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Smoking, a weak predictor of periodontitis in older adults

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Abstract

Background: The impact of smoking habits on periodontal conditions in older subjects is poorly studied.

Aims: To assess if a history of smoking is associated with chronic periodontitis and medical history in older subjects.

Material and Methods: The medical and dental history was collected from 1084 subjects 60–75 years of age. Smoking history information was obtained from self-reports. Periodontal variables [clinical probing depth (PD) $\geqslant$ 5.0 mm, clinical attachment levels (CALs) $\geqslant$ 4.0 mm], and radiographic evidence of alveolar bone loss were assessed.

Results: 60.5% had never smoked (NS), 32.0% were former smokers (FS) (mean smoke years: 26.1 years, SD $\pm$ 13.1), and 7.5% were current smokers (CS) (mean smoke years 38.0 years, (SD $\pm$ 12.1). The proportional distribution of CAL $\geqslant$ 4.0 mm differed significantly by smoking status (NS and CS groups) (mean difference: 12.1%, 95% confidence interval (CI): 1.5–22.6, $p < 0.02$). The Mantel–Haenszel common odds ratio between smoking status (CS+FS) and periodontitis (>20% bone loss) was 1.3 ($p < 0.09$, 95% CI: 0.9–2.0) and changed to 1.8 ($p < 0.02$, 95% CI: 1.3–2.7) with 30 years of smoking as cutoff. A weak correlation between number of years of smoking and CAL $\geqslant$ 4.0 mm was demonstrated (r^2 values 0.05 and 0.07) for FS and CS, respectively. Binary logistic forward (Wald) regression analysis demonstrated that the evidence of carotid calcification, current smoking status, gender (male), and the number of remaining teeth were explanatory to alveolar bone loss.

Conclusions: A clinically significant impact on periodontal conditions may require 30 years of smoking or more. Tooth loss, radiographic evidence of carotid calcification, current smoking status, and male gender can predictably be associated with alveolar bone loss in older subjects.

Key words: bone loss; clinical attachment; older; periodontitits; smoking habit

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It has been demonstrated that although smoking has an impact on alveolar bone loss this relationship almost disappears when controlling for current oral hygiene levels (Bolin et al. 1986). A reduced tendency of gingival bleeding in smokers has been reported (Preber & Bergström 1985). Contradictory findings with higher bleeding index among smokers, although also associated with poor oral hygiene in smokers, have been reported (Amarasena et al. 2003). Analysis of the National Health and Nutrition Examination Survey III (NHANES III) data has demonstrated difficulties in distinguishing the effects of periodontitis from those of smoking with respect to a smoking-related outcome (Spiekerman et al. 2003).

Limited differences in periodontal status have been reported among periodontal care seeking smokers and non-smokers (van der Weijden et al. 2001). It has also been pointed out that smoking is not a prerequisite for developing periodontitis and that the clinical significance of smoking may be limited (van der Weijden et al. 2001). The effects of initial cause-related therapy at 1 month after completion of treatment have been shown to be similar between smokers and non-smokers (Preber & Bergström 1986).

However, smoking habits as a risk factor for periodontitis have been reported in a large number of studies (i.e. Ismail et al. 1983, Bergström 1989, Haber et al. 1993). In further analysis of the NHANES III, studies have demonstrated that smokers have a higher prevalence of periodontal sites with increased probing depth (PD) (Calsina et al. 2003, Hyman & Reid 2003). Loss of clinical attachment level (CAL) has been associated with smoking in subjects 50 years of age or more (Hyman & Reid 2003). Such findings are consistent with reports that long time smoking is associated with chronic

periodontitis (Bergström 2003, Teng et al. 2003).

The combination of smoking and being carrier of a genetic factor such as the interleukin 1 polymorphism gene significantly increases the risk for periodontitis (Meisel et al. 2003). The impact of smoking on the subgingival microflora remains unclear (Stoltenberg et al. 1993, Darby et al. 2000, Boström et al. 2001, Eggert et al. 2001).

