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Investigation of periodontal destruction patterns in smokers and non-smokers

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Abstract

Background: Previous work has suggested that tobacco smoking has a local as well as a systemic effect on the severity of periodontal disease.

Objective: To test the hypothesis that smokers have more disease in the upper anterior region.

Methods: A retrospective stratified random sample of 49 non-smokers and 39 heavy smokers ($\geqslant$20 cigarettes/day) was obtained from a total of 3678 referred patients with adult periodontitis. Probing depth data were collected from clinical records and radiographic measurements were carried out on existing dental panoramic tomographs to assess the inter-proximal bone levels.

Results: The proportion of sites with ''bone loss'' 4.5 mm or greater was higher in smokers, the greatest difference being observed in upper anterior sites (smokers: 73.3 $\pm$ 25.5%, non-smokers: 48.3 $\pm$ 31.2%, $p < 0.001$). A difference was also observed when the number of palatal sites probing 4 mm or greater in the upper anterior region was expressed as a proportion of all such sites in the mouth (smokers: 12.3 $\pm$ 6.8%, non-smokers: 9.8 $\pm$ 8.8%; $p = 0.050$).

Conclusion: The overall pattern of tissue destruction was consistent with a systemic effect of smoking. The suggestion of a marginal local effect of the smoking habit in maxillary anterior palatal sites requires further investigation.

Key words: bone loss; periodontitis; smoking

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The effect of smoking on the periodontium has been the subject of studies for the past 20 years. There is now a considerable body of evidence demonstrating the association of periodontal destruction and cigarette smoking. Smokers have a greater risk of exhibiting more periodontal attachment loss (Bergström & Preber 1994, Grossi et al. 1994, Machtei et al. 1997, Axelsson et al. 1998, Machtei et al. 1999) a larger number of deep periodontal pockets and higher mean probing pocket depth (Preber & Bergström 1986, Bergström & Eliasson 1987, Bergström 1989, Haber et al. 1993) and more extensive and more severe alveolar bone loss (Bergström & Eliasson 1987, Grossi et al. 1995, Razali et al. 2005). Substantial epidemiological data indicate that smokers have fewer teeth, a higher prevalence of edentulism and a greater incidence of tooth loss than non-smokers (Mohlin et al. 1979, Osterberg & Mellstrom 1986, Ahlqwist et al. 1989, Holm 1994). Furcation involvement at molar teeth is also more frequent in smokers than non-smokers (Mullally & Linden 1996, Axelsson et al. 1998).

It is established that different parts of the dentition have different rates of progression of periodontitis. Björn & Halling (1987) reported variation in periodontal breakdown between regions of the dentition in healthy, middle-aged, non-smoking women. They found more maxillary teeth were severely involved, with the exception of upper incisors. Higher bone loss in molar/pre-molar areas especially of the maxilla was also reported in the earlier study of Schei et al. (1959).

The majority of evidence in the literature is inconclusive on the effect of smoking on the microflora, but suggests that the main effect of smoking is on the immune and inflammatory response (MacFarlane et al. 1992, Persson et al. 1999, Palmer et al. 2005), which frequently reduces the clinical signs of gingival inflammation such as redness and bleeding (Preber & Bergström 1985, 1986, Bergström & Preber 1986, Bergström et al. 1988, Bergström 1990). However, some studies have reported differences in the ''pattern'' of periodontal destruction and tooth loss among smokers and non-smokers that imply a localized effect of the smoking habit in

the upper anterior region and especially at palatal sites (Preber & Bergström 1985, 1986, Haber & Kent 1992, Axelsson et al. 1998, Haffajee & Socransky 2001, van der Weijden et al. 2001).

The aim of this study was to establish retrospectively whether there is any potential difference in the pattern of destructive periodontal disease between smokers and non-smokers in a group of adult periodontitis patients; in particular, to test the hypothesis that smokers have proportionally more disease in the upper anterior region.

Material and Methods

The records of 3678 patients referred to and seen in the periodontal clinic at Guy's and St. Thomas' Hospital were considered, and subjected to the following inclusion and exclusion criteria:

Inclusion criteria

1. Patients diagnosed with moderate-to-severe periodontitis.
2. Aged between 46 and 60 years.
3. Patient with no significant medical history.
4. Smoking patients reported smoking $\geqslant 20$ cigarettes/day.
5. Non-smoking patients reported never smoking.

Exclusion criteria

1. Patients with abnormal tooth and root form.
2. Patients with a significant medical history, such as diabetes or drugs affecting inflammation.
3. Past smokers.

