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Dental restorations: a risk factor for periodontal attachment loss?

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Abstract

Background—Dental caries and restorations in proximal tooth surfaces often impinge upon the periodontal biological width.

Aim—This study examines whether these factors may contribute to risk for periodontal attachment loss at these sites.

Methods—The study is based upon data from the Dunedin Multidisciplinary Health and Development Study, a long-standing cohort study. Approximal tooth surfaces of 884 study members were evaluated for restorations and caries at age 26 and again at 32 years, and probing depth and gingival recession were recorded in millimetres at age 32. Attachment loss was computed as the sum of pocket depth and gingival recession. Data were analysed using generalized estimating equations.

Results—Where a caries/restorative event had occurred on an inter-proximal tooth surface before age 26, the age-32 attachment loss at the corresponding periodontal site was approximately twice more likely to be ≥3 mm than if the adjacent tooth surface had remained sound to age 32. This was also true where a caries/restorative event had occurred subsequent to age 26. The association remained after controlling for potential confounders, including smoking.

Conclusions—Site-specific periodontal attachment loss due to dental caries or restorative events occurs in adults in their third and fourth decades of life.

Keywords

biological width; caries; cohort study; longitudinal study; periodontal attachment loss; periodontal disease

Over the past 40 years, numerous studies have considered the effects of the location of restoration margin placement, restoration surface integrity, and type of restorative material on post-intervention periodontal tissue status. There is little doubt that poorly contoured restorations can increase plaque retention and/or violate the biologic width. However, there is controversy about whether the placement of a new restoration pre-disposes the adjacent periodontal tissues to future breakdown (Albandar et al. 1995, Schatzle et al. 2001, Paolantonio et al. 2004).

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One 5-year study of 114 adults reported a mean increase in periodontal pocket depth of 1.2 mm following the placement of crowns with subgingival margins (even when professionally administered prophylaxis was provided every 6 months); where the crowns were placed with supragingival margins, a mean increase of only 0.6 mm was reported by the end of the study period (Valderhaug & Birkeland 1976). Another study compared periodontal measurements from abutment teeth and non-abutment teeth in 55 individuals 15 years following the placement of fixed restorations: gingival index scores and pocket depths were slightly greater for abutment teeth; however, a majority of sites (57%) had pocket depths at 2 mm, and those sites that lost attachment did so within the first 5 years following restoration placement (Valderhaug et al. 1993).

Two separate longitudinal studies performed on adolescents and adults males in Norway confirmed the relationship between caries and restorations and future periodontal disease breakdown. A Scandinavian study followed a randomly selected group of Norwegian males for 26 years, and obtained periodontal data for 160 individuals at seven intervals during the study period. Data collected included measures of the gingival index, plaque index, gingival caries index, gingival restoration index, and gingival recession and loss of attachment [clinical attachment level (CAL)] at each observation period. “Test” and “Control” sites were defined based on the presence or absence of restorations at baseline and throughout the study period. The plaque index scores increased in each group over time, with no difference between the test and control sites. In contrast, gingival index and gingival caries index scores were greater for the test sites at several observation periods. With respect to the “gold standard” for periodontal destruction – clinical attachment loss – there were only small differences between the test and control sites across time. The greatest CAL occurred between 2 and 4 years in the test sites group; however, the pattern of CAL over time was similar for test and control sites, with differences being relatively equivalent after 26 years, suggesting that the presence of restorations was not associated with periodontal attachment loss (Schatzle et al. 2001). Another longitudinal study of periodontal disease examined the association between caries and restorations, and periodontal disease in a sample of 227 13-year-old adolescents over a 3-year-period. These authors reported significantly greater odds for having gingival inflammation and radiographic bone loss at sites approximal to teeth with manifest caries, defective fillings, and non-defective fillings. While these results were statistically significant, the odds ratios (ORs) and 95% confidence intervals (CIs) subsequently computed by us from the *b* estimates and SEs were very small at 1.07 (1.05, 1.08), 1.12 (1.10, 1.13), and 1.09 (1.08, 1.11), respectively. CAL was determined by measuring the distance to the alveolar crest to the nearest 0.1 mm using bitewing radiographs taken at yearly intervals. The impact of other confounding variables was not considered in their analyses. None of the longitudinal studies reported to date has controlled for the potential effects of other covariates known to be associated with periodontal inflammation while concomitantly allowing for the clustering of sites within individuals (Albandar et al. 1995).

