



NFIS

National Fluoridation
Information Service

Dental fluorosis – is it more than an aesthetic concern?

National Fluoridation Information Service Advisory

Regional Public Health
Better Health For The Wellington Region



MASSEY UNIVERSITY
WELLINGTON



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National Fluoridation Information Service

The National Fluoridation Information Service (NFIS) is a consortium funded by the Ministry of Health, led by Regional Public Health working in partnership with:

- Hutt Valley DHB Community Dental Services,
- Environmental Science and Research,
- Centre for Public Health Research at Massey University and the
- National Poisons Centre.

Our work includes:

- Following public debate and choices on water fluoridation.
- Monitoring international research on the usefulness of water fluoridation.
- Critically reviewing emerging research.
- Working with District Health Boards and Councils to provide accurate and up-to-date information to their communities.
- Providing clinical advice to the Ministry of Health (MoH).
- Monitoring water fluoridation policy.
- Providing access to New Zealand oral health data and research.
- Sharing information via quarterly e-newsletters and e-briefings and the NFIS website.

GLOSSARY

- **Acute:** within a 24 hour period.
- **Adequate Intake:** the level of average daily intake estimated to give an individual an adequate amount of the nutrient.
- **Aesthetic:** relates to the appearance of the teeth.
- **Caries Experience:** the cumulative effect of the caries process through a person's lifetime, measured as teeth that are decayed, missing or filled (DMFT).
- **Central Incisors:** the two front upper and lower teeth.
- **CWF:** community water fluoridation, which involves adding low levels of fluoride (0.7-1.0mg/l) to reticulated water supplies to help prevent tooth decay. The levels of fluoride used in CWF in New Zealand are within the guidelines of the World Health Organization and other international public health agencies. The fluoride levels in areas with CWF are monitored regularly.
- **Deciduous Teeth:** first (or baby) teeth.
- **Demarcated opacities:** an alteration in the translucency of the enamel, but the thickness of the enamel is normal. It is marked out from adjacent normal enamel, with a distinct and clear boundary causing white, yellow or brown 'marks' on the teeth. These defects are usually due to a local cause such as previous decay and are generally more visible than diffuse enamel defects.
- **Dental fluorosis:** a condition of altered enamel formation caused by excessive intake of fluoride during tooth formation (Burt and Eklund, 2005), with a wide range of severity. Fluorosis is only one of a wide range of developmental defects that can occur in tooth enamel. Clinically, dental fluorosis is characterised by opaque white areas in the enamel in its milder forms, while more severe fluorosis can be characterised by brown stains or pitting (MoH, 2009).
- **Dentition:** the set of natural teeth. The adult dentition comprises 32 teeth, while the primary dentition comprises 20 teeth.

- **Diffuse opacities:** enamel defects that do not have a clearly defined border. They occur as opaque white areas in the enamel. Diffuse enamel defects are sometimes due to fluoride exposure (i.e. fluorosis), but can also be due to other factors such as early childhood fevers or malnutrition. The fluoride intake and medical history of a child with diffuse enamel defects needs to be assessed to determine whether they are due to fluorosis or other factors (Cutress and Suckling, 1990).
- **Discrete and confluent pitting of enamel:** brown stains that are widespread; teeth often present a corroded-like appearance.
- **dmft/DMFT:** An index of dental caries experience measured by counting the number of Decayed,(D) Missing(M) and Filled (F) Teeth (T), shown (in lower case) as dmft for first (baby) teeth and (in upper case) DMFT for permanent teeth.
- **Labial surface:** the surface of a front tooth facing the lips.
- **Maxillary incisors:** the four front upper teeth.
- **Maximum Acceptable Value (MAV):** the concentration of a determinand, below which the presence of the determinand does not result in any significant risk to a consumer over a lifetime of consumption.
- **Molar:** one of the twelve back teeth used in grinding food.
- **Upper level of intake (UL):** the highest average daily intake level likely to pose no adverse health effects to almost all individuals in the general population while meeting nutritional needs. Usual or routine intake of a nutrient above this level may place an individual at risk of adverse effects from excessive nutrient intake. The upper level for fluoride is set on the basis of moderate enamel fluorosis.

