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Long-term effect of smoking on vertical periodontal bone loss

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Abstract

Objectives: The objective of the present study was to investigate the influence of smoking on vertical periodontal bone loss over 10 years.

Material and Methods: The study base consisted of a population that was examined on two occasions with a 10-year interval, including 91 individuals, 24 smokers, 24 former smokers, and 43 non-smokers. The assessment of vertical bone loss was based on full sets of intra-oral radiographs from both time points. The severity of vertical bone loss was expressed as the proportion of proximal sites with vertical defects per person

Results: The 10-year increase in the proportion of vertical defects was statistically significant in all groups ($p < 0.001$) and, in addition, significantly associated with smoking ($p < 0.05$). In particular, the difference between smokers and non-smokers was significant ($p < 0.01$) whereas former smokers did not differ from non-smokers. Moreover, the 10-year vertical bone loss was significantly greater in heavy exposure smokers than in light exposure smokers suggesting an exposure-response effect ($p < 0.01$). Compared with non-smokers the unadjusted 10-year relative risk was 2.3-fold increased in light exposure smokers and 5.3-fold increased in heavy exposure smokers ($p < 0.05$).

Conclusions: The present observations indicate a significant long-term influence of smoking on vertical periodontal bone loss, yielding additional evidence that smoking is a risk factor for periodontal bone loss.

Key words: angular bone loss; periodontal disease; radiography; smoking; vertical defects

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Smoking is associated with increased prevalence and severity of destructive periodontal disease in terms of periodontal pocketing, periodontal bone loss, and tooth loss (Bergström & Flodérus-Myrhed 1983, Feldman et al. 1983, Ismail et al. 1983, Preber & Bergström 1986, Haber & Kent 1992, Holm 1994, Bergström et al. 2000a, b, Jansson & Lavstedt 2002, Bergström 2003, 2004, Khader et al. 2003). The smoking destructive effect on periodontal bone is of even ''horizontal'' (Feldman et al. 1987, Bolin et al. 1993, Norderyd et al. 1999, Payne et al. 2000, Bergström et al. 2000b, Jansson & Lavstedt 2002) and vertical ''angular'' pattern (Persson et al. 1998b, Baljoon et al. 2004). Radiographically, a vertical defect is characterized by an asymmetrical destruction around the tooth and the alveolar crest is not parallel to the level connecting the cemento-enamel junctions of adjacent teeth but the base of the defect is located apically to the alveolar crest (Pepelassi et al. 2000, Carranza 2002). Vertical bone loss has been associated with further periodontal bone loss and tooth loss and, therefore, the early detection of this phenomenon is considered clinically important (Papapanou & Wennström 1991).

The occurrence of vertical bone loss as estimated from cross-sectional studies varies from 23% to 61% in patients seeking dental care (Nielsen et al. 1980, Persson et al. 1998b) and from 30% to 40% in population studies (Wouters et al. 1989, Baljoon et al. 2003). The mechanism through which tobacco smoking deleteriously affects the periodontium remains obscure. We have earlier in a cross-sectional study reported on the association between smoking and prevalence as well as severity of vertical periodontal bone loss. The results suggested smoking to be a potential risk factor for this phenomenon (Baljoon et al. 2004). The present study, therefore, aimed to investigate the influence of smoking on vertical bone loss in a prospective study over 10 years.

Material and Methods

Study cohort

In 1982, a comprehensive periodontal health study was carried out in 249 professional musicians in Stockholm, Sweden (Bergström & Eliasson 1985). Ten years later, a new study of the same population was performed (Eliasson & Bergström 1997). The present study is based on 101 individuals who took part in both studies and for whom a com-

plete radiographic examination from both occasions was available. Because of incomplete smoking data or changed smoking habits, 10 individuals were excluded leaving 91 individuals to be accounted for. They form a prospective cohort including 24 individuals who were smokers in 1982 (baseline) and had continued smoking over the 10-year period (smokers), 24 individuals who had quit smoking already before the commencement of the baseline investigation and not taken up smoking again (former smokers), and 43 individuals who denied smoking both at baseline and follow-up (non-smokers). The distribution of the present cohort at baseline according to age and smoking is presented in Table 1. The smoking exposure was expressed in terms of life-time exposure, i.e., the accumulated exposure over time as formed by the product of daily consumption and years of duration (''cigarette years''). The smoking exposure characteristics are presented in Table 2.

The study was approved by the local ethical committee of Karolinska Institutet at the Karolinska University Hospital, Stockholm, in accordance with the Helsinki Declaration of 1975 and as revised in 1983.

