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Exposure to water fluoridation and caries increment

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Objective The objective of this cohort study was to examine the association between exposure to water fluoridation and the increment of dental caries in two Australian states: Queensland (Qld) – 5 per cent fluoridation coverage; and South Australia (SA) - 70 per cent fluoridation coverage. ***Method*** Stratified random samples were drawn from fluoridated Adelaide and the largely non-fluoridated rest-of-state in SA, and fluoridated Townsville and non-fluoridated Brisbane in Qld. ***Participants*** Children were enrolled between 1991 and 1992 (SA: 5-15 yrs old, n=9,980; Qld: 5-12 yrs old, n=10,695). Follow-up caries status data for 3 years (± ½ year) were available on 8,183 children in SA and 6,711 children in Qld. ***Main outcome measures*** Baseline data on lifetime exposure to fluoridated water, use of other fluorides and socio-economic status (SES) were collected by questionnaire, and tooth surface caries status by dental examinations in school dental service clinics. ***Results*** Higher per cent lifetime exposure to fluoridated water (6 categories: 0;1–24;25–49;50–74;75–99;100 per cent) was a significant predictor (ANOVA, p<0.01) of lower annualised Net Caries Increment (NCI) for the deciduous dentition in SA and Qld, but only for Qld in the permanent dentition. These associations persisted in multiple linear regression analyses controlling for age, gender, exposure to other fluorides and SES (p<0.05). ***Conclusions*** Water fluoridation was effective in reducing caries increment, even in the presence of a dilution effect from other fluorides. The effect of fluoridated water consumption was strongest in the deciduous dentition and where diffusion of food and beverages from fluoridated to non-fluoridated areas was less likely.

Keywords: Children, dental caries; fluoridation

Introduction

By the mid 1990s, the prevalence of dental caries in Australian children had decreased to its lowest level since WWII. In 1996, fewer than half of all children had any clinical caries experience in either their deciduous or permanent teeth and most children with experience of clinical caries had only one or two teeth affected in either dentition. Currently, only a small minority of children experience higher levels of caries experience, four or more teeth affected in either dentition, although their proportion of all disease experienced is high (Armfield et al., 2003).

The improvement in caries experience in Australia has been largely attributed to the use of a number of fluoride vehicles (NHMRC, 1991). Water fluoridation, for example, was introduced in Beaconsfield, Tasmania in 1953. By the late 1960s and early 1970s, most of the larger capital cities of states and territories initiated water fluoridation, with coverage of the Australian population reaching 66 per cent by 1978 (Spencer, 1984). Only Brisbane, of all the Australian capital cities, remained non-fluoridated.

Fluoride toothpaste was tested in a clinical trial in Adelaide, South Australia in the 1960s and by the 1970s market-share of fluoride toothpaste in Australia exceeded 95 per cent. Fluoride supplements, although promoted in non-fluoridated areas, were used regularly only by a minority of children (Spencer, 1986b). School dental services provided professionally applied fluorides but the rate of provision of these services has greatly slowed and was more targeted from the 1980s.

An ecological study using data available across 1965 to 1978 examined the association of proportion of lifetime exposure to water fluoridation, market share of fluoride toothpaste and regular use of fluoride supplements with caries experience in adolescents (Spencer, 1986a). The strongest association was with exposure to water fluoridation, followed by fluoride supplements, then fluoride toothpaste. When these associations were used to retrospectively predict caries experience, exposure to water fluoridation contributed most to the decline in caries experience, followed by fluoride toothpaste, then fluoride supplements (Spencer, 1986b).

Such ecological studies provide only one level of evidence on the role of fluorides in caries prevention. It has been more common to base claims of the effectiveness of water fluoridation on cross-sectional comparisons with either historical or concurrent controls. The Canberra, Tamworth, Perth and Townsville studies in Australia in the 1970s represent the use of historical controls, while later studies in Perth and Bunbury in Western Australia and Melbourne and Geelong in Victoria, represent the use of concurrent controls.

The evidence-base for the effectiveness of water fluoridation in caries protection has also varied considerably in terms of focus on measures of exposure. Early analyses mention continuous residence in the fluoridated and/or non-fluoridated community. A trend in more recent analyses has been to include some categorisation of exposure across the subject's lifetime. Brown et al. (1990) categorised lifetime exposure to fluoridated water in thirds of a lifetime. Riordan (1991) categorised exposure from birth to four years and four to 12 years of

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age as either short-, medium- or long-term within each separate period.

The separation of continuous residents for analysis is becoming increasingly problematic in the highly mobile communities in which people now live. An alternative approach may therefore be to place individual exposure to water fluoridation along a gradient. The ecological study by Spencer (1986a) had introduced the use of per cent lifetime exposure to water fluoridation. It was found to be strongly associated with caries experience and the nature of the exposure variable helped explain the lagged effect of caries reductions. Stockwell et al. (1990) computed a per cent lifetime measure of residence in the fluoridated or non-fluoridated sites for their research.

A similar approach to calculating exposure to fluoridated water was used in a large Australian study, the Child Fluoride Study (Slade et al., 1995; 1996), where individual per cent lifetime exposure to water fluoridation was found to be associated with caries experience. This study indicated that the benefit of exposure to water fluoridation was more pronounced in the deciduous than permanent teeth, that reductions in caries are relatively small in absolute terms, and that exposure to water fluoridation explains rather little of the variation observed in caries experience in children (Locker, 1999). Slade et al. (1995) speculated that differences between study sites in the strength of association between fluoride and caries experience may have been due to a 'halo effect', with the spread of fluoride from fluoridated to non-fluoridated communities occurring via the medium of food and beverages produced in fluoridated areas. The Child Fluoride Study included one Australian state, Queensland, where only five per cent of the population consumed optimally fluoridated water, thus minimising the potential for a halo effect.

