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Family history and oral health: findings from the Dunedin Study

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Abstract

Context—The effects of the oral health status of one generation on that of the next within families are unclear.

Objective—To determine whether parental oral health history is a risk factor for oral disease.

Methods—Oral examination and interview data were collected during the age-32 assessments in the Dunedin Study. Parental data were also collected on this occasion. The sample was divided into two familial-risk groups for caries/tooth loss (high risk and low risk) based on parents' self-reported history of tooth loss at the age-32 assessment interview.

Main outcome measures—Probands' dental caries and tooth loss status at age 32, together with lifelong dental caries trajectory (age 5–32).

Results—Caries/tooth-loss risk analysis was conducted for 640 proband-parents groups. Referent groups were the low-familial-risk groups. After controlling for confounding factors (sex, episodic use of dental services, socio-economic status and plaque trajectory), the prevalence ratio (PR) for having lost 1+ teeth by age 32 for the high-familial-risk group was 1.41 (95% confidence interval [CI] 1.05, 1.88) and the rate ratio for DMFS at age 32 was 1.41 (95% CI 1.24, 1.60). In the high-familial-risk group, the PR of following a high caries trajectory was 2.05 (95% CI 1.37, 3.06). Associations were strongest when information was available about both parents' oral health. Nonetheless, when information was available for one parent only, associations were significant for some proband outcomes.

Conclusions—People with poor oral health tend to have parents with poor oral health. Family/parental history of oral health is a valid representation of the intricacies of the shared genetic and environmental factors that contribute to an individual's oral health status. Associations were strongest when data from both parents can be obtained.

Keywords

oral health; family history; intergenerational; risk

Introduction

The life-course approach to chronic disease epidemiology considers the long-term biological, behavioural, psychological, social and environmental influences that link adult health and adult disease risk to exposures acting during the individual's life span (1). It is also concerned with how these influences can contribute to health across generations (1). Research suggests that the health status of one generation can have a profound effect on that of the next; studies have found intergenerational and familial associations for cardiovascular disease (2–7), non-insulin-dependent diabetes mellitus (8–12), metabolic syndrome (13, 14), cancer (15–18), asthma (19), obesity (20, 21), and health-related behaviours, including smoking, drug and alcohol use (22–25), and diet and exercise (26, 27). However, little research has been carried out on intergenerational associations for oral health.

Is family history a risk factor for oral disease? Intriguingly, twin studies suggest a strong genetic contribution to dental caries experience (28–30). An early attempt was made in 1974 to investigate caries experience across three generations; unfortunately, the study suffered from high rates of attrition and insufficient power (31). Since then, little research has investigated the mechanisms that link oral health (and oral disease risk) to exposures across generations. What has been done has almost exclusively concentrated on oral health outcomes in children or young adolescents (32–41). Most researchers have focussed on narrow aspects of intergenerational continuity, such as oral cancer (42), or *Streptococcus mutans* acquisition (36). Some studies are cross-sectional in design (and so do not offer strong evidence), and many have tended to examine the effects of exposures at only one point in time. Others have suffered from methodological problems (such as selection bias, attrition, or insufficient statistical power). Many studies have not controlled for confounding factors. In summary, it is unclear whether the oral health status of one generation has an effect on that of the next.

Oral health, in common with virtually all traits, is likely to be the result of gene-environment interaction (43–45). Family history reflects the results of shared genetic variations, and shared non-genetic factors (environmental factors, exposures and common behaviours) (46). While research has increased awareness of the numerous and diverse risk factors contributing to oral disease, an understanding of these shared genetic and environmental influences acting through generations on the oral health of adults is a clear gap in the knowledge base. Using intergenerational data from an adult sample with sufficient statistical power, and controlling for putative confounders, the aim of this study was to determine whether an individual's oral health and disease risk is influenced by that of his or her parents.