Radiographic assessment

All individuals were examined at baseline and follow-up by means of a complete set of intra-oral radiographs including 16 periapical and four bitewing projections. The modified parallel long-distance technique was used. The film was placed in a film holder as parallel as possible to the long axis of the teeth. The X-ray machines used operating at 65–70 kVp, were equipped with a rectangular tube giving at least 20 cm target-to-skin distance. Kodak Ekta Speed film (speed group E, Eastman Kodak Company, Rochester, NY, USA) was used. The radiographic assessment of vertical defects was performed by one or other of two observers (M. B. and S. N.) under $\times 2$ magnification using a Mattsson viewer and a light table with good illumination. A vertical bone loss was defined as a resorption of the inter-dental marginal bone of at least 2 mm that had a typical angulation towards either the mesial or distal aspect of the root (Baljoon et al. 2003). All teeth except third molars were assessed as to the presence or absence of a vertical defect. However, if a first or second molar was missing the third molar of the same quadrant if normally erupted was included. Altogether, 9464 sites were examined. Out of these, 205 sites (2%) were unreadable. The prevalence of vertical bone loss was estimated from the number of individuals exhibiting one or more vertical defects. The 10-year cumulative incidence was estimated from the proportion of individuals who became affected over time. The term severity of vertical bone loss was used to describe the frequency of sites with a vertical defect in relation to the frequency of sites measured in the individual. The severity was expressed as % per person. Radiographic assessments were performed blinded with reference to the smoking status of the individual. Furthermore, the radiographic data from baseline and 10-year follow-up were assessed independently. When an examiner was in doubt the final decision was taken after a consensus of all authors.

The inter-dental bone height at baseline was measured from the same set of intra-oral radiographs. The bone height was measured mesially and distally to all teeth and expressed as % of the root length (Bergström & Eliasson 1986).

Clinical data

The inflammatory condition of the gingiva was evaluated according to the gingival index method of Löe & Silness (1963) and supragingival dental plaque was scored following the plaque index system of Silness & Löe (1964). All teeth in the individual were examined and four sites per tooth (buccal, mesial, distal, lingual) were given a score. Pocket probing depth of the above sites of all teeth was measured with a 2 mm graduated probe, and the periodontal clinical condition of the individual was expressed as the mean probing depth or the frequency of sites with a probing depth of 4 mm or more (Bergström & Eliasson 1986).

Error of measurement

The inter-examiner reliability with respect to vertical bone defect measurements was estimated from 30 randomly selected individuals (representing 1584 sites) using Cohen's κ statistic according to the formula

$$\kappa = \frac{A_o - A_c}{1 - A_c},$$

where A_o is the proportion of agreements that was actually observed and A_c the proportion of agreements that could be expected by chance (Cohen 1960). The inter-examiner reliability found was $\kappa = 0.89$ indicating ''perfect'' agreement (Landis & Koch 1977). It is concluded that the error related to inter-examiner variability of assessments did not substantially influence the outcome.

Statistical analysis

The proportion of proximal sites with vertical defects per person was presented as means and 95% confidence intervals (CIs). This variable was non-normally distributed and, therefore, primarily tested with Kruskal–Wallis ANOVA. Additional statistical analysis was performed by means of one-factor ANOVA, including *post hoc* multiple comparisons testing according to Scheffe. Also the 10-year differences in the number of teeth and proportion of vertical defects were non-normally distributed and, therefore, tested with Friedman ANOVA. Additionally, one- or two-factor repeated-measures ANOVA was applied. In two-factor analyses, age at baseline (two groups), plaque level at baseline (three levels), and vertical defects at baseline (presence/absence), respectively, were introduced as co-factors. Prevalence differences were tested with the chi-squared test. Multiple linear regression analysis was

Table 1. Study cohort at baseline by age and smoking

Age	Smokers		Former smokers		Non-smokers		Total	
	n	%	*n*	%	*n*	%	*n*	%
20–40	10	42	6	25	24	56	40	44
41–60	14	58	18	75	19	44	51	56
Total	24	100	24	100	43	100	91	100
Mean	49.5		55.5		49.2		51.0	
95% CI	45.8; 53.3		52.5; 58.5		46.2; 52.2		49.1; 52.9	

CI, confidence interval.

