Slade, Gary Douglas; Sanders, Anne Elizabeth; Bill, C. J.; Do, Giang Loc
Risk factors for dental caries in the five-year-old South Australian population Australian Dental Journal, 2006; 51(2):130-139

Australian Dental Journal 2006;51:(2):130-139

# Risk factors for dental caries in the five-year-old South Australian population

GD Slade,* AE Sanders,* CJ Bill,† LG Do*

**Abstract**

***Background:*** This study tested the hypothesis that risk behaviours in disadvantaged groups would explain socio-economic inequality in dental caries prevalence among preschool children.

***Methods:*** Using a case-control study, children with caries experience (one or more decayed, missing or filled primary tooth surfaces) and with no caries experience were sampled with known probabilities from among five year olds attending the South Australian Dental Service (SADS). Dental caries experience of primary teeth was recorded by SADS clinicians. Social and behavioural information was collected using a questionnaire mailed to parents. Prevalence rates, prevalence ratios (PR) and 95 per cent confidence intervals (95% CI) were computed, taking into account sampling probabilities.

***Results:*** Questionnaires were obtained for 64.6 per cent of sampled children (n=1398) and 40.2 per cent (95% CI=37.8–42.6) of them had caries experience. Five statistically significant risk factors were identified relating to previous feeding, current oral hygiene and parent's own oral health perceptions. The prevalence of four risk factors was greater in low-income households compared with high-income households ($P \leq 0.01$). In multivariate analysis, after adjusting for age of tooth cleaning onset, age at which toothpaste was introduced was not significantly associated with caries prevalence. Behavioural risk factors did not explain income-related gradients in caries prevalence but modified the level of risk associated with delayed onset of tooth cleaning. Children who delayed tooth cleaning until the age of 24 months or more and who were from low-income households had a 2.7-fold increase in caries prevalence (95% CI=2.1–3.4).

***Conclusions:*** Caries prevention efforts need to target behaviours in infancy and non-behavioural risk factors among preschoolers in low-income households.

*Key words:* Dental caries, risk factors, child, preschool, epidemiology.

*Australian Research Centre for Population Oral Health, Dental School, The University of Adelaide, South Australia.
†South Australian Dental Service.

***Abbreviations and acronyms:*** dmft = decayed, missing or filled primary teeth; SADS = South Australian Dental Service.

*(Accepted for publication 23 May 2005.)*

## INTRODUCTION

Surveillance surveys of dental caries among children enrolled in Australian school dental services revealed a steady decline in disease prevalence in all States and Territories in the 20 years from 1977. However, in 1996 the decline ceased and small but consistent increases in prevalence and number of teeth affected per child have since been observed in both primary and permanent dentitions.[1] Increases are most marked among five year olds where the mean number of primary teeth with clinically detectable untreated decay increased by 22 per cent in the three years to 1999.[1] Dental caries therefore is widespread at school entry. In 1999, 40 per cent of Australian five year olds had one or more decayed, missing or filled primary teeth (dmft) and the decayed component accounted for more than 80 per cent of the disease experience.[1]

Increases in population rates of dental decay are of particular concern among preschool-aged children. Unlike primary school children who have high participation rates in the school dental services, few preschool children make dental visits. In South Australia, the participation rate of four year olds in the school dental service is 40.2 per cent and is only 0.6 per cent in children aged less than four years (Lis Williams, personal communication). Thus preventive and treatment services delivered through school dental services will not be effective in reaching preschool children. It can be difficult to manage behaviour of preschool children if restorative dental treatment is required, with the result that some of them require general anaesthesia. Moreover caries in the primary dentition is not a self-limiting condition. A prospective cohort study from New Zealand showed that caries at five years predicted caries in adulthood even after controlling for childhood socio-economic status.[2] The appropriate public health response needs to be informed by epidemiologic inquiry to better understand how risk factors interact and why some preschool children are at elevated risk for dental caries.

A recent systematic review of 73 studies in the international literature identified 106 risk factors that were associated with the prevalence or incidence of caries in the primary teeth of children aged up to six years.[3] Collectively these included demographic, dietary, breast and/or bottle-feeding, oral hygiene practice, oral bacterial flora and other factors such as enamel hypoplasia and parental oral health. Existing Australian research into risk factors for caries in young children is mostly confined to small areas or groups that are not representative of the wider preschool population. One Melbourne study found that parental birthplace, ethnicity and occupation were significantly associated with caries in children aged five and six years.[4] Another Melbourne study that reported an eightfold difference in levels of tooth decay between preschoolers of Lebanese and Anglo-Saxon backgrounds found that maternal education explained 23 per cent of the variation in disease.[5] A study of children aged four to six years living in northern Brisbane identified non-Caucasian ethnicity, family language other than English, low socio-economic status[6] and single parent status[7] as sociodemographic risk factors and behavioural risk factors of sleeping with a bottle and sipping from a bottle. Breast-feeding between three and 12 months of age was protective.[8] Other research among children in Brisbane reported elevated levels of tooth decay among indigenous preschool children. The authors hypothesized that developmental enamel defects, low fluoride exposure, poor oral hygiene and a high sucrose diet increased risk of primary dental caries among children aged between three and six years.[9] Recently, a larger South Australian study[10] of children aged four to nine years found positive associations between primary caries experience and the consumption of water from sources other than the public water supply. In contrast, little is known about behavioural and sociodemographic risk factors for dental caries among South Australian preschool children.

The aim of this study was to identify the influence of behavioural and sociodemographic risk factors during preschool life on dental caries prevalence among the population of five-year-old South Australian children receiving care in the School Dental Service. Here, the term 'risk factor' is used to mean 'an attribute or exposure that is associated with an increased probability of . . . disease. Not necessarily a causal factor'.[11] Agreement is yet to be reached on a case definition for epidemiological studies of early childhood caries. For example, in their evaluation of NHANES III estimates of early childhood caries, Kaste and colleagues[12] used more than one definition to define cases. Furthermore, the most liberal definition recommended by Drury and colleagues[13] includes pre-cavitated lesions, which were not measured in our study. For these reasons, we used a more conservative approach to case definition and use the term 'dental caries' when reporting our results from this study. The study hypothesis was that children from socio-economically disadvantaged backgrounds would have greater prevalence of caries than children from socio-economically advantaged backgrounds but that this inequality would be accounted for by a disproportionate concentration of behavioral risk factors among socio-economically disadvantaged groups.

