J Clin Periodontol 2005; 32: 81–88, doi: 10.1111/j.1600-051X.2004.00638.x

Journal of Clinical Periodontology

Tobacco smoking and subgingival dental calculus

Jan Bergström

Institute of Odontology, Karolinska Institutet, Stockholm, Sweden

Bergström J: Tobacco smoking and subgingival dental calculus. J Clin Periodontol 2005; 32: 81–88. doi: 10.1111/j.1600-051X.2004.00638.x. © Blackwell Munksgaard, 2004.

Abstract

Objective: A radiographic investigation into the relationship between tobacco smoking and subgingival dental calculus was conducted in an adult population, including 48 current smokers, 57 former smokers, and 125 non-smokers.

Material and Methods: Assessment of subgingival calculus was based on a full set of radiographs. Mesial and distal root surfaces were assessed as to presence or absence of radiopaque deposits apical to the cemento-enamel junction. The severity of subgingival calculus deposition, labeled subgingival calculus load, was estimated from both the total number and the proportion of proximal sites affected.

Results: The overall prevalence of individuals exhibiting at least one subgingival calculus positive site was 43%, ranging from 15% in age stratum 20–34 years to 72% in age stratum 50–69 years. The prevalence among current smokers, former smokers, and non-smokers was 71%, 53%, and 28%, respectively. The differences between smoking groups were statistically significant ($p < 0.001$). The mean subgingival calculus load of current smokers, former smokers, and non-smokers was 3.4, 1.2, and 0.6 affected sites per person, respectively, or expressed as mean proportions, 6.2%, 2.4%, and 1.1%, respectively. The association between smoking and subgingival calculus load was statistically significant ($p < 0.001$). The subgingival calculus load increased with increasing smoking exposure, suggesting a dose–response relationship.

Conclusion: The present observations in dentally aware adults indicate a strong and independent impact of tobacco smoking on subgingival calculus deposition.

Key words: calcification; dental calculus; periodontal disease; smoking; tobacco

Accepted for publication 27 April 2004

In a previous communication elevated levels of supragingival dental calculus in smokers as compared with non-smokers and former smokers in a population of dentally aware adults was reported (Bergström 1999). Concordant with some earlier clinical and epidemiologic findings in various populations (Pindborg 1949, Alexander 1970, Ainamo 1971, Sheiham 1971, Anerud et al. 1991), the observations suggested that tobacco smoking is accompanied by an increased likelihood of supragingival dental calculus deposition. In the past, only a few studies have investigated the relationship between smoking and subgingival dental calculus. Although some of these studies do not strictly separate subgingival from supragingival calculus, they are largely suggestive of a positive association also between subgingival calculus and smoking (Pindborg 1949, Alexander 1970, Anerud et al. 1991, Linden & Mullally 1994). Subgingival calculus formation is associated with and probably consequential to the establishment of chronic inflammatory periodontal disease (Christersson et al. 1992). Because it is generally agreed that smoking is associated with increased rates of periodontal morbidity, it may be a priori presumed that smoking would be linked to subgingival calculus as well. In a recent study of patients with periodontal disease, however, it was reported that subgingival calculus, although increasingly prevalent with increasing disease severity, was inversely related to smoking (Martinez-Canut et al. 1999). Thus, there seems to be some controversy as to the role of smoking in subgingival calculus deposition. In order to further elucidate whether or not smoking is associated with excess risk of dental calculus deposition, the purpose of the present investigation was to estimate the prevalence and severity of the subgingival category of dental calculus in the previously described population. It was hypothesized that smoking, regardless of periodontal disease status, may promote subgingival calculus deposition by way of rendering subgingival plaque increasingly liable to calcification.

Material and Methods

Study population

The population to be investigated comprised a total of 242 individuals in the age range 20–69 years, 185 men and 57 women. The participation rate was 80%. The population has been previously described and is characterized by a high

standard of dental awareness (Bergström 1999, Bergström et al. 2000a). More than 90% claimed to be regular dental attenders visiting the dentist at least once every 2 years for the last 5–10 years. All claimed to brush their teeth on a regular daily basis and 66% used one or several additional oral hygiene aids such as dental floss and interdental brush. The oral health condition also was of a high standard as seen from, e.g. a great number of retained teeth and minimum bone loss (Eliasson & Bergström 1997). The average number of retained teeth was 28.4 ranging from 18 to 32. Also, in individuals 50 years of age and over the number of remaining teeth was high, the average being 26.6.

The fractions of current smokers, former smokers, and non-smokers were 20%, 23%, and 52%, respectively, whereas in 5% ($n = 12$) information about smoking was missing. The latter fraction was excluded. The frequency distribution of the study population according to age, gender, and smoking is presented in Table 1. The smoking exposure of current and former smokers was expressed in terms of smoking consumption (cigarettes smoked per day), smoking duration (number of years of smoking), and lifetime exposure, i.e. the product of consumption and duration (cigarette-years). The mean (SD) consumption, duration and lifetime exposure of current smokers was 13.8 (7.2) cigarettes/day, 20.4 (12.8) years and 299.3 (260.9) cigarette-years, respectively, and the previous exposure of former smokers 15.6 (8.6) cigarettes/day, 12.8 (9.4) years, and 238.9 (225.85) cigarette-years, respectively. Former smokers had given up smoking since on the average 12.8 (9.1) years. The distributions of current and former smokers according to exposure are presented in Table 2. The mean (SD) number of retained teeth in current smokers, former smokers, and non-smokers was 28.0 (2.5), 27.8 (2.8), and 28.7 (2.6), respectively. The study was approved by the local ethical committee of Karolinska Institutet, Huddinge, Sweden.