Methods

Study design and participants

This study is an analysis of data from the Dunedin Multidisciplinary Health and Development Study (DMHDS) using dental data collected from Study members (hereafter referred to as “probands”) and their parents during the age-32 assessments. The DMHDS is a longitudinal epidemiological study of a birth cohort of 1,037 children born at the Queen Mary Hospital, Dunedin, New Zealand between 1 April 1972 and 31 March 1973. These 1037 children represent 91% of the 1139 eligible children born between these dates, and 972 (95.8% of the surviving cohort) were assessed at age 32. Of these 972, 932 (95.9%) were dentally examined (Figure 1). Of those who were dentally examined, the majority (882, 94.6%) had one or both parents participate in the family health history study; two-thirds (648, 69.5%) had both parents participate. Data from 16 probands were excluded from the analysis due to incomplete parental information. Analyses were carried out for two samples.

The first sample (N = 866) comprised probands who had one or both parents interviewed at the age-32 assessment (generalisable to households where data from either one parent only, or both parents were available); the second (N = 640) consisted of probands who had both parents interviewed at the age-32 assessment (a more complete parental history is obtained, but is only generalisable to two-parent families). As the “one or both parents interviewed” sample encompasses the “both parents interviewed” sample, the samples are not mutually exclusive.

Māori (7.5%) were under-represented (in comparison to 15% in the total New Zealand population) in the cohort at age 32. There were statistically significant differences between those included and those excluded in the analyses; those excluded had a greater prevalence of one or more missing teeth ($p < 0.05$), higher mean DMFS ($p < 0.05$), and higher mean MS ($p < 0.05$) at age 32 than those included (unpublished data, available on request). Those excluded did not significantly differ from those included with respect to sex, SES, self-rated oral health, use of dental services, smoking status, caries prevalence, severe caries prevalence, mean DS, and mean FS at age 32. Ethics approval for the study was granted by the Otago Research Ethics Committee, and participants gave informed consent.

Measurements

The study used data collected from probands’ oral examinations and interviews, and from interviews with their parents, at the age-32 assessments.

Proband caries and tooth loss measurement—In the age-32 assessments (conducted in 2004–2005), calibrated examiners carried out dental examinations for caries and missing teeth on 932 (95.6%) of the 972 probands who were assessed at age 32. Accumulated tooth loss due to caries was determined by recording the presence or absence of each tooth, and noting the reason for its absence. Third molars were not included in the estimation of caries-associated tooth loss. Teeth were examined for caries and restorations; four surfaces were coded for the anterior teeth (buccal, lingual, mesial and distal), and five (including the occlusal surface) for the posterior teeth.

As reported previously, longitudinal caries experience data were used to identify three distinct trajectories of dental caries experience between ages 5–32 (47). Briefly, a modified DMFS was computed for participants who had three or more dental examinations (and dental caries data) between ages 5 and 32. Group-based trajectory analysis, based on the zero-inflated Poisson distribution, was performed with the PROC TRAJ macro in SAS 9.1 to identify low, moderate and high caries trajectory groups. Regarding the family history samples, 849 individuals had a caries trajectory in the “one or both parents in” sample (low, N = 349, 41.1%; moderate, N = 374, 44.1%; and high, N = 126, 14.8%), and 626 individuals had a caries trajectory in the “both parents in” sample (low, N = 256, 40.9%; moderate, N = 283, 45.2%; and high, N = 87, 13.9%).

Plaque measurement—The simplified oral hygiene index was used to quantify plaque accumulation on six index teeth (48), and the overall plaque score was the sum of the scores divided by the number of teeth scored. Long-term plaque exposure was described through trajectory analysis. The longitudinal data on plaque scores measured at three or more assessments between the ages 5–32 (including at age 32) were used to split the cohort into three distinct ‘plaque groups’ using a group-based trajectory analysis model, based on the censored normal distribution, in SAS 9.2 (49). In the family history samples, 848 individuals had a plaque trajectory in the “one or both parents in” sample (Group 1, low levels of plaque, N = 333, 39.3%; Group 2, moderate levels of plaque, N = 419, 49.4%; and Group 3, high levels of plaque, N = 96, 11.3 %), and 625 individuals had a plaque trajectory in the

“both parents in” sample (Group 1, low levels of plaque, N = 257, 41.1%; Group 2, moderate levels of plaque, N = 306, 49.0%; and Group 3, high levels of plaque, N = 62, 9.9%).