## METHODS

Data for this analysis came from a case-control study that was designed to examine effects of a preschool dental programme on dental caries experience of five-year-old children. Because cases and controls were sampled with known probability from the target population (see below), we were able to use the same dataset for this current analysis of the influence of sociodemographic and behavioral risk factors on dental caries prevalence.

### Study population and sampling

The target population was five-year-old South Australian children attending the South Australian Dental Service (SADS). At the time of sampling, 12 436 five year olds were enrolled in SADS, which represents 66.6 per cent (n=18 662) of the estimated State population of five year olds in June 2003.[14] Clinical findings from dental examinations undertaken by SADS' dentists and dental therapists were recorded electronically on a computerized dental chart which captured surface level caries experience for each primary and permanent tooth. Dental decay was recorded at the level of enamel cavitation and could be detected wholly by clinical examination, radiographs or both. Clinical examinations were made using visual criteria, and clinicians elected whether or not to additionally use compressed air and/or an explorer. Bitewing radiographs were used when needed, based on the judgment of the examining clinician. Apart from clinical details, the child's name, address and date of birth were also obtained from the computerized database.

The computerized examination database was used to define four sampling strata for this study: (1) Group A cases were defined as children with one or more decayed (d) primary tooth surfaces, but no primary tooth surfaces that had been filled (f) or extracted (m) due to caries (d≥1; m+f=0); (2) Group B cases were defined as children with no decayed primary tooth surfaces, but one or more primary tooth surfaces that had been filled or extracted due to caries (d=0; m+f ≥1); (3) Group C cases were defined as children with one or more decayed primary tooth surfaces and one or more primary tooth surfaces that had been filled or extracted due to caries (d≥1; m+f≥1); (4) Controls were defined as children who had no caries experience in their primary teeth (d=0; m+f=0).

Variation between uncalibrated examiners at this level of classification was less likely to affect case definitions, and hence unlikely to affect the results of the study, compared with studies that report dmf indices.

Sampling was achieved using the computerized records of all comprehensive dental examinations conducted among children in the target population in the period July 2002–June 2003. Where children had more than one comprehensive oral examination recorded in the period, data from the first examination were selected. Excluded from the sample were children examined at the Minda home for the disabled and at the Pika Wiya Health Service for indigenous children, yielding examination data from 77 SADS clinics distributed throughout the State. After selecting all 77 clinics, sampling was clustered within each clinic in the following way: (a) Children were first categorized into one of the four sampling strata defined above. (b) All children in the numerically smallest stratum were sampled. (c) Then, an equal number of children was sampled at random from each of the three remaining strata. This yielded a total of 2164 sampled children, 541 in each of the four sampling groups described above.

### Questionnaires mailed to parents

A 12-page questionnaire booklet was developed inquiring about aspects of oral health of preschool children that have been reported previously. Questionnaires were mailed to the parent or guardian of the sampled child at the residential address of the child recorded in the SADS computerized patient database. A cover letter, signed by the Executive Director of SADS, explained that participation was voluntary, and encouraged parents to complete the questionnaire and to mail it to the researchers using a reply-paid envelope included for that purpose. Parents/guardians who did not respond were sent up to one reminder card and two reminder letters with replacement questionnaires. Eighty-one questions were grouped into seven sections: birth characteristics (birth weight, Apgar score, gestational age and singleton or multiple birth status); family structure and sociodemographic details of the child's household; history of toothache; access to and utilization of information and services for child dental care, early feeding practices; past and present tooth cleaning practices and parental knowledge and ratings of general and oral health status. Questionnaire items comprised a combination of open-ended and pre-defined response categories to which parents/guardians responded by ticking a box (e.g., for answer categories such as 'yes' or 'no') or recording a number (e.g., age in months that tooth cleaning began). Parents/guardians consenting to have their child's dental records included in the study also signed a consent form attached to the back of the questionnaire.

Data from questionnaires were keypunched and merged with information from the examination database used for sampling. Data were checked and edited for out-of-range errors and internal inconsistencies. To address the study aim, caries prevalence was chosen as the dependent variable, defined as the weighted percentage of children in the target population who had one or more primary tooth that was decayed, missing or filled due to caries (i.e., the weighted percentage of children in any of the three groups of cases).

### Data analysis

Response rates were compared among the four sampling strata using unweighted data. Subsequent analyses evaluating our study aim used weighted data. The analytic strategy began by grouping children into mutually exclusive categories of each hypothesized risk factor. For hypothesized risk factors that were not dichotomous, this entailed inspection of univariate distributions for each variable, and subsequent categorization into two or three groups, based either on precedents reported in the literature (e.g., to define two categories of birth weight <2500g versus ≥2500g), clinically meaningful distinctions (e.g., feeding practices that occurred before or after tooth eruption) and distribution of data (e.g., annual household income ≤1200, ≤$31 200, $31 200 to less than $52 000 and ≥$52 000). For each hypothesized risk factor, caries prevalence was then compared between categories and a prevalence ratio and its corresponding 95 per cent confidence interval (95% CI) was computed. Prevalence ratios were computed by dividing the observed prevalence for the hypothesized high-risk (exposed) group by observed prevalence for a nominated low-risk (unexposed) group (referred to hereafter as the reference group). Under the null hypothesis of no association between the risk factor and caries prevalence, the prevalence ratio had a value of one, and the alternative to the null hypothesis could be rejected statistically if the 95 per cent confidence interval for the prevalence ratio excluded the value of one. To evaluate the study aims prevalence ratios were calculated, rather than odds ratios. Although commonly used in case-control studies, the latter were a poor approximation of risk when the condition being investigated was not infrequent,[15] as occurred in this study.

