



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

FINAL ASSESSMENT

APPLICATION A588

**VOLUNTARY ADDITION OF FLUORIDE TO
PACKAGED WATER**

SUPPORTING DOCUMENT 2

NUTRITION RISK ASSESSMENT REPORT

SUMMARY

The primary purpose of the nutrition risk assessment is to establish if fluoridated packaged water is nutritionally equivalent to fluoridated reticulated water.

The benefits and risks associated with fluoride exposure were assessed in relation to rates and trends for dental caries and dental fluorosis and potential associations with other conditions. As part of this consideration on benefits and risks, FSANZ has reviewed the effect of fluoridated packaged waters on dental erosion. This assessment has been included following submitter concerns that the acidity of fluoridated packaged waters may promote dental erosion.

In respect to nutritional equivalence, FSANZ found that:

- Packaged water is nutritionally equivalent to reticulated water in relation to nutrients other than fluoride. If fluoride is added to packaged water between 0.6-1.0 mg/litre, then it will be nutritionally equivalent to fluoridated reticulated water supplies in Australia and New Zealand.
- The requested forms of fluoride have been added to community water supplies in Australia and New Zealand for many years, some forms of which are also used in dental products such as toothpaste.
- The requested forms of fluoride are highly bioavailable when added to water.

The benefit of fluoride in reducing the prevalence of dental caries has long been established. Dental caries in adults have decreased over the last few decades due to the natural attrition of older generations with higher rates of dental caries and since the introduction of water fluoridation. Lower rates of dental caries are observed in people who reside in areas with fluoridated water compared to non-fluoridated areas for both Australia and New Zealand.

The main health risk associated with the fluoridation of water is dental fluorosis. Dental fluorosis is generally recognised as an irreversible condition, particularly in relation to severe forms. Very mild and mild dental fluorosis are observed in around 10-25% of Australian and New Zealand children but these signs are not considered to be a health concern. Moderate dental fluorosis (the basis of the Upper Level for fluoride and a clinical concern) is rarely seen in Australia and New Zealand. The prevalence of very mild and mild dental fluorosis is usually higher in fluoridated compared to non-fluoridated areas. However, there is no evidence of skeletal fluorosis that is attributable to fluoridated water supply sources in Australia or New Zealand.

Available evidence indicates that there is no evidence of any adverse effects other than very mild or mild dental fluorosis, from current levels of water fluoridation or dietary intakes in Australia or New Zealand. Also it has been determined that packaged waters with a low pH (including those with added fluoride) are likely to have a negligible effect on the potential for tooth enamel erosion due to a low buffering capacity.

FSANZ therefore concludes that the benefits of water fluoridation in relation to the reduction of dental caries outweigh the risk of developing dental fluorosis. It is recognised that the fluoridation of water (including packaged water) may result in some mild dental fluorosis. The fluoridation of packaged water at levels permitted in Australia and New Zealand reticulated water supplies is unlikely to result in any other adverse health effects.

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1. INTRODUCTION

Food Standards Australia New Zealand (FSANZ) received an Application from the Australian Beverages Council Ltd on 23 August 2006 seeking to permit the voluntary addition of fluoride to packaged water. The Applicant requested permission for sodium fluoride, sodium fluorosilicate (also called sodium silicofluoride) and hydrofluorosilicic acid to be voluntarily added to packaged water to a level of between 0.6-1.0 mg fluoride/L. Current levels of water fluoridation in Australia and New Zealand range between 0.6 and 1.0 mg/L.

Fluoride is the ionic form of fluorine (F). Fluoride is ubiquitous in the environment and is a natural constituent of the body involved in the mineralisation of teeth and bones. About 99% of the body's fluoride is found in calcified tissues (such as bone and teeth), to which it is strongly but not irreversibly bound. This function is discussed later in this document.

2. NUTRITION RISK ASSESSMENT

2.1 Scope of Assessment

In accordance with the Ministerial Policy Guideline *Fortification of Foods with Vitamins and Minerals*, the Application is being assessed on the basis of nutritional equivalence because fluoridated packaged water can be considered a substitute beverage for fluoridated reticulated (tap) water.

The purpose of the nutrition risk assessment is to establish if packaged water is nutritionally equivalent to fluoridated reticulated water. The bioavailability of the proposed permitted forms was also assessed. The benefits and risks associated with fluoride exposure have also been assessed in relation to rates and trends for dental caries and dental fluorosis and potential association with other conditions.

Food technology and safety considerations of adding fluoride to packaged water are discussed in the main body of the report and Supporting Document 4.

2.2 Literature Reviewed

The nutrition assessment included consideration of the information provided by the Applicant in relation to nutritional issues but also had regard to other available information, including from the scientific literature, general technical information, key reports, position statements, independent scientists and experts, other regulatory agencies and international agencies and the general community. Information from reliable websites was also used.

Some relevant references included in submissions were also reviewed. The reference lists of the papers reviewed were assessed for further relevant information.

A literature search was conducted using the internet and in particular, PubMed. Topics searched included dental caries and fluorosis, acidic beverages and dental erosion, bioavailability of fluoride and other adverse effects. The search for evidence for dental caries and fluorosis was primarily restricted to articles from Australia and New Zealand. However, international studies were reviewed where relevant. Dental experts in Australia and New Zealand also highlighted key papers for review.

The majority of the studies reviewed on dental caries and fluorosis were cross sectional studies, with some case-control studies. Some systematic reviews on water fluoridation were also reviewed. These studies had some limitations which included one or more of the following:

- no national data for Australia or New Zealand;
- reviewed different adverse effects;
- used different scales for rating adverse effects;
- the concentration of fluoride in the water the subjects have consumed and the level of dietary intake are not reported, not investigated or are unknown;
- participants move residence during their lives therefore have varying exposure to different levels of fluoride from water and the diet and different exposure times at different levels of intake; and
- were older studies prior to changes in dental policies in Australia.

The first limitation is partially accounted for by several studies being available for consideration for different areas of each country. The second and third limitations meant it was sometimes difficult to compare studies. Not knowing the fluoride intakes, and only knowing the fluoride content in the water, made it difficult to determine what levels of intake are directly associated with development of dental fluorosis. The age of the studies were considered and greater emphasis placed on more recent data when assessing current prevalence of dental caries and fluorosis.

The literature search for acidic beverages and dental erosion was complicated by the lack of *in vivo* studies and epidemiological research that included packaged waters within their study designs. As a result, FSANZ was limited to using a small number of *in vitro* studies on the subject. Because of this limitation, FSANZ broadened its assessment to include material relating to the chemistry of dental erosion and to compositional data on low pH packaged waters, to better inform an understanding of how fluoridated packaged waters influence dental erosion outcomes.

3. NUTRITIONAL EQUIVALENCE

This Application is being assessed on the basis of nutritional equivalence. Nutritional equivalence in the context of this assessment is between fluoridated reticulated water and fluoridated packaged water. Similarities in nutrient content between packaged water and reticulated water in Australia and New Zealand, whether fluoridated or not, were assessed in order to determine their comparability as equivalent beverages. This included an evaluation of whether the contribution to the diet of other nutrients in water would be affected by substituting reticulated water with packaged water. FSANZ also considered the fluoride content of the waters.

3.1 Composition of reticulated and packaged water

3.1.1 Australian data

The current publicly available food composition data for Australia, NUTTAB 2006, contains information on the nutritional composition of both ‘tap’ and ‘bottled’ water. This information is shown in Table 1 excluding fluoride.

Table 1: The nutritional composition of tap and bottled water in Australia

Nutrient	Value per 100 g	
	Tap water	Bottled water
Energy	0 kJ	0 kJ
Moisture	100 g	100 g
Nitrogen	0 g	0 g
Protein	0 g	0 g
Fat*	0 g	0 g
Sugars, total	0 g	0 g
Starch	0 g	0 g
Total dietary fibre	0 g	0 g
Alcohol	0 g	0 g
Calcium	1 mg	1 mg
Iodine	0.7 µg	0.2 µg
Iron	0 mg	0 mg
Magnesium	1 mg	5 mg
Phosphorus	0 mg	0 mg
Potassium	0 mg	0 mg
Sodium	0 mg	1 mg
Thiamin	0 mg	0 mg
Riboflavin	0 mg	0 mg
Niacin	0 mg	0 mg
Niacin derived from Tryptophan or Protein	0 mg	0 mg
Niacin equivalents	0 mg	0 mg
Folate	0 µg	0 µg
Dietary Folate Equivalents	0 µg	0 µg
Vitamin C	0 mg	0 mg
Beta Carotene	0 µg	0 µg
Beta Carotene Equivalents	0 µg	0 µg
Retinol	0 µg	0 µg
Retinol Equivalents	0 µg	0 µg
Alpha Tocopherol	0 mg	0 mg
Vitamin E	0 mg	0 mg

Source: (FSANZ, 2006)

Notes: kJ = kilojoules, g = grams, mg = milligrams, µg = micrograms

* All lipids when reported separately, including saturated, monounsaturated, polyunsaturated, cholesterol, long chain Omega 3 etc, were also all zero concentrations.

Only nutrients where the information was available for both types of water have been included in the table. The composition data for manganese was left out of the table due to the unreliability of the data. The values represent average, or the most likely values, as natural variation occurs in the range of tap and bottled waters available for consumption.

Only three nutrients (shown in bold in the Table) had a slightly different concentration between 'tap' and 'bottled' water. These nutrients were iodine, magnesium and sodium.

3.1.2 New Zealand data

The latest publically available nutrient composition data for New Zealand (Athar *et al.*, 2006) were also used to assess the nutritional equivalence of ‘tap’ water compared to ‘bottled’ water. Table 2 shows the nutrient profile for these two waters for nutrients other than fluoride. Only one nutrient (shown in bold in the Table) showed a slight difference in concentration between ‘tap’ and ‘bottled’ water, which was sodium.

Table 2: The nutritional composition of tap and bottled water in New Zealand

Nutrient	Value per 100 g	
	Tap water	Bottled water
Energy	0 kJ	0 kJ
Water	100 g	100 g
Protein	0 g	0 g
Fat*	0 g	0 g
Sugars, total	0 g	0 g
Starch	0 g	0 g
Total dietary fibre	0 g	0 g
Calcium	2 mg	2 mg
Iron	Trace	Trace
Potassium	2 mg	Trace
Selenium	Trace	Trace
Sodium	1 mg	3 mg
Zinc	0 mg	Trace
Thiamin	0 mg	0 mg
Riboflavin	0 mg	0 mg
Total Niacin Equivalents	0 mg	0 mg
Folate, Total	0 µg	0 µg
Vitamin B6	0 mg	0 mg
Vitamin B12	0 µg	0 µg
Vitamin C	0 mg	0 mg
Beta Carotene Equivalents	0 µg	0 µg
Total Vitamin A Equivalents	0 µg	0 µg

Source: (Athar *et al.*, 2006)

Notes: kJ = kilojoules, g = grams, mg = milligrams, µg = micrograms

* All lipids when reported separately, including saturated, monounsaturated, polyunsaturated, cholesterol, were also all zero concentrations.