The Child Fluoride Study has added weight to evidence available to support the effectiveness of water fluoridation in the 1990s. There has been an emphasis on individual exposure measurement, control for confounders and surface level observation of caries experience. However, the finding of a statistically significant association in that particular study does not establish a casual relationship. In terms of the general criteria formalised by Hill (1971) for assessing the extent to which available evidence supports a casual relationship there is a temporal ambiguity. This has been a common occurrence in ecological research studies on the effectiveness of water fluoridation.

Few prospective cohort studies have been published on exposure to water fluoridation and caries development in children. Hardwick et al. (1982) analysed the caries increment over three years in children aged 12 at the start of water fluoridation. They found a statistically significant lower increment in DMFT and DMFS score in the fluoridated area compared with the non-fluoridated area after the three-year period. Systematic reviews like that by the University of York found no further evidence on the issue of exposure to water fluoridation and caries incidence or increment in children (McDonagh et al., 2000).

The purpose of the present study was to investigate the effectiveness of water fluoridation in a cohort of contemporary Australian children using a longitudinal analysis in a cohort study design. The specific aim was to examine the association between exposure to water fluoridation and the increment of dental caries in two Australian states: Queensland (Qld) with five per cent fluoridation coverage; and South Australia (SA) with 70 per cent fluoridation coverage.

Methods

This paper reports on the longitudinal findings of a cohort study designed to examine exposure to water fluoridation and dental caries in Australian children. The Child Fluoride Study was initiated in 1991 and this report includes data collected across the 1991 to 1995 period. Results from cross-sectional analyses of baseline data in this study have been reported previously (Slade et al., 1995).

The Child Fluoride Study was designed as a multi-site cohort study, with two contrasting states of Australia, so as to improve the generalisability of the results. In SA, over 70 per cent of the population of 1.5 million people is served by fluoridated water. All residents of Adelaide, the state capital of 1.1 million people were served by fluoridated water. Most residents in the rest-of-state were served by non-fluoridated water. In Qld, only a small number of regional cities and towns are served by fluoridated water. The major provincial city served by fluoridated water was Townsville, population of 87,274 in 1991. A total of 5.1 per cent of the state's 3.1 million residents live in fluoridated areas. The capital city Brisbane, population of 1.4 million in 1991, was non-fluoridated. Across both non-fluoridated strata natural fluoride levels in water supplies were negligible.

Source of subjects and sampling

The sampling frame included all children enrolled in the school dental service (SDS) of SA and in Townsville and Brisbane, Qld who received an examination between June 1991 and May 1992. The SDS provided regular dental care to over 85 per cent of the age groups included in both states and at the time of enrolment the care was provided at no cost to families.

In SA a stratified random sample of subjects aged 5 to 15 years was selected from two strata: Adelaide, where the sampling ratio was 1:12 children, and rest-of-state, where the sampling ratio was 1:5 children. In Qld a stratified random sample of subjects aged 5 to 12 years was selected from two strata: Townsville, where the sampling ratio was 1:1 children and Brisbane where the sampling ratio was 1:5 children. This sampling strategy was adopted to provide approximately equivalent numbers of children in each of the two states and for both strata within each state. While the strata were basically comprised of a fluoridated and non-fluoridated stratum, it was recognised that individual residential history would vary. Children were therefore subsequently classified by exposure to water fluoridation based on their residential history. Variation in the age range reflected the effective age at which the SDS in each state ceased provision of school-based dental care and the oldest age for which three years of follow-up care was potentially available.

The target sample size was set on the assumption that the three-year increment in caries was 1.0 DMFS

with a standard deviation of 3.0. It was thought necessary to detect a mean difference of 0.15 DMFS (15 per cent) between lifetime versus no exposure to fluoridated water groups. Type I and II error was set at 0.05. This created a requirement for 17,212 subjects at completion of the 3 years.

Caries experience data were collected by SDS staff, including dentists and dental therapists. Written instructions were provided to the staff concerning criteria for the dmfs and DMFS index based on WHO (1987) criteria for the diagnosis of caries and US NIDR (1987) criteria for surface demarcation.

In the primary dentition, additional guidelines were used to distinguish between teeth missing due to caries and teeth that were exfoliated. In view of the large number of SDS staff involved in two states, there were no additional procedures for standardising examiners or assessing reliability through replicate examinations. The effectiveness of water fluoridation as examined in the present study therefore reflected the perspective of the clinical staff who diagnose and manage most caries experience in Australian children.

At the time of sampling, parents were invited to take part in the study and complete a questionnaire. The questionnaire asked about the child's residential history and sources of drinking water, use of other fluoride vehicles and household characteristics (educational attainment, occupation, pre-tax annual household income). Up to two reminder notices were sent to parents who did not respond to the questionnaire.

Ethical approval and informed consent

The Child Fluoride Study was approved by The University of Adelaide Human Research Ethics Committee. Informed positive consent was obtained from parents at the time of their completion of the parental questionnaire. Parents gave consent to access to their child's oral examination data from the SDS at baseline and across the follow-up period.

Exposure to fluoride in water was calculated using responses to questions on residential history and sources of drinking water. Parents were asked to list each suburb, town or location in which the child had lived for longer than six months, the years in which the child had lived at that place and whether the child usually drank water from the mains water or another source.

An electronic database of Australian postal codes and their fluoridation status was established and linked to postal codes of residence at data entry. Overseas locations were individually coded for their fluoridation status. Fluoridation status was categorised in this database as less than 0.3 ppm F (imputed as 0.0 ppm F) 0.3 to less than 0.7 ppm F (imputed as 0.5 ppm F) and 0.7 ppm F and above (imputed as 1.0 ppm F). In the two fluoridated strata included in the sampling the fluoride concentration was 0.9 ppm in Adelaide and 0.7 ppm in Townsville.

