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Influence of tobacco smoking on periodontal bone height. Long-term observations and a hypothesis

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Abstract

Aim: The aim of the investigation was to estimate the magnitude of the long-term influence of chronic smoking on the periodontal bone height.

Methods: The study population included 19 continuous smokers, 28 former smokers and 44 non-smokers in the age range 20–60 years at baseline. The participants were examined at two points in time with an interval of 10 years. The height of the periodontal bone was determined from bite-wing radiographs of the first and second premolars of the maxilla and the mandible and measured from the cemento-enamel junction (CEJ) to the periodontal bone crest (PBC) mesially and distally to the preselected teeth.

Results: The mean (SD) CEJ–PBC distance at baseline was 1.82 (1.01) mm for smokers, 1.65 (0.81) mm for former smokers, and 1.16 (0.59) mm for non-smokers ($p = 0.016$). The mean (SD) 10-year bone height reduction was 0.74 (0.59) mm for smokers as against 0.26 (0.31) mm for former smokers and 0.27 (0.29) mm for non-smokers. Controlling for age and baseline bone height level, the magnitude of the reduction was significantly dependent of smoking ($p = 0.000$). The widening gap between smokers and non-smokers over time suggested that the bone height reduction of smokers took place at an accelerated rate.

Conclusion: On the basis of the observations it is hypothesized that smoking induces an acceleration of the periodontal bone height reduction rate and that smoking cessation results in a return towards non-smoker rate.

Key words: bone height; bone loss; periodontal disease; smoking; tobacco

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During the past decade and a half, clinical and epidemiologic research have provided new insight into the harmful effects of tobacco smoking on the health of the periodontium. Most if not all studies utilizing a variety of measures or endpoints indicate that prevalence as well as severity of chronic periodontal disease are negatively influenced by smoking, from a modified gingival hemorrhagic response (Bergström & Flodérus-Myrhed 1983, Bergström & Preber 1985, Bergström & Boström 2001) to an elevated tooth mortality rate (Feldman et al. 1987, Holm 1994, Krall et al. 1999).

Radiographic assessment of periodontal bone height or bone loss is a reliable means of studying the periodontal health condition that has been used as a surrogate endpoint in some previous longitudinal studies on the relationship between smoking and periodontal disease (Feldman et al. 1987, Bolin et al. 1993, Machtei et al. 1997, Krall et al. 1999, Payne et al. 2000). These studies, although rather heterogeneous by design and length of follow-up term, indicate that smoking is associated with excess bone loss. Although consistent in general, these studies do not allow a closer estimation of the magnitude of the effect. Investigations that can provide quantitative estimates of the long-term effect of smoking, therefore, remain to be done.

We have previously reported on excess bone loss in smokers as compared to non-smokers and past smokers (Bergström et al. 2000). In an attempt to estimate the magnitude of the effect of chronic smoking in a long-term perspective, the present investigation was carried out with the object to determine the periodontal bone height changes related to the maxillary and mandibular

premolar regions in smokers, former smokers, and non-smokers over 10 years.

Material and Methods

Study population

The population to be studied was derived from two consecutive cross-sectional samples drawn from the same population with an interval of 10 years. The two samples included 264 and 258 individuals, respectively, the participation rate was 80% and 81%, respectively, and smoking prevalence was 33% and 20%, respectively. Those individuals who participated in both cross-sectional studies form the present prospective longitudinal cohort. From the aspect of smoking, the present cohort can be regarded as a random sample.

Originally, the cohort included 95 individuals who had been clinically and radiographically examined at the two points in time (baseline and 10-year follow-up). The participants were predominantly men in the age range 20–60 years at baseline, including 19 individuals who had regularly smoked cigarettes over the 10 years and still held the habit (smokers or continuous smokers), 28 individuals who had quit smoking before the commencement of the study (former smokers) and 44 individuals who claimed not to smoke and never to have used any tobacco products (non-smokers or never smokers). Another four individuals who had changed smoking behaviour during the interval between the two examinations were excluded, leaving a total of 91 individuals to be considered.