Introduction

Dental and skeletal fluorosis are the only two evidence-based effects that have been associated with extended periods of high fluoride intake from any source. While dental fluorosis is associated with high fluoride intake during the critical time of tooth development, skeletal fluorosis is associated with prolonged high intake over many years. Over recent years it has been suggested to local government councillors, and in many letters to the editor around New Zealand, that dental fluorosis is indicative of the toxic accumulation of fluoride within the human body. This claim is alarmist, and may be concerning to those new to the debate over the benefits and risks of community water fluoridation (CWF) in New Zealand, which involves adding low levels of fluoride (0.7-1.0mg/l) to reticulated water supplies to help prevent tooth decay.

As review of CWF programmes by councils around New Zealand is part of the on-going democratic process, it is important that clearly referenced coherent information is available on this topic to help councillors and ratepayers make informed decisions for their communities.

KEY SCIENTIFIC QUESTIONS

What is the relationship, if any, between severe dental fluorosis, skeletal fluorosis and CWF at the levels of 0.7 to 1.0 mg/L?

What evidence is there that dental fluorosis is an indicator of toxic fluoride accumulation in the body?

Methods

A literature review was carried out using the Pubmed database to identify key reports and papers about CWF and any adverse effects of fluoride, including fluorosis and the possible toxic accumulation of fluoride in the body. Specific papers and reports that were relevant to New Zealand were selected for review.

Key Findings

Evidence does not indicate there are any health risks associated with CWF at the levels of 0.7 to 1.0 mg/L in New Zealand, and no severe dental fluorosis, or skeletal fluorosis, has been found. While fluoride is incorporated into teeth and bones, there is no robust evidence of toxic accumulation of fluoride in other tissues in the body. CWF in New Zealand has been found to not lead to anything more than very mild or mild dental fluorosis for a small

number of people. Dental fluorosis is not a biomarker or indicator of skeletal fluorosis. Although they are both related to fluoride intake, dental and skeletal fluorosis are different biological processes.

REVIEW OF EVIDENCE

Background - Dental fluorosis in New Zealand

Fluoride Intake

Total fluoride intake (from all sources) is the most important consideration in dental fluorosis risk in New Zealand. Sources of fluoride, other than fluoridated water, include toothpaste, mouth rinses, fluoride varnishes, fluoride tablets, beverages made with fluoridated water, infant formula reconstituted with fluoridated water, some marine fish and other foods. Fluoride is also permitted as an addition to bottled water. Fluoride intake from most foods is low (National Health and Medical Research Council (NHMRC), 2006), with foods typically having a fluoride content in the range of 0.1 to 1.0 mg/kg (Cressey, Gaw & Love, 2010). Main fluoride intake from food is from common foods such as bread and potatoes, beverages (particularly tea, soft drinks, and beer), and fluoridated drinking water (Cressey et al., 2010). A study estimating fluoride intake in New Zealand found that it is unlikely that significant numbers of New Zealanders will have fluoride intakes above the recommended upper level and the risk of dental fluorosis in the general population is thought to be low (Cressey et al., 2010).

New Zealand has low levels of naturally occurring fluoride in water (Ministry for the Environment, 2014). CWF involves the controlled adjustment of fluoride in reticulated water supplies to bring fluoride concentration to a level that will help prevent dental caries while minimising the risk of dental fluorosis (MoH, 2010). In New Zealand it is recommended that fluoride levels in drinking water are adjusted to between 0.7 and 1.0 ppm (MoH, 2014a). Fluoridation of the water supply is recognised as one of the most cost effective public health methods of preventing dental caries (NFIS, 2013a). Fifty six per cent of the New Zealand population on reticulated water supplies have access to fluoridated water (MoH, 2014b). More information about areas of New Zealand with CWF can be found on the website of the Institute of Environmental Science and Research Limited (ESR), (ESR, 2014).

The current New Zealand guidelines for the use of toothpaste in young children recommend that a smear of regular strength (approximately 1000ppm) fluoride toothpaste is used until 5 years of age. From age 6 years, a pea-sized amount of regular strength fluoride toothpaste should be used (New Zealand Guidelines Group, 2009, p. vii). Children should be supervised

when brushing their teeth, and toothpaste should not be swallowed (New Zealand Guidelines Group, 2009).

