Journal of
PERIODONTAL RESEARCH

J Periodont Res 2013; 48: 623–631

JOURNAL OF PERIODONTAL RESEARCH
doi:10.1111/jre.12048

Comparison of periodontal destruction patterns among patients with and without the habit of smokeless tobacco use – a retrospective study

P. S. Anand[1], K. P. Kamath[2], A. Bansal[1], S. Dwivedi[1], S. Anil[3]

[1]Department of Periodontics, People's College of Dental Sciences & Research Centre, [2]Department of Oral Pathology, People's Dental Academy, Bhopal, Madhya Pradesh State, India and [3]Department of Periodontics and Community Dentistry, College of Dentistry, King Saud University, Riyadh, Kingdom of Saudi Arabia

Anand PS, Kamath KP, Bansal A, Dwivedi S, Anil S. Comparison of periodontal destruction patterns among patients with and without the habit of smokeless tobacco use – a retrospective study. J Periodont Res 2013; 48: 623–631. © 2013 John Wiley & Sons A/S. Published by John Wiley & Sons Ltd

Background and Objective: The effects of tobacco smoking on the prevalence and severity of periodontal disease have been well documented. However, very few studies have assessed the effects of oral smokeless tobacco (ST) on the periodontium. Considering the widespread use of ST products globally, the effects of such products on the periodontal tissues may be important. The present study was performed to compare retrospectively the patterns of periodontal destruction among oral ST users and never-users with periodontitis.

Material and Methods: Data from 149 patients with periodontitis (60 ST users and 89 never-users) were compared for mean scores of probing depth, recession (REC) and clinical attachment loss (CAL) and the mean percentage of sites with different ranges of probing depth, REC and CAL.

Results: For full-mouth scores, mean REC and CAL were significantly higher in ST users than in never-users ($p < 0.001$ and $p = 0.008$, respectively). For different regions of the dentition, mean scores of REC were significantly higher among ST users than among never-users ($p < 0.001$ for all regions), and mean scores of CAL were significantly higher for ST users in mandibular ($p < 0.001$), buccal ($p = 0.008$), lingual ($p = 0.022$), anterior ($p = 0.012$) and molar ($p = 0.009$) sites. Generally, there were higher percentages of sites with shallow pockets (0–3 mm), REC of $\geq$ 1 mm and CAL of $\geq$ 3 mm in ST users than in never-users. However, only differences for REC categories were significant for all regions ($p < 0.001$). ST users had a significantly higher proportion of sites with CAL of $\geq$8 mm for full-mouth sites ($p = 0.003$), mandibular teeth ($p < 0.001$), buccal sites ($p = 0.002$), anterior teeth ($p = 0.040$) and molars ($p = 0.007$).

Conclusion: ST users tend to have more severe REC and CAL and a greater proportion of sites with higher values of REC and CAL compared with never-users. The greatest increase in severity of CAL was found to be localized to sites on mandibular teeth, buccal surfaces, anteriors and molars, which may be a result of the retention of the ST product in the oral cavity.

Pradeep S. Anand, Department of Periodontics, People's College of Dental Sciences & Research Centre, Karond-Bhanpur Bypass Road, Bhanpur, Bhopal 462037, Madhya Pradesh State, India
Tel: +91-9981511098
Fax: +91-755-4005315
e-mail: deepusanand@yahoo.co.in

Key words: attachment loss; gingival recession; periodontitis; pockets; smokeless tobacco

Accepted for publication December 07, 2012

The effects of tobacco smoking on the prevalence and severity of periodontal disease have been well documented. Studies have clearly demonstrated that tobacco smoking can result in an increased loss of periodontal attachment as well as of alveolar bone (1–3). Current smokers have been reported to be at a greater risk of developing periodontitis than former smokers or nonsmokers (3,4), and quitting the habit of smoking has been shown to reduce the odds of having periodontitis (4,5). It has been reported that current smokers tend to have higher mean probing depths, recession scores and attachment loss, and lower gingival bleeding scores, compared with former smokers and nonsmokers (6–9). Smoking has also been shown to be associated with a reduction in alveolar bone height and an increase in the prevalence and severity of vertical bone loss and furcation involvement (1,2,7,8,10,11). Studies have shown that tobacco smoking also leads to a less favorable response to periodontal therapy (12–15). Pocket depth reduction and gain in attachment levels following nonsurgical and surgical periodontal therapy have been reported to be significantly less in smokers than in nonsmokers (12–14). Moreover, smokers also tend to show a greater loss of attachment than nonsmokers during the maintenance phase (14,15). Studies have also shown that the deleterious effects of smoking on the periodontium are most commonly seen on the palatal aspects of the maxillary teeth and on the mandibular anterior teeth (16–18). Probing depths and attachment loss in smokers have been shown to be higher in the palatal aspects of maxillary anterior teeth than on mandibular teeth and on facial aspects (16,18). Bone loss, in smokers, also tends to be more severe in the maxillary anterior region than in other areas of the dentition (17).

Furthermore, it has also been demonstrated that the other different forms of smoking tobacco, such as cigar (19,20), pipe (19,20) and water pipe (21), are all strongly associated with periodontal destruction. However, the effects of oral smokeless tobacco (ST) on the periodontium have not been studied as much as the effect of tobacco smoking on the periodontium.

Gingival recession, particularly adjacent to the site of tobacco placement or the tobacco-related mucosal lesion, has been reported to be the most common periodontal change associated with the use of ST, especially among younger individuals (22–25). ST use has also been reported to cause increase in gingival blood flow (26), gingival bleeding (27) and inflammation (28).

