INTRODUCTION

Cigarette smoking currently kills 4 million people annually and is estimated to kill up to 10 million annually by the late 2020’s (1).

Effects of smoking on oral and dental tissues include oral mucosal, dental and aesthetic stains and periodontal effects amongst others. Despite its known health hazards and life shortening effects, smoking still goes on un-abated in many parts of the world, Nigeria inclusive (2).

Literature exists on the effects of smoking on general health (3, 4) but literature on the oral hygiene and periodontal status of Nigerian smokers is almost non-existent. One of the very few studies in this area in Nigeria was carried out among factory workers (5). This current study on factory workers therefore, is an attempt at replicating existing studies in this area in order to have a basis for comparison. Tobacco smoking as it affects the periodontium is the primary focus of this study.

Nicotiana Tabacum was cultivated by Jean Nicot for medicinal purposes, about the time Christopher Columbus discovered “New World” in the 16th Century (5). Since the first observation of the adverse effects of smoking on the periodontium were made more than 160 years ago (6), literature has continued to build up on the effect of smoking on the periodontium on both periodontal status and treatment outcome. (7)
TAF Preventive Medicine Bulletin, 2010: 9(2)

Pindborg found that as high as 98% of soldiers with cases of acute ulcerative gingivitis (AUG) were smokers. Kowolik and Nisbet made similar observations in a more recent study. Studies have found poorer oral hygiene among smokers. Gingival bleeding has been consistently reported to occur less in smokers due to the nicotine–induced vasoconstriction in smokers’ gingivae as well as heavy gingival keratinization. Pocket depth measurements have also been found to be greater in smokers due to increased alveolar bone loss which is in turn due to decreased calcium absorption in the alveolar bone of smokers.

The periodontal effects of smoking have been found to be systematically mediated. For instance, chemotactic and phagocytotic ability of neutrophils have been found to be significantly defective in smokers. Still on systemic mediation of periodontal effects of smoking is the fact that nicotine alters the structure of fibroblasts, preventing their firm attachment to root surfaces, the biological effect on bone tissue, effects on the subgingival microflora, lower pocket oxygen tension, warmer subgingival temperature and also damage to deoxyribonucleic acid (DNA).

MATERIALS AND METHODS

This was a cross-sectional survey carried out among factory workers in Lagos state between August 2003 and December 2004 using a purposive sampling method.

A total of 254 adult factory workers (70 females, 184 males) aged 19 to 74 years were examined. One hundred and twenty three (66.8%) of the males examined were non-smokers, while 61 (33.2%) were smokers. All the females examined were non-smokers. Females were therefore not included in the analyses.

Oral Hygiene Index (OHI) and Community Periodontal Index (CPI) were utilized in this study to assess periodontal pocketing, five index teeth per arch (first and second molars bilaterally and the central incisor) making a total of ten index teeth per subject and findings were recorded in examiner-administered questionnaires.

The choice of sample size was based on the statistical formula for the comparison of two groups and from the example of similar studies. In the current study 123 non-smokers and 61 smokers were included in the analysis. The females were not included because they were non-smokers. Smokers on 10 cigarettes or less per day in the current study were classified as light smokers and more than 10 cigarettes per day, as heavy smokers a modification of the classification used by Devorah et al.

The subjects were clinically healthy adult factory workers with no signs and symptoms of any debilitating disease such as diabetes mellitus, significant crowding of teeth, known history and oral clinical signs of immunosuppressive therapy or epileptic therapy (phenytoin) and aggressive periodontitis. The control group in this study consisted of 123 non-smokers.

This study was authorized by the Medical Ethics Committee of the Lagos University Teaching Hospital and consent was obtained from the workers.

The Epi info (version 6.04d) was used for data analysis. Statistical tests yielding p-values less than 0.05 were considered significant.

RESULTS

Though subjects and controls in this study were found to be between 19 and 74 years of age (Table 1).

<table>
<thead>
<tr>
<th>Age group (Years)</th>
<th>Non-smokers</th>
<th>Smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td>19-24</td>
<td>36</td>
<td>29.3</td>
</tr>
<tr>
<td>25-34</td>
<td>32</td>
<td>26.0</td>
</tr>
<tr>
<td>35-44</td>
<td>21</td>
<td>17.1</td>
</tr>
<tr>
<td>&gt;45</td>
<td>34</td>
<td>27.6</td>
</tr>
<tr>
<td>Total</td>
<td>123</td>
<td>100</td>
</tr>
</tbody>
</table>

There were no statistically significant differences in self-reported gingival bleeding between smokers and non-smokers (p>0.05). (Table 2).