Assessments

The present analyses were based on 242 individuals with a full set of radiographs including 16 intra-oral projections, five anterior and four posterior projections of maxillary teeth, and three anterior and four posterior projections of mandibular teeth, and allowing an unambiguous assessment of subgingival calculus. The radiographs were taken according to a standardized paralleling and long-distance technique and the films (Kodak Ekta Speed E, Chalon-Sur-Saône, France) were processed according to the manufacturer's instructions at the Department of Oral Radiology, Karolinska Institutet, Huddinge, Sweden. Subgingival calculus was assessed on the mesial and distal root surfaces with the aid of a light table and a viewer allowing × 2 magnification. All teeth except third molars were dichotomously assessed according to presence or absence of subgingival calculus, where score 0 indicated no visible radiopaque deposit and score 1 a distinctly visible radiopaque deposit. Any radiopaque deposit on the root surface apical to the cemento-enamel junction was considered, whereas deposits coronal to the cemento-enamel junction only were disregarded. The severity of subgingival calculus within the individual, labeled subgingival calculus load, was expressed as (1) the absolute frequency of sites affected and (2) the frequency of affected sites as percentage of all sites at risk. Assessments were performed blinded as to the smoking status of the individual.

Supragingival calculus was bilaterally assessed on the lingual surfaces of the mandibular anterior teeth and the vestibular surfaces of the maxillary premolar and molar teeth as described previously (Bergström 1999). The occurrence of deposits in each region was assessed by clinical examination and the arithmetic mean across the four regions formed the supragingival calculus mean score of the individual. Supragingival plaque was assessed according to the plaque index of Silness & Löe (1964). Assessment was made on all buccal, distal, lingual, and mesial tooth surfaces in the individual following erythrosin staining. The mean of all assessments formed the plaque index of the individual. Gingival inflammation was assessed according to the gingival index of Löe & Silness (1963). The marginal gingivae along the buccal, distal, lingual, and mesial aspects of all teeth were assessed and the mean of all assessments formed the gingival index of the individual. Periodontal probing depth and bone height were assessed on the basis of all teeth as described elsewhere (Bergström & Eliasson 1986, Eliasson & Bergström 1997, Bergström et al. 2000a, b).

Table 1. Study population according to age, gender and smoking

Age (years)	Current smoker		Former smoker		Non-smoker		Other		Total	
	men	women	men	women	men	women	men	women	men	women
20–34	12	3	6	7	39	19	3	1	60	30
35–49	13	5	13	4	26	11	6	0	58	20
50–69	14	1	25	2	26	4	2	0	67	7
total	39	9	44	13	91	34	11	1	185	57

Table 2. Distribution of current and former smokers according to smoking exposure; n = number of individuals

Smoking status	Consumtion (cigarettes/day)				Duration (years)				Life-time exposure (cigarette-years)			
	light		heavy		light		heavy		light		heavy	
	n	range	*n*	range	*n*	range	*n*	range	*n*	range	*n*	range
current smoker	21	1–10	27	12–30	14	2–10	34	11–50	25	2–200	23	225– 980
former smoker	24	2–10	30	15–40	32	1–10	24	12–40	33	4–200	21	225–1050
total	45	1–10	57	12–40	46	1–10	58	11–50	58	2–200	44	225–1050

Statistics

Data are presented as means and 95% confidence intervals (CI) or standard errors of the mean (SEM). Statistical significance of differences between groups was tested according to Kruskal–Wallis ANOVA. Variables with a non-normal distribution were log transformed. Possible confounders were run together with smoking as co-variables in two-factor ANOVAS. Post hoc multiple comparisons were performed according to Scheffe's test. For proportions, sig-

nificance was tested using the χ^2-distribution with the application of Yates' correction where indicated. Multivariate testing was performed using multiple linear and multiple logistic regression analyses. Logistic regression was run with subgingival calculus load as the dependent response variable, transformed into a dichotomous variable with values greater than 0 recoded into 1, else 0. The relative risk was estimated from the odds ratios (OR) and 95% CI. When included in the multivariate logistic models, supragingival calculus was recoded into presence ($n = 168$) or absence ($n = 71$); periodontal pocketing into presence ($n = 146$) or absence ($n = 82$); bone height into low ($< 82.0\%$, $n = 59$) or high ($\geqslant 82.0\%$, $n = 170$); gingival index into low (0.07–0.49, $n = 71$), medium (0.50–0.97, $n = 85$), and high (1.00–2.00, $n = 71$); plaque index into low (0.05–0.64, $n = 81$), medium (0.65–0.89, $n = 78$), and high (0.90–2.00, $n = 69$). Age was stratified according to Table 1. Smoking consumption was stratified according to (1) 1–10 cigarettes/day ($n = 44$) and (2) > 10 cigarettes/day ($n = 57$); smoking duration according to (1) 1–10 years ($n = 46$) and (2) > 10 years ($n = 58$); and lifetime exposure according to (1) 1–200 cigarette-years ($n = 58$) and (2) > 200 cigarette-years ($n = 44$). In the linear regressions, smoking was introduced as two dummy variables, current smoking, i.e. current smokers versus former smokers and non-smokers, and former smoking, i.e. former smokers versus current smokers and non-smokers.