By contrast, a 1-year longitudinal study found that neither periodontal clinical parameters nor site-specific microbiology changed 12 months after the placement of amalgam or glass ionomer restorations. There was, however, a statistically significant increase in total bacterial counts obtained from subgingival sites adjacent to composite resin restorations at 8- and 12-month follow-up assessments. The authors concluded that composites may create a greater hazard to periodontal health than other restorative materials. It is not possible to generalize from these findings, however, as they were obtained from a sample of only 16 healthy individuals for whom oral hygiene was continually reinforced throughout the short 1-year study period (Paolantonio et al. 2004).

While these studies comprise the best longitudinal evidence to date, the generalizability of their findings is limited due to design and statistical problems (Cook & Campbell 1979). Most

studies did not account for the inter-dependence among multiple sites observed within individuals. In addition, most used simple bivariate analyses to attempt to capture differences between groups and trends without controlling for inflation of the type 1 error rate. There is a need to investigate the relationship between restorations and periodontal health while controlling for the confounding effect of other known risk factors. Multi-level statistical analyses are most appropriate for periodontal data because of the non-independence of sites within individuals; moreover, differences among individuals often result in unbalanced designs (Albandar & Goldstein 1992).

Several cross-sectional and retrospective studies have identified other potential risk factors for periodontal attachment loss. While these have had methodological differences, they have consistently shown that smoking is a primary risk factor. The findings from NHANES III have shown that cigarette smoking, caries rate, and gender are independently associated with clinical attachment loss in 20–49-year-olds (Hyman & Reid 2003). Similar findings with respect to gender and smoking as risk factors for periodontal attachment loss have also been reported elsewhere (Beck et al. 1990; Norderyd & Hugoson 1998, Ogawa et al. 2002, Paulander et al. 2004), but have not always confirmed the association of gender with periodontal attachment loss (Norderyd & Hugoson 1998). To date, no longitudinal studies have examined the role of dental restorations and untreated carious lesions in periodontal attachment loss in adults, independent of other known risk factors. Therefore, this study aimed to determine the degree to which dental restorations and carious lesions are independent risk factors for future periodontal breakdown (while controlling for other periodontal disease risk factors).

Methods

The Dunedin Multidisciplinary Health and Development Study (DMHDS) is a longitudinal study of a birth cohort of children who were born at the Queen Mary Hospital, Dunedin, New Zealand, between 1 April 1972 and 31 March 1973 (Silva & Stanton 1996). 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, 26, and most recently at 32. Barriers to study members' participation were minimized by the Unit assuming the costs of participation (such as travel, lost wages, child care). Over 90% of the cohort self-identify as being New Zealand European.

The various assessments (e.g. oral health, mental health, physical health) are presented as standardized modules in counterbalanced order, and each is conducted by a different examiner who is kept blind to all study data. Periodic collections of health and developmental data (including dental examinations) have been undertaken as the study's inception, and the current study uses data collected from dental examinations at ages 26 and 32.

The Otago Ethics Committee granted ethics approval for each assessment phase. Study members gave informed consent before participating.

Dental health measures

Dental examination data from ages 26 and 32 years are included for analysis in the current study. Examinations for periodontal disease were not undertaken before the assessment at age 26 (apart from measurement using the CPITN index), and the age 26 periodontal assessment was half-mouth only. For this reason, the periodontal outcome variable was restricted to age 32, and only the age 26 and 32 data were used (Thomson et al. 2000, Thomson & Williams 2002).

At age 26 years, dental examinations were conducted by three examiners who had been previously calibrated and who examined 84.4%, 10.8%, and 4.8% of the study members,

respectively. At age 32, dental examinations were conducted by two calibrated examiners, who, respectively, examined 53.1% and 46.9% of the dentally assessed study members. The examiner who assessed 84.4% of the study members at age 26 was the same who assessed 46.9% at age 32. Information on how the examinations for dental caries were conducted is available in previous publications (Broadbent et al. 2006, Thomson & Locker 2000). Before the age-32 examination, clinical report forms were adjusted to account for teeth that had been recorded as missing at the age-26 assessments.