In order to consider the joint effects of behavioural and socio-economic risk factors, we next compared the percentage of children with identified risk factors in low-income (<$31 200) versus high-income households ($31 200 or more). Where the difference in percentage reached a nominal statistical threshold of $P<0.10$, we undertook stratified analyses to evaluate potential effect modification by computing prevalence ratios separately for income strata. Prevalence ratios that were found not to be homogenous among strata, as evidenced using the Breslow-Day test[16] were identified. Finally, to consider joint effects of all risk factors found to be statistically significant in preceding analyses, we constructed a multivariate, binary logistic regression model. Because the logistic regression model computed the log (odds) of caries, we converted parameter estimates for odds ratios to prevalence ratios and calculated their corresponding 95 per cent confidence intervals using methods we have described previously.[17] In this way, parameter estimates from the logistic regression model were used to compute predicted prevalence rates and ratios for the separate risk factors after controlling for other risk factors.

To test our hypothesis that elevated risk associated with low socio-economic status could be accounted for by behavioural risk factors, we first entered behavioural risk factors into this model, then added socio-economic status. If the preceding stratified analysis provided evidence of effect modification (i.e., Breslow-Day test P<0.05), we included interaction terms between socio-economic status and the risk factor. We reasoned that our study hypothesis would be supported if low socio-economic status did not persist as a statistically significant risk factor in this multivariate model (i.e., if the 95 per cent confidence interval for socio-economic status included one).

### Adjustment for different probabilities of sampling and clustered sampling design

In order to generate valid estimates of prevalence in the population, it was necessary to calculate sampling weights that adjusted for the unequal probability of inclusion of children due to sampling design and because of differences among sampling strata in response rates. Consequently, sampling weights were calculated as the inverse of the probability of response for each child. Within each clinic, probability of response was calculated for each of the four sampling strata. This unit record weight was computed as the number of respondent children in the stratum divided by the number of sampled children in the stratum.

Because children were sampled within clinics, these data were clustered, and therefore variance estimates that assume simple random sampling are invalid.[18] We therefore undertook statistical analyses using SUDAAN software, Release 9.[19] For these analyses, we used SUDAAN's Taylor series approximation to calculate variance, 95 per cent confidence intervals and P-values. However, SUDAAN does not compute Breslow-Day statistics from stratified analyses, so we instead computed the Breslow-Day statistic in SAS then divided the result by 1.91 which was the sampling design effect obtained from SUDAAN, to yield an adjusted chi-square test. The sampling design effect was the factor by which variance in this study was greater than that which would have been found had we used a simple random sampling design.

The primary benefit of undertaking weighted analysis was to produce estimates of prevalence and prevalence ratios that could be generalized to the target population of five year olds enrolled in SADS. The primary drawback of undertaking weighted analysis was to reduce statistical power by a factor of 1.4 (the square root of the sampling design effect). This means that we had less capacity to identify statistically significant risk factors than would have been the case had we undertaken a cross-sectional study with simple random sampling.

### Sample size

The study was designed to provide sample size requirements for its case-control study design, in which we aimed to have power of 0.80 to detect an odds ratios of 2.0 or more (equivalent to a prevalence ratio of 1.5 or more) for associations between risk factors differences between individual strata, with type I error of P<0.05. This created a requirement of 307 children in each of the four sampling strata, and therefore a total of 1228 subjects. For the current analysis of caries prevalence, the final sample size of 1398 children had power of 0.80 to detect a prevalence ratio of at least 1.3 with type I error of P<0.05 comparing dichotomized risk groups, where as few as 25 per cent were in the high-risk group, and after adjusting for an expected sampling design effect of 1.9 due to clustering.

### Ethical conduct of research

Parents/guardians of children reported in this analysis provided informed, signed consent for their participation. This project was reviewed and approved by The University of Adelaide's Human Research Ethics Committee and by the Board of the South Australian Dental Service.

**Table 1. Response rates among sampling strata and clinic locations**

| Sub-group* | | No. of children sampled | No. of children with returned questionnaire | % with returned questionnaire |
|---|---|---|---|---|
| Sampling stratum† | d=0, m+f=0 | 541 | 373 | 69.0 |
| | d=0, m+f>0 | 541 | 359 | 66.4 |
| | d>0, m+f=0 | 541 | 329 | 60.8 |
| | d>0, m+f>0 | 541 | 337 | 62.3 |
| Clinic location‡ | Adelaide | 1316 | 826 | 62.8 |
| | rest of state | 848 | 572 | 67.5 |

*d=no. of decayed primary teeth; m+f=no. of missing or filled primary teeth.
†P=0.02 (Chi-square test, 3df) for null hypothesis that response rates are equal among four strata.
‡P=0.02 (Chi-square test, 1df) for null hypothesis that response rates are equal in Adelaide and rest of state.