Error of measurement

The radiographic assessment was independently performed by two observers, each observer assessing the total number of individuals and blinded as to the smoking status of the individual. The inter-observer agreement was tested according to the Pearson product–moment correlation. The correlation coefficient was $r = 0.93$ and $p = 0.000$. The intra-observer reproducibility was tested in 25 randomly selected individuals and estimated from duplicate measurements within a time frame of 2 days. The reproducibility was expressed as the precision (s), i.e. the standard deviation of a single measurement, according to

$$s = \sqrt{\sum d_i^2 / 2n},$$

where d_i is the difference between duplicates and n the number of duplicates. The estimates of the precision referring to a single measurement of subgingival calculus load were $s_1 = 0.9$ and $s_2 = 0.1$ for the two observers, respectively. Because each individual was represented by the average of the two observers' readings, the precision referring to a single mean is $s_3 = 0.6$. The precision pertaining to group means (s_m) was the order of 0.05–0.10. It is concluded that the influence of measurement error on group means is negligible.

Results

Prevalence

The prevalence of individuals exhibiting radiographic evidence of subgingival calculus at one or more proximal sites was 43% for the total population, increasing from 15% in age stratum 20–34 years to 72% in age stratum 50–69 years (Fig. 1). The increase with age was statistically significant ($\chi^2 = 62.1$, $p = 0.000$).

The prevalence among current smokers was 71% as against 53% among former smokers and 28% among non-smokers (Fig. 2). The difference across smoking groups was statistically significant ($\chi^2 = 28.3$, $p = 0.000$). The specific differences between current smokers and non-smokers, and former smokers and non-smokers were statistically significant (Yates' $\chi^2 = 24.4$, $p = 0.000$ and 9.1, $p = 0.002$, respectively). The difference between current and former smokers was almost significant (Yates' $\chi^2 = 2.9$, $p = 0.087$). The significant predominance of current smokers held true throughout all age groups. The prevalence was significantly higher in heavy exposure smokers than light exposure smokers in terms of smoking duration and lifetime exposure. This held true for current smokers alone (Yates' $\chi^2 = 5.7$, $p = 0.017$ and 4.2, $p = 0.041$, respectively) as well as for current and former smokers combined (Yates' $\chi^2 = 17.0$, $p = 0.000$ and 9.0, $p = 0.003$, respectively).

Severity

The frequency distribution of individuals according to subgingival calculus load was skewed to the left, suggesting a decreased frequency with increasing load. With the exception of one individual exhibiting an extreme of 50, the maximum frequency of affected sites per person was 11. Only 14 individuals (5%) exhibited more than six (10%) affected sites. The overall mean subgingival calculus load was 1.4 (2.5%) affected sites per person, increasing with age from 0.3 (0.5%) in age stratum 20–34 years to 2.3 (4.4%) in age stratum 50–69 years (Fig. 3). The association between age and subgingival calculus load was statistically significant both in absolute and relative terms (Kruskal–Wallis $H = 52.7$ and 54.5, respectively, $p = 0.000$). Gender was not statistically significantly associated with subgingi-

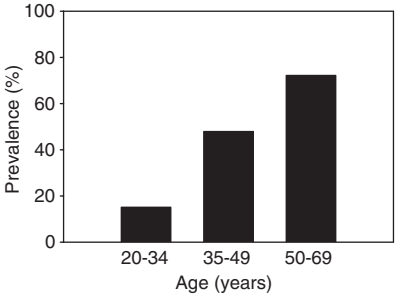

Fig. 1. Prevalence (%) of individuals with subgingival calculus according to age.

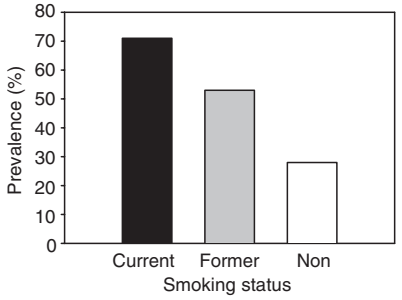

Fig. 2. Prevalence (%) of individuals with subgingival calculus according to smoking status.

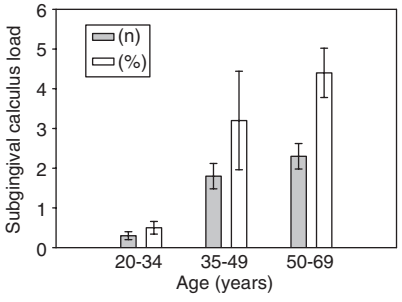

Fig. 3. Subgingival calculus load as number (n) and proportion (%) of affected sites. Mean and SEM by age.

val calculus load when age was controlled for.

The mean subgingival calculus load for current smokers was 3.4 (6.2%) affected sites per person as against 1.2 (2.4%) for former smokers, and 0.6 (1.1%) for non-smokers (Fig. 4). The association between smoking and subgingival calculus load was statistically significant both in absolute and relative terms (Kruskal–Wallis $H = 32.8$, and 33.2, respectively, $p = 0.000$). The association remained statistically significant when controlling for age ($F(2, 2) = 24.1$ and 32.8, respectively, $p = 0.000$). Post hoc multiple comparisons testing indicated statistically significant differences, in both absolute and relative terms, between current smokers and non-smokers ($F = 19.5$ and 18.6, respectively, $p = 0.000$), and between current and former smokers ($F = 6.0$ and 5.4, respectively, $p = 0.000$). Further, the association between smoking and subgingival calculus load remained statistically significant when controlling for the influence of other significant factors, one at a time, such as gingival index, periodontal bone height, periodontal pocketing, plaque index, or supragingival calculus in two-factor ANOVAS (data not shown).

