

## reports

verifiable  
cpd paper

## Prevalence of enamel defects and dental caries among 9-year-old Auckland children

PHILIP J. SCHLUTER, SATHANANTHAN KANAGARATNAM, CALLUM S. DURWARD, AND ROBYN MAHOOD

*New Zealand Dental Journal 104, No. 4: 145-152; December 2008*

## ABSTRACT

**Objectives:** To report the prevalence and severity of enamel defects and dental caries in a probability-based sample of 9-year-old children in the Auckland region, both overall and by residence in fluoridated and non-fluoridated areas.

**Design:** A two-stage clustered design with stratification. Strata were defined by fluoridated and non-fluoridated regions, school size, and school decile status.

**Setting:** Invitations, consent forms and questionnaires were distributed to eligible participants at school for completion at home. Participants were examined at school-based clinics or in a mobile clinic.

**Participants/Materials and Methods:** The source population was 9-year-old children attending schools in the Auckland region and enrolled with the Auckland Regional School Dental Service. Participants returned a completed consent form and questionnaire by post and then had a dental examination. Regression analyses accommodating probability sampling weights, stratification and clustering were employed.

**Main outcome measures:** The modified Developmental Defects of Enamel index was used to classify enamel defects in permanent teeth. Diagnosis of dental caries on deciduous and permanent teeth was visually-based.

**Results:** Overall, 612 children participated, 310 in fluoridated and 302 in non-fluoridated areas. Diffuse opacities are prevalent in Auckland, with 28.0 per 100 children affected. Significant regional differences by fluoridation status were apparent, with diffuse opacity rates of 29.1 per 100 and 14.7 per 100 children in fluoridated and in non-fluoridated areas respectively ( $P < 0.001$ ). Conversely, the prevalence of deciduous teeth dental caries was significantly lower in fluoridated areas (54.9 per 100) than in non-fluoridated areas (62.0 per 100),  $P = 0.05$ .

**Conclusions:** Diffuse opacities were the predominant tooth defect found in this study, but their prevalence appears largely unchanged from estimates reported within New Zealand over the last 25 years.

## INTRODUCTION

Community water fluoridation has been identified by the Centres for Disease Control and Prevention (CDC) as one of 10 great public health achievements of the 20<sup>th</sup> century (Centers for Disease Control and Prevention, 2007). It reduces inequalities in oral health among children, adolescents and adults. However, it is widely recognised that fluoride can have both beneficial and harmful effects on the dentition (McDonagh et al., 2000). Dental caries is one of the common oral diseases, and fluoride reduces the rate of development of carious lesions. The effect of fluoride on dental caries is due primarily to the topical effect of fluoride after the teeth have

erupted into the oral cavity. The harmful effects of fluoride are due to its systemic absorption during tooth development, resulting in dental fluorosis (Ellwood and Fejerskov, 2003). Dean's data showed that fluorosis was prevalent even in communities exposed to concentrations of fluoride below 1 ppm (Dean, 1934). The dose-response relationship is linear and, for every 0.01 mg F/kg body weight increase in exposure, there is a corresponding detectable increase in dental fluorosis in the population (Ellwood and Fejerskov, 2003).

In addition to optimally fluoridated water, children today are potentially exposed to many different forms of supplemental fluoride, such as that in infant formulas and fluoride toothpastes. A child's potential risk for fluorosis therefore increases as his or her exposure increases with the number of fluoride-containing products used (Mascarenhas, 2000). The swallowing of fluoride toothpaste by very young children has been identified as a risk factor for fluorosis (Rock and Sabieha, 1997). It is difficult to quantify the precise amount of toothpaste that a small child might swallow; however, it is believed that small children may swallow around half the toothpaste placed on the brush (Rock, 1994). The age brushing commenced, the frequency of brushing, the fluoride concentration of the toothpaste (Tavener et al., 2004) and the amount of toothpaste applied to the toothbrush (and subsequently swallowed) have all been implicated as potential fluorosis risk factors. Fluoride tablets provide little pre-eruptive effect on preventing caries development, but present a clear risk for fluorosis, particularly if ingested by young children (Ellwood and Fejerskov, 2003).

In the past, mild dental fluorosis manifesting as whitish discolorations of the teeth was regarded as being an acceptable alternative to having dental caries. However, concern has been raised in recent decades that fluoride may be causing an increase in aesthetically unacceptable dental fluorosis in both fluoridated and non-fluoridated communities (Clark, 1994). It is therefore becoming increasingly critical that the balance between the beneficial and harmful effects of fluoride in young children is continuously monitored and maintained in order to reassure the public and the dental profession (Medical Research Council, 2002; World Health Organization, 1994).

After reviewing numerous studies, the York Review concluded that, at water supply fluoride levels of 1 ppm, 48% of the population was affected by dental fluorosis, and, of those, 12% had fluorosis of aesthetic concern (McDonagh et al., 2000). However, the United Kingdom's (UK) Medical Research Council (MRC) suggested that the prevalence of dental fluorosis of aesthetic concern in UK populations drinking artificially fluoridated water was probably lower than the estimates presented by the York Review (Medical Research Council, 2002). Recent UK and Irish studies support the MRC contention finding that the prevalence of dental fluorosis of aesthetic concern was between only 0% and 4% (Cochran et al., 2004; Tabari et al., 2000; Whelton et al., 2003).