A random sample, stratified for age, was selected from the 650 records that satisfied these criteria. The selection was achieved by dividing the records into 3-year age bands, and using a random number generation programme (Stata Co., College Station, TX, USA). One hundred and twenty records were generated and 88 records (smoker = 39, non-smoker = 49) were obtained with dental panoramic tomographs (DPTs) that allowed assessment of bone levels in all regions of the mouth. The mean age of the subjects was similar between smokers (52.2 years, standard deviation (SD) $\pm$ 4.4) and non-smokers (52.9 $\pm$ 4.2 years).

Probing depth data

The probing depth data were obtained from the measurements that were performed during the initial consultation by a senior clinician or students under supervision. A calibrated probe (Williams markings) was used to measure mesio-buccal, mid-buccal, disto-buccal, mesio-lingual, mid-lingual and disto-lingual of each tooth. Probing depth charts were photocopied with the patients' details masked so that the examiner was blind to the patients' information when the data were transferred for analysis.

Radiographic measurement of "bone loss"

DPTs were coded and bone levels measured by a single examiner (B. B.) masked as to the patient's age, gender and smoking status.

All DPTs were viewed on a screen (Rinn Viewer and Magnifier, Dentsply, Elgin, IL, USA) at $\times$ 3 magnification. The outline of the cemento-enamel junction (CEJ)and the proximal alveolar bone crest on both mesial and distal surfaces were marked with a fine pencil (0.5 mm tip). On each proximal site, the bone level was measured directly from the CEJ to the alveolar bone crest using callipers calibrated to the nearest 0.5 mm. The landmarks were defined as follows:

1. CEJ—If the CEJ could not be identified, the most apical margin of a crown or proximal restoration was used as a reference point. If the margin of the restoration was too apical to the average level of the CEJ, the site was defined as immeasurable.
2. Alveolar bone crest—the most coronal level where the periodontal membrane retained its normal width (Björn et al. 1969). When an infrabony defect was present, the measurement was made to the apical border of the radiolucency (Suomi et al. 1971). When the radiographic image indicated differing heights of alveolar bone crest, the most apical level was chosen.

If either of the landmarks could not be visualized clearly according to the criteria described above, the site was regarded as immeasurable. This measure was termed "bone loss", acknowledging that not all of this measure could be attributed to actual loss of bone through periodontitis.

The number of remaining teeth was also charted. Only teeth that were fully erupted and charted in the clinical record were considered as present.

Statistical analysis

All data were analysed using the statistical package STATA 8 (Stata Co.). The patients constituted the statistical unit. The data from four distinct regions were investigated, the anterior region (canine to canine) and the combined posterior regions (pre-molar and molars) in the upper and lower jaw (maxilla and mandible). In addition, probing depth data from the maxilla was submitted to separate analysis of the buccal and palatal sites in the anterior and posterior regions. The mean probing depth, the proportion of sites with probing depth greater or equal to 4 mm, the mean bone loss and the proportion of sites with bone loss greater or equal to 4.5 mm were calculated for each region. The dividing line of 4.5 mm bone loss was chosen as evidence of definite bone loss taking account of the magnification factor inherent in DPTs. A two-sample *t* test was used to test differences between smokers and non-smokers.

In order to determine whether smokers had *proportionally* more disease in the upper anterior region, the number of sites with probing depth greater or equal to 4 mm and the number of sites with bone loss greater or equal to 4.5 mm were calculated as proportions of all such sites in the whole mouth. This data did not exhibit a normal distribution and the Mann–Whitney *U* test was used to test differences between smokers and non-smokers.

In all analyses, statistical significance was implied when a p value < 0.05 was observed.

Eleven randomly selected DPTs were re-examined under masked conditions for establishing intra-examiner reproducibility.

Results

Table 1 presents the mean number of teeth remaining within each region of the mouth. There were no statistically significant differences. Although smokers had slightly fewer teeth overall (24.6 $\pm$ 3.0) than non-smokers

Table 1. Mean (standard deviation) number of teeth present in smokers and non-smokers by region

Region	Smoker ($n = 39$)	Non-smoker ($n = 49$)	Difference	p value*
Upper				
Anterior	5.4 (1.7)	5.5 (1.2)	0.1	0.415
Posterior	6.2 (2.0)	6.6 (2.1)	0.4	0.927
Lower				
Anterior	5.9 (0.3)	5.7 (0.9)	0.2	0.171
Posterior	6.8 (1.6)	7.3 (2.1)	0.5	0.294

*Student's two-group *t*-test.