The number of years spent by a child at each residential location was multiplied by the imputed fluoride concentration for that locality during the period of residence. If the public water supply was not the usual source of drinking water several adjustments were made on the assumption that tanks, bores and bottled water had fluoride concentrations of less than 0.3 ppm F and the proportion of total fluoride intake from the substitution of these non-public water supplies would diminish fluoride exposures (NHMRC, 1991). The product of years of residency and imputed fluoride concentration was summed for all residential locations listed for a child. The figure was expressed as a rate by dividing by the total number of documented years of residency and is referred to as the per cent lifetime exposure to 1 ppm F water.

There was an underlying assumption that for a specific child the exposure during any short period, up to 6 months, of unknown exposure occurred at the same level as the known years of exposure. For a limited number of children the fluoride concentration of a postal code or overseas location was unknown, or a gap of greater than two years existed in the residential history. Such children were excluded from the analyses due to missing fluoride exposure data.

All the clinical records of children enrolled and examined at baseline were flagged so that at routine recall courses of care at the SDS the children would again be examined and data recorded on the supplied forms. The number of courses of care determined the number of follow-up oral examinations across the subsequent three and a half year period. Children were recalled at different intervals in the SDS depending on their level of risk judged by clinical staff. For the present study only the baseline and last oral examination in the subsequent three and a half year period were used for the analysis of caries increment. If a child had a follow-up oral examination one year into the follow-up period and then was lost to the study, then this last examination was used in the calculation of caries increment.

Eligibility for SDS care is age dependent. Older children at baseline therefore had set periods of time, varying with their age, that they would remain eligible for the SDS and in the study.

The dependent variable in this study was derived from the tooth surface data from the baseline and last follow-up oral examination and was annualised to obtain caries increment per year. Increment was calculated on a surface-by-surface basis where changes or events were mapped out in a grid (DePaola, 1990; see Table 1). The same principles applied to both the deciduous and permanent teeth. Net caries increment (NCI) involved increments (I) at the follow-up examination (T_2) that follow a baseline (T_1) tooth surface status of either unerupted (U), sound (S) or fissure sealed (FS) as well as increments associated with filled surfaces at baseline (T_1), either filled unsatisfactory (F_{UN}) or filled (F), progressing to decay (F_{RC} or D) at follow-up (T_2). Caries increment excluded teeth missing teeth due to reasons other than caries. However, NCI subtracted examiner reversals. These reversals included any filled or decayed surface (F_{UN}, F, F_{RC} or D) at baseline (T_1) that changed to sound (S) or fissure sealed (FS) at follow-up (T_2).

As the study children included those with a mixed dentition, all deciduous teeth exfoliated at follow-up were considered not to have any increment from their baseline status.

Table 1. Classification of events in tooth surface diagnosis between baseline and follow-up examinations.

(Baseline) T_1	Follow-up (T_2)								
	U	*S*	M_O	*FS*	F_{UN}	*F*	F_{RC}	*D*	M_C
U	NP	NP	NP	NP	I	I	I	I	I
S	E	NP	NP	NP	I	I	I	I	I
M_O	E	E	NP	E	E	E	E	E	E
FS	E	NP	NP	NP	I	I	I	I	I
F_{UN}	E	R	NP	R	NP	NP	I	I	I
F	E	R	NP	R	NP	NP	I	I	I
F_{RC}	E	R	NP	R	NP	NP	NP	NP	NP
D	E	R	NP	R	NP	NP	NP	NP	NP
M_C	E	R_E	E	R_E	E	E	E	E	NP

Note: U = Unerupted; S = Sound; M_O = Missing (due to reasons other than caries); FS = Fissure Seal; F_{UN} = Filled (unsatisfactory); F = Filled; F_{RC} = Filled (recurrent caries); D = Decayed; M_C = Missing (due to caries). NP = Non-progression of caries; I = Increment; R = Reversal; E = Error; R_E = Reversal (erroneous).

= Events included in Net Caries Incidence (NCI)

Caries increment was calculated separately for deciduous teeth for those aged 5–7 at baseline and permanent teeth for those 6–12 in Qld and 6–15 in SA. Annualised caries increment was calculated by dividing the increment measure for each child by the number of years between baseline and the last follow-up examination for the respective child.

Weighting of data

All data were weighted before the analyses to adjust for the different sampling ratios.

Results

There were 13,911 children sampled for the study in SA. A total of 9,988 children participated at baseline (71.8%) with oral examination data available for 9,714 of these children. There were 8,183 children (84.2% of baseline participants) who had at least one follow-up oral examination across the three-year period. In Qld 18,348 children were sampled. A total of 10,695 children participated at baseline (58.3%) and 6,711 (62.7% of baseline participants) children had at least one follow-up oral examination across the three-year period.

Descriptive statistics and bivariate analyses

Table 2 presents a comparison of characteristics of baseline participants who were lost to follow-up and those participants who had at least one follow-up examination in SA and Qld. Significant differences were observed between children lost to follow-up and those involved in follow-up examinations for age, parental education, family income, occupational prestige, number of residences and fluoridated water status in both states. Children lost to follow-up were older and more residentially mobile in both states. In SA children lost to follow-up were more likely to be from the extremes of the socio-economic status variables while children lost to follow-up from Qld were more likely to be merely from low socio-economic status groups. In both states, children lost to follow-up were more likely to be from non-fluoridated areas.

The distribution of lifetime exposure to water fluoridation was substantially different between the two states. Table 3 presents the per cent lifetime exposure to optimally fluoridated water for children with follow-up data. Among the SA children, less than 10% had 0% lifetime exposure. Over one third had 100% lifetime exposure. Other children were distributed predominantly across the 25–49, 50–74 and 75–99 per cent lifetime exposure groups. In contrast, over two-thirds of the children in Qld had 0% lifetime exposure and all remaining children were evenly spread across the five higher exposure groups. Only 5% of Qld children had 100% lifetime exposure.