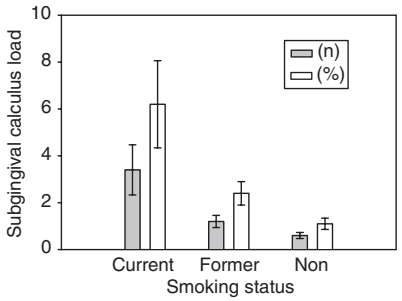

Fig. 4. Subgingival calculus load as number (*n*) and proportion (%) of affected sites. Mean and SEM by smoking status.

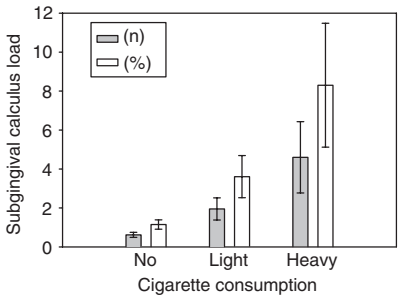

Fig. 5. Subgingival calculus load as number (*n*) and proportion (%) of affected sites. Age adjusted means and SEM by cigarette consumption for current smokers.

The subgingival calculus load was significantly associated with smoking exposure measures in a dose-dependent manner (Fig. 5). Controlling for age, the association between cigarette consumption, smoking duration, and lifetime exposure, respectively, and subgingival calculus load was statistically significant in both absolute and relative terms for current smokers alone ($F(2, 2) = 25.3$ (21.6), 12.6 (17.6), and 13.7 (15.7), respectively, $p = 0.000$) as well as for current and former smokers combined ($F(2, 2) = 12.9$ (12.2), 9.0 (9.2), and 8.6 (9.0), respectively, $p = 0.000$).

Multivariate modeling and risk assessment

Multiple linear regression including subgingival calculus load as the dependent variable and age, current smoking, former smoking, gingival index, periodontal bone height, plaque index, and supragingival calculus as predictors, introduced in one block, suggested that subgingival calculus load could be linearly predicted from a combination of these variables accounting for 43% of the variance in the dependent variable ($F = 20.3$, $p = 0.000$, $R^2(\text{adj}) = 0.43$). The strongest predictors were age, current smoking, and supragingival calculus (Table 3).

Multiple logistic regression was run with subgingival calculus load as the dependent response variable (recoded into presence = 1, absence = 0) and age, gender, gingival index, periodontal bone height, periodontal pocketing, plaque index, smoking, and supragingival calculus as predictors. Univariate analysis indicated that age, smoking, gingival index, periodontal bone height, periodontal pocketing, and supragingival calculus were significantly associated with subgingival calculus load, whereas gender and plaque index were not (Table 4). In a multivariate analysis including all significant factors, age (OR = 1.1, $p = 0.000$), smoking (OR = 3.1, $p = 0.007$), and supragingival calculus

Table 3. Multiple regression analysis with subgingival calculus load as dependent variable. $R^2 = 0.43$

Variable	Parameter	SEM	*t*-value	*p*
intercept	0.8			
age	0.01	0.004	3.7	0.000
current smoking	0.48	0.096	5.0	0.000
former smoking	0.11	0.089	1.2	0.232
gingival index	0.01	0.003	2.4	0.019
periodontal bone height	−0.02	0.009	1.9	0.060
plaque index	−0.00	0.001	0.2	0.800
supragingival calculus	0.37	0.125	2.9	0.004

Table 4. Potential risk factors for subgingival calculus. Odds ratios (OR) and 95% confidence intervals (CI). Univariate analysis

Factor	OR	95% CI	*p*
Age			
young	1		
middle	3.8	2.6; 5.6	
old	14.4	6.6; 31.0	0.000
Periodontal bone height			
high	1		
low	4.9	2.6; 9.3	0.000
Periodontal pockets			
no	1		
yes	2.3	1.3; 4.1	0.000
Gingival index			
low	1		
medium	1.9	1.3; 2.7	
high	3.5	1.8; 7.0	0.000
Smoking			
no	1		
yes	3.4	1.8; 6.5	0.000
Supragingival calculus			
no	1		
yes	6.0	2.9; 12.3	0.000

(OR = 11.0, $p = 0.000$) remained statistically significant. Further, the risk of exhibiting subgingival calculus was 8.5-fold elevated among current smokers compared with non-smokers after adjustment for age and other confounders (OR = 8.5, $p = 0.000$, Table 5). The risk among former smokers was slightly elevated (OR = 2.2, $p = 0.051$).

The relative risk associated with exposure variables indicated a dose–response relation (Table 6). Significant trends for increasingly elevated relative risks with increasing exposure were observed regarding consumption, duration, as well as lifetime exposure among current smokers alone and among current and former smokers combined. Compared with a non-smoker, a smoker that had smoked 15–20 cigarettes/day for 15–20 years ran a 15–20-fold elevated risk of exhibiting subgingival calculus after adjustment for age and other possible confounders.

