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Cigarette smoking and periodontal disease among 32-year-olds: a prospective study of a representative birth cohort

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Abstract

Background—Smoking is recognized as the primary behavioural risk factor for periodontal attachment loss (AL), but confirmatory data from prospective cohort studies are scarce.

Aim—To quantify the association between cigarette smoking patterns and AL by age 32.

Methods—Periodontal examinations were conducted at ages 26 and 32 in a longstanding prospective study of a birth cohort born in Dunedin (New Zealand) in 1972/1973. Longitudinal categorization of smoking exposure was undertaken using data collected at ages 15, 18, 21, 26 and 32.

Results—Complete data were available for 810 individuals of whom 48.9% had ever smoked (31.5% were current smokers). Compared with never-smokers, long-term smokers (and other age-32 smokers) had very high odds ratios (ORs of 7.1 and 5.7, respectively) for having 1 +sites with 5 +mm AL, and were more likely to be incident cases after age 26 (ORs of 5.2 and 3.2, respectively). Two-thirds of new cases after age 26 were attributable to smoking. There were no significant differences in periodontal health between never-smokers and those who had quit smoking after age 26.

Conclusions—Current and long-term smoking in young adults is detrimental to periodontal health, but smoking cessation may be associated with a relatively rapid improvement in the periodontium.

Keywords

cohort study; periodontal disease; smoking; tobacco

Second only to dental caries as a cause of tooth loss, periodontitis (commonly known as “gum disease”) has the added characteristic of an accumulating body of epidemiological and clinical evidence (largely circumstantial) for its involvement in systemic conditions such as cardiovascular disease, stroke, pulmonary disease and adverse pregnancy outcomes (Pihlstrom et al. 2005). It is now well-established that smoking is the primary behavioural risk factor for periodontitis (Gelskey 1999, Johnson & Hill 2004), but the scarcity of information on the

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condition's natural history and risk factors among adult populations (particularly younger adults) compromises current understanding of the nature of smoking's role as a risk factor for periodontal attachment loss (AL) through what is arguably a key period in the condition's development. Bergstrom et al. (2000) recently highlighted the need for population-based longitudinal data on the relationship, not only to determine the effects of continued smoking but also to quantify the positive effects of smoking cessation, for which there is some evidence from cross-sectional or clinical studies (Bergstrom 2003, Johnson & Hill 2004), but not from longitudinal studies.

We recently reported on the occurrence of AL from ages 26 to 32 among participants in a longstanding prospective study of a birth cohort of 1037 persons (Thomson et al. 2006). An earlier report (Hashim et al. 2001) from that study described a strong, "dose-dependent" association between adult periodontitis and smoking from late adolescence, with chronic smokers at ages 15, 18, 21 and 26 being almost three times more likely than never-smokers to have established periodontitis by age 26. Despite the strength of the evidence from this and other studies, a number of unanswered research questions about that association remain, not least among these being the nature of the periodontal benefits of smoking cessation. The aims of this study were to describe and quantify the association between long-term smoking and periodontitis by age 32, and to determine the effects of recent smoking cessation on age-32 periodontal status.

Material and 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 (NZ) between 1 April 1972 and 31 March 1973 (Silva & Stanton 1996). The sample that formed the basis for the longitudinal study was 1037 children; they were assessed within a month of their third birthdays. Periodic collections of health and developmental data (including dental examinations) have been undertaken since then, and this study uses data collected from assessments conducted at ages 15, 18, 21, 26 and 32. Over 90% of the cohort self-identified as being of European origin. Ethical approval for the study was obtained from the Otago Ethics Committee, and informed consent was obtained from each participant.