Periodontal measurements at the age-32 assessments were not conducted on individuals who reported a history of cardiac valvular anomalies or rheumatic fever. Three sites per tooth were examined (mesiobuccal, buccal, and distolingual). A recent study suggests that this partial recording protocol results in the smallest bias in estimating full-mouth measures (Susin et al. 2005). Probing depth (PD; the distance from the tip of the probe to the gingival margin) and gingival recession (the distance from the gingival margin to the cementoenamel junction) were recorded using a National Institute of Dental Research (NIDR) probe (manufactured by Hu-Friedy, Chicago, IL, USA, Product Number PCP2). This probe has alternating black and silver bands, with the black bands at 2–4, 6–8, and 10–12 mm. 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. Replicate periodontal examinations were not possible during the assessments because of time constraints (due to the busy assessment day undergone by study members). However, replicate examinations were conducted on a separate sample of 16 adults on four occasions during the Dunedin Study's age-32 data-collection phase, giving data for 1423 measured sites. Intraclass correlation coefficients for the periodontal measurements pooled for the two examiners (with the individual examiner coefficients in brackets) were 0.93 (0.94, 0.89) for mean GR, 0.68 (0.46, 0.83) for mean PD, and 0.69 (0.66, 0.86) for mean CAL. Of the 1423 replicated pairs of measurements, 99.6% were within ± 2 mm, meaning that only 0.4% of replicated pairs differed by 3+ mm.

Plaque accumulation was measured using the Simplified Oral Hygiene Index (Green & Vermillion 1964) at ages 5, 9, 15, 18, 26, and 32. For the purposes of creating a categorical variable to allow for controlling by long-term plaque, the longitudinal plaque scores were split into three distinct "plaque groups" using a censored normal group-based trajectory analysis model in the PROC TRAJ macro for SAS 9.1 (Jones et al. 2001, Nagin 2005). The mean OHI-S plaque scores for each of these groups were substantially different. The scores were as follows: group 1, low levels of plaque (group mean = 0.61, N = 370, 40.7%); group 2, moderate levels of plaque (group mean = 0.93, N = 438, 48.1%); and group 3, high levels of plaque (group mean = 1.42, N = 102, 11.2%).

Risk measures

In order to assess the change in tooth surface integrity between ages 26 and 32 as a risk factor, the mesial and distal surfaces of all teeth (with the exception of third molars) were separately coded as either sound, carious, or filled at age 26, and again at age 32. Based on the change in status between these two evaluation periods, each mesial surface was subsequently "dummy coded" into one of five categories to serve as a primary predictor variable: these categories were (age 26 years → 32 years), "sound → carious", "sound → restored", "carious → carious", "carious → restored", and "restored → restored". Surfaces that remained unaffected by caries or restorations by age 32 formed the reference category. Reversals were excluded from the analysis. Distal surfaces were coded in the same way, and were analysed separately. A restoration on one side of an inter-proximal space may affect the periodontal attachment levels on the other side of the inter-proximal space. Thus, mesial and distal surfaces were analysed separately in order to prevent overcomplicating the analytical model used for the analysis (with corresponding simplicity in interpretation).

Smoking status was determined based on the study members' self-reported smoking status at ages 21, 26, and 32. Smoking status was measured at ages 21, 26, and 32 years by asking the study members' current smoking status. Study members who reported smoking at all of ages 21, 26, and 32 years were classified as long-term smokers. Dental visiting pattern was classified in a similar way: study members who reported their dental visiting pattern as episodic at both ages 26 and 32 were classified as episodic users of dental care.

Dental flossing may independently affect an individual's inter-proximal periodontal attachment level. The dental flossing frequency of each participant was determined at age 32, and this frequency was categorized as "Never/Rarely" and "Sometimes/Every day".

Information on each study member's occupation (obtained during the age-26 interview) was used to obtain a measure of adult socioeconomic status (SES). Occupation was scaled into one of six categories based on the Elley–Irving index (Elley & Irving 1985), a commonly used New Zealand index of occupation-based social status. Study members were classified as growing up in families whose mean SES was low (groups 6 and 5 – e.g. oyster canner, car painter), medium (groups 4 and 3 – e.g. butcher, secretary), or high (groups 2 and 1 – e.g. architect, dentist) on the basis of the educational levels and income associated with that occupation in contemporary data from the New Zealand census (6 = unskilled labourer, 1 = professional).