Few studies have provided long-term longitudinal data on alveolar bone loss in smokers and non-smokers. In one such study only marginal and clinically insignificant differences in periodontal status were found between smokers and non-smokers (Laurell et al. 2003). The effect of long-term smoking habits on periodontal conditions in older subjects is not well known. There is also limited information on the impact of smoking cessation and its effects on future periodontal status.

The purposes of the present study were to: (I) address the effect of smoking on periodontal status and on tooth loss in older subjects from diverse ethnic backgrounds, (II) to assess what factors known from the medical and dental histories (including smoking status) were explanatory to alveolar bone loss.

Materials and Methods

The data used in the present study were derived from the baseline clinical findings of the ''Trials to Enhance Elders'' ''Teeth and Oral Health'' (TEETH trial). The medical and dental history of all subjects was collected. Details of the TEETH study including the recruitment strategies and methods of data collection have been described elsewhere (MacEntee et al. 2002, Persson et al. 2002a, b). The TEETH trial was approved by the Institutional Review Board at the University of British Columbia and the University of Washington; data collections and subject safety are monitored by a Data and Safety Managing Board (DSMB). All study subjects had signed consent to participate in the TEETH trial.

The inclusion criteria specified that only adults who were at least 60 years of age and with at least four remaining teeth could participate. Efforts were made to obtain an ethnically diverse sample representing the predominant ethnic groups in the Pacific Northwest. All subjects responded to a comprehensive health questionnaire and interviews regarding self-perceptions of periodontal disease risk, also including information on smoking habits, the number of years of smoking, marital status, and medical conditions. The latter information was confirmed by communication with subjects' physicians as deemed necessary and/or reviews of prescription lists or actual medications brought to the examination. Subjects were identified as never smokers (NS) if they had never smoked, current smokers (CS) if they currently had a smoking habit, and as former smokers (FS) if they reported that they had quit smoking.

Subjects were asked to identify their perceived risk for future caries, periodontitis, and tooth loss on a 0–100 scale. Clinical periodontal conditions were assessed at six surfaces per tooth, including PD and clinical attachment level measurements (Persson et al. 2002a, b). Subject-based data using the computed proportions of sites with a PD $\geqslant 5.0$ mm and the proportions of sites with a loss of clinical attachment $\geqslant 4.0$ mm were used for the analysis. Likewise, subject-based data for the number of sites with PD $\geqslant 5.0$ mm and CAL $\geqslant 4.0$ mm were also studied. CALs were calculated from data on clinical PD and measurements of gingival recession.

Panoramic radiographs were taken and radiographic evidence of horizontal alveolar bone loss and vertical bone defects were identified. Bone loss was defined as the distance between the bone level and the cement-enamel junction $\geqslant 4.0$ mm. Subjects were classified as; ''0'' if they had no radiographic evidence of horizontal, vertical or any other inter-radicular bone loss, ''1'' if they had evidence of alveolar bone loss $<25\%$ of bone height, and ''2'' if the extent of alveolar bone loss varied, on average, between 25% and 50% of bone height, and ''3'' if the extent of alveolar bone loss exceeded 50% of the root length as a generalized pattern of bone loss (Persson et al. 2002a, b). In the dichotomous analysis bone conditions were defined as having no evidence of alveolar bone loss (score ''0'') while any other condition was assigned a score of ''1''.

Statistical analysis

Descriptive statistics were used to characterize the sample. The One-way ANOVA using Bonferroni post hoc or Kruskal–Wallis ANOVA was used to ascertain if there were differences in periodontal parameters by smoking status (never, current, former-smokers). Spearman rank correlations and Mantel–Haenszel common odds ratios were used to assess the risk of having periodontitis and a smoking history. Binary logistic forward (Wald) regression analysis was performed to study which study variables were explanatory to evidence of alveolar bone loss. The SPSS 11.5 statistical software program for PC was used (SPSS Inc., Chicago, IL, USA).

