessment of dietary exposure to Ace K in chewing gum

As the overall mean and the 90th percentile estimated exposures to Ace K for populations aged 12 years and over in Australia and New Zealand from the 2004 sweetener survey were low, and the current proposed change is to increase the MPL for intensely sweetened chewing gums only (a food that is consumed in small quantities by a small proportion of the general population), the chosen dietary modelling approach for Ace K was simplified. This included determining:

- the consumption amount for intensely sweetened chewing gum based on the latest nutrition surveys for Australia and New Zealand
- The impact on estimated baseline dietary exposure from the proposed increase in MPL for the intensely sweetened chewing gum as a percent of the ADI. I.e. a *Baseline* model reflecting the current MPL for intensely sweetened chewing gum and a *Scenario* model including the proposed MPL as specified in Table 4.1.

This estimate of dietary exposure from chewing gum only was combined with the exposure from the whole diet from the 2004 sweetener survey for baseline and scenario.

Table 4.1: Current and proposed MPLs for Ace K in intensely sweetened chewing gum, and the levels used for the dietary exposure assessment

Food Category Code	Food Category Name	MPL (mg/kg)		Concentration used in dietary exposure estimation (mg/kg)	
		Current	*Proposed*	*Baseline*	*Scenario*
5.2.1.1	Low joule chewing gum	2000	5000	2000	5000

A summary of the general FSANZ approach to conducting the dietary exposure assessment for this Application is at Appendix 1. A detailed discussion of the FSANZ methodology and approach to conducting dietary exposure assessments is set out in *Principles and Practices of Dietary Exposure Assessment for Food Regulatory Purposes* (FSANZ 2009).

4.1.3 Consumption data used

The food consumption data for chewing gum used to estimate dietary exposures to Ace K from this product for the Australian and New Zealand populations were:

- 2011–12 Australian National Nutrition and Physical Activity Survey (2011 NNPAS)
- 2008–09 New Zealand Adult Nutrition Survey (2008 NZANS)
- 2002 New Zealand National Children's Nutrition Survey (2002 NZNNS).

The design of each of these surveys varies somewhat and key attributes of each, including survey limitations, are set out in Appendix 1 – Dietary Exposure Assessments at FSANZ.

The hazard identification and characterisation (Section 3) did not identify any population sub-groups for which there were specific safety considerations in relation to exposure to Ace K. A dietary exposure assessment for Ace K from chewing gum was conducted for children aged 2–6, 7–11 years, for the general population aged 2 years and over and for the population aged 12 years and over for Australia; and for children aged 5–14 years and the population aged 15 years and over for New Zealand.

Based on the latest Australia and New Zealand nutrition surveys, the proportion of the population consuming intensely sweetened chewing gum were 0–1%. Mean consumer consumption for these products was 2.5–7.4 g/day, equivalent to 1–4 pieces or tabs of chewing gum assuming 1 serve is ~2 g (for more details on the consumption data for each country and population group see Table A2.2 in Appendix 2.

4.1.4   Concentration data for Ace K

Ace K is permitted in a wide range of foods and beverages at specified MPLs, except for table top sweeteners which are permitted at a level determined by Good Manufacturing Practice (GMP). Ace K is often used in combination with other sweeteners to achieve the required sweetener profile for the chosen product, if used in combination with other sweeteners and complying with the section 1.3.1—6, Ace K may not be used at the MPL in the Code. Therefore, undertaking a calculation to determine a realistic estimate of dietary exposure to Ace K, including all Ace K permissions in Schedule 15, requires current industry use level data and market share data. No new information on the use of Ace-K in other foods was provided in the Application. The Ace-K dietary exposure estimates from the 2004 sweetener survey (section 4.2.2) were used as the baseline estimated exposure for this assessment because industry use levels for intense sweeteners formed the basis of reported dietary exposure estimates, making it the best available estimate for this purpose.

4.1.5   Assumptions and limitations of the dietary exposure assessment

The aim of the dietary exposure assessment was to make as realistic an estimation of dietary exposure to Ace K as possible. However, where significant uncertainties in the data existed, conservative assumptions were generally used to ensure that the estimated dietary exposure was not an underestimate of exposure, for example, all intensely sweetened chewing gum was assumed to contain Ace K at the proposed maximum level, while in reality Ace K may be used at lower levels in combination with other sweeteners.