It has been suggested that ST use may be significantly associated with severe periodontal disease (29). Moreover, studies conducted among subjects in Asian countries have also indicated that oral ST use may be associated with increased periodontal destruction and attachment loss (30,31). However, at present, very little is known about the pattern of periodontal destruction among oral ST users. Nationwide surveys have shown that, in India, the habit of oral ST use is more prevalent than tobacco smoking (32,33). Several different forms of oral ST products, which differ in their contents, are available in India. These products include betel quid with tobacco, *zarda* (prepared by boiling pieces of tobacco leaves in water with slaked lime), *gutka* and *pan masala* (powdered tobacco mixed with areca nut, slaked lime and catechu), *khaini* (tobacco with slaked lime) and *mawa* (a mixture of areca nut, tobacco and slaked lime) (34). In the context of the Indian population, because of the widespread use of ST products in the country, the effects of oral ST on the periodontal tissues are important. Hence, the present study was undertaken to compare retrospectively the patterns of periodontal destruction among oral ST users and never-users with periodontitis.

Material and methods

Subjects

The study was performed as a retrospective study utilizing data collected from patients with periodontitis who had undergone a full-mouth periodontal examination at the Department of Periodontics, People's College of Dental Sciences & Research Centre, in Bhopal, Madhya Pradesh state, India, between November 2007 and March 2011. The patients whose data were available were categorized into two groups: oral ST users (who had the habit of oral ST consumption at least 10 times a day for the past 5 years and were still continuing the habit); and never-users (who never consumed any form of tobacco). Data collected from patients who had any systemic disease, a history of use of other forms of tobacco (smoking or dry snuff, which is inhaled), a history of discontinuing ST use or a history of any form of periodontal treatment or antibiotic therapy during the 6 mo period before the study, were excluded from the study. Data from 196 patients (81 ST users and 115 never-users) who had undergone periodontal examination were examined for the purpose of the study. Of these 196 patients, data from 21 ST users were excluded from the final analysis either because of the presence of the habit of smoking or because of a history of periodontal therapy within the previous 6 mo. Data from 26 never-users were also excluded either because of the presence of systemic disease or because of a history of periodontal therapy within the past 6 mo. Thus, data from 149 patients were available for the final analysis. These included 60 ST users and 89 never-users.

Data collection

The mean number of total teeth and the mean number of teeth in the maxillary arch, the mandibular arch and the anterior, premolar and molar regions were calculated. Probing depth, recession (REC) and clinical attachment loss (CAL) were recorded at six points on all permanent teeth excluding the third molars. All patients were examined by a single examiner. The clinical measurements were made using a periodontal probe (UNC-15; Hu-Friedy Chicago, IL, USA.) and were rounded off to the nearest millimeter.

Clinical variables

The probing depth measurements were categorized into four ranges (0–3, 4–5, 6–7 and $\geq$ 8 mm); the REC values into four ranges (0, 1–2, 3–4 and $\geq$ 5 mm); and CAL into five ranges (0, 1–2, 3–4, 5–7 and $\geq$ 8 mm). For each subject, the number of sites falling in different ranges of probing depth, REC and CAL were calculated for different regions (full-mouth sites, maxillary sites, mandibular sites, buccal sites, lingual sites, anterior teeth, premolars and molars) and were expressed as a percentage of the total sites available for the respective regions. Thus, the percentage of sites falling in different ranges of probing depth, REC and CAL were calculated for each subject separately for the different regions mentioned above. The mean scores for probing depth, REC and CAL, and the mean percentage of sites in the different ranges of probing depth, REC and CAL, were compared between ST users and never-users for all sites and for sites in the maxillary arch, mandibular arch, buccal surfaces, lingual surfaces, anterior teeth, premolars and molars. The proportion of subjects in each group with mean probing depth of $\geq$ 3 mm, mean REC of $\geq$ 1 mm and mean CAL of $\geq$ 4 mm were also compared. The present study was performed as part of a project to determine the effects of tobacco use on dental health and mortality, and the protocol for this study was approved by the Institution's Human Ethics Committee.

Statistical analysis

All data were entered into a personal computer and statistical analyses were performed using a statistical software package (SPSS version 16; SPSS Inc, Chicago, IL, USA.). One-way analysis of variance was used to compare the mean age, number of teeth, probing depth, REC and CAL between the ST users and the never-users. The chi-square test was used to compare the proportion of subjects in each group with mean probing depth of $\geq$ 3 mm, mean REC of $\geq$ 1 mm and mean CAL of $\geq$ 4 mm. The percentage of sites in different ranges of probing depth, REC and CAL among both the groups was transformed by arcsine transformation and subsequently analysed using the Student's

Table 1. Summary of data collected for the two study groups - smokeless tobacco (ST) users and never-users

Variable	ST users (n = 60)	Never-users (n = 89)	p-Value
Age	37.65 ± 8.15	31.99 ± 8.23	< 0.001*
Mean no. of teeth	27.32 ± 1.16	27.24 ± 1.51	0.726*
Mean no. of maxillary teeth	13.70 ± 0.59	13.64 ± 0.80	0.623*
Mean no. of mandibular teeth	13.62 ± 0.88	13.59 ± 0.96	0.892*
Mean no. of anterior teeth	11.80 ± 0.48	11.66 ± 1.09	0.360*
Mean no. of premolars	7.85 ± 0.51	7.97 ± 0.18	0.052*
Mean no. of molars	7.67 ± 0.65	7.61 ± 0.94	0.668*
Probing depth (mm)	3.37 ± 0.71	3.57 ± 0.82	0.119*
REC (mm)	0.91 ± 0.52	0.35 ± 0.39	< 0.001*
CAL (mm)	4.23 ± 0.88	3.82 ± 0.93	0.008*
Proportion of subjects with mean probing depth $\geq$3 mm	70 (42)	79.8 (71)	0.172**
Proportion of subjects with mean REC $\geq$ 1 mm	40 (24)	6.7 (6)	< 0.001**
Proportion of subjects with mean CAL $\geq$ 4 mm	61.7 (37)	31.5 (28)	< 0.001**

Values are given as mean ± standard deviation or as % (n). ST-users, smokeless tobacco users. Recession (REC) and clinical attachment loss (CAL).
*Analysis of variance.
**Chi-square test.