Dental examinations conducted at age 26 included periodontal measurements made in two quadrants (quadrants 1 and 3 for study members whose study ID number was odd; quadrants 2 and 4 for those with an even ID number; the mix of odd and even ID numbers was approximately 50:50) because of time constraints. Three sites (mesiobuccal, buccal and distolingual) per tooth were examined, and gingival recession (GR; the distance in millimetres from the cemento-enamel junction, or CEJ, to the gingival margin) and probing depth (PD; the distance from the tip of the probe to the gingival margin) were recorded, using an NIDR probe (which has a rounded 1 mm tip and six alternating 2 mm bands). Where the gingival margin was situated more than 1 mm coronally to the CEJ, a negative value for GR was recorded. Periodontal measurements were not conducted on those reporting a history of cardiac valvular anomalies or rheumatic fever. The combined AL for each site was computed by summing the measurements for gingival recession and adjusted PD (third molars were not included). The dental examinations were repeated 6 years later at age 32. The clinical procedures were identical, except that a full-mouth examination was now possible. Because of the high prevalence of negative GR measures in this cohort (Thomson et al. 2006) – indicating gingival enlargement, known previously as "gingival hyperplasia" – we adjusted the measures for PD by subtracting the distance from the free gingival margin to the point 1 mm coronal to the CEJ. In this analysis, we report only the adjusted PD, as using the unadjusted measures was found in an earlier analysis (Thomson et al. 2006) to overestimate PD.

Two calibrated examiners were used. Replicate periodontal examinations were not possible because of time constraints (due to the busy assessment day undergone by participants, who spend an entire day being assessed in groups of four, with the dental assessment taking place last). However, replicate examinations were conducted on a separate sample of 16 adults on four occasions during the age-32 data-collection phase, giving data for 1423 measured sites. Intra-class 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. The κ value for the prevalence of 1+ sites with 4+ mm CAL was 0.5 (0.7, 0.8). Of the 1423 replicated pairs of measurements, 99.6% were within ± 2 mm (only 0.4% of pairs differed by 3+ mm). Thus, 2+ mm was chosen as the minimum threshold representing true change (a low probability of being due to examiner error) for PD and CAL in this study.

Periodontitis case definitions

Periodontitis prevalence was determined (at two levels of severity) by identifying individuals with 1 or more sites with 4+ mm AL or 1 or more sites with 5+ mm AL. For changes in periodontal attachment over time (based upon half-mouth data), a site that had been examined (and found to be non-diseased at age 26), which increased at least 2 mm and resulted in a periodontal pocket of at least 4 mm, was classified as having *incident* disease, while a site showing *progression* was defined as one with AL of 4+ mm at age 26 that had increased by at least 2 mm by age 32. An "incident case" was an individual with one or more sites experiencing incident disease or progression.

Measurement of smoking exposure

At age 15, smoking was determined with the question "Have you smoked in the last 4 weeks?". At age 18, we used "Have you been smoking every day for the last month?". At ages 21, 26 and 32, we used "Have you smoked every day for 1 month or more of the previous 12 months?". The number of pack-years exposure (i.e., the number of packs of cigarettes smoked per day multiplied by the number of years smoked at that rate) was computed based on the following questions about the number of cigarettes smoked at ages 18, 21, 26 and 32:

- **a.** Have you ever smoked for as long as a year? ("No" means <20 packs of cigarettes in your lifetime or less than one cigarette/day for as long as a year).
- **b.** How old were you when you started smoking regularly?
- **c.** Have you cut down or stopped smoking?
- **d.** How long ago did you cut down or stop smoking?
- **e.** How many cigarettes per day did you smoke (on average) before the change?
- **f.** How many cigarettes per day do you now smoke on average?

Cigarette consumption was then calculated for the following periods: up to age 18, 18–21, 21–26 and 26–32. If data were not collected from a participant at an assessment, his/her responses to the same questions at the next assessment were used, and calculations made retrospectively. Those who had not smoked during the period were taken to have smoked zero cigarettes; those who had were taken to have smoked the sum of their current consumption rate multiplied by the number of years during the period they had smoked at that rate, and, if applicable, their consumption rate before they cut down multiplied by the number of years during the period they had smoked at the previous rate. This provided the number of cigarettes smoked per day multiplied by the number of years of smoking, and this estimate was divided by 20 to give the number of pack years.