Table 2. Study cohort according to smoking exposure at baseline and follow-up

Smoking exposure	Baseline					Follow-up	
	n	consumption (cigarette/day), mean (95% CI)	duration (years), mean (95% CI)	life-time exposure (cigarette years), mean (95% CI)	ex-time* (years), mean (95% CI)	consumption (cigarette/day), mean (95% CI)	duration (years), mean (95% CI)	life-time exposure (cigarette years), mean (95% CI)
Smokers								
Light	12	10.5 (7.2; 13.8)	11.9 (6.8; 17.0)	125.0 (63.5; 174.8)	–	11.5 (5.2; 17.8)	19.5 (9.8; 29.2)	239.3 (81.6; 396.9)
Heavy	12	17.3 (14.6; 20.0)	27.9 (22.4; 33.5)	476.7 (350.7; 602.6)	–	17.1 (13.6; 20.6)	38.5 (32.5; 44.6)	661.3 (495.7; 826.8)
Total	24	13.9 (11.4; 16.3)	19.9 (15.0; 24.8)	297.9 (198.2; 397.7)	–	13.6 (10.0; 17.2)	27.9 (20.7; 35.0)	424.9 (272.3; 577.6)
Former								
Light	15	13.6 (9.2; 18.0)	9.7 (6.0; 13.4)	112.9 (76.0; 149.8)	11.5 (6.0; 16.9)	–	–	–
Heavy	9	21.3 (14.6; 27.9)	24.0 (17.7; 30.3)	470.6 (367.1; 574.1)	12.5 (7.9; 19.5)	–	–	–
Total	24	15.6 (11.8; 19.4)	14.0 (9.8; 18.3)	227.5 (144.3; 310.6)	11.8 (7.3; 16.3)	–	–	–

*Number of years since giving up smoking.
CI, confidence interval; *n*, number of individuals.

run with the 10-year difference in proportion of vertical defects as the dependent variable. Logistic regression was used to estimate the relative risk expressed as odds ratio (OR) and 95% CI (OR and 95% CI). The 10-year difference in the number of vertical defects was used as the dependent variable, dichotomized ($\geqslant 2 = 1$, else $= 0$). In the logistic regression analyses, age at baseline was stratified according to (1) 20–40 years ($n = 40$) and (2) 41–60 years ($n = 51$); life-time exposure at baseline into (1) no exposure ($n = 43$), (2) light exposure < 250 cigarette years (mean 115.7 cigarette year, $n = 27$; 12 smokers, 15 former smokers), and (3) heavy exposure $\geqslant 250$ cigarette years (mean 474.3 cigarette years, $n = 21$; 12 smokers, nine former smokers); bone height at baseline into (1) low (63.9–80.2%, $n = 24$), (2) medium (80.3–85.7%, $n = 39$), and (3) high (85.8–93.0%, $n = 28$); vertical defects at baseline into (1) absence (2) presence. Pairwise correlations were carried out by means of Pearson's product moment method. The data were analyzed using the STATISTICA (6.0) program. Statistical significance was accepted at $p < 0.05$.

Results

Dental awareness and oral hygiene condition

The vast majority (94%) claimed to visit a dentist on a regular basis at least once every 2 years, and 87% that they regularly brushed their teeth twice daily or more. This dental behaviour held true for the total population investigated at baseline as well as for the prospective cohort. The same standard of dental awareness was consistent in all three smoking groups.

The mean (95% CI) plaque index at baseline was 1.1 (0.9; 1.2), 0.9 (0.7; 1.0); and 0.7 (0.6; 0.8) in smokers, former smokers, and non-smokers, respectively; and 0.8 (0.6; 1.0); 0.8 (0.6; 0.9); and 0.7 (0.6; 0.8), respectively, at 10-year follow-up. The differences between smoking groups were not statistically significant (Kruskal–Wallis $H = 3.2$ and 1.5, respectively, $p > 0.05$). The change in plaque index over the 10 years was not statistically significant neither within nor between smoking groups (Friedman's ANOVA, $p > 0.05$; Kruskal–Wallis ANOVA, $p > 0.05$).

Table 3. Number of teeth and vertical bone defects (VD) at baseline and follow-up and number of teeth lost to follow-up

Smoking habit	Teeth lost, *N*	Teeth at baseline, mean	Teeth at follow-up, mean	10-year loss, mean	VD at baseline, *N*	VD at follow-up, *N*	VD per tooth at baseline, mean	VD per tooth at follow-up, mean	Relative increase	VD 10-year change, mean
Smoker										
Loser ($n = 5$)	9	27.4	25.6	1.8	10	14	0.073	0.109	1.5	0.036
Non-loser ($n = 19$)	0	27.2	27.2	0	13	48	0.025	0.093	3.7	0.068
Former smoker										
Loser ($n = 11$)	23	26.0	23.9	2.1	10	18	0.035	0.069	2.0	0.034
Non-loser ($n = 13$)	0	27.5	27.5	0	2	19	0.006	0.053	8.8	0.047
Non-smoker										
Loser ($n = 13$)	23	25.5	23.7	1.8	10	15	0.030	0.049	1.6	0.019
Non-loser ($n = 30$)	0	27.5	27.5	0	3	32	0.004	0.039	9.7	0.035
Total										
Loser ($n = 29$)	55	26.0	24.1	1.9	30	47	0.040	0.067	1.7	0.027
Non-loser ($n = 62$)	0	27.4	27.4	0	18	99	0.011	0.058	5.3	0.047

Means in loser and non-loser individuals according to smoking. *n*, number of individuals; *N*, number of teeth or vertical defects.

