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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-parent groups. Reference 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 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 are strongest when data from both parents can be obtained.

Key words: family history; intergenerational; oral health; risk

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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 lifespan (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,

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probands were excluded from the analysis because of 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 (generalizable 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 generalizable 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 with 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 caused by 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 and 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 and 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 socio-economic status (SES) at phase 32 was obtained from each study member using standard New Zealand indices that 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).

Table 1. 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)

	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 socio-economic status	106 (33.9)	154 (27.8)[a]	260 (30.0)
Medium socio-economic status	164 (52.4)	291 (52.6)	455 (52.5)
High socio-economic 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 (owing 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 (owing 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 $N = 543$.

high-familial-risk group was 1.30 [95% confidence interval (CI) 1.16, 1.47] times greater than that in the low-familial-risk group, and the PR of having a high caries trajectory between ages 5 and 32 in the high-familial-risk group was 1.66 (95% CI 1.20, 2.30) times greater than that in 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 in 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 in the low-familial-risk group; and the PR of having a high caries trajectory between ages 5 and 32 in the high-familial-risk group was 2.05 (95% CI 1.37, 3.06) times greater than that in 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 (than the low-familial-risk group) of having lost one or more teeth owing 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 categorize 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

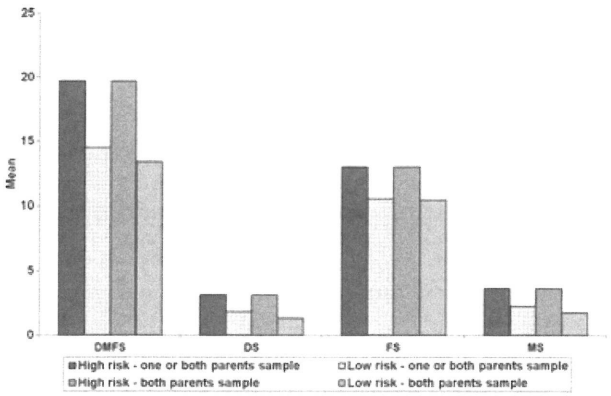

Fig. 4. Proband caries and tooth loss severity by caries and tooth loss familial-risk category at phase 32.

the possibility of error because of 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 because of the lack of data on the oral health status of a nonattending 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

Table 3. 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 and 32 (one or both parents sample)

	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

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, socio-economic status.
$N = 848$.
*Adjusted for sex, use of dental services, SES and plaque trajectory at phase 32.

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 and 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

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, socio-economic status.
$N = 625$.
*Adjusted for sex, use of dental services, SES and plaque trajectory at phase 32.

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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