Over time, several studies have been conducted in regions of New Zealand in order to compare the prevalence and severity of enamel defects among children living in fluoridated and non-fluoridated areas (Cutress et al., 1985; de Liefde and Herbison, 1985; 1989; Mackay and Thomson, 2005; Suckling and Pearce, 1984; Suckling et al., 1985). All of these studies have used the Developmental Defects of Enamel (DDE) index (or a variant). Moreover, for direct age-comparability, almost all New Zealand studies have investigated 9-year-old children, with the sole exception being that by Suckling and Pearce (1984), who examined 12-14-year-old children in Richmond. There has been considerable variability in the reported prevalence of enamel defects in these studies. The reported prevalence of diffuse opacities in fluoridated regions ranged from 28% in a 1981-82 Dunedin study (Suckling et al., 1985) to 51% in a 1985 Hawke's Bay study (de Liefde and Herbison, 1989), whereas the prevalence in non-fluoridated regions ranged from 14%—in both the 2002 Southland study (Mackay and Thomson, 2005) and 1985 Auckland study (Cutress et al., 1985)—to 24% in the 1985 Hawke's Bay study (de Liefde and Herbison, 1989). Moreover, all of the data themselves are historical. Except for the 2002 Southland study, no other studies on fluorosis or developmental defects of enamel have been carried out in New Zealand since 1985.

The World Health Organization (WHO) recommends that countries monitor changes in the prevalence of dental caries and fluorosis, and base their recommendations concerning fluoridation and the use of fluoride toothpastes on these findings (World Health Organization, 1994). Using a methodologically rigorous design, thorough clinical examination, and careful statistical analysis, the current study aimed to provide robust and contemporary estimates of the prevalence and severity of enamel defects in 9-year-old children in the Auckland region as a whole, and for fluoridated and non-fluoridated areas. When investigating the prevalence of diffuse opacities, it is also important to report also on the prevalence and severity of dental caries (in line with the WHO recommendations), and this comprises the second aim for the study.

## METHODS

### Source population

The source population was nine-year-old children attending schools in the Auckland region and enrolled with the Auckland Regional School Dental Service (SDS). In the Auckland region, approximately 95% of all schoolchildren (including those who are home-schooled) are enrolled with the SDS (Hagre et al., 2003).

### Study design

A two-stage clustered design with stratification was used. Strata were defined by fluoridated and non-fluoridated regions, school size (smaller, 5-39 9-year-old children; and larger, 40- 9-year-old children), and school decile (lower, deciles 1-3; middle, deciles 4-7; and higher, deciles 8-10). A school's decile score indicates the extent to which it draws its students from communities of low socio-economic status (SES). Decile-1 schools comprise the 10% of New Zealand schools with the highest proportion of students from low-SES communities, whereas decile-10 schools are the 10% of schools with the lowest proportion of those students (Ministry of Education, 2007). For efficiency reasons, schools with fewer than five 9-year-old children were excluded from the sampling frame, and resource

constraints meant that a maximum of 38 schools could be included. The number of schools and the students from each school were probabilistically sampled in order to reflect the overall decile and school size distribution which was representative of Auckland schools yet produce a sample that was approximately balanced between fluoridated and non-fluoridated regions. Moreover, sampling was designed so that at least two schools were selected within each stratum.

### Procedures

Detailed information about the procedures is described elsewhere (Kauagaratnam et al., under review). In brief, project information and an invitation to participate were sent to principals and Boards of Trustees of all selected primary schools. Once consent was obtained, school student lists were used to randomly select potential participants. Selected children and their parents/caregivers were provided with an information sheet, questionnaire, consent form and return envelope. A research assistant ensured that the appropriate forms were sent to parents/caregivers with instructions to return them by the due date (2 weeks after issue). If the material was not received on time, a reminder letter with a new complete set of forms was sent. Only children for whom the signed consent form and completed questionnaire were returned were eligible for examination. One section of the questionnaire asked for information on the current area of residence and all other addresses where the child had lived for more than 3 months since birth (specifically: suburb, town and length of domicile, in months and years).

A dental examination date was scheduled for each child (and subsequently rescheduled if the first appointment date was missed through illness, class trip, etc). All children were examined at school-based clinics or in the mobile clinic by author RM after training and calibration by a registered dentist. The teeth were examined in wet condition, and labial/buccal, lingual/palatal and occlusal surfaces of all erupted permanent teeth were visually inspected for enamel defects. The modified Developmental Defects of Enamel (DDE) index was used to identify and classify enamel defects in permanent teeth (FDI Working Group, 1992). Single defects smaller than 1mm in diameter were not recorded. Doubtful areas, such as suspected hypoplastic pits, were explored with a blunt probe to confirm a diagnosis. Both defect type and extent were recorded. The extent of a defect in a tooth was measured by the surface area of the enamel affected. When two different types of defect were present, then the recorded extent related to the combined size of the defects.

Diagnosis of dental caries in deciduous and permanent teeth was visually-based, and a blunt probe was used only for the removal of debris when necessary. Compressed air and radiographs were not used. Classification of the status of individual tooth surfaces was based on the decayed, missing, and filled teeth (dmft for deciduous and DMFT for permanent teeth), and decayed, missing, and filled surfaces (dmfs for deciduous and DMFS for permanent teeth) indices (Pitts et al., 1997).

Clinical data were recorded at the time of the examination on a laptop computer using Dental SurveyPlus 2 version 2.1 (University of Dundee, Dundee, Scotland). Following the examination, each child was given a sheet to take home informing the parent/caregiver of whether any dental caries (or abnormalities) requiring possible treatment had been identified, and advising the seeking of care. This information was also passed to the local dental therapist.