Results

Subject characteristics

Data were collected from 882 subjects from whom a smoking record could be verified. The mean age of the subjects was 67.1 years (SD $\pm$ 4.7) and 53.7% were women. The three major ethnic groups were subjects with European descent (47.6%), Asian descent (primarily Chinese) (29.7%), and those with African descent (7.6%). In this population 60.5% reported that they had never smoked, 32.0% reported that they had quit smoking, and 7.5% reported that they were current smokers. The number of years of smoking among current and former smokers varied between 2 and 64 years.

Subjects in the CS group had been smoking for on average 38.0 years (SD $\pm$ 12.1 years, median: 40.0, range: 10.0–61.0 years). Subjects in the FS group had been smoking for on average 26.1 years (SD $\pm$ 13.1 years, median: 25.0, range: 2–64 years). Smoking habits differed by ethnicity ($p<0.001$, Kruskal–Wallis ANOVA) in that those of European descent (51%) and those of African descent (60.8%) reported that they either were current or former smokers as compared with those with Asian descent (17.5%) ($p<0.001$). The ethnic differences in the proportions of those who were current smokers was however limited (10.1%, 4.9%, and 7.6%, respectively).

Subjects in the NS group perceived their future risks for caries, periodontitis and tooth loss as lower than those in the CS or FS groups ($p<0.001$) with no differences between the two groups of smokers. The average number of remaining teeth in the NS, CS, or FS groups was 22.7 teeth (SD $\pm$ 6.3), 21.0 teeth (SD $\pm$ 7.5), and 20.9 teeth (SD $\pm$ 6.9), respectively. Notwithstanding, One-way ANOVA Bonferroni post hoc test demonstrated a statistically sig-

nificant difference in that subjects in the NS group had significantly more remaining teeth than subjects in the CS or FS groups ($p<0.001$) but with no difference between CS and FS groups.

Periodontal clinical measures

The distribution of sites with PD $\geqslant$ 5.0 mm (0, 1–4, 5–8, >8 sites) and the proportional distributions of PD $\geqslant$ 5.0 mm are presented (Figs 1 and 2). One-way ANOVA (Bonferroni post hoc test) failed to demonstrate smoking group differences in the distribution of PD $\geqslant$ 5.0 mm ($p = 0.49$), the proportional distribution of sites with PD $\geqslant$ 5.0 mm ($p = 0.34$), or the distribution of sites with a CAL $\geqslant$ 4.0 mm ($p = 0.34$). The proportional distribution of CAL $\geqslant$ 4.0 mm, however, differed significantly by smoking status, with a mean difference of 12.1% between NS and CS groups (mean difference: 12.1%, 95% confidence interval (CI): 1.5–22.6, $p<0.02$). Differences were also found between CS and FS groups (mean difference 13.2%, 95%CI: 2.0–24.2, $p<0.01$), but the difference between NS and FS groups was marginal (mean difference 0.3, $p<1.0$) (Fig. 3). The association between the proportions of sites with CAL $\geqslant$ 4.0 mm in relation to the estimated number of years of smoking is illustrated in a scatterplot diagram demonstrating r^2 values of 0.0.5 and 0.07 (Fig. 4).

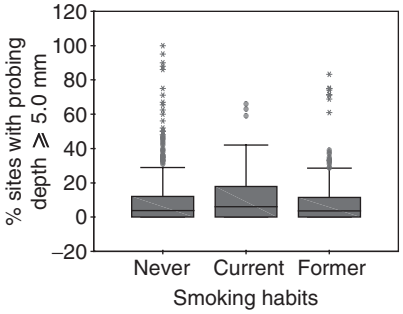

Fig. 1. Boxplot diagram illustrating the proportional distribution of sites with probing depth (PD) $\geqslant$ 5.0 mm in older subjects who; had never smoked, in current smokers, and in former smokers. (* = outliers, ■ = extreme outlier values).