Fluorosis

Fluorosis is the only evidence-based effect of extended periods of high fluoride intake; it manifests in two forms: dental and skeletal fluorosis. Dental fluorosis is the more common (Food Standards Australia New Zealand (FSANZ), 2009) and is discussed below. Skeletal fluorosis is discussed on page 8.

Dental Fluorosis

Dental fluorosis occurs due to an excess intake of fluoride during tooth formation. The severity of dental fluorosis is related to the duration, timing and dose of fluoride intake at the critical time when teeth are developing. The process of enamel formation occurs in the permanent dentition from around three to four months of age for central incisors with completion at 4 to 5 years. Initiation of the process for the remainder of the dentition, excluding second and third permanent molar teeth, commences at or after that of the central incisor teeth but in general the process for these teeth is completed between 6 and 7 years of age. There is a dose response relationship with more severe dental fluorosis found with higher levels of fluoride consumption (Burt, 1992). Milder forms of dental fluorosis result in opaque white areas in the tooth enamel while more severe forms are characterised by brown stains or pitting (MoH, 2010).

The mild and very mild forms of fluorosis that are seen in New Zealand are aesthetic in nature only, and have been shown to result in stronger and whiter teeth (FSANZ, 2009), while the more severe forms of dental fluorosis, which can produce adverse effects on dental health, are rare (FSANZ, 2009). Moderate fluorosis, when it does occur, is commonly attributed to the use of dental products containing high concentrations of fluoride, e.g. swallowing toothpaste, and not CWF (FSANZ, 2009). More severe forms of dental fluorosis are found in areas which have very high levels of fluoride occurring naturally in their drinking water, for example parts of China (Fawell et al., 2006).

Measuring Dental Fluorosis

The different types of dental fluorosis were first described by Dean (1934) as normal, questionable, very mild, mild, moderate, moderately severe and severe. Other indices have since been developed (for example, see Horowitz, Driscoll, Meyers, Heifetz, and Kingman, 1984; and Pendry, 1990). The Dean's index measures fluorosis by scoring the appearance of the second most severely affected tooth in the mouth. Mackay and Thomson (2005)

indicate that international research frequently uses Dean's index. In contrast, most New Zealand studies use the Developmental Defect of Enamel (DDE) index to record enamel defects (as described by Suckling, 1998; Mackay & Thomson, 2005). This method is not strictly a fluorosis index, but rather systematically records information on the nature, extent and position of any enamel defects.

Different indices investigating the prevalence of fluorosis may provide different results because they are examining for slightly different appearances on the teeth. For example, a comparison of Dean's index with the DDE index found that the two indices differed markedly in their estimates of the prevalence of fluorosis (Mohamed, Thomson & Mackay, 2010). Among 9 year old children from Southland, New Zealand, just over half the examined children had one or more enamel defects when the DDE index was used, in comparison to one in ten determined to have fluorosis when using Dean's index (Mohamed et al., 2010). The DDE does allow for a wider and more comprehensive range of information to be collected, but may have little agreement with other methods such as Dean's index, and this may hinder comparability with studies in other countries (Kanagaratnam, Schluter, Durward, Mahood, & Mackay, 2009).

CWF and rates of dental fluorosis in New Zealand

A New Zealand study (using the DDE index) that examined the prevalence of enamel defects and dental decay among 9 and 10 year old children in fluoridated and non-fluoridated areas of Southland (Mackay & Thomson, 2005), found that about half the children had at least one enamel defect. Demarcated opacities were the most common, 38.8%, while diffuse opacities affected almost one in four (24.1%). In the majority of cases (75.5%), the enamel defects were limited to less than one third of the labial surface of the affected teeth. Children who were continuous residents in communities with fluoridated water were found to be at a greater risk of having diffuse opacities (OR=2.23; 95% CI 1.37, 3.63); however, their dental caries experience (DMFS) was half that of children living in non-fluoridated areas (IRR=0.50; 95% CI 0.39, 0.65). The prevalence of demarcated opacities between children in fluoridated and non-fluoridated areas was found to be similar (38.7% and 38.8% respectively). The authors commented that the findings of this study were similar to previous New Zealand studies carried out in the 1980s, particularly in regards to prevalence of diffuse opacities among children in fluoridated or non-fluoridated areas, suggesting there has been no significant increase in dental fluorosis over that time.

