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Inter-generational continuity in periodontal health: findings from the Dunedin Family History Study

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Abstract

Objective: To determine whether parental periodontal disease history is a risk factor for periodontal disease in adult offspring.

Methods: Proband periodontal examination [combined attachment loss (CAL) at age 32, and incidence of CAL from ages 26 to 32] and interview data were collected during the age-32 assessments in the Dunedin Study. Parental data were also collected. The sample was divided into two familial-risk groups for periodontal disease (high- and low-risk) based on parents' self-reported periodontal disease.

Results: Periodontal risk analysis involved 625 proband-parent(s) groups. After controlling for confounding factors, the high-familial-risk periodontal group was more likely to have 1+ sites with 4+ mm CAL [relative risk (RR) 1.45; 95% confidence interval (CI) 1.11–1.88], 2+ sites with 4+ mm CAL (RR 1.45; 95% CI 1.03–2.05), 1+ sites with 5+ mm CAL (RR 1.60; 95% CI 1.02–2.50), and 1+ sites with 3+ mm incident CAL (RR 1.64; 95% CI 1.01–2.66) than the low-familial-risk group. Predictive validity was enhanced when information was available from both parents.

Conclusions: Parents with poor periodontal health tend to have offspring with poor periodontal health. Family/parental history of oral health is a valid representation of the shared genetic and environmental factors that contribute to an individual's periodontal status, and may help to predict patient prognosis and preventive treatment need.

Key words: family history; inter-generational; periodontal; risk

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Conflict of interest and source of funding statement

No conflict of interest declared.

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The concept of inter-generational continuity in periodontal health is not new. It was observed almost a century ago, and during the 1940s and 1950s, researchers conducted investigations into inter-generational effects, including family studies and twin studies (Gorlin et al. 1967, Hassell & Harris 1995). Clear evidence for a substantial genetic component in periodontal disease susceptibility was demonstrated in animal models (Baer et al. 1961). However, the main focus of research from the 1960s to 1990s shifted from hereditary factors to the role of bacteria and other environmental factors in disease risk (Löe 1993). More recently, the idea that virtually all characteristics are the result of gene–environment interaction has become the paradigm for considering many common, preventable disorders of adulthood (Collins 2004, Hunter 2005, Moffitt et al. 2005). An increasing interest in gene–environment interactions is reflected in greater awareness of the role of family history and inter-generational continuity in health as a practical, inexpensive approach to categorizing gene–environment risk for these disorders, including periodontal disease (Scheuner et al. 1997, Khoury et al. 2005, Valdez et al. 2010).

Research suggests that the health status of one generation can have a profound effect on that of the next. Studies have found inter-generational