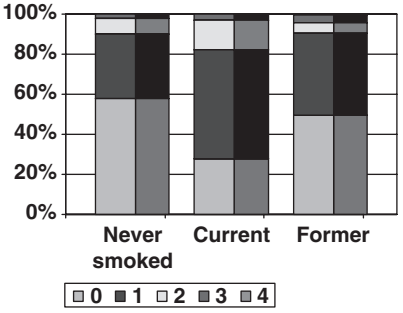

Fig. 2. Distribution of sites with probing depth (PD) $\geqslant$ 5.0 mm categorized in five categories from no evidence of such probing depths to having more than eight such sites in older subjects with no smoking habit, current smokers, and former smokers. (0 = none to two sites, 1 = three to four sites, 2 = five to six sites, 3 = seven to eight sites, 4 > eight sites).

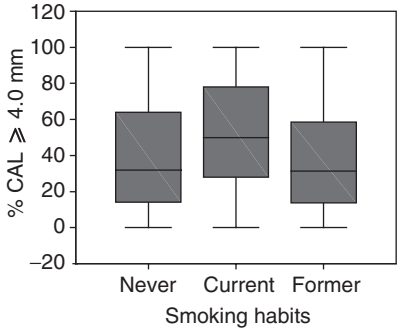

Fig. 3. Proportional distribution of sites with clinical attachment level loss $\geqslant$ 4.0 mm in older subjects who had never smoked, in current smokers, and in former smokers.

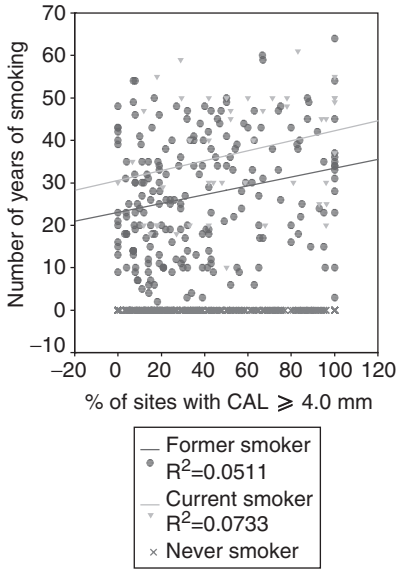

Fig. 4. Scatterplot diagram demonstrating the relationship between years of smoking and the proportional distribution of sites with clinical attachment loss $\geqslant$ 4.0 mm. (Notice that each dot may represent several cases.)

Radiographic evidence of bone loss

The extent of alveolar bone loss differed significantly by smoking status ($p<0.001$, Kruskal–Wallis ANOVA). Thus, 56.9% of the subjects in the NS group had no radiographic evidence of alveolar bone loss. In the FS group 48.8% had no radiographic evidence of alveolar bone loss whereas only 28.8% of the subjects in the CS group had no radiographic evidence of alveolar bone. Specifically, subjects in the CS and FS groups had significantly more evidence of both horizontal and vertical site specific alveolar bone loss than those in the NS group ($p<0.001$) and ($p<0.01$), respectively. Statistical analysis, however, failed to demonstrate a difference in the extent of vertical defects between NS and FS groups ($p = 0.60$). Thus 67.1% of subjects in the NS group and 69.3% of subjects in the FS group had no evidence of vertical defects whereas 54.5% of subjects in the CS group had no evidence of vertical defects. A dose–response relationship between the number of years of smoking and alveolar bone loss is presented suggesting that it might require 30 years or more of smoking to result in clinically relevant effects (Fig. 5).