**Table 2. Bivariate relationships between sociodemographic and behavioural characteristics and caries prevalence**

| | | n [a] | % with characteristic[b] | Prevalence: % with dmft>0 | Prevalence ratio | 95% CI[c] |
|---|---|---|---|---|---|---|
| Sociodemographic characteristics | | | | | | |
| Sex of child | Male | 699 | 50.1 | 42.5 | 1.12 | 0.95-1.33 |
| | Female | 699 | 49.9 | 37.9 | ref | |
| Indigenous status of child | Indigenous | 28 | 1.5 | 66.6 | 1.68 | 1.05-2.68 |
| | Non-indigenous | 1364 | 98.3 | 39.8 | ref | |
| Single parent status | Yes | 242 | 16.2 | 51.3 | 1.35 | 1.10-1.65 |
| | No | 1145 | 83.2 | 38.0 | ref | |
| Language spoken by parent at home | Not English | 213 | 14.4 | 46.5 | 1.19 | 0.90-1.58 |
| | English | 1173 | 84.9 | 39.1 | ref | |
| Concession status | Health card holder | 526 | 35.5 | 47.5 | 1.31 | 1.08–1.59 |
| | Non-card hold | 858 | 63.7 | 36.2 | ref | |
| Annual household income | Low (<$31 200) | 419 | 27.5 | 49.7 | 1.55 | 1.21–1.98 |
| | Mod ($31 200-$51 999) | 390 | 27.2 | 39.7 | 1.24 | 0.9 –1.59 |
| | High ($52 000+) | 444 | 36.0 | 32.1 | ref | |
| Behavioural characteristics | | | | | | |
| Consumed sweet drinks regularly when aged <6 months | Yes | 511 | 34.3 | 46.3 | 1.26 | 1.04-1.52 |
| | No | 887 | 65.7 | 36.9 | ref | |
| Slept with bottle containing sweet drink | Yes | 487 | 32.1 | 48.4 | 1.35 | 1.14–1.59 |
| | No | 891 | 67.1 | 35.9 | ref | |
| Used sweetened dummy | Yes | 79 | 5.0 | 48.6 | 1.23 | 0.87–1.73 |
| | No | 1309 | 69.1 | 39.6 | | |
| Age weaned (breast) | 18 months or more | 153 | 8.5 | 51.3 | 1.39 | 1.09–1.77 |
| | By 18 months | 948 | 71.7 | 36.9 | ref | |
| Age started tooth cleaning | 24 months or more | 359 | 23.7 | 52.8 | 1.64 | 1.35–2.00 |
| | 12-<24 months | 511 | 39.2 | 39.8 | 1.24 | 1.00–1.53 |
| | By 12 months | 412 | 28.1 | 32.2 | ref | |
| Age started toothpaste | 24 months or more | 593 | 41.2 | 46.6 | 1.29 | 0.92-1.81 |
| | >12, <24 months | 595 | 17.8 | 36.1 | 1.00 | 0.73–1.37 |
| | By 12 months | 103 | 32.3 | 36.1 | ref | |
| Parent's self-rated oral health | Fair, poor | 438 | 27.9 | 50.8 | 1.41 | 1.18–1.69 |
| | Good, very good or excellent | 952 | 71.6 | 36.0 | ref | |
| Birthweight | <2500gms | 69 | 4.6 | 40.3 | 1.01 | 0.69–1.46 |
| | 2500gms or more | 1210 | 86.8 | 40.1 | ref | |

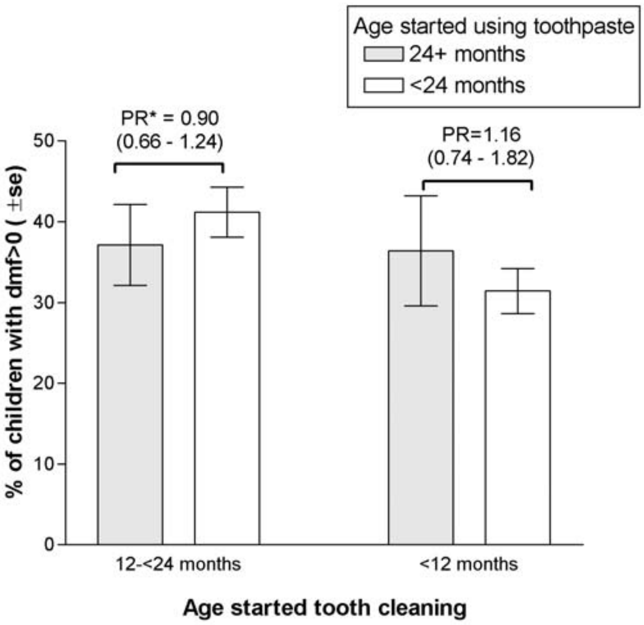
(a) Unweighted number of subjects.
(b) Weighted percentage of subjects.
(c) 95% confidence interval for prevalence ratio.

## RESULTS

Completed questionnaires were obtained from 1398 children, representing a response rate of 64.6 per cent among the 2164 children sampled for the study. Response rates varied among the four case-control sampling strata from 61 per cent of children who had decay but no filled or missing teeth to 69 per cent of children who had no caries experience (Table 1). Children sampled from Adelaide had a lower response rate than children sampled from the rest of the State. Although not shown in Table 1, the response rate of non-Adelaide residents was greater by an average of five per cent within each sampling stratum compared with the rate for Adelaide residents (Cochran-Mantel-Haenszel chi-square, 1 df, P=0.02).

The weighted percentage of children with caries experience (dmf>0) was 40.2 per cent (95% CI=37.8–42.6 per cent). This weighted estimate of 40.2 per cent with dmft>0 was lower than the unweighted percentage of sampled children who were cases because cases were sampled with a greater probability than non-cases. They consisted of 7.9 per cent (95% CI=7.2–8.7 per cent) with d=0 and m+f>0; 21.5 per cent (95% CI=19.9–23.2 per cent) with d>0 and m+f=0; and 10.8 per cent (95% CI=9.6–12.1 per cent) with d>0 and m+f>0. Among respondents, half of the children were male, 1.5 per cent were Aboriginal or Torres Strait Islander, 14.4 per cent had a parent who spoke a language other than English, 16.1 per cent lived in a single-parent household and 35.6 per cent had a parent who had a health care concession card. Similar proportions of children lived in households that had low (27.5 per cent) or moderate levels (27.2 per cent) of annual income and the remainder lived in households with higher income. Caries prevalence was significantly elevated among indigenous children, children living in single-parent households, children whose parent had a health care concession card, and children living in low-income households, as evidenced by prevalence ratios with 95 per cent confidence intervals that excluded one (Table 2). The largest sociodemographic inequality in prevalence was observed for indigenous children whose caries prevalence was 1.7-fold greater than non-indigenous children.

Five putative risk factors were associated with statistically significant increases in prevalence of caries (Table 2). Children whose tooth cleaning began after the age of 24 months had a 1.64-fold increase in




*PR=Prevalence ratio (95% confidence interval in parentheses).
Breslow-Day test for homogeneity of association=1.29 (SAS estimate) ÷1.91 (SUDAAN design effect)=0.67, P=0.41.
Mantel-Haenszel adjusted prevalence ratio=0.97, 95%CI=0.75-1.25.