Table 2. Mean (standard deviation) probing depth (mm) in smokers and non-smokers by region

Region	Smoker ($n = 39$)	Non-smoker ($n = 49$)	Difference	p value*
Upper				
Anterior	3.61 (0.98)	3.26 (1.30)	0.35	0.070
Posterior	3.97 (0.90)	3.75 (1.11)	0.22	0.163
Lower				
Anterior	2.87 (0.83)	2.96 (1.29)	0.09	0.753
Posterior	3.69 (0.76)	3.46 (1.25)	0.23	0.046

*Student's two-group t-test.

Table 3. Mean (standard deviation) percentage of sites with probing depth $\geqslant 4$ mm within each region in smokers and non-smokers

Region	Smoker ($n = 39$)	Non-smoker ($n = 49$)	Difference	p value*
Upper				
Anterior				
Buccal	37.8 (25.2)	28.7 (25.0)	9.1	0.056
Palatal	50.4 (31.6)	31.4 (28.4)	19.0	0.005
Posterior				
Buccal	45.0 (23.5)	37.1 (23.6)	7.9	0.099
Palatal	57.7 (26.5)	46.2 (25.3)	11.5	0.039
Lower				
Anterior	25.5 (23.1)	24.8 (26.9)	− 0.7	0.903
Posterior	43.1 (20.6)	33.7 (25.5)	9.4	0.067

*Student's two-group t-test.

(25.2 ± 4.4), this was not statistically significant.

Mean probing depth (mm)

There was no statistically significant difference in whole-mouth mean probing depth between smokers (3.53 ± 0.70 mm) and non-smokers (3.41 ± 1.14 mm), although the lower posterior region (Table 2) showed a marginal difference ($p = 0.046$).

Percentage of sites with probing depth $\geqslant 4$ mm

Smokers showed a trend towards a higher percentage of sites with probing depth $\geqslant 4$ mm in most regions compared with non-smokers (Table 3). However, the difference was only statistically significant for palatal sites in the maxilla, with anterior palatal sites showing the greatest difference ($p = 0.005$).

Number of sites with probing depth $\geqslant 4$ mm in the maxilla as a proportion of such sites in the whole mouth

Table 4 shows the number of sites with probing depth $\geqslant 4$ mm in the maxilla as a proportion of such sites in the whole mouth. There were no statistically significant differences between smokers and non-smokers, except the proportion in the anterior palatal region, which was 25% higher in smokers than non-smokers ($p = 0.050$).

Mean bone loss (mm)

Repeated measurement of bone loss on radiographs showed strong agreement with an intra-class correlation coefficient of 0.973. Eighty-three per cent of repeated measurements were within ± 0.5 mm of the original.

The mean bone loss was greater in smokers than non-smokers in all regions (Table 5). More bone loss was measured in the upper jaw compared with the lower jaw in both smokers and non-smokers ($p < 0.05$). The difference between smokers and non-smokers was very similar in all regions.

Percentage of sites with bone loss $\geqslant 4.5$ mm

Smokers had a higher proportion of sites with bone loss $\geqslant 4.5$ mm in all regions. This difference was greatest in the upper anterior region (smokers: $73.3 \pm 25.5\%$, non-smokers: $48.3 \pm 31.2\%$; $p < 0.001$) (Table 6).

Number of sites with bone loss $\geqslant 4.5$ mm in the maxilla and mandible as a proportion of such sites in the whole mouth

Table 7 shows the number of sites with bone loss $\geqslant 4.5$ mm in the maxilla and mandible as a proportion of such sites in the whole mouth. There was no difference between smokers and non-smokers.

Discussion

This study was carried out to determine whether there were differences in the regional pattern of probing depth and bone loss between smokers and non-smokers. In particular, as to whether previous papers that reported a local effect of the smoking habit might produce a greater proportion of disease in the upper anterior region were correct. Ideally, attachment level measurements would have been helpful, but the available data did not permit reliable calculation of attachment level in this retrospective study.

Some advantages of the retrospective nature of the present study were that the subjects could be randomly chosen from a large population of subjects with moderate-to-severe periodontal disease matched for age between groups and that measurements could be made and analysed under masked conditions. The study design also benefited from the exclusion of past smokers and the selection of smokers who smoked 20 or more cigarettes daily in order to maximize the possibility of finding differences.

