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# Socioeconomic inequalities in oral health in childhood and adulthood in a birth cohort

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Abstract – *Objectives:* To determine whether adult oral health is predicted by (a) childhood socioeconomic advantage or disadvantage (controlling for childhood oral health), or (b) oral health in childhood (controlling for childhood socioeconomic advantage or disadvantage), and whether oral health in adulthood is affected by changes in socioeconomic status (SES). *Methods:* Participants in a longstanding cohort study underwent systematic dental examination for dental caries and tooth loss at ages 5 and 26 years. The examination at age 26 years included the collection of data on periodontal attachment loss and plaque level. Childhood SES was determined using parental occupation, and adult SES was determined from each study member's occupation at age 26 years. Regression models were used to test the study hypotheses. *Results:* Complete data were available for 789 individuals (47.4% female). After controlling for childhood oral health, those who were of low SES at age 5 years had substantially greater mean DFS and DS scores by age 26 years, were more likely to have lost a tooth in adulthood because of caries, and had greater prevalence and extent of periodontitis. A largely similar pattern was observed (after controlling for childhood SES) among those with greater caries experience at age 5 years. For almost all oral health indicators examined, a clear gradient was observed of greater disease at age 26 years across socioeconomic trajectory groups, in the following order of ascending disease severity and prevalence: 'high–high', 'low–high' (upwardly mobile), 'high–low' (downwardly mobile) and 'low–low'. *Conclusion:* Adult oral health is predicted by not only childhood socioeconomic advantage or disadvantage, but also by oral health in childhood. Changes in socioeconomic advantage or disadvantage are associated with differing levels of oral health in adulthood. The life-course approach appears to be a useful paradigm for understanding oral health disparities.

Key words: caries; cohort study; periodontal disease; socioeconomic status; tooth loss

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The life-course approach is useful in documenting and explaining differences in health. The basic tenet underlying the approach is that, throughout life, adverse exposures gradually accumulate by way of ill-health episodes, environmental factors or individual behaviours which increase the risk of chronic disease and mortality. In the context of chronic health conditions, the accumulation of risk occurs through a range of biological events and social experiences over time. Depending on the condition, this may occur gradually throughout the entire life course, or there may be certain critical or sensitive periods when adverse exposures are likely to be more detrimental. Through the resultant chain of risk or advantage, certain experiences or exposures in early life increase the likelihood of future events which, in turn, lead to greater or lower risk of adult disease (1). Those chains of risk

may be biological or social; with the former, exposures to causal factors during gestation, early childhood and early adulthood contribute towards health in later adulthood. The latter includes a socioeconomically compromised start to life and is linked to adult socioeconomic conditions which influence disease risk through later-life exposures to causal factors. Social inequalities in health and disease contribute to these processes of accumulating advantage or disadvantage (2). Of course, the context is not the only consideration; for a given setting, an individual's behaviour helps to shape not only his/her current circumstances, but also his/her future health pathway.

The life-course concept appears to be well-suited to oral health (Fig. 1). Dental caries is a good exemplar, being not only chronic and highly prevalent, but largely irreversible and cumulative in nature. The evidence of an individual's past and present disease experience is readily revealed by dental examination: sequelae such as tooth loss or restorations provide readily measured end-points. Where dental caries is concerned, an example of the direct environmental effect would be having lower access to health-promoting environmental exposures such as water fluoridation. The indirect effect could operate through having sub-optimal oral hygiene practices (and an associated lower exposure to fluoride-containing toothpaste), poorer diet, and lower access to health-promoting exposures such as clinical preventive dentistry.

Constitutional vulnerability (the heritable elements of health) is also pertinent to the life-course approach, and is represented in Fig. 1 as an underlying determinant of the individual's susceptibility to disease. This notion is probably more appropriate when considering a disease such as periodontal disease, where genetic susceptibility is thought to play a large role in the level of tissue destruction occasioned by the individual's chronic inflammatory response to the bacterial challenge (3).

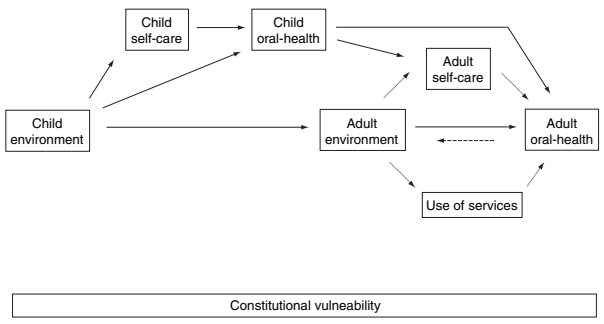


*Fig. 1.* Chains of oral health risk through the life course [adapted from Ref. (1)].

