Prevalence of caries in urban Australian aborigines aged 1-3.5 years

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Abstract

Purpose: This study investigated the prevalence and etiological factors associated with caries in a group of young Australian aboriginal children from 1 to 3.5 years of age.

Methods: One hundred and forty-seven healthy infants, aged from 1 to 3.5 years, attending a community health center in Brisbane, a nonfluoridated state capital city, were randomly selected for the study.

Results: The caries prevalence was 39% by subjects and 32% by the total number of teeth present. The mean number of decayed, filled teeth (dtf) was 2.5±0.4, which is more than twice the figure for 3-year-old children in Australia. Furthermore, the filled (f) component represented only 1% of the total dft, suggesting very low treatment levels. Increased caries experience of the infants was strongly associated with high dental plaque scores, high levels of Streptococcus mutans infection, and sleep-time consumption of milk containing added sugar.

Conclusion: The results suggest that urban Australian aboriginal infants are at high risk for caries, and that preventive strategies are urgently required. (Pediatr Dent 21:91–96, 1999)

Like other types of dental decay, caries in young children has been found to be polarized to certain population groups,[1–4] and is now thought to have reached serious levels in the socially disadvantaged, such as low income children,[5] indigenous populations[6–11] and poor immigrants.[12] A recent review[13] on the biological mechanisms involved in caries in young children suggests that biological risk factors are essentially similar to those of other types of caries, namely, early acquisition of *Streptococcus mutans*,[14] high frequency of sugar consumption,[15] poor oral hygiene[16] and possibly, enamel hypoplasia.[17–19] However, the etiological factors associated with caries in young children remain poorly understood. Although previous dental literature[2–4] emphasized the significance of sleep time use of the nursing bottle, other studies suggest that children subjected to similar feeding patterns did not develop dental caries.[20] Another related controversy centers on the cariogenicity of milk. Although in vitro and animal studies have suggested that milk is not cariogenic,[21–24] human clinical studies to support the laboratory results are not available. In this regard, whether plain cow's milk or breast milk consumed at sleep time may cause caries is of importance. In addition, the relative cariogenicity of milk containing added sugar compared to other sugar solutions is still unclear.

Although there have been two previous studies on older preschool Australian aboriginal children,[18, 25] there is little information on the oral health of very young children of this ethnic group. In particular, little is known about the prevalence of caries in these communities and the predisposing factors involved. This paper aims to determine the caries prevalence and etiological factors associated with caries in a group of urban Australian aboriginal infants and young children. It is likely that the results of this study may be applicable to other disadvantaged groups in other parts of the world.

Methods

Subjects

The subjects were Australian aboriginal young children, from 1 to 3.5 years of age, who resided in the metropolitan areas of Brisbane, a nonfluoridated state capital city. The infants were recruited from those presenting to the Aboriginal and Islander Community Health Center, which is one of two major community health centers for the indigenous population in the metropolitan area. All infants presenting to the center from the period November 1996 to July 1997 were invited for a dental examination as part of a health promotion program initiated by the dental team of the center.

Health, feeding, and brushing histories

Parents were requested to fill in a medical history form for the children, and were interviewed with regard to feeding histories during infancy and consumption of sugar in drinks and solid foods. In addition, information was collected regarding dental health habits such as previous dental visits, fluoride supplementation, frequency of tooth brushing, and whether parents assisted with toothbrushing. The data were recorded in comprehensive forms.

Dental examination

The subjects were examined in the dental clinic located at the community health center. Cooperative, older children were examined in the dental chair while the younger infants were examined using lap examination and normal dental lighting. The examinations were performed by two pediatric dentists (Dr. W. Kim Seow and Dr. A. Amaratunge), whose interexaminer variability has been calibrated as nonsignificant.[25]

Table 1. Demography of Study Population, and Prevalences of Dental Caries and Enamel Defects