3.2 Fluoride concentrations

At present, packaged water is not permitted to contain added fluoride. A recent analytical survey (Cochrane *et al.*, 2006) assessed nine brands of packaged spring and filtered water in Australia and determined that all brands had fluoride concentrations of below 0.08 mg/L.

The mean fluoride contents in reticulated water supplies in Australia are between 0.6-1.0 mg/L in fluoridated areas, with lower levels in some places in the Northern Territory at around 0.5 mg/L. These data correspond with the target range in the National Health and Medical Research Council (NHMRC) Public Statement (NHMRC, 2007a) on water fluoridation of 0.6-1.1 mg/litre. Actual concentrations of fluoride in reticulated water in New Zealand averages around 0.8-0.9 mg/L in fluoridated areas (Water Care Services Limited, 2007).

This corresponds with the target range in *Drinking-water standards for New Zealand 2005* of 0.7–1.0 mg/L (Ministry of Health, 2005).

3.3 Evaluation of nutritional equivalence

The macronutrient content is equivalent between the two types of waters with zero concentrations for energy, fat, protein, carbohydrates, fibre and alcohol. This is also the case for a range of vitamins which also have zero concentrations.

There are some very minor differences between some minerals in ‘tap’ compared to ‘bottled’ water as outlined above. This is expected given the natural variation of nutrients in foods, beverages and water. The natural variation in mineral content in water can be influenced by time of year, the region where the product originated and any effects of processing. Variation in reported nutrient content can also be attributable to the analytical method used to measure mineral levels. In relation to differences in the concentration of iodine for example, the area from which the water was collected can strongly influence the concentrations in water, therefore, this variation could be attributed to the different sources of the samples.

While there are minor differences for the small number of minerals indicated, the results are still similar and generally in the same order of magnitude. Therefore, the intake of these minerals would not be expected to differ considerably as a result of substituting reticulated water with packaged water. To demonstrate this, a simple calculation to determine the variation in dietary intake was undertaken based on a mean consumption of water. The estimated intake was compared to the Recommended Dietary Intake (RDI) or Adequate Intake (AI) to determine if the variation in nutrient intakes due to the different concentrations would be important considering normal daily dietary requirements (see Table 3). For magnesium for example, the estimated intakes from both ‘tap’ and ‘bottled’ water are well within the RDI for adults of 310-420 mg/day (NHMRC and NZ MoH, 2006). This is also seen with the other nutrients

Table 3: Estimated variation in intake of certain minerals for adults aged 19 years and over for tap and bottled water based

Country	Mineral	Concentration (units/100 mL)		Intake (units/day) [#]		RDI* (units/day)	AI** (units/day)
		Tap	Bottled	Tap	Bottled		
Australia	Iodine	0.7 µg	0.2 µg	6.0 µg	1.7 µg	150 µg	
	Magnesium	1 mg	5 mg	8.5 mg	42.5 mg	310-420 mg	
	Sodium	0 mg	1 mg	0 mg	8.5 mg		460-920 mg
New Zealand	Sodium	1 mg	3 mg	8.5 mg	25.5 mg		460-920 mg

[#] Based on mean consumption of 850 ml/per person/day for *Mineral Waters and Water* aged 19 years and over from the 1995 Australian National Nutrition Survey (McLennan and Podger, 1999).

* Range for magnesium is females to males.

**Range for sodium is the range for both males and females.

Overall it is considered by FSANZ that the variation in mineral content from either type of water would be well within daily variation in dietary intake. Additionally, over time, different sources of water would be consumed with varying levels of the same minerals which would average out to similar dietary intakes over time.

The forms of fluoride requested to be added to packaged water by the Applicant are the same as those added to fluoridated reticulated water, therefore reticulated and packaged water would be nutritionally equivalent in relation in this respect.

FSANZ concludes that packaged water which does not have added fluoride is nutritionally equivalent to non-fluoridated reticulated water. Should packaged water be permitted to contain between 0.6 to 1.0 mg/L of fluoride, then it would be nutritionally equivalent to fluoridated reticulated water supplies in Australian and New Zealand.

4. BIOAVAILABILITY

The bioavailability of fluoride, particularly of the requested permitted forms in drinking water, was evaluated. Some submitters at Initial Assessment suggested that different forms have different bioavailabilities.

4.1 Bioavailability from food and water

Fluoride is absorbed by the body by passive diffusion, primarily from the stomach (the mechanism and rate of which is affected by gastric acidity) and the intestine (World Health Organization, 2002). Fifty per cent of orally ingested fluoride is absorbed from the gastrointestinal tract after approximately 30 minutes. Fluoride binds to calcium, magnesium, aluminium, iron and other cations forming insoluble and poorly absorbed compounds, therefore reducing its bioavailability (Institute of Medicine, 1997; NHMRC and NZ MoH, 2006). In the absence of calcium and other cations, absorption may be as high as 80% (Institute of Medicine, 1997). If fluoride is ingested with milk, infant formula or solid foods the bioavailability may be reduced to 10 to 25% (Institute of Medicine, 1997; NHMRC, 2007b). However, the bioavailability would not be expected to differ from infant formula prepared using fluoridated reticulated water compared to fluoridated packaged water.

The addition of fluoride to water supplies has been used as a public health measure in part due to the high bioavailability of fluoride obtained from this source.

Water soluble forms of fluoride such as sodium fluoride and fluorosilicic acid, which are both used for water fluoridation, are nearly completely absorbed from the intestine and utilised by the human body (World Health Organization, 2002; Maguire *et al.*, 2005; NHMRC and NZ MoH, 2006).

Less soluble forms of fluoride (e.g. calcium fluoride, magnesium fluoride and aluminium fluoride) are poorly absorbed (World Health Organization, 2002), however, these forms are not usually added to drinking water.

Children tend to absorb more fluoride due to development of the skeleton and teeth, with around 90% of ingested fluoride being absorbed (World Health Organization, 2002). Children may retain up to 80% of fluoride ingested, whereas young and middle aged adults may retain only 50% (Institute of Medicine, 1997).

In terms of chemistry and bioavailability, there is no difference between added and naturally occurring fluoride in drinking water and the effect of cations (calcium, magnesium) in water, even packaged water, on the bioavailability is very small (Jackson *et al.*, 2002). These were *in vitro* studies. Maguire *et al* (2005) conducted a human study using naturally and artificially fluoridated water with fluoride close to 1 mg/Litre, and a reference water.

There were no statistically significant differences in absorption between the naturally versus artificially fluoridated or reference water. There was more difference between and within individual subjects.

The 'hardness' of the water is characterised by the amount of calcium and magnesium in the water. Harder waters have higher levels of these elements. It has been found that the hardness of the water does not affect the bioavailability of the fluoride (Jackson *et al.*, 2002; Maguire *et al.*, 2005).

The evidence suggesting caffeine increases the bioavailability of fluoride is contradictory (Institute of Medicine, 1997).

In conclusion, the requested forms of fluoride are highly bioavailable when added to drinking water.

4.2 Bioavailability from dental products and supplements

If toothpaste is ingested, whether it contains sodium monofluorophosphate or sodium fluoride, the fluoride is almost 100% absorbed (Institute of Medicine, 1997; Riordan, 2002). Sodium fluoride from tablets is rapidly absorbed as is fluoride from other dental products if swallowed, however if supplements are taken with milk or food, the bioavailability is decreased by around 30-40% (World Health Organization, 2002).

5. HEALTH BENEFITS

Fluoride is well recognised for its dental health benefits in relation to preventing dental caries (Fawell *et al.*, 2002; World Health Organization, 2002; NHMRC, 2007b). In requesting permission for the addition of fluoride to packaged water, the Applicant touched on a number of public health issues relating to the population intake of fluoride and methods for maintaining dental health through sufficient access to fluoride. Submissions to the Draft Assessment Report also commented on the health benefits of fluoride.

5.1 Nutritional role for bones

Fluoride has the ability to stimulate the formation of new bone (Institute of Medicine, 1997). As with teeth, an increased fluoride intake has the potential to improve the structural integrity of bone through the promotion of bone mineralisation. However the evidence on the relationship between fluoride intake and bone mineral density is too variable to demonstrate any positive health outcome (NHMRC, 1999).

5.2 Nutritional role for teeth

Dental caries is a condition defined as a destructive process causing decalcification of the tooth enamel and leading to continued destruction of enamel and dentin, and cavity formation in the tooth (W.B.Saunders Company, 1995).

Dental caries begin when some of the enamel is destroyed by acid. The acid is produced by bacteria that grow on the surfaces of teeth to form plaque. When teeth are exposed to foods or drinks containing sugars, the bacteria rapidly convert some of the sugars into acid. The plaque can hold the acid in contact with the tooth surface for up to two hours before it is neutralised by saliva. Acid exposure causes a loss of calcium and phosphate minerals from the tooth surface (demineralisation). Once the plaque acids have been neutralised the minerals can return to enamel via the saliva (remineralisation).

Fluoride intake is a significant factor in the maintenance of dental health, as it not only maintains tooth integrity but prevents tooth deterioration. Fluoride protects tooth enamel by:

- promoting repair of early damage to teeth;
- improving the chemical structure of tooth enamel making it more resistant to acid attack; and
- reducing the ability of plaque to produce acid.

The relationship between fluoride intake and dental caries is an inverse one (Institute of Medicine, 1997). However, as dental caries is a multi-factorial condition, an increase in fluoride intake by itself may not necessarily prevent dental caries formation. For example, prolonged exposure to dietary sugars and starches may also have an impact, as can many factors that affect saliva quality such as smoking, substance abuse, some medications, ageing and radiation therapy (Australian Dental Association, 2007).

Some submissions stated that there is insufficient evidence to suggest a benefit from fluoride. However, there is a very large body of evidence demonstrating that increased fluoride intake can decrease the prevalence of dental caries. The World Health Organization has classified the strength of this evidence as ‘convincing’ (World Health Organization, 2003). Water fluoridation and its contribution to the reduction of dental caries has also been described as one of the top 10 best public health measures in the United States in the twentieth century (Centres for Disease Control and Prevention, 1999). There are many studies from Australia and New Zealand in the published literature showing lower rates of dental caries in areas where water is fluoridated. Due to its role in dental health, fluoride is considered an essential nutrient by the NHMRC and the MoH (NHMRC and NZ MoH, 2006).

Due to the low natural level of fluoride in some water supplies and high levels of dental caries, many authorities world wide, including Australia and New Zealand, have permitted fluoridation of water supplies (World Health Organization, 2003). The aim of water fluoridation is the adjustment of the natural fluoride concentration in fluoride-deficient water to that recommended for optimal dental health (NHMRC and NZ MoH, 2006).

The NHMRC state the optimal level for Australia is between 0.6 mg/L and 1.1 mg/L (NHMRC, 2007a) where as the MoH recommend between 0.7 mg/L to 1.0 mg/L (Ministry of Health, 2005). The American Dental Association reports that the optimal level of water fluoridation for preventing tooth decay is 0.7-1.2 mg/L.