The mean consumption of smokers was 14.9 cig/day (range 10–20 cig/day) at baseline and 13.5 cig/day (range 2–20 cig/day) at follow-up, and the mean smoking duration of smokers at follow-up 30.3 years (range 10–50 years). The previous mean consumption of former smokers was 15.6 cig/day (range 5–40 cig/day), and the mean duration of previous smoking 15.8 years (range 3–32 years). Former smokers had stopped smoking an average of 10.9 years before baseline (range 1–25 years). The distribution of the present cohort according to age and smoking is presented in Table 1. The periodontal condition with reference to other clinical characteristics is demonstrated in Table 2. The study was approved by the local ethical committee of Karolinska Institutet at Huddinge University Hospital.

Measurements

Radiographs of all participants were obtained at the same examination occasion at baseline as well as follow-up using the same equipment. The radiographical examinations were performed by two specially trained assistants. Two bilateral pairs of bite-wing radiographs per person from each of the two examinations were available for measurement. The radiographs were of a high technical standard allowing a maximum of 16 determinations per person. The height of the interdental periodontal bone of the individual was determined from the original radiographs of the first and second premolars of the maxilla and the mandible. The distance from the cemento-enamel junction (CEJ) to the periodontal bone crest (PBC), i.e., the most coronal point of the interdental bone crest, was measured mesially and distally to each preselected tooth. Measurements were made under $\times 7$ magnification with the aid of a magnifying lens equipped with a 0.1 mm graduated scale. All single measurements were made to the nearest 0.1 mm. Measurements were made blinded as to the smoking status of the individual.

Comparison of baseline and follow-up means in the individual (case-means) was based (1) on determinations of all available sites in the individual at baseline and follow-up, respectively, and (2) on determinations of pairwise identical sites at baseline and follow-up allowing ''exact'' comparisons in the individual. In the first instance 82% of case-means at baseline and 70% at follow-up were based on 10 or more determinations. In the second instance 68% of case-means were based on 10 or more paired determinations.

The calculation of constant acceleration (a) was performed following the standard formulations

$$v_i = v_0 + at$$

and

$$v_i^2 = v_0^2 + 2as,$$

where v_i is the velocity at time point i, v_0 the initial velocity at time point 0, t the length of time interval, and s the distance.

Methodological error

A random subsample of 25 individuals was selected to estimate the magnitude of measurement error. Based on duplicate measurements the precision of the method (s) was calculated according to

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

where d denotes the difference between duplicates and n the number of duplicates.

The precision related to a single determination (standard error of unit weight) was estimated to $s = 0.122$. The standard error related to the mean (s_m) across all determinations within an individual (case-mean), following the general law of error propagation as found from

$$s_m = s/\sqrt{n-1},$$

was estimated to be $s_m = 0.035$ for an average of 12 determinations per

Table 1. Study cohort according to age at baseline and smoking

Age group (years)	Smoker					Former smoker					Non-smoker	
	N	age at baseline (years) mean	consumption baseline (cig/day) mean	consumption follow-up (cig/day) mean	duration (years) mean	*N*	age at baseline (years) mean	consumption (cig/day) mean	duration (years) mean	quit-time* (years) mean	*N*	age at baseline (years) mean
20–35	3	27.7	17.5	12.3	14.0	6	28.8	11.7	9.0	9.0	15	28.6
36–45	10	41.4	14.0	12.1	28.6	9	40.7	15.8	12.6	15.3	15	39.0
46–60	6	48.5	15.7	16.2	41.2	13	50.5	17.5	21.3	17.5	14	52.1
Total	19	41.5	14.9	13.5	30.3	28	42.7	15.6	15.8	15.0	44	39.8

*Number of years since giving up smoking.

individual. The error related to the comparison of group means (s_c) was estimated to be $s_c = 0.010$. The calculations were based on an a priori unit weight of the 16 single measurement sites.

Statistics

The distributions of the dependent variables expressing the CEJ–PBC distance at baseline and follow-up as well as the resulting 10-year change were approximately normal (Kolmogorov–Smirnov test). The statistical analyses were performed by means of 1-, and 2-factor ANOVA as well as repeated measures ANOVA, fixed effects, or ANCOVA with baseline bone height level as a covariate. Post hoc testing was performed according to Scheffe. Variables with non-normal distributions were tested with non-parametric methods (χ^2, Wilcoxon matched pairs, and Kruskal–Wallis ANOVA).