Table 4 presents the mean deciduous and permanent caries experience at baseline for those who were continuous participants and those who were lost to follow-up, and the follow-up caries experience for the continuous subjects in SA and Qld. Among SA baseline participants, those lost to follow-up had a significantly lower dmfs, driven by their significantly lower number of filled surfaces. No significant differences existed in deciduous caries experience between Qld baseline participants who were lost or continued through to follow-up. At follow-up the deciduous caries experience of both the SA and Qld participants had significantly decreased, with decreases in all components of their caries experience. This apparent paradox reflects the exfoliation of deciduous teeth with past caries experience at the baseline 5–7 year olds age. It was for this reason that subsequent analyses using caries increment were limited to teeth that were not exfoliated during the period of follow-up. Of course if the exfoliation of deciduous teeth with surfaces with past caries experience exceeds the increment of new caries in those that remain, then deciduous caries experience of the children followed can decrease while these same children have a positive net caries increment across the follow-up period.

Permanent caries experience showed different trends. Those subjects lost to follow-up had significantly higher permanent caries experience compared to subjects who had at least one follow-up, predominantly due to their significantly higher number of filled surfaces. Those lost

Table 2. Characteristics of children at baseline who were lost to follow-up and those children retained for follow-up (weighted)

	South Australia		*Queensland*	
	Baseline lost to follow-up	*Follow-up*	*Baseline lost to follow-up*	*Follow-up*
Number of children	1,613	8,187	3,984	6,711
Age at baseline (yrs)	10.7	9.3	9.1	8.0
	$F = 307.81$, $p < 0.001$		$F = 739.33$, $p < 0.001$	
Sex (% male)	52.0	51.0	51.3	50.7
	$\chi^2 = 0.53$, $p > .05$		$\chi^2 = 0.09$, $p > 0.05$	
Parental education (%)				
Not completed secondary	29.3	28.3	26.4	23.0
Completed secondary	31.4	35.3	34.3	32.6
Some university/college	10.7	11.3	12.4	13.1
Completed university/college	25.6	22.2	23.9	28.3
Missing	3.0	2.9	3.0	2.8
	$\chi^2 = 13.24$, $p = 0.004$		$\chi^2 = 20.10$, $p < 0.001$	
Family income				
Up to $20,000	26.8	22.8	22.5	17.6
$20,001 to $30,000	19.7	23.3	21.7	22.5
$30,001 to $40,000	18.1	19.9	21.1	22.1
$40,001 to $50,000	11.7	11.5	12.0	13.1
$50,001 and over	15.4	13.9	15.4	17.2
Missing	8.3	8.6	7.3	7.5
	$\chi^2 = 21.97$, $p < 0.001$		$\chi^2 = 37.62$, $p < 0.001$	
Occupational prestige				
No usual occupation	3.0	1.9	2.3	1.7
56 to 69 (lowest)	14.3	15.3	16.4	13.6
42 to 55	31.7	33.9	35.0	34.4
28 to 41	37.7	37.8	31.7	35.4
12 to 27 (highest)	8.2	6.5	8.4	9.9
Missing	5.0	4.7	6.1	5.0
	$\chi^2 = 16.84$, $p < 0.001$		$\chi^2 = 24.26$, $p < 0.001$	
Residences				
1 residence	35.9	45.9	26.8	35.9
2 residences	26.0	25.0	25.4	27.4
3 residences	15.3	14.9	20.9	18.0
4+ residences	22.7	15.6	26.6	18.5
Missing	0.0	2.3	0.4	0.3
	$\chi^2 = 97.29$, $p < 0.001$		$\chi^2 = 90.38$, $p < 0.001$	
Fluoridation				
Fluoridated	11.2	14.9	87.1	90.8
Part fluoridated	9.1	10.8	0.0	0.0
Not fluoridated	79.7	74.2	12.9	9.2
	$\chi^2 = 22.52$, $p < 0.001$		$\chi^2 = 36.76$, $p < 0.001$	

Table 3. Per cent lifetime exposure to optimally fluoridated water for South Australia and Queensland children retained for follow-up (weighted).

Percent lifetime F exposure	*South Australia*		*Queensland*	
	n	*%*	*n*	*%*
0%	799	9.8	4,639	69.1
1 to 24%	291	3.6	497	7.4
25 to 49%	962	11.8	463	7.0
50 to 74%	1,795	21.9	426	6.3
75 to 99%	1,555	19.0	323	4.8
100%	2,771	33.8	340	5.1
Missing	14	0.2	16	0.2
Total	8,187	100.0	6,704	100.0

Table 4. Mean (SD) number of deciduous and permanent decayed, missing and filled surfaces among South Australian and Queensland children at baseline and follow-up (weighted).

	South Australia						*Queensland*					
	Baseline lost to follow-up		*Baseline followed-up*		*Follow-up*		*Baseline lost to follow-up*		*Baseline followed-up*		*Follow-up*	
	(n = 753)		*(n = 5,252)*		*(n = 5,252)*		*(n = 2,076)*		*(n = 5,891)*		*(n = 5,891)*	
dmfs	2.84	(4.89)	3.29	(5.52)	2.37	(4.30)	3.63	(5.63)	3.62	(5.93)	2.94	(4.97)
d	0.67	(1.97)	0.58	(1.50)	0.37	(1.02)	0.85	(2.27)	0.73	(2.14)	0.42	(1.31)
m	0.09	(1.06)	0.20	(1.87)	0.10	(1.07)	0.27	(1.78)	0.24	(1.80)	0.08	(0.83)
f	2.08	(3.98)	2.51	(4.46)	1.91	(3.68)	2.51	(4.23)	2.65	(4.56)	2.44	(4.29)
	(n = 1,453)		*(n = 7,241)*		*(n = 7,241)*		*(n = 3,262)*		*(n = 5,969)*		*(n = 5,969)*	
dmfs	1.28	(2.65)	0.75	(1.67)	1.01	(1.95)	0.91	(1.88)	0.52	(1.35)	0.95	(1.87)
d	0.15	(0.60)	0.12	(0.46)	0.17	(0.58)	0.24	(0.74)	0.16	(0.59)	0.26	(0.75)
m	0.04	(0.75)	0.02	(0.34)	0.01	(0.35)	0.02	(0.40)	0.01	(0.20)	0.01	(0.41)
f	1.09	(2.26)	0.62	(1.46)	0.82	(1.72)	0.65	(1.50)	0.36	(1.12)	0.68	(1.50)