Another New Zealand study also published in 2005 (Broadbent, Thomson & Williams), examined dental decay in primary teeth at age 5 and enamel defects in the following permanent teeth at age 9 among participants of the Dunedin Multidisciplinary Health and Development Study, a prospective cohort study of 1,037 children born in Dunedin between

1 April 1972 and 31 March 1973. Only enamel defects on the outer surfaces of the participants' maxillary incisors (the upper front teeth) were measured (using the DDE index). The study found that diffuse opacities were the most common defects among the participants (15.2%), while 6% of the participants had a demarcated opacity. Children who lived in a fluoridated area were found to be more likely to have a diffuse enamel defect at age 9 (adjusted OR 1.11, 95% CI 1.00, 1.23), while children who had decay in their upper front teeth at age 5 were found to be more likely to have a demarcated opacity in the following teeth at age 9 (adjusted OR=2.19, 95% 1.12-4.29). The authors suggested in their conclusion that while living in a fluoridated area increases the risk of diffuse opacities, CWF may indirectly help to prevent demarcated opacities as these were found to be associated with previous dental decay.

A more recent study among 9 year old children in Auckland examined whether or not CWF is associated with dental caries and enamel defects (Kanagaratnam et al., 2009). This study also used the DDE index to measure enamel defects. Among the 612 participating children in this study, diffuse opacities were found to be slightly more common (19%, n=117) than demarcated opacities (19%, n=115). The proportion of children with diffuse opacities was highest among those who had lived continuously in fluoridated areas (25%) and lowest among the children living continuously in non-fluoridated areas (11%) (adjusted OR=4.17; 95% CI 1.94, 8.94). However, caries experience (dmfs) in primary teeth was lowest amongst children from the fluoridated areas (51%) and highest amongst children in the non-fluoridated areas (67%) (adjusted OR=0.42; 95% CI 0.25, 0.70). Of the total teeth found with enamel defects (n=990), 25% had defects affecting less than one third of the labial surface. The authors concluded that CWF in Auckland has reduced dental caries, but is associated with a higher risk of diffuse opacities among 9 year old children. They also concluded that the prevalence and severity of diffuse opacities does not seem to have increased during the preceding 25 years (Kanagaratnam et al., 2009)

The 2009 New Zealand Oral Health Survey (2009 NZOHS) (MoH, 2010) examined the prevalence of fluorosis between fluoridated and non-fluoridated areas (using Dean's index). The prevalence of very mild dental fluorosis among 8-30 year olds was found to be the same in fluoridated and non-fluoridated areas (10.2% and 10.3% respectively). The prevalence of mild dental fluorosis was higher among those in the non-fluoridated areas (7.8%) than those in fluoridated areas (3%). Moderate fluorosis was found to occur in only 2.0% of 8-30 year olds, and virtually no one (0.0%) was found to have severe fluorosis. Adjusted results for fluorosis were not provided. The 2009 NZOHS also found that the dental caries experience (dmfs/DMFS) of children (aged 2-17 years) living in non-fluoridated areas was significantly higher than children living in fluoridated areas (3.9 and 2.5 respectively, adjusted ratio of means=1.7, p <0.05) (MoH, 2010).

All the above studies (except the 2009 NZOHS), found significant associations between living in a fluoridated area and an increased risk of diffuse opacities that are consistent with mild or very mild dental fluorosis. In addition, all the studies (including the 2009 NZOHS), found that there was significantly lower dental decay experience among children living in fluoridated areas than among those in non-fluoridated areas. It appears that the prevalence of dental fluorosis has not increased over the last two decades.

SCIENTIFIC QUESTIONS

What is the relationship, if any, between severe dental fluorosis, skeletal fluorosis and CWF at the levels of 0.7 to 1.0 mg/L?

What evidence is there that dental fluorosis is an indicator of toxic fluoride accumulation in the body?