Discussion

The overall prevalence of subgingival calculus in the present population of dentally aware individuals was 43% suggesting that a majority remained unaffected. Moreover, only 5% displayed a subgingival calculus load in excess of six (10%) affected sites, suggesting that affected individuals had predominantly low to moderate levels. Radiographically detectable calculus assessed at the root surface apical to the cemento-enamel junction may not clinically be equivalent to calculus of a subgingival localization. Along with the ongoing periodontal retraction and bone height reduction with age and/or disease, portions of the root may become exposed and some deposits radiographically assessed as subgingival may clinically be localized supragingivally. Vice versa, deposits coronal to the cemento-enamel junction might clinically represent subgingival calculus even in the absence of a true pocket as the result of gingival inflammation. Unfortunately, clinical assessment was not performed in parallel, why an unambiguous distinction between subgingival and supragingival calculus may not have been fully possible. However, because supragingival calculus was accounted for in the calculations, any confounding from this factor did not influence the comparison between smoking groups. Slight uncertainty as to calculus localization, therefore, is unlikely to affect group comparisons. A further limitation inherent in the radiographic assessment method is the fact that only proximal aspects of the root surfaces are assessable. Although subgingival calculus is predominantly observed at proximal sites (Anerud et al. 1991, Corbett & Dawes 1998), the use of radiographic assessment alone will inevitably result in an underestimation. This type of systematic error, however, will not greatly distort comparisons between smoking groups. The presently used radiographic method, producing assessments of a high precision as found by the reassuring tests of intra- and inter-observer reproducibility, therefore, is considered appropriate for the purpose of the study.

The hypothesis to be tested in the present investigation was that tobacco smoking enhances the subgingival deposition of calculus onto root surfaces. Consistent with the hypothesis, the observations demonstrated significantly elevated levels of subgingival calculus in association with smoking. Together with earlier findings in various populations (Pindborg 1949, Alexander 1970, Ainamo 1971, Sheiham 1971, Anerud et al. 1991) as well as in the present one (Bergström 1999), reflecting a positive association between smoking and supragingival calculus, the results reported herein suggest that also subgingival calcification is promoted by smoking. The observations confirm and further strengthen the findings of a few earlier reports (Alexander 1970, Linden & Mullally 1994). It may be argued that the association between subgingival calculus and smoking simply reflects a confounding influence of periodontal morbidity as periodontal disease is more prevalent and severe in smokers (Bergström & Flodérus-Myrhed 1983, Bergström 1989, Haber & Kent 1992, Haber et al. 1993, Bergström & Preber 1994, Grossi et al. 1994, 1995, Martinez-Canut et al. 1995, Bergström et al. 2000a, b). This explanation is unlikely, however, because the effect of smoking remained when the possible confounding of factors

Table 5. Relative risk for exhibiting subgingival calculus

	Current smokers			Former smokers			Current and former smokers		
	OR	95% CI	*p*	OR	95% CI	*p*	OR	95% CI	*p*
crude	6.2	2.9–12.9	0.000	2.8	1.5–5.4	0.002	3.4	1.8–6.5	0.000
adjusted*	8.4	3.6–19.9	0.000	1.4	0.9–2.1	0.075	2.6	1.2–5.5	0.011
adjusted†	8.5	3.2–22.3	0.000	2.2	1.0–5.1	0.051	3.4	1.5–7.4	0.003

Crude and adjusted OR and 95% CI for current and former smokers with reference to non-smokers.
*Adjusted for age.
†Adjusted for age, periodontal bone height, pocket frequency, gingival index, and supragingival calculus.

Table 6. Relative risk for current smokers of exhibiting subgingival calculus with reference to non-smokers

	Consumption (cigarettes/day)					Duration (years)					Life-time exposure (cigarette-years)				
	light		heavy		trend	light		heavy		trend	light		heavy		trend
	OR	95% CI	OR	95% CI	*p*	OR	95% CI	OR	95% CI	*p*	OR	95% CI	OR	95% CI	*p*
crude	3.0	1.9–4.8	8.9	3.5–22.7	0.000	3.2	2.1–5.1	10.4	4.2–25.7	0.000	3.8	2.2–6.5	14.6	5.0–42.7	0.000
adjusted*	3.5	2.0–6.0	12.2	4.1–36.2	0.000	3.5	2.1–5.8	12.1	4.3–33.8	0.000	4.4	2.3–8.4	19.1	5.2–70.3	0.000
adjusted†	3.2	1.7–5.8	10.2	3.1–33.7	0.000	3.4	1.9–5.9	11.5	3.7–35.4	0.000	4.2	2.1–8.5	18.0	4.4–72.8	0.000

Crude and adjusted OR and 95% CI according to smoking exposure.
*Adjusted for age.
†Adjusted for age, periodontal bone height, pocket frequency, gingival index, supragingival calculus.

such as gingival inflammation, periodontal pocketing, periodontal bone loss, supragingival plaque and supragingival calculus was accounted for. Thus, the observed effect of smoking was independent of disease severity and oral hygiene condition. This contention is further supported by the additional observation that subgingival calculus deposition increased with increasing smoking exposure, suggesting a dose–response relation. By the same token, giving up smoking seemed to reverse the effect as suggested by the observation that former smokers who had given up smoking in the past were less likely to be affected than current smokers.