to follow-up were older and many subjects were lost due to reaching the age ceiling for eligibility in the SDS. Permanent caries experience was significantly higher at follow-up than at baseline for continuous participants for both those in SA and Qld. The SA continuous participants' baseline permanent caries experience was significantly higher than that for Qld participants (DMFS SA 0.75 vs Qld 0.52) due to the older age range in SA. There was no difference in permanent caries experience at follow-up between children in the two states (DMFS SA 1.01 vs Qld 0.95). However, the difference in age range needs to be considered in interpreting these results.

At baseline, between 39.0% and 56.9% of 5-10 year old South Australians and between 38.9 and 57.1% of 5-10 year old Queensland children had some caries experience in the deciduous teeth (dmfs>0). Mean dmfs levels for 6-year-olds were 2.61 in South Australia and 3.62 in Queensland. The percentage of deciduous tooth surfaces with caries experience that were decayed (d/dmfs) was similar between strata and only differed a little between states. For example, among 5-year-olds, d/dmf was 37.6% in Adelaide, 38.2% in the remainder of SA, 44.4% in Brisbane, and 47.8% in Townsville.

The prevalence of caries experience in permanent teeth (DMFS>0) was less than 40% among children aged 10 years or less but increased to 69.0% among 15-year-olds in South Australia and to 51.0% among 12-year-olds in Queensland. The mean DMFS for 12-year-olds was 1.16 in South Australia and 1.70 in Queensland. The percentage of permanent tooth surfaces with caries experience (D/DMFS) among 12-year-olds varied from 16.4% in Adelaide to 15.2% in the remainder of SA, 17.4% in Brisbane and 30.1% in Townsville.

The annualised increment for the deciduous dentition (5–7-year-olds at baseline) was 0.34 (SD = 1.17) in SA and 0.50 (SD = 1.60) in Qld. In the permanent dentition (6–12-year-olds Qld, 6–15-year-olds SA) annualised caries increment was 0.12 (SD = 0.61) in SA and 0.24 (SD = 0.83) in Qld. The caries increment was 30% less in SA than Qld for the deciduous dentition and 50% less in SA than Qld for the permanent dentition.

The annualised caries increment for SA and Qld varied according to children's per cent lifetime exposure to fluoridated water (Table 5). A reasonably consistent linear trend occurred in the deciduous dentition of children in SA, with children with 0% lifetime exposure having sig-

Table 5. Annualised Net Caries Increment (NCI) by per cent lifetime exposure to fluoridated water for deciduous and permanent dentition for South Australia and Queensland (weighted).

% lifetime F exposure	*Annualised Net Caries Increment*			
	South Australia		*Queensland*	
	Deciduous (aged 5–7 at baseline)			
	(n = 4,310)		(n = 4,971)	
0%	442	0.50	3,496	0.54
1 to 24%	118	0.24	341	0.38
25 to 49%	474	0.45	337	0.37
50 to 74%	946	0.35	327	0.44
75 to 99%	751	0.31	211	0.43
100%	1,578	0.28	260	0.29
	F = 3.71, p=0.002		F = 2.32, p=0.040	
	Permanent (aged 6–15 in SA; 6–12 in Qld)			
	(n = 7,232)		(n = 5,968)	
0%	704	0.15	4,120	0.27
1 to 24%	260	0.12	448	0.19
25 to 49%	851	0.13	405	0.22
50 to 74%	1,612	0.13	385	0.14
75 to 99%	1,398	0.11	301	0.14
100%	2,406	0.10	309	0.15
	F = 0.92, p=0.466		F = 4.17, p=0.001	

Table 6. Exposure to other fluorides (weighted)

Other fluoride variables	*South Australia*	*Queensland*
	Retained for followed-up	*Retained for followed-up*
Brushing frequency (per day)		
Once or less	38.9	27.5
Twice	57.9	69.4
More than twice	1.9	1.6
Missing	1.9	1.4
Fluoride treatments		
0	67.9	19.0
1–2	23.0	42.9
3–4	6.6	22.9
5+	2.5	15.2
Fluoride supplement use (month)		
0	86.6	75.8
>0–50	9.8	15.2
>50	3.6	9.0

nificantly higher caries increment than children with 100% exposure. The magnitude of this difference was 78.6%. A significant effect was also found in Qld with children with 0% exposure having an annualised caries increment 86.2% higher than children with 100% exposure. In the permanent dentition, although the caries increment was 50.0% and 80.0% higher in SA and Qld respectively for children with 0% exposure than for children who had 100% exposure, the association between increment and per cent lifetime fluoride exposure was statistically significant only in Qld.

The distribution of exposure to other fluorides in both SA and Qld is shown in Table 6. The majority of children in both states brushed their teeth with fluoridated

toothpaste twice per day on average, although a sizeable minority (38.9% and 27.5% in SA and Qld respectively) only brushed their teeth once per day or less. There was a difference between the states with Qld children brushing their teeth more often than SA children. More than three times the percentage of children had received fluoride treatments in Qld compared to SA. In SA, 67.9% of children had never received a fluoride treatment while in Qld 81.0% of children had received at least one fluoride treatment. The use of fluoride supplements in both states was uncommon, with only 13.4% and 24.2% of SA and Qld children respectively having used them. For those children who had taken fluoride tablets or drops, approximately two-thirds consumed them for 50 months or less.

Multivariate modelling

Due to possible confounding with demographic, socio-economic or other fluoride source variables, multivariate modelling was used to test the hypothesis that caries increment was associated with per cent lifetime exposure to fluoridated water.