Periodontal bone height and pocket probing depth

The mean periodontal bone height at baseline was 80.3%, 80.7%, and 85.1% in smokers, former smokers, and non-smokers, respectively. The corresponding means at 10-year follow-up were 78.0%, 80.0%, and 84.2%, respectively. The 10-year difference between smokers and non-smokers controlling for age and baseline bone height level was statistically significant ($F = 5.3$, $p < 0.01$).

The mean pocket probing depth at baseline was 2.2, 2.1, and 2.0 mm in smokers, former smokers, and non-smokers, respectively. The corresponding means at 10-year follow-up were 2.9, 2.2, and 2.2 mm, respectively. The 10-year difference between smokers and non-smokers controlling for age and baseline probing depth was statistically significant ($F = 6.3$, $p < 0.01$).

Influence of 10-year tooth loss on vertical defects

The mean (95% CI) frequency of retained teeth at baseline was 27.3 (26.1; 28.0), 26.8 (25.2; 27.9), and 26.9 (24.7; 27.8) for smokers, former smokers, and non-smokers, respectively. The corresponding mean (95% CI) frequencies at follow-up were 26.9 (25.5; 27.3), 25.8 (23.3; 27.3) and 26.3 (23.7; 27.9), respectively. There were no statistically significant differences between smoking groups ($p > 0.05$). The decrease in the number of teeth over the 10 years was statistically significant in all three smoking groups (Friedman's ANOVA, chi-squared = 4.2, 3.7, and 3.1, respectively, $p < 0.05$). The 10-year decrease was not significantly associated with smoking (repeated-measures ANOVA, $F = 0.9$, $p > 0.05$).

The mean (95% CI) number of teeth at baseline and follow-up among 29 individuals who lost teeth to follow-up was 26.0 (23.3; 27.7) and 24.1 (21.7; 26.5), respectively, as against 27.4 (26.3; 27.9) among 62 individuals who did not lose teeth. As further described in Table 3, the 29 loser individuals lost a total of 55 teeth or on average 1.9 teeth per individual. Five smokers lost nine teeth affected by six vertical defects, 11 former smokers lost 23 teeth affected by two vertical defects, and 13 non-smokers lost 23 teeth affected by seven vertical defects. The mean (95% CI) number of vertical defects at baseline was 1.0 (0.06; 2.6) in individuals who lost teeth over 10 years (2.0, 0.9, and 0.8 in smokers, former smokers, and non-smokers, respectively) compared with 0.3 (0.6; 1.2) in individuals who did not lose any teeth (0.7 in smokers, 0.2 in former smokers, and 0.1 in non-smokers). The difference between loser and non-loser individuals was statistically significant (Kruskal–Wallis, $H = 8.1$, $p < 0.01$).

The increase in the number of vertical defects over the 10-year period was greater in non-loser than loser individuals. The increase was 1.5, 2.0, and 1.6-fold in smokers, former smokers, and non-smokers, respectively, who lost teeth to follow-up compared with 3.7, 8.8, and 9.7-fold, respectively, in smokers, former smokers, and non-smokers who did not lose teeth to follow-up (Table 3). Individuals who lost teeth were significantly older ($p < 0.001$) and had an inferior bone height level at baseline ($p < 0.001$). There was an interaction effect of smoking and bone height on lost teeth ($p < 0.05$).

In the following presentation of changes in vertical bone loss only teeth that were present both at baseline and follow-up were considered.

Incidence

Including teeth lost to follow-up the overall prevalence of individuals with one or more vertical defects at baseline was 34% (38% in smokers, 33% in former smokers, and 21% in non-smokes). Excluding teeth lost to follow-up the prevalence at baseline was 21% (33% in smokers, 25% in former smokers, and 12% in non-smokers, Table 4). The prevalence at follow-up was 55% (67% in smokers, 55% in former smokers, and 49% in non-smokers). Although the prevalence estimates were throughout greater in smokers, the differences between smoking groups were not statistically significant ($p > 0.05$).

Excluding teeth lost to follow-up, the 10-year cumulative incidence was 50% in smokers, 39% in former smokers, and 42% in non-smokers (Table 4). The 10-year cumulative incidence of individuals who became affected by one or more vertical defects was not significantly different between smoking groups (chi-squared = 1.3, $p > 0.05$). In addition, the 10-year cumulative incidence was 58% in age group 20–40 years and 29% in age group 41–60 years. The difference between age groups was significant (chi-squared = 9.2, $p < 0.01$).

Table 4. Frequency of individuals with ''affected'' or without ''unaffected'' vertical defects at baseline and follow-up, and 10-year cumulative incidence

Smoking habit	Baseline		Follow-up		10-year cumulative incidence (%)
	non-affected (*n*)	affected (*n*)	non-affected* (*n*)	affected* (*n*)	
Smoker	16	8	8	8	50
Former smoker	18	6	11	7	39
Non-smokers	38	5	22	16	42
Total	72	19	41	31	43

Estimates according to smoking after exclusion of teeth lost to follow-up.
*Out of non-affected at baseline.
n, number of individuals.