	Overall	Boys	Girls
Demography			
Number (%) of children	147 (100%)	59 (40%)	88 (60%)
Age at examination (mean yrs ± SD)	3.1 ± 0.1	3.1 ± 0.2	3.1 ± 0.1
Range 1 to 3.5 yrs			
Birthweight (mean kg ± SD)	3.10 ± 0.8	3.3 ± 0.9	3.0 ± 0.8
Mother's education:			
Primary school	64 (44%)		
Secondary school	36 (24%)		
Not mentioned	47 (32%)		
Dental health habits			
No. (%) been to the dentist before	24 (16%)		
No. (%) taking F tablets	8 (5%)		
Mean daily brushing frequency	1.4 ± 0.7		
Parental assistance with brushing	89 (61%)		
Caries prevalence			
By subjects			
N (%) children with caries	58 (39%)	27 (45%)	31 (55%)
N (%) caries free	89 (61%)	33 (36%)	56 (64%)
Mean no. decayed, filled teeth	2.5 ± 0.4		
Mean decayed, filled teeth/Total teeth (dft/T ± SD)	0.32 ± 0.03	0.26 ± 0.03	0.35 ± 0.04
Filled teeth: N (%) carious teeth	8 (1%)		
By teeth			
N teeth	2541		
N (%) carious teeth	813 (32%)		
Developmental Defects of Enamel (DDE)			
Prevalence by subjects			
N (%) with at least one tooth with DDE	29 (20%)	13 (55%)	16 (45%)
Mean N ± SD of DDE teeth	3.4 ± 1.2	3.6 ± 1.4	3.1 ± 1.2
Prevalence by teeth			
Total (%) of teeth affected with DDE	104 (4%)		

World Health Organization (WHO) criteria were used for caries diagnosis[26] and caries experience was reported as number of decayed, filled teeth (dft). As there were no teeth extracted for caries in the children, the component "missing teeth" was not employed. Furthermore, as some of the children did not have the full primary dentition, it was pertinent to express caries experience (dft) as a percentage of total number of teeth present (T)—i.e., dft/T.

A modified Developmental Defects of Enamel (DDE) Index[27] was used for charting enamel defects. An opacity was recorded if there was a change in the translucency of enamel, and enamel hypoplasia was charted if there was quantitative loss of enamel or break in the enamel surface.[28]

Modified indices[29] were used for the charting of gingival inflammation and plaque scores. Gingival inflammation was assessed by gentle gingival probing on the facial and lingual surfaces of six key primary teeth (maxillary right central incisor, maxillary right and left second molars, mandibular right and left second molars and mandibular left central incisor). A score of 1 or 0 denoted the presence or absence of gingival bleeding respectively. The total score divided by the total number of surfaces obtained the percentage score.

Oral plaque scores were obtained using the same key teeth and surfaces as those used to assess gingival inflammation. A score of 1 or 0 denoted the presence or absence of plaque respectively.

Salivary *S. mutans* counts

A modified technique[30] using commercially available Dentocult Strip mutans kits (Orion Diagnostica, Espoo, Finland) was used to assess levels of salivary *S. mutans* in a group of randomly selected patients (presenting on alternate weeks). Briefly, a plastic spatula from the kit was rotated on the patient's tongue 10 times and placed into the vial of supplied culture broth containing bacitracin. The bacterial cultures were incubated at 37°C for 48 h, after which the plastic strips were removed and air dried. The density of *S. mutans* colony growth was assessed as being greater than 100,000 or less than 100,000 per mL of saliva using the standard charts provided by the manufacturer.

Statistical analysis

Student's *t*-tests, and Chi-square (χ^2) tests were used where appropriate. The c value for tests of significance was set at $P<0.05$.

Results

Demography

Altogether, 147 parents agreed to a dental examination of their children (consent rate of 99%). Table 1 shows the demography of the study group. There were 88 (60%) girls and 59 (40%) boys. Their mean age at dental examination was 3.1±0.1 yrs (range from 1 to 3.5 years). The majority of children were born full-term (97%) with mean birthweight 3.10 ± 0.87 kg. The remaining 3% were prematurely born with low birthweight (<2.5 kg). Except for asthma, which affected 22 (15%), most of the children were healthy.

Dental health habits

Only 24 (16%) had visited a dentist before. In spite of the fact that Brisbane is a nonfluoridated city, only eight (5%) were taking fluoride supplements. The mean daily brushing frequency of the children was reported to be 1.4±0.7, and 89 (61%) of the parents indicated that they assisted with brushing.

Caries prevalence

Table 1 shows the caries prevalence of the subjects. There were 58 children (39%) with caries, and 89 (61%) were caries-free. The mean dft index was 2.5±0.4. Out of a total number of 2541 teeth examined, 813 were carious, giving a total caries prevalence by teeth of 32%. There were eight filled teeth, which comprised only 1% of all the carious teeth.