Assessment of the likelihood of smoking as a predictor of increased risk for periodontitis

The odds ratio of having alveolar bone loss and a history of smoking was assessed by combining the two groups

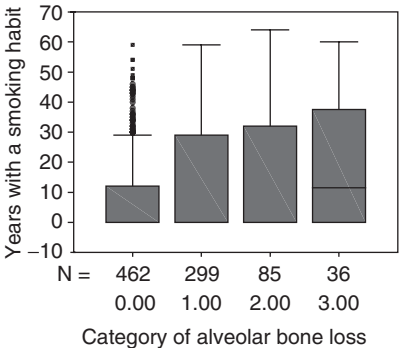

Fig. 5. Boxplot diagram representing the extent of horizontal alveolar bone loss in relation to years of smoking. •, outlier values; ■, extreme outlier values. (Definition of the extent of bone loss see Persson et al. 2002a, b). Notice that the median value for the number of years of smoking is ''0'' for all but the bone loss category ''3''.

Table 1. Results from logistic binary forward (Wald) regression analysis demonstrating that radiographic evidence of carotid calcification, smoking status (CS), gender (male) and the number of remaining teeth were variables explanatory to the presence of alveolar bone loss

	B	SE	Wald	Df	Sig.	Exp(*B*)
Evidence of carotid calcification	1.440	0.628	5.257	1	0.022	4.222
Smoking status	0.624	0.253	6.098	1	0.014	1.867
Number of remaining teeth	−0.111	0.019	32.767	1	0.000	0.895

of smokers and defining periodontitis as having evidence of horizontal alveolar bone loss >20%. The Mantel–Haenszel common odds ratio between smoking and periodontitis was 1.3 ($p<0.09$, 95% CI: 0.9–2.0 with $n=992$). Smoking for 30 years or more (current or former) changed the odds ratio of having periodontitis to 1.8 ($p<0.02$, 95% CI: 1.3–2.7).

Factors that demonstrated a significant correlation (Spearman rank correlation) with smoking status were included in binary and linear regression analysis. Binary logistic regression analysis demonstrated that using the dichotomous registration of bone loss as the dependent variable the following factors were predictable of bone loss; (1) radiographic evidence of carotid calcification, (2) smoking status (current smoker), (3) gender (male), and (4) the number of remaining teeth (Table 1).

Discussion

One of the shortcomings of the present study as in many other studies on the role of smoking, might be that subjects were asked about their smoking habits and these self-reports could not be objectively confirmed. However, while collecting smoking history information from the subjects there was very little to no hesitation among the subjects when asked about their smoking history. Collection of serum to assess cotinine levels would not be useful in that it would only be accurate for current smokers and not representative for former smokers. The same approach to collect information on smoking habits via a questionnaire has been used in most other studies. Validation studies, however, have shown that subject information on smoking habits is in agreement with analysis of cotinine levels (Rebagliato 2002).

In 1985, US and Canadian statistics reported that 19.7% of subjects 65 years and older were current smokers (National Center for Health Statistics et al. 1988). Between 1965 and 1990, the prevalence of smoking in the United States declined by 40%, but has since then remained virtually unchanged. In 1999 among the age group >65 years prevalence rates had been reduced to 10.6% (CDC html: www.doc.gov/mmmr/preview/mmwrhtml/mm5040al.htm). Thus, the findings in the present study that 7.9% of the subjects reported a current smoking habit and that 32% had quit smoking were consistent with national findings, suggesting that the study sample was representative of the current older US population.

Many studies on smoking and periodontitis have recruited subjects from dental school populations or otherwise health care-seeking subjects; this may have biased study conditions and been over-representative of subjects with periodontitis. Few studies, if any, have focused on the role of smoking on periodontal conditions in a general population of older subjects. In the present study, subjects were recruited from other sources and efforts were made to enroll an ethnically representative population (MacEntee et al. 2002).

Another shortcoming of the present study was the cross-sectional nature of data on smoking. However, a masked randomized longitudinal clinical trial on the impact of smoking on periodontal conditions would be ethically unacceptable. It would therefore be impossible to conduct a prospective study to ascertain the dose response effect of smoking on periodontal conditions. In the present study of older subjects, it was possible to enroll a cohort of subjects who had been smoking for many years. The study participants also represented a generation of adults who not until lately had access to information on smoking cessation and health benefits of not smoking.