### Statistical analysis

Data were downloaded from the Dental Survey Plus database into specialist statistical databases and packages, combined with questionnaire information, and then consistency and range checks were performed. Descriptive statistics were calculated and reported, and group comparisons of the demographic characteristics of participants between fluoridation and non-fluoridation regions was undertaken using Pearson's  $\chi^2$  test (or, where appropriate, Fisher's exact test) for categorical variables, and Student's *t*-test for continuous variables. The *svy* procedure in Stata 10 (StataCorp, TX, USA) was used to estimate prevalence and associated 95% confidence intervals (95% CI), after accounting for the two-stage clustered design with stratification. Sampling weights inversely proportional to the probability of being selected (and adjusted for non-response within schools) were employed. A finite population correction factor at both levels was used, accounting for the sampling without replacement of schools within strata and the sampling without replacement of students within selected schools. Logistic regression models were used in the analysis of binary outcome data (prevalence), whereas linear regression models were used to model the means of caries severity data. For subgroup analysis, important differences identified by age and/or sex were also included in the regression models. Residual checks and influence diagnostics were checked for all statistical models (Dupont, 2002), but these have not been reported unless important assumption violations were noted. Significance was set at  $\alpha=0.05$  for all statistical comparisons.

### Ethics

Ethical clearance was obtained from the Auckland Branch of the National Ethics Committee (NTX/06/06/067).

### RESULTS

Of the 437 schools in Auckland, 331 (75.7%) had more than five 9-year-old children. There was no systematic difference in the fluoride status (Fisher's exact  $P=0.15$ ) or decile status (Fisher's exact  $P=0.35$ ) between eligible and excluded schools. All selected schools agreed to participate in the study, including 22 schools in fluoridated areas and 16 schools in non-fluoridated areas. The student pool of eligible 9-year-old children in these 38 selected schools totalled 1,113, of whom 612 (55%) consented, completed the questionnaire and underwent examination. Response rates varied widely by

school, with between 29% and 98% of children participating in fluoridated areas ( $n=310$ , average response rate 61%) and 30% and 84% of children participating in non-fluoridated areas ( $n=302$ , average response rate 50%).

The average age of children examined was about 9.5 years, with a small but statistically significant difference by fluoridation status (Table 1). A significant SES difference was seen among participants from fluoridated and non-fluoridated areas, despite the stratified study design. More children from higher-decile schools in fluoridated areas and more children from lower-decile schools in non-fluoridated areas participated in this study. Moreover, a significant ethnic difference was seen among participants from fluoridated and non-fluoridated areas. More children of European descent and fewer children of Asian descent attended schools within non-fluoridated areas than those within fluoridated areas.

Of the 310 children in fluoridated areas who participated in the study, 175 (56%) had lived continuously in a fluoridated area from birth. Similarly, of the 302 children in non-fluoridated areas participating in the study, 149 (49%) had lived continuously in a non-fluoridated area from birth. Of the 288 children with intermittent or unknown residential fluoridation history status, 50% had lived more than 50% of their lives within the current region, and 25% had lived more than 70% of their lives within the current region. Almost half (47%) of those children with intermittent or unknown residential fluoridation history had immigrated from overseas. Others had moved within Auckland or within regions of New Zealand, but failed to provide a complete residential history.

### Prevalence of children with enamel defects

The frequencies and estimated prevalence of enamel defects for all 612 participating children and those in fluoridated and non-fluoridated areas are presented in Table 2. Enamel defects of any type were observed in just over one-third of children overall, with an estimated prevalence of 40.7 per 100 children. Overall, the estimated prevalence of diffuse opacities (28.0 per 100 children) was higher than that for demarcated opacities (19.8 per 100 children) or hypoplastic defects (7.3 per 100 children). Children living in fluoridated areas had a higher prevalence of diffuse opacities than their counterparts living in non-fluoridated areas. A similar association was seen for the prevalence of any enamel defects ( $P=0.03$ ).

Table 1. Demographic characteristics of the participating 9-year-old children presented by their fluoridation status.

	Overall	Fluoridation region status		P-value
		Fluoridated	Non-fluoridated	
	n (%)	n (%)	n (%)	
Years of age: mean (SD)	9.5 (0.2)	9.5 (0.2)	9.6 (0.2)	<0.001*
Gender				0.87
Male	320 (52)	161 (52)	159 (53)	
Female	295 (48)	149 (48)	143 (47)	
Ethnicity				<0.001
European	364 (59)	151 (49)	213 (71)	
Maori	113 (18)	66 (21)	47 (16)	
Pacific	66 (11)	40 (13)	26 (9)	
Asian	41 (7)	34 (11)	7 (2)	
Other	28 (5)	19 (6)	9 (3)	
Socio-economic status				<0.001
High (decile 8-10)	181 (30)	124 (40)	57 (19)	
Middle (decile 4-7)	260 (42)	127 (41)	133 (44)	
Low (decile 1-3)	171 (28)	59 (19)	112 (37)	

P-values calculated using Fisher's exact test except for \* which used Student's *t*-test.

Table 2. Frequencies (percentages) and prevalence estimates for enamel defects, adjusted for child's age centred around 9.5 years, with associated 95% confidence intervals (95% CI) for the overall sample and presented by fluoridation status.