5.2.1 Topical action of fluoride

There are some conflicting views regarding the timing of fluoride exposure (i.e. before or after tooth eruption) on the beneficial effect on dental caries. Review articles have attributed the anti-cariogenic effects of fluoride to be mostly topical, however a pre-eruptive role continues to be suggested. Products such as toothpastes are designed to provide post-eruptive exposure to fluoride through a topical action (Singh *et al.*, 2003).

Some reports indicate that there is convincing evidence that both locally applied (i.e. direct contact with teeth) and systemic fluoride (from fluoride that has been ingested) prevent dental caries (World Health Organization, 2003; Singh *et al.*, 2003). Systemic fluoride from birth is thought to build fluoride ions directly into the developing enamel (Singh *et al.*, 2003). Early exposure to fluoride, during the pre-eruptive stage, can protect the newly erupting deciduous teeth (Institute of Medicine, 1997; Do and Spencer, 2007b). There is also a beneficial post eruptive effect from fluoride contained in saliva and dental plaque due to reduced acid production by plaque bacteria and increased mineralisation (Institute of Medicine, 1997). The Australian Dental Association Inc (ADA) report that the effect of fluoride added to water is predominantly topical, with some systemic influence in children (Australian Dental Association Inc, 2007).

An Australian study (Singh *et al.*, 2003) examined the topical effects of water fluoridation on caries in permanent molars of children aged 6-15 years. Subjects were classified into groups according to the proportion of their life exposures to different levels of water fluoridation and the level of exposure both pre- and post tooth eruption. There was an exposure-response relationship that showed significantly lower level of dental caries among the groups that had the highest pre-eruption exposure. Exposure to fluoridated water post-eruption did not result in significantly lower rates of dental caries, which was the case for pre-exposure alone. The most preventative effect was shown by both pre- and post-eruption exposure.

5.2.2 Rates of dental caries

Dental caries are often categorised or analysed collectively and are given a 'dmft' (decayed, missing and filled teeth) score (sometimes referred to as 'dmfs' (decayed, missing and filled surfaces)). The acronym is usually lower case when referring to deciduous (baby or first) teeth and upper case when referring to permanent (adult or second) teeth. This score represents a person's dental caries experience over a lifetime. Another good measure of oral health status is the percent of a population group that is caries free. It is recognised that if there are dental caries in deciduous teeth, the person is more likely to get dental caries in permanent teeth (Broadbent *et al.*, 2005).

The evidence for the rates and trends of dental caries in Australia and New Zealand were reviewed. This information is described below.

5.2.2.1 Adults

The overall trend for adults is that the prevalence of dental caries is lower now compared to decades ago.

The 2004-2006 National Survey of Adult Oral Health in Australia assessed 15-97 year olds by interview (n=14123) and dental examination (n=5505). Over 95% of the population born before 1970 had dental decay, whereas for those born after 1970 only 76% had dental decay. Across all adults, the DMFT was 12.8. This was lower for the 1970-1990 generation at 4.5 compared to the pre-1930s generation at 24.3 (Roberts-Thomson and Do, 2007). In 1973 the DMFT for adults 35-44 years was 19 (Spencer *et al.*, 1996).

Trends in caries were also examined between the 2004-06 survey and the 1987-88 national survey (Slade and Sanders, 2007). On a national basis across all ages, the DMFT decreased by 16% in the 17 years between the surveys from 14.9 DMFT in 1987-88. This reduction was influenced by the passing of older generations who had poorer dental health with higher rates of decay and tooth loss, and the emergence of the 'fluoride generation' with access to fluoridated water and toothpaste. Those born after the introduction of fluoridation had half the dental caries of those of the same age born pre-fluoridation (Spencer *et al.*, 2007).

A study on oral health specifically in South Australia (Ellershaw *et al.*, 2005) assessed the prevalence of dental caries in young adults (20-24 years). The survey showed that oral health deteriorates significantly after leaving school and the School Dental Service. Young adults have an average of 3.68 decayed teeth. Only one in five were free of decay.

National oral health surveys were conducted in New Zealand in 1976 and 1988, however the results from these surveys are not publicly available. A national oral health survey is planned for New Zealand for 2008 which will include both adults and children (NZ MoH, 2008). One study stated that dental caries in New Zealand have decreased over the 40 years prior to the late 1990's (de Liefde, 1998).

As adults get older, their dental health deteriorates and there is an increase in caries (Thomson, 2004; Broadbent *et al.*, 2006). From the Australian Adult Oral Health Study, when assessing the same population group between 1987-88 and 2004-06, dental decay increased for both those born after the 1930's and for those in the 'fluoride generation'. For example, for those born between 1916-32 the DMFT was 22.9 in 1987-88 and 24.3 in 2004-06 and for those born 1967-83 the DMFT was 2.5 in 1987-88 and 6.4 in 2004-06. These data show the success of water fluoridation on the generation exposed who had lower rates of dental caries into adulthood. One New Zealand study (Broadbent *et al.*, 2006) showed that for the same cohort at age 26, 94.9% had dental caries and at 32 years, 96.8% had caries. Tooth loss was also higher at the older age.

5.2.2.2 Children

The data reviewed for Australian children indicate that the prevalence of dental caries is increasing. This is also the case for younger children in New Zealand. The data are summarised in Table 4.

The prevalence of dental caries in Australian children decreased until about the mid-1990's after which it increased. An annual Child Dental Health Survey is conducted in Australia by the Australian Institute of Health and Welfare (AIHW) Dental Statistics and Research Unit (DSRU), which is located in the Australian Research Centre for Population Oral Health (ARCPOH) at the University of Adelaide. The latest data from 2002 (which exclude NSW due to methodological reasons) show that around half of the six year olds and 40% of 12 year olds had some experience of dental decay.

The mean dmft for 5-12 year olds was 1.68 with and the mean DMFT was 0.49. The proportion of 5-12 year olds who were caries free was 60%. Children from Queensland had the highest decay experience in deciduous teeth, while for permanent teeth it was Tasmania (Australian Institute of Health and Welfare, 2007). Higher rates of caries in Queensland can be attributed to a greater proportion of residents in the state not having access to fluoridated water.

A national time series shows that there has been an increase in dental caries among children since 1996 for deciduous teeth and since 1998/99 for permanent teeth (see Figure 1). The data are adjusted for under-reporting in the NSW data which occurred between 1996-2000 due to changes in the methodology used to obtain the data.

Other studies have shown that between 1977 and 1998 dmft scores decreased in Australian children from over three in deciduous teeth to around 1.6 and DMFT scores from 4.8 to 0.89 respectively (Armfield and Spencer, 2008). Low rates of caries (DMFT 0.13) in the 1990/1991 study of Perth children can be attributed to the majority of children having lived in fluoridated areas and the short time since the eruption of the teeth examined (Riordan, 1993a).

A study on oral health specifically in South Australia (Ellershaw *et al.*, 2005) has also highlighted the increase in dental caries in children since the late 1990's. For example, 6-7 year olds had an average of 1.44 decayed deciduous teeth in 1999 and 1.83 in 2002. For children aged 14-15 years, there was an average of 1.12 decayed permanent teeth in 1999 and 1.53 in 2000.

Socioeconomic status has been shown to have an impact on caries experience. Children from lower socioeconomic backgrounds have a significantly higher incidence of dental caries for both fluoridated and non-fluoridated areas, however, this was not as strong in permanent dentition (11-12 year olds) compared to deciduous dentition (5-6 year olds) (Armfield, 2005).

Table 4: Summary of data for the prevalence of dental caries in Children

Year	dmft/DMFT	%Caries free	Region	Age (years)	Fluoridation (mg/L in the water)	Study
2002	dmft 1.68 DMFT 0.49	60	Australia, National (excluding NSW)	5-12		AIHW, 2007
2002	dmft 1.83 DMFT 1.53		South Australia	dmft 6-7	NS	Ellershaw <i>et al.</i> , 2005
1999	dmft 1.44 DMFT 1.12			DMFT 14-15		

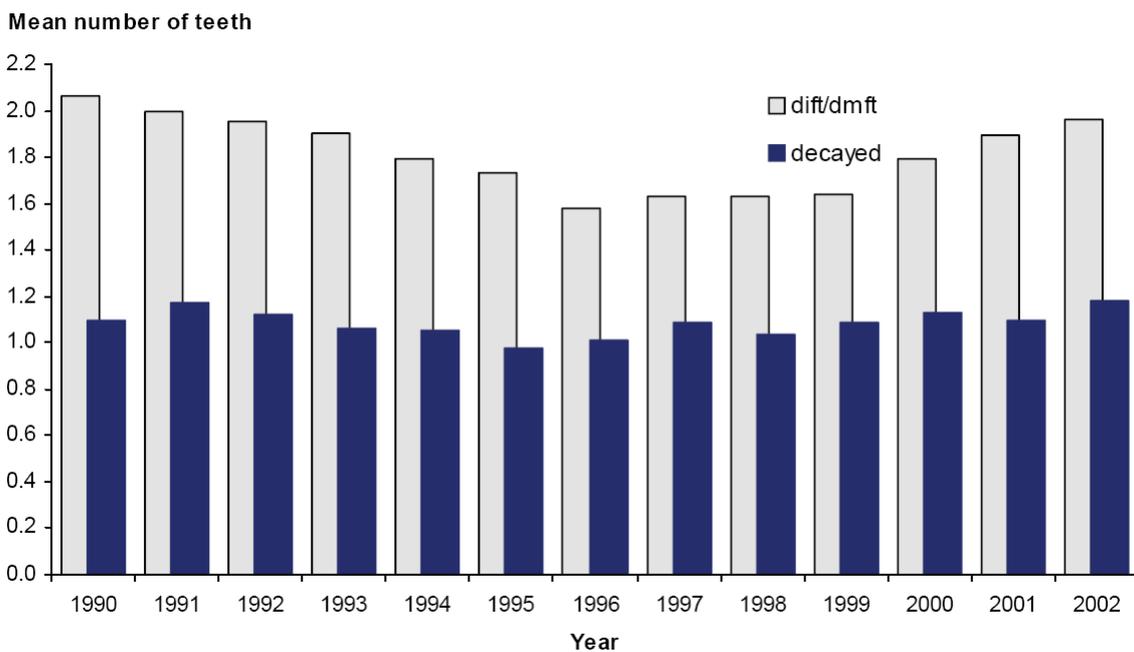
Year	dmft/DMFT	% Caries free	Region	Age (years)	Fluoridation (mg/L in the water)	Study
2002	dmfs 4.4 DMFS 1.05 F area dmfs 3.42 DMFT 0.70 Non-F area dmfs 5.11 DMFT 1.22	Deciduous 33 Permanent 57	Southland, New Zealand	Mean 9.8		Mackay & Thomson, 2005
2000	DMFT 0.3 overall DMFT 0.31 F area DMFT 0.28 NF area	83*	Perth, Bunbury, WA	10	F area 0.85 NF area 0.2- 0.3	Riordan, 2002
1998 1977	dmft 1.6 DMFT 0.89 dmft >3 DMFT 4.8		Australia			Armfield & Spencer, 2008
1996	F area dmfs 2.63 NonF area dmfs 3.8 F area DMFS 1.39 NonF area dmfs 2.37		New Zealand	5 12		Lee & Dennison, 2004
1990/ 1991	DMFT 0.13	90	Perth, WA	7 years		Riordan, 1993
1989/ 1990	DMFT 0.89 F area DMFT 1.57 NF area		Perth, Bunbury, WA	12	F area 0.85 NF area 0.2- 0.3	Riordan, 2002

Notes: F area = fluoridated area 0.8-0.85 mg/L, NF area = non-fluoridated area <0.2-0.3 mg/L.