The population attributable risk (PAR) from smoking between ages 26 and 32 was calculated for the incidence of periodontal disease using two incident-case definitions: for the first, the incidence of new cases of 4+ mm AL between ages 26 and 32 was used; the second used the incidence of new cases of 5+ mm AL between ages 26 and 32. For each, we identified those individuals who were not cases at 26 but were cases at age 32. The PAR (expressed as a percentage) was computed as the number exposed multiplied by the difference between incidence rates in those exposed and not exposed, divided by the total number in the sample multiplied by their incidence rate.

Covariates

Each participant's adult socioeconomic status (SES) was measured at age 26 by categorizing his/her adult occupation using standard NZ occupationally based indices (Irving & Elley 1977, Elley & Irving 1985), which use a six-interval classification (where, e.g., a doctor scores "1" and a labourer scores "6"). Those with a score of "5" or "6" were categorized as low SES. To determine their usual dental utilization pattern, participants were asked (at 26 and 32) whether they usually visited the dentist for a check-up or because of a problem. Those who gave the latter response at both ages were designated "episodic users" of dental care. Dental plaque accumulation at age 32 was measured using the Simplified Oral Hygiene Index (Greene & Vermillion 1964).

Data analysis

Chi-square tests were used to examine the statistical significance of differences observed with categorical dependent variables. Analysis of variance was used for continuous variables. Logistic regression modelling was used to examine smoking exposure and periodontitis prevalence and incidence while controlling for sex, SES, dental plaque accumulation and the use of dental services. Those covariates were chosen because either they had been found in earlier studies to be confounders of the smoking–periodontitis relationship, or they were found to be associated with the dependent variables in the bivariate analyses.

Results

At age 26, 1019 (98.3%) of the 1037 original participants were alive, and 980 (96.2% of the surviving cohort) participated in the assessments. Dental examination data at age 26 were available for 930 individuals; 914 (98.3%) of those were periodontally examined, one refused and 15 (1.6%) were not examined because of a medical contraindication to periodontal probing. Of those for whom periodontal data were available, there were approximately equal numbers of males and females. At age 32, periodontal examination data were available for 915 individuals, of whom 882 (96.4%) were examined at both ages. Of those, smoking history information from ages 15 to 32 was available for 810 (88.5%). All subsequent analyses are limited to those 810 (50.7% male). There were no significant differences by gender or by any of the periodontal measures between those included in the study and the 105 who were excluded because of incomplete smoking data (data available on request).

Overall, 414 participants (51.1%) had never smoked. Of the remaining 396 "ever-smokers" (48.9%), 95 (24.0%) were smokers at each of ages 15, 18, 21, 26 and 32 (their mean pack-years exposure was 13.9, SD = 5.3); 160 (40.4%) were smokers at age 32 but not at all of those ages (10.5 pack-years, SD = 5.5); 69 (17.4%) had given up smoking after age 26 (6.8 pack-years, SD = 4.1); and 72 (28.1%) had given up earlier than age 26 (2.2 pack-years, SD = 2.6). Overall, smoking prevalence increased steadily from age 15 to 26 (being 25.8% at age 15, 28.4% at 18, 36.7% at 21 and 38.3% at 26), after which a decline to 31.5% was observed by age 32. There was considerable intra-individual fluctuation over the observation period (data available upon request).

Summary data on age-32 AL are presented by smoking exposure category and other characteristics in Table 1. Overall, more than one in four met the 4+ mm AL case definition, and one in eight met the 5+ mm AL case definition. Just over one in eight had 1+ sites with a 3+ mm AL increase. The periodontal disease gradients across the smoking exposure categories were largely consistent, with the prevalence and extent of AL being greatest among the long-term smokers and other age-32 smokers, lower among the ex-smokers, and lowest among the never-smokers (similar gradients were observed with respect to mean CAL and mean PD; data not presented here). A similar pattern was evident with the incidence of AL between ages 26 and 32 (experienced by almost one in 12 individuals). Those who had given up smoking after age 26 had periodontal disease experience, which was very close to that of the never-smokers, while those who had given up before age 26 showed a similar pattern. There were differences in the prevalence and incidence of AL by gender, SES, dental visiting pattern and plaque score.