Table 2. Values obtained for the clinical variables probing depth, recession and clinical attachment loss at different oral sites in the smokeless tobacco (ST) and never-user study groups

Variable and site	ST users (n = 60)	Never-users (n = 89)	p-Value*
Probing depth			
Maxillary	3.49 ± 0.76	3.68 ± 0.93	0.185
Mandibular	3.24 ± 0.75	3.46 ± 0.80	0.096
Buccal	3.50 ± 0.84	3.62 ± 0.89	0.387
Lingual	3.24 ± 0.69	3.53 ± 0.82	0.030
Anterior	3.03 ± 0.80	3.36 ± 0.97	0.026
Premolar	3.24 ± 0.76	3.37 ± 0.87	0.350
Molar	4.02 ± 0.82	4.09 ± 0.86	0.614
Recession			
Maxillary	0.55 ± 0.44	0.26 ± 0.34	< 0.001
Mandibular	1.27 ± 0.76	0.45 ± 0.51	< 0.001
Buccal	0.83 ± 0.54	0.32 ± 0.38	< 0.001
Lingual	0.99 ± 0.65	0.39 ± 0.46	< 0.001
Anterior	1.21 ± 0.66	0.50 ± 0.51	< 0.001
Premolar	0.50 ± 0.45	0.16 ± 0.28	< 0.001
Molar	0.86 ± 0.62	0.35 ± 0.53	< 0.001
Clinical attachment loss			
Maxillary	3.99 ± 0.99	3.84 ± 1.08	0.394
Mandibular	4.48 ± 0.95	3.81 ± 0.92	< 0.001
Buccal	4.26 ± 0.98	3.81 ± 1.01	0.008
Lingual	4.21 ± 0.98	3.84 ± 0.94	0.022
Anterior	4.17 ± 0.99	3.72 ± 1.11	0.012
Premolar	3.70 ± 0.90	3.44 ± 0.92	0.089
Molar	4.86 ± 1.06	4.39 ± 1.06	0.009

Values are given in mm, as mean ± standard deviation.
*ANOVA.

t-test. As a statistically significant difference was observed in the mean age of the patients in the two groups, and age is known to be related to the clinical parameters of periodontitis, logistic regression analysis was performed to determine the association of ST habits, after adjustment for age, with mean REC $\geq$ 1 mm and mean CAL $\geq$ 4 mm. The statistical significance was fixed at 0.05.

Results

The mean age, the mean number of teeth and the full-mouth mean values for probing depth, REC and CAL for ST users and never-users is shown in Table 1. The mean age $\pm$ standard deviation of ST users was 37.65 $\pm$ 8.15 years, whilst that of never-users was 31.99 $\pm$ 8.23 years. There were no significant differences between the two groups in terms of the mean number of teeth. Although the mean probing depth was slightly higher in never-users (3.57 $\pm$ 0.82 mm) than in ST users (3.37 $\pm$ 0.71 mm), the differences were not statistically significant. However, mean REC and mean CAL were significantly higher in ST users than in never-users (REC: 0.91 $\pm$ 0.52 mm vs. 0.35 $\pm$ 0.39 mm, $p < 0.001$; and CAL: 4.23 $\pm$ 0.88 mm vs. 3.82 $\pm$ 0.93 mm, $p = 0.008$). Similarly, although there were a higher proportion of never-users with mean probing depth of $\geq$ 3 mm, the differences were not statistically significant. A higher proportion of ST users had mean REC of $\geq$ 1 mm (40%) and mean CAL of $\geq$ 4 mm (61.7%) compared with never-users (6.71% and 31.5%, respectively) and these differences were statistically significant ($p < 0.001$).

Probing depth

The mean probing depths for different regions of the dentition in ST users and never-users are shown in Table 2. As observed with the mean full-mouth probing depth, the mean scores of probing depth for the different regions of the dentition were higher in never-users. However, the differences in mean scores of probing depth were significant only for lingual sites ($p = 0.030$) and anterior teeth ($p = 0.026$). Table 3 shows the mean percentage of sites in different ranges of probing depth for the different regions in ST users and never-users. Generally, although shallow pockets (0–3 mm) were more prevalent in ST users and deeper pockets (> 3 mm) were more prevalent in never-users, the differences were not statistically significant except for pocket depths 0–3 and 6–7 mm for mandibular sites ($p = 0.036$ and $p = 0.024$, respectively), lingual sites ($p = 0.019$ and $p = 0.033$ respectively) and anterior teeth ($p = 0.012$ and $p = 0.008$ respectively).

Gingival recession

The mean REC scores were higher in ST users than in never-users for all the different regions (Table 2) and these differences were statistically significant ($p < 0.001$ for all regions). Figures 1–3 show the mean percentage of sites in different ranges of REC, for the different regions, in ST users and in never-users. Generally, never-users had a greater proportion of sites without REC (0 mm), whilst

Table 3. Mean percentage of sites in different ranges of probing depth at different oral regions in the smokeless tobacco (ST) and never-user study groups