Fluoride is readily incorporated into calcified tissues, such as bone and teeth. Most of the fluoride in the body, about 99%, is contained in the bones (National Research Council, 2006). Chronic fluoride intoxication can occur after ingesting excessive quantities of fluoride through drinking water with very high fluoride content over a prolonged period of time. This results in skeletal fluorosis, which causes the development of lesions and adverse changes in the bone structure (Perumal, Paul, Govindarajan, & Panneerselvam, 2013). Dental fluorosis, on the other hand, can only develop as a result of fluoride exposure at a critical time during tooth development.

Skeletal fluorosis

Skeletal fluorosis is a serious medical condition, and can lead to significant bone degradation and adverse neurological manifestations (FSANZ, 2009). In the FSANZ risk assessment for the addition of fluoride to bottled water, no evidence was identified of more severe forms of fluorosis, including skeletal fluorosis, attributable to CWF in New Zealand (FSANZ, 2009).

Endemic skeletal fluorosis is well documented, and is known to occur with a range of severity in several parts of the world, including India, China and northern, eastern, central and southern Africa (Fawell et al., 2006). Recent research has identified skeletal fluorosis as a public health problem in parts of Ethiopia (Melaku, Assefa, Enqusilassie, Bjorvatn, & Tekle-Haimanot., 2012), West Bengal (Majumdar, 2011), Brazil (de Souza et al., 2013) and Tanzania (Jarvis et al., 2013). In these regions, naturally occurring fluoride levels in drinking

water can be very high. The Brazilian study, for example, found that fluoride concentration in the water varied from 0.11 to 9.33 mg/L, with 30% of all samples analysed showing values above 1.5 mg/L (de Souza et al., 2013), the internationally recognized maximum acceptable value for CWF. In Tanzania, the fluoride levels in well water have been found to be very high, ranging from 9.3 to 35 mg/L, with surface water sources containing fluoride in the range of 2.1 to 9.5 mg/L (Jarvis et al., 2013).

It was estimated by the National Research Council (1997) that based on epidemiological research, an intake of at least 10 mg/day of fluoride for at least 10 years is required before the clinical symptoms of skeletal fluorosis occur. A further report suggested that there is clear evidence from India and China that skeletal fluorosis and increased bone fractures occur at total intakes of 14 mg fluoride/day and suggestive evidence of an increased risk of effects on the skeleton at total intakes above about 6 mg fluoride/day (International Programme on Chemical Safety (IPCS), 2002).

By way of comparison, concentrations of fluoride in public water supplies in New Zealand average around 0.8 to 0.9mg/L in areas with CWF, and around 0.15mg/L in non-fluoridated areas (FSANZ, 2009). The Maximum Acceptable Value of fluoride in drinking water in New Zealand is 1.5 mg/L, which is much lower than the levels that occur in areas of the world where skeletal fluorosis is prevalent.

Although severe dental fluorosis and skeletal fluorosis are associated with high total fluoride intake, skeletal fluorosis has not been reported in New Zealand in communities whether or not they are receiving fluoridated or non-fluoridated water.

Fluorosis in other body tissues

Case reports and *in vitro* and animal studies have indicated that exposure to fluoride at concentrations greater than 4 mg/L can be irritating to the gastrointestinal system, can affect renal tissues and function, and can alter hepatic and immunologic parameters (National Research Council, 2006). However, there is no robust evidence that fluoride intakes at the level of CWF in New Zealand (0.7 to 1.0 mg/L) will lead to toxic effects in other body tissues.

In addition, it is impossible to experience acute fluoride toxicity from drinking water optimally fluoridated at levels between 0.7 to 1 mg/L (MoH, 2009). NFIS (2013b) have estimated that a probable acute toxic dose ranges from 140 glasses of water a day for infants aged 0-6 months, to 1,520 glasses of water a day for men aged over 19 years.

Effects of dental fluorosis

Given that the more severe forms of dental fluorosis have **not** been found in New Zealand (MoH, 2010), the key concern is whether the aesthetic impact of mild or very mild levels of dental fluorosis has any adverse effect on quality of life, such as, embarrassment, unhappiness with appearance, or hindering people smiling.