**Fig 1.** Association between age that toothpaste usage began and caries prevalence in children with early and late initiation of tooth cleaning.

prevalence of caries compared with children whose tooth cleaning began in the first year of life, whereas initiation of toothbrushing between the ages of 12 and 24 months was associated with a 1.24-fold increase in prevalence. Other statistically significant prevalence ratios ranged in magnitude from 1.35 to 1.41. Using a sweetened dummy and weighing less than 2500g at birth were not associated with statistically significant variations in caries prevalence (Table 2).

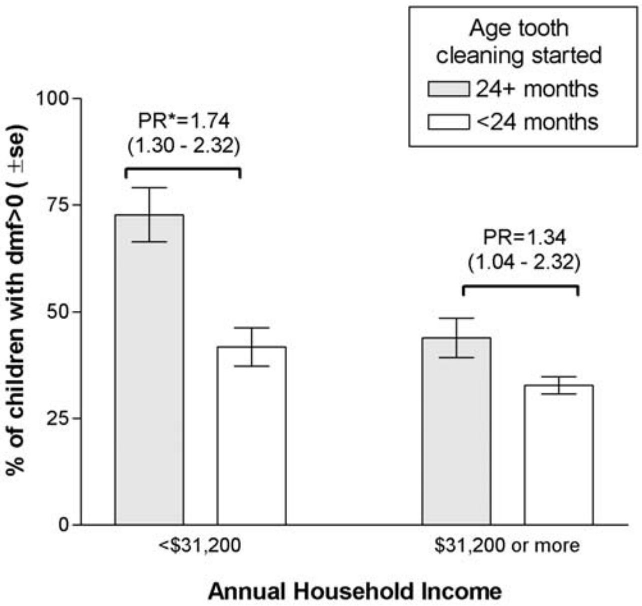
Some 45 per cent of children delayed initiation of toothpaste beyond the age of 24 months, and while this was associated with a 1.29-fold increase in caries prevalence, it was not a statistically significant increase (95% CI=0.92–1.81) compared with children who began using toothpaste before the age of 12 months (Table 2). Furthermore, unlike the gradient in prevalence observed across three age groups of tooth cleaning initiation, caries prevalence among children who began using toothpaste in the intermediate age range of 12–24 months did not differ compared with children who began using toothpaste before the age of 12 months. Although not reported in the Tables, 87 per cent of children reportedly began using a children's low concentration fluoride toothpaste, while 11 per cent reportedly began with a standard fluoride toothpaste and two per cent began with a non-fluoridated toothpaste. By five years of age, 67 per cent of children reportedly were using a children's low concentration fluoride toothpaste and 31 per cent were using a standard fluoride toothpaste. The type of fluoridated toothpaste first used or used at the age of five years was not associated with caries prevalence.

Effects of tooth cleaning and use of toothpaste were isolated in stratified analyses of the subgroup of 896 children who began cleaning before the age of 24 months and dichotomizing toothpaste initiation into two age groups: less than 24 months versus 24 months or more (Fig 1). This dichotomy was used because 24 months was the age at which toothpaste usage was associated with caries prevalence in bivariate analysis (Table 2). Within the stratum of children who began tooth cleaning before the age of 12 months, the age at which toothpaste was started was not associated with caries prevalence (prevalence ratio=1.16) (Fig 1). There was a similar lack of association between toothpaste initiation and caries prevalence in the stratum of children who began tooth cleaning between the ages of 12 and 24 months and the non-significant Breslow-Day test (P=0.41) supported pooling of these prevalence ratios. The pooled Mantel-Haenszel adjusted prevalence ratio for delayed (≥24 month) toothpaste initiation among the two strata of tooth cleaning initiation was 0.97 (95% CI=0.75–1.25), indicating no significant influence of the age of toothpaste initiation on caries after adjustment for the age of tooth cleaning initiation.

Of the five risk factors identified in Table 2, four were observed more frequently among low-income households compared with high-income households at a nominal threshold of P≤0.10 (Table 3). When the relationship between each risk factor and dental caries was stratified between the two income categories, statistically significant effect modification was found between income and the age of tooth cleaning initiation (Fig 2). Among children in low-income households, tooth cleaning that was initiated at the age of 24 months or more was associated with a 1.74-fold increase in caries prevalence compared with cleaning initiated at a young age, whereas among children in high-income households it was associated with only a 1.34-fold increase in prevalence. This difference in prevalence ratios was statistically significant based on the Breslow-Day test (P=0.05).

**Table 3. Distribution of risk factors between income two categories**

| Risk factor | Income <$31 200 | Income ≥$31 200 | Chi-square P-value |
|---|---|---|---|
| % who consumed sweet drinks regularly when aged <6 months | 41.7 | 30.5 | 0.02 |
| % who slept with bottle containing sweet drink | 39.6 | 29.8 | 0.04 |
| % weaned from breast when aged ≥18 months | 11.1 | 10.1 | 0.74 |
| % who started tooth cleaning when aged ≥24 months | 29.9 | 23.7 | 0.10 |
| % with parent having 'fair' or 'poor' self-rated oral health | 32.4 | 25.3 | 0.08 |




PR = Prevalence ratio (95% confidence interval in parentheses). Breslow-Day test for homogeneity of association=7.52 (SAS estimate) ÷1.91 (SUDAAN design effect)=3.94, P=0.05.

**Fig 2.** Association between age that tooth cleaning began and caries prevalence in two income groups.

In multivariate analysis, three risk factors persisted as independent influences (P<0.05) on caries prevalence: sleeping with a bottle containing sweet drink, regular consumption of sweet drinks in the first six months of life and parent's fair/poor self-rated oral health (Table 4). Age of initiating tooth cleaning was an additional statistically significant risk factor, but its effect was conditional upon household income, as evidenced by a multiplicative interaction (P=0.04) (Table 4). Other sociodemographic characteristics and putative risk factors considered in previous tables were excluded from the multivariate model because they were not statistically significant. When parameter estimates from the logistic regression model were used to compute predicted prevalence rates, the group with the greatest elevation of prevalence was children from low-income households whose tooth cleaning began at the age of 24 months or more (prevalence ratio = 2.66) (Table 5). In contrast, children from high-income households whose tooth cleaning began at the age of 24 months or more had a smaller increase in caries prevalence (prevalence ratio = 1.41) while prevalence was not significantly elevated for children from low-income households whose tooth cleaning began before 24 months of age.