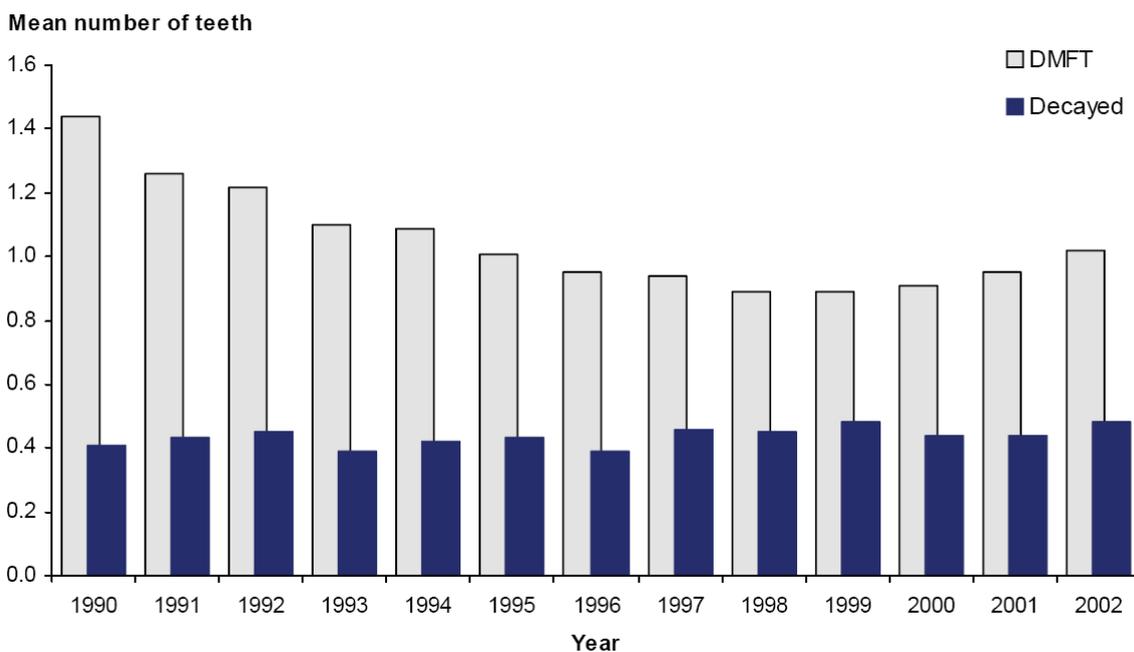
* No significant difference between fluoridated and non-fluoridated areas.

NS = not specified.

a. 6 year old children



b. 12 year old children



Source: (Armfield *et al.*, 2007)

* Adjusted for under-reporting in NSW between 1996-2000.

Figure 1: Decay experience in Australian children 1990-2002*

It has also been shown that children living in rural and remote areas have a higher prevalence of caries and higher mean dmft/DMFT scores (AIHW Dental Statistics and Research Unit, 2006). The results are shown in Table 5. About 8-12% fewer children from metropolitan areas had dental caries experience in deciduous teeth. This was also the case for permanent teeth for 9 year olds and above. This trend remained the same after also adjusting for socioeconomic status.

Table 5: Dental caries (dmft/DMFT) rates for children living in urban and rural areas

Age (years)	Measure	Metropolitan	Rural	Remote
5-6	dmft	1.53	2.14	2.31
7-8	dmft	1.80	2.54	2.51
11-12	DMFT	0.65	0.84	0.85
13-14	DMFT	1.11	1.40	1.28

Source: (AIHW Dental Statistics and Research Unit, 2006)

A summary of data for New Zealand children from the School Dental Service (Ministry of Health, 2008) are provided in Figure 2 (and Table 1 in Appendix 1). The data for the 5 year olds show a slight increase in both the dmft and percent caries free parameters between 1990 and 2006. Over the last 10 years of data examined there appears to be an increase in the dmft and decrease in the proportion that are caries free. The data show that for 12 year olds, the DMFT has decreased between 1990 and 2006, while the proportion that is caries free has slightly increased. Both parameters for 12 year olds appear to have remained relatively stable over the last 10 years examined. These data are descriptive data only and have not been corrected for any factors (e.g. area of fluoridation, socioeconomic status etc) or statistically adjusted.

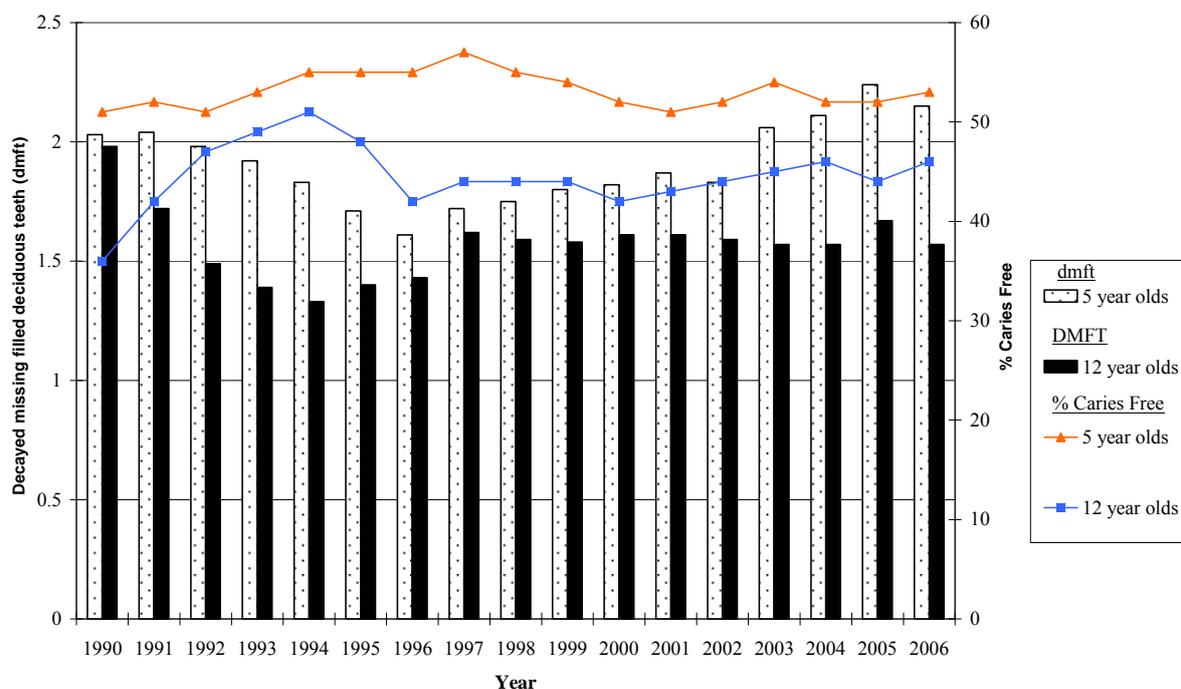


Figure 2: Dental Caries in New Zealand Children from School Dental Service data 1990-2006

Other data from New Zealand indicate that two thirds of Southland children in 2002 had caries in deciduous teeth with a mean dmfs of 4.4 (Mackay and Thomson, 2005).

Possible explanations for the rise in dental caries in children include the availability of lower fluoride toothpastes for children, increased consumption of bottled and rain water, a reduction in the number of fissure sealants and changes in the diets of children in relation to sugar and fermentable carbohydrate intake (Ellershaw *et al.*, 2005; Draper *et al.*, 2005).

5.2.2.3 *Difference between fluoridated and non-fluoridated areas*

It has been shown that children with exposure to fluoridated water have a lower prevalence of dental caries.

On a national basis, data from 2002 (Armfield *et al.*, 2007) show that children from areas with negligible fluoride in the water had poorer dental health (higher dmft/DMFT scores). This is shown in Figure 3. This difference remained when assessed by residential location and socioeconomic status.

A number of studies in Australia were reviewed assessing the difference in the rates of dental caries between fluoridated and non-fluoridated areas (Spencer *et al.*, 1996), including a study in Tamworth that evaluated dental caries before and after the implementation of water fluoridation. All studies showed a lower prevalence of caries due to water fluoridation.

One study on South Australian children (Armfield and Spencer, 2004) showed use of non-public water supplies (from bottles or rain water tanks) resulted in a significantly higher rate of dental caries in deciduous teeth but not permanent teeth. Another study on South Australian children (Do and Spencer, 2007b) evaluated rates of dental caries based on history of exposure to fluoridated water and toothpaste use.

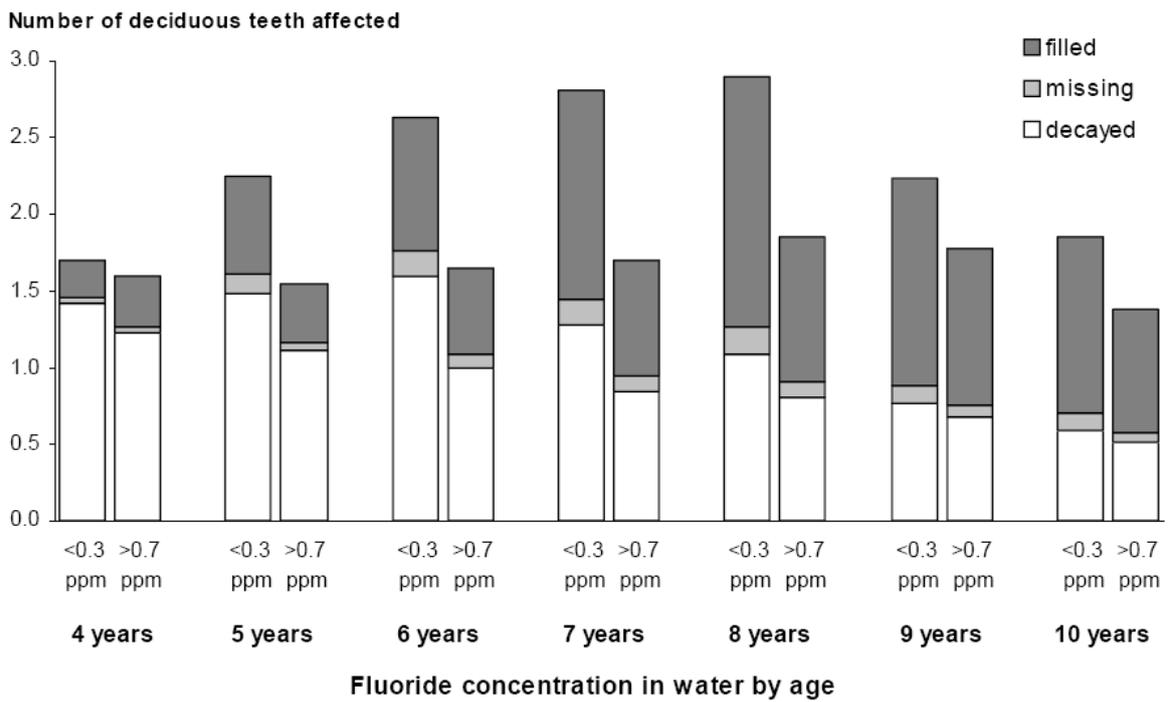
The authors found that subjects with no exposure to fluoridated water had significantly higher rates of dental caries. Commencing tooth brushing after 30 months of age was significantly associated with higher prevalence and severity of dental caries. The use of children's toothpaste (i.e. 400-500 ppm fluoride), swallowing brushing slurry and eating/licking toothpaste were not significantly associated with lower rates of dental caries.