AL prevalence at age 32 was modelled using logistic regression (Table 2). Compared with never-smokers, long-term smokers (and other age-32 smokers) had high odds of being a case (and those odds were considerably higher with the more stringent case definition of 5+ mm AL), and they were more likely to have become new cases after age 26. By contrast, those who had given up smoking after age 26 were not significantly different from the reference category (never-smokers) in any of the models. While the other ex-smokers also did not differ statistically from the reference category for two of the models, they did have higher odds (than the more recent quitters) for 1+ sites with 5+ mm AL at age 32.

The PAR for new cases of 4+ mm AL was 34.2%, while that for the more stringent case definition of 5+ mm AL was 67.1% (meaning that two-thirds of the new cases between ages 26 and 32 were attributable to smoking). When only smoking since age 21 was considered, those estimates were 33.8% and 63.8%, respectively.

Discussion

This prospective cohort study investigation strongly supports the role of long-term smoking as a risk factor for periodontitis in adulthood. It has also provided the first epidemiological confirmation that smoking cessation after the mid-20s may be associated with an improvement in the periodontal tissues which is detectable by the early 30s.

Before discussing the findings in detail, it is appropriate to consider the study's weaknesses and strengths. Principal among the former are the use of self-reported smoking status data and the partial-mouth periodontal examination data. First, it is possible that the prevalence of smoking at each age was underreported (particularly at ages 15 and 18) because of the reliance on self-reported smoking exposure data. However, a systematic review of the literature found generally high levels of concordance between self-report and biological measures of smoking exposure, particularly where (as in the current study) data are collected prospectively and interviewer-administered questionnaires are used (Patrick et al. 1994). This has been supported by more recent studies (Dolcini et al. 2003, Fendrich et al. 2005, Mak et al. 2005). The recency of the self-report reference period is an important issue, with shorter periods leading to greater validity (Dolcini et al. 2003); in the current study, the reference period at ages 15 and 18 was the previous 4 weeks, while the previous year was used for ages 21, 26 and 32. Given these considerations, and the participants' life-long exposure to questionnaires through their ongoing involvement in the Dunedin study, it is probable that the validity of self-reported smoking status was acceptable in the current study. Furthermore, if smoking prevalence was under-reported, it would tend to strengthen our findings because such underreporting would have resulted in more conservative estimates of the association with periodontal disease. Second, time constraints during the oral examination at age 26 meant that only half-mouth periodontal examinations were possible (this was rectified at age 32), meaning that, while the age-32

prevalence estimates were based on up to 28 teeth, the measure of age 26–32 disease incidence (in turn) was based on half-mouth data. Accordingly, it is likely that the longitudinal data presented here are underestimates of the true incidence of disease. The fact that measurements were made at only three sites per tooth (instead of six sites) will also have led to some underestimation of both prevalence and incidence, but its magnitude is unknown. However, a recent study of the effect of partial recording protocols on estimates of periodontal disease prevalence found that the three-site combination used in the current study was associated with the least bias when compared with estimates from the use of all six sites per tooth (Susin et al. 2005), although it was still more than would have been from random partial-mouth recording (Beck et al. 2006). Third, in controlling for the use of dental services by identifying those who were episodic users at 26 and 32, we assumed that they were episodic users in the intervening years and, as such, had no access to any professionally provided periodontal care. This is a reasonable assumption, but it is by no means a certainty. Moreover, we were unable to determine whether any participant had periodontal surgery.