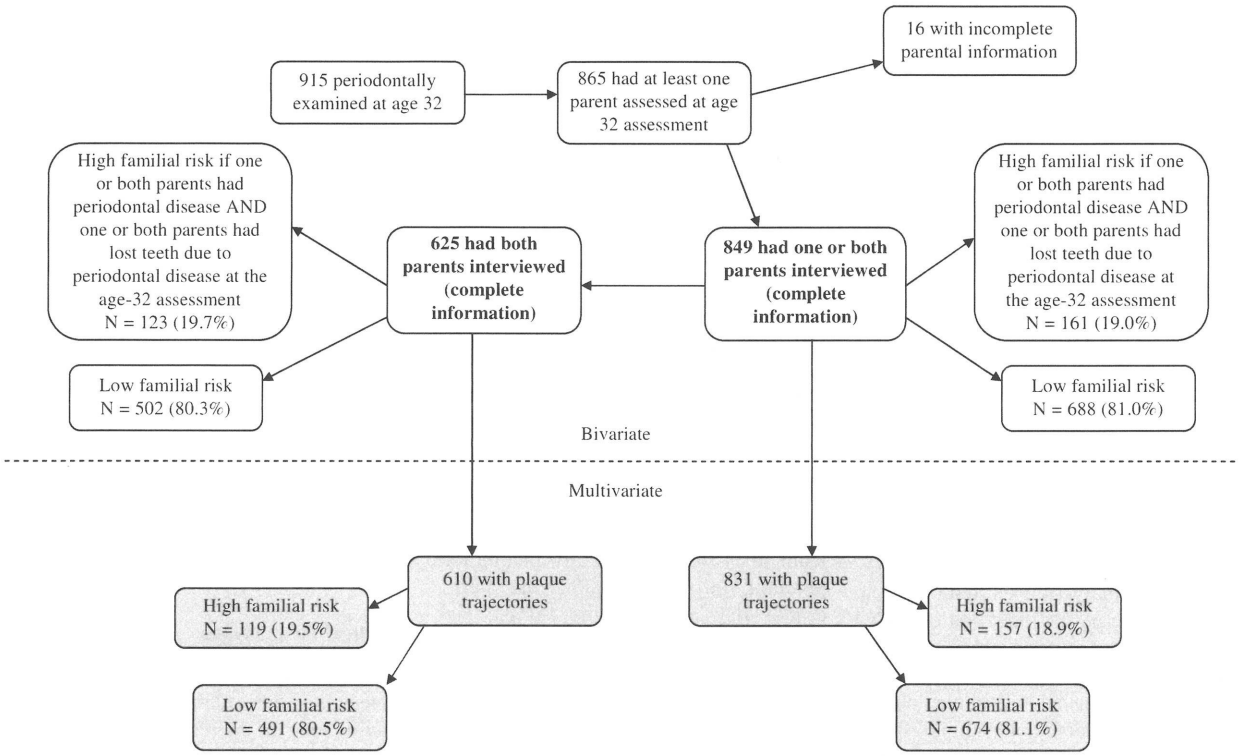

Fig. 1. Periodontal disease familial-risk groups.

plaque exposure was described through trajectory analysis. The longitudinal data on plaque scores measured at ages 5, 9, 15, 18, 26, and 32 years 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. The scores were as follows: group 1, low levels of plaque (group mean = 0.59, $N = 328$, 39.5% of the cohort); group 2, moderate levels of plaque (group mean = 0.93, $N = 408$, 49.1%); and group 3, high levels of plaque (group mean = 1.45, $N = 95$, 11.4%). Overall, plaque trajectory data were available for 953 study members, but analyses were restricted to those 831 Study members who were periodontally examined at age 32, who had at least one parent attend for interview, and for whom plaque data were available at age 32 years (Broadbent et al. 2011).

Proband interviews

Probands were questioned on their smoking history; in addition, tobacco usage data had been collected during previous assessments. Current and ex-smokers were asked about the number of cigarettes smoked per day, and the number of years at this level of consumption. These data were used to compute an individual's exposure as the number of pack-years to age 32.

A measure of SES at age 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'' (Irving & Elley 1977, Elley & Irving 1985). 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 (Thomson et al. 2010).

Parental interviews

Around the same time as the age-32 assessment (2003–2006), the parents of probands took part in an interview on their oral health status and history (Milne et al. 2009a). They were asked whether they had ever been told they had periodontal disease, whether they had lost any teeth (for any reason) and if so, how many. Finally, they were asked about the main reason for their tooth loss (tooth decay, periodontal disease, trauma or another reason). Two of these variables (prevalence of periodontal disease, and prevalence of tooth loss due to periodontal disease) formed the basis of the familial-risk grouping for periodontal disease (Fig. 1). Probands were allocated to the high-familial-risk category if one or both of their parents reported having periodontal disease, and one or both parents had lost teeth due to periodontal disease, at the age-32 assessment. All other probands were grouped in the low-familial-risk category.

Statistical analysis

The parental interview information was used to allocate each proband (their child) to either a ''high-familial-risk'' group or a ''low-familial-risk'' group for periodontal disease (Fig. 1). The utility of those familial-risk groups was

services, SES, plaque trajectory, and tobacco use. For the ''one or both parents interviewed'' sample, the RR for those in the high-familial-risk group did not reach statistical significance for any of these variables (Table 2).