A study in South East Queensland (Teo *et al.*, 1997) showed mean DMFS were significantly lower in subjects who had fluoride from water in the first 12 years of life (14%) or those who had fluoride supplements (8%) compared to those with no form of fluoride (25%).

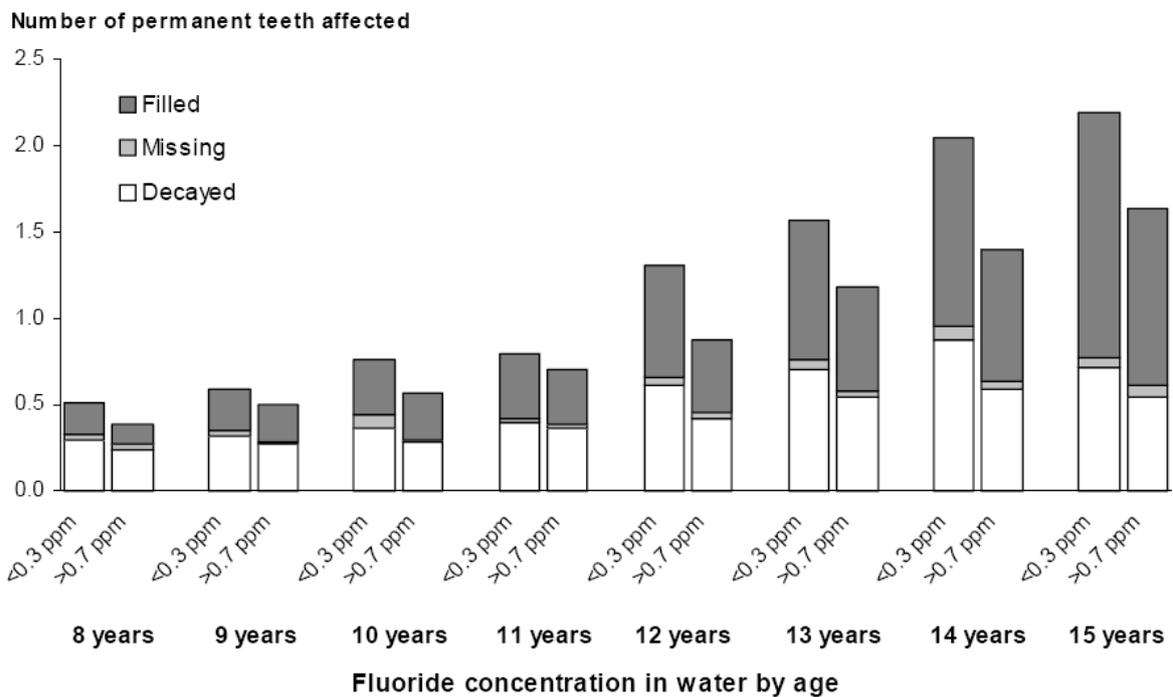
The difference in dental caries between fluoridated and non-fluoridated areas has been assessed for NSW children (Armfield, 2005). For 5-6 year olds, there were significantly higher dmft scores in six of the eight Area Health Service regions assessed. In only two regions were the dmft scores higher for fluoridated areas, but these differences were not significant. The same trend was seen for DMFT scores for 11-12 year olds where six of the 10 regions had higher scores in non-fluoridated areas and where this was the opposite, the results were again not significant.

A study on New Zealand children (Lee and Dennison, 2004) showed that caries were more prevalent in an area with no water fluoridation (Wellington) compared to an area with fluoridation (Canterbury). Five year olds in a fluoridated area had a dmfs of 2.63, and in a non-fluoridated area it was 3.8. Twelve year olds had a DMFS of 1.39 if a fluoridated area and a DMFS of 2.37 in a non-fluoridated area. Macaky and Thomson (2005) also showed this with a study in Southland children, with higher caries in non-fluoridated areas (dmfs 5.11; DMFT 1.22) compared to fluoridated areas (dmfs 3.42; DMFT 0.70).

a. Deciduous teeth



b. Permanent teeth



Source: (Armfield *et al.*, 2007)

Figure 3: Decayed, missing and filled teeth by age and fluoride concentration in water for Australian children, 2002

New Zealand data from the School Dental Service (Ministry of Health 2008) also show that in fluoridated areas the mean dmft is lower and percent caries free higher compared to non-fluoridated areas.

Several of the studies reviewed for dental caries in children included a comparison between exposure to, and not to, fluoridated water. These studies show the success of water fluoridation in the reduction of dental caries. These data dispel the notion that dental health in non-fluoridated areas can be better than those in fluoridated areas and provides evidence for the effectiveness of water fluoridation.

6. HEALTH RISKS

Fluorosis is a term that refers to the effects associated with excess fluoride intake. It manifests in two forms: dental and skeletal fluorosis (United States Institute of Medicine, 1997), of which the former is more common.

6.1 Dental fluorosis

Dental fluorosis refers to the incorporation of fluoride into the enamel of the teeth. It can range from a beneficial effect, making teeth stronger and whiter, to an adverse health effect. It varies from very thin, almost invisible, white patches or lines over the tooth surface or mottling, to significant areas of pitted enamel with brown stains arising from very high fluoride exposure. The milder forms are generally considered to be a cosmetic adverse effect.

Dental fluorosis is generally recognised as an irreversible condition, particularly in relation to moderate and severe forms. Mottling of teeth, as occurs in the mild form of fluorosis, can have numerous other causes (Australian Dental Association Inc, 2007). Instances of severe dental fluorosis are now rare in Australia (Australian Dental Association Inc, 2007).

While early exposure to fluoride can protect newly erupting teeth, it can also be a risk factor for fluorosis (Do and Spencer, 2007b). The pre-eruptive development period for teeth is considered to be the most vulnerable for the development of dental fluorosis (Institute of Medicine, 1997). Deciduous teeth erupt anywhere from 6 months (usually the central incisors) to 33 months (molars) (Dental Practice Education Research Unit, 2008). Permanent teeth develop around 3-4 months of age and erupt between 6-12 years of age (Department of Human Services Victoria, 2007). However, there is some uncertainty for the key exposure period with claims of 18-30 months of age (Puzio *et al.*, 1993), 0-6 years (Do and Spencer, 2007a), from 22 months to 36 months (Evans and Stamm, 1991) or after 12 months of age, or whether there are any post eruption influences. The NHMRC concluded that the critical time for exposure is after the first 12 months of life (NHMRC, 2007a). Mature enamel is not susceptible to the condition. Therefore, dental fluorosis only affects children up to around eight years.

Fluorosis development relates to both the time of exposure in relation to enamel formation and the cumulative duration of elevated fluoride intake (Hong *et al.*, 2006). One study suggests that it can take as little as four months to develop fluorosis (Evans and Stamm, 1991). Fluoride intake from a range of sources cumulates to produce fluorosis if ingestion occurs when the teeth are mineralising (Riordan, 2002). Fluorosis can be found on both the deciduous and permanent teeth.

Dental fluorosis has been associated with exposure from several sources, both individually or collectively, including fluoridated water (NHMRC, 1999), toothpaste, other dental products and supplement use. The Australian Drinking Water Guidelines says levels above 1.5 mg/L can cause dental fluorosis (NHMRC and NRMCC, 2004).

The clinical signs of very mild and mild dental fluorosis are not specific and can also be attributed to other enamel defects (e.g. enamel demineralisation disorders). Some of the studies assessing fluorosis may report figures that are slightly overestimated as enamel opacities not caused by fluoride may have been included according to the indices used to score fluorosis (McDonagh *et al.*, 2000). The degree of the overestimate is unknown and could be different for different studies depending on factors such as examiner training, examiner bias or poor examiner randomisation.

There are many rating scales for fluorosis. Some scales that are commonly referred to include Dean's Classification, the Thylstrup-Fejerskov (TF) Index, the Tooth surface index of fluorosis (TSIF) and the Fluorosis Risk Index (FRI). The most commonly used indexes seem to be Dean's, which was developed in the 1940's and in countries such as the United States of America, and the TF index, which is commonly used in Australian studies. The scales have different numbers of level of classification and it is difficult to match classifications in one scale to those in another. Appendix 2 shows the Dean's classification, TF index and the TSIF scale. There is also different reporting of fluorosis, from 'any' fluorosis to 'fluorosis of aesthetic concern' as well as terms such as 'mild' or 'moderate', which can be based on the rating scales mentioned above. All of these classification scales and descriptions of dental defects make it difficult to compare results between studies.

Apart from the Iowa Fluoride Study (Hong *et al.*, 2006), there is little information directly linking levels of dietary intake to rates of fluorosis. The Iowa study showed a strong significant positive relationship between total fluoride intakes (diet, water, dental products, supplements) and the prevalence of any fluorosis. Cumulative average daily intakes of more than 0.04 mg/kg bw/day result in a significantly higher prevalence of fluorosis. Intakes below 0.04 mg/kg bw had a less than 20% probability of developing any fluorosis, and the majority of the fluorosis was the mild form. The highest rates of fluorosis are associated with intakes >0.06 mg/kg bw/day. Intakes at 0.04 mg/kg bw/day have a low risk of developing any fluorosis, intakes at 0.04-0.06 have a significantly elevated risk and above 0.06 is a high risk.

FSANZ investigated the prevalence of dental fluorosis in Australia and New Zealand. The results are discussed below.