Data analysis

The level of statistical significance was set at $p < 0.05$. In order to account for the nesting of tooth surfaces by mouth, multivariate analysis by generalized estimating equations was conducted using the STATA procedure XTGEE (Intercooled Stata 8.0, Stata Corporation, College Station, TX, USA, 2003), with exchangeable working matrices and robust standard errors. Two definitions of periodontal destruction, CAL≥3 and CAL≥4 mm, were used as outcome measures. In order to simplify the multilevel model used for the analysis, mesial and distal approximal sites were analysed separately. This meant that the sampling unit was people, but that the unit of analysis was the site.

Results

A total of 980 study members (96.2% of the surviving 1019) participated in the age-26 assessments; at age 32, 972 study members (95.8% of the surviving 1015) participated. Dental examination data were available for 930 and 932 individuals at ages 26 and 32, respectively, with dental examination data available for 901 at both ages (note that this figure excludes two completely edentulous study members). Approximately equal numbers of males and females were examined at both ages. Periodontal examination data were available for 915 individuals at age 32. The analysis was restricted to the 884 individuals who had been dentally examined at both ages 26 and 32, and who had also been periodontally examined at age 32 (Table 1).

A total of 40,633 inter-proximal surfaces were included in the analysis: 20,318 were mesial, and 20,315 were distal sites. The vast majority of inter-proximal surfaces were sound at both age-26 and age-32 assessments. However, a total of 1647 mesial and 1556 distal surfaces had been restored by age 32, while 238 mesial and 313 distal surfaces were carious at age 32 (Table 2). Of the 40,633 surfaces included in the analysis, 40,542 of these abutted an adjacent tooth. This could be interpreted as a total of 20,271 inter-proximal sites. At age 32, some 1067 (5.3%) of these sites were restored or carious on both the mesial and distal, 854 (4.2%) were carious or restored on only the mesial (with the adjacent distal surface being sound), and a further 769 (3.8%) were carious or restored on only the distal surface. The remainder of the carious and restored surfaces were in inter-proximal surfaces that did not abut an adjacent tooth.

An inter-proximal attachment loss of 3 mm or more was predicted by the restoration category of the site, whether newly carious at age 32, newly filled at age 32, carious since before age 26, carious since before age 26 but filled by age 32, or filled since before age 26. This finding held for both mesial and distal inter-proximal sites. The odds of attachment loss were significantly greater for sites that were newly carious or filled and ranged from 1.8 (1.1, 3.0) to 4.4 (2.2, 9.1). Other significant predictors of 3+ mm inter-proximal periodontal attachment loss included episodic dental attending, medium or low SES, long-term smoking, male gender, and moderate or poor long-term oral hygiene. Poor long-term oral hygiene and smoking were consistently the potential confounders independently related to attachment loss. Flossing showed a protective influence against the forming of inter-proximal periodontal pockets. Similar associations were observed for 4+ mm pockets, but those with some variables were not significant (Table 3).

An analysis of mesial inter-proximal periodontal attachment levels by all tooth-surface transitions (including occlusal surface transitions) was also conducted. This revealed that the mesial tooth-surface transitions and the occlusal-restoration-associated surface transitions were the only transitions to be associated with a mesial periodontal attachment loss of 3+ mm (Table 4). Mesial and distal restorations are, of course, “Class 2 restorations” and are normally associated with occlusal restorations. Thus, a separate bivariate analysis of the age-32 data for occlusal restorations on posterior teeth only (i.e., where no mesial, buccal, distal, or lingual restorations were also present on the tooth) was conducted. It revealed that of 10,037 posterior teeth with no occlusal restoration, 1237 (12.3%) had a mesial periodontal attachment loss of 3+mm, while, of 2066 teeth with occlusal restorations only, some 259 (12.5%) had a mesial periodontal attachment loss of 3+ mm ($\chi = 0.0710$, $p = 0.790$).

After adjusting for potential confounding variables, long-term carious sites were not significant predictors of 3+ mm of attachment loss. However, the remainder of the restoration-category associations with periodontal attachment loss of 3+ mm held after adjusting for potential confounding variables, although all but the association between newly carious mesial sites and CAL for the 4+ mm data lacked statistical significance (Table 5).