	Total	Mouth			P-value	Total	Tooth			P-value
		n (%)	prevalence (95% CI)				n (%)	prevalence (95% CI)		
Any defect										
Overall	612	215 (35.1)	40.7 (32.3, 49.0)		9,033	990 (11.0)	14.1 (11.3, 16.9)			
Fluoridation status				0.03						<0.001
Fluoridated	310	114 (36.8)	41.4 (31.5, 51.2)		4,673	594 (12.7)	14.5 (11.2, 17.8)			
Non-fluoridated	302	101 (33.4)	32.3 (26.7, 37.8)		4,360	396 (9.1)	9.3 (7.9, 10.7)			
Demarcated opacities										
Overall	612	115 (18.7)	19.8 (14.9, 24.8)		9,033	268 (3.0)	3.0 (0.9, 5.1)			0.64
Fluoridation status				0.95						
Fluoridated	310	54 (17.4)	19.8 (14.0, 25.6)		4,673	121 (2.6)	3.0 (0.5, 5.4)			
Non-fluoridated	302	61 (20.2)	20.1 (14.8, 25.5)		4,360	147 (3.4)	3.4 (2.2, 4.6)			
Diffuse opacities										
Overall	612	117 (19.1)	28.0 (18.0, 37.9)		9,033	642 (7.1)	10.6 (7.2, 14.0)			<0.001
Fluoridation status				0.001						
Fluoridated	310	74 (23.9)	29.1 (17.3, 40.8)		4,673	432 (9.2)	11.0 (7.0, 15.1)			
Non-fluoridated	302	43 (14.2)	14.7 (11.1, 18.3)		4,360	210 (4.8)	5.1 (4.0, 6.2)			
Hypoplastic defect										
Overall	612	57 (9.3)	7.3 (4.4, 10.1)		9,033	110 (1.2)	1.0 (0.5, 1.5)			0.64
Fluoridation status				0.34						
Fluoridated	310	27 (8.7)	7.1 (3.7, 10.4)		4,673	59 (1.3)	1.0 (0.4, 1.6)			
Non-fluoridated	302	30 (9.9)	9.6 (6.4, 12.7)		4,360	51 (1.2)	1.1 (0.8, 1.5)			

### Prevalence of teeth with enamel defects

The frequencies and prevalence estimates for enamel defects found in all 9,033 examined teeth are also presented in Table 2. Overall, 990 (11%) were observed with enamel defects, yielding a population prevalence of 14.1 per 100 teeth. Of those 990 affected teeth, 237 (24%) had demarcated opacities only, 614 (62%) had diffuse opacities only, 107 (11%) had hypoplastic defects only, 28 (3%) had both demarcated and diffuse opacities, 3 (0.3%) had demarcated opacities and hypoplastic defects, and 1 (0.1%) had all three defects. No significant differences in teeth prevalence estimates were seen between fluoridated and non-fluoridated areas for demarcated opacities or hypoplastic defects, but teeth in fluoridated areas had a significantly higher prevalence of diffuse opacities than teeth in non-fluoridated areas (11.0 per 100 teeth and 5.1 per 100 teeth respectively;  $P < 0.001$ ).

### Distribution and severity of enamel defects

On average, 14.8 permanent teeth were examined in each child (range: 6-28 teeth). Data on the distribution of enamel defects for each tooth within the sample are presented in Table 3.

Maxillary and mandibular teeth differed significantly in their distributions of demarcated opacities ( $\chi^2=28.7$ ,  $df=13$ ,  $P < 0.001$ ), with teeth 11 and 21 in particular having relatively more demarcated opacities than teeth 31 and 41. However, there was no evidence of left-side/right-side asymmetry in the distribution of demarcated opacities in teeth ( $\chi^2=6.5$ ,  $df=13$ ,  $P=0.93$ ). There was significant non-uniformity in the distribution of demarcated opacities in maxillary teeth ( $\chi^2=56.3$ ,  $df=13$ ,  $P < 0.001$ ) with teeth 11 and 21 having relatively more demarcated opacities than the other teeth. By contrast, demarcated opacities were more evenly distributed among mandibular teeth ( $\chi^2=7.8$ ,  $df=13$ ,  $P=0.85$ ).

Where diffuse opacities are concerned, maxillary and mandibular teeth differed significantly in their distribution ( $\chi^2=78.1$ ,  $df=13$ ,  $P < 0.001$ ), but there was no evidence of left-side/right-side asymmetry in the distribution of diffuse opacities in teeth ( $\chi^2=3.4$ ,  $df=13$ ,  $P=0.99$ ). There was significant non-uniformity in the distribution of diffuse opacities in teeth in the maxillary teeth ( $\chi^2=30.0$ ,  $df=13$ ,

$P=0.005$ ), with teeth 11 and 21 having relatively more (and teeth 13, 23, 14 and 24 having relatively fewer) diffuse opacities than the other teeth. Similarly, there was significant non-uniformity in the distribution of diffuse opacities in mandibular teeth ( $\chi^2=23.8$ ,  $df=13$ ,  $P=0.03$ ), with teeth 36 and 46 having relatively more diffuse opacities.

For hypoplastic defects, there was no difference in their distributions in the maxilla and mandible ( $\chi^2=12.9$ ,  $df=13$ ,  $P=0.46$ ), and neither was there evidence of left-side/right-side asymmetry ( $\chi^2=6.3$ ,  $df=13$ ,  $P=0.94$ ). There was significant non-uniformity in the distribution of hypoplasia in maxillary teeth ( $\chi^2=49.9$ ,  $df=13$ ,  $P < 0.001$ ), with teeth 16 and 26 having relatively more (and teeth 11, 21, 12 and 22 having relatively fewer) instances of hypoplasia than the other teeth. Similarly, there was significant non-uniformity in the distribution of hypoplasia in mandibular teeth ( $\chi^2=65.5$ ,  $df=13$ ,  $P < 0.001$ ), with teeth 36 and 46 having relatively more (and teeth 32 and 42 having relatively fewer) instances of hypoplasia than the other teeth.