Proband interviews—A measure of socioeconomic status (SES) at phase 32 was obtained from each study member using standard New Zealand indices which apply a six-interval classification according to occupation; for example, a doctor scores “1” and a labourer scores “6” (50, 51). Study members with a score of “1” or “2” were allocated to the “high SES group; those with a score of “3” or “4” were assigned to the “medium SES group; and those with a score of “5” or “6” were assigned to the “low SES group. Participants were asked to indicate whether they were routine or episodic users of dental care services. Routine users were those who usually visited for a check-up, and had made a dental visit in the previous 12 months (52).

Parental interviews—Around the same time as the age-32 assessment (2003 to 2006), the parents of probands took part in an interview on their oral health status and history (53). They were asked whether they had lost any teeth (for any reason) and if so, how many? In addition, they were asked about the main reason for their tooth loss (tooth decay, periodontal disease, trauma or another reason).

The parental interview information on their dental caries and tooth-loss experience was used to allocate each proband (their child) to either a “high-familial-risk” group or a “low-familial-risk” group for dental caries (Figure 1). Probands were allocated to the high-familial-risk category if one or both of their parents reported having lost six or more teeth, due to caries, at the age-32 assessment. All other probands (those whose parents did not report having lost six or more teeth, due to caries, at the age-32 assessment) were grouped in the low-familial-risk category. These groups were further refined for some analyses according to whether the probands had been assigned to a caries trajectory, a plaque trajectory, or both (Figure 1)

Statistical analysis

Descriptive and bivariate analyses were conducted using SPSS version 16.0 (SPSS Inc. Chicago, Illinois). Multivariable analyses used Stata version 10.0 (StataCorp, College Station, Texas 77845, USA). Chi-square tests were used to examine the statistical significance of associations observed between familial-risk groups and proband caries and tooth loss prevalence at age 32. Independent sample t-tests were used for continuous dependent variables mean DMFS, mean DS, mean MS, mean FS, and mean plaque score at age 32. Statistical tests were two-tailed and the threshold for statistical significance was set at $p < 0.05$. In the multivariable analysis, the generalized linear model (GLM) command with modified Poisson regression analysis was used to estimate prevalence ratio (with confidence intervals) for proband prevalence of one or more missing teeth due to caries, and of having a high caries trajectory, and a rate ratio for mean DMFS, at age 32, using a robust error variance procedure. Confounding factors (sex, episodic use of dental services, SES and plaque trajectory) were selected on the basis of biological plausibility, and were guided by the literature (49, 54–56). In the causal directed acyclic graph (DAG), an arrow connecting two variables indicates causation (Figure 2). This graphical representation of causal effects between variables facilitates the identification of confounders that should be adjusted for in order to minimise bias (57, 58).

Results

Dental caries and tooth loss by familial-risk category

In bivariate analyses for both samples, there were marked caries/tooth loss risk category differences between the familial-risk groups in the prevalence of: severe caries experience (DMFS > 20); one or more teeth missing due to caries; having a DMFS score greater than the median (median DMFS = 12) at age 32; and having a high caries trajectory between ages 5–32 (Tables 1 and 2, and Figure 3). This was also the case for mean DMFS, mean DS, mean FS, mean MS, and mean plaque score (Tables 1 and 2, and Figure 4).