A bivariate analysis was also conducted to determine whether a surface identified as carious or restored facing another surface (whether sound, carious, or restored) could affect the prevalence of periodontal attachment loss on the other side of the periodontal space. The prevalence of periodontal attachment loss of 3+ and 4+ mm was the greatest where both the mesial and distal tooth surfaces were carious/restored, and was the least when both of these surfaces were sound. The prevalence of a mesial periodontal attachment loss of 3+ mm was greater where mesial restorations only had been placed than where distal restorations only had been placed, and the reverse was true for a distal periodontal attachment loss of 3+ mm. No such difference was found for the 4+ mm data (Table 6).

Discussion

The findings of this investigation support our hypothesis that approximal restorations are related to future periodontal breakdown. Our findings are consistent with those of Albandar et al. (1995) with respect to the role of defective and non-defective dental restorations on gingival inflammation and alveolar bone level in a population of adolescents. Both studies utilized multilevel statistical modelling; however, Albandar et al. (1995) did not control for other confounding variables, and the determination of CAL was accomplished using radiographic changes rather than clinical measures. It is possible that the measurement of the dependent variable (as well as differences in sample size) may explain the differences found between these studies. The ORs computed from *b* estimates reported by Albandar et al. (1995) were considerably smaller than those reported in the current study; however, it is unclear whether

the multi-level analyses modelled each 0.1 mm unit decrease in bone height or some other unit of measure.

There was no attempt to determine whether the relationship between CAL and restorations in the current investigation was attributable to the type of dental materials used, restoration contour/irregularities, or violation of the biologic width. Data on such details are not recorded in the Dunedin Study. However, previous studies suggest that a large proportion of approximal dental restorations (between 20% and 76%) have overhanging or poorly contoured margins, although estimates vary widely depending on both the methods for determining overhangs and the population studied (Brunsvold & Lane 1990, McDonald & Pack 1990, Albandar et al. 1995). Other studies have suggested that different dental materials may pose differential risks for periodontal CAL (Paolantonio et al. 2004). Researchers in future longitudinal studies may want to examine the effect over time of various dental materials on periodontal attachment loss.

The accuracy of periodontal attachment loss measurement has been shown to be dependent on the sites measured, the probe used, measurement error, and the operational definition of CAL (Breen et al. 1999, Jeffcoat & Reddy 2003). That measurements were made at only three sites per tooth (instead of six sites) may have led to some underestimation. While its magnitude is unknown, a recent study of the effect of partial recording protocols on estimates of periodontal disease prevalence found that the combination of the mesiobuccal, midbuccal, and distolingual sites was associated with the least bias when compared with estimates from the use of all six sites per tooth (Susin et al. 2005). This suggests that the use of these three sites in the current study may have minimized this partial-recording bias. Methodologically, measurement error was minimized in this study by use of a standard assessment technique and well-calibrated examiners. The NIDR periodontal probe is frequently used in large epidemiological studies of periodontal disease, and we are confident that its use in the current study has provided us with accurate measurements. We also opted to use two approaches to operationalizing periodontal attachment loss in order to validate the study's findings, and thus enhance their validity (Cook & Campbell 1979). Using two definitions of periodontal destruction, CAL≥3 mm and CAL≥4 mm, presents a realistic and conservative estimate of early CAL, as ± 2 mm error is an acceptable standard of error in clinical CAL assessment (Lopez et al. 2003, Thomson et al. 2004). Additionally, the use of full-mouth measures of posterior and anterior sites should produce stable estimates of OR (Thomson & Williams 2002).

Inherent in any periodontal study is the challenge of providing sufficient evidence to imply a causal relationship between risk factors and attachment loss. Two necessary criteria for demonstrating risk or a cause–effect relationship are (a) biologic plausibility and (b) an appropriate temporal relationship between exposure to risk factors and the occurrence of clinical attachment loss (Hennekens et al. 1987). Prospective cohort studies arguably provide the best evidence for identifying risk factors and quantifying the degree of risk (Albandar 2002). With regard to the temporal relationship, the current investigation captured changes in adjacent tooth surface integrity during that time. The data were unique in that full clinical datasets, obtained under standardized conditions, were available for 884 individuals across the study interval, along with life-long SES, dental utilization, and smoking-exposure-over-time measures.