## DISCUSSION

While this study confirmed that a number of sociodemographic and behavioural risk factors influence caries prevalence in this population of five year olds, our hypothesis that behavioural risk factors would account for socio-economic inequalities in caries was not supported. Behavioural risk factors attenuated the socio-economic gradient but did not completely explain why dental caries is more prevalent among children from households with low income. Instead, a major finding of this study was that caries prevalence was influenced by an interaction between household income and the age that tooth cleaning began (Table 4). Although delayed onset of tooth cleaning (beyond two years of age) was a risk factor for caries prevalence in both household income groups, the risk of delayed cleaning onset was greater for children from low-income households (Table 5). This suggests that for more advantaged children the risk of delayed tooth cleaning on oral health is buffered by other protective factors associated with socio-economic status. In a UK study, Gibson and Williams[20] reported an interaction

**Table 4. Multivariate logistic regression model for odds of caries**

| Effect | df | Parameter estimate | se | P-value |
|---|---|---|---|---|
| Intercept | 1 | -1.17 | 0.13 | <0.01 |
| Slept with bottle containing sweet drink | 1 | 0.44 | 0.17 | 0.01 |
| Regular consumption of sweet drinks aged <6 months | 1 | 0.43 | 0.20 | 0.03 |
| Fair/poor parent's self rated oral health | 1 | 0.62 | 0.18 | <0.01 |
| Started cleaning teeth aged ≥24 months | 1 | 0.48 | 0.22 | 0.03 |
| Low annual household income (<$31 200) | 1 | 0.29 | 0.22 | 0.19 |
| Interaction: age of brushing x annual household income | 1 | 0.94 | 0.26 | 0.04 |

For full model with 7 degrees of freedom, Wald F-statistic=20.0, P<0.001.

**Table 5. Independent effects of risk factors on predicted caries prevalence**

| Risk factor | Predicted prevalence[(a)] | Prevalence ratio (95% CI)[(b)] | |
|---|---|---|---|
| Slept with bottle containing sweet drink | 33% | 1.4 | (1.1-1.7) |
| Regular consumption of sweet drinks aged <6 months | 32% | 1.4 | (1.0-1.8) |
| Fair/poor parent's self rated oral health | 37% | 1.5 | (1.2-2.0) |
| Started cleaning teeth at age ≥24 months and income <$31 200† | 63% | 2.7 | (2.1-3.4) |
| Started cleaning teeth at age ≥24 months and income ≥$52 000† | 33% | 1.4 | (1.1-1.9) |
| Started cleaning teeth at age <24 months and income <$31 200† | 29% | 1.2 | (0.9-1.7) |
| Reference group | 24% | | |

(a) Prevalence rates and ratios are computed from multivariate logistic regression model in Table 4.
(b) 95% CI is 95% confidence interval for prevalence ratio. Prevalence ratios are relative to the reference group comprising children who had none of the risk factors listed in this table.

between social class and oral hygiene among children of preschool age that suggested a protective effect of social advantage. They found that toothbrushing frequency was associated with significantly lower caries prevalence in children whose father's occupation was non-manual. In fact, the odds of having dental caries halved in children who brushed twice a day or more often compared with those who brushed less often. Yet for children whose father's occupation was classified as manual, toothbrushing frequency was not significantly associated with caries prevalence. The authors suggested that toothbrushing may be more effective in the non-manual groups and supported this argument with evidence that children in the manual occupation group were more likely to brush their own teeth than were the children in the non-manual occupation group.[20]

Consistent with earlier studies[3] we found that sleeping with a bottle of sweet drink and regular exposure to sweet drinks in the first six months of life were behavioural risk factors. In addition, parental self-rating of their own oral health as fair or poor was significantly associated with caries prevalence in their child, independently of the effects of the behavioural and sociodemographic risk factors investigated. We interpreted parent's rating of their own oral health as a marker of increased risk for children's decay due to parental transmission of cariogenic bacteria, general attitudes and behaviours regarding oral health within the family, or both.

An unexpected finding in this study was that, after controlling for the age of initiating tooth cleaning, the age at which toothpaste usage began was not significantly associated with caries prevalence. To date few studies have tested the effectiveness of fluoride toothpaste on caries incidence in the primary dentition.[21] Of these studies, only one reported a significant reduction in caries increment compared to placebo.[22] However, that study in 1982 sampled older children aged six to eight years. This and most other randomized clinical trials of fluoride toothpaste were conducted more than two decades ago when caries prevalence was substantially higher. In addition, there is little research examining the effectiveness of low-concentration fluoride toothpaste which was the predominant form reported by the study participants. Winter *et al.*[23] reported lower, but statistically non-significant, differences in caries levels when comparing low-concentration toothpaste to an adult-strength toothpaste in a three-year clinical trial of preschool aged children. However, Winter *et al.*[23] did not use a placebo toothpaste, so it is not known if either fluoride concentration differed significantly from a non-fluoridated toothpaste in this young study population that had low levels of caries.

This finding implies that early onset of tooth cleaning reduces the risk of caries but that early introduction of toothpaste does not confer any additional protection from dental caries. While 98 per cent of children reportedly used fluoride toothpaste, at least two-thirds of parents said it was a low-concentration children's formulation, and fluoride concentration was not associated with caries prevalence. However, we are cautious in interpreting these results because parents responding to these questions were asked to recall details of oral hygiene practices that occurred some three to four years earlier. It is a maxim of epidemiological research that estimates of association (in this study, prevalence ratios) are not biased due to recall error so long the likelihood of recall error is similar among cases with disease and non-cases without disease.[15] Instead, 95 per cent confidence intervals become wider than would have been the case in the absence of recall error. We have no reason to believe that the presence of caries in a five-year-old child would alter the extent of error in parent's recall of oral health behaviours, so we think there is reasonable grounds to believe that the magnitude of prevalence ratios have not been markedly affected by recall error.