In addition to the specific assumptions made in relation to this dietary exposure assessment, there are a number of limitations associated with the nutrition surveys from which the food consumption data used for the assessment are based. A discussion of these limitations is included in Section 6 of the *Principles and Practices of Dietary Exposure Assessment for Food Regulatory Purposes* (FSANZ 2009).

4.2   Estimated population dietary exposures to Ace K

The estimated dietary exposures to Ace K were calculated for 'consumers' only[2]. Mean and 90th percentile dietary exposures to Ace K, as a percentage of the ADI, were derived from each individual's ranked daily exposures.

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[2] A consumer is a respondent from the national nutrition survey who reported consuming a food(s) that has permissions for adding Ace K.

4.2.1 Estimated dietary exposures for consumers of intensely sweetened chewing gum

The estimated dietary exposures to Ace K from intensely sweetened chewing gum only, expressed as a percentage of the ADI, for all the population groups investigated were well below the ADI for both the *Baseline* and increased MPL *Scenario*.

Estimated mean dietary exposures for Ace K were at or below 2% of the ADI for the *Baseline* and 4% of the ADI for the *Scenario* for all population groups assessed. The 90th percentile dietary exposures were 1–6% of the ADI at *Baseline* and 3–14% of the ADI for the *Scenario* for the population groups assessed.

Based on the 2011 NNPAS, Australian children aged 2–6 years were not consumers of either sugar sweetened chewing gum or intensely sweetened chewing gum and thus no exposure was reported for this group. Estimated exposures for 12 years and above were the same as for the population aged 2 years and above as there were only three consumers for intense sweetened chewing gum below 12 years of age (see Figure 4.1 and Table A2.3 in Appendix 2 for Australia and New Zealand). Estimated exposures for New Zealand children were higher than those for adults when expressed as a percentage of the ADI, likely due to the higher ratio of food consumption to body weight for children compared to adults.
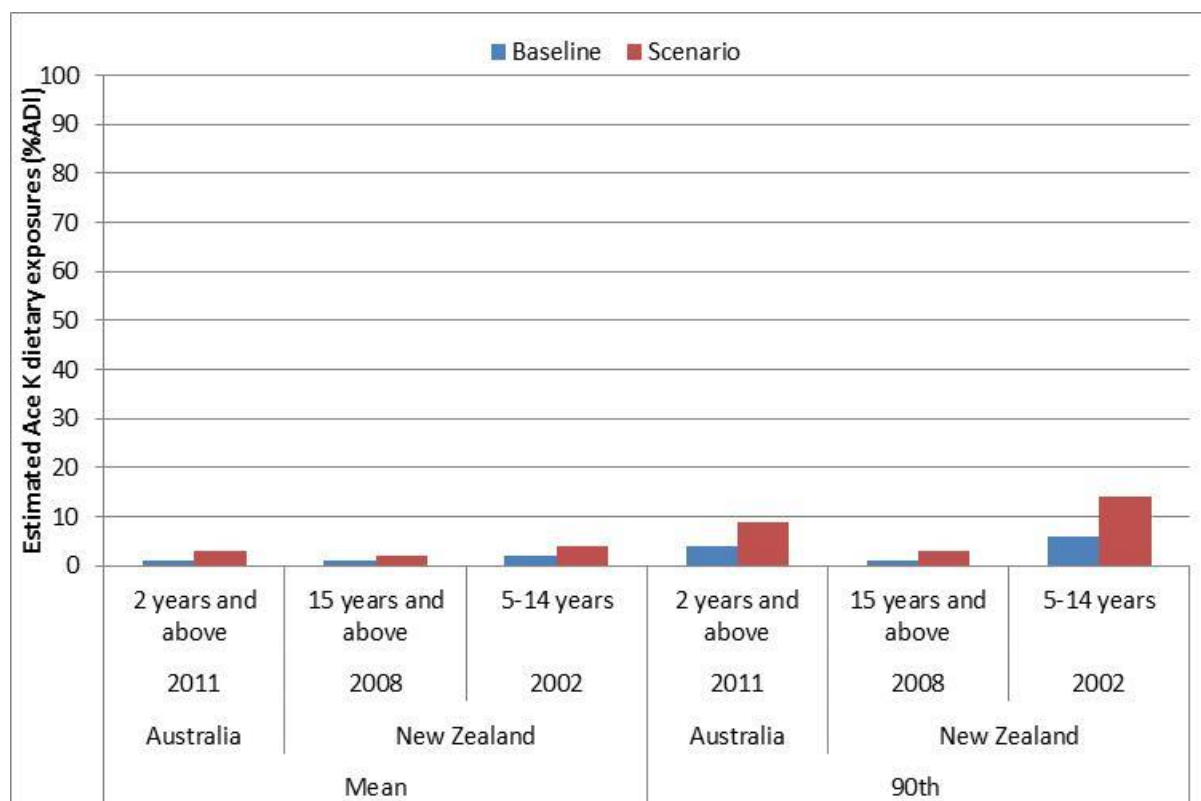
Figure 4.1: Estimated mean and 90th percentile dietary exposures to Ace K from intensely sweetened chewing gum as a percentage of the ADI for all Australian and New Zealand population groups assessed[3]