Where biologic plausibility is concerned, that for CAL as a result of restorative treatment has been well established in previous literature (Kois 1996). This is further supported in that we found that only mesial restoration/caries transitions significantly affect the odds for mesial periodontal attachment loss. The fact that the odds for CAL were the greatest for periodontal sites adjacent to tooth surfaces that had permanent restorations at ages 26 and 32 –compared

with newly restored teeth and teeth with caries – provides additional evidence to support our hypothesis.

Implication

In general, dentists should consider inter-proximal caries and dental restorations to be local risk factors for localized periodontal attachment loss. Accordingly, they should take appropriate steps to minimize the occurrence of either, and to monitor carefully (and where necessary treat) inter-proximal sites that have (or require) adjacent restorations.

Clinical Relevance

Scientific rationale for study

Poorly contoured restorations can increase plaque retention and/or violate the biologic width; however, there is controversy about whether inter-proximal restorations predispose the adjacent periodontal tissues to future breakdown.

Principal findings

We found periodontal attachment loss to be approximately twice more likely to be ≥3 mm at sites corresponding to inter-proximal restorations or caries.

Practical implications

In general, dentists should consider inter-proximal caries and dental restorations to be local risk factors for periodontal attachment loss. Accordingly, dentists should take appropriate steps to minimize the occurrence of either, and to monitor carefully inter-proximal sites that have adjacent restorations.

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Table 1

Numbers of individuals in each examination, by gender (brackets contain percentages that use the number surviving at each age as the denominator)

	Males	Females	All
At baseline (age 3)	535	502	1037
At age 26 (1019 surviving)			
Dentally examined	472 (88.2)	458 (91.2)	930 (91.3)
Periodontally examined	465 (86.9)	449 (89.4)	914 (89.7)
At age 32 (1015 surviving):			
Dentally examined	476 (89.0)	456 (90.8)	932 (91.8)
Periodontally examined	469 (87.7)	446 ((88.8)	915 (90.1)
At both ages			
Dentally examined	459 (85.8)	442 (88.0)	901 (86.8)
Dentally examined at both ages, and periodontally examined at 32	452 (84.5)	432 (86.1)	884 (85.2)

Table 2

Number of surfaces in each transition category, by type of interproximal site

Age 26 → Age 32 transition	Site type	
	mesial	distal
Sound → Sound	18,433 (90.7)	18,436 (90.8)
Sound → Carious	160 (0.8)	189 (1.0)
Sound → Filled	356 (1.8)	431 (2.1)
Carious → Carious	78 (0.4)	134 (0.7)
Carious → Filled	166 (0.8)	123 (0.6)
Filled → Filled	1,125 (5.5)	1,002 (4.9)
Total	20,318	20,315

Table 3

Bivariate analyses of periodontal attachment loss outcomes for mesial and distal sites at age 32 by dental restoration status at ages 26 and 32, as well as bivariate analyses by periodontal outcomes by potential confounding variables