Social inequalities in oral health have been well documented in the dental scientific literature, providing plenty of evidence for the poorer oral health of lower-socioeconomic status (SES) groups compared with their higher-SES counterparts (4–10). The association between the prevalence of caries and SES is less marked among children living in areas with community water fluoridation (5, 7, 8, 11–14). In New Zealand, the first evidence of this was from an analysis of the age 5-year data in the Dunedin Multidisciplinary Health and Development Study (DMHDS), which showed that the observed social gradient in primary caries prevalence was greater among cohort members who had been living in areas without community water fluoridation (5).

All the studies cited above have been cross-sectional analyses; this approach precludes examining the antecedents and persistence of the observed socioeconomic inequalities. Key research questions include: (a) Is poor adult oral health predicted by socioeconomic disadvantage in childhood, after controlling for childhood oral health?; (b) Is poor adult oral health predicted by poor oral health in childhood, after controlling for childhood socioeconomic disadvantage?; and (c) Is oral health in adulthood affected by changes in SES? In other words, what currently remains unclear is the extent to which childhood disadvantage is associated with poor oral health in adulthood, and whether changes in social circumstances can modify that relationship. Concerning the latter, the *upward mobility* hypothesis holds that rising in the SES hierarchy from childhood to adulthood has a beneficial effect on health, while *downward mobility* would be expected to have a detrimental effect; contrasting with these is the *social-origins hypothesis*, by which growing up in low-SES conditions has detrimental effects for adult health irrespective of the individual's adult-SES destination (15). The applicability of these hypotheses to oral health outcomes such as tooth loss has yet to be examined. Watt (16) has recently called for research identifying 'windows of opportunity' when health promotion interventions may have the greatest long-term benefit in promoting oral health and the reduction of inequalities. Essential to that process is investigating the natural history – and amenability to change – of oral health inequalities.

A comprehensive study of oral health inequalities through the life course should: (i) use the cohort study design; (ii) examine a representative sample; (iii) commence early in the participants' lives; (iii) collect data on a wide range of social, physical and oral characteristics; and (iv) follow-up participants for as long as possible, in order to determine the natural history of those inequalities. Poulton et al. (17) recently reported their analysis of whether childhood advantage or disadvantage predicted a wide range of health conditions in a longitudinal study of New Zealanders. They found that, after controlling for neonatal health, children who grew up in low-SES households were likely to have poorer physical and oral health as young adults. The aim of this study was to undertake a detailed examination of the nature and persistence of social inequalities in the occurrence of dental disease among participants in a longstanding cohort study of New Zealanders.

## Methods

### *The sample*

Data for this study were obtained from assessments conducted at ages 0, 3, 5 and 26 years as part of the DMHDS, a longitudinal study of children born in Dunedin during 1972–73 (18). Perinatal data were obtained and the sample for the longitudinal study was defined at age 3 years. This initially comprised 1037 children assessed within a month of their third birthdays and again at ages 5, 7, 9, 11, 13, 15, 18, 21 years and, most recently, at age 26 years, when 980 (96%) of the surviving 1019 study members were assessed. Barriers to study members' participation were minimized by the unit assuming the costs of participation (such as travel, lost wages, child care). The various assessments (e.g. oral health, mental health, physical health) are presented as standardized modules in a counterbalanced order and each is conducted by a different examiner who is kept blind to all study data.

Ethical approval for the current study was obtained from the Otago Ethics Committee, and informed consent was obtained from all participants.

### *Measures*

Two estimates of social class were obtained for each participant by using data collected on parental SES, using standard New Zealand occupationally based indices (19, 20), which employ a six-interval classification (where, e.g. a doctor scores '1' and a labourer scores '6'). In the current analyses, early childhood SES was determined by the SES level of the child's father at the time of the study member's birth. Where the father was unable to be categorized in this way, data on the mother were used. Where neither parent could be categorized (as in the situation where the father was unemployed and the mother was categorized as a housewife), the corresponding data from the age-3-year assessments were used in the same way; similarly, where an SES category was unable to be assigned, data from the age-5-year assessments were used in the same way. This enabled the individuals to be assigned to one of two early childhood SES groups: those with a score of '1', '2' or '3' were allocated to the 'high-SES' group, while the remainder were allocated to the 'low-SES' group. A measure of adult SES was obtained using the study member's adult occupation, assessed during the age-26-year interview, and study members were assigned to low or high groups using the same algorithm. Having separate measures of early childhood ('origin') and adult ('destination') SES allowed each participant to be allocated to one of the following four separate SES 'trajectories': those who were in the high-SES group up to the age of 5 years and at age 26 years were categorized as the 'high–high' group; those in the low-SES group at ages 5 and 26 years were designated the 'low–low' group; and the 'high–low' and 'low–high' groups comprised those who (respectively) were downwardly and upwardly mobile.