In the present study of clinical periodontal measurements, only the proportion of sites with CAL$\geqslant$4.0 mm was higher in the CS group than in the NS or FS groups. The limited clinically relevant effects of smoking was further demonstrated in the analysis suggesting that it required approximately 30 or more years of smoking to have a significant impact on alveolar bone loss. Studies have suggested that gingival recession in older people may be common and not necessarily the effect of periodontitis. Furthermore, the small difference in the number of remaining teeth between groups cannot be taken as evidence that prior tooth loss may have masked the impact of smoking.

Rather, the present study suggested that a combination of factors including signs of atherosclerosis, being of male gender, older, current smoker for many years, and having previously lost several teeth are among conditions associated with alveolar bone loss in older subjects. Thus several co-risk factors in addition to longtime smoking may be required for periodontitis. Significant issues on the potential mechanism and impact on the periodontium from smoking remain unclear and must be researched. This could include studies on the impact of nicotine on capillary blood flow, on host-immunity, on the development of a pathogenic biofilm and to other attributed risks such as genetic predisposition, socio-economic and socio-behavioural factors in addition to cigarette consumption and impact of nicotine concentrations.

Whereas clinical measures of PD and CAL reflect current presence of periodontitis, radiographic evidence of alveolar bone loss reflects the long-term effect caused by all factors on alveolar bone. However, the analysis of risk of bone loss as an effect of smoking failed to identify smoking as a clinically significant risk. In fact, the current data suggested that it might take a minimum of about 30 years of smoking before smoking becomes a relevant clinical risk for periodontitis. Whether tooth loss in these older adults had occurred because of caries, periodontitis, prosthetic, or other reasons would have been impossible to evaluate. However the difference in the mean number of remaining teeth, although statistically significant, was clinically of limited importance with a mean difference of two teeth at the age of 60 or more.

A smoking habit has been associated with an increased risk for cardiovascular diseases in several studies. Studies have, however, also shown that former smokers who quit for 9 years or more had an

odds ratio of 1.1 to develop clinical evidence of cardiovascular disease in contrast with an odds ratio of 2.2 in current heavy smokers (Tavani et al. 2001). It is of interest that the odds that a current/former smoker had periodontitis as reported here was similar to that of being at risk for cardiovascular disease. Further details of the associations between periodontitis and cardiovascular disease and the impact of smoking in the present study population have been reported elsewhere (Persson et al. 2002a, b). The confounding effect of smoking on the association between cardiovascular disease and periodontitis may require studies other than epidemiological studies.

Quitting smoking has been associated with a substantial reduction in risk of all-cause mortality among patients and specifically for coronary heart diseases (Critchley & Capewell 2004). Whether quitting smoking would decrease future tooth loss similar to decrease in cardiovascular disease morbidity remains to be studied (Burns 2000, Suskin et al. 2001).

The present study demonstrated that having evidence of carotid calcification was another primary condition associated with smoking. Several studies have associated smoking with mortality by cardiovascular disease in older subjects (i.e. LaCroix et al. 1991). A smoking habit has been associated with osteoporosis, especially in older women (Law & Hackshaw 1997). In terms of periodontal conditions the data suggested that a large number of years of smoking is needed to cause measurable effects on periodontal status in older dentate subjects.

In conclusion, the study demonstrated that it may take a minimum of 30 years of smoking or 50% of a current adult lifespan, and continuing smoking before smoking has an impact on periodontal conditions (alveolar bone loss). Non-smoking older subjects perceive their risks for periodontitis as lower than do current/past smokers. A profile of having radiographic evidence of atherosclerosis, previous tooth loss, being male and a current older smoker is associated with periodontitis defined as alveolar bone loss.

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