	Mesial surfaces				Distal surfaces			
	CAL≥3 mm OR (95% CI)	**number sites with CAL≥3 mm (%)**	**CAL≥4 mm OR (95% CI)**	**number sites with CAL≥4 mm (%)**	**CAL≥3 mm OR (95% CI)**	**number sites with CAL≥3 mm (%)**	**CAL≥4 mm OR (95% CI)**	**number sites with CAL≥4 mm (%)**
Restoration category (Age 26 → 32)								
Sound → Sound (reference)	1.0	1811 (10.0)	1.0	368 (2.1)	1.0	1362 (7.8)	1.0	394 (2.2)
Sound → Carious	1.8 (1.0, 3.0)	36 (25.5)	2.1 (0.6, 7.3)	13 (8.9)	1.7 (1.1, 2.8)	23 (17.2)	1.3 (0.4, 4.1)	10 (5.6)
Sound → Filled	1.8 (1.4, 2.3)	59 (16.6)	2.0 (1.3, 3.1)	15 (3.8)	2.3 (1.8, 2.9)	61 (18.7)	1.9 (1.2, 2.9)	17 (4.0)
Carious → Carious	1.9 (1.1, 3.3)	18 (26.1)	2.5 (0.7, 9.3)	6 (5.1)	1.2 (0.6, 2.2)	13 (19.2)	0.7 (0.2, 2.6)	3 (2.4)
Carious → Filled	2.5 (1.7, 3.7)	25 (22.5)	1.5 (0.7, 3.1)	4 (3.6)	2.2 (1.4, 3.5)	15 (13.9)	1.4 (0.5, 43.5)	4 (3.3)
Filled → Filled	1.8 (1.5, 2.1)	174 (15.7)	1.0 (0.7, 1.5)	26 (2.8)	2.3 (1.8, 2.8)	152 (14.7)	1.4 (1.0, 2.1)	32 (3.2)
Dental attendance at age 26								
Routine (reference)	1.0	723 (7.5)	1.0	133 (1.4)	1.0	566 (5.9)	1.0	118 (1.2)
Episodic	1.9 (1.5, 2.4)	1471 (13.6)	2.3 (1.4, 3.8)	346 (3.2)	2.0 (1.6, 2.6)	1216 (11.1)	2.8 (1.8, 4.3)	368 (3.4)
Socioeconomic status at age 26								
High (reference)	1.0	242 (6.0)	1.0	36 (0.9)	1.0	182 (4.5)	1.0	46 (1.1)
Medium	1.8 (1.3, 2.5)	1058 (10.0)	2.2 (1.0, 5.2)	205 (2.0)	1.9 (1.3, 2.7)	849 (8.0)	1.8 (0.8, 3.9)	213 (2.0)
Low	2.8 (1.9, 4.0)	767 (14.9)	4.2 (1.8, 9.9)	188 (3.7)	3.0 (2.0, 4.5)	642 (12.3)	3.2 (1.5, 6.8)	184 (3.5)
Smoked at all ages 21, 26, and 32								
Non-smoker (reference)	1.0	1227 (7.7)	1.0	192 (1.2)	1.0	940 (5.9)	1.0	225 (1.4)
Smoker	3.3 (2.6, 4.1)	1055 (21.5)	5.5 (3.6, 8.4)	310 (6.3)	3.5 (2.8, 4.4)	894 (17.9)	4.2 (2.7, 6.4)	277 (5.6)
Gender								
Female (reference)	1.0	952 (9.4)	1.0	225 (2.2)	1.0	735 (7.2)	1.0	221 (2.2)
Male	1.4 (1.1, 1.7)	1331 (12.5)	1.2 (0.8, 1.8)	277 (2.6)	1.5 (1.1, 1.9)	1100 (10.2)	1.2 (0.8, 1.9)	281 (2.6)
Long-term oral hygiene category								
Good (reference)	1.0	533 (6.3)	1.0	92 (1.1)	1.0	444 (5.3)	1.0	99 (1.2)
Moderate	1.8 (1.4, 2.3)	1114 (11.5)	1.8 (1.1, 3.1)	206 (2.1)	1.7 (1.3, 2.2)	880 (9.0)	1.7 (1.1, 2.9)	210 (2.1)
Poor	5.1 (3.7, 7.0)	573 (26.0)	8.3 (4.7, 14.5)	190 (9.6)	4.7 (3.3, 6.6)	463 (20.7)	6.8 (3.7, 12.6)	174 (7.8)
Flossing at age 32								
Never flosses (reference)	1.0	1466 (12.7)	1.0	333 (2.9)	1.0	1181 (10.1)	1.0	310 (2.7)
Flosses at least occasionally	0.7 (0.5, 0.8)	811 (8.8)	0.6 (0.4, 1.0)	169 (1.8)	0.7 (0.5, 0.9)	652 (7.1)	0.8 (0.5, 1.2)	191 (2.1)
Overall*		2283 (11.0)		502 (2.4)		1835 (8.8)		502 (2.4)

*Columns may not add up perfectly due to a small amount of missing data for some items.

CAL, clinical attachment level; OR, odds ratio.