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[3] Australian children aged 2-6 years were not consumers of both sugar sweetened chewing gum and intensely sweetened chewing gum. Also estimated exposures for 12 years and above were same as the 2 years above as there were only three consumers for intense sweetened chewing gum between 7-11 years in Australia.

4.2.2 Estimated population dietary exposures from intensely sweetened chewing gum combined with the 2004 sweetener survey results

Combined estimated dietary exposures from the 2004 sweetener survey (baseline) and the Ace K exposures from the intensely sweetened chewing gums were estimated for populations aged 12 years and over for Australian and 15 years and over for New Zealand and expressed as a percentage of the ADI.

There are some limitations with this approach given they were estimates from different time periods and methodologies. However, it does provide an indication of the potential impact of the current request for extended Ace K permissions on public health and safety in broad terms. In addition, it should be noted that, using this approach, exposure to Ace K from intensely sweetened chewing gum would be counted twice, as it was also included in the 2004 sweetener survey, hence it is likely an over-estimate.

With this combined estimate, the mean and 90th percentile dietary exposures for consumers were well below the ADI for the 12 years and over population group for Australia and 15 years and over group for New Zealand when the increased MPL *Scenario* was considered. Combined estimated mean dietary exposures for Ace K were 5-7% ADI and the 90th percentile dietary exposures were 9–15% of the ADI for the population groups assessed. See Figure 4.2 and Table A2.4 in Appendix 2 for Australia and New Zealand).

Figure 4.2: Estimated mean and 90th percentile population dietary exposures to Ace K as a percentage of the ADI for all population groups assessed

4.2.3 Estimated total dietary exposures to Ace K for young children

As noted above, the 2004 sweetener survey considered the Australian and New Zealand populations aged 12 years and above.

Although estimated dietary exposure to Ace K from intensely sweetened chewing gum was estimated for Australian children aged 2–11 years and New Zealand children aged 5–14 years, a total dietary exposure estimate was not calculated at this point in time because there were no baseline data.

Further analysis of the 2011 NNPAS indicates the proportion of children consuming a range of intense sweetened food groups (i.e. carbonated soft drinks, cordials, flavoured yoghurts, confectioneries, desserts) was similar to or lower than that for the general population. For example, within the food groups investigated, the largest difference was for intensely sweetened cola soft drinks which had the highest proportion of consumers (8% for the general population aged 2 years and above but less than 1% for Australian children aged 2–6 years (ABS 2014b). Therefore, the baseline mean dietary exposure for consumers of Ace K is likely to be similar to or lower for the 2–11 year old age group than for the population aged 12 years and over.

However, as children 2–6 years in Australia did not report consuming intense sweetened chewing gum in the 2011 NNPAS, there would be no expected impact on dietary exposure to Ace K for this group due to an increased MPL for Ace K in chewing gum (see Table A2.3 in Appendix 2). As the proportion of Australian 7–11 years olds consuming intensely sweetened chewing gum (<1%) was similar to that for the population aged 12 years and over (<1%) the impact of increasing the MPL for Ace K in chewing gum on estimated total dietary exposure is also expected to be similar.

The same trend was noted for the New Zealand populations, where the proportion of New Zealand children consuming intensely sweetened chewing gum (1%) was similar to that for the population aged 15 years and over (<1%) so the impact of changing the MPL for Ace K in chewing gum on estimated total dietary exposures is also expected to be similar.

4.2.4   Major foods contributing to Ace K dietary exposure

From the 2004 sweetener survey, for those participating in the 7-day diary survey the contribution to total Ace K dietary exposure from confectioneries (i.e. chewing gum, lollies, chocolate etc), was 7% for the Australian and 10% for the New Zealand population. Food groups with the highest contribution for Ace K dietary exposure were non-alcoholic beverages (including carbonated soft drinks, cordial and fruit drinks) for both Australian and New Zealand populations (61% and 82%, respectively), followed by flavoured yoghurts and mousses (22% and <5% respectively). An increase in the MPL for Ace K in chewing gum might be expected to increase the percentage contribution to total estimated Ace K dietary exposure from chewing gum by a limited amount depending on its share of the chewing gum market, with a subsequent small decrease in percentage contribution from all other foods.