Table 5. Proportion (%) of vertical bone defects at baseline and 10-year follow-up

Smoking habit	Baseline, mean (95% CI)	Follow-up, mean (95% CI)	*F*	*p*
Smoker	1.3 (0.2; 2.4)*	4.5 (1.8; 7.1)*	3.5	< 0.001
Former smoker	0.9 (0.0; 1.8)	2.9 (1.3; 4.5)	3.2	< 0.01
Non-smoker	0.3 (0.0; 0.6)	1.7 (1.0; 2.5)	4.1	< 0.001
Total	0.7 (0.3; 1.1)	2.8 (1.9; 3.6)	3.6	< 0.01

Mean and 95% CI according to smoking.
*Significantly different from non-smokers ($p<0.05$).
CI, confidence interval.

Severity

Excluding teeth lost to follow-up the mean (95% CI) proportion of vertical defects per person at baseline was 1.3% (0.2; 2.4) in smokers, 0.9% (0.0; 1.8) in former smokers, and 0.3% (0.0; 0.6) in non-smokers. The association between smoking and the proportion of vertical defects at baseline was almost statistically significant (Kruskal–Wallis, $H = 2.7$, $p = 0.071$). The *post hoc* difference between smokers and non-smokers using one-factor ANOVA and Scheffe's test was statically significant ($p<0.05$).

The 10-year change in the proportion of vertical defects implied a significant increase in all smoking groups (Friedman's ANOVA, chi-squared = 13.0 in smokers; chi-squared = 12.0 in former smokers; and chi-squared = 22.0 in non-smokers, respectively, $p<0.001$). The increase was significantly associated with smoking (repeated-measures ANOVA, $F = 3.7$, $p<0.05$, Table 5). The *post hoc* difference between smokers and non-smokers was statistically significant (Scheffe's test, $p<0.01$). Controlling for baseline condition of vertical defects the significance was attenuated ($F = 3.0$, $p = 0.052$). The *post hoc* difference between smokers and non-smokers, however, remained significant (Scheffe's test, $p<0.01$). The same held true controlling for age or plaque level at baseline ($F = 3.0$, $p = 0.053$ and $F = 2.9$, $p = 0.060$, respectively, Scheffe's test, $p<0.01$).

The effect of life-time smoking exposure at baseline on the 10-year change in the proportion of vertical defects was statistically significant (repeated-measures ANOVA, $F = 5.6$, $p<0.001$) with a significant *post hoc* difference between heavy and light exposure smokers (Scheffe's test, $p<0.01$) but not between light exposure smokers and non-smokers. The same held true controlling for baseline condition of vertical bone defects or plaque ($F = 3.5$ and 7.3, respectively, $p<0.05$, Scheffe's test, $p<0.01$). Controlling for age, the overall significance was lost ($F = 2.4$, $p>0.05$), but the *post hoc* difference between heavy and light exposure smokers remained significant (Scheffe's test, $p<0.01$). The life-time exposure effect became stronger as the analysis was restricted to smokers (repeated-measures ANOVA, $F = 8.1$, $p<0.001$, Fig. 1a) and disappeared as run in former smokers (repeated-measures ANOVA, $F = 1.7$, $p>0.05$, Fig. 1b).

Multiple linear regression and risk assessment

The 10-year change in the proportion of vertical defects as the dependent variable could be predicted using multiple linear regression from the variables age, life-time exposure at baseline, number of teeth at baseline, bone height at baseline, number of pockets at baseline, gingival index at baseline, and plaque index at baseline as predictors entered in one block. The variables explained 19% of the variance in the dependent variable (R^2 (adj) = 0.19, F (7, 84) = 3.5, $p<0.01$, Table 6). The strongest predictors were life-time exposure and number of teeth at baseline. Also in a forward stepwise approach, life-time exposure and number of teeth at baseline turned out the only significant factors (R^2 (adj) = 0.20, F (4, 88) = 6.0, $p<0.001$).

In smokers, the bivariate correlation between life-time exposure and 10-year vertical periodontal bone loss was statistically significant using the correlation method of Pearson's ($r = 0.57$, $p<0.01$, Fig. 2).

Logistic regression analysis was run to estimate the relative risk of vertical bone loss associated with life-time smoking exposure. The relative risk of smokers and former smokers combined compared with non-smokers was 2.3-fold increased in light exposure smokers and 5.3-fold increased in heavy exposure smokers (OR = 2.3, 95% CI 1.1–4.9 and OR = 5.3, 95% CI 1.2–24.3, $p<0.05$). Adjustment for age, bone height at baseline, vertical defects at baseline, plaque level at baseline, or number of teeth at baseline did not substantially influence the relative risk estimates. The relative risk of light and heavy smokers was 2.4-fold (OR = 2.4, 95% CI 1.0–5.6) and 5.8-fold (OR = 5.8, 95% CI 1.1–13.2), respectively, increased compared with non-smokers. The relative risk was not significantly elevated neither in light nor heavy exposure former smokers when compared with non-smokers.