Developmental defects of enamel (DDE)

As shown in Table 1, the prevalence of DDE was 20% by subjects (29 out of 147), and 4% by teeth (104 out of 2541). In subjects showing enamel hypoplasia, there was a significant association of enamel hypoplasia with dental caries *(P*<0.001, Table 2).

Table 2. Association of Dental Caries with Enamel Hypoplasia

Dental Caries	Developmental Defects of Enamel (DDE)		Total
	Present	Absent	
Present	37	55	92
Absent	72	329	401
Total	109	384	493

The association of dental caries with enamel hypoplasia was statistically significant ($\chi^2 = 21.49$, df = 1, $P < 0.001$).

Table 3. Association of *Streptococcus Mutans* Counts and Caries Experience *S. Mutans* Counts

	<100,000 bacteria per mL	>100,000 bacteria per mL
N (%) of subjects (N=42)	23 (55%)	19 (45%)
Mean dft/T	0.04±0.08	0.15±0.22

The difference in dft/T between the two groups of bacterial counts is statistically significant ($P<0.026$, $t=2.32$, df=40).

Table 4. Association of Gingival Inflammation Scores with Caries Experience

Gingivitis* (% of total score)	dft/T			
	0	0.01–0.25	0.26–1.0	Total
0	65	35	18	118 (80%)
1–49	3	1	0	4 (3%)
50–100	21	2	2	25 (17%)
Total	89	38	20	147 (100%)

*The differences in dft/T among the various groups are not statistically significant ($P > 0.1$).

S. mutans infection

Of the 42 children who were tested for *S. mutans* infection, 23 (55%) were scored as having >100,000 bacteria per mL of saliva, and 19 (45%) as having <100,000 bacteria per mL of saliva Table 3. The mean dft/T of the two groups was significantly different ($P<0.026$), suggesting that levels of *S. mutans* infection were directly correlated with levels of caries experience.

Gingival inflammation

As shown in Table 4., the majority of children (118, 80%) had no gingival inflammation (Score 0). Four (3%) had gingival scores of 1–49%, while 25 (17%) had gingival scores of 50–100%. In contrast to plaque scores, there were no significant association between gingival inflammation scores and caries experience ($P>0.1$).

Dental plaque scores

Sixty-seven children (46%) had low plaque scores of 0–25%, 17 (11%) had moderately high scores of 26–74%, and 63 (43%) had high oral debris scores of 75–100%. As can be observed from Fig 1, there was a strong association between high oral debris scores with high dft/T, and low oral debris scores with low dft/T ($\chi^2=127.5$, df=4, $P<0.001$).

Feeding patterns of children with and without caries

To determine the association of caries prevalence with various feeding patterns, the caries prevalence of those who consumed plain milk, milk containing added sugar, and breast milk during waking periods, and those who consumed milk during sleep were compared. The results (Table 5) showed that only seven out of 32 (22%) children who consumed plain milk during sleep had caries, compared with 13 out of 22 (59%) children who consumed milk with added sugar, and six out of 12 (50%) children who were breast fed during sleep. The difference in caries prevalence among the groups which consume different types of milk was statistically significant ($P<0.02$).

In contrast, when each milk group was considered separately, there were no statistical differences in caries prevalence between groups which consumed milk during sleep, and those which consumed milk during waking periods only ($P>0.1$).

Caries experience (dft/T) of children with different feeding patterns

Significantly higher caries experience was noted in children who consumed plain milk during sleep (0.42±0.06, $P<0.001$), or milk containing added sugar during sleep (0.37±0.06, $P<0.01$) compared to those who only consumed plain milk (0.24±0.09), or milk containing sugar (0.23±0.10) during waking periods only (Table 6).

Discussion

In general, there is a paucity of caries data of children from 1 to 3 years of age worldwide probably due to difficulties in accessing this young age group. Hence, although limited by the relatively small numbers of subjects, this study provides the first report on caries prevalence and treatment needs of a group of urban Australia aboriginal children from 1 to 3.5 years of age. Our results showed a high caries prevalence of 39%,