Multiple regression analysis was run with dummy variables for smoking (smokers versus non-smokers and former smokers) and former smoking (former smokers versus non-smokers and smokers). Logistic regression was used to estimate the relative risk associated with smoking, expressed as odds ratio and 95% confidence interval (OR and 95% CI). The 10-year change in CEJ–PBC distance served as the dependent variable. The dependent variable was transformed into a dichotomous variable following the 80th %-ile of the distribution (>0.70 mm = 1, else = 0), a reduction of more than 0.7 mm signifying ''true'' bone loss. Statistical significance was accepted at $p<0.05$.

In the analyses age at baseline was stratified according to (1) 20–35 years ($n = 24$), (2) 36–45 years ($n = 34$), and (3) 46–60 years ($n = 33$). The CEJ–PBC distance at baseline was stratified according to (1) 0.0–1.0 mm ($n = 31$), (2) 1.1–1.55 mm ($n = 30$), and (3) 1.56–4.32 mm ($n = 30$). Smoking exposure in terms of duration (i.e., years of smoking) was stratified into (1) no smoking ($n = 44$), (2) 1–19 years (mean 9.7 years, $n = 19$; four smokers, 15 former smokers), and (3) 20 or more years (mean 30.2 years, $n = 26$; 15 smokers, 11 former smokers). Lifetime exposure, i.e., the accumulated exposure in terms of the product of daily consumption and duration was stratified into (1) no smoking ($n = 44$), (2) 20–340 cig-years (mean 174.6 cig-years, $n = 24$; 10 smokers, 14 former smokers), and (3) 350–900 cig-years (mean 554.4 cig-years, $n = 18$; nine smokers, nine former smokers).

Table 2. Study cohort

Characteristic	Smoker		Former smoker		Non-smoker	
	mean	SD	mean	SD	mean	SD
plaque index	0.8	0.30	0.8	0.35	0.7	0.33
gingival bleeding (%)	35.8*	32.2	18.9	16.7	20.3	16.6
pocket frequency (n)	12.7*	3.22	3.1	2.56	3.2	1.88

Clinical characteristics at baseline according to smoking.
*Significantly different from non-smokers and former smokers ($p<0.05$).

Table 3. Number of missing premolar teeth according to quadrant region

Premolar quadrant region	Missing teeth (n)		
	baseline	follow-up	10-year change
maxillary right 1st	12	14	−2
maxillary right 2nd	6	7	−1
maxillary left 1st	10	12	−2
maxillary left 2nd	8	10	−2
mandibular right 1st	5	5	0
mandibular right 2nd	2	4	−2
mandibular left 1st	6	6	0
mandibular left 2nd	3	3	0
total	52	61	−9

Results

Missing teeth and measurability

The numbers of missing premolar teeth at baseline and follow-up as well as the 10-year premolar tooth mortality are presented in Table 3. Maxillary first premolars were the most frequently missing teeth, 13% of individuals at baseline and 15% at follow-up missing at least one maxillary first premolar. The mandibular second premolars were the least frequently missing teeth, 3% of individuals at baseline and 4% at follow-up missing at least one mandibular second premolar. A total of 52 premolar teeth (7%) in 31 individuals was missing at baseline and 61 (8%) in 37 individuals at follow-up. Nine premolar teeth (1%) in 8 individuals with a maximum of two teeth in any one individual were lost to follow-up. One or more premolar teeth were missing in 6 (31%) smokers, 8 (29%) former smokers, and 17 (39%) non-smokers at baseline, and in 8 (42%) smokers, 11 (39%) former smokers, and 18 (41%) non-smokers at follow-up. The differences between smoking groups were not statistically significant neither at baseline nor at follow-up ($\chi^2 = 0.8$ and 2.6, respectively, $p>0.05$). The mean (SD) number of missing premolar teeth per person at baseline and follow-up was 0.6 (0.97) and 0.7 (1.01), respectively, implicating a 10-year premolar mortality rate of 0.10 (0.33) per person. The differences between smoking groups were small throughout and not statistically significant (Kruskall–Wallis $H = 0.3$, $p>0.05$).