Where the presence of any enamel defect was concerned, maxillary and mandibular teeth differed significantly in their distribution ( $\chi^2=82.8$ ,  $df=13$ ,  $P < 0.001$ ). Again, there was no evidence of left-side/right-side asymmetry ( $\chi^2=6.7$ ,  $df=13$ ,  $P=0.92$ ). There was significant non-uniformity in the distribution of enamel defects in maxillary teeth ( $\chi^2=53.9$ ,  $df=13$ ,  $P < 0.001$ ), with teeth 11 and 21 having relatively more defects and teeth 13, 23, 14 and 24 having relatively fewer defects than the other teeth. Similarly, there was significant non-uniformity in the distribution of enamel defects in mandibular teeth ( $\chi^2=32.4$ ,  $df=13$ ,  $P=0.002$ ) with teeth 36 and 46 having relatively more and teeth 32 and 42 having relatively fewer defects.

Of the teeth observed with enamel defects, 251 (25%) had defects involving less than one-third of the labial surface, 331 (33%) had defects covering between one-third and two-thirds of the labial surface, and 408 (41%) had defects extending over at least two-thirds of the labial surface.

### Prevalence and severity of dental caries

Data on the prevalence and severity of dental caries in deciduous and permanent teeth are presented in Table 4. Dental caries in deciduous teeth was observed in 370

Table 3. Distribution of enamel defects across the permanent dentition.

Tooth	Number examined N	Demarcated opacity		Diffuse opacity		Hypoplastic defect		Any defects	
		n (%)	n (%)	n (%)	n (%)	n (%)	n (%)		
11	607	43 (7)	73 (12)	4 (1)	114 (19)				
12	581	23 (4)	55 (9)	4 (1)	81 (14)				
13	107	0 (0)	3 (3)	1 (1)	4 (4)				
14	182	2 (1)	11 (6)	2 (1)	15 (8)				
15	78	0 (0)	6 (8)	1 (1)	7 (9)				
16	610	15 (2)	59 (10)	16 (3)	86 (14)				
17	9	0 (0)	0 (0)	0 (0)	0 (0)				
21	609	36 (6)	72 (12)	2 (0)	106 (17)				
22	583	14 (2)	50 (9)	1 (0)	63 (11)				
23	104	1 (1)	1 (1)	1 (1)	3 (3)				
24	175	2 (1)	10 (6)	2 (1)	13 (7)				
25	70	0 (0)	4 (6)	2 (3)	6 (9)				
26	611	15 (2)	60 (10)	23 (4)	94 (15)				
27	13	0 (0)	0 (0)	0 (0)	0 (0)				
31	611	19 (3)	25 (4)	4 (1)	49 (8)				
32	607	14 (2)	24 (4)	0 (0)	38 (6)				
33	215	4 (2)	7 (3)	1 (0)	12 (6)				
34	182	7 (4)	12 (7)	0 (0)	17 (9)				
35	82	1 (1)	4 (5)	1 (1)	6 (7)				
36	611	12 (2)	43 (7)	19 (3)	71 (12)				
37	33	2 (6)	1 (3)	0 (0)	3 (9)				
41	612	19 (3)	25 (4)	5 (1)	49 (8)				
42	606	11 (2)	24 (4)	0 (0)	35 (6)				
43	216	6 (3)	9 (4)	1 (0)	16 (7)				
44	191	5 (3)	12 (6)	0 (0)	16 (8)				
45	79	2 (3)	5 (6)	3 (4)	10 (13)				
46	611	14 (2)	47 (8)	17 (3)	75 (12)				
47	38	1 (3)	0 (0)	0 (0)	1 (3)				
Total	9033	268 (3)	642 (7)	110 (1)	990 (11)				

children (61%). The prevalence of deciduous caries was significantly lower for children living in fluoridated areas than among children residing in non-fluoridated areas (54.9 per 100 children and 62.0 per 100 children respectively;  $P=0.05$ ). Mean dmft scores were significantly lower among children living in fluoridated areas than among those residing in non-fluoridated areas. While mean dmfs scores were lower in fluoridated areas than in non-fluoridated areas, no statistically significant difference was observed (due to the higher variability associated with this measure). Permanent dentition caries was observed in 95 children (16%), with an overall prevalence of 15.6 per 100 children. There were

no significant differences between fluoridated and non-fluoridated areas in caries prevalence or severity.

## DISCUSSION

Diffuse opacities are prevalent in Auckland, with 28.0 per 100 9-year-old children. There were clinically and statistically important differences in diffuse opacity mouth and tooth prevalence estimates between fluoridation and non-fluoridation regions, and these were consistent with international (Cochran et al., 2004; Milsom and Mitropoulos, 1990; Nik-Hlissein et al., 1999) and previous national estimates (Cutress et al., 1985; de Liefde and Herbison,

Table 4. Frequencies (percentages) and prevalence and severity estimates for dental caries in the deciduous and permanent dentitions, adjusted for child's age centred around 9.5 years, with associated 95% confidence intervals (95% CI) for the overall sample and presented by fluoridation status.