Dental examinations for caries and missing teeth at ages 5 and 26 years were conducted by trained dental examiners. Repeat examinations were not possible because of the logistical constraints imposed by the tightly scheduled assessment that study members underwent (while this absence of reliability examinations is an important theoretical consideration, its actual importance to the current study's findings is questionable, particularly in the light of the magnitude of the observed differences). Dental examiners were not aware of study members' SES at the time of the examinations. An estimate of accumulated tooth loss because of caries was obtained by observing the presence or absence of each tooth at age 26 years, and ascertaining the reason for its absence at that age by asking the study member at the time of the examination. In this study, third molars were not included in the

computation of tooth loss; only those teeth which had been lost because of caries were included in the analysis.

At age 26 years, periodontal measurements (not conducted on the 15 individuals who reported a history of cardiac valvular anomalies or rheumatic fever) were made in two quadrants: the upper right and lower left for study members whose study ID number was odd; the upper left and lower right for those with an even ID number; the mix of odd and even numbers was approximately 50 : 50. Three sites (mesiobuccal, buccal, and distolingual) per tooth were examined. Probing depth (PD; the distance from the tip of the probe to the gingival margin) and gingival recession (GR; the distance from the gingival margin to the amelocemental junction) were recorded using an NIDR probe. Midbuccal measurements for molars were made at the midpoint of the mesial root. All measurements were rounded down to the nearest whole millimetre at the time of recording. Plaque accumulation was measured using the Simplified Oral Hygiene Index (OHI-S; 21). Third molars were not included in the analysis of the periodontal data. The case definition for periodontitis identified individuals with one or more sites with 4+ mm combined attachment loss (CAL). Measurements made at age 18 years on six index teeth using the Community Periodontal Index of Treatment Needs (CPITN) (22) indicate that no study members had PD >3 mm at that age; thus, there were no cases of juvenile periodontitis in the cohort (24).

Residential fluoride exposure up to age 5 years was computed as the percentage of those years spent living in an area with community fluoridation. Similarly, residential fluoride exposure up to age 26 years was computed as the percentage of those years spent living in an area with community fluoridation. Each of the residential fluoride exposure variables was dichotomized to distinguish those who had spent all their lives in fluoridated communities from the remainder.

Making use of dental services was determined by asking study members whether they usually visited the dentist for a check-up or because of a problem. Those who reported the latter were designated 'episodic users' of dentistry. Study members were categorized as 'chronic smokers' if they gave a positive response to the question 'Have you smoked daily for one month or more in the previous 12 months?' at both ages 21 and 26 years.

### *Data analysis*

Following the computation of univariate statistics, bivariate associations were tested for statistical significance ($\alpha = 0.05$) using chi-square tests for categorical data, and nonparametric tests (Mann–Whitney or Kruskal–Wallis tests, where appropriate) for continuous variables. Regression analyses were conducted to test the study hypotheses and derive adjusted estimates for the dependent variables. Poisson regression modelling was used for count data (such as DFS and DS), linear regression was used for continuous variables (such as plaque scores), and logistic regression was used for dichotomous oral health outcomes (such as the prevalence of periodontal disease). The estimation of the association of childhood SES and adult oral health controlled for sex, adult dental visiting pattern and age-5-year dmfs (representing childhood oral health). Estimates for the caries and tooth-loss variables were also adjusted by the length of residence in a fluoridated community, while the estimates for periodontitis prevalence and extent were also adjusted for smoking (as 31.7% of study members smoked at both 21 and 26 years of age). Estimation of the association between child and adult oral health involved controlling for sex, adult dental visiting pattern and age-26 SES.