A total of 306 sites at baseline and 409 at follow-up were non-measurable, representing 21% and 28%, respectively, of the ideal maximum number of preselected sites. The increase implied a reduction in measurability by 8.8% in relation to available sites at baseline. At baseline, 104 sites were non-measurable due to missing teeth whereas 202 sites were strictly non-readable. The corresponding figures at follow-up were 122 and 287, respectively. Thus, non-readable sites constituted the greater part of non-measurability, 66% at baseline and 70% at follow-up, the ratio of non-readable to missing sites being approximately 2:1. The main reason for non-readability was lack of unambiguously identifiable landmarks due to projectional overlapping and dental restoration artefacts.

Bone height level at baseline and follow-up

The mean CEJ–PBC distance at baseline according to premolar quadrant

region and smoking is presented in Table 4. For all quadrants there were statistically significant differences between smoking groups. The overall mean (SD) CEJ–PBC distance for the total sample was 1.46 (0.80) mm; 1.82 (1.01) mm for smokers, 1.65 (0.81) mm for former smokers, and 1.16 (0.59) mm for non-smokers. Controlling for age the association between smoking and baseline bone height level was statistically significant (ANOVA $F(2,2) = 4.3$, $p = 0.016$). The post hoc difference between smokers and non-smokers was statistically significant (Scheffe $p = 0.014$), whereas the differences between smokers and former smokers, and between former smokers and non-smokers, respectively, were not (Scheffe $p > 0.05$). The median (IR) valid number of determinations per person related to the overall mean was 14.0 (5.0), and there were no statistically significant differences between smoking groups (Kruskal–Wallis $H = 0.3$, $P > 0.05$).

The observations regarding the CEJ–PBC distance at the 10-year follow-up according to premolar quadrant region and smoking are presented in Table 5. For all quadrants there were statistically significant differences between smoking groups. The overall mean (SD) CEJ–PBC distance was 1.83 (0.91) mm for the total sample; 2.56 (1.18) mm for smokers, 1.91 (0.72) mm for former smokers, and 1.43 (0.67) mm for non-smokers, respectively. Controlling for age the association between smoking and follow-up bone height level was statistically significant (ANOVA $F(2,2) = 11.6$, $p = 0.000$). Smoking remained significant when baseline bone height level was introduced as a co-variate in the model (ANCOVA $F(2,2) = 8.8$, $p = 0.000$). The post hoc differences between smokers and non-smokers, and between smokers and former smokers, respectively, were statistically significant (Scheffe $p = 0.000$ and 0.011, respectively). The difference between former smokers and non-smokers was not significant (Scheffe $p > 0.05$). The median (IR) valid number of determinations per person related to the overall mean was 12.0 (6.0), which was significantly less than that at baseline (Wilcoxon $Z = 4.6$, $p = 0.000$). There were no statistically significant differences between smoking groups with reference to the valid number of determinations per person (Kruskal–Wallis $H = 0.9$, $p > 0.05$).

Table 4. CEJ–PBC distance (mm) at baseline

Premolar quadrant	Determinations per person (mean)	Smoker		Former Smoker		Non-Smoker		ANOVA	
		mean	SD	mean	SD	mean	SD	*F*	*p*
maxillary right	3.0	2.03	1.20	1.89	1.06	1.28	0.70	5.4	0.006
maxillary left	2.8	1.89	0.85	1.59	0.72	1.19	0.67	5.9	0.004
mandibular right	3.4	1.50	0.84	1.41	0.76	0.98	0.52	4.0	0.021
mandibular left	3.4	1.61	0.84	1.39	0.69	1.07	0.57	3.6	0.031
right side	6.4	1.75	0.98	1.63	0.82	1.16	0.54	5.7	0.005
left side	6.2	1.82	0.98	1.55	0.70	1.16	0.59	4.7	0.012
total	12.6	1.82	1.01	1.65	0.81	1.16	0.59	5.5	0.006

Mean and SD according to smoking and premolar quadrant.
CEJ, cemento-enamel junction; PBC, periodontal bone crest.