No plausible hypothesis has yet been advanced to explain the evidence linking dental calculus deposition with smoking. Such a hypothesis would enable an explanation of both the supragingival and subgingival categories of dental calculus and, in addition, patho-physiological calcification elsewhere in the body. The formation of subgingival calculus is generally considered to be a process by which calcium and phosphorus emanating from the gingival crevicular fluid is absorbed into the subgingival plaque or biofilm (White 1997). The process of calcification is favored by the presence of cells within the biofilm. Certain elements of bacterial or cellular origin such as lipoteichoic acid and proteins may play an essential role as contributing factors (Nancollas & Johnson 1994). Whether various bacterial species would have different calcification potential is not well known and may not be important in the context of smoking, because most studies agree that the subgingival microflora is independent of smoking (Preber et al. 1992, 1995, Stoltenberg et al. 1993, Boström et al. 1998, 1999, 2001, Renvert et al. 1998). It seems unlikely, therefore, that smoking would influence the process of calcification by interfering with the subgingival microflora. A stimulatory effect by smoking on inflammatory events and crevicular fluid production could be a possible mechanism. Contrary to expectation, however, smoking is frequently associated with less severe clinical signs of inflammation, and evidence has been provided that smoking suppresses periodontal signs and symptoms of inflammation including crevicular fluid flow rate (Preber & Bergström 1985, Bergström & Preber 1986, Bergström et al. 1988, Bergström 1990, Persson et al. 1999, Bergström & Boström 2001, Rezavandi et al. 2002). Suppression of gingival crevicular fluid flow, however, might retard the clearance and prolong the contact time between fluid and root surfaces allowing an increased exchange of fluid constituents, including mineral ions, within the gingival sulcus or periodontal pocket. Further, it can be conceived that a reduced flow may cause elevation of the calcium and phosphate concentrations. Thus, paradoxically, smoking might promote the calcification of subgingival plaque notwithstanding its suppressive action on some inflammatory events.

Subgingival calculus formation may have similarities with ectopic calcification elsewhere in the body such as atherosclerosis of vessel wall endothelium of major vessels, and pancreatic, placental and renal calcification (Anderson 1983, Wexler et al. 1996, Wallin et al. 2001). Although the mechanisms of calcification are incompletely understood, a common feature of calcific diseases is the formation of crystalline, insoluble, calcium–phopsphate mineral in the form of hydroxyapatite (Anderson 1983, LeGeros 2001, Proudfoot & Shanahan 2001, Doherty et al. 2003). Tobacco smoking is a major risk factor for the occurrence of ectopic calcification and calcific diseases (Danielsen et al. 1996, Maher et al. 1996, Klesges et al. 1998, Imoto & DiMagno 2000, Iribarren et al. 2000, Newman et al. 2001, Manger et al. 2003).

It has been frequently reported that ectopic calcification can occur at the same time as loss of bone tissue from the skeleton, and many osteogenic regulatory factors of bone development are also expressed in atherosclerotic lesions, suggesting that the same mechanisms are operating in mineral deposition and resorption (Demer 2002). Furthermore, inflammation is thought to play a role in the regulation of vascular calcification. It has been demonstrated that tumor necrosis factor-α (TNF-α), among several soluble factors, is involved in the process of ectopic calcification (Tintut et al. 2002). Inflammation is an integral part of destructive periodontal disease and several inflammatory mediators such as interleukins (IL)-1 and -6, and (TNF)-α are expressed in tissue destruction (Birkedal-Hansen 1993, Alexander & Damoulis 1994). Interestingly, we have previously provided evidence of elevated levels of TNF-α in crevicular fluid of smoker patients who respond with inadequate bone formation following therapy (Boström et al. 1998). It might therefore be speculated that periodontal bone resorption induced by smoking locally acts in concert with inflammatory mediators to produce a crevicular exudate enriched in certain elements that enhance calcification of cellular material on dental root surfaces. Thus, the increased amounts of subgingival calcification observed in smokers may well be a sequel of the elevated rate of periodontal bone resorption caused by smoking (Bergström et al. 2000a, b, Bergström 2004).

In summary, the present observations have demonstrated a strong association between smoking and subgingival calculus deposition beyond any influence of age, oral hygiene and periodontal morbidity. The association observed most likely reflects a generalized susceptibility to calcification brought about by tobacco smoking. The present observations merit further investigation into the relations between tobacco smoking, calcification of dental deposits, and ectopic calcification of other tissues.

Acknowledgments

The authour is grateful to Dr A. Homayounfar, Växjö, Sweden and Dr P. Bahman, London, UK for their assistance in the study.

References

Ainamo, J. (1971) The seeming effect of tobacco consumption on the occurrence of periodontal disease and dental caries. *Suomen Hammaslääkinen Toimistu* **67**, 87–94.

Alexander, A. G. (1970) The relationship between tobacco, plaque accumulation and gingivitis. *Dental Health* **9**, 6–9.

Alexander, M. & Damoulis, P. (1994) The role of cytokines in the pathogenesis of periodontal disease. *Current Opinion in Periodontology* 39–53.

Anderson, H. C. (1983) Calcific diseases. A concept. *Archives of Pathology and Laboratory Medicine* **107**, 341–348.

Anerud, A., Löe, H. & Boysen, H. (1991) The natural history and clinical course of calculus formation in man. *Journal of Clinical Periodontology* **18**, 160–170.

Bergström, J. (1989) Cigarette smoking as risk factor in chronic periodontal disease. *Community Dentistry and Oral Epidemiology* **17**, 245–247.

Bergström, J. (1990) Oral hygiene compliance and gingivitis expression in cigarette smokers. *Scandinavian Journal of Dental Research* **98**, 497–503.