The analytical approach also merits examination. The fundamental issue is the exposures that have led to the disease observed at age 32. Our approach to operationalizing those exposures has been to use a five-category classification of cumulative smoking exposure to that point ("continuous" smokers, other current smokers, those who gave up after 26, other ex-smokers and never-smokers). This admittedly "broad-brush" categorization captures the time dependence of the exposure to smoking, but it can be argued that a more rigorous modelling approach should have been used, which allowed the inclusion of time-dependent covariates (such as a generalized estimating equation model, but we have only two observation points for the dependent variable). However, if it is accepted that the most important dependent variable here is indeed periodontitis case status at age 32, then some form of logistic regression analysis is appropriate, and the covariates can be treated as fixed (rather than time-dependent) covariates, because they occurred before age 32.

Among the study's strengths are the follow-up rate, the approach to determining smoking exposure (and the length of time over which those exposure data were collected), and the use of a dynamic (rather than static) disease measure. At 96% after three decades, the follow-up rate is exceptional. The generalizability of the findings to the source population has been established, and we have addressed the issue of the degree to which they can be generalized to similar populations in both NZ and the United States in an earlier report (Thomson et al. 2006). Finally, the use of not only two cumulative disease measures (prevalence and extent) at age 32 but also a dynamic measure of disease incidence enabled a robust examination of the smoking-periodontitis association (as well as allowing calculation of the PAR).

Our findings on the periodontal consequences of long-term smoking build on earlier work with this cohort (Hashim et al. 2001). Recent reviews have underlined that a lack of data from population-based prospective cohort studies with high follow-up rates has meant that knowledge of the smoking–periodontitis relationship was incomplete (Borrell & Papapanou 2005, Bergstrom 2006). Our findings should assist by highlighting the oral health detriment that accrues from continuing to smoke from adolescence into adulthood: those who smoked at every assessed age had five times the odds of being incident cases between ages 26 and 32, and approximately two-thirds of those incident cases were due to smoking. Moreover, the biological gradient was as expected.

Where the study provides potentially interesting new evidence, however, is on the periodontal benefits of smoking cessation. Disease prevalence at age 32 among the group who had ceased smoking after age 26 was almost as low as among those who had never smoked, suggesting that some periodontal improvement (attachment gain) had occurred. If so, it would most likely manifest as reductions in PD rather than as gingival recession. However, such an assertion

should be supported by data showing evidence of that gain between ages 26 and 32. We were unable to find any evidence of this (Table 3), with the exposure group in question showing neither greater prevalence (nor greater extent) of PD gain than the others. In fact, when the age-26 (baseline) periodontal disease experience of the various smoking exposure groups is examined (Table 3), those who ceased smoking after age 26 already had considerably lower AL prevalence and extent. This raises the question of whether they had, in fact, been progressively reducing their smoking in the years before giving up (which would be consistent with the personal experiences of two of the authors, who are ex-smokers). There is some evidence for this in Table 3, with that group having similar (if slightly fewer) pack-years exposure (on average) up to age 21, but less exposure over the period from ages 21 to 26. While the difference is not overly substantial, it does offer some support for the "tapering" hypothesis. The absence of periodontal examinations during the age-21 Dunedin study assessments is unfortunate. In the final analysis, however, the data in the final column of Table 2 are the crux of the matter: the incidence of periodontal AL from ages 26 to 32 among those who had ceased smoking after age 26 (and also among the other former smokers) was not significantly different from that observed among never smokers. Clearly, smoking cessation has periodontal benefits; the issue of their timing relative to that of cessation is a secondary one. How might these benefits come about? Current understanding of the nature of periodontal disease is that it is a dynamic phenomenon, with cyclical patterns of progression and resolution (Gilthorpe et al. 2003) at any given site. Smoking is thought to tip the balance towards progression by impairing the immune response and compromising the periodontal tissue's ability to heal following a period of disease activity (Johnson & Hill 2004). Thus, smoking cessation would (*ceteris paribus*) be expected to favour resolution over progression at a given site.