Table 5. CEJ–PBC distance (mm) at 10-year follow-up

Premolar quadrant	Determinations per person (mean)	Smoker		Former smoker		Non-Smoker		ANOVA	
		mean	SD	mean	SD	mean	SD	*F*	*p*
maxillary right	2.6	2.79	1.83	2.23	0.94	1.60	0.84	6.4	0.003
maxillary left	2.6	2.62	1.10	1.92	0.82	1.47	0.78	11.5	0.000
mandibular right	3.2	2.10	0.85	1.66	0.72	1.33	0.72	6.7	0.002
mandibular left	3.1	2.43	1.21	1.67	0.79	1.26	0.69	11.8	0.000
right side	5.8	2.40	1.23	1.91	0.73	1.48	0.73	7.9	0.001
left side	5.7	2.62	1.17	1.81	0.73	1.37	0.66	14.8	0.000
total	11.5	2.56	1.18	1.91	0.72	1.43	0.67	12.3	0.000

Mean and SD according to smoking and premolar quadrant.
CEJ, cemento-enamel junction; PBC, periodontal bone crest.

Table 6. Ten-year change in CEJ–PBC distance (mm)

Premolar quadrant	Determinations per person (mean)	Smoker		Former smoker		Non-smoker		ANOVA	
		mean	SD	mean	SD	mean	SD	F	*p*
maxillary right	2.5	0.94	1.06	0.57	0.43	0.56	0.34	6.8	0.002
maxillary left	2.4	0.69	0.49	0.25	0.44	0.29	0.33	8.5	0.001
mandibular right	3.2	0.56	0.56	0.28	0.45	0.33	0.40	6.7	0.002
mandibular left	3.0	0.80	0.80	0.34	0.43	0.20	0.43	9.3	0.000
right side	5.7	0.74	0.56	0.38	0.34	0.43	0.33	7.8	0.001
left side	5.4	0.81	0.64	0.33	0.37	0.22	0.33	9.8	0.000
total	11.1	0.74	0.59	0.26	0.31	0.27	0.29	10.9	0.000

Differences between identical sites at baseline and follow-up. Mean and SD according to smoking and premolar quadrant.
CEJ, cemento-enamel junction; PBC, periodontal bone crest.

Ten-year bone height change

The 10-year change in the CEJ–PBC distance according to premolar quadrant region and smoking is demonstrated in Table 6. For all quadrants there were significant bone height reductions over time. The magnitude of the reduction was throughout significantly greater in smokers as compared to non-smokers. The overall mean (SD) 10-year bone height reduction for the total sample was 0.37 (0.42) mm; 0.74 (0.59) mm for smokers as against 0.26 (0.31) mm for former smokers, and 0.27 (0.29) mm for non-smokers (Fig. 1). The changes within smoking groups over time were statistically significant (paired $t = 5.5$, 4.3, and 5.9, respectively, $p = 0.000$). Controlling for age the 10-year bone height reduction was significantly associated with smoking (repeated measures ANOVA $F(2,2) = 8.2$, $p = 0.000$). The association remained significant when, in addition, baseline bone height level was introduced as a co-variate (ANCOVA $F(2,2) = 5.0$, $p = 0.009$). The post hoc difference between smokers and

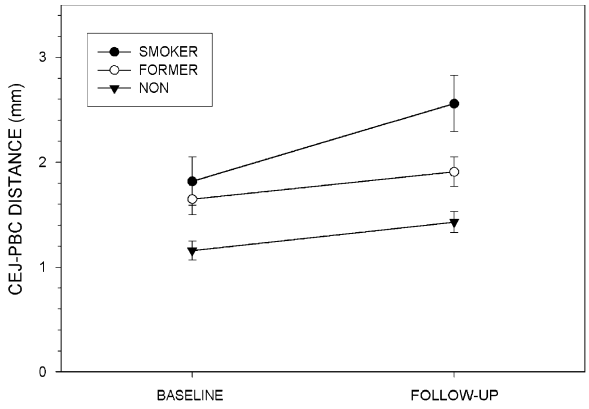

Fig. 1. Ten-year change in periodontal bone height (CEJ–PBC distance). Mean and 95% CI according to smoking. Interaction effect $F(2,85) = 11.5$, $p = 0.000$.