Probing-depth ranges	ST users ($n = 60$)	Never-users ($n = 89$)	*p*-Value*
Full mouth			
0–3 mm	62.67 $\pm$ 18.79	56.63 $\pm$ 21.23	0.073
4–5 mm	28.05 $\pm$ 14.85	31.24 $\pm$ 15.34	0.192
6–7 mm	7.27 $\pm$ 6.02	9.64 $\pm$ 8.39	0.058
$\geq$ 8 mm	2.01 $\pm$ 3.01	2.49 $\pm$ 3.82	0.701
Maxilla			
0–3 mm	58.83 $\pm$ 21.38	54.05 $\pm$ 23.04	0.180
4–5 mm	30.69 $\pm$ 16.04	32.48 $\pm$ 15.81	0.438
6–7 mm	8.42 $\pm$ 7.18	10.50 $\pm$ 9.96	0.170
$\geq$ 8 mm	2.07 $\pm$ 3.37	2.98 $\pm$ 5.05	0.348
Mandible			
0–3 mm	66.70 $\pm$ 19.15	59.22 $\pm$ 21.61	0.036
4–5 mm	25.28 $\pm$ 15.63	30.00 $\pm$ 16.82	0.087
6–7 mm	6.08 $\pm$ 6.10	8.79 $\pm$ 8.06	0.024
$\geq$ 8 mm	1.94 $\pm$ 3.51	1.99 $\pm$ 3.46	0.761
Buccal			
0–3 mm	59.48 $\pm$ 22.58	55.47 $\pm$ 22.81	0.289
4–5 mm	29.21 $\pm$ 17.80	30.88 $\pm$ 16.57	0.488
6–7 mm	8.63 $\pm$ 7.17	10.85 $\pm$ 9.50	0.180
$\geq$ 8 mm	2.69 $\pm$ 3.73	2.81 $\pm$ 4.40	0.841
Lingual			
0–3 mm	65.86 $\pm$ 18.26	57.78 $\pm$ 21.73	0.019
4–5 mm	26.89 $\pm$ 14.04	31.60 $\pm$ 16.30	0.071
6–7 mm	5.92 $\pm$ 6.26	8.45 $\pm$ 8.18	0.033
$\geq$ 8 mm	1.34 $\pm$ 2.79	2.17 $\pm$ 3.90	0.116
Anterior			
0–3 mm	71.51 $\pm$ 19.22	61.84 $\pm$ 23.65	0.012
4–5 mm	22.11 $\pm$ 13.95	27.23 $\pm$ 16.50	0.074
6–7 mm	5.01 $\pm$ 7.32	8.76 $\pm$ 9.18	0.008
$\geq$ 8 mm	1.37 $\pm$ 3.23	2.17 $\pm$ 4.19	0.232
Premolar			
0–3 mm	65.64 $\pm$ 22.35	61.17 $\pm$ 24.18	0.272
4–5 mm	27.49 $\pm$ 17.70	30.04 $\pm$ 18.21	0.396
6–7 mm	5.90 $\pm$ 7.21	7.26 $\pm$ 9.82	0.428
$\geq$ 8 mm	0.99 $\pm$ 2.62	1.55 $\pm$ 3.29	0.229
Molar			
0–3 mm	46.19 $\pm$ 23.11	44.28 $\pm$ 21.70	0.600
4–5 mm	37.76 $\pm$ 19.52	38.49 $\pm$ 17.34	0.738
6–7 mm	12.10 $\pm$ 8.63	13.27 $\pm$ 10.30	0.446
$\geq$ 8 mm	3.98 $\pm$ 6.16	3.99 $\pm$ 7.59	0.821

*Student's *t*-test.

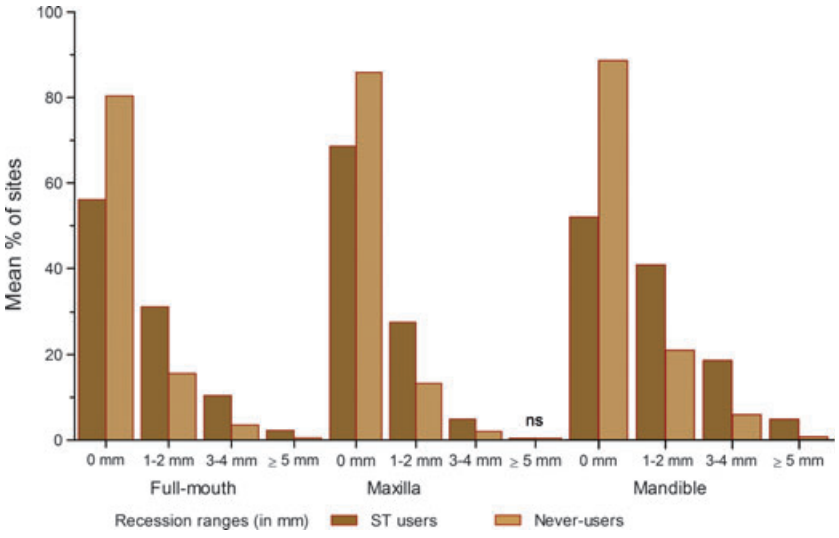

Fig. 1. Mean percentage of sites in different ranges of recession (REC) for full-mouth, maxillary and mandibular regions in the smokeless tobacco (ST) and never-user study groups. ns, not significant.

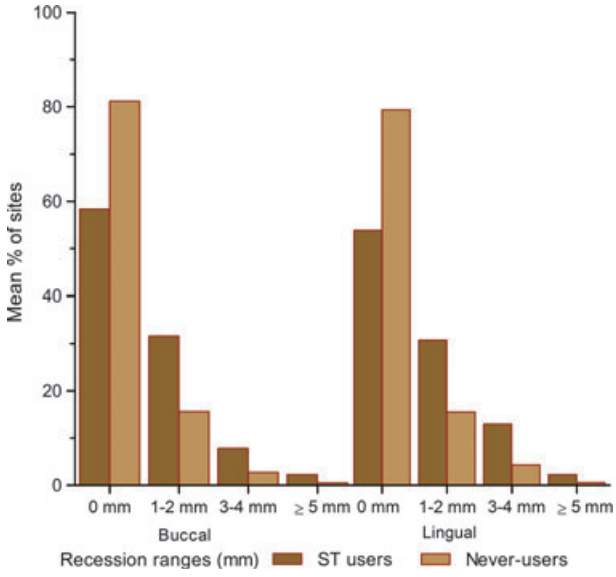

Fig. 2. Mean percentage of sites in different ranges of recession (REC) for buccal and lingual regions in the smokeless tobacco (ST) and never-user study groups.