Two models were run in the deciduous dentition for both SA and Qld using caries increment as the dependent variable (Table 7). In Model 1, the effect of lifetime exposure to fluoridated water was statistically significant in both states after controlling for gender and age. The beta coefficient was approximately 1.5 times larger in SA than in Qld with the negative coefficient indicating that increased exposure to fluoridated water was associated with decreased caries increment in the deciduous dentition. In Model 2 a sequential regression model was again used, this time with gender and age entered into the model at step 1, toothbrushing frequency, number of fluoride treatments and length of time taking fluoride supplements entered at step 2, the socio-economic variables of education, income and occupational prestige entered at step 3, and finally per cent lifetime exposure to fluoridated water entered at step 4. In both SA and Qld, the demographic variables were significantly related to caries increment, as were the other fluoride exposure variables as a group. However, the relationship between socio-economic variables and caries increment was not statistically significant. After statistically controlling for

Table 7. Multivariate analyses of per cent lifetime exposure to fluoridated water on annualised net caries increment for deciduous dentition (weighted).

		South Australia				*Queensland*			
		Beta	*Δ R^2*	*F Ch.*	*p**	*Beta*	*Δ R^2*	*F Ch.*	*p**
Model 1									
Step 1			0.011	24.06	<0.001		0.019	47.00	<0.001
	Sex	-0.035			0.023	-0.049			0.001
	Age	-0.100			<0.001	-0.131			<0.001
Step 2			0.003	12.98	<0.001		0.001	7.18	0.007
	% F exposure#	-0.055			<0.001	-0.038			0.007
		(n = 4,311, Model R^2 = 0.014)				(n =4,906, Model R^2 = 0.021)			
Model 2									
Step 1			0.011	24.03	<0.001		0.019	54.87	<0.001
	Sex	-0.035			0.022	-0.051			<0.001
	Age	-0.101			<0.001	-0.129			<0.001
Step 2			0.002	2.83	0.037		0.006	9.55	<0.001
	Brushing Freq.	-0.041			0.008	-0.075			<0.001
	F treatments	-0.001			0.953	0.000			0.993
	F supplements	0.020			0.198	-0.010			0.497
Step 3			0.002	0.80	0.643		0.002	1.06	0.394
	Education								
	Income								
	Occupat. Prestige								
Step 4			0.002	9.80	0.002		0.002	7.56	0.006
	% F exposure#	-0.050			0.002	-0.040			0.006
		(n = 4,240, Model R^2 = 0.018)				(n = 4,846, Model R^2 = 0.028)			

* p values are for the change in the F statistic (F Ch.) in relation to change in R^2 (Δ R^2) and for t statistics in relation to Beta coefficients

Percent lifetime exposure to fluoridated water

Table 8. Multivariate analyses of per cent lifetime exposure to fluoridated water on annualised net caries increment for permanent dentition (weighted).

		South Australia				*Queensland*			
		Beta	Δ R^2	*F Ch.*	*p**	*Beta*	Δ R^2	*F Ch.*	*p**
Model 1									
Step 1			0.004	15.01	<0.001		0.003	9.74	<0.001
	Sex	0.018			0.091	-0.022			0.237
	Age	0.059			<0.001	0.053			<0.001
Step 2			0.000	3.58	0.059		0.003	18.01	<0.000
	% F exposure[#]	-0.022			0.059	-0.055			<0.001
		(n = 7,235, Model R^2 = 0.004)				(n = 5,968, Model R^2 = 0.006)			
Model 2									
Step 1			0.004	13.31	<0.001		0.003	8.04	<0.001
	Sex	0.017			0.159	-0.019			0.152
	Age	0.058			<0.001	0.049			<0.001
Step 2			0.003	6.24	<0.001		0.006	11.12	<0.001
	Brushing Freq.	-0.035			0.003	-0.057			<0.001
	F treatments	0.035			0.004	0.032			0.030
	F supplements	-0.012			0.314	-0.036			0.006
Step 3			0.002	1.48	0.133		0.004	2.30	0.008
	Education								
	Income								
	Occupat. Prestige								
Step 4			0.000	3.14	0.076		0.003	16.66	<0.001
	% F exposure[#]	-0.022			0.076	-0.055			<0.001
		(n = 7,188, Model R^2 = 0.010)				(n = 5,891, Model R^2 = 0.015)			

* p values are for the change in the F statistic (F Ch.) in relation to change in R^2 (Δ R^2) and for t statistics in relation to Beta coefficients

[#] Percent lifetime exposure to fluoridated water

all the variables entered in steps 1 to 3, per cent lifetime exposure to fluoridated water exhibited a significant association with caries increment in both states. Again, the negative standardised beta coefficient indicated that increased exposure to fluoridated water was associated with decreased caries increment.

The series of sequential linear regression models calculated for the deciduous dentition was repeated using the annualised caries increment in the permanent dentition as the dependent variable (Table 8). The results differed between SA and Qld. In Model 1 where the relationship between per cent lifetime fluoride exposure and caries increment was examined after statistically controlling for gender and age, a statistically significant effect was found in Qld but not in SA. Although the standardised beta coefficients for both states were negative, indicating an inverse relationship between increased per cent lifetime exposure to fluoridated water and caries increment, the coefficient was 2.5 times larger in Qld than in SA. These results were repeated in Model 2, where the relationship between per cent lifetime fluoride exposure and caries increment was examined after statistically controlling for the demographic variables of age and gender, other fluoride exposure variables and the socio-economic variables. In this model the standardised beta coefficients for per cent lifetime exposure to fluoridated water remained unchanged from Model 1, meaning that a statistically significant effect was found in Qld only.

Discussion

The main findings from this study supported the hypothesis that there would be a negative association between per cent lifetime exposure to fluoridated water and dental caries increment. This effect was found in the deciduous dentition of two Australian states of very different water fluoridation coverage, but only in the low water fluoridation coverage situation in Qld in the permanent dentition.