Multivariable analyses

Multivariable modelling was used to determine the prevalence ratio (PR) for one or more missing teeth at age 32, the rate ratio (RR) for DMFS at age 32, and the PR of having a high caries trajectory between ages 5–32 in the high-familial-risk group (using the low-familial-risk group as a referent) while controlling for the confounding factors of sex, episodic use of dental services, SES and plaque trajectory. For the “one or both parents interviewed” sample, the age-32 mean DMFS in the high-familial-risk group was 1.30 (95% confidence interval [CI] 1.16, 1.47) times greater than that for the low-familial-risk group; and the PR of having a high caries trajectory between ages 5–32 in the high-familial-risk group was 1.66 (95% CI 1.20, 2.30) times greater than that for the low-familial-risk group (Table 3). For the “both parents interviewed” sample, the PR of having lost at least one tooth by age 32 for those in the high-familial-risk group was 1.41 (95% CI 1.05, 1.88) times greater than that for the low-familial-risk group; the age-32 mean DMFS in the high-familial-risk group was 1.41 (95% CI 1.24, 1.60) times greater than that for the low-familial-risk group; and the PR of having a high caries trajectory between ages 5–32 in the high-familial-risk group was 2.05 (95% CI 1.37, 3.06) times greater than that for the low-familial-risk group (Table 4).

Discussion

These data from a prospective cohort study provide clear support for the continuity of oral health across generations within families. Study members (probands) were grouped according to their parents’ self-reported oral health status, recorded by interview, when probands were aged 32. It was found, after controlling for confounding factors, that those in the high-familial-risk group for tooth loss had significantly greater risk (in comparison with the low-familial-risk group) of having higher cumulative dental caries experience, and of having a high caries trajectory, by age 32. If both parents were interviewed, those in the high-familial-risk group for tooth loss were also found to have a significantly greater risk (in comparison with the low-familial-risk group) of having lost one or more teeth due to caries.

This study had some limitations. No detailed record was made of participants’ dietary habits, nor of their overall fluoride exposure, so these could not be adequately controlled for. The study relied on parental self-report data to categorise the proband into familial-risk groups, and on proband self-report data on SES and use of dental services. The issue of the reliability and validity of self-report data has been addressed by other authors (59, 60). Interview/examiner-based assessments, as used in the family health history study, are more likely to yield valid data than “self-completed” data. In addition, Dunedin Study participants and their parents are familiar with interviews, are aware of the importance of accurate responses, and there is a long history of mutual trust and respect between participants and researchers. However, the possibility of error due to parents being unaware of their oral health status at the age-32 assessment of probands must be considered. This error would most likely have been in the direction of undiagnosed disease leading to misclassification, and may have increased the likelihood of a null finding. In the case of the “one or both

parents interviewed” sample, the possibility of error due to lack of data on the oral health status of a non-attending co-parent cannot be overlooked.

The use of a birth cohort, and the high retention rate, means that the sample is representative of its source population (the South Island of New Zealand). The issue of whether the findings can be generalised to the New Zealand population, and to other populations (particularly the United States), has been addressed by another paper using data from this sample (61). It was cautiously concluded that findings from the DMHDS can be generalised to these populations. In support of the wider generalisability of the sample, oral health data from similar studies may be considered. While the intergenerational findings of the DMHDS are unique, the principal cross-sectional and descriptive findings at phase 32 are reasonably consistent with the limited data on this age group available from overseas studies (62–66). This is true also of the findings for the parents at the age-32 assessment (65, 67). This increases confidence in the validity of the intergenerational findings. In addition, validity is further enhanced by previous research demonstrating associations between family history and oral health outcomes in children or young adolescents (28–41), and intergenerational and familial associations found for other chronic disease outcomes in adults (2–27). We suggest that the intergenerational findings of the DMHDS are consistent with existing biological and medical knowledge.