For the ''both parents interviewed'' sample, the RR for having one or more sites with 4+ mm CAL by age 32 for those in the high-familial-risk group was 1.45 times greater than that for the low-familial-risk group (Table 2). The RR for having two or more sites with 4+ mm CAL by age 32 for those in the high-familial-risk group was 1.45 times greater than that for the low-familial-risk group. For those in the high-familial-risk group, the RR for having one or more sites with 5+ mm CAL by age 32 was 1.60 times that of the low-familial-risk group, and the RR for having one or more sites with 3+ mm incident CAL between ages 26 and 32 was 1.64.

Multivariate modelling revealed effect modification between plaque and smoking to substantially increase smokers' relative risk (in either sample) of having one or more sites with 4+ mm CAL, two or more sites with 4+ mm CAL, and one or more sites with 5+ mm CAL, by age 32; and of having one or more sites with 3+ mm incident CAL between ages 26 and 32 (Supporting Information, Tables S1–S8 and Figs S1–S6). Likewise, effect modification was found between familial-risk group and smoking whereby there was no difference between the reference group and the high-familial-risk group in non-smokers (with the exception of the prevalence of 1+ sites with 3+ mm incident CAL between ages 26 and 32, in the ''both parents in'' sample), but both high- and low-familial-risk groups experienced greater risk for all outcomes in smokers. In general, effect modification between familial-risk group and plaque trajectory was not found.

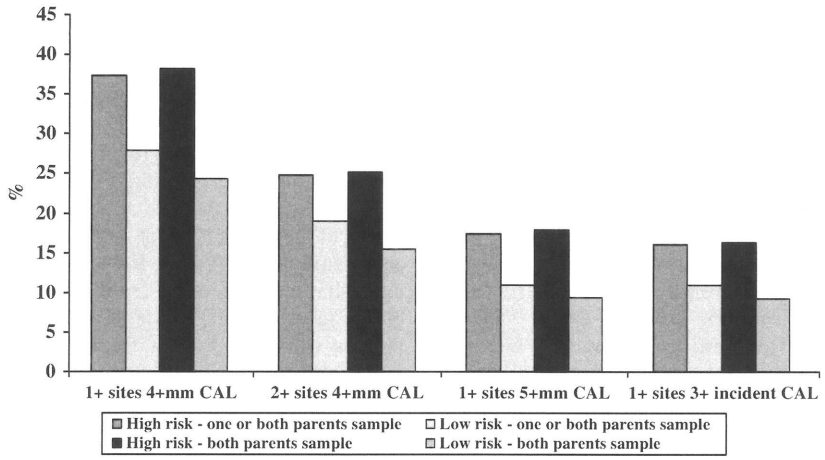

Fig. 2. Proband periodontal disease prevalence by periodontal familial-risk category at age 32.

Discussion

These data from a prospective cohort study suggest a degree of continuity of periodontal health across generations within families. Study members (probands) were grouped according to their parents' self-reported periodontal health status, recorded by interview, when pro-

Table 2. Outcomes of multivariate modelling, and smoking-plaque effect modification, for proband prevalence of one or more sites with 4+ mm combined attachment loss (CAL), prevalence of two or more sites with 4+ mm CAL, and prevalence of one or more sites with 5+ mm CAL at age 32, and prevalence of one or more sites with 3+ mm incident CAL between ages 26 and 32