4.3   Dietary exposure assessment conclusion

The estimated mean and 90th percentile dietary exposures to Ace K for population groups aged 12 years and over were well below the ADI including when the increased MPL *Scenario* to intensely sweetened chewing gum was considered. As a similar or lower proportion of younger children reported consuming intensely sweetened products compared to the population aged 12 years and over and only 0–1% reported consuming intensely sweetened gum in recent surveys, the impact on increasing the MPL for Ace K in chewing gum on this age group is likely to be minimal. While no information has been considered on the changes in food industry use of Ace K since the 2004 sweetener survey, it is considered extremely unlikely that any changes would be of a degree that would result in estimated dietary exposures for any population group in Australia or New Zealand exceeding the ADI.

5 Risk characterisation

The proposed increase in the MPL for Ace K in chewing gum from 2000 mg/kg to 5000 mg/kg results in estimated dietary exposure to Ace K that is well below the ADI of 15 mg/kg bw. There are no public health and safety concerns associated with the proposed increase in the MPL for Ace K in chewing gum.

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Appendix 1 – Dietary Exposure Assessments at FSANZ

A dietary exposure assessment is the process of estimating how much of a food chemical a population, or population sub group, consumes. Dietary exposure to food chemicals is estimated by combining food consumption data with food chemical concentration data. The process of doing this is called 'dietary modelling'.

Dietary exposure = food chemical concentration x food consumption

FSANZ's approach to dietary modelling is based on internationally accepted procedures for estimating dietary exposure to food chemicals. Different dietary modelling approaches may be used depending on the assessment, the type of food chemical, the data available and the risk assessment questions to be answered. In the majority of assessments FSANZ uses the food consumption data from each person in the national nutrition surveys to estimate their individual dietary exposure. Population summary statistics such as the mean exposure or a high percentile exposure are derived from the ranked individual person's exposures from the nutrition survey.

An overview of how dietary exposure assessments are conducted and their place in the FSANZ Risk Analysis Process is provided on the FSANZ website at http://www.foodstandards.gov.au/science/riskanalysis/Pages/default.aspx.

FSANZ has developed a custom-built computer program 'Harvest' to calculate dietary exposures. Harvest is a newly built program and replaces the program 'DIAMOND' that has been used by FSANZ for many years. Harvest has been designed to replicate the calculations that occurred within DIAMOND using a different software package. Harvest was only used for this assessment to extract the consumption data for intensely sweetened chewing gum for New Zealand consumers as the confidentialised unit record files (CURF) for the most recent Australian survey are not yet incorporated into Harvest.

Further detailed information on conducting dietary exposure assessments at FSANZ is provided in *Principles and Practices of Dietary Exposure Assessment for Food Regulatory Purposes* (FSANZ 2009), available at http://www.foodstandards.gov.au/science/exposure/documents/Principles%20_%20practices%20exposure%20assessment%202009.pdf.

A1.1   Food consumption data used

The most recent food consumption data available were used to estimate exposures to Ace K for the Australian and New Zealand populations. The national nutrition survey (NNS) data used for these assessments were:

- The 2011-12 Australian National Nutrition and Physical Activity Survey (2011 NNPAS)
- The 2002 New Zealand National Children's Nutrition Survey (2002 NZNNS)
- The 2008-09 New Zealand Adult Nutrition Survey (2008 NZANS).

The design of each of these surveys varies somewhat and key attributes of each are set out below. Further information on the National Nutrition Surveys used to conduct dietary exposure assessments is available on the FSANZ website at http://www.foodstandards.gov.au/science/exposure/Pages/dietaryexposureandin4438.aspx.

A1.1.1 2011-12 Australian National Nutrition and Physical Activity Survey (2011 NNPAS)

The 2011–12 Australian National Nutrition and Physical Activity Survey (NNPAS) undertaken by the Australian Bureau of Statistics is the most recent food consumption data for Australia. This survey includes dietary patterns of a sample of 12,153 Australians aged from 2 years and above. The survey used a 24-hour recall method for all respondents, with 64% of respondents also completing a second 24-hour recall on a second, non-consecutive day. Only the Day 1 24-hour recall data for all respondents were used for this assessment. These data were weighted for use in the calculation (CURF data set used directly).