6.1.1 Rates of dental fluorosis

There were no data found on the prevalence of dental fluorosis in Australian or New Zealand adults. Studies on dental fluorosis are generally done on children. Given that the condition is generally accepted to be irreversible, rates in adults should be the same as, or higher than, the rates for children. It is likely to be higher given that today's younger adults would have had higher exposures to fluoride as children before policy changes on the use of fluorides were implemented in the early 1990's (e.g. introduction of child strength toothpastes, decrease in fluoride in infant formulas). A summary of the data on the prevalence of fluorosis in children is shown in Table 6.

There are no national data on fluorosis for Australian or New Zealand children, but there is some information from some Australian states and for some different areas in New Zealand. It is accepted that in areas with optimal water fluoridation, there will be a prevalence of around 10-12 % of very mild to mild dental fluorosis. The more recent studies in Australia (in the last decade) show a prevalence of fluorosis ranging between around 10 to 20%, but which includes primarily very mild and mild fluorosis. The prevalence of diffuse opacities in New Zealand children, which is the marker assessed in New Zealand and is similar to the milder forms of fluorosis, is reported to be between 20 and 25%.

The prevalence of fluorosis in children is decreasing. Fluorosis in Australian children was more prevalent in the early 1990's (Riordan, 2002; Do and Spencer, 2007a). Following work in the early 1990s through the NHMRC, the levels in infant formula powders were reduced, low fluoride toothpastes for children and advice on using a 'pea sized' amount were introduced and new dosage schedules for supplement use in children were recommended. Therefore, children born after 1993 should have a lower prevalence of fluorosis.

A study was undertaken in South Australia to assess the difference in the prevalence of fluorosis across three birth cohorts: before (born 1989/1990); during (born 1991/1992); and after (born 1993/1994) policy changes (Do and Spencer, 2007a). The majority of the fluorosis in south Australian children was very mild to mild (see Table 6). Overall there was a decrease in the prevalence of fluorosis across the birth cohorts: TF 1 16% earliest cohort, 15% latest cohort; and TF 2+ 18% earliest cohort and 8% latest cohort. Both the whole sample and the analysis by birth cohort showed an association between fluorosis and starting tooth brushing before 2 years of age, eating/licking toothpaste, using high fluoride toothpaste (1000 mg/kg) and exposure to fluoridated water. Supplement use was not associated with the prevalence of fluorosis.

The strongest effect on the decrease in fluorosis was due to the increased use of lower fluoridated toothpastes. These results imply that the policy changes had a beneficial effect on decreasing the prevalence of fluorosis.

One study (Spencer and Do, 2008) stated a decline in fluorosis from 45% in 1992/1993 to 26% in 2002/2003 in South Australian children (using a TF score of 1+). The main reason for the decrease appears to be changes in policy from around 1990 and due primarily to the change in use of dental products.

Fluorosis has been associated with exposure from several sources, both individually or collectively, including fluoridated water, toothpaste, other dental products, supplement use and use of infant formula. At the same time, some studies may show no association from these sources, which may be due to factors such as the small number of people in the study taking supplements, for example (Riordan, 1993a). Fluorosis prevalence is generally higher in children who had lived for longer periods of time in fluoridated areas (Riordan and Banks, 1991; Riordan, 1993a; Riordan, 2002). Residence in a fluoridated area as a risk factor for mild fluorosis ($TF \geq 1$) was not a significant risk factor for a more severe rating of fluorosis ($TF \geq 2$) in one study, unlike swallowing toothpaste (Riordan, 1993a), or supplement use (Riordan and Banks, 1991). Even no exposure to fluoridated water or supplements can lead to fluorosis (11-17%) (Riordan and Banks, 1991; Riordan, 2002), suggesting other factors such as toothpaste as the cause. In one study, there was a fluorosis prevalence ($TF > 0$) of 53% for persons who used supplements in accordance with directions (Riordan and Banks, 1991).

Table 6: Summary of data for the prevalence of dental fluorosis in children

Year	% with any fluorosis	Fluorosis index and score [#]	Region	Age (years)	Teeth examined	Reference
Recent ^{##}	19	Diffuse opacities	Auckland, New Zealand	9		Schluter et al, 2008
2003/2004	12 (TF=1) 11 (TF=2-3) 14 (>50% life F area) 11 (0-50% life F area) 3% (0% life F area)	TF TF ≥2	South Australia	8-13	Maxillary central incisors	Do and Spencer, 2007b
2002/2003	15 (TF=1) 12 (TF=2-3)	TF	South Australia	8-14	Maxillary central incisors	Do & Spencer, 2007a
2002	24%	Diffuse opacities	Southland, New Zealand	Mean 9.8	check	Mackay & Thomson, 2005
2000	18 (overall) 15 (TF=1) 3 (TF=2) <1 (TF=3) 22 F area* 11 NF area	TF>0	Perth (F area), Bunbury (NF area), WA	10	Not specified as to permanent or deciduous	Riordan, 2002
1992/1993	57 F area** 29 NF area 34 F area 19 NF area	TF ≥1 Deans ≥ very mild	South Australia	10-17		Puzio 1993
1990/1991	48 (overall) 31 (TF=1) 13 (TF=2) 5 (TF=3)	TF Any > 0 More severe ≥ 2	Perth, WA	7	Permanent	Riordan, 1993
1989/1990	40 F area (0.8) 33 NF area (0.2) (p>0.05) 27 (TF=1) 9 (TF≥2)	TF Any > 0	Perth (F area), Bunbury (NF area), WA	12	Permanent	Riordan and Banks, 1991

Notes: F area = fluoridated area, NF area = non-fluoridated area

* Water fluoride concentration F area 0.85 mg/L, NF area <0.2-0.3 mg/L.

** Water fluoride concentration F area 1.0 mg/L, NF area 0.3 mg/L. Same data classified using two different indexes of fluorosis.

See Appendix 2 for index definitions.

##Exact year unknown, study still in press.

A study on South Australian children between 2003 and 2004 (Do and Spencer, 2007b) evaluated rates of fluorosis based on history of exposure to fluoridated water and toothpaste use. The authors found that exposure to fluoridated water from birth to three years was significantly associated with the prevalence of fluorosis whereas the group with no exposure to fluoridated water had significantly lower rates of fluorosis. Commencing tooth brushing after 30 months of age was not significantly associated with lower rates of fluorosis. The use of children's toothpaste (i.e. 400-500 ppm fluoride) was significantly associated with lower prevalence of fluorosis. Swallowing brushing slurry was associated with higher odds of fluorosis and eating/licking toothpaste was significantly associated with a higher risk of fluorosis.

For the two studies 10 years apart in two areas of Western Australia; Perth, a fluoridated area and Bunbury, a non-fluoridated area (Riordan and Banks, 1991; Riordan, 2002), a reduction of the prevalence of fluorosis occurred between 1990 and 2000. The author attributed a decline in fluorosis to changes in education. After 1989/1990, the School Dental Service discouraged fluoride supplements and promoted low fluoride toothpaste for children under 6 years. No increase in dental caries was recorded.

Different assessment methods are used in New Zealand. Australia assesses 'dental fluorosis' and New Zealand assesses 'enamel defects'. The enamel defects observed are consistent with mild dental fluorosis, however these defects may or may not be attributed to fluoride. The prevalence of enamel defects in New Zealand children has not increased since the 1980s. The prevalence of diffuse opacities in New Zealand children has not increased in children who have lived their whole lives in fluoridated areas, and severe enamel defects have decreased (Mackay and Thomson, 2005). There was no significant difference in severe enamel defects between fluoridated and non-fluoridated areas. Preliminary data for a study in the Auckland region (Schluter *et al.*, 2008) showed that children living continuously in fluoridated areas were four times more likely to have diffuse opacities compared to those living in non-fluoridated areas. Other studies have shown that enamel defects are higher in children in New Zealand from fluoridated areas compared to non fluoridated (Suckling and Pearce, 1984; Cutress *et al.*, 1985).

Different scales of rating fluorosis can result in different estimates of rates of fluorosis in the population groups being assessed. Table 6 shows one study where the same children were rated using two different scales (Deans and TF) (Puzio *et al.*, 1993) and resulted in rates of fluorosis that were higher using the TF index. The majority of studies also just report any fluorosis and do not distinguish between mild or moderate. Moderate fluorosis on the Dean scale was the basis of the Upper Level (UL) for children aged up to 8 years. Also, different teeth are examined (e.g. deciduous versus permanent; incisors versus molars), which can also influence the result. Permanent central maxillary incisors are reported to have a higher prevalence of fluorosis than first permanent molars (Hong *et al.*, 2006). Different individuals in the same exposure category have different susceptibilities to fluorosis (Hong *et al.*, 2006).

A study conducted in Perth in 1989-90 asked parents, dentists and student dentists to rate the appearance of children's teeth. The majority (85-90%) were neutral or agreed that teeth with no fluorosis (TF scale=0) looked nice, approximately 80% had this opinion for teeth with barely discernable fluorosis (TF=1) and 65-80% for teeth graded as TF=2. However only 40-50% agreed or were neutral that teeth graded as TF=3 looked nice (Riordan, 1993b). Therefore, the majority of mild fluorosis seen in Australia at present is not generally perceived as cosmetically adverse.

6.2 Skeletal effects, including fluorosis

Skeletal fluorosis is a serious medical condition, and can lead to significant bone degradation and neurological manifestations. This condition is, however, extremely rare even with excessive fluoride intake. Only five cases have been diagnosed in the U.S. over the last 45 years. It is more common in India and Pakistan where natural fluoride levels in water may be as high as 18 mg/L. The rarity of skeletal fluorosis is due primarily to the need for continued chronic exposure to very high fluoride intakes (in the order of about 10 mg/day for >10 years to produce mild forms of skeletal fluorosis). The Australian Drinking water Guidelines says levels above 4 mg/L can cause skeletal fluorosis (NHMRC and NRMCC, 2004), but the maximum set in the Guidelines was 1.5 mg/L.

No evidence for occurrence of skeletal fluorosis in Australia or New Zealand was found. Levels of fluoride in drinking water in Australia and New Zealand are much lower than those attributed to the development of skeletal fluorosis.

An Australian review in 2001 assessing 33 individual studies concluded that fluoride at 1 mg/L does not have an adverse effect on bone strength, mineral density, or evidence of fractures. A systematic review of fluoridation in McDonagh *et al* (2000), followed by a subsequent systematic review by the NHMRC (2007b), also found no clear evidence of an association between any other adverse bone related conditions including fracture or bone development where water is fluoridated around 1 mg/litre. .

6.3 Dental Erosion

Comments to the Draft Assessment Report for Application A588 have expressed concern that due to the potential for an increase in acidity of the final product (a reduction in pH), the fluoridation of packaged waters may create conditions that could promote dental erosion. As a result of this feedback from submitters, FSANZ has conducted an additional review of available literature to further explore the process of dental erosion, and to determine how packaged waters may or may not contribute to this process, especially packaged waters with an acidic (low pH) profile.