	High-familial-risk group for periodontal disease			
	unadjusted	adjusted model 1*	adjusted model 2†	adjusted model 3‡
Periodontal disease prevalence at 32				
One or both parents sample				
RR 1+ sites with 4+ mm CAL (95% CI)	**1.33 (1.05,1.69)**	**1.32 (1.05,1.67)**	1.24 (0.98,1.55)	1.23 (0.98,1.54)
RR 2+ sites with 4+ mm CAL (95% CI)	1.32 (0.96,1.80)	1.31 (0.96,1.78)	1.20 (0.89,1.63)	1.18 (0.88,1.57)
RR 1+ sites with 5+ mm CAL (95% CI)	**1.59 (1.06,2.38)**	**1.57 (1.05,2.33)**	1.40 (0.94,2.09)	1.36 (0.92,1.99)
RR 1+ sites with 3+ mm incident CAL (95% CI)	1.47 (0.97,2.21)	1.45 (0.97,2.18)	1.35 (0.90,2.04)	1.34 (0.90,2.01)
Both parents sample				
RR 1+ sites with 4+ mm CAL (95% CI)	**1.57 (1.19,2.08)**	**1.55 (1.19,2.03)**	**1.46 (1.12,1.89)**	**1.45 (1.11,1.88)**
RR 2+ sites with 4+ mm CAL (95% CI)	**1.65 (1.14,2.40)**	**1.62 (1.12,2.33)**	**1.51 (1.07,2.12)**	**1.45 (1.03,2.05)**
RR 1+ sites with 5+ mm CAL (95% CI)	**1.93 (1.19,3.11)**	**1.88 (1.18,2.99)**	**1.64 (1.04,2.59)**	**1.60 (1.02,2.50)**
RR 1+ sites with 3+ mm incident CAL (95% CI)	**1.79 (1.10,2.92)**	**1.75 (1.08,2.83)**	**1.65 (1.01,2.69)**	**1.64 (1.01,2.66)**

*Model 1 adjusted for sex and SES.

†Model 2 adjusted for sex, SES, use of dental services, plaque trajectory, and pack years to age 32 (smoking history).

‡Model 3 adjusted for sex, SES, use of dental services, and interaction between smoking and plaque trajectory.

Reference categories: male (female, coded 0), medium or low SES at age 32 (high SES coded 0), episodic user of dental services at age 32 (routine user of dental services at age 32, coded 0), moderate or high plaque trajectory (low plaque trajectory coded 0), non-smoker at age 32+ moderate plaque trajectory, non-smoker at age 32+ high plaque trajectory, smoker at age 32+ low plaque trajectory, smoker at age 32+ moderate plaque trajectory or smoker at age 32+ high plaque trajectory (non-smoker at age 32+ low plaque trajectory coded 0), high-familial-risk for periodontal disease (low-familial-risk for periodontal disease coded 0)

RR, relative risk; CAL, combined attachment loss; CI, confidence interval; SES, socioeconomic status. Significant findings in bold type.

viewed'' sample. It seems reasonable to assume that the potential for misclassification in the direction of undiagnosed disease would be an issue for periodontal disease. While most people are aware of having lost a tooth (for example), a substantial proportion may be unaware of having periodontal disease. It is possible that this error is compounded in the ''one or both parents interviewed'' sample, whereby no data were obtained from a non-attending parent (although it is not possible to speculate on which direction this misclassification may lie; i.e., whether or not it favoured the null hypothesis). This potential error may be one reason why the associations differ for the two samples; however, it is likely that other unknown factors may also be involved. In any case, it appears that predictive validity is enhanced if data from both parents can be obtained.

The interaction between smoking and plaque trajectory is not an unexpected finding. Smokers were more likely to be at a greater risk of periodontal disease than non-smokers, and smokers with high plaque trajectories were at greatest risk. These findings suggest that smoking and plaque trajectory combine to raise the risk of periodontal disease to a higher level than either of these factors acting independently, and highlight the necessity of assessing effect modification between smoking and plaque levels in periodontal disease research (Hyman 2006). Likewise, effect modification between smoking and familial-risk group is a reasonable finding; smokers in both low- and high-familial-risk groups are at greater risk of periodontal disease than non-smokers, and smokers in the high-familial-risk group are at greatest risk. Smoking exacerbates the impact of being in the high-familial-risk group for periodontal disease.