Table 4

Odds ratios (OR), 95% confidence intervals, and predicted % for the prevalence of a periodontal attachment loss of 3 + mm CAL at mesial sites by all surface transitions*

Surface	Transition category	Odds ratio	95% Confidence interval	Predicted % with 3 + mm CAL
Mesial	Sound → Sound (reference)	1.0		11.7
	Sound → Carious	1.8	1.1, 2.9	20.9
	Sound → Filled	1.4	1.0, 1.9	16.8
	Carious → Carious	1.0	0.3, 3.1	11.6
	Carious → Filled	3.4	2.2, 5.1	33.4
	Filled → Filled	1.5	1.2, 1.8	17.7
Buccal	Sound → Sound (reference)	1.0		12.4
	Sound → Carious	0.8	0.5, 1.5	13.0
	Sound → Filled	1.2	0.8, 1.6	17.1
	Carious → Carious	1.1	0.6, 2.1	14.9
	Carious → Filled	1.2	0.7, 2.3	15.7
	Filled → Filled	0.8	0.6, 1.0	12.4
Distal	Sound → Sound (reference)	1.0		12.4
	Sound → Carious	0.9	0.6, 1.5	13.7
	Sound → Filled	0.8	0.6, 1.1	12.6
	Carious → Carious	1.4	0.8, 2.5	17.0
	Carious → Filled	0.6	0.3, 1.2	10.2
	Filled → Filled	0.8	0.7, 1.1	13.9
Lingual	Sound → Sound (reference)	1.0		12.3
	Sound → Carious	1.2	0.6, 2.3	18.7
	Sound → Filled	1.2	0.7, 2.0	17.9
	Carious → Carious	0.3	0.0, 3.9	5.1
	Carious → Filled	1.8	0.7, 4.3	22.7
	Filled → Filled	1.3	1.0, 1.7	18.5
Occlusal	Sound → Sound (reference)	1.0		11.2
	Sound → Carious	1.3	0.8, 2.0	15.9
	Sound → Filled	1.1	0.8, 1.4	12.4
	Carious → Carious	1.2	0.6, 2.4	13.9
	Carious → Filled	1.4	1.1, 1.9	17.5
	Filled → Filled	1.2	1.1, 1.4	14.6

* *N* for the full model (mesials of posterior teeth only) = 827 individuals and 12,103 observations. CAL, clinical attachment level.

Table 5

Odds ratios (OR) and 95% CI of periodontal attachment loss at age 32 for mesial and distal proximal surfaces after adjusting for potential confounding variables

	Mesial surfaces*		Distal surfaces†	
	CAL≥3 mm OR (95% CI)	CAL≥4 mm OR (95% CI)	CAL≥3 mm OR (95% CI)	CAL≥4 mm OR (95% CI)
Restoration category (Age 26 → 32)				
Sound → Sound (reference)	1.0	1.0	1.0	1.0
Sound → Carious	1.8 (1.0, 3.1)	2.1 (0.7, 5.7)	1.8 (1.3, 2.6)	1.2 (0.5, 2.7)
Sound → Filled	1.9 (1.4, 2.4)	1.8 (1.0, 3.1)	2.5 (1.9, 3.3)	2.0 (1.3, 3.2)
Carious → Carious	1.6 (1.0, 2.8)	1.7 (0.6, 4.8)	1.3 (0.8, 2.0)	0.8 (0.4, 1.7)
Carious → Filled	2.7 (1.8, 4.1)	1.5 (0.7, 3.1)	2.1 (1.3, 3.5)	1.1 (0.4, 3.1)
Filled → Filled	1.9 (1.6, 2.3)	1.0 (0.7, 1.6)	2.5 (2.0, 3.1)	1.5 (1.0, 2.4)

* *N* for full model (mesials) = 827 individuals and 18,827 observations

† *N* for full model (distals) = 827 individuals and 18,960 observations

‡ The model controlled for the gender, oral hygiene, dental visiting, SES, smoking, and flossing measures.

CAL, clinical attachment level; CI, confidence interval.

Table 6

Bivariate analysis using interproximal site as the unit of analysis to demonstrate periodontal attachment loss on either side of the papilla by the presence of mesial and/or distal tooth surface caries/restoration

Caries/restoration		Number of sites with 3+ mm CAL (%)		Number of sites with 4+ mm CAL (%)	
distal	mesial	mesial	distal	mesial	distal
No	No	1729 (10.0)	1325 (7.7)	377 (2.2)	378 (2.2)
No	Yes	120 (16.4)	112 (14.8)	23 (3.2)	26 (3.4)
Yes	No	114 (13.6)	125 (15.2)	25 (3.0)	28 (3.4)
Yes	Yes	197 (18.8)	175 (17.0)	46 (4.4)	41 (3.9)

CAL, clinical attachment level.