The findings of this study are consistent with previous research within an Australian context that has found a significant association between caries experience and exposure to fluoridated water. For instance, Slade et al. (1995) found that greater exposure to fluoridated water

was associated with both lower dmfs and lower DMFS. Similarly, Stockwell et al. (1990) found that after adjusting for a number of potential confounding factors the relative risk of having caries in the non-fluoridated Bunbury region compared to the fluoridated city of Perth was 1.43 for the deciduous dentition and 1.39 for the permanent dentition.

One of the primary strengths of this study is the prospective longitudinal cohort design, with participants followed for up to three and a half years. Such a design allows temporal association to be established, as exposure is determined prior to disease outcome. Due to the time consuming nature and often high cost of longitudinal prospective studies there have been few such studies conducted to look at the effect of exposure to water fluoridation on caries increment. Those studies that have been reported have generally relied on a small sample size, been restricted in follow-up time or have looked at caries progression rather than increment. For example, Groeneveld (1985) reported on 93 children in fluoridated Tiel, Netherlands and 103 children in non-fluoridated Culemborg, finding that the progression of lesions occurred more often for children in the non-fluoridated area compared to the fluoridated area. Lawrence and Sheiham (1997) reported on 290 12- and 16-year-olds followed over one year, finding that the mean rate of approximal caries progression was 62 per cent lower in fluoridated compared to non-fluoridated areas. A more extensive study reported by Maupome et al. (2001) looked at caries progressions and reversals in 2,964 Canadian children over three years from both a fluoridated community and a community that had ceased water fluoridation. They found odds ratios almost twice as large for caries progression in the non-fluoridated compared to the fluoridated community. Our current study adds to this body of evidence, showing that exposure to fluoridated water is associated with a decrease in caries increment.

Another strength of this study is the large sample size and the small number of cases excluded due to missing data. Only 1.7 per cent of cases were excluded because there were two or more years of missing fluoride exposure data. Slade et al. (1995) identified missing data as a methodological issue with their paper on the association between exposure to fluoridated drinking water and caries experience. Differences in the classification of unknown exposure levels were identified as affecting the outcome of the study's results. In contrast, the current study excluded only a small number of cases with missing data as considerable effort was put into contacting the parents of children sampled for the study and clarifying errors or lapses in fluoride exposure reporting.

One of the most notable findings in this study was the difference between SA and Qld in the association of lifetime exposure to fluoridated water and caries increment in the permanent dentition. While a significant effect was found in Qld the effect in SA was not statistically significant. Slade et al. (1995) also grappled with this finding and attributed the difference between the states to four possible explanations. First, the lower DMFS in SA reduces the variability in caries experience making statistically significant effects harder to obtain. Second, there were differences in the sampling methodology for both states and it may be that differences in what they termed "unspecified community effects" may have been responsible for differences in the parameter estimates for the two states. Third, because a high percentage of children in SA consume non-fluoridated water from rainwater tanks (Armfield and Spencer, 2004) it is possible that those children recorded as consuming high proportions of non-fluoridated water might have been consuming considerable quantities of fluoridated water from their school. Finally, in SA a 'halo effect' (Newbrun, 1989) may have been in operation, whereby children from non-fluoridated areas were actually consuming fluoridated foods and beverages prepared in and distributed from the fluoridated capital city of Adelaide. The same phenomenon has been described as a 'diffusion effect' which, in the United States, was found to provide protective benefit in children's permanent teeth that was similar in magnitude to the direct effect of living in a fluoridated are (Griffin et al, 2001). The current study was undertaken in only two Australian states, and hence it was not possible to replicate the methodology use by Griffin et al (2001) to calculate the diffused benefit. However, Queensland represented the state with low coverage of the population (approximately 5 per cent) by water fluoridation, and it is therefore unlikely that a substantial quantity of manufactured foods or beverages would be transported to Brisbane from fluoridated Townsville, which is 1,117 km to the north of non-fluoridated Brisbane.

While the halo or diffusion effect may contribute to an explanation of the difference between SA and Qld in the association of lifetime exposure to fluoridated water and caries increment in the permanent dentition, it was noteworthy that exposure to other fluorides were controlled in the multivariate analyses. This was necessary as both the frequency of fluoride treatments (topical gel) and frequency of longer term use of fluoride supplements were greater in Qld than SA. This reflected some level of compensation for the limited exposure to water fluoridation in Qld. However, the associations of such exposures were in the same direction and of similar magnitude in the two states.

A number of methodological issues should be considered when interpreting the results of this study. First, the study used a large number of uncalibrated examiners across multiple sites. Some 216 and 95 SDS clinics were involved in Qld and SA respectively. It must be expected that using many uncalibrated examiners will lead to greater error variance within the study making it harder to detect statistically significant results. Also, systematic geographic variations in the diagnosis of caries cannot be ruled out as contributing to reported differences between areas. However, several features of this study setting suggest that there should be little bias due to geographic variation in diagnosis. First, the majority of dental therapists in the school dental services were trained through the their own state health department's dental therapy training school and hence, within each state, they underwent identical training in caries diagnosis. Second, clinical staff in the school dental service were salaried, and they have no financial incentives or disincentives to diagnose caries. This was borne out in the findings regarding the baseline percentage of surfaces with caries experience that were diagnosed with decay, where there was no consistent trend for differences in

percentages between the fluoridated and non-fluoridated regions within each state. In fact among 12-year-olds, Townsville children had the highest D/DMFT (30.1%) suggesting that any bias was probably in the direction of over-diagnosis in that fluoridated city. Finally, regardless of potential examiner bias, these findings identify a pragmatic public health problem, namely that caries increment is elevated among children with relatively less exposure to fluoride in water. Given the focus of this study on a community perspective of disease experience, the approach used in this study is instructive. Because it is school dental service staff who are required to diagnose and treat children's dental caries, it is in many ways appropriate to use the methodology employed within their clinical practice.