A literature review (Chankanka, Levy, Warren, & Chalmers, 2010) assessed studies investigating the relationships between aesthetic perceptions of dental fluorosis and oral health related quality of life. The authors commented that the increased concern about dental fluorosis prevention have led to some infants at risk of caries possibly not being given enough fluoride for caries prevention. In addition, the authors pointed out that because dental fluorosis is not a condition that causes pain or has clinical symptoms, reports of prevalence and severity alone do not provide enough information to understand the potential public health impacts of dental fluorosis. The authors therefore reviewed the findings of studies that have investigated the relationship between dental fluorosis, aesthetic perceptions and quality of life.

Results from this review showed that mild and very mild fluorosis were not associated with negative effects on oral health related quality of life, and there was some evidence that mild fluorosis is associated with an enhanced oral health related quality of life (Chankanka et al., 2010). However, severe fluorosis was reported less favourably. The authors state that fluorosis cases occurring in the USA, and other nations without high levels of naturally occurring fluoride in the water supplies, are mainly of the very mild and mild types found not to have any adverse effects on oral health related quality of life. Therefore, the study recommended that dental professionals should focus on balancing the need for appropriate use of fluoride to prevent dental caries, while preventing the occurrence of moderate and severe types of fluorosis (Chankanka et al., 2010.).

Conclusion from evidence

There are no known health risks associated with CWF in New Zealand, and no severe dental fluorosis, or skeletal fluorosis, has been found. While fluoride is incorporated into teeth and bones, there is no robust evidence of toxic accumulation of fluoride in other tissues in the body.

Overall, recent studies of enamel defects in New Zealand show that those living in areas with CWF are more likely to have diffuse opacities and less dental decay than those living in areas without CWF (Kanagaratnam et al., 2009; Mackay & Thomson, 2005; Broadbent, et al. 2005). Fluoride intake estimates indicate it is unlikely that significant numbers of New

Zealanders will have fluoride intakes above the recommended upper level (Cressey et al., 2010). In New Zealand, CWF has been found to not lead to anything more than very mild or mild dental fluorosis for a small number of people (FSANZ, 2009).

As with many vitamins and minerals, such as iron, and vitamins A and D, fluoride intakes at high levels can be toxic. However, it is impossible to experience acute fluoride toxicity from drinking water optimally fluoridated at levels between 0.7 mg/L to 1.0 mg/L (MoH, 2009), and there is no evidence of skeletal fluorosis resulting from CWF in New Zealand. It makes sound clinical sense to ingest a substance at a level that achieves maximum benefit with minimal adverse effects (Bowen, 2002).

IMPLICATIONS FOR NEW ZEALAND

What implications, if any, can be concluded from the evidence for

a. The MoH's current CWF policy

Evidence does not indicate that there is a link between CWF at the levels of 0.7 to 1.0 mg/L, and the occurrence of severe dental fluorosis or skeletal fluorosis in New Zealand.

Evidence does not indicate that fluoride accumulates to toxic levels in body tissues with CWF at the levels of 0.7 to 1.0 mg/L.

Each year ESR prepares a report for the Ministry of Health on New Zealand's drinking-water quality. This records whether fluoridating water supplies have complied with the Drinking-water Standards for New Zealand. The latest report on drinking water quality found there were no fluoride exceedances in water leaving any fluoridating treatment plant in New Zealand (MoH, 2014).

b. Research needs within the NZ context

It is important that the prevalence, severity and aesthetic impact of fluorosis continues to be monitored in both fluoridated and non-fluoridated areas of New Zealand. In particular, the impact of different sources of fluoride should be investigated, with on-going recommendations made in relation to guidelines on total fluoride use (Kanagaratnam et al., 2009).

Future research on the oral health of the New Zealand population will enable comparisons to be made with existing data in relation to the impact of CWF on the condition and retention of teeth within different population groups, and on any adverse effects such as the prevalence and severity of fluorosis.

c. Research needs within the international context

Internationally, there is also a need for on-going research on dental and skeletal fluorosis, particularly in those parts of the world where populations are exposed to very high levels of naturally occurring fluoride since this can lead to severe dental and skeletal fluorosis. Providing clean, piped optimally-fluoridated water to such communities would provide obvious health benefits (Jarvis et al., 2013).

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