6.3.1 Background information on dental erosion

Dental erosion involves the chemical etching and irreversible loss of dental hard tissue by exposure to non-bacterial acids (Moynihan and Petersen, 2004; British Nutrition Foundation, 2009). The aetiology of dental erosion is complex and multi-factorial, and depends on an individual being predisposed to the development of the condition, either through non-dietary behaviours or physiological characteristics (Moss, 1998; Lussi *et al.*, 2004; Australian Dental Association, 2006). Some of the biological and behavioural factors for dental erosion include the sipping of drinks, use of chewable vitamin tablets, and dental soft and hard tissue composition.

Available scientific literature cites the acidity of beverages as an important factor in the development of dental erosion (Lussi *et al.*, 1993; Jendsdottir *et al.*, 2005; Featherstone and Lussi, 2006). However, FSANZ has been unable to identify any controlled experimental trials on the relationship between packaged water and dental erosion.

A pH level less than 5.5 is widely reported within scientific literature and through public health advice as the critical pH for the relationship between beverages and dental erosion (derived from the pH at which tooth demineralisation occurs) (Stephen, 1940; Meurman and ten Cate, 1996; Milosevic, 1997; Australian Dental Association, 2005).

The evidence based on epidemiological studies (observational studies) has shown that there is an association between acidic beverages such as juice and carbonated soft drinks and the development of dental erosion (Moynihan and Petersen, 2004). The World Health Organization has reviewed this epidemiological evidence, and classified the strength of the relationship between acidic beverages and dental erosion as 'probable' (World Health Organization, 2003).

There are a large number of *in vitro* studies investigating beverages and dental erosion, however as with epidemiological studies, this evidence is dominated by juice and carbonated soft drinks. Therefore, it is not clear whether the relationship between these beverages and dental erosion can be applied to packaged waters.

6.3.2 Determining whether low pH packaged water presents a dental erosion risk

Due to the limitations of the epidemiological evidence, FSANZ has conducted a review of the literature to determine the influence of low pH packaged waters on dental erosion outcomes. Three *in vitro* studies were identified by FSANZ (Parry *et al.*, 2001; Seow and Thong, 2005; Kitchens and Owens, 2007) that investigated packaged water as one of a number of a test beverages (with a pH range of 2.1-8.1) on human dental tissue. Only one of these studies involved packaged water at a pH of 5.5.

The results of these three studies are consistent with the trend shown in other studies on juice and carbonated soft drinks, that as the pH of the beverages declines there is a corresponding increase in dental erosion.

Because of the limited direct evidence on packaged water, FSANZ has investigated the chemical processes involved in dental erosion. An important aspect of the chemistry of dental erosion is that the buffering capacity of a beverage may modify the relationship of pH to dental erosion, where beverages with a high buffering capacity have a greater erosive potential than beverages with a lower buffering capacity at a similar pH. The trend in dental erosion observed when beverage pH is considered in isolation is not, therefore, the full determinant of a beverage's erosive potential. As a result, the buffering capacity of low pH packaged water has been a key consideration in this assessment.

6.3.2.1 In vitro studies on packaged waters and dental erosion

The three studies mentioned above all used similar experimental methods. Samples of human enamel were immersed in test beverages (packaged waters, juice, and carbonated soft drinks), with part of the enamel coated with either an acid resistant wax or varnish. The samples were held in the test beverage for a defined period of time, with 30 minutes for Parry *et al.* (2001) and Seow and Thong (2005), and 350 hours for Kitchens and Owens (2007). Following immersion, Seow and Thong (2005) and Kitchens and Owens (2007) assessed the effects on enamel surface erosion, whereas Parry *et al.* (2001) measured erosion through spectrophotometric assays of phosphorus dissolution from the enamel.

The results of these studies show that beverages with low pH levels produce greatest levels of dental erosion, with the packaged waters producing minimal dental erosion outcomes of the range of test beverages.

Unfortunately none of these studies investigated the buffering capacity of their packaged water test beverages, and so the influence of this factor on the outcomes is unknown. Also, the pH of the packaged waters used in Parry *et al.* (2001) and Seow and Thong (2005) were within a non-acidic range (pH 7.12-8.1), and so these results may not represent the erosive potential of packaged waters with a pH less than 5.5.

Kitchens and Owens (2007) assessment of a packaged water showed a slightly higher but non-significant ($p > 0.05$) difference in dental erosion compared to the tap water control. However with a pH near to 5.5 (pH = 5.48), this packaged water may not have been acidic enough to produce a noticeably different level of erosion compared to the tap water control.

These three studies are considered insufficient to determine the effect of low pH water on dental erosion, as the pH of the test waters was too high.

6.3.2.2 *Chemistry of dental erosion*

6.3.2.2.1 Beverage pH and buffering capacity

Recent investigations into the dental erosion process have indicated that the buffering capacity of a beverage is also important in addition to its pH. A beverage with a high buffering capacity¹ can depress the pH inside the oral cavity for longer periods of time than a beverage with a lower buffering capacity, and can increase the exposure period in which dental erosion may develop (Meurman and ten Cate, 1996; Jensdottir *et al.*, 2005).

To determine the relevance of the combination of pH and buffering capacity for local packaged waters, FSANZ obtained chemical analysis data for nine brands of bottled mineral water sold in Australia (Water ECOscience, 2005). These data show that the waters with low pH (range 4.0 – 4.1) had very low levels of buffering ions relative to the higher pH waters (range 5.3 – 5.9). The levels of bicarbonate (an important buffering ion) were 20 to 130 times lower in the low pH waters compared to the higher pH waters. Levels of carbon dioxide were also much lower in the lower pH waters.

Because of the lower levels of chemical species (carbon dioxide and mineral ions) that can play a buffering role, the lower pH waters would have a much lower buffering capacity than the higher pH waters. The pH of local packaged waters with a pH less than 5.5 would therefore be expected to rise more markedly upon contact with a higher pH buffered solution (such as saliva) compared to the higher pH waters.

¹ The amount of base required to titrate to a state of neutral pH, also expressed as the amount of titratable acid.

6.3.2.2.2 Addition of fluoride to packaged water

Section 11 of the main report (Food Technology Considerations) discusses the potential reduction in water pH due to the addition of hydrofluorosilicic acid and the other two alternate forms of fluoride. This section reports that the very small amount of added hydrofluorosilicic acid that are required for a packaged water containing 0.6-1.0 mg fluoride/L will normally have very little, if any, effect on the pH of the water.

For waters with a low pH and a low buffering capacity, treatment with hydrofluorosilicic acid would be expected to lower the pH even further. This pH reduction will apply to a much lesser extent to the other two forms of fluoride under consideration for addition to packaged water. The addition of the proposed fluoride compounds to low pH packaged waters is unlikely to introduce a significantly greater amount of buffering ions, and thus is unlikely to substantially change the existing low buffering capacity of these waters.

6.3.2.2.3 Saliva

Saliva has the capacity to counteract acidic challenges that enter the oral cavity, such as those from beverages. As such, saliva may act as a protective factor against dental erosion. There are several methods by which saliva can protect against dental erosion (Meurman and ten Cate, 1996; Hara *et al.*, 2006; Dawes, 2008):

- The formation of a protective barrier (pellicle) on the tooth surface;
- The dilution and buffering of acids, which helps to prevent enamel demineralisation. Saliva contains a mixture of different buffering agents. In unstimulated saliva, a phosphate buffer system predominates while in stimulated saliva the carbonic acid-bicarbonate system is more important;
- Saliva is supersaturated with calcium and phosphorus, which slows the dissolution process of enamel and encourages enamel remineralisation following an acidic attack; and
- The flow of saliva, which dilutes and allows for the clearance of acidic substances from the mouth.

However, there are several caveats to this protective role for saliva. The pH of saliva is normally in the range of 6 to 7, and varies with salivary flow from 5.3 to 7.8. The flow of stimulated saliva is not high, with flow rates averaging from 1 to 3 mL per minute, and a residual volume of saliva in a normal adult at approximately 0.8 mL (de Almeida *et al.*, 2008). Available literature is also unclear on how the salivary flow rate changes in response to water, however evidence on hydration status suggests that even the saliva of a dehydrated person may not be stimulated by water consumption (de Almeida *et al.*, 2008).

In addition to these caveats, it should be noted that saliva is not a uniform substance, and can vary between individuals. A study by Wetton *et al.* (2007) suggests that saliva is not always able to protect against dental erosion in some individuals, although it should be noted that citric acid was used as a test medium rather than packaged water in this study.

The retention time in the oral cavity, and its exposure to saliva, is unlikely to be of significance for beverages with a low buffering capacity, as their pH is able to increase rapidly when in contact with salivary buffering agents. Retention time may be of greater importance for the dental erosion outcomes associated with beverages that have a high

buffering capacity, as these beverages can maintain a low pH in the oral environment for longer periods of time.

6.3.3 Conclusion on low pH packaged waters and dental erosion

FSANZ has been unable to locate suitable studies, including epidemiological studies, investigating the potential relationship between the pH of packaged water and dental erosion.

With regard to studies on other beverages such as fruit juices and carbonated soft drinks, correlations between the pH of those beverages alone and measures of dental erosion are often weak. The available evidence suggests that the erosive potential of these beverages depends on a complex interaction of factors including pH, buffering capacity, the presence of specific acids (e.g. citric acid in fruit juices and phosphoric acid in cola type drinks), and the overall mineral profile of the beverage. In addition, most of the published studies investigating the effect of beverages on dental erosion are *in vitro* studies and do not include saliva which has a protective buffering effect on teeth.

The packaged waters available in Australia with the lowest pH have a chemical composition that indicates a very low buffering capacity, and this buffering capacity is unlikely to change with the addition of fluoride. FSANZ considers that these lower pH, low buffering capacity packaged waters are therefore likely to have a negligible effect on the potential for tooth enamel erosion.

6.4 Cancer

It was raised in submissions to the Draft Assessment that long-term exposure to levels of fluoride, even as low as those found in Australian water, may cause cancer particularly bone cancer.

From a systematic review of the evidence for fluoridation, the studies assessing the relationship with cancer did not indicate that fluoride caused cancer (McDonagh *et al.*, 2000). Additional studies on cancer published after the McDonagh *et al* review were evaluated by the NHMRC as part of its systematic review (NHMRC, 2007b).. The NHMRC did not find any evidence to the contrary. The NHMRC concluded that the studies examining the association between fluoride and cancer provide insufficient evidence to reach a conclusion. These reviews included an assessment of the Bassin *et al.* (2006) osteosarcoma study which is often used as evidence that shows an association between fluoride exposure and osteosarcoma in teenage boys but not teenage girls.

6.5 Vulnerable sub-population groups

The issue of susceptible sub-population groups to intakes of fluoride was raised in submissions.

6.5.1 Infants

Infants, particularly those fed formula that is made up with fluoridated water, have often been described as an at risk group for exposure to fluoride. FSANZ included an evaluation of dietary intakes and risk for infants for this Application. See the main body of the report and the Dietary Intake Assessment report at Supporting Document 3 for further details.