A1.1.2 2002 New Zealand National Children's Nutrition Survey (2002 NZNNS)

The 2002 NZNNS was a cross-sectional and nationally representative survey of 3,275 New Zealand children aged 5–14 years. The data were collected during the school year from February to December 2002. The survey used a 24-hour food recall and provided information on food and nutrient intakes, eating patterns, frequently eaten foods, physical activity patterns, dental health, anthropometric measures and nutrition-related clinical measures. It was also the first children's nutrition survey in New Zealand to include a second day diet recall data for about 15% of the respondents, and dietary intake from both foods (including beverages) and dietary supplements. Only the Day 1 24-hour recall data for all respondents (excluding supplements) were used for this assessment. These data were weighted for use in Harvest.

A1.1.3 2008-09 New Zealand Adult Nutrition Survey (2008 NZANS)

The 2008 NZANS provides comprehensive information on the dietary patterns of a sample of 4,721 respondents aged 15 years and above. The survey was conducted on a stratified sample over a 12 month period from October 2008 to October 2009. The survey used a 24-hour recall methodology with 25% of respondents also completing a second 24-hour recall. The information collected in the 2008 NZANS included food and nutrient intakes, dietary supplement use, socio-demographics, nutrition related health, and anthropometric measures. Only the Day 1 24-hour recall data for all respondents were used for this assessment. These data were weighted for use in Harvest.

A1.2 Limitations of dietary exposure assessments

Dietary exposure assessments based on 2011 NNPAS, 2002 NZNNS and 2008 NZANS food consumption data provide the best estimation of actual consumption of a food and the resulting estimated dietary exposure assessment for the Australian population aged 2 years and above, as well as the New Zealand populations aged 5–14 years and 15 years and above, respectively. However, it should be noted that NNS data do have limitations. Further details of the limitations relating to dietary exposure assessments undertaken by FSANZ are set out in the FSANZ document, *Principles and Practices of Dietary Exposure Assessment for Food Regulatory Purposes* (FSANZ 2009).

Appendix 2 – Dietary Exposure Assessment Results

Table A2.1: Estimated dietary exposures to Ace K based on 2004 sweetener survey (consumers only)

Country	Age group	2004 sweetener survey dietary exposures	
		Mean	90th
		%ADI	%ADI
Australia and New Zealand combined	12 years and above	3	6
Australia	12 years and above	4	6
New Zealand	12 years and above	3	6

Table A2.2: Estimated consumption of intense sweetened intensely chewing gum

Country	Survey	Age group	No. Respondents	Consumers		Consumption (Day1 consumers only)	
				%	count	Mean g/day	90th g/day
Australia	2011	2 years and above	12153	<1%	50	7.4	25
		12 years and above	10572	<1%	47	7.7	25
		7-11 years	802	<1%	3	2.5	NA
		2-6 years	779	Not consumed		NA	NA
New Zealand	2008	15 years and above	4721	<1%	13	4.9	5.8
	2002	5-14 years	3275	1%	37	4.6	16

Table A2.3: Estimated dietary exposures of Ace K from consumption of intense sweetened Chewing gum (consumers only)

Country	Survey	Age group	Consumers	Baseline (current permissions in Schedule 15)		Scenario (baseline + increased MPL)	
			%	Mean %ADI	90th %ADI	Mean %ADI	90th %ADI
Australia	2011	2 years and above	<1%	1%	4%	3%	9%
		12 years and above	<1%	1%	4%	3%	9%
		7-11 years	<1%	1%	NA	3%	NA
		2-6 years	Not consumed	NA	NA	NA	NA
New Zealand	2008	15 years and above	<1%	<1%	1%	2%	3%
	2002	5-14 years	1%	2%	6%	4%	14%

Table A2.4: Estimated dietary exposures to Ace K combined with consumers of intense sweetened Chewing gum and 2004 sweetener survey (consumers only)[4]

Country	Survey	Age group	Baseline (Including current permissions in Schedule 15)		Scenario (baseline + increased MPL)	
			Mean %ADI	90th %ADI	Mean %ADI	90th %ADI
Australia	2011	12 years and above	5	10	7	15
New Zealand	2008	15 years and above	4	7	5	9

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[4] Combined estimated dietary exposures from the 2004 sweetener survey (baseline) and the Ace K exposures from the intensely sweetened chewing gums were estimated for populations aged 12 years and over for Australia and 15 years and over for New Zealand (consumers only) and expressed as a percentage of the ADI