We did not find low birth weight to be a significant risk factor for caries prevalence in bivariate analysis. This was perhaps not surprising given the complexity of the relationship between low birth weight and primary caries and the inconsistent findings reported previously. A UK study, for example, found that children aged three to four years with birth weight in the normal range had a significantly higher dmft score than had pre-term low birth weight children.[24] Many epidemiologic studies both in developed and developing nations, some using nationally representative samples, have reported negative associations between socio-economic resources and low birth weight. For example, in a representative sample of US births, Parker and colleagues[25] found that all measures of socio-economic status were associated with birth weight, irrespective of race. Several investigators have hypothesized a relationship between low birth weight and early childhood caries that may be mediated by nutritional deficiencies *in utero* and the subsequent development of enamel defects and hypoplasia in the primary dentition. Yet there is no epidemiological evidence to support this relationship at this time,[26] and we did not collect information about enamel defects.

Despite finding that exposure to sweetened drinks was a risk factor, we did not find that the low use of a sweetened dummy was associated with statistically significant variations in caries prevalence in bivariate analysis. Our non-significant finding was consistent with the conclusions of an evaluation of the evidence for the association between dummy use, with and without sweetener, and early childhood caries that found no strong or consistent associations. In fact, some evidence reviewed pointed to dummy use as offering a mildly protective effect.[27]

While our study evaluated the joint influences on dental caries of common behavioural risk factors and sociodemographic characteristics, we did not address other risk factors such as enamel hypoplasia or oral bacterial flora. There is good evidence that the intra-oral presence of high levels of *Streptococcus mutans* is associated with increased risk for caries in preschool children, yet there is also evidence that socially mediated behaviours such as good oral hygiene and a non-cariogenic diet can moderate the adverse impact of these bacteria.[3] Furthermore, while a number of studies show that dental treatment,[28] chlorhexidine gel[29] and behavioural interventions[30] can reduce levels of salivary mutans streptococci in preschool children, there is not good evidence that this reduction of oral bacteria is associated with a subsequent reduction in the risk of incident caries.[28]

An additional limitation was that questionnaires were not returned from 35 per cent of sampled children and we have no information about potential differences in distribution of risk factors between respondents and non-respondents. While the weighting scheme adjusted for non-response, it made an implicit assumption that, within each of the four sampling strata, those risk factors were distributed identically in respondents and non-respondents. Yet we found that non-respondents were more likely to be Adelaide residents than non-Adelaide residents in all four sampling strata and hence it was probable that some other characteristics influencing caries prevalence also differed between respondents and non-respondents.

We chose to quantify associations between putative risk factors and dental caries using prevalence ratios, rather than odds ratios, because when prevalence is high (e.g., greater than 20 per cent), odds ratios are a poor approximation of the prevalence ratio. One consequence is that the observed strength of association between risk factors and caries reported here appears considerably smaller than findings from other studies that have reported odds ratios. To illustrate this point, the prevalence ratio of 1.7 for indigenous children reported in Table 2 was equivalent to an odds ratio of 3.0. This highlights the bias inherent in interpreting odds ratios as the ratio of increased disease probability associated with a risk factor when disease is relatively common.

The high prevalence of dental caries observed in this study is consistent with the national estimate (40 per cent) for five year olds.[1] Even among children with none of the identified risk factors, caries prevalence was about 20 per cent (Table 5). By comparison the prevalence of reported wheeze – the most common symptom for asthma – is 20 per cent among children aged six to seven years in Melbourne.[31] Moreover, while a 26 per cent decrease in wheeze prevalence among children aged six to seven years occurred in the 10 years from 1993,[31] in children aged five years, dental caries prevalence increased 22 per cent nationally in the years 1996 to 1999.[1] Asthma in Australia is highly prevalent by international standards and rates of hospitalization and emergency department attendance are higher for preschool children than they are for adults.[32] In 1999, asthma, but not dental caries, was declared as one of Australia's seven National Health Priority Areas.

In their 2000 Standards of Care policy document, the Australasian Academy of Paediatric Dentistry[33] recommended that the first dental visit that includes a thorough oral examination should follow within six months of the eruption of the first primary tooth and no later than 12 months.

On the one hand, very few preschool children are accessing free and accessible school dental care while on the other hand medical general practitioners, who are visited almost universally in children's preschool years are not effective in preventing and managing dental caries.[34] These limitations in dental services provision for preschool children highlight the need for primary prevention efforts to prevent caries among preschool children. Based on these findings, these efforts need to be targeted at behaviours that begin as early as the first six months of life, and they need to address non-behavioural risk factors which contributed to disproportionately high caries prevalence among children in low-income households. In particular, public health interventions need to target families with low household income. Although recognized sweetened beverage consumption in early life and the delayed onset of tooth cleaning are associated with dental caries in the primary dentitions of children from all income groups, the adverse impact of these behaviours on oral health is greater among children from low-income households.

## ACKNOWLEDGEMENT

The financial support of the Channel 7 Children's Research Foundation of South Australia Inc is gratefully acknowledged.