While this sample under-represents Maori with respect to the total New Zealand Maori population, it is generally representative of the South Island. According to the 2006 Census, 7.6% of adults aged 25–34 self-identified as Maori in Otago (Dunedin is the capital of the Otago region); this is in accord with the DMHDS sample. Furthermore, as Maori, on average, suffer poorer oral health than the general population (68), the under-representativeness of Maori in the DMHDS may have led to an under-estimation of the strength of the observed associations. Similarly, the differences between those included in and those excluded from the analyses (prevalence of one or more missing teeth, mean dmfs, and mean ds at age 32) may have increased the likelihood of a null finding.

Turning to the study findings, we believe that these are unprecedented. Until now, it has not been possible to examine the nature and extent of intergenerational continuity in oral health because such data have not been available. The Dunedin Study is unique in its longevity, sample size, retention rate, oral health data (including intergenerational data), and information on a range of potential risk, ameliorating, exacerbating and confounding factors. It offers a particularly valuable opportunity to investigate intergenerational associations in oral health, and to broaden our understanding of the causal associations between parental oral health and the oral health of their offspring.

The grouping of individuals into risk categories was informed by cross-sectional and longitudinal studies which indicate associations between parental and child oral health (32–35, 37–40). The age-32 assessment bivariate data provided support for the groupings, as did DMHDS bivariate associations in previous years. For example, at the age-5 assessment, statistically significant associations were found between maternal self-reported oral health and the child’s caries severity at age 5, and between maternal denture status and the child’s caries severity at age 5 (unpublished data, available on request). The parental cut-off point of six or more teeth lost due to caries for membership of the high-risk familial group ensured an adequate sample size for this group, and so enhanced statistical power.

For caries and tooth loss, a largely consistent pattern of higher prevalence and greater severity of disease was clearly seen across the familial-risk groups. The prevalence of “any caries experience” (1+DMFS) at age 32 was so high that only a minimal difference between familial-risk groups can be seen; however, the differences for severe caries (DMFS >20),

having one or more missing teeth, having a DMFS greater than the median, and caries and tooth loss severity were striking. This was the case for both the “one or both parents interviewed” and “both parents interviewed” samples; there was minimal difference between the two samples in the strength of the associations.

It is currently unclear how disease risk is transmitted from one generation to the next. Genetically-determined traits may present in similar ways across generations. Environmental risk factors (such as SES, smoking and episodic use of dental health services) may persist across generations. A genetic predisposition coupled with exposure to environmental risk factors forms the basis for gene-environment interplay; that is, the situation where both genetic and environmental factors interact to influence health in individuals and populations (43–45).

Our findings provide clear evidence—in the case of dental caries and tooth loss—in support of a causal association between parental oral health and proband oral health. The combination of genetic and environmental risk factors over the life course is a biologically plausible mechanism whereby parental oral health can influence the oral health of offspring.

Conclusions

This study provides clear evidence that the children of parents with poor oral health are more likely to have poor oral health in adulthood than the children of parents with good oral health. Family/parental history of oral health appears to be a valid representation of the complex interplay between shared genetic factors and shared environmental factors, exposures and behaviours that contribute to an individual’s oral health status. Future research is required to examine associations between family/parental history and other oral health outcomes, such as periodontal disease and oral health-related quality of life.

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Fig. 1.
Participation rates, sample sizes, and familial-risk groups

Fig. 2.
DAG for exposure (familial-risk), outcome (proband caries/tooth loss status at age 32) and confounding factors

Fig. 3. Proband caries and tooth loss prevalence by caries and tooth loss familial-risk category at phase 32 (error bars represent the 95% confidence interval)

Fig. 4. Proband caries and tooth loss severity by caries and tooth loss familial-risk category at phase 32 (error bars represent the 95% confidence interval)

Proband caries and tooth loss prevalence and severity, and plaque score, at age 32 by familial-risk category for caries and tooth loss (one or both parents sample).