## Results

Dental examinations were conducted on 922 study members at age 5 years, and on 930 members at age 26 years. Dental examination data from both ages were available for 838 individuals (90.1% of those examined at age 5 years). Allocation to a SES category in both early childhood and at age 26 years was possible for 789 (94.2%) of those, of whom 374 (47.4%) were female, and 58 (7.4%) were Māori. By age 5 years, 386 (48.9%) had never lived in an area with community water fluoridation, while 377 (47.8%) had spent all of their lives in one. By age 26 years, only 99 (12.5%) had never lived in an area with community water fluoridation, while 235 (29.8%) had spent all of their lives (to date) in one.

Comparison of those who were dentally examined at ages 5 and 26 years – but were not able to be allocated to a SES score at both ages – with those who were included in this study shows some important differences between the two groups

(Table 1). More of the former were female, had lost one or more teeth because of caries, or had established periodontal disease. Subsequent analyses are confined to the 789 study members for whom complete data were available.

### *Oral health and SES at age 5 years*

The cohort's dental caries experience at age 5 years is presented in Table 2 by sex, early childhood SES group and time spent living in an area with community water fluoridation. The prevalence and severity of caries were higher among the low-SES group, who also had a higher number of untreated carious lesions. The number of untreated carious lesions was greater among males.

### *Socioeconomic disadvantage in early childhood and adult oral health*

Adjusted estimates of the cohort's age-26-year dental disease experience are presented by age-5-year SES group in Table 3. There were statistically significant differences between the high- and low-SES groups in all oral health measures examined.

### *Oral health in early childhood and adult oral health*

Adjusted estimates of the cohort's age-26-year dental caries experience by age-5-year dental caries experience are presented in Table 4. There were statistically significant differences between those who had and had not had caries by age 5 years in mean DFS, mean DS, and the mean number of teeth lost because of caries. When the cohort was dichotomized according to the highest quartile for dmfs at age 5 years (dmfs > 4), those in the highest quartile had higher mean scores for DFS, DS and the mean number of missing teeth by age 26 years, and also had more extensive periodontal loss of attachment.

### *Changes in socioeconomic status and oral health in adulthood*

Of the 313 study members who were in the high-SES group in early childhood, 151 (48.2%) had moved to the low-SES group by age 26 years; of the 476 who were in the low-SES group in early childhood, 169 (35.5%) had moved to the

Table 1. Comparison of characteristics of those included and not included in this analysis (data refer to age 26 years unless otherwise specified, and to the 838 study members who were dentally examined at both ages 5 and 26 years)

| | Not included | Included |
|---|---|---|
| Number in group | 49 (5.8) | 789 (94.2) |
| Number of males (%) | 7 (14.3) | 415 (52.6)* |
| Brush less than once daily (%) | 8 (16.3) | 66 (8.4) |
| Mean plaque score (SD) | 0.92 (0.48) | 0.86 (0.53) |
| Mean dmfs at age 5 years (SD) | 3.04 (5.44) | 3.63 (5.63) |
| Mean DFS (SD) | 13.00 (10.26) | 11.42 (9.92) |
| Number with 1+ teeth missing because of caries (%) | 13 (26.5) | 76 (9.6)* |
| Mean number of missing teeth because of caries (SD) | 0.43 (0.87) | 0.17 (0.64)* |
| Number with 1+ sites with 4+ mm CAL (%) | 22 (44.9) | 140 (17.7)* |
| Mean extent of sites with 4+ mm CAL (SD) | 3.44 (5.35) | 0.83 (2.59)* |

*$P < 0.01$.

Table 2. Age-5-year dental caries experience by early childhood SES, sex and exposure to community water fluoridation

| | Number | Number with caries (%) | Mean dmfs (SD) | Mean ds (SD) | Number missing 1 or more teeth (%) |
|---|---|---|---|---|---|
| Sex | | | | | |
| Female | 374 | 219 (58.6) | 3.41 (5.28) | 0.63 (1.97)* | 20 (5.3) |
| Male | 415 | 244 (58.8) | 3.83 (5.92) | 0.92 (2.33) | 23 (5.5) |
| SES group in early childhood | | | | | |
| High | 313 | 165 (52.7)** | 2.70 (4.31)** | 0.45 (1.33)** | 11 (3.5) |
| Low | 476 | 298 (62.6) | 4.24 (6.28) | 1.00 (2.55) | 32 (6.7) |
| Time spent living in fluoridated area | | | | | |
| 0–4 years | 412 | 243 (59.0) | 3.67 (5.47) | 0.85 (2.35) | 22 (5.3) |
| 5 years | 377 | 220 (58.4) | 3.59 (5.80) | 0.71 (1.95) | 21 (5.6) |
| All combined | 789 | 463 (58.7) | 3.63 (5.63) | 0.78 (2.17) | 43 (5.4) |

*$P < 0.05$; **$P < 0.01$.