	Deciduous teeth				Permanent teeth			
	Total	N (%)	Prevalence (95% CI)	P-value	n (%)	Prevalence (95% CI)	P-value	
<i>Caries</i>								
Overall	612	370 (60.5)	55.5 (48.9, 62.0)	0.05	95 (15.5)	15.6 (11.5, 19.7)	0.14	
Fluoridation status								
Fluoridated	310	177 (57.1)	54.9 (47.2, 62.6)		54 (17.4)	15.9 (11.0, 20.8)		
Non-fluoridated	302	193 (63.9)	62.0 (57.3, 66.7)	41 (13.6)	11.7 (8.0, 15.5)			
		Mean (SD)	Mean (95% CI)		Mean (SD)	Mean (95% CI)		
<i>Severity of caries in teeth</i>								
Overall	612	2.07 (2.38)	1.72 (1.42, 2.07)	0.02	0.25 (0.69)	0.21 (0.15, 0.27)	0.88	
Fluoridation status								
Fluoridated	310	1.87 (2.28)	1.69 (1.33, 2.04)		0.25 (0.62)	0.21 (0.14, 0.28)		
Non-fluoridated	302	2.26 (2.48)	2.10 (1.86, 2.33)	0.26 (0.75)	0.20 (0.13, 0.27)			
<i>Severity of caries in surfaces</i>								
Overall	612	4.04 (5.17)	3.41 (2.77, 4.06)	0.15	0.35 (1.10)	0.26 (0.20, 0.32)	0.58	
Fluoridation status								
Fluoridated	310	3.76 (5.07)	3.37 (2.61, 4.13)		0.31 (0.90)	0.25 (0.18, 0.33)		
Non-fluoridated	302	4.32 (5.25)	3.95 (3.46, 4.44)	0.39 (1.27)	0.30 (0.19, 0.42)			

Table 5. Comparison of the findings of New Zealand studies on the mouth and tooth prevalence developmental defects of enamel in 9-year-old children in fluoridated areas (F) and non-fluoridated areas (NF).

Enamel defects	Dunedin <sup>1</sup> 1981-1982 <sup>a,c</sup>		Hawke's Bay <sup>2</sup> 1982 <sup>a,c</sup>		Hawke's Bay <sup>3</sup> 1985 <sup>a,c</sup>		Southland <sup>4</sup> 2002 <sup>b,d</sup>		Auckland <sup>5</sup> 1985 <sup>a,c</sup>		Auckland 2007 <sup>a,d</sup>	
	F	NF	F	NF	F	NF	F	NF	F	NF	F	NF
Mouth prevalence												
Number of participants	581	51	191	237	260	263	137	183	105	28	310	302
Demarcated opacities	35%	37%	47%	44%	28%	27%	37%	36%	46%	43%	19.8%	20.1%
Diffuse opacities	28%	16%	37%	23%	51%	24%	29%	14%	30%	14%	29.1%	14.7%
Hypoplasia	12%	28%	15%	14%	-	-	5%	4%	13%	14%	7.1%	9.6%
Any defect	56%	63%	69%	56%	-	-	52%	48%	64%	54%	41.4%	32.3%
Tooth prevalence												
Demarcated opacities	-	-	11%	8%	3%	4%	9%	8%	7%	9%	3.0%	3.4%
Diffuse opacities	-	-	11%	5%	19%	7%	8%	5%	13%	4%	11.0%	5.1%
Hypoplasia	-	-	2%	2%	-	-	1%	1%	1%	1%	1.0%	1.1%
Any defect	-	-	22%	14%	-	-	18%	16%	21%	13%	14.5%	9.3%

Key: <sup>1</sup>Suckling et al., 1985; <sup>2</sup>de Liefde and Herbison, 1985 & 1989; <sup>3</sup>de Liefde and Herbison, 1989; <sup>4</sup>Mackay and Thomson, 2005; <sup>5</sup>Cutress et al., 1985; <sup>a</sup>Full mouth survey; <sup>b</sup>Ten index teeth only (WHO); <sup>c</sup>Teeth dried not wiped; <sup>d</sup>Teeth not wiped or dried; <sup>e</sup>Teeth wiped not dried.

1985; 1989; Mackay and Thomson, 2005; Suckling and Pearce, 1984; Suckling et al., 1985). Diffuse opacities are the predominant type of tooth defect found in this study, a finding also reported in overseas studies (Cochran et al., 2004; Milsom and Mitropoulos, 1990; Nik-Ilissein et al., 1999). In contrast, all other New Zealand studies reported that demarcated opacities were the predominant type of enamel defect. The mouth and tooth prevalence estimates of enamel defects from previously published New Zealand studies are summarised in Table 5. The current study's estimates for diffuse opacity prevalence fall within the previously published range, but its estimated prevalence for demarcated opacities is considerably lower than previously-published estimates. This apparent shift in enamel defect type ranking within New Zealand needs further epidemiological confirmation.

The current study also demonstrated a significant association between deciduous caries prevalence and residential fluoridation history status. The severity of dental caries in children's teeth (as represented by the mean dmft score) showed a pattern similar to that seen with caries prevalence, but it was not observed at surface level (dmfs). In addition, no significant association was found between residential fluoridation history and dental caries in the permanent dentition. This may be partly because, at 9 years of age, only some of the permanent teeth are present, and differences in caries prevalence and severity with differing exposures to fluoride have yet to become fully apparent, unlike the situation with older children, whose permanent teeth have been exposed for longer (Kanagaratnam, 1997). Moreover, many of the permanent first molars of the children in the current study had been fissure-sealed.

In this descriptive epidemiological study, we employed a study design that investigated and reported prevalence based on current fluoridation residential status, rather than partitioning the sample into those with life-long exposure and examining that sub-group only. Although it made comparisons between our findings and those of other studies more difficult, we undertook this approach for three related primary reasons. First, a complete picture of the prevalence of the condition of interest over an easily identifiable population has, we believe, greater utility for policy-makers and health promoters than empirical evidence from incomplete or more-difficult-to-identify populations. Moreover, if families' propensity for residential mobility changes over time, then benchmarking prevalence on different population

proportions is likely to be confounded if those who move have different oral health profiles, behaviours or risk factors compared to those who stay. Second, subgroup analyses introduce analytic challenges and can lead to overstated and misleading findings in both clinical (Lagakos, 2006; Wang et al., 2007) and observational studies (Vandenbroucke et al., 2007). Issues of treatment effect heterogeneity and test multiplicity—and how joint effects and interactions between risk factors should be evaluated and reported are debated—remain unresolved and are generally poorly handled by most authors (Vandenbroucke et al., 2007; Wang et al., 2007). Third, probability weights for the two-stage cluster design with stratification adopted here can be readily calculated, thereby yielding valid and robust prevalence estimates for all contributing participants. When residential status and life-long exposure are used to define sub-groups, then both the probability weighting and complex-sample analysis (accounting for the study design) become considerably more complex in determining meaningful population prevalence. However, if the epidemiologic aim differs from determining population prevalence, such as identifying important associations and correlates within a sample (Kanagaratnam et al., under review), then categorisations based on exposure are appropriate.