ST users had greater proportion of sites in all other categories of REC and these differences were statistically significant ($p < 0.001$) except for the REC category of $\geq$ 5 mm for maxillary, premolar and molar teeth, where the differences were not statistically significant.

Clinical attachment loss

The mean CAL values for different regions of the dentition in ST users and never-users are shown in Table 2. The mean CAL scores were higher in ST users than in never-users for all regions and these differences were significant except for maxillary teeth and premolars. Table 4 shows the mean percentage of sites in different ranges of CAL for the different regions. ST users had a significantly higher proportion of sites with $\geq$ 8 mm CAL for full-mouth sites ($p = 0.003$), mandibular teeth ($p < 0.001$), buccal sites ($p = 0.002$), anterior teeth ($p = 0.040$) and molars ($p = 0.007$). ST users had a significantly higher proportion of sites with 5–7 mm CAL also for mandibular sites ($p = 0.008$) and anterior teeth ($p = 0.022$).

Logistic regression analysis

Logistic regression analysis, performed to determine the association of ST habits with mean REC $\geq$ 1 mm and mean CAL 4 mm, showed that, after adjusting for age, ST habit was strongly associated with mean REC of $\geq$ 1 mm (odds ratio = 6.665, p = 0.001) and mean CAL of $\geq$ 4 mm (odds ratio = 2.911, p = 0.003) (Table 5).

Discussion

This retrospective study was performed to compare the patterns of periodontal destruction among periodontitis patients with and without the habit of oral ST use. The results of the present study indicate that the patterns of periodontal destruction among ST users are different from those in never-users, with ST users having a higher severity and proportion of sites with REC and CAL than never-users.

Although the role of smoking on the prevalence and severity of periodontal diseases is well documented, very little data are available regarding the pattern of periodontal destruction among ST users. In the state of Madhya Pradesh, the location of the institution of the present study, the prevalence of oral ST use was reported to be significantly higher than smoking tobacco use, among male subjects as well as female subjects (35). The widespread use of ST products among this population provides an opportunity to understand, in greater detail, the effects of ST products on the periodontal tissues.

Comparison of various parameters of probing depth between ST users and never-users in the present study showed that the two groups did not differ significantly in terms of mean probing depth or percentage of sites with different ranges of probing depth. Similar results for probing

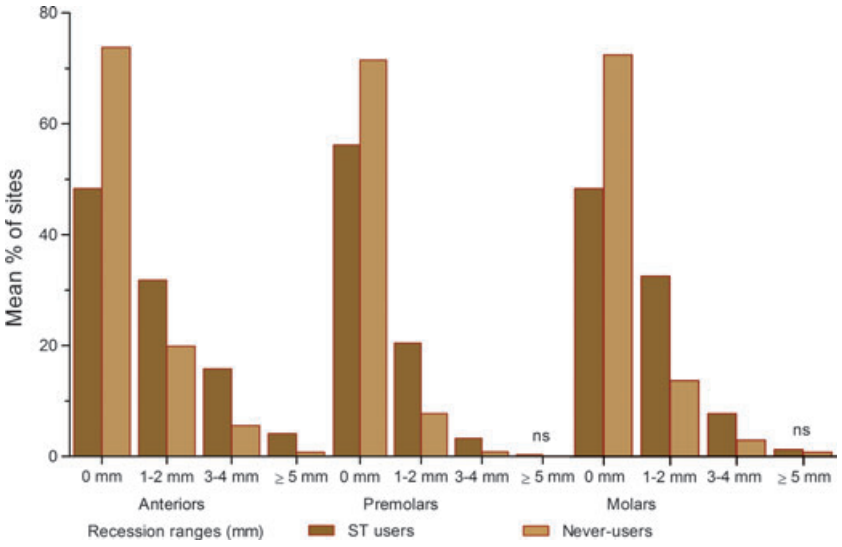

Fig. 3. Mean percentage of sites in different ranges of recession (REC) for anterior, premolar and molar regions in the smokeless tobacco (ST) and never-user study groups (ns, not significant).

depths have been reported by other investigators (22,23). However, in studies among south Indian pan chewers (30) and Bangladeshi subjects (31), it was reported that tobacco chewing was associated with greater pocket depths. In the present study, although it was observed that ST users had lower mean probing depth and a smaller percentage of sites with probing depth > 3 mm, these differences were not statistically significant. This may be a result of the fact that the present study was conducted among patients with periodontitis who, in either category, tend to have higher probing depths.

In the present study, it was observed that ST users had higher scores of mean REC and a greater proportion of sites with gingival REC than never-users. These differences for the various parameters of gingival REC were significant for all regions except for sites with REC of $\geq$ 5 mm in maxillary, premolar and molar sites. Whilst earlier studies showed that ST use was associated with increased gingival REC mainly at the site of tobacco placement (23–25), the findings of the present study showed that ST use may be associated with a generalized increase in gingival REC and that the habit can result in an increased prevalence, extent and severity of REC that may not be as localized as previously considered. Monten *et al.* (22), in a study conducted among Swedish adolescents, showed that ST use was associated with increased gingival REC in the maxillary anterior regions.

The generalized increase, observed in the present study, in the prevalence, extent and severity of gingival REC may be explained by the fact that during the habit of tobacco chewing, the harmful ingredients contained in the tobacco product may be moved around from one region of the oral cavity to the other, thus exposing all areas of the dentition to the deleterious effects of the tobacco contents. Although these products are not retained at all locations for a significant length of time, persistence of the habit over a long period of time may have a cumulative effect resulting in generalized damage to the periodontium. This may also explain why, unlike in smoking, the changes in the periodontal tissues resulting from ST use are not localized to a specific area of the dentition. It was also observed that the largest differences between the two groups in the mean scores of gingival REC and the percentage of sites with $\geq$ 3 mm of REC were seen for mandibular sites and anterior regions. This may suggest that retention of the ST product, which is usually in the buccal vestibule or anterior regions, can also cause a localized aggravation of the generalized injury to the periodontium resulting from ST use.