Table 3. Adjusted values for age-26-year oral disease experience by SES at age 5 years

| | SES group at age 5 years | | |
|---|---|---|---|
| | High | Low | *P*-value |
| Number in group | 313 (39.7%) | 476 (60.3%) | |
| Dental caries | | | |
| Mean DFS[a] | 10.52 | 11.54 | <0.001 |
| Mean DS[a] | 1.60 | 1.88 | 0.003 |
| Tooth loss from caries | | | |
| Percentage with >1 teeth missing[a] | 4.66 | 9.93 | 0.007 |
| Mean no. missing teeth[a] | 0.08 | 0.17 | <0.001 |
| Periodontal disease | | | |
| Percentage with periodontitis[b] | 12.80 | 19.75 | 0.013 |
| Mean % affected sites[b] | 0.53 | 0.96 | <0.001 |
| Self-care | | | |
| Plaque score[c] | 0.81 | 0.90 | 0.02 |

[a]Adjusted for sex, fluoride exposure, dental visiting pattern and age-5-year dmfs.
[b]Adjusted for sex, smoking at ages 21 and 26 years, dental visiting pattern and age-5-year dmfs.
[c]Adjusted for sex and dental visiting pattern.

high-SES group by age 26 years. Adjusted age-26-year estimates for oral disease experience and plaque scores by SES trajectory are presented in Table 5. The severity of dental caries experience (mean DFS) was lowest in the 'low–high' group and highest in the 'low–low' group, with the 'high–high' and 'high–low' groups occupying an intermediate position (the mean score in the 'low–high' group being lower than that in the 'high–low' group). A more consistent biological gradient was apparent with mean DS, with the lowest score in the 'high–high' group, the next lowest in the 'low–high' group, followed by the 'high–low' and 'low–low' groups. The same gradient across the SES trajectory groups was observed with respect to tooth loss (with respect to both prevalence and the mean number of missing teeth) and plaque scores. Where the prevalence and extent of periodontal attachment loss are concerned, the 'high–high' and 'high–low' groups had the lowest scores, followed by the 'low–high' and 'low–low' groups. As with mean DFS by age 26 years, the gradient across the SES trajectory groups was not as clear-cut for periodontal disease experience (despite the highly significant *P*-values for linear trend).

The outcomes from testing the life-course hypotheses are also presented in Table 5. To test the social-origins hypothesis, the combined estimates from the 'low–high' and 'low–low' groups were compared with combined estimates from the 'high–high' and high–low' groups: the differences were statistically significant for all oral health outcomes examined. The upward mobility hypothesis was tested by comparing the estimates from the 'low–high' group with those from the 'low–low' group: the differences were statistically significant for all but the periodontal disease outcomes (and the latter approached statistical significance; $P = 0.09$). To test the downward mobility hypothesis, the estimates from the 'high–low' group were compared with those from the 'high–high' group: the differences were statistically significant for the mean DS, the mean number of teeth missing because of caries, and plaque scores, but not for the other outcomes.

Table 4. Adjusted values for age-26-year oral disease experience by dental caries experience at age 5 years

| | Any caries at age 5 years (dmfs > 0) | | | High caries at age 5 years (dmfs > 4) | | |
|---|---|---|---|---|---|---|
| | No | Yes | *P*-value | No | Yes | *P*-value |
| Number in group | 326 (41.3) | 463 (58.7) | | 577 (73.1) | 212 (26.9) | |
| Dental caries | | | | | | |
| Mean DFS[a] | 8.46 | 13.36 | <0.001 | 9.55 | 16.19 | <0.001 |
| Mean DS[a] | 1.45 | 2.00 | <0.001 | 1.60 | 2.24 | <0.001 |
| Tooth loss from caries | | | | | | |
| Percentage with >1 teeth missing[a] | 5.66 | 8.75 | 0.09 | 6.61 | 9.76 | 0.10 |
| Mean no. missing teeth[a] | 0.08 | 0.17 | <0.001 | 0.11 | 0.19 | 0.002 |
| Periodontal disease | | | | | | |
| Percentage with periodontitis[b] | 14.98 | 17.99 | 0.27 | 15.69 | 19.64 | 0.19 |
| Mean % affected sites[b] | 0.67 | 0.83 | 0.08 | 0.67 | 1.02 | <0.001 |
| Self-care | | | | | | |
| Plaque score[c] | 0.90 | 0.84 | 0.12 | 0.88 | 0.83 | 0.27 |

[a]Adjusted for sex, fluoride exposure, dental visiting pattern and age-5-year SES.
[b]Adjusted for sex, dental visiting pattern, smoking at ages 21 and 26 years, and age-5-year SES.
[c]Adjusted for sex and dental visiting pattern.