Discussion

The objective of the present study was to investigate the influence of smoking on vertical periodontal bone loss over 10 years. The observations suggested that vertical bone loss was influenced by smoking, since the 10-year severity increase was significantly greater in smokers compared with non-smokers. In addition, the vertical bone loss increased comparably more in heavy exposure smokers than in light exposure smokers indicating an exposure-response relation. On the other hand, former smokers who had given up

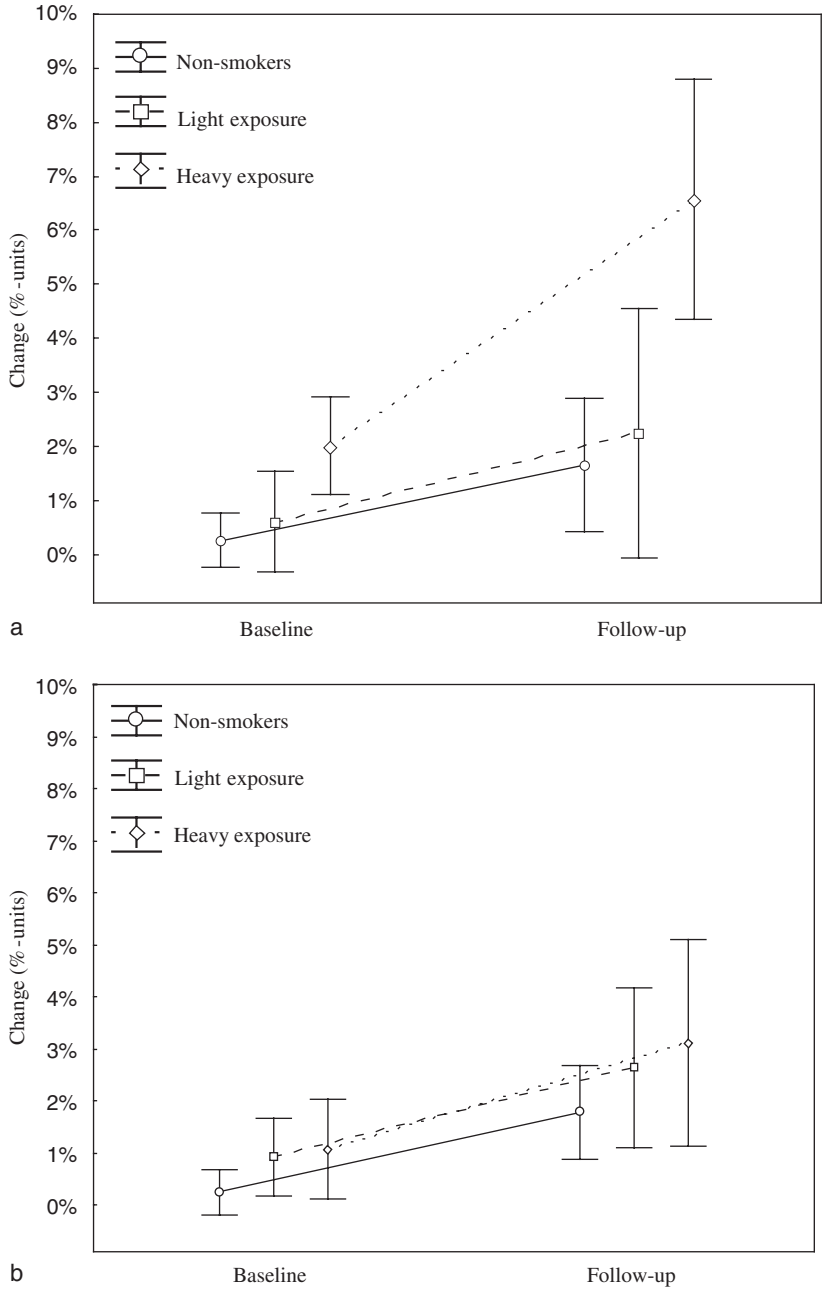

Fig. 1. (a) Ten-year change in proportion (%) of vertical bone defects. Mean and 95% confidence interval (CI) according to life-time exposure in smokers and with non-smokers as control. (b). Ten-year change in proportion (%) of vertical bone defects. Mean and 95% CI according to life-time exposure in former smokers and with non-smokers as control.