There were a similar number of teeth remaining in all regions of the mouth in smokers and non-smokers under the age of 60 years. This finding is supported by the work of Axelsson et al. (1998),

Table 4. Mean (standard deviation) number of sites probing 4 mm and above within each region as a proportion of the sites probing 4 mm and above in the whole mouth

Region	Smoker ($n = 39$)	Non-smoker ($n = 49$)	Difference	p value*
Upper				
Anterior				
Buccal	9.9 (6.1)	9.9 (9.7)	0.0	0.482
Palatal	12.3 (6.8)	9.8 (8.8)	2.5	0.050
Posterior				
Buccal	14.7 (8.2)	15.7 (9.7)	1.0	0.600
Palatal	18.8 (9.8)	19.5 (10.2)	0.7	0.399
Lower				
Anterior	13.3 (8.7)	15.0 (15.8)	1.7	0.649
Posterior	31.0 (14.1)	30.1 (17.9)	0.9	0.718

*Mann–Whitney U-test.

Table 5. Mean (standard deviation) bone loss (mm) in smokers and non-smokers by region

Region	Smoker ($n = 39$)	Non-smoker ($n = 49$)	Difference	p value*
Upper				
Anterior	6.22 (1.91)	5.05 (2.35)	1.17	0.002
Posterior	6.80 (1.74)	5.64 (2.39)	1.16	0.003
Lower				
Anterior	6.03 (1.99)	4.94 (2.35)	1.09	0.016
Posterior	6.00 (1.54)	5.09 (2.61)	0.91	0.001

*Student's two-group t-test.

Table 6. Mean (standard deviation) percentage of sites with bone loss $\geqslant 4.5$ mm within each region in smokers and non-smokers

Region	Smoker ($n = 39$)	Non-smoker ($n = 49$)	Difference	p value*
Upper				
Anterior	73.3 (25.5)	48.3 (31.2)	25.0	<0.001
Posterior	79.0 (16.3)	58.3 (27.2)	20.7	<0.001
Lower				
Anterior	69.5 (29.6)	51.8 (38.2)	17.7	<0.001
Posterior	68.0 (24.2)	45.1 (33.5)	22.9	0.019

*Student's two-group t-test.

Table 7. Mean (standard deviation) number of sites with bone loss $\geqslant 4.5$ mm and above within each region as a proportion of the sites with bone loss $\geqslant 4.5$ mm and above in the whole mouth

Region	Smoker ($n = 39$)	Non-smoker ($n = 49$)	Difference	p value*
Upper				
Anterior	22.0 (12.3)	22.7 (11.8)	0.7	0.916
Posterior	30.9 (14.2)	29.3 (13.3)	1.6	0.743
Lower				
Anterior	21.1 (10.6)	22.1 (18.8)	1.0	0.711
Posterior	27.6 (9.5)	24.3 (14.9)	3.3	0.194

*Mann–Whitney U-test.

which showed only minor differences in the number of missing teeth except in the age group above 65 years. This similarity in the number of teeth present in both groups allowed reasonably valid comparison between smokers and non-smokers, and is in contrast to some previous reports where differences in the number of teeth present may have affected the measurable disease level (Haffajee & Socransky 2001).

Many previous studies (Bergström 1989, Linden & Mullally 1994, Haffajee & Socransky 2001, Bergström 2003) have shown that current smokers had deeper probing depths than non-smokers. The present study, while not showing universal differences in mean probing depth, did find a higher proportion of sites probing 4 mm and above in smokers. Preber & Bergström (1986) suggested that higher local exposure to cigarette smoke on the palatal maxillary surfaces had led to a significant difference in probing depth between smokers (4.17 ± 0.86 mm) and non-smokers (3.78 ± 0.74 mm). However, the ''significant'' status of this difference was based entirely on the fact that a statistically significant p value was observed for maxillary palatal surfaces, while the differences in other regions were not significant. In contrast to the present investigation, their study included a wide age range (26–79 years) and smoking exposure including the lower range of 1–20 cigarettes a day. Van der Weijden et al. (2001) reported that the palatal surfaces of the upper anterior teeth showed the largest differences between smokers and non-smokers and they claimed a possible local effect of smoking. However, the subjects in their study also differed from those in the present investigation, ranging in age from 36 to 66 years and cigarette consumption from 1 to 60 cigarettes/day. Haffajee & Socransky (2001) have also reported a difference in pocket depth data between smokers and non-smokers at inter-proximal and palatal maxillary sites. Once again, their sample showed a wide age range from 20 to 86 years, although the difference in the pocket depth was only found in subjects 50 years and over. None of these previous reports compared the proportional representation of disease at upper anterior sites between smokers and non-smokers.