While this study has many strengths (including its methodological and clinical robustness, sample-frame coverage that exceeds 95% of the 9-year-old population, large sample size, and sophisticated statistical analysis), it also has several weaknesses. Overall, 55% of eligible children participated in this study. Even though many schools were visited two or three times, in many cases, children were either absent or involved in other school activities on the days scheduled for examinations. Some children failed to return the questionnaire even after the second set of forms were either sent or given to the children by the dental therapists. A small number of parents (and, in one case, the child) did not consent for examination. Should the non-participants have a different enamel defect or dental caries profile, then our prevalence estimates will be biased. Moreover, there were differences in participant distributions between fluoridated and non-fluoridated areas by children's age, SES and ethnicity characteristics. Unlike the SES differences seen between participants from fluoridated and non-fluoridated areas, the ethnic distribution differences seen are likely due to different ethnic geographical residential preferences rather than differential systematic patterns of response. As



the statistical weights adjusted for the non-response within schools (and included weights for the strata), the reported prevalence estimates accommodated and adjusted for the SES differences. Children's age was centred around 9.5 years and corrected for in all reported statistical analyses determining prevalence.

Using a methodologically rigorous design, thorough clinical examination, and careful statistical analysis, this study provides contemporary estimates of the prevalence and severity of enamel defects and dental caries in 9-year-old children in the Auckland region as a whole, and for fluoridated and non-fluoridated areas. Moreover, a detailed description of the distribution of enamel defects for each permanent tooth examined within our large sample is provided. Such benchmarking descriptive epidemiological information is critical to clinicians, policy-makers and health promoters in understanding the population rates and surrounding issues within the contemporary environment and to monitor broad changes within a historical context. Armed with this empirical evidence, informed policies and recommendations might be made.

#### ACKNOWLEDGMENTS

We are grateful to Auckland University of Technology, Counties-Manukau District Health Board and the New Zealand Dental Association Research Foundation for funding this study. We are particularly thankful to Waitemata District Health Board for allowing us to use the clinics, Tim Mackay for training the examiner, and Cheryl Brookes and Hewa Baas for recording the data. All staff of Auckland Regional Dental Service and Dr Robin Whyman are thanked for their valuable support.

#### REFERENCES

- Centers for Disease Control and Prevention (2007). Community Water Fluoridation. Atlanta, GA: CDC.
- Clark DC (1994). Trends in prevalence of dental fluorosis in North America. *Community Dentistry and Oral Epidemiology* 22(3):148-152.
- Cochran JA, Ketley CE, Arnadóttir IB, Fernandes B, Koletsis-Kouari H, Oila AM, et al. (2004). A comparison of the prevalence of fluorosis in 8-year-old children from seven European study sites using a standardized methodology. *Community Dentistry and Oral Epidemiology* 32(S1):28-33.
- Cutress TW, Suckling GW, Pearce FF, Ball ME (1985). Defects of tooth enamel in children in fluoridated and non-fluoridated water areas of the Auckland region. *New Zealand Dental Journal* 81:12-19.
- de Liefde B, Herbison GP (1985). Prevalence of developmental defects of enamel and dental caries in New Zealand children receiving differing fluoride supplementation. *Community Dentistry and Oral Epidemiology* 13(3):164-167.
- de Liefde B, Herbison GP (1989). The prevalence of developmental defects of enamel and dental caries in New Zealand children receiving differing fluoride supplementation, in 1982 and 1985. *New Zealand Dental Journal* 85:2-7.
- Dean HT (1934). Classification of mottled enamel diagnosis. *Journal of the American Dental Association* 21:1421-1426.
- Dupont WD (2002). Statistical Modelling for Biomedical Researchers. A Simple Introduction to the Analysis of Complex Data. Cambridge: Cambridge University Press.
- Ellwood R, Fejerskov O (2003). Clinical use of fluoride. In: Dental Caries: The Disease and its Clinical Management O Fejerskov and E Kidd editors. Munksgaard, Denmark: Blackwell.
- FDI Working Group (1992). A review of the developmental defects of enamel index (DDE Index). Commission on Oral Health, Research & Epidemiology. *International Dental Journal* 42(6):411-426.
- Hagre K, Kiro C, Stewart L, Logan R, Forgere G, Pearce N (2003). Improving Child Oral Health and Reducing Child Oral Health Inequalities. Wellington: Public Health Advisory Committee, National Advisory Committee on Health and Disability.
- Kanagaratnam S (1997). Dental caries patterns and the utilisation of dental services among 15-year-old adolescents in the Southern Regional Health Authority region. *New Zealand Dental Journal* 93(412):44-46.
- Kanagaratnam S, Schluter PJ, Durward C, Mahood R, Mackay T (under review). Enamel defects and dental caries in 9 year old children living in fluoridated and non-fluoridated areas of Auckland, New Zealand. *Community Dentistry and Oral Epidemiology*.
- Lagakos SW (2006). The challenge of subgroup analyses — reporting without distorting. *New England Journal of Medicine* 354(16):1667-1669.
- Mackay TD, Thomson WM (2005). Enamel defects and dental caries among Southland children. *New Zealand Dental Journal* 101(2):35-43.
- Mascarenhas AK (2000). Risk factors for dental fluorosis: a review of the recent literature. *Pediatric Dentistry* 22(4):269-277.
- McDonagh MS, Whiting PF, Wilson PM, Sutton AJ, Chestnut I, Cooper J, et al. (2000). Systematic review of water fluoridation. *British Medical Journal* 321(7265):855-859.
- Medical Research Council (2002). Water Fluoridation and Health. London: MRC.
- Milsom K, Mitropoulos CM (1990). Enamel defects in 8-year-old children in fluoridated and non-fluoridated parts of Cheshire. *Caries Research* 24(4):286-289.
- Ministry of Education (2007). Deciles information. Wellington: Ministry of Education.
- Nik-Hlissein NN, Majid ZA, Mutalib KA, Abdullah F, Abang A, Wan MN (1999). Prevalence of developmental defects of enamel among 16-year-old children in Malaysia. *Annals of Dentistry, University of Malaya* 6:11-16.
- Pitts NB, Evans DJ, Pine CM (1997). British Association for the Study of Community Dentistry (BASCD) diagnostic criteria for caries prevalence surveys -1996/97. *Community Dental Health* 14(S1):6-9.
- Rock WP (1994). Young children and fluoride toothpaste. *British Dental Journal* 177(1):17-20.
- Rock WP, Sabieha AM (1997). The relationship between reported toothpaste usage in infancy and fluorosis of permanent incisors. *British Dental Journal* 183(5):165-170.
- Suckling GW, Pearce EI (1984). Developmental defects of enamel in a group of New Zealand children: their prevalence and some associated etiological factors. *Community Dentistry and Oral Epidemiology* 12(3):177-184.
- Suckling GW, Brown RH, Herbison GP (1985). The prevalence of developmental defects of enamel in 696 nine-year-old New Zealand children participating in a health and development study. *Community Dental Health* 2(4):303-313.
- Tabari ED, Ellwood R, Rugg-Gunn AJ, Evans DJ, Davies RM (2000). Dental fluorosis in permanent incisor teeth in relation to water fluoridation, social deprivation and toothpaste use in infancy. *British Dental Journal* 189(4):216-220.
- Tavener JA, Davies GM, Davies RM, Ellwood RP (2004). The prevalence and severity of fluorosis and other developmental defects of enamel in children who received free fluoride toothpaste containing