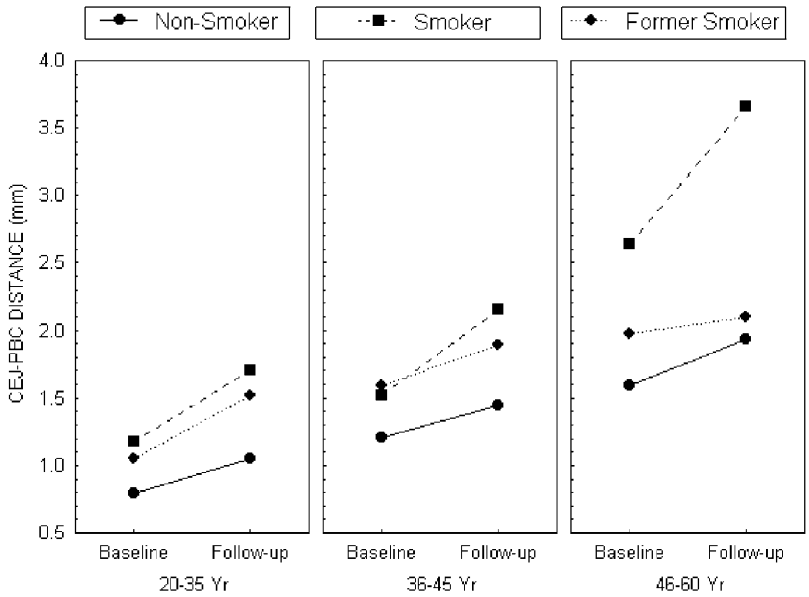

Fig. 2. Ten-year change in periodontal bone height (CEJ–PBC distance). Mean according to smoking and age at baseline. Interaction effect $F(4,79) = 2.2$, $p = 0.072$.

non-smokers was statistically significant (Scheffe $p = 0.000$). The differences between smokers and former smokers, and between former smokers and non-smokers were almost significant (Scheffe $p = 0.082$ and 0.077, respectively). The results were very similar when the 10-year change in the CEJ–PBC distance was evaluated via determinations of pairwise identical sites (ANOVA $F(2,2) = 8.5$, $p = 0.000$). This also held true when baseline bone height level was included as a co-variate (ANCOVA $F(2,2) = 5.7$, $p = 0.005$).

The median (IR) valid number of determinations per person related to the overall mean was 12.0 (7.0) which was statistically significantly less than the valid numbers of determinations at baseline and follow-up (Wilcoxon $Z =$ 6.3 and 3.9, respectively, $p = 0.000$). The differences between smoking groups with reference to the valid number of determinations per person were not statistically significant (Kruskal–Wallis $H = 1.6$, $p > 0.05$).

Effects of baseline bone height level and age

The 10-year change in CEJ–PBC distance was statistically significantly associated with baseline bone height level, controlling for age (repeated measures ANOVA $F(2,2) = 29.0$, $p = 0.000$). There was no significant interaction effect between smoking and baseline bone height level on the 10-year bone height change.

The 10-year change in CEJ–PBC distance was not significantly associated with age, when baseline bone height level was accounted for (repeated measures ANOVA $F(2,2) = 0.5$, $p > 0.05$), and there was no significant interaction effect between age and smoking. However, a borderline significant interaction effect between smoking, age and time point ($p = 0.072$) was observed, suggesting that the gradual bone height reduction over time was comparably greater in smokers with increasing age (Fig. 2).

Exposure effect

Smoking exposure expressed as duration and accumulated lifetime exposure, respectively, was significantly associated with 10-year change in CEJ–PBC distance. Controlling for age the association with smoking duration (ANOVA $F(2,2) = 7.1$, $p = 0.002$) as well as lifetime exposure (ANOVA $F(2,2) = 26.6$, $p =$ 0.000) was statistically significant in continuous smokers alone, as well as in continuous smokers and former smokers combined (ANOVA $F(2,2) = 13.2$, and $F(2,2) = 11.5$, respectively, $p = 0.000$).

Multiple regression analyses

Multiple linear regression was run with 10-year change in CEJ–PBC distance as the dependent variable, and age, continuous smoking, former smoking, baseline bone height level, plaque, gingival index and number of retained teeth as independent variables entered in one block. The analysis resulted in a significant model (adjusted $R^2 = 0.19$, $F(6,84) = 4.5$, $p = 0.001$). Continuous smoking was the most important and only statistically significant variable ($t = 3.7$, $p = 0.001$). Former smoking, thus, was not a significant factor.