In summary, long-term (and current) smoking is detrimental to periodontal health, and quitters are likely to reap not only the direct periodontal benefits –and the direct general health benefits of smoking cessation (Wen et al. 2005) –but also the indirect general health benefits of the associated decrease in periodontal risk. The latter is, of course, conditional upon epidemiological validation of the accumulating body of evidence implicating periodontal disease as a contributory factor for a number of systemic health conditions (Beck & Offenbacher 2001, Iso et al. 2005, Pihlstrom et al. 2005).

Clinical Relevance

Scientific rationale for the study

While cigarette smoking is recognized as the primary behavioural risk factor for periodontitis, information on the association among younger adults is scarce, and there is a lack of population-based data on the putative periodontal benefits of smoking cessation.

Principal findings

We found that those who had smoked since their mid-teens were more than seven times as likely (as those who had never smoked) to have established periodontitis. The periodontal health of people who had given up smoking in the previous 6 years was very similar to that of never smokers.

Practical implications

It appears that smoking cessation may confer periodontal benefits, which accrue relatively rapidly. Dentists should be at the forefront of efforts to identify and counsel smokers.

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Table 1

Periodontal status at age 32 by summary smoking exposure category, gender, socioeconomic status (SES) and use of dental services

	No (%)	Disease measures at age 32			Incidence from ages 26 to 32
		prevalence of AL		extent of AL	
		no with 1+ sites with 4+ mm (%)	no with 1+ sites with 5+ mm (%)	mean % of sites with 4+ mm AL (SD)	no with 1+ sites with 3+ mm AL increase (%)
Smoking exposure group					
Smoked at all ages	95 (11.7)	53 (55.8)	28 (29.5)	6.1 (11.2)	27 (28.4)
Other age-32 smokers	160 (19.8)	67 (41.9)	39 (24.4)	3.8 (8.3)	32 (20.0)
Gave up smoking after 26	69 (8.5)	17 (24.6)	4 (5.8)	1.4 (4.8)	7 (10.1)
Other ex-smokers	72 (28.1)	16 (22.2)	8 (11.1)	1.5 (6.1)	6 (8.3)
Never smoked	414 (51.1)	73 (17.6)*	18 (4.3)*	0.6 (2.2)*	31 (7.5)*
Gender					
Female	399 (49.3)	93 (23.3)*	41 (10.3)	1.9 (6.2)	48 (12.0)
Male	411 (50.7)	133 (32.4)	56 (13.6)	2.1 (6.3)	55 (13.4)
Socioeconomic status at 26†					
High	244 (31.3)	55 (22.5)*	30 (12.3)	1.7 (5.6)	25 (10.2)
Medium	397 (50.9)	114 (28.7)	47 (11.8)	1.8 (5.4)	45 (11.3)
Low	139 (17.8)	47 (33.8)	17 (12.2)	2.8 (8.5)	25 (18.0)
Dental visiting pattern					
Episodic at both 26 and 32	312 (38.5)	120 (38.5)*	62 (19.9)*	3.4 (8.3)*	47 (15.1)
Other	498 (61.5)	106 (21.3)	35 (7.0)	1.1 (4.3)	56 (11.2)
Plaque score at age 32					
Below median	461 (56.9)	104 (22.6)*	46 (10.0)*	1.4 (5.0)*	57 (12.4)
Above median	349 (43.1)	122 (35.0)	51 (14.6)	2.8 (7.5)	46 (13.2)
All groups combined	810 (100.0)	226 (27.9)	97 (12.0)	2.0 (6.3)	103 (12.7)

*$p<0.001$.

†30 individuals with unclassifiable SES at age 26.

AL, attachment loss.