Being breast fed was not associated with lower levels of fluorosis in one study in Western Australian children and weaning before 9 months of age was a significant risk factor for fluorosis ($TF \geq 1$) (Riordan, 1993a). Being formula fed as an infant was not associated with the prevalence of fluorosis in a 2002/2003 study in South Australian Children (Do and Spencer, 2007a).

Guidelines on the use of fluorides (Australian Research Centre for Population Oral Health, 2006) noted that there was no evidence from Australian population based studies to indicate levels of dental fluorosis from the consumption of infant formulas made up with either fluoridated or unfluoridated water.

6.5.2 Other at risk groups

An evaluation of the risk to other potential at risk groups such as high consumers of water (elite or endurance athletes and those working and/or living in hotter climates) was considered as part of the Draft Assessment Report based on data included in the Dietary Intake Assessment (in Supporting Document 3).

Pregnant women are assigned the same Nutrient Reference Values for fluoride as non-pregnant women, as they are thought to not have increased requirements or be at any greater risk of skeletal fluorosis. Therefore this group was not evaluated further.

6.6 Other health risks

Many potential adverse conditions or risks associated with fluoride were highlighted in submissions. These included associations with kidney disease, thyroid function (especially in the iodine deficient), those with brain disease, heart disease, neurological conditions (e.g. Alzheimer's dementia, lowered IQ in children, Parkinson's disease), goitre, birth defects, Down's Syndrome, kidney failure, arthritis.

No evidence of haematological, hepatic or renal effects has been found (World Health Organization, 2002). An association between fluoridated water and other effects (Down's Syndrome, mortality, dementia, goitre, IQ) was not found in a recent systematic review of water fluoridation (McDonagh *et al.*, 2000). A similar conclusion was reached following the systematic review conducted by the NHMRC (NHMRC, 2007b), that there is no evidence for the contribution of fluoride to any adverse effects other than fluorosis.

6.7 Conclusion regarding health risks

The only observed adverse effect in Australia and New Zealand from exposure to fluoride is very mild and mild dental fluorosis in children.

Other effects have not been proven (cancer, birth defects) or only occur in areas of extremely high fluoride content of water over many years (e.g. skeletal fluorosis), which are well above the concentrations in Australian or New Zealand water supplies. There is also no appreciable risk of dental erosion from low pH packaged waters (including those with added fluoride).

7. RISK VERSUS BENEFIT

The literature indicates that levels of fluoridation of around 1 mg/L will result in some fluorosis, usually very mild to mild in nature, but that the benefit of water fluoridation on dental caries outweighs the risk of fluorosis. This is also reflected in the advice and positions of government agencies and dental and health associations (see Table 7). Dental associations and experts also suggest a reduction in the exposure to fluoride from sources other than water fluoridation (e.g. toothpaste, supplements) as a means of lowering the incidence of fluorosis, while and retaining beneficial effects from increased fluoride intakes.

Available literature supports this position (Riordan, 1993a). Water fluoridation is seen as a cost effective measure (Wright *et al.*, 2001) in relation to dental caries and is socially equitable.

Table 7: Statements in relation to the risks and benefits of fluoridation

Agency	Statement	Reference
NHMRC public statement	‘Fluoridation of drinking water remains the most effective and socially equitable means of achieving community-wide exposure to the caries prevention effects of fluoride. It is recommended that water be fluoridated in the target range of 0.6 to 1.1 mg/L, depending on climate, to balance reduction of dental caries and occurrence of dental fluorosis.’	(NHMRC, 2007a)
Australian Dental Association (ADA) Policy 2007	The ADA recommends: ‘Water fluoridation as the most effective, equitable and efficient measure for achieving reduction in dental caries incidence across a community.’ The ADA recommends that the control of additional fluoride sources (e.g. toothpaste, supplements etc) should be undertaken to reduce levels of fluorosis, and not the reduction or removal of fluoridating drinking water which is the preferred way to maintain the low incidence of dental caries.	(Australian Dental Association Inc, 2007)
Australian Dental Association Queensland (ADAQ) – Position statement	‘ADAQ acknowledges that all sources of fluoride availability (toothpastes, mouth rinses, tables and water fluoridation) have the potential to contribute to varying levels of incidence and severity of dental fluorosis, a white mottled specking of tooth enamel. This is a condition of aesthetic concern only and may occur in a small number of individuals as a result of inappropriate exposure to fluorides during the period of development of the secondary dentition.’ ‘ADAQ endorses optimal community water fluoridation is the safest, most effective and most equitable means of improving the oral health of Queenslanders regardless of age, educational level or socio-economic situation.’	(Australian Dental Association (Queensland Branch), 2007)

Agency	Statement	Reference
Public Health Association of Australia – Oral Health Policy (2006)	‘Fluoridation of reticulated water supplies is the most effective socially equitable and safe method of prevention of dental caries.’ Supports the National Oral Health Plan 2004-2013 that ‘The fluoridation of reticulated water supplies should be continued and extended to all communities across Australia of 1000 or more people.’	Public Health Association of Australia (2006)
Public Health Association of New Zealand – Water Fluoridation Policy (2003)	‘The adjustment of fluoride to between 0.7 and 1.0 ppm in drinking water is the most effective and efficient way of preventing dental caries in communities receiving a reticulated water supply.’	Public Health Association of New Zealand (2003)

9. CONCLUSION FROM THE NUTRITION RISK ASSESSMENT

In summary it is concluded that reticulated and packaged waters are nutritionally equivalent. The permitted forms have been used for many years and are highly bioavailable from drinking water.

FSANZ therefore concludes that the benefits of water fluoridation in relation to the reduction of dental caries outweigh the risk of developing dental fluorosis. The prevalence of dental caries in children in Australia and New Zealand is increasing, which highlights the importance of continuing water fluoridation, which could include the provision of fluoridated packaged water. It is recognised that the fluoridation of water (including packaged water) will result in some mild dental fluorosis. There is evidence showing mild forms of fluorosis in Australian and New Zealand children.

Outside of contributions to mild dental fluorosis, the fluoridation of packaged water at levels permitted in Australia and New Zealand reticulated water supplies is unlikely to result in any other adverse health effects.

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APPENDIX 1

NEW ZEALAND DATA ON DENTAL CARIES FOR CHILDREN

Table 1: Dental Caries in New Zealand Children from School Dental Service data 1990-2006

Year	5 year old children		12 year old children*	
	Mean dmft	% Caries free	Mean dmft	% Caries free
1990	2.03	51	1.98	36
1991	2.04	52	1.72	42
1992	1.98	51	1.49	47
1993	1.92	53	1.39	49
1994	1.83	55	1.33	51
1995	1.71	55	1.40	48
1996	1.61	55	1.43	42
1997	1.72	57	1.62	44
1998	1.75	55	1.59	44
1999	1.80	54	1.58	44
2000	1.82	52	1.61	42
2001	1.87	51	1.61	43
2002	1.83	52	1.59	44
2003	2.06	54	1.57	45
2004	2.11	52	1.57	46
2005	2.24	52	1.67	44
2006	2.15	53	1.57	46

* Specified as Year 8 children 2002-2006 and Form 2 Children 2000-2001.

Source: Ministry of Health

COMMON RATING SCALES FOR DENTAL FLUOROSIS**Criteria for Dean's Fluorosis Index**

Score	Criteria
Normal	The enamel presents the usual translucent semi-vitriform type of structure. The surface is smooth, glossy, and usually of a pale creamy white colour.
Questionable	The enamel discloses slight aberrations from the translucency of normal enamel, ranging from a few white flecks to occasional white spots. This classification is utilized in those instances where a definite diagnosis of the mildest form of fluorosis is not warranted and a classification of 'normal' is not justified.
Very Mild	Small opaque, paper white areas scattered irregularly over the tooth but not involving as much as approximately 25 per cent of the tooth surface. Frequently included in this classification are teeth showing no more than about 1-2 mm of white opacity at the tip of the summit of the cusps of the bicuspid or second molars.
Mild	The white opaque areas in the enamel of the teeth are more extensive but do not involve as much as 50 per cent of the tooth.
Moderate	All enamel surfaces of the teeth are affected, and surfaces subject to attrition show wear. Brown stain is frequently a disfiguring feature.
Severe	Includes teeth formerly classified as 'moderately severe' and 'severe'. All enamel surfaces are affected and hypoplasia is so marked that the general form of the tooth may be affected. The major diagnostic sign of this classification is the discrete or confluent pitting. Brown stains are widespread and teeth often present a corroded-like appearance.

Source: (Dean, 1942)

Clinical Criteria and Scoring for the TF (Thylstrup-Fejerskov) Index

Score	Criteria
0	Normal translucency of enamel remains after prolonged air-drying.
1	Narrow white lines corresponding to the perikymata.
2	<i>Smooth surfaces:</i> More pronounced lines of opacity that follow the perikymata. Occasionally confluence of adjacent lines. <i>Occlusal surfaces:</i> Scattered areas of opacity <2 mm in diameter and pronounced opacity of cuspal ridges.
3	<i>Smooth surfaces:</i> Merging and irregular cloudy areas of opacity. Accentuated drawing of perikymata often visible between opacities. <i>Occlusal surfaces:</i> Confluent areas of marked opacity. Worn areas appear almost normal but usually circumscribed by a rim of opaque enamel.
4	<i>Smooth surfaces:</i> The entire surface exhibits marked opacity or appears chalky white. Parts of surface exposed to attrition appear less affected. <i>Occlusal surfaces:</i> Entire surface exhibits marked opacity. Attrition is often pronounced shortly after eruption.
5	<i>Smooth surfaces and occlusal surfaces:</i> Entire surface displays marked opacity with focal loss of outermost enamel (pits) <2 mm in diameter.
6	<i>Smooth surfaces:</i> Pits are regularly arranged in horizontal bands <2 mm in vertical extension. <i>Occlusal surfaces:</i> Confluent areas <3 mm in diameter exhibit loss of enamel. Marked attrition.
7	<i>Smooth surfaces:</i> Loss of outermost enamel in irregular areas involving <1/2 of entire surface. <i>Occlusal surfaces:</i> Changes in the morphology caused by merging pits and marked attrition.
8	<i>Smooth and occlusal surfaces:</i> Loss of outermost enamel involving >1/2 of surface.
9	<i>Smooth and occlusal surfaces:</i> Loss of main part of enamel with change in anatomic appearance of surface. Cervical rim of almost unaffected enamel is often noted.

Source: Thylstrup and Fejerskov, 1978. As reproduced in (NHMRC, 2007b)

Clinical Criteria and Scoring for the TSIF (Tooth surface index of fluorosis)

Score	Criteria
0	No evidence of fluorosis
1	Definite evidence of fluorosis
2	Parchment-white fluorosis on at least 1/3 visible surface
3	Parchment-white fluorosis on at least 2/3 visible surface
4	Enamel staining with any preceding levels fluorosis
5	Discrete enamel pitting
6	Discrete pitting and staining of intact enamel
7	Confluent pitting of enamel surface

Source: (NHMRC, 2007b)