<table>
<thead>
<tr>
<th>Gingival bleeding</th>
<th>Non-smokers</th>
<th>Smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Did not know</td>
<td>n</td>
<td>%</td>
</tr>
<tr>
<td></td>
<td>8</td>
<td>6.5</td>
</tr>
<tr>
<td>Yes (Bleeding occurs)</td>
<td>29</td>
<td>23.6</td>
</tr>
<tr>
<td>No (no bleeding)</td>
<td>86</td>
<td>69.9</td>
</tr>
<tr>
<td>Total</td>
<td>123</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 2 shows history of gingival bleeding in subjects and controls. Twenty-nine non-smokers (23.6%) and 18 smokers (29.5%) had experienced gingival bleeding while cleaning. (X²=0.55, df=1, p=0.46).
Smokers however had significantly poorer oral hygiene than the non-smokers (p<0.001). Non-smokers had a significantly higher number of healthy sextants (Code 0) than the smokers in all age groups (p<0.001) while smokers were found to have a highly significant higher number of sextants with calculus than the non-smokers in all age groups. (p<0.001). (Table 3).

The number of sextants with code 1 was few. On analysis of sextants, smokers had less gingival bleeding (1.13%) than non-smokers (1.90%) It was however, not statistically significant (p=0.41).

Smokers were found to have a significantly higher number of teeth with pockets than the non-smokers in all age groups. (p=0.036) (Table 4).

Table 4 shows that smokers had 16 teeth with periodontal pockets (2.7%), while non-smokers had 11 teeth with periodontal pockets (0.9%). Smokers had relatively more periodontal pockets in all age groups. (X²=8.52, df=3, p=0.036).

Table 5 shows a significant association between smoking status and higher OHIS (poorer oral hygiene). (p<0.001)

This corroborates previous studies which have adduced different reasons including smokers’ neglect of oral health, (30) and higher mineralization potential of plaque fluid in smokers (5). The CPI is a poor index in the assessment of gingival bleeding since it could be clouded by higher cores because that status codes per individual lead to overestimation and
loss of sensitivity in the analysis (31). Analyses of sextants seem to give a clearer picture. However, in this study, neither the analysis of sextants with code 1 nor self-reported experience of gingival bleeding reflected the kind of differences reported in most studies (12,13). The possible explanation for this could be in the fact that most of the smokers seen were light smokers (ninety percent of the smokers seen in this study were light smokers and 61% had smoked for less than 5 years). The differences seen were therefore marginal (as seen in 1.13% in non-smokers versus 1.90% in smokers) and not strong enough to attain statistical significance (p=0.575).

Considering each CPI score, (i.e. code 0, 1, 2, 3, and 4) it was found that a significantly lower number of smokers had healthy sextants (code 0) than non-smokers (p<0.001). This corroborates previous studies (5,32). The current study however found a paradoxical relationship in the fact that older smokers had healthier sextants (Code 0) than the younger smokers. The reason for this is not clear. The older age group however, was found to have a highly significant greater number of pockets (p<0.001).

Most subjects and controls in previous studies had calculus (code 2) (5,32). Dafi (32) and other workers (30) found more deposits of calculus in smokers. The current study made similar observations. Dafi found calculus to be the greatest problem in smokers and non-smokers between 45 and 60 years of age (32). The current study however found more calculus in the younger smokers and non-smokers aged 19 to 34 years.

Pathological periodontal pocketing and attachment loss have been identified as the most important indicators of periodontal disease (33). In this study, actual number of pockets in ten index teeth was used to assess pockets to avoid losing data due to the scoring method of CPI. Several studies have found smokers to have deeper pathological periodontal pockets and alveolar bone loss (code 3 and 4) than non-smokers (5, 14, 15, 17). The findings in the current study agree with these previous studies. A significant association (p=0.036) was found to exist between smoking status and periodontal pocketing. These differences observed have two possible explanations. One plausible explanation is that the differences seen in periodontal pockets among smokers and non-smokers in the current study were merely due to poorer oral hygiene among the smokers seen and not a reflection of the impact of smoking. This explanation seems strengthened by the fact that there were higher calculus scores among smokers in all age groups (Table 3) and a strong association between oral hygiene index scores (OHIS) and periodontal pocketing (Table 5). While this explanation is very possible, two facts seem to suggest the contrary. One is the paradoxical relationship observed in the current study where the older age groups had fewer sextants with calculus and yet more periodontal pocketing than the younger age group while the second fact is the findings from several studies which have reported that the effects of smoking on the periodontium occur independent of local factors like plaque and calculus (18,19,26,34).

From the findings of this study, the major treatment needs of the smokers are for oral health education, professional oral prophylaxis then to lesser degree subgingival scaling and root planning and periodontal surgery in very few instances. This is in consonance with a previous study. (32) The non-smokers’ major treatment needs are in the area of oral health education and oral prophylaxis.

CONCLUSIONS

Smokers in this study had poorer oral hygiene, less healthy periodontium, more calculus, and more periodontal pocketing than non-smokers. The major treatment need for smokers and non-smokers was oral health and oral prophylaxis.

REFERENCES

7. Preber H, Bergstrom J. Effect of cigarette smoking on periodontal healing following