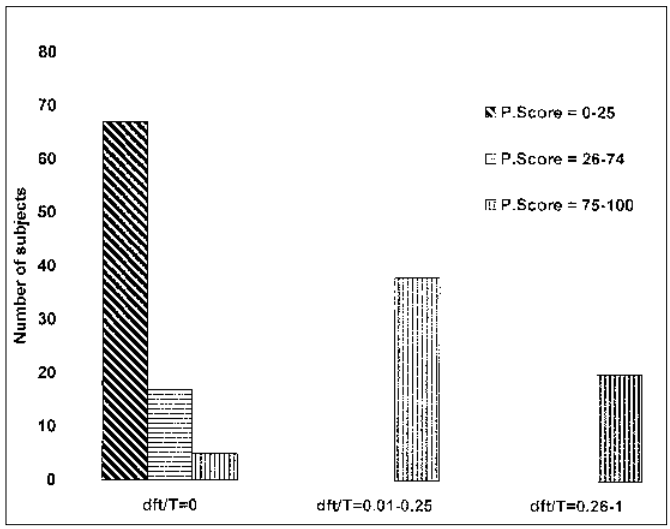

Fig 1. Relationship of plaque (p) scores and dft/T. The differences in dft/T among the various plaque score groups are statistically significant (χ^2 test=127.5, df=4, $P<0.001$).

Table 5. Feeding Patterns of Children With and Without Caries

	Milk Consumption During Sleep Time	Milk Consumption During Waking Periods Only	Total	*P* value*
Plain milk				
Caries Present	7	8	15	NS
Caries Free	25	27	52	
Total	32	35	67	
Breast milk				
Caries Present	6	11	17	NS
Caries Free	6	19	25	
Total	12	30	42	
Milk with added sugar				
Caries Present	13	5	18	NS
Caries Free	9	5	14	
Total	22	10	32	
P value**	*P*<0.02 (χ^2=8.20, df=2)	NS		

* Comparing caries prevalence in groups which consumed milk during sleep time with those which consumed milk during waking periods.
** Comparing caries prevalence in groups using different types of milk consumed.

and filled (restoration) component of only 1%. Also, the mean dft of this aboriginal group was 2.5±0.4 which is more than double the figure of 1.2 for all 3 year olds for other racial groups in Australia.[31]

For comparison, the overall prevalence of caries in children younger than 4 years of age in developed countries has been estimated to be no greater than 3-6%,[3] and may be even less.[32] In contrast, high caries prevalences in indigenous population groups and low socioeconomic communities have been reported. For example in the native North American populations, prevalences of 50%,[6] 53%,[7] 50–70%,[8] 44–90%,[9] 47%,[10] and 65%[11] have been demonstrated in separate studies of different communities. Other groups with similarly high caries rates are those in Head Start programs, which demonstrated caries prevalences of around 11–18% in 3–5 year old children,[33, 34] and immigrant children in whom prevalence figures have been reported to be as high as nearly 30%.[12] In one of the first large scale epidemiological studies of children younger than three years in the United States, Tang et al.[5] reported on the dental caries prevalence and its treatment in more than 5000 Arizona children aged five months through four years enrolled in Head Start or WIC programs or recruited at health fairs or day care centers. In that study, higher caries scores were associated with lower socioeconomic status, and the overall caries prevalence varied from 6% in 1 year olds, 25% in 2 year olds, 35% in 3 year olds, and 49% in 4 year olds.[5]

The low treatment rate of only 1% found in our study is similar to those reported for many disadvantaged groups at high risk.[5] In this study, the main reasons for the low treatment levels are probably related to lack of parental awareness of the significance of caries in the primary dentition which prevented them from bringing the children for dental examinations. Other reasons may be inadequate recommendations from health care personnel, and unwillingness or inability of practitioners to provide care for young children.

As with other at risk groups, in the Australian aboriginal children, the high predisposition to caries is likely to reflect their lower standards of general health compared to other racial groups.[35] The deterioration in health status in Australian aborigines has been related to the land dispossession and loss of hunter-gatherer lifestyles as well as the severe social dislocations into missions and reserves.[35] Although there are increasing numbers of aborigines settling in the major Australian cities in the last half-century, they generally have less education, fewer marketable skills, greater poverty, and a high unemployment rate.[36]

In the present study, high caries scores are strongly associated with high plaque scores, high *S. mutans* counts, and consumption of milk during sleep. Furthermore, moderate to high levels of plaque in more than half the children, indicating ineffective or nonexistent toothbrushing, is another significant caries risk factor in our study population. Also closely related to this finding is the observation that children with higher levels of *S. mutans* infection have higher caries experience.