A second methodological issue concerns the differences in caries experience between those children who had baseline only examinations and those children for whom follow-up examinations were conducted. Children lost to follow-up had less caries experience at baseline in the deciduous dentition and more caries experience at baseline in the permanent dentition. However, much of this variation can be attributed to the differences in the age of these groups.

Finally, the lack of follow-up information on place of residence meant that exposure to fluoridated water across follow-up could not be determined. For some children exposure from baseline to follow-up may have constituted up to approximately 40% of the total possible lifetime exposure to fluoridated water at follow-up. However, we found that between 85% and 90% of children in both states were still recorded as either attending the same school or clinic at follow-up as at baseline, meaning that exposure to fluoridated water at follow-up would have likely remained the same as at baseline for the vast majority of children.

The current study adds further to the weight of evidence supporting the effectiveness of water fluoridation in reducing caries in children. Caries increment was less for children with higher lifetime exposure to fluoridated water. Of note, the associations between caries increment and per cent lifetime consumption of fluoridated water persisted despite the study taking place in a low caries population of children with a relatively high exposure to other fluorides vehicles. The association between exposure to water fluoridation was stronger for the deciduous than for the permanent dentition which indicates the difficulties in finding statistically significant comparisons when caries experience is low and where prominent diffusion or 'halo' effects may occur.

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References

Armfield, J.M., Roberts-Thomson, K.F. and Spencer, A.J.(1997): *The Child Dental Health Survey, Australia 1999: trends across the 1990s*. AIHW Cat. No. DEN 95 (AIHW Dental Statistics and Research Series No. 27). Adelaide: The University of Adelaide.

Armfield, J.M. and Spencer, A.J. (2004): Consumption of non-public water: implications for children's caries experience. *Community Dentistry Oral Epidemiology* **32,** 283–296.

Brown, L.P., Mulqueen, T.F. and Storey, E. (1990): The effect of fluoride consumption and social class on dental caries in 8-year–old children. *Australian Dental Journal* **35,** 61–68.

DePaola, P.F. (1990): Measurement issues in epidemiology of dental caries. In: *Risk assessment in dentistry*, ed.Bader, J.D. pp19-26. Chapel Hill: University of North Carolina, Dental Ecology.

Griffin, S.O., Gooch, B.F., Lockwood, S.A. and Tomar, S.L. (2001): Quantifying the diffused benefit from water fluoridation in the United States. *Community Dentistry Oral Epidemiology* **29,** 120-129.

Groeneveld, A. (1985): Longitudinal study of prevalence of enamel lesions in a fluoridated and non-fluoridated area. *Community Dentistry Oral Epidemiology* **13,** 159–163.

Hardwick, J. and Teasdale, J. and Bloodworth, G. (1982): Caries increments over 4 years in children aged 12 at the start of water fluoridation. *British Dental Journal* **153,** 217–222.

Hill, A.B. (1971): *Principles of medical statistics*. 9th edition. New York: Oxford University Press.

Lawrence, H.P. and Sheiham, A. (1997): Caries progression in 12 and 16-year-old schoolchildren in fluoridated and fluoride-deficient areas in Brazil. *Community Dental Oral Epidemiology* **25,** 402–411.

Locker, D. (1999):*Benefits and risks of water fluoridation; an update of the 1996 Federal-Provincial Sub-Committee Report*. Toronto: Faculty of Dentistry, University of Toronto.

Maupome, G., Shulman, J.D, Clark, D.C, Levy, S.M and Berkowitz, J. (2001): Tooth-surface progression and reversal changes in fluoridated and no-longer-fluoridated communities over a 3-year period. *Caries Research* **35,** 95–105.

McDonagh, M., Whiting, P., Bradley, M, et al. (2000): *A systematic review of public water fluoridation*. York; NHS Centre for Reviews and Dissemination, University of York.

National Health and Medical Research Council (NHMRC).(1991): *The effectiveness of water fluoridation*. pp122–127. Canberra: AGPS.

National Institute of Dental Research (NIDR).(1987): *Oral health of United States adults, the national survey of oral health in U.S. employed adults and seniors: 1985-86* (NIH Publication no. 87-2868). Bethesda: National Institute of Dental Research.

Newbrun, E. (1989): Effectiveness of water fluoridation. *Journal of Public Health Dentistry* **49**, 279–289.

Riordan, P.J. (1991): Dental caries and fluoride exposure in Western Australia. *Journal Dental Research* **70,** 1029–1034.

Slade, G.D., Davies, M.J., Spencer, A.J. and Stewart, J.F. (1995): Associations between exposure to fluoridated drinking water and dental caries experience among children in two Australian states. *Journal Public Health Dentistry* **55,** 218–228.

Slade, G.D., Spencer, A.J., Davies, M.J. and Stewart, J.F.(1996): The influence of exposure to fluoridated water on socio-economic inequalities in children's caries experience. *Community Dental Oral Epidemiology* **24,** 89-100.

Spencer, A.J. (1984): Time trends in exposure to optimally fluoridated water supplies among Australian adolescents. *Community Dental Oral Epidemiology* **12,** 1–4.

Spencer,A.J. (1986a): Past association of fluoride vehicles with caries severity in Australian adolescents. *Community Dental Oral Epidemiology* **14,** 233–237.

Spencer, A.J. (1986b): Contribution of fluoride vehicles to change in caries severity in Australia adolescents. *Community Dental Oral Epidemiology* **14,** 238–241.

Stockwell, A.J., Medcalf, G.W, Rutledge, G.J., Holman, C.D.J. and Roberts, M. (1990): Dental caries experience in schoolchildren in fluoridated and non-fluoridated communities in Western Australia. *Community Dental Oral Epidemiology* **18,** 184–189.

World Health Organisation (WHO) (1987): *Oral health surveys. Basic methods*. 3rd ed. Geneva: WHO.