Table 5. Adjusted values for age-26-year oral disease experience by SES trajectory

| | SES trajectory (early childhood SES → age-26-year SES) | | | | | *P*-values for life-course hypotheses[d] | | |
|---|---|---|---|---|---|---|---|---|
| | High–high | Low–high | High–low | Low–low | *P*-value for linear trend | Social origins | Upward mobility | Downward mobility |
| Number in group | 162 | 169 | 151 | 307 | | | | |
| Dental caries | | | | | | | | |
| Mean DFS[a] | 10.41 | 10.09 | 10.62 | 12.38 | <0.001 | <0.001 | <0.001 | 0.56 |
| Mean DS[a] | 1.26 | 1.61 | 1.94 | 2.05 | <0.001 | 0.003 | 0.001 | <0.001 |
| Tooth loss from caries | | | | | | | | |
| Percentage with >1 teeth missing[a] | 3.00 | 5.45 | 6.38 | 12.53 | <0.001 | 0.007 | 0.02 | 0.14 |
| Mean no. missing teeth[a] | 0.04 | 0.09 | 0.12 | 0.23 | <0.001 | <0.001 | <0.001 | 0.012 |
| Periodontal disease | | | | | | | | |
| Percentage with periodontitis[b] | 12.52 | 14.79 | 13.22 | 22.45 | 0.006 | 0.013 | 0.09 | 0.66 |
| Mean % affected sites[b] | 0.51 | 0.82 | 0.55 | 1.04 | <0.001 | <0.001 | 0.05 | 0.35 |
| Self-care | | | | | | | | |
| Plaque score[c] | 0.75 | 0.81 | 0.88 | 0.95 | <0.001 | 0.04 | 0.006 | 0.03 |

[a]Adjusted for sex, fluoride exposure, dental visiting pattern, and age-5-year dmfs.
[b]Adjusted for sex, smoking at ages 21 and 26 years, dental visiting pattern, and age-5-year dmfs.
[c]Adjusted for sex and dental visiting pattern.
[d]Comparisons for these were as follows: for the social-origins hypothesis, the combined data for the 'low–high' and 'low–low' groups were compared with the combined data for the other two groups; for the upward mobility hypothesis, the 'low–high' group was compared with the 'low–low' group; and, for the downward mobility hypothesis, the 'high–low' group was compared with the 'high–high' group.

## Discussion

This investigation used the prospective observational study design to examine the nature and persistence of social inequalities in the occurrence of dental disease among participants in a long-standing study of a representative birth cohort. Each of the three research questions is addressed, in turn, below.

### *Is poor adult oral health predicted by socioeconomic disadvantage in childhood, after controlling for childhood oral health?*

The study has found that not only were oral health inequalities present at age 5 years (Table 2), but they were also apparent at age 26 years when the early childhood SES categories were used (Table 3), suggesting that early socioeconomic inequalities in a number of important oral health indicators do persist well into the third decade of life. Although state-funded dental care is provided for New Zealand children under the age of 5 years, there are social differences in access to, and uptake of, that care (23), and it is not until children are attending school that the principle of universality of access is actually realised (24). The current study's evidence that childhood disadvantage has an enduring effect on oral health is particularly noteworthy, because the New Zealand dental care system ensures access to free state-funded dental care until age 18 years, when there is an abrupt transition to self-funded dental care (24). By age 18 years, only two study members had lost permanent teeth due to caries, whereas almost one-tenth of the cohort had done so by age 26 years (25), when profound socioeconomic differences had re-emerged after being very much reduced during the years of schooling (26). This finding strongly suggests that, while universal access to dental health care from childhood through adolescence may act protectively to dampen the effect of SES inequity, the effect may not persist once that universal access ends.

### *Is poor adult oral health predicted by poor oral health in childhood, after controlling for childhood socioeconomic status?*

The data on this issue were less clear-cut. The evidence was unequivocal where dental caries is concerned: having high disease experience early in life predicted having greater disease experience in adulthood, other factors being equal. The pattern was not as clear with periodontal disease, however: while those in the high-caries group at age 5 years had (on average) more extensive attachment loss by age 26 years, their greater disease prevalence was not statistically significant.