Table 6. Multiple regression analysis with 10-year change in the proportion of vertical defects as dependent variable

Variable	Parameter	Standard error	*t*	*p*
Age baseline	0.00034	0.00049	0.80	0.423
Plaque index baseline	0.00007	0.00018	0.60	0.547
Gingival index baseline	− 0.00005	0.00013	− 0.03	0.969
Bone height baseline	− 0.00091	0.00068	− 1.37	0.172
Number of pockets baseline	0.00028	0.00023	1.44	0.153
Number of teeth baseline	0.00407	0.00184	2.21	0.029
Life-time exposure baseline	0.00005	0.00002	2.67	0.009

Standard model (R^2 (adj) = 0.19).

smoking on average about 10 years prior to the study did not differ from non-smokers. To our knowledge, the present observations are the first ones to show an effect of smoking on long-term vertical periodontal bone loss. Our results confirm previous cross-sectional findings demonstrating an association between smoking and vertical bone loss (Persson et al. 1998b, Baljoon et al. 2004), and, furthermore, agree with previous long-term studies on smoking and periodontal bone loss in general (Feldman et al. 1987, Bolin et al. 1993, Norderyd et al. 1999, Payne et al. 2000, Bergström et al. 2000b, Jansson & Lavstedt 2002, Bergström 2004). The present observations also are consistent with ample documentation that smoking increases the rate of bone loss in other parts of the skeleton such as the radius, femoral neck, hip, and lumbar spine (Vogel et al. 1997, Krall & Dawson-Hughes 1999, Hannan et al. 2000, Tanaka et al. 2001, Naves et al. 2004).

The prevalence of vertical bone loss estimated at baseline was 34% before exclusion of teeth lost to follow-up. This was close to the prevalence found in the total population (Baljoon et al. 2003) suggesting that the present cohort was largely representative of the total population at baseline. The present population is considered dentally aware since most participants claimed to be regular dental attenders for many years and to exert daily tooth brushing (Bergström & Eliasson 1985). The generally high standard of oral hygiene was confirmed by low plaque levels observed both at baseline and follow-up. An advantage of utilizing a population of dentally aware individuals for the study of vertical bone loss is the circumstance that the number of retained teeth in such individuals is high. Most individuals of the present study had a minimum of 26 teeth at baseline and follow-up and the tooth mortality rate was low being on average 0.05 teeth per year during the 10-year period.

In spite of a low tooth mortality rate, there was a significant loss of teeth over the 10 years in all three smoking groups. Furthermore, tooth loss influenced the estimation of vertical defects since individuals who lost teeth developed fewer new defects than individuals who did not lose any teeth. Loss of teeth reduces the probability of attracting new vertical defects and at the same time increases the probability of losing

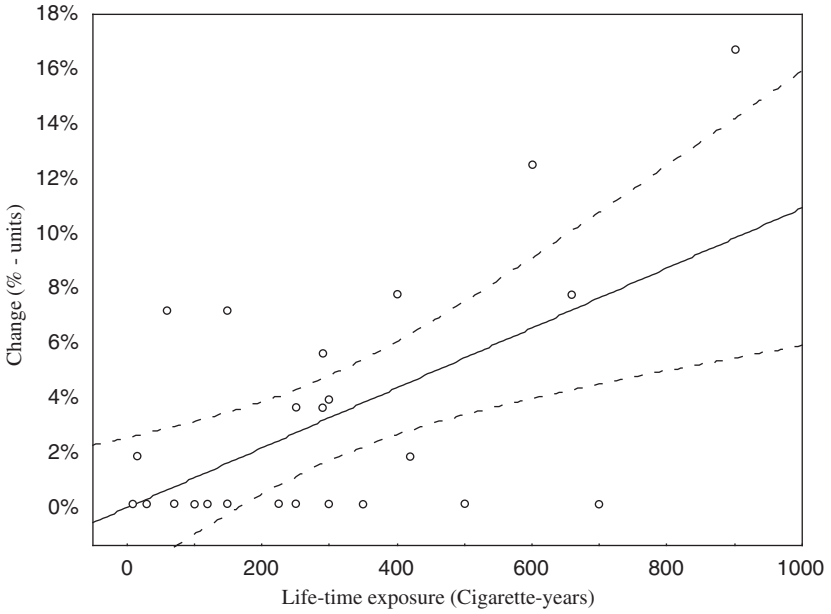

Fig. 2. Scatterplot of the relationship between 10-year change in proportion (%) of vertical bone defects and life-time exposure. Regression line and 95% confidence interval in smokers. $r = 0.57$, $p = 0.004$.

existing defects, thus resulting in an underestimation. Although loss of teeth was not significantly different in the smoking groups, the effect of tooth loss on vertical bone loss seemed to be greater in smokers since they were more affected at baseline. For the same reason, the 10-year relative increase in the number of vertical defects in individuals who did not lose teeth was comparably less pronounced in smokers (Table 3). This might have resulted in a comparably greater underestimation in smokers. The effect of tooth loss on estimates of vertical bone loss has to be considered in longitudinal studies.