While the mechanisms underlying inter-generational continuity in periodontal health are unclear and are undoubtedly complex, there are a number of potential pathways whereby disease risk can be transmitted across generations. Inter-generational transmission of genetically or epigenetically determined traits may be one mechanism (Nadeau 2009, Skinner et al. 2010). Risk factors such as SES, smoking, and episodic use of dental care services may continue across generations, manifesting as poor health capital (Zimmerman 1992, Corcoran 1995, Chassin et al. 1998, Shenassa et al. 2003, Hill et al. 2005). Poor maternal health before and during pregnancy (and/or during the early post-natal period) can have an unfavourable impact on intrauterine foetal growth and neonatal development, in turn leading to adverse outcomes for the offspring later in the life course (Frankel et al. 1996, Lithell et al. 1996, Barker 1998, De Stavola et al. 2000, Power & Jefferis 2002). In fact, poor maternal periodontal health has been associated with an increased risk of pre-term birth and low birth weight in some populations (Wimmer & Pihlstrom 2008). Another mechanism involving a genetic predisposition coupled with exposure to environmental risk factors forms the basis for the gene–environment interaction model that is, the situation where both genetic and environmental factors interact to produce health outcomes in individuals and populations (Collins 2004, Hunter 2005, Moffitt et al. 2005).

Our findings provide evidence to suggest a causal association between parental periodontal health and proband periodontal health. Generally, periodontal disease has a later onset than other oral disease such as caries, and the association in this cohort between parental periodontal health and proband periodontal health may accordingly strengthen with age. The predictive validity of parental periodontal health information is enhanced if data from both parents can be obtained.

Conclusions

This study suggests that the children of parents with poor periodontal oral health are more likely to have poor periodontal health in adulthood than the children of parents with good periodontal health. Family/parental history of periodontal health appears to be a valid representation of the complex inter-play between shared genetic factors and shared environmental factors, exposures and behaviours that contribute to an individual's periodontal health status. Generally, it could be quickly and inexpensively assessed by clinicians, and along with assessment of SES and smoking history, may improve prediction of patient prognosis and preventive treatment need.

Acknowledgements

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Fig. S1. Smoking × Plaque Trajectory Effect Modifications – One or both parents sample.
Fig. S2. Smoking × Plaque Trajectory Effect Modifications – Both parents sample.
Fig. S3. Familial Risk × Plaque Trajectory Effect Modifications – One or both parents sample.
Fig. S4. Familial Risk × Plaque Trajectory Effect Modifications – Both parents sample.
Fig. S5. Smoking × Familial Risk Effect Modifications – One or both parents sample.
Fig. S6. Smoking × Familial Risk Effect Modifications – Both parents sample.
Table S1. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of one or more sites with 4+ mm combined attachment loss at age 32 (one or both parents in).
Table S2. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of two or more sites with 4+ mm combined attachment loss at age 32 (one or both parents in).
Table S3. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of one or more sites with 5+ mm combined attachment loss at age 32 (one or both parents in).
Table S4. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of one or more sites with 3+ mm incident combined attachment loss between ages 26 and 32 (one or both parents in).
Table S5. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of one or more sites with 4+ mm combined attachment loss at age 32 (both parents in).
Table S6. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of two or more sites with 4+ mm combined attachment loss at age 32 (both parents in).
Table S7. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of one or more sites with 5+ mm combined attachment loss at age 32 (both parents in).
Table S8. Outcomes of multivariate modelling, smoking-plaque effect modification (model 3), familial risk-plaque effect modification (model 4), and smoking- familial risk effect modification (model 5) for proband prevalence of one or more sites with 3+ mm incident combined attachment loss between ages 26 and 32 (both parents in).

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Clinical Relevance

Scientific rationale for the study: Family history of periodontal disease may be an early marker of shared genetic, epigenetic, and environmental influences associated with periodontal disease risk.

Principal findings: The children of parents with poor periodontal health are more likely to have poor periodontal health in adulthood than the children of parents with good periodontal health.

Practical implications: Generally, family/parental history of periodontal health could be quickly and inexpensively assessed by clinicians to improve prediction of patient prognosis and preventive treatment need.