### *Is oral health in adulthood affected by changes in socioeconomic status?*

The third research issue addressed in this study was the extent to which changes in socioeconomic circumstances are associated with different oral health outcomes in adulthood. Is the news all bad for those born into low-SES households, or does adult SES also exert an influence? The data in Tables 3 and 5 offer compelling support for the *social-origins hypothesis* (15), indicating that those born in low-SES households will, on average, have poorer oral health than their high-SES counterparts. For example, the two groups with the highest prevalence and extent of periodontitis by age 26 years were the 'low–high' and 'low–low' groups; that is, the individuals who originated in low-SES households. That gradient persisted irrespective of whether the analysis controlled for smoking exposure. However, the data in Table 5 also confirm that destination SES matters. For example, support for the *upward mobility* hypothesis is provided in the observation that those who rose to the high-SES group from the low-SES group were second only to the 'high–high' group in almost all oral health outcomes measured, and that the differences were statistically significant for most outcomes examined. There is also some support for the *downward mobility* hypothesis (although it is equivocal): those on the downward trajectory had (on average) dirtier teeth, more missing teeth, and more untreated decayed surfaces than those who remained in the high-SES group, but they did not differ with respect to overall DFS, tooth-loss prevalence, or their periodontal disease experience. In essence, from an oral health viewpoint, it appears that it is better to be born into a high-SES household; if that cannot be achieved, then ascending in the SES hierarchy is likely to assuage most (if not all) of that earlier disadvantage.

From a methodological viewpoint, it is appropriate to consider the study's potential shortcomings. First, the measures of SES were limited to occupational status (parental occupation for childhood SES; study member occupation for adult SES), ignoring other potential indicators of social inequality. Accordingly, we replicated the trajectory analysis with this cohort using participants' educational achievement (university graduates versus the remainder) for the measure of adult SES, and there was no change to the overall patterns observed (data available on request). Secondly, we have assumed that SES at age 26 years reflects each individual's final socioeconomic destination, notwithstanding the fact that subsequent life events or achievements may have an influence. However, most Study members had completed their formal education by age 26 years, and it is unlikely that many will change their SES, given that educational attainment is a strong predictor of mid-life SES (27).

A third potential shortcoming is the possibility of some misclassification arising from our division of the cohort by allocating those with Elley-Irving SES scores 1, 2 or 3 to the high-SES group (and scores 4, 5 or 6 to the low-SES group), as it could be argued that there may be little difference between those near the cut-off point; that is, those with scores 3 or 4. Examples of occupations which score 3 in the occupational classification are electricians, clerks and nurses, while grocers, motor mechanics and farmers would score 4. In a similar analysis with the same cohort, Poulton et al. (17) divided participants into three SES groups (scores 1 and 2, 3 and 4, and 5 and 6) and then eliminated all for whom the middle was the origin or destination SES group. Their rationale for this approach was maximizing the contrast between the groups being examined. For the current analysis, statistical power considerations necessitated the modified approach used here. We did, however, repeat the analyses after omitting those with scores 3 or 4, and there was no change to the patterns which were observed, with the gradients in the adjusted values across the SES trajectory groups similar to those in Table 5 for all measures (data available on request).

### *Implications*

Data such as those reported here beg the question of the extent to which dental health services interventions can reduce oral health inequalities. Efforts to do so must take cognisance of the enduring effect of childhood disadvantage on oral health. Three distinct strategies have been identified for reducing inequalities in oral health (26): (a) changing basic socioeconomic determinants, such as reducing the prevalence of poverty and improving educational and employment opportunities; (b) changing intermediary factors between oral health and its socioeconomic determinants, such as increasing the availability of topical fluoride; and (c) developing oral health services which are more suitable, both by developing new services where appropriate, and by making appropriate changes to existing services. The findings of this study suggest not only that the

first of these has the greatest potential for reducing inequalities, but that it is likely to be the most difficult and involve the greatest delay. Future oral health analyses with the Dunedin cohort will examine the nature of the association between ethnicity, SES and oral health through the life course.