Table 2

Logistic regression models for the prevalence and incidence of attachment loss (AL) at age 32 (reference category for smoking variables = never smokers)

	Prevalence				Incidence	
	1+ sites with 4+ mm AL*		1+ sites with 5+ mm AL†		1+ sites with 3+ mm AL increase‡	
	OR	95% CI	OR	95% CI	OR	95% CI
Female§	0.66	0.47, 0.93	0.77	0.48, 1.24	0.85	0.54, 1.32
Low SES at 26	1.17	0.76, 1.80	0.77	0.42, 1.43	1.41	0.84, 2.37
Plaque score at 32¶	1.64	1.19, 2.25	1.21	0.80, 1.83	1.06	0.70, 1.58
Episodic user of dentistry at 26 and 32	1.30	0.91, 1.85	1.71	1.04, 2.82	0.81	0.50, 1.30
Long-term smoker (from age 15 to 32)	5.01	2.97, 8.45	7.13	3.53, 14.38	5.16	2.73, 9.76
Other age-32 smokers	2.75	1.98, 4.20	5.68	3.06, 10.54	3.20	1.83, 5.58
Gave up smoking after age 26	1.59	0.86, 2.95	1.32	0.43, 4.05	1.47	0.62, 3.50
Other ex-smokers	1.50	0.80, 2.79	2.75	1.14, 6.68	1.21	0.48, 3.03

*Nagelkerke R^2 = 0.161; Hosmer & Lemeshow test p = 0.771.

†Nagelkerke R^2 = 0.182; Hosmer & Lemeshow test p = 0.692.

‡Nagelkerke R^2 = 0.087; Hosmer & Lemeshow test p = 0.706.

§Reference categories: female (male, coded 0), Low SES at 26 (higher SES, coded 0); episodic dental user at 26 and 32 (routine dental visitor, coded 0); smoking exposure variables (never smoker; coded 0).

¶This is a continuous variable representing the extent of plaque on six index teeth (range 0–3).

CI, confidence interval; OR, odds ratio; SES, socioeconomic status.

Table 3

Probing depth gains between ages 26 and 32, periodontal status at age 26, and earlier smoking exposure, by summary smoking exposure category

	Smoking exposure group					All combined
	smoked at all ages	**other age-32 smokers**	**gave up after 26**	**other ex-smokers**	**never smoked**	
Probing depth gains between ages 26 and 32						
Number showing PD gain of 2+ mm (%)	52 (54.7)	63 (39.4)	30 (43.5)	41 (56.9)	195 (47.1)	381 (47.0)
Number showing PD gain of 3+ mm (%)	9 (9.5)	14 (8.8)	2 (2.9)	2 (2.8)	17 (4.1)	44 (5.4)*
Mean % of sites showing PD gain of 2+ mm (SD)	3.2 (4.8)	2.0 (3.4)	1.7 (2.3)	2.9 (3.6)	2.4 (4.2)	2.4 (4.0)
Mean % of sites showing PD gain of 3+ mm (SD)	0.4 (2.0)	0.2 (0.8)	0.1 (0.4)	0.1 (0.4)	0.2 (1.5)	0.2 (1.3)
Periodontal disease measures at age 26						
No. with 1+ sites with 4+ mm AL	33 (34.7)	35 (21.9)	9 (13.0)	14 (19.4)	56 (13.5)*	147 (18.1)
No. with 1+ sites with 5+ mm AL	8 (8.4)	7 (4.4)	1 (1.4)	4 (5.6)	8 (1.9)*	28 (3.5)
Mean % of 1+ sites with 4+ mm AL (SD)	2.5 (5.2)	1.4 (4.1)	0.4 (4.8)	1.0 (2.5)	0.7 (2.6)*	1.0 (3.3)
Earlier smoking exposure						
Mean pack-years (SD) up to age 21	5.2 (2.3)	2.8 (2.3)	2.6 (2.2)	1.3 (1.6)	0.0 (0.0)†	1.5 (2.3)
Mean pack-years (SD) from age 21–26	4.1 (1.7)	3.3 (1.9)	2.6 (2.2)	0.6 (0.9)	0.0 (0.0)‡	1.4 (2.0)

*$p<0.05$.

†$p<0.05$; oneway ANOVA: those who gave up smoking after age 26 differ from all other groups except for the other age-32 smokers.

‡$p<0.05$; oneway ANOVA: those who gave up smoking after age 26 differ from all other groups.

PD, probing depth.