## REFERENCES


1. Armfield JM, Roberts-Thomson KF, Spencer AJ. The Child Dental Health Survey, Australia 1999: Trends across the 1990s. AIHW Dental Statistics and Research Series No. 27. AIHW Cat. No. DEN 95. Adelaide: The University of Adelaide, 2003.
2. Thomson WM, Poulton R, Milne BJ, Caspi A, Broughton JR, Ayers KM. Socioeconomic inequalities in oral health in childhood and adulthood in a birth cohort. Community Dent Oral Epidemiol 2004;32:345-353.
3. Harris R, Nicoll AD, Adair PM, Pine CM. Risk factors for dental caries in young children: a systematic review of the literature. Community Dent Health 2004;21(1 Suppl):71-85.
4. Spencer AJ, Wright FA, Brown LM, Brown LP. Changing caries experience and risk factors in five- and six-year-old Melbourne children. Aust Dent J 1989;34:160-165.
5. Stacey MA, Wright FA. Diet and feeding patterns in high risk pre-school children. Aust Dent J 1991;36:421-427.
6. Hallett KB, O'Rourke PK. Dental caries experience of preschool children from the North Brisbane region. Aust Dent J 2002;47:331-338.
7. Hallett KB, O'Rourke PK. Early childhood caries and infant feeding practice. Community Dent Health 2002;19:237-242.
8. Hallett KB, O'Rourke PK. Social and behavioural determinants of early childhood caries. Aust Dent J 2003;48:27-33.
9. Seow WK, Amaratunge A, Bennett R, Bronsch D, Lai PY. Dental health of aboriginal pre-school children in Brisbane, Australia. Community Dent Oral Epidemiol 1996;24:187-190.
10. Armfield JM, Spencer AJ. Consumption of nonpublic water: implications for children's caries experience. Community Dent Oral Epidemiol 2004;32:283-296.
11. Last JM. Dictionary of Epidemiology. 4th edn. Oxford: Oxford University Press, 2001.

12. Kaste LM, Drury TF, Horowitz AM, Beltran E. An evaluation of NHANES III estimates of early childhood caries. J Public Health Dent 1999;59:198-200.
13. Drury TF, Horowitz AM, Ismail AI, Maertens MP, Rozier RG, Selwitz RH. Diagnosing and reporting early childhood caries for research purposes. A report of a workshop sponsored by the National Institute of Dental and Craniofacial Research, the Health Resources and Services Administration, and the Health Care Financing Administration. J Public Health Dent 1999;59:192-197.
14. Australian Bureau of Statistics. Population by Age and Sex, Australian States and Territories 2004. Cat. No. 3201.0.
15. Kleinbaum D, Kupper L, Morgenstern H. Epidemiologic Research: Principles and Quantitative Methods. New York: Van Nostrand Reinhold, 1982.
16. Breslow NE, Day NE. Statistical methods in cancer research. Volume 1: The analysis of case-control studies. Lyon: International Agency for Research on Cancer, 1980:185-187.
17. Slade GD, Gansky SA, Spencer AJ. Two-year incidence of tooth loss among South Australians aged 60+ years. Community Dent Oral Epidemiol 1997;25:429-437.
18. Caplan DJ, Slade GD, Gansky SA. Complex sampling: implications for data analysis. J Public Health Dent 1999;59:52-59.
19. Research Triangle Institute. SUDAAN Language Manual, Release 9.0. Research Triangle Park, North Carolina: Research Triangle Institute, 2004.
20. Gibson S, Williams S. Dental caries in pre-school children: associations with social class, toothbrushing habit and consumption of sugars and sugar-containing foods. Further analysis of data from the National Diet and Nutrition Survey of children aged 1.5-4.5 years. Caries Res 1999;33:101-113.
21. Marinho VC, Higgins JP, Sheiham A, Logan S. Fluoride toothpastes for preventing dental caries in children and adolescents (2003). The Cochrane Database of Systematic Reviews: CD002278.
22. Cahen PM, Frank RM, Turlot JC, Jung MT. Comparative unsupervised clinical trial on caries inhibition effect of monofluorophosphate and amine fluoride dentifrices after 3 years in Strasbourg, France. Community Dent Oral Epidemiol 1982;10:238-241.
23. Winter GB, Holt RD, Williams BF. Clinical trial of a low-fluoride toothpaste for young children. Int Dent J 1989;39:227-235.
24. Davenport ES, Litenas C, Barbayiannis P, Williams CE. The effects of diet, breast-feeding and weaning on caries risk for pre-term and low birth weight children. Int J Paediatr Dent 2004;14:251-259.
25. Parker JD, Schoendorf KC, Kiely JL. Associations between measures of socioeconomic status and low birth weight, small for gestational age, and premature delivery in the United States. Ann Epidemiol 1994;4:271-278.
26. Burt BA, Pai S. Does low birthweight increase the risk of caries? A systematic review. J Dent Educ 2001;65:1024-1027.
27. Peressini S. Pacifier use and early childhood caries: an evidence-based study of the literature. J Can Dent Assoc 2003;69:16-19.
28. Chase I, Berkowitz RJ, Mundorff-Shrestha SA, Proskin HM, Weinstein P, Billings R. Clinical outcomes for Early Childhood Caries (ECC): the influence of salivary mutans streptococci levels. Eur J Paediatr Dent 2004;5:143-146.
29. Wan AK, Seow WK, Purdie DM, Bird PS, Walsh LJ, Tudehope DI. The effects of chlorhexidine gel on Streptococcus mutans infection in 10-month-old infants: a longitudinal, placebo-controlled, double-blind trial. Pediatr Dent 2003;25:215-222.
30. Seow WK, Cheng E, Wan V. Effects of oral health education and tooth-brushing on mutans streptococci infection in young children. Pediatr Dent 2003;25:223-228.
31. Robertson CF, Roberts MF, Kappers JH. Asthma prevalence in Melbourne schoolchildren: have we reached the peak? Med J Aust 2004;180:273-276.
32. Australian Centre for Asthma Monitoring 2003. Asthma in Australia 2003. AIHW Asthma Series 1. AIHW Cat. No. ACM 1. Canberra: Australian Institute of Health and Welfare, 2003.
33. Australasian Academy of Paediatric Dentistry. Standards of Care. Published by The Australasian Academy of Paediatric Dentistry Inc. February 2002.
34. Bader JD, Rozier RG, Lohr KN, Frame PS. Physicians' roles in preventing dental caries in preschool children: a summary of the evidence for the US Preventive Services Task Force. Am J Prev Med 2004;26:315-325.

*Address for correspondence:*
Professor Gary D Slade
Australian Research Centre for Population Oral Health
Dental School
The University of Adelaide
Adelaide, South Australia 5005
Email: gary.slade@adelaide.edu.au