In the present study, smokers showed the greatest numerical difference in the mean proportion of sites probing 4 mm and above on upper anterior palatal surfaces. More convincing was the fact that the number of sites probing 4 mm and above was greater *as a proportion of such sites in the whole mouth* ($p = 0.050$). This suggests that, as far as probing depths are concerned, there is a small contribution to this measure of disease in these sites, which may be associated with smoking. However, it must be stressed that probing depth is not equated with attachment loss and the measurements were obtained from clinical recordings by multiple examiners.

The most valid measure of disease severity in the present study is the amount of bone loss. However, the inaccuracy of DPTs in interpreting marginal bone height has been an issue of

debate in a number of previous studies (Åkesson et al. 1989). Adriens et al. (1982) found the DPT had a lower number of measurable sites (70–87%) when compared with a periapical radiograph (87–97%). Kaimenyi & Ashley (1988) showed a similar result (74%) for DPTs. The deficiencies of the DPT include image distortion, poor imaging at maxillary pre-molar and lower anterior regions due to overlapping and superimposition of the cervical spine. However, in the present study, the number of measurable sites which were obtained from the DPTs (87.3%) was more comparable with those previously reported for periapicals, which may be due to improved quality of DPTs in the intervening years. However, the increased number of measurable sites in this study could also be due to direct measurements being taken compared with relative proportions in other studies (Albandar & Abbas 1986), as the identification of the root apex is not required in the former. In any case, the proportion of unreadable sites was not very different between smokers and non-smokers allowing a reasonable comparison to be made.

The results from radiographic measurement are in agreement with many previous studies that have shown more bone loss among smokers than non-smokers (Bergström & Eliasson 1987, Bergström et al. 1991, Grossi et al. 1995, Razali et al. 2005). All the regions of the mouth showed smokers have greater mean bone loss and a higher proportion of sites with $\geqslant$4.5 mm bone loss. However, there was only a marginally greater difference observed in the upper anterior sites compared with other regions. The more pertinent analysis of the regional differences between smokers and non-smokers, represented by the comparison of the number of sites with bone loss $\geqslant$4.5 mm in the upper anterior region as a proportion of all such sites, did not show any difference.

The evidence for the systemic effects of smoking on the periodontium is overwhelming and has been proposed as the most important mechanism (Palmer et al. 1999, 2005). However, the possibility of an additional local effect of tobacco use cannot be ruled out. In the present study, although the difference in bone loss and probing depth between smokers and non-smokers at all four regions was small, the upper anterior region showed the greatest and most consistent difference between smokers and non-smokers. This is in agreement with a few studies where the greatest difference of pockets and attachment loss was found in the anterior maxillary region (Preber & Bergström 1986, Haber & Kent 1992, Haffajee & Socransky 2001, van der Weijden et al. 2001). The variables which provided a means of testing the hypothesis that smokers have proportionally more disease in the upper anterior region were the number of sites with probing depth $\geqslant$4 mm and the number of sites with bone loss $\geqslant$4.5 mm in the maxilla as a proportion of such sites in the whole mouth. These variables have not previously been used in studies of local effects of smoking. Although there was no indication of differences related to bone loss, a marginally significant difference was noted between smokers and non-smokers in probing depths for sites in the upper anterior palatal region. However, the present study was not able to specifically measure palatal sites for either attachment or bone loss. Further work, preferably including attachment loss and perhaps with a larger number of subjects, needs to be carried out to demonstrate that the effect represented by the present finding is of significant clinical magnitude to implicate a local effect.

Conclusion

Within the limitations of the present study, we conclude that smokers have more severe periodontal disease for age, as demonstrated by a higher proportion of sites probing $\geqslant$4 mm and more bone loss in all regions of the mouth. The effect of smoking is likely to be mainly due to a systemic effect, but a marginal local contribution cannot be entirely ruled out in the palatal sites of the upper anterior teeth. Further research is required to confirm this latter finding.

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Clinical Relevance

Scientific rationale for study: The systemic effects of tobacco smoking are widespread and well documented. Although some authors have proposed significant local periodontal effects, the evidence is not strong.

Principal findings: This study confirmed that smokers have significantly more periodontal destruction in all parts of the mouth. However, the maxillary anterior palatal sites in smokers may exhibit proportionally more disease.

Practical implications: The maxillary anterior teeth in smokers may be at greater risk of periodontitis and consequent tooth loss.