Table 1

	Risk category for caries and tooth loss according to parental tooth loss history		
	High risk N = 313 (36.1%)	Low risk N = 553 (63.9%)	Total N = 866 (100%)
Proband characteristics (%)			
Male	169 (54.0)	270 (48.8)	439 (50.7)
Low socioeconomic status	106 (33.9)	154 (27.8) ^a	260 (30.0)
Medium socioeconomic status	164 (52.4)	291 (52.6)	455 (52.5)
High socioeconomic status	43 (13.7)	108 (19.5) ^a	151 (17.4)
Episodic user of dental services	172 (56.4)	285 (52.1)	457 (53.6)
Proband caries and tooth loss prevalence (%)			
Any caries	306 (97.8)	529 (95.7)	835 (96.4)
Severe caries (DMFS > 20)	118 (37.7)	138 (25.0) ^b	256 (29.6)
One or more teeth missing (due to caries)	86 (27.5)	109 (19.7) ^a	195 (22.5)
DMFS > median	187 (59.7)	229 (41.4) ^b	416 (48.0)
Moderate caries trajectory age 5–32 *	148 (48.4)	226 (41.6)	374 (43.2)
High caries trajectory age 5–32 *	63 (20.6)	63 (11.6) ^c	126 (14.5)
Proband caries and tooth loss severity (SD)			
Mean DMFS	19.7 (16.0)	14.5 (14.0) ^d	16.4 (14.9)
Mean DS	3.1 (5.3)	1.8 (4.1) ^d	2.3 (4.6)
Mean FS	13.0 (10.4)	10.5 (10.0) ^d	11.4 (10.2)
Mean MS(due to caries)	3.6 (8.8)	2.2 (6.3) ^e	2.7 (7.3)
Plaque measure (SD)			
Mean plaque score	0.8 (0.6)	0.7 (0.5) ^e	0.8 (0.5)

^a p<0.05; chi-square test.

^b p<0.001; chi-square test.

^c p<0.005; chi-square test.

^d $p < 0.001$; independent samples t-test.

^e $p < 0.05$; independent samples t-test.

* Total N = 849; High risk N = 306; Low risk N = 543.

Proband caries and tooth loss prevalence and severity, and plaque score, at age 32 by familial-risk category for caries and tooth loss (both parents sample).

Table 2

	Risk category for caries and tooth loss according to parental tooth loss history		
	High risk N = 254 (39.7%)	Low risk N = 386 (60.3%)	Total N = 640 (100%)
Proband characteristics (%)			
Male	131 (51.6)	181 (46.9)	312 (48.8)
Low socioeconomic status	82 (32.3)	98 (25.4) ^a	180 (28.1)
Medium socioeconomic status	138 (54.3)	206 (53.4)	344 (53.8)
High socioeconomic status	34 (13.4)	82 (21.2) ^a	116 (18.1)
Episodic user of dental services	137 (55.0)	193 (50.3)	330 (52.1)
Proband caries and tooth loss prevalence (%)			
Any caries	249 (98.0)	365 (94.6) ^a	614 (95.9)
Severe caries (DMFS > 20)	97 (38.2)	88 (22.8) ^b	185 (28.9)
One or more teeth missing (due to caries)	71 (28.0)	66 (17.1) ^c	137 (21.4)
DMFS > median	151 (59.4)	152 (39.4) ^b	303 (47.3)
Moderate caries trajectory age 5–32 [*]	121 (48.8)	162 (42.9)	282 (44.2)
High caries trajectory age 5–32 [*]	51 (20.6)	36 (9.5) ^b	87 (13.6)
Proband caries and tooth loss severity (SD)			
Mean DMFS	19.7 (16.0)	13.4 (12.1) ^d	15.9 (14.1)
Mean DS	3.1 (5.5)	1.3 (2.8) ^d	2.0 (4.2)
Mean FS	13.0 (10.3)	10.4 (9.9) ^e	11.5 (10.2)
Mean MS(due to caries)	3.6 (9.1)	1.7 (4.5) ^e	2.4 (6.8)
Plaque measure (SD)			
Mean plaque score	0.8 (0.5)	0.7 (0.5) ^f	0.7 (0.5)

^a p<0.05; chi-square test.