Whilst earlier studies (22,23,25) have shown that ST use results mainly in increased REC, with little effect on CAL except for that associated with gingival REC, the findings of the present study indicate that ST use is also associated with a generalized increase in attachment loss. The ST users in the present study showed significant increase in mean CAL and in the percentage of sites with $\geq$ 8 mm of CAL in all regions, except for maxillary sites and premolar teeth. The greatest differences in the parameters for CAL were observed for mandibular sites and anterior teeth. This increased attachment loss associated with ST use was observed in these locations, even in the presence of reduced probing depths, compared with never-users.

In the present study, it was observed that the mean age of ST users was significantly higher than that of never-users. As the prevalence and severity of periodontal diseases are known to increase with age (36,37), the higher mean age of ST users could have been a factor for the increased severity of periodontal destruction observed among this group of subjects in the present study. However, the results of the logistic regression analyses showed that the association between ST habit and increased severity of REC and CAL among the study population existed, even after adjusting for age.

The majority of the earlier studies have failed to show any effect of ST use on CAL. However, a few studies have shown that the habit may be associated with increased attachment loss (24,29,31). A recent study among rural male ST users reported significantly higher buccal attachment loss adjacent to ST lesions compared with contralateral sites that were not associated with the ST lesion (24). Another study among subjects in a south Indian population who had the habit of pan chewing, with or without tobacco, showed that more subjects among pan chewers with tobacco had higher scores for loss of attachment (30).

Table 4. Mean percentage of sites in different ranges of clinical attachment loss (CAL) at different oral regions in the smokeless tobacco (ST) and never-user study groups

CAL ranges	ST users (n = 60)	Never-users (n = 89)	p-Value*
Full mouth			
0 mm	0.42 ± 0.99	0.85 ± 2.32	0.132
1–2 mm	20.11 ± 14.93	26.69 ± 17.58	0.013
3–4 mm	39.00 ± 10.14	38.58 ± 11.23	0.773
5–7 mm	33.96 ± 14.67	29.85 ± 14.37	0.104
≥ 8 mm	6.52 ± 6.37	4.04 ± 5.81	0.003
Maxilla			
0 mm	0.66 ± 1.72	0.82 ± 2.06	0.424
1–2 mm	24.28 ± 18.73	27.86 ± 19.55	0.193
3–4 mm	39.94 ± 11.55	37.81 ± 13.28	0.288
5–7 mm	29.73 ± 16.41	28.84 ± 15.65	0.834
≥ 8 mm	5.40 ± 7.50	4.66 ± 7.28	0.334
Mandible			
0 mm	0.18 ± 0.62	0.88 ± 3.03	0.067
1–2 mm	16.04 ± 14.02	25.54 ± 18.54	0.001
3–4 mm	37.88 ± 13.29	39.29 ± 12.77	0.544
5–7 mm	38.18 ± 16.13	30.87 ± 15.79	0.008
≥ 8 mm	7.73 ± 8.36	3.43 ± 5.34	< 0.001
Buccal			
0 mm	0.46 ± 1.41	1.03 ± 2.62	0.088
1–2 mm	20.84 ± 17.81	28.36 ± 19.72	0.007
3–4 mm	37.47 ± 11.40	36.82 ± 13.11	0.726
5–7 mm	34.04 ± 15.55	29.40 ± 15.52	0.068
≥ 8 mm	7.19 ± 7.10	4.39 ± 6.18	0.002
Lingual			
0 mm	0.38 ± 1.35	0.66 ± 2.45	0.367
1–2 mm	19.37 ± 14.65	25.01 ± 17.65	0.036
3–4 mm	40.53 ± 13.43	40.33 ± 12.00	0.936
5–7 mm	33.88 ± 16.82	30.29 ± 15.20	0.249
≥ 8 mm	5.84 ± 7.79	3.69 ± 5.89	0.061
Anterior			
0 mm	0.63 ± 1.61	1.17 ± 3.03	0.143
1–2 mm	20.90 ± 18.19	29.16 ± 21.14	0.009
3–4 mm	38.90 ± 14.38	37.69 ± 14.01	0.581
5–7 mm	33.95 ± 16.54	27.89 ± 18.08	0.030
≥ 8 mm	5.62 ± 6.46	4.10 ± 6.92	0.040
Premolar			
0 mm	0.42 ± 1.21	0.77 ± 2.71	0.486
1–2 mm	26.11 ± 18.27	32.45 ± 21.57	0.057
3–4 mm	45.14 ± 13.72	41.55 ± 14.64	0.121
5–7 mm	25.32 ± 18.06	23.30 ± 16.96	0.479
≥ 8 mm	3.03 ± 5.57	1.95 ± 4.09	0.187
Molar			
0 mm	0.10 ± 0.46	0.43 ± 1.62	0.153
1–2 mm	12.86 ± 13.19	17.04 ± 14.02	0.026
3–4 mm	32.95 ± 14.24	36.86 ± 14.22	0.108
5–7 mm	42.80 ± 18.43	39.28 ± 16.44	0.253
≥ 8 mm	11.31 ± 12.02	6.42 ± 10.87	0.007

*Student's t-test.