either 440 or 1450 ppm F from the age of 12 months. *Community Dental Health* 21(3):217-223.

Vandenbroucke JP, von Elm E, Altman DG, Gøtzsche PC, Mulrow CD, Pocock SJ, et al. (2007). Strengthening the Reporting of Observational Studies in Epidemiology (STROBE): Explanation and Elaboration. *Epidemiology* 18(6):805-835.

Wang R, Lagakos SW, Ware JH, Hunter DJ, Drazen JM (2007). Statistics in medicine - reporting of subgroup analyses in clinical trials. *New England Journal of Medicine* 357(21):2189-2194.

Whelton H, Crowley E, O'Mullane D, Cronin M, Kelleher V (2003). Oral Health in Ireland 2002: Preliminary Results. Dublin: University College Cork.

World Health Organization (1994). Fluorides and Oral Health Report of a WHO Expert Committee on Oral Health Status and Fluoride Use. Technical Report Series: 846. Geneva: WHO.

PHILIP J. SCHLUTER<sup>ab</sup>, MSC, PHD  
SATHANANTHAN KANAGARATNAM<sup>c</sup>, BDS, MCOMDENT,  
DDPHRCS  
CALLUM S. DURWARD<sup>bd</sup>, BDS, MDSc, MPH, FRACDS  
ROBYN MAHOOD<sup>a</sup>, CERTDENTHER  
<sup>a</sup>AUT University, School of Public Health & Psychosocial  
Studies, Auckland, New Zealand  
<sup>b</sup>The University of Queensland, School of Nursing &  
Midwifery, QLD 4072, Australia  
<sup>c</sup>Auckland Regional Dental Service, Waitemata District  
Health Board, Auckland, New Zealand  
<sup>d</sup>Faculty of Dentistry, International University, Phnom  
Penh, Cambodia

Corresponding author: Professor Philip Schluter, Head of Research, School of Public Health and Psychosocial Studies, AUT University. Phone: +64 9 921 9999. Private Bag 92006. Fax: +64 9 921 9877, AUCKLAND 1142. Email: philip.schluter@aut.ac.nz

### CLINICAL ROOMS AVAILABLE (East Coast Bays)

Located centrally on Auckland's sunny east coast bays, our practice rooms have prominent street front appeal.

In an area of huge residential and commercial development, you will enjoy excellent market presence.

Well established podiatrist, sunny spacious rooms and sea views help ensure a pleasant, interactive environment.

Available due to redevelopment, these rooms (approx 18sq metres and 12 sq metres) enjoy very competitive rates and become available December 2008.

For expressions of genuine interest, please contact:  
Paul on (09) 478 5589  
or paulhames@woosh.co.nz

## on Handpiece Service



Repairing  
a broken handpiece is  
usually your most  
cost-effective option

# HAYES

THE LOCAL HANDPIECE REPAIR CENTER

Freephone 0800 426 374

Hayes Handpiece Centre  
11 Barrys Point Rd, Takapuna, Auckland  
Tel: 09 489 2494 Mob: 0274 723 807  
Email: jenk@clear.net.nz

[www.dentalstuff.co.nz](http://www.dentalstuff.co.nz)