Logistic regression was run with 10-year ''true'' bone loss as the dependent variable. A cut-off point of 0.70 mm corresponding to the 80th %-ile of the distribution of the 10-year change in CEJ–PBC distance was arbitrarily selected to signify 10-year ''true'' bone loss. It was observed that the relative risk (odds ratio, OR) of 10-year ''true'' bone loss associated with smoking was OR = 17.0 (95% CI 3.1–92.8, $p = 0.001$) after adjustment for age, baseline bone height, plaque, gingival index, and number of retained teeth. Former smoking was not associated with an increased risk.

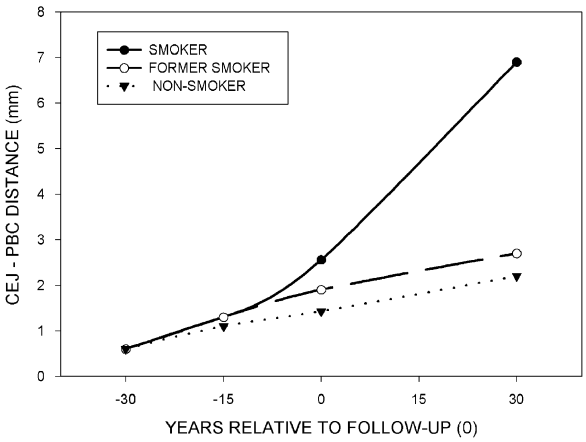

Fig. 3. Smoking-induced acceleration of periodontal bone height reduction rate. Hypothetical model based on present observations and the assumption of a constant reduction rate in non-smokers. 0 = time point of present 10-year follow-up, −15 years = time point of smoking cessation in former smokers, −30 years = time point for starting to smoke in current and former smokers, 30 years = prediction found by extrapolation.

Discussion

The object of the present investigation was to determine the magnitude of the long-term effect of smoking on the periodontal bone height over 10 years. The longitudinal change implied a reduction of the periodontal bone height irrespective of smoking. The bone height reduction observed in smokers, however, was 2.7 times greater than that observed in non-smokers, suggesting on the average an almost three-fold elevated bone height reduction rate under the influence of smoking. The bone height reduction rate of former smokers who had given up smoking in the distant past was identical to that of non-smokers. Changes were consistent throughout the four quadrants of the dentition. The observations are in general agreement with earlier longitudinal studies (Feldman et al. 1987, Bolin et al. 1993, Machtei et al. 1997, Krall et al. 1999, Bergstrom et al. 2000, Payne et al. 2000) and lend additional support to the contention that smoking is a major factor contributing to periodontal bone loss in humans. The overall average 10-year bone height reduction rate was estimated at 0.037 mm per year, which appears to be of the same order as recently reported for a large-scale longitudinal investigation in Sweden over 17 years (Hugoson & Laurell 2000).

Smoking-induced acceleration of bone loss. A hypothesis

In the present study, the bone height reduction rate was remarkably elevated in smokers. This was observed already at baseline (at a population median age of 44 years) as the CEJ–PBC distance was approximately 0.6 mm more reduced in smokers compared to non-smokers. At the 10-year follow-up the gap had increased to 1.1 mm. With reference to such a widening gap between smokers and non-smokers over time, it may be hypothesized that the bone height reduction in smokers takes place at an accelerated rate. The plausibility of this hypothesis is illustrated in Fig. 3. Under the assumption that the bone height reduction in the *absence* of smoking occurs at a constant average rate of 0.027 mm per year, the mean CEJ–PBC distance 30 years prior to follow-up can be estimated to approximately 0.6 mm (at a population median age of 22 years). Using this value as the initial bone height level and 0.027 mm per year as the initial bone height reduction rate, the acceleration in the *presence* of smoking can be estimated to approximately 0.0025 mm per year^2. Assuming, further, a constant acceleration the additional bone height reduction in smokers 30 years after follow-up can be predicted to approximately 4.4 mm. The extrapolated 30-year future CEJ–PBC distance in smokers, then, will approximate 7.0 mm. This level may be compared with a predicted additional reduction of approximately 0.8 mm in non-smokers resulting in an extrapolated CEJ–PBC distance of approximately 2.2 mm 30 years after follow-up. Following these trends, the gap between smokers and non-smokers 30 years after follow-up (at a population median age of 82 years) is expected to have increased to approximately 4.7 mm.