In the present study, a vertical defect was defined as a resorption of the interdental marginal bone of at least 2 mm that had a typical angulation towards either the mesial or distal aspect of the root. No quantitative criteria of defect size or defect depth were applied in the assessment. Therefore, the severity of vertical bone loss was based on the horizontal extension within the dentition only whereas the depth dimension was not taken into account as has been done by others (Nielsen et al. 1980, Persson et al. 1998a). This limitation may have resulted in an underestimation of changes in severity over time.

It has been shown in earlier cross-sectional studies that prevalence as well as severity of vertical bone loss increase with increasing age (Nielsen et al. 1980, Wouters et al. 1989, Persson et al. 1998a, Baljoon et al. 2003). According to the present observations the effect of age seemed to be the result of an accumulation of vertical defects over time. This was evidenced by the observations that the 10-year cumulative incidence was relatively greater in young (20–40 years) compared with old (41–60 years) individuals. Furthermore, the 10-year severity increase, i.e. the vertical bone loss rate was not dependent of age. This seems to agree with some previous long-term observations suggesting that the bone loss rate is not influenced by age (Bergström 2004).

The 10-year increase in vertical periodontal bone loss depended on smoking exposure confirming our earlier cross-sectional observations suggesting that the vertical bone loss was more pronounced in comparably more exposed individuals (Baljoon et al. 2004). The effect of heavy exposure increased the 10-year risk for vertical bone loss by five to six times compared with non-smokers. Furthermore, the observation that former smokers who had given up smoking in the past exhibited a 10-year progression rate on a par with non-smokers suggests that an exposure decrease has a beneficial effect. The observation of an exposure response effect is important since it is commonly accepted that this strengthens the plausibility of a causal relationship between the risk factor and the dependent variable (Hill 1983, Rothman 1986). Although the risk estimates were uncertain as is seen from the comparably large CIs, the magnitude of the estimated risk is reasonable when compared with other studies on smoking associated periodontal bone loss (Norderyd & Hugoson 1998, Norderyd et al. 1999).

The biological mechanisms responsible for the effect of smoking on the periodontal tissues are still elusive. Several possibilities have been described, and both locally and systemically induced effects have been suggested. Cytotoxic substances such as nicotine and its major metabolic cotinine can be detected in the saliva, gingival cervicular fluid, serum, and urine demonstrating their systemic availability (McGuire et al. 1989). It is possible that the effect of nicotine is related to vascular changes, resulting in insufficient vascular supply and indirectly leads to bone tissue breakdown. There is experimental evidence to suggest that nicotine as well as cigarette smoke have detrimental effects on bone cells and osteoprogenitor cells (Liu et al. 2001, 2003, Walker et al. 2001, Akmal et al. 2004, Oda et al. 2004). It is further readily realized that several other agents in cigarette smoke may exert a toxic action on bone cell metabolism causing retardation or obstruction of regenerative functions. Such an action would lead to an imbalance between build-up and breakdown functions.

Based on present and previous long-term observations we argue that exposure to cigarette smoke exerts an effect on the periodontal bone such that the probability of being affected by severe bone loss, including vertical defects, is elevated in chronic smokers. Since smoking was antecedent to the effect observed, whereas the abolishment of smoking was related to no effect, the argument favours the contention that smoking is a cause of severe bone loss. We do not argue, however, that smoking is the only cause of such an event – nor that it necessarily is a sufficient one. It is highly likely that additional factors – usually referred to as component causes – are needed to form a sufficient cause which eventually can provoke the effect. This is synonymous to stating that for severe bone loss to occur in a smoker, the smoker has to be susceptible. Unfortunately, little is presently known bout what makes a smoker susceptible (Kocher et al. 2002). Other sets of component causes may exist that can cause sever bone loss. They have,

however, not been shown to possess the same strength as sets where smoking is a component cause.

The main limitations of the study are the limited size and the gender bias towards male predominance. These circumstances together with the fact that the dental awareness was above average may put some restraints on the generalization of the findings.

The influence of age, number of pockets, gingival index, and plaque index on 10-year vertical bone loss was marginal if any as found from the multiple regression analysis. Of the local or ''periodontal'' factors included in the analyses baseline condition of tooth frequency contributed most to the explanation of vertical bone loss. However, only about 20% of the variation in the dependent variable was explained by the factors studied, and, therefore, uncontrolled confounding might remain from other factors such as trauma from occlusion (Glickman & Smulow 1965) and deficient root cementum (Blomlöf et al. 1987).

In conclusion, the chief novelty of the present 10-year prospective study is that tobacco smoking exerts an effect on vertical periodontal bone loss. The observations offer additional evidence that smoking is a risk factor for periodontal bone loss.

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