^b p<0.001; chi-square test.

^c p<0.005; chi-square test.

^d p<0.001; independent samples t-test.
^e p<0.005; independent samples t-test
^f p<0.05; independent samples t-test.
^{*} Total N = 626; High risk N = 248; Low risk N = 378.

Modified Poisson regression model* for proband prevalence of one or more missing teeth at age 32, for proband caries severity (decayed, missing and filled surfaces) at age 32, and for moderate and high caries trajectories between ages 5–32 (one or both parents sample).

Table 3

	Prevalence 1+ missing teeth			DMFS		High caries Trajectory	
	PR	95% CI	RR	95% CI	PR	95% CI	
Male	1.03	0.79, 1.32	0.90	0.79, 1.02	0.89	0.63, 1.25	
Episodic user of dental services at age 32	2.37	1.72, 3.25	1.23	1.09, 1.40	1.31	0.92, 1.86	
Low SES at age 32	2.24	1.36, 3.67	1.22	1.01, 1.47	1.20	0.72, 1.99	
Medium SES at age 32	1.54	0.94, 2.52	1.05	0.89, 1.24	0.98	0.60, 1.61	
Moderate plaque trajectory at age 32	2.29	1.60, 3.29	1.28	1.13, 1.46	1.71	1.15, 2.55	
High plaque trajectory at age 32	2.84	1.85, 4.36	1.59	1.29, 1.95	1.89	1.09, 3.30	
High familial-risk group for tooth loss	1.22	0.96, 1.56	1.30	1.16, 1.47	1.66	1.20, 2.30	

* Adjusted for sex, use of dental services, SES and plaque trajectory at phase 32.

Reference categories: male (female, coded 0), episodic user of dental services at phase 32 (routine user of dental services at phase 32, coded 0), low or medium SES at phase 32 (high SES coded 0), moderate or high plaque trajectory (low plaque trajectory coded 0), high familial-risk for tooth loss (low familial-risk for tooth loss coded 0)

PR, prevalence ratio; RR, rate ratio; CI, confidence interval; SES, socioeconomic status.

N = 848

Table 4

Modified Poisson regression model* for proband prevalence of one or more missing teeth at age 32, for proband caries severity (decayed, missing and filled surfaces) at age 32, and for moderate and high caries trajectories between ages 5–32 (both parents sample).

	Prevalence 1+ missing teeth		DMFS		High caries Trajectory	
	PR	95% CI	RR	95% CI	PR	95% CI
Male	1.11	0.82, 1.51	0.88	0.77, 1.01	0.87	0.58, 1.31
Episodic user of dental services at age 32	2.47	1.69, 3.61	1.31	1.15, 1.50	1.47	0.96, 2.23
Low SES at age 32	2.32	1.29, 4.15	1.15	0.95, 1.40	1.05	0.57, 1.94
Medium SES at age 32	1.57	0.89, 2.77	1.05	0.88, 1.26	1.03	0.57, 1.83
Moderate plaque trajectory at age 32	2.04	1.35, 3.09	1.21	1.04, 1.40	1.64	1.02, 2.64
High plaque trajectory at age 32	2.40	1.44, 4.01	1.42	1.04, 1.77	1.70	0.86, 3.34
High familial-risk group for tooth loss	1.41	1.05, 1.88	1.41	1.24, 1.60	2.05	1.37, 3.06

* Adjusted for sex, use of dental services, SES and plaque trajectory at phase 32.

Reference categories: male (female, coded 0), episodic user of dental services at phase 32 (routine user of dental services at phase 32, coded 0), low or medium SES at phase 32 (high SES coded 0), moderate or high plaque trajectory (low plaque trajectory coded 0), high familial-risk for tooth loss (low familial-risk for tooth loss coded 0)

PR, prevalence ratio; RR, rate ratio; CI, confidence interval; SES, socioeconomic status.

N = 625