Table 5. Results of logistic regression analyses performed with mean recession (REC) of ≥ 1 mm and mean clinical attachment loss (CAL) of ≥ 4 mm as the dependent variables

Dependent variable	Independent variables	Odds ratio	p-Value	95% confidence interval
Mean REC ≥ 1 mm	Smokeless tobacco use	6.665	0.001	2.277–19.507
	Age	1.151	<0.001	1.079–1.229
Mean CAL ≥ 4 mm	Smokeless tobacco use	2.911	0.003	1.422–5.960
	Age	1.038	0.080	0.996–1.083

The distribution of sites in different CAL categories in the present study showed that subjects using ST may demonstrate a greater percentage of sites with severe attachment loss compared with never-users, particularly in the mandibular sites, buccal sites, anterior teeth and molars. This localized effect on attachment loss may be caused by the retention of ST products in the oral cavity, which results in the harmful ingredients of these products remaining in contact with the tissues in these regions for extended periods of time. The increased attachment loss occurring in relation to the mandibular teeth probably explains the increased loss of mandibular teeth observed among oral ST users in our study, which was conducted among subjects from central India (38).

In the present study, lack of significant differences, between the two groups, in CAL parameters for maxillary sites and premolar teeth suggests that, although ST use may result in an increase in attachment loss, it may have very little effect on attachment loss in these regions.

Although the findings of the present study suggest that ST use may be associated with a generalized increase in REC and CAL, certain aspects of the study need to be considered when interpreting the study findings. As the present study was conducted exclusively among patients with periodontitis who were seeking treatment, we could not examine those subjects who were periodontally healthy or patients who had not reported to the department for periodontal treatment, and this may be considered a limitation of the study. As mentioned earlier, there are different types of commercially available ST products that differ in their contents and hence may also differ in their effects on the periodontal tissues (31). However, in the present study the ST users were not categorized according to the type of ST product used, and this may be considered as a limitation of the study. Moreover, we had included, among ST users, those subjects who had been using ST products for at least 5 years. The effect of ST on the

periodontium of patients who continued the habit for a longer duration of time (> 10 years), might have been different (24, 31) and was not studied. Nevertheless, by excluding subjects who used other forms of tobacco and subjects who had discontinued ST use, we were able to study the effects of current ST use on the pattern of periodontal destruction.

To conclude, the findings of the present study suggest that ST use may lead to a generalized increase in the severity of gingival REC and CAL, with ST users having a larger proportion of sites with severe REC and CAL. The greatest increase in severity of CAL was found to be localized to sites on mandibular teeth, buccal surfaces, anterior teeth and molars, and this is possibly related to retention of the ST product in the oral cavity. However, further studies evaluating the effects of ST on periodontal tissues in different regions of maxillary and mandibular arches, and in interproximal, midbuccal and midlingual sites, also need to be performed in order to obtain a clearer picture of the pattern of periodontal destruction among ST users. Currently, such a study, utilizing the same data set, is in progress. Moreover, further cross-sectional and prospective studies among the general population, including patients with periodontitis as well as periodontally healthy subjects, need to be performed with improved methodology and use of other variables such as alveolar bone level and microbial changes, and the stratification of patients based on type of ST product and duration of ST use, in order to improve our understanding of the effects of ST use on the periodontium. The improved knowledge regarding the effects of ST use on periodontal tissues may be helpful in implementing public health programs to increase the awareness of the general public regarding the potential health hazards associated with this highly prevalent habit. It may also serve as a guideline for a tobacco-cessation intervention in the periodontal treatment protocol for the management of patients using ST products. Tobacco cessation in dental-care settings can play an important role in improving the outcome of periodontal treatment as well as in improving the oral health of patients with the habit of ST use. Studies have shown that the tobacco-cessation intervention in dental-care settings can be effective in increasing the proportion of ST users who quit the habit (39–42). In India, as in other south-Asian countries, the habit of ST use is culturally ingrained among the population and is generally considered to be part of their lifestyle. This, combined with a general belief that ST is less harmful than smoking, accounts for the widespread use of ST products. These beliefs may also undermine the success of tobacco-cessation programs in these populations. Hence, tobacco-cessation programs initiated by dentists and periodontists should also aim at improving the knowledge of the patients regarding such misconceptions in order to tackle, more effectively, the burden of health hazards associated with ST consumption.