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## References

1. Kuh D, Ben Shlomo Y. A life course approach to chronic disease epidemiology. Oxford: Oxford University Press; 1997.
2. Blane D. The life course, social gradient and health. In: Marmot M, Wilkinson RG, editors. Social inequalities and health. Oxford: Oxford University Press, 1999. p.64–80.
3. Page RC. Milestones in periodontal research and the remaining critical issues. J Periodont Res 1999;34:331–9.
4. Cutress TW, Hunter PBV, Davis PB, Beck DJ, Croxson LJ. Adult oral health and attitudes to dentistry in New Zealand 1976. Wellington: Medical Research Council of New Zealand; 1979.
5. Evans RW, Beck DJ, Brown RH, Silva PA. Relationship between fluoridation and socioeconomic status on dental caries experience in 5-year-old New Zealand children. Community Dent Oral Epidemiol 1984;12:5–9.
6. Hunter PBV, Kirk R, de Liefde B. The study of oral health outcomes. Wellington: Department of Health; 1992.
7. Provart SJ, Carmichael CL. The use of an index of material deprivation to identify groups of children at risk to dental caries in County Durham. Community Dent Health 1995;12:138–42.
8. Fergusson DM, Horwood LJ. Relationships between exposure to additional fluoride, social background and dental health in 7-year-old children. Community Dent Oral Epidemiol 1986;14:48–52.
9. Brown RH, Treasure ET. Inequities in oral health: implications for the delivery of care and health promotion. N Z Dent J 1992;88:132–8.
10. Schou L, Uitenbrook D. Social and behavioural indicators of caries experience in 5-year-old children. Community Dent Oral Epidemiol 1995;23:276–81.
11. Jones CM, Taylor GO, Whittle JG, Evans D, Trotter DP. Water fluoridation, tooth decay in 5 year olds, and social deprivation measured by the Jarman score: analysis of data from British dental surveys. Br Med J 1997;315:514–7.
12. Treasure ET, Dever JG. The prevalence of caries in 5-year-old children living in fluoridated and non-fluoridated communities in New Zealand. N Z Dent J 1991;88:9–13.
13. Treasure ET, Dever JG. Relationship of caries with socioeconomic status in 14-year-old children from communities with different fluoride histories. Community Dent Oral Epidemiol 1994;22:226–30.
14. Slade GD, Davies MJ, Spencer AJ, Stewart JF. Influence of exposure to fluoridated water on socioeconomic inequalities in children's caries experience. Community Dent Oral Epidemiol 1996;24:89–100.
15. Rutter M. Psychosocial influences: critiques, findings, and research needs. Develop Psychopathol 2000;12: 375–405.
16. Watt RG. Emerging theories into the social determinants of health: implications for oral health promotion. Community Dent Oral Epidemiol 2002;30:241–7.
17. Poulton R, Caspi A, Milne BJ, Thomson WM, Taylor A, Sears MR et al. Children's experience of socioeconomic disadvantage predicts their poor adult health: a longitudinal study. The Lancet 2002;360: 1640–5.
18. Silva PA, Stanton W. From child to adult: the Dunedin Multidisciplinary Child Development Study. Auckland: Oxford University Press; 1996.
19. Irving JC, Elley WB. A socio-economic index for the female labour force in New Zealand. N Z J Educ Stud 1977;12:154–63.
20. Elley WB, Irving JC. The Elley-Irving socio-economic index 1981 Census revision. N Zeal J Educ Stud 1985;20:115–28.
21. Greene JC, Vermillion JR. The simplified oral hygiene index. J Am Dent Assoc 1964;68:7–13.
22. Cutress TW, Ainamo J, Sardo-Infirri J. The community periodontal index of treatment needs (CPITN) procedure for population groups and individuals. Int Dent J 1987;37:222–33.
23. Beautrais AL, Fergusson DM, Shannon FT. Use of preschool dental services in a New Zealand birth cohort. Community Dent Oral Epidemiol 1982;10:249–52.
24. Thomson WM. Use of dental services by 26-year-old New Zealanders. N Z Dent J 2001;97:44–8.
25. Thomson WM, Poulton R, Kruger E, Boyd DH. Socioeconomic and behavioural risk factors for tooth loss from age 18 to 26 among participants in the Dunedin Multidisciplinary Health and Development Study. Caries Res 2000;34:361–6.
26. National Health Committee. Improving child oral health and reducing child oral health inequalities. Report to the Minister of Health from the Public Health Advisory Committee. Wellington: Ministry of Health; 2003 (Available from http://www.nhc.govt.nz/phac_pubs.htm).
27. Hauser RM, Warren JR, Haung MS, Carter WY. Social stratification across three generations. In: Arrow K, Bowles S, Durlauf S, eds. Meritocracy and inequality. Princeton, NJ: Princeton University Press; 2000. p. 179–229.