Bergström, J. (1999) Tobacco smoking and supragingival dental calculus. *Journal of Clinical Periodontology* **26**, 541–547.

Bergström, J. (2004) Influence of tobacco smoking on periodontal bone height. Long-term observations and a hypothesis. *Journal of Clinical Periodontology* **31**, 260–266.

Bergström, J. & Boström, L. (2001) Tobacco smoking and periodontal hemorrhagic responsiveness. *Journal of Clinical Periodontology* **28**, 680–685.

Bergström, J. & Eliasson, S. (1986) Alveolar bone height in professional musicians. *Acta Odontologica Scandinavica* **44**, 141–147.

Bergström, J., Eliasson, S. & Dock, J. (2000a) Exposure to tobacco smoking and periodontal health. *Journal of Clinical Periodontology* **27**, 61–68.

Bergström, J., Eliasson, S. & Dock, J. (2000b) A 10-year prospective study of tobacco smoking and periodontal health. *Journal of Periodontology* **71**, 1338–1347.

Bergström, J. & Flodérus-Myrhed, B. (1983) Co-twin control study of the relationship between smoking and some periodontal disease factors. *Community Dentistry and Oral Epidemiology* **11**, 113–116.

Bergström, J., Persson, L. & Preber, H. (1988) Influence of cigarette smoking on vascular reaction during experimental gingivitis. *Scandinavian Journal of Dental Research* **96**, 34–39.

Bergström, J. & Preber, H. (1986) The influence of cigarette smoking on the development of experimental gingivitis. *Journal of Periodontal Research* **21**, 668–676.

Bergström, J. & Preber, H. (1994) Tobacco use as a risk factor. *Journal of Periodontology* **65**, 545–550.

Birkedal-Hansen, H. (1993) Role of cytokines and inflammatory mediators in tissue destruction. *Journal of Periodontal Research* **28**, 500–510.

Boström, L., Bergström, J., Dahlén, G. & Linder, L. E. (2001) Smoking and subgingival microflora in periodontal disease. *Journal of Clinical Periodontology* **28**, 212–219.

Boström, L., Linder, L. E. & Bergström, J. (1998) Clinical expression of TNF-α in smoking-associated periodontal disease. *Journal of Clinical Periodontology* **25**, 767–773.

Boström, L., Linder, L. E. & Bergström, J. (1999) Smoking and crevicular fluid levels of IL-6 and TNF-α in periodontal disease. *Journal of Clinical Periodontology* **26**, 352–357.

Christersson, L., Grossi, S., Dunford, R., Machtei, E. & Genco, R. (1992) Dental plaque and calculus. Risk indicators for their formation. *Journal of Dental Research* **71**, 1425–1430.

Corbett, T. L. & Dawes, C. (1998) A comparison of the site-specificity of supragingival and subgingival calculus deposition. *Journal of Periodontology* **69**, 1–8.

Danielsen, R., Sigvaldson, H., Thorgeirsson, G. & Sigfusson, N. (1996) Predominance of aortic calcification as an atherosclerotic manifestation in women: the Reykjavik study. *Journal of Clinical Epidemiology* **49**, 383–387.

Demer, L. (2002) Vascular calcification and osteoporosis: inflammatory responses to oxidized lipids. *International Journal of Epidemiology* **31**, 737–741.

Doherty, T., Asotra, K., Fitzpatric, L., Qiao, J.-H., Wilkin, D., Detrano, R., Dunstan, C., Shah, P. & Rajavashisth, T. (2003) Calcification in atherosclerosis: bone biology and chronic inflammation at the arterial crossroads. *Proceedings of the National Academy of Science USA* **100**, 1120–1126.

Eliasson, S. & Bergström, J. (1997) Minimum periodontal bone loss in dentally aware adults. A 10-year prospective study. *Journal of Clinical Periodontology* **24**, 34–38.

Grossi, S. G., Genco, R. G., Machtei, E. E., Ho, A. W., Koch, G., Dunford, R. G., Zambon, J. J. & Hausmann, E. (1995) Assessment of risk for periodontal disease. II. Risk indicators for alveolar bone loss. *Journal of Periodontology* **66**, 23–29.

Grossi, S. G., Zambon, J. J., Ho, A. W., Koch, G., Dunford, R. G., Machtei, E. E., Norderyd, O. M. & Genco, R. G. (1994) Assessment of risk for periodontal disease. I. Risk indicators for attachment loss. *Journal of Periodontology* **65**, 260–267.

Haber, J. & Kent, R. L. (1992) Cigarette smoking in a periodontal practice. *Journal of Periodontology* **63**, 100–106.

Haber, J., Wattles, J., Crowley, M., Mandell, R., Joshipura, K. & Kent, R. (1993) Evidence for cigarette smoking as a major risk factor for periodontitis. *Journal of Periodontology* **64**, 16–23.

Imoto, M. & DiMagno, E. (2000) Cigarette smoking increases the risk of pancreatic calcification in late-onset but not early-onset idiopathic chronic pancreatitis. *Pancreas* **21**, 115–119.

Iribarren, C., Sidney, S., Sternfeld, B. & Browner, W. (2000) Calcification of the aortic arch. Risk factors and association with coronary heart disease, stroke, and peripheral vascular disease. *Journal of the American Medical Association* **283**, 2810–2815.