References

1. Bergstrom J, Eliasson S, Preber H. Cigarette smoking and periodontal bone loss. *J Periodontol* 1991;**62**:242–246.
2. Baljoon M, Natto S, Bergstrom J. The association of smoking with vertical periodontal bone loss. *J Periodontol* 2004;**75**:844–851.
3. Thomson WM, Broadbent JM, Welch D, Beck JD, Poulton R. Cigarette smoking and periodontal disease among 32-year-olds: a prospective study of a representative birth cohort. *J Clin Periodontol* 2007;**34**:828–834.
4. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. National Health and Nutrition Examination Survey. *J Periodontol* 2000;**71**:743–751.
5. Torrungruang K, Nisapakultorn K, Sutdhibhisal S *et al*. The effect of cigarette smoking on the severity of periodontal disease among older Thai adults. *J Periodontol* 2005;**76**:566–572.
6. Haffajee AD, Socransky SS. Relationship of cigarette smoking to attachment level profiles. *J Clin Periodontol* 2001;**28**:283–295.
7. Kerdvongbundit V, Wikesjo UM. Prevalence and severity of periodontal disease at mandibular molar teeth in smokers with regular oral hygiene habits. *J Periodontol* 2002;**73**:735–740.
8. Bergstrom J, Eliasson S. Noxious effect of cigarette smoking on periodontal health. *J Periodontal Res* 1987;**22**: 513–517.
9. Calsina G, Ramon JM, Echeverria JJ. Effects of smoking on periodontal tissues. *J Clin Periodontol* 2002;**29**:771–776.
10. Schuller AA, Holst D. An "S-shaped" relationship between smoking duration and alveolar bone loss: generating a hypothesis. *J Periodontol* 2001;**72**: 1164–1171.
11. Bergstrom J, Eliasson S. Cigarette smoking and alveolar bone height in subjects with a high standard of oral hygiene. *J Clin Periodontol* 1987;**14**:466–469.
12. Preber H, Bergstrom J. The effect of non-surgical treatment on periodontal pockets in smokers and non-smokers. *J Clin Periodontol* 1986;**13**:319–323.
13. Preber H, Bergstrom J. Effect of cigarette smoking on periodontal healing following surgical therapy. *J Clin Periodontol* 1990;**17**:324–328.
14. Ah MK, Johnson GK, Kaldahl WB, Patil KD, Kalkwarf KL. The effect of smoking on the response to periodontal therapy. *J Clin Periodontol* 1994;**21**:91–97.
15. Kaldahl WB, Johnson GK, Patil KD, Kalkwarf KL. Levels of cigarette consumption and response to periodontal therapy. *J Periodontol* 1996;**67**:675–681.
16. van der Weijden GA, de Slegte C, Timmerman MF, van der Velden U. Periodontitis in smokers and non-smokers: intra-oral distribution of pockets. *J Clin Periodontol* 2001;**28**:955–960.
17. Baharin B, Palmer RM, Coward P, Wilson RF. Investigation of periodontal destruction patterns in smokers and non-smokers. *J Clin Periodontol* 2006;**33**: 485–490.
18. Anil S. Study of the patterns of periodontal destruction in smokers with chronic periodontitis. *Indian J Dent Res* 2008;**19**:124–128.
19. Albandar JM, Streckfus CF, Adesanya MR, Winn DM. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. *J Periodontol* 2000;**71**:1874–1881.
20. Krall EA, Garvey AJ, Garcia RI. Alveolar bone loss and tooth loss in male cigar and pipe smokers. *J Am Dent Assoc* 1999;**130**:57–64.
21. Natto S, Baljoon M, Bergstrom J. Tobacco smoking and periodontal health in a Saudi Arabian population. *J Periodontol* 2005;**76**:1919–1926.
22. Monten U, Wennstrom JL, Ramberg P. Periodontal conditions in male adolescents using smokeless tobacco (moist snuff). *J Clin Periodontol* 2006;**33**:863–868.
23. Robertson PB, Walsh M, Greene J, Ernster V, Grady D, Hauck W.

Periodontal effects associated with the use of smokeless tobacco. *J Periodontol* 1990;**61**:438–443.
24. Chu YH, Tatakis DN, Wee AG. Smokeless tobacco use and periodontal health in a rural male population. *J Periodontol* 2010;**81**:848–854.
25. Rolandsson M, Hellqvist L, Lindqvist L, Hugoson A. Effects of snuff on the oral health status of adolescent males: a comparative study. *Oral Health Prev Dent* 2005;**3**:77–85.
26. Mavropoulos A, Aars H, Brodin P. The acute effects of smokeless tobacco (snuff) on gingival blood flow in man. *J Periodontal Res* 2001;**36**:221–226.
27. Amarasena N, Ekanayaka AN, Herath L, Miyazaki H. Association between smoking, betel chewing and gingival bleeding in rural Sri Lanka. *J Clin Periodontol* 2003;**30**:403–408.
28. Poore TK, Johnson GK, Reinhardt RA, Organ CC. The effects of smokeless tobacco on clinical parameters of inflammation and gingival crevicular fluid prostaglandin E2, interleukin-1 alpha, and interleukin-1 beta. *J Periodontol* 1995;**66**:177–183.
29. Fisher MA, Taylor GW, Tilashalski KR. Smokeless tobacco and severe active periodontal disease, NHANES III. *J Dent Res* 2005;**84**:705–710.
30. Sumanth S, Bhat KM, Bhat GS. Periodontal health status in pan chewers with or without the use of tobacco. *Oral Health Prev Dent* 2008;**6**:223–229.
31. Akhter R, Hassan NM, Aida J, Takinami S, Morita M. Relationship between betel quid additives and established periodontitis among Bangladeshi subjects. *J Clin Periodontol* 2008;**35**:9–15.
32. Gupta PC. Survey of sociodemographic characteristics of tobacco use among 99,598 individuals in Bombay, India using handheld computers. *Tob Control* 1996;**5**:114–120.
33. Subramanian SV, Nandy S, Kelly M, Gordon D, Davey Smith G. Patterns and distribution of tobacco consumption in India: cross sectional multilevel evidence from the 1998–9 national family health survey. *BMJ* 2004;**328**:801–806.
34. Sinha DN. *Report on Oral Tobacco Use and its Implications in South East Asia*. 2004.
35. Rani M, Bonu S, Jha P, Nguyen SN, Jamjoum L. Tobacco use in India: prevalence and predictors of smoking and chewing in a national cross sectional household survey. *Tob Control* 2003;**12**:e4.
36. Beck JD. Periodontal implications: older adults. *Ann Periodontol* 1996;**1**: 322–357.
37. Papapanou PN. Periodontal diseases: epidemiology. *Ann Periodontol* 1996;**1**: 1–36.
38. Anand PS, Kamath KP, Chandrashekar BR, Anil S. Relationship of smoking and smokeless tobacco use with tooth loss in a central Indian population. *Oral Health Prev Dent* 2012;**10**:243–252.
39. Severson HH, Eakin EG, Stevens VJ, Lichtenstein E. Dental office practices for tobacco users: independent practice and HMO clinics. *Am J Public Health* 1990;**80**:1503–1505.
40. Stevens VJ, Severson H, Lichtenstein E, Little SJ, Leben J. Making the most of a teachable moment: a smokeless-tobacco cessation intervention in the dental office. *Am J Public Health* 1995;**85**:231–235.
41. Gordon JS, Severson HH. Tobacco cessation through dental office settings. *J Dent Educ* 2001;**65**:354–363.
42. Gansky SA, Ellison JA, Kavanagh C, Hilton JF, Walsh MM. Oral screening and brief spit tobacco cessation counseling: a review and findings. *J Dent Educ* 2002;**66**:1088–1098.