The 10-year bone height reduction observed in former smokers suggested an average reduction rate of 0.026 mm per year, i.e., the same as for non-smokers. The CEJ–PBC distance difference between former smokers and non-smokers at baseline was 0.5 mm, which was maintained at the 10-year follow-up. The mean CEJ–PBC reduction rate in former smokers during approximately 15 years of previous smoking (before quitting) is supposed to have equalled that of smokers, assuming the same exposure. The average bone height level approximately 15 years prior to follow-up, then, can be estimated to approximately 1.3 mm (same as for smokers). Following smoking cessation, the reduction rate of former smokers regresses toward the non-smoker rate and subsequently equals it. The extrapolated 30-year future CEJ–PBC distance for former smokers, then, can be predicted to be approximately 2.7 mm, and the gap between former smokers and non-smokers can be expected to remain constant at approximately 0.5 mm (Fig. 3).

According to this hypothesis the reduction rate of the periodontal bone in the absence of smoking is modest and the longevity of teeth not seriously challenged. In the average lifelong non-smoker some 80% of the premolar supporting bone will remain intact until old age as against some 40% in the average chronic smoker. This suggests that it is highly likely that smokers, subjected to an accelerated bone loss rate, are at increased risk for premature tooth loss. This contention is supported by some earlier observational data (Feldman et al. 1987, Holm 1994, Krall et al. 1999).

Although strongly indicative of a causal role of smoking in periodontal bone loss, the present observations do not immediately provide evidence that smoking (alone) causes periodontal disease. Bone loss is a surrogate endpoint and not synonymous with periodontal disease. It seems more likely that the effect of smoking is exerted in consort with other component causes. Other component causes that may act synergistically with smoking to cause periodontal disease are genetic susceptibility factors and other environmental factors including microbial factors. The understanding of the interplay between component causes, particularly gene–environment interactions, in periodontal disease as

in other chronic diseases is currently limited.

Age apparently had not a pivotal role in the process of bone height reduction, an observation which appears to be in agreement with the observations of Hugoson & Laurell (2000), suggesting that age per se is no risk factor for periodontal bone loss. It is conceivable, however, that with the passage of time the cumulative influence of other as yet unidentified factors, may exert an influence that is currently captured under the collective term of ''age''. Following the above reasoning, a considerable part of the effect traditionally ascribed to age is likely to be due to smoking.

The present observations are based on the radiographic assessment of premolar teeth, i.e., a selected fraction of the dentition. The reasons for selecting premolars were an expected high tooth retention frequency together with a moderate tooth restoration frequency. In addition, comparably good reproduction characteristics of the image material were obtained by the use of standardized bite-wing radiographs, securing an overall high standard of measurability. As indicated by the precision of the method, measurement quality was high, minimizing the influence of random error. It should be recognized, however, that the influence of systematic error due to, e.g., parallactic distortion, is unknown. As indicated by the findings that changes over time were consistent throughout the four quadrants of the dentition, the representativity of the selected fraction was judged satisfactory.

A major strength of the present investigation is the great number of retained teeth at all ages during the course of the investigation, the proportion of retained premolars being 93% at baseline and 92% at follow-up. Moreover, 85% of available sites at baseline and 78% at follow-up were measurable. Interestingly, the observations based on determinations of identical sites within the individual at both time points were very similar to those based on determinations of maximum available sites at each time point. It is concluded, therefore, that the 10-year comparisons rest on a robust basis of high-quality data. A limitation of the investigation is the small size of the study cohort, particularly on the part of smokers. This limitation is to some extent counterbalanced by the cohort homogeneity in terms of socio-economic status and oral health behaviour as described elsewhere (Bergström & Eliasson 1985).

In summary, the present observations, estimating the long-term influence of chronic smoking on the periodontal bone height in quantitative terms, provide evidence that the bone destruction rate under the influence of smoking is elevated, most likely accelerated, resulting in a gradually increasing deviation from normality along with the passage of time. On the basis of the observations it is hypothesized that

- the periodontal bone height reduction in non-smokers occurs at a modest constant rate,
- the periodontal bone height reduction rate in smokers is accelerated,
- the smoking induced acceleration will return toward the non-smoker rate following cessation of smoking.

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