Klesges, L., Murray, D., Brown, J., Cliver, S. & Goldenberg, R. (1998) Relations of cigarette smoking and dietary antioxidants with placental calcification. *American Journal of Epidemiology* **147**, 127–135.

LeGeros, R. (2001) Formation and transformation of calcium phosphates: relevance to vascular calcification. *Zeitschrift fur Kardiologie* **90** Suppl 3, 116–124.

Linden, G. & Mullally, B. (1994) Cigarette smoking and periodontal destruction in young adults. *Journal of Periodontology* **65**, 718–723.

Löe, H. & Silness, J. (1963) Periodontal disease in pregnancy. I Prevalence and severity. *Acta Odontologica Scandinavica* **21**, 533–551.

Maher, J., Raz, J., Bielak, L., Sheedy, P., Schwartz, R. & Peyser, P. (1996) Potential of quantity of coronary artery calcification to identify new risk factors for asymptomatic atherosclerosis. *American Journal of Epidemiology* **144**, 943–953.

Manger, K., Kusus, M., Forster, C., Ropers, D., Daniel, W., Kalden, J., Achbach, S. & Manger, B. (2003) Factors associated with coronary artery calcification in young female patients with SLE. *Annals of the Rheumatic Diseases* **62**, 846–855.

Martinez-Canut, P., Benlloch, D. & Izcuierdo, R. (1999) Factors related to the quantity of subgingival calculus in proximal root surfaces. *Journal of Clinical Periodontology* **26**, 519–524.

Martinez-Canut, P., Lorca, A. & Magan, R. (1995) Smoking and periodontal disease severity. *Journal of Clinical Periodontology* **22**, 743–749.

Nancollas, G. H. & Johnson, M. A. S. (1994) Calculus formation and inhibition. *Advances in Dental Research* **8**, 307–311.

Newman, A., Naydeck, B., Sutton-Tyrrel, K., Feldman, A., Edmundowicz, D. & Kuller, L. (2001) Coronary artery calcification in older adults. *Circulation* **104**, 2679–2689.

Persson, L., Bergström, J., Gustafsson, A. & Åsman, B. (1999) Tobacco smoking and gingival neutrophil activity in young adults. *Journal of Clinical Periodontology* **26**, 9–13.

Pindborg, J. J. (1949) Tobacco and gingivitis II. Correlation between consumption of tobacco, ulceromembranous gingivitis and calculus. *Journal of Dental Research* **28**, 460–463.

Preber, H. & Bergström, J. (1985) Occurrence of gingival bleeding in smoker and non-smoker patients. *Acta Odontologica Scandinavica* **43**, 315–320.

Preber, H., Bergström, J. & Linder, L. E. (1992) Occurrence of periopathogens in smoker and non-smoker patients. *Journal of Clinical Periodontology* **19**, 667–671.

Preber, H., Linder, L. E. & Bergström, J. (1995) Periodontal healing and periopathogenic microflora in smokers and non-smokers. *Journal of Clinical Periodontology* **22**, 946–952.

Proudfoot, D. & Shanahan, C. (2001) Biology of calcification in vascular cells: intima versus media. *Hertz* **26**, 245–251.

Renvert, S., Dahlén, G. & Wikstrom, M. (1998) The clinical and microbiological effects of non-surgical therapy in smokers and non-smokers. *Journal of Clinical Periodontology* **25**, 153–157.

Rezavandi, K., Palmer, R., Odell, E., Scott, D. & Wilson, R. (2002) Expression of ICAM-1 and E-selectin in gingival tissues of smokers and non-smokers with periodontitis. *Journal of Oral Pathology and Medicine* **3**, 59–64.

Sheiham, A. (1971) Periodontal disease and oral cleanliness in tobacco smokers. *Journal of Periodontology* **42**, 259–263.

Silness, J. & Löe, H. (1964) Periodontal disease in pregnancy. II Correlation between oral hygiene and periodontal condition. *Acta Odontologica Scandinavica* **22**, 121–135.

Stoltenberg, J. L., Osborn, J. B., Pihlstrom, B. L., Herzberg, M. C., Aeppli, D. M., Wolff, L. F. & Fisher, G. E. (1993) Association between cigarette smoking, bacterial

pathogens and periodontal status. *Journal of Periodontology* **64**, 1225–1230.

Tintut, Y., Patel, J., Territo, M., Saini, T., Parhami, F. & Demer, L. (2002) Monocyte/macrophage regulation of vascular calcification in vitro. *Circulation* **105**, 650–655.

Wallin, . R., Wajih, N., Greenwood, G. & Sane, D. (2001) Arterial calcification: a review of mechanisms, animal models, and the prospects for therapy. *Medical Research Reviews* **21**, 274–301.

Wexler, L., Brundage, B., Crouse, J., Detrano, R., Fuster, V., Maddahi, J., Rumberger, J., Stanford, W., White, R. & Taubert, K. (1996) Coronary artery calcification: pathophysiology, epidemiology, imaging methods, and clinical implications. A statement for health professionals. *Circulation* **94**, 1175–1192.

White, D. J. (1997) Dental calculus: recent insights into occurrence, formation, prevention, removal and oral health effects of supragingival and subgingival calculus deposits. *European Journal of Oral Sciences* **105**, 508–522.

Address:
Jan Bergström
Institute of Odontology
The Karolinska Institute
Box 4064, Alfred Nobels Allé 8
S-141 04 Huddinge
Sweden
E-mail: Jan.bergstrom@ofa.ki.se