Although there is widespread belief that milk has cariogenic potential, a recent review[13] into the cariogenicity of milk suggests that plain milk in laboratory studies does not induce caries. Furthermore, laboratory studies suggest that milk has many cariostatic properties such as a potential to decrease demineralization and increase remineralization.[21, 22] Decreased adherence of *S. mutans* to milk-treated enamel has also been observed.[23, 24] However, the results of laboratory and animal studies have not yet been confirmed clinically. In this regard, although the present study may be limited by problems associated with parental recall of feeding histories, it was found that sweetened milk ingested during sleep was associated with the highest caries prevalence. While the highly cariogenic ef-

Table 6. Caries Experience (dft/T) in Children with Different Feeding Patterns

	Milk Consumption During Sleep Time	Milk Consumption During Waking Periods Only	*P* value*
Plain milk	0.42±0.06 (N=32)	0.24±0.09 (N=35)	*P*<0.001 (*t*=4.29, df=13)
Breast milk	0.20±0.04** (N=12)	0.23±0.07 (N=30)	NS
Milk with added sugar	0.37±0.06 (N=22)	0.23±0.10 (N=10)	*P*<0.01 (*t*=3.50, df=14)

* Comparing dft/T in groups which consumed milk during sleep time with those which consumed milk during waking periods.
** *P*<0.001 (t=7.33, df=1. Comparing dft/T in groups which consume plain cow's milk and groups which consumed breast milk during sleep time).

fects of consuming sweet foods and drinks at sleep time have already been demonstrated in several previous studies,[37, 38] our results also showed that ingestion of breast milk or plain milk during sleep was associated with significant caries scores. Furthermore, there was a two-fold increase in mean caries experience (dft/T) in the group who drank either plain or sweetened milk during sleep, compared to the group who drank milk only during waking periods. The increase in caries in the group consuming milk at sleep time is probably related to the substantial decrease in salivary flow during sleep.[39] This causes a reduction in oral clearance of carbohydrate substrates, as well as decreases the concentration of protective factors from saliva such as buffers, remineralizing and antibacterial factors, thus leading to higher caries risk.

However, other factors may also influence caries experience. For example, it is likely that our results may have been influenced by the infants consumption of other fluids and solids containing sugar. It is likely that different patterns of milk consumption may be associated with particular dietary behaviors. For example, it is likely that children who take a bottle of milk to bed may also be given bottles of sweetened fluids such as cordials to take to bed,[40] as well as consuming frequent sugar snacks during the day.[41] Similarly, although rampant caries have been associated with exclusively breastfed children,[42] "at will" breastfeeding during sleep may reflect a more indulgent form of parenting which includes more liberal consumption of sweet snack foods.[15] Therefore, in those children who consume only plain milk during sleep time, foods and drinks containing sugars ingested at other times may put the teeth at even greater risk to caries.[43]

The significance of bottle feeding during sleep in the etiology of early childhood caries is currently controversial. Although the majority of previous studies[2–4] have suggested that rampant caries in infants is associated with prolonged use of the feeding bottle, in other investigations[44, 45] the results appear equivocal. In one well-controlled study,[20] children who took a bottle to bed did not experience more caries than a control group who did not. In Beijing, where baby bottles are generally not used, the prevalence of maxillary anterior caries was as high as 45%,[46] suggesting that other factors are also involved, although this fact does not indicate that the bottle has no role in caries.

Enamel hypoplasia, which was present in 20% of the children, is another likely predisposing factor in the etiology of infant caries, particularly in the case of preterm or medically compromised children.[28] The loss of surface integrity[47] and increased *S. mutans* colonization[48] in the defects are likely to predispose affected teeth to increased caries. As in previous studies on other Australian aboriginal children,[18] preterm children with low birthweight,[17] and rural Thai children,[19] the present study showed a strong association of enamel hypoplasia with caries.

Prevention of caries is one of the most important aspects of management. Although preventive strategies may be complicated by many complex psychoscocial and behavorial issues associated with socially disadvantaged groups,[49] parental educational programs[50, 51] regarding appropriate nursing and dietary changes, as well as oral hygiene and fluoride supplement use,[52] should be encouraged. As the low restoration rate of only 1% of all carious teeth found in this study suggests significant unmet dental need, early dental examination should be recommended.[53] In addition, screening for early caries and appropriate intervention such as application of fluoride varnish by dental auxiliaries may be considered.[54]

Conclusions

1. A high subject prevalence rate of caries of 39% was found in a group of urban Australian aboriginal children from 1 to 3.5 years of age.
2. Caries prevalence was associated with high levels of *S. mutans* infection, sleep time consumption of sweetened milk, and high plaque scores.
3. The treatment rate was only 1%, suggesting high unmet restorative need in this group of children.

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