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Research Article

Gingival Recession and Smoking in Young Adults: A Cross-sectional Survey

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Abstract

Background and aims. The aim of the present cross-sectional study was to evaluate the effect of smoking on gingival recession in smoking and non-smoking young adults.

Materials and methods. The subjects consisted of 158 adults (75 males and 83 females), including 85 smokers and 73 non-smokers, with an age range of 18-33 years. All the subjects were clinically examined and answered questions regarding their smoking habits. The clinical examination involved assessment of plaque, calculus, buccal probing depth and buccal gingival recession. In addition, the association between gingival recession and the following variables was assessed: age, plaque index, duration of smoking and the number of cigarettes smoked daily, educational status and plaque control methods. Data were analyzed using Student's *t*-test and the coefficient correlation test.

Results. Statistical analysis of data showed a significantly higher mean of recession in smokers compared to non-smokers ($P < 0.001$). The analysis of correlation of coefficients showed a positive association between gingival recession and the plaque index, duration of smoking and the number of cigarettes smoked daily. In addition, a significant negative association was noted between gingival recession and plaque control methods, as well as the educational status.

Conclusion. The results of the present study suggest that cigarette smoking causes gingival recession and in conjunction with several other risk factors may affect its prevalence and extent.

Key words: **Adults, gingival recession, Periodontal disease, smoking.**

Introduction

Gingival recession is a very common and undesirable condition.[1] It is defined as an apical shift of the gingival margin over the cemento-enamel junction (CEJ) and the exposure of the root surface to the oral environment.[2]

Gingival recession usually creates an aesthetic problem, especially when it affects the anterior teeth. There might be anxiety over tooth loss due to pro-

gressive destruction; it may also be associated with dentin hypersensitivity and/or root caries, abrasion and/or cervical wear, and erosive lesions because of the exposure of the root surface to the oral environment and an increase in dental plaque index.[3,4]

This condition can easily occur in patients with very vigorous oral hygiene, especially in association with malpositioned teeth and the use of a hard toothbrush. Other factors may play a role in recession, including destructive periodontal disease, inadequate toothbrushing, alveolar bone dehiscence, thin marginal tissue covering a non-vascularized root surface, high muscle attachment, frenum pull and occlusal trauma.[4]

Other causative factors that have been reported are iatrogenic factors related to reconstructive, conservative, periodontal, orthodontic or prosthetic treatments.[4,5]

Tobacco smoke is regarded as one of the main risk factors for destructive forms of periodontal disease.[6-10]

The relative risk of smokers for periodontitis has been estimated in several studies to range between 2.5 and 6.0; however, in young populations it may essentially be greater.[11] In addition, adolescents and young adults who smoke may have a higher risk to acquire more aggressive forms of the disease.[12]

Cigarette smoking and the presence of supragingival calculi are the factors most significantly associated with localized and generalized recession.[13]

Even though similar levels of plaque may be found in smokers and non-smokers, smokers have considerably more supra-gingival calculus.[14,15] Several studies have shown that smoking is a major risk factor for periodontal disease, affecting the prevalence, extent and severity of the disease.

Despite the fact that gingival recession has been associated with brushing frequency and technique, it should be noted that excessive use of a hard toothbrush and abrasive toothpaste might account for the development of more recession in smokers compared to non-smokers.[16]

The response of the microcirculation to plaque accumulation appears to be altered in smokers when compared with non-smokers.[17] In addition, the oxygen concentration in healthy gingival tissues appears to be lower in smokers than in non-smokers, although this condition is reversed in the presence of moderate inflammation.[18]

The aim of the present study was to evaluate the effect of smoking on gingival recession in a group of smoking and non-smoking young adults.

Materials and Methods

Subjects

The subjects included 158 young adults, 73 non-smokers (as the case group) and 85 smokers (as the control group), with an age range of 18-37 years. Mean ages of smokers and non-smokers were 24.5 ± 2.8 and 25.2 ± 2.4, respectively. The subjects were a sample of young males and females who sought dental treatment in a private clinic in Patra, one of the biggest cities in Greece.

All the examinations were performed by the author of the present article.

Both groups, non-smokers and smokers, were divided into 8 subgroups according to the age range: Group 1: 18-19 years; Group 2: 20-21 years; Group 3: 22-23 years; Group 4: 24-25 years; Group 5: 26-27 years; Group 6: 28-29 years; Group 7: 30-31 years; Group 8: 32-33 years old.

The subjects were in good general health as determined by a health questionnaire.

Ethics

All the subjects were informed about the evaluation to which they would be subjected and gave their informed consent to participate in the study.

Questionnaire

Before the clinical examination all the subjects filled in a questionnaire regarding personal data such as age, smoking habits (duration of smoking and the number of cigarettes smoked daily), educational status (primary, secondary, college, university) and use of dental home care devices (toothbrush, toothbrush and dental floss, none) and the last visit to a dentist.

The subjects were classified as smokers if they had smoked more than 10 cigarettes daily regularly for at least 2 years (all the smokers were cigarette smokers). The duration of smoking included smokers who had smoked for less than 2 years and smokers who had smoked for more than 2 years.[19]

Non-smokers were subjects who had never smoked.

Smoking was quantified by the number of cigarettes smoked daily (10-20 cigarettes daily and more than 20 cigarettes daily).

Clinical Examination

The subjects were clinically examined by the author of the present article.

The following indices were measured on each tooth: plaque index (O'Leary et al, 1973) (classified as <30%, 30-70%, and >70%) and gingival recession

rate from CEJ to the gingival margin measured by William's mm-graduated probe (Goldman-Fox/Williams DE Probe PD: PGF/W, Chicago, IL) in the mid-facial/buccal surfaces of all the teeth except for the third molars; linear measurements were obtained from the CEJ up to the gingival margin in the teeth presenting with gingival recession in order to estimate the vertical (apico-coronal) width of recession.

Presence or absence of supra-gingival plaque was recorded after disclosing soft deposits using a piece of cotton impregnated with 3% erythrocin solution and coating buccal tooth surfaces for a period of 30 seconds; the teeth and gingiva were dried with compressed air under dental unit light used as the light source for inspections.

In cases in which the CEJ was covered by calculus, hidden by a restoration or lost due to caries or wear, the location of the junction was estimated on the basis of the adjacent teeth.[20]

Exclusion criteria

None of the participants had received scaling and root planing procedures or periodontal treatment during the previous six months.[20]

Occasional and former smokers were excluded and subjects with systemic diseases were not selected.

Statistical analysis

The individual was the statistical unit.

For each participant average values of variables of recession and percentage of buccal surfaces covered by supra-gingival plaque or calculus were calculated.

As appropriate, Student's *t*-test and coefficient correlation test were employed to test the hypothesis of no differences or no correlations between non-smokers and smokers.

Data analysis was performed using the statistical package of SPSS ver.16.0 (SPSS Inc., Chicago, IL, USA).

Statistical significance was defined at $P<0.05$.

The correlation coefficient, typically denoted by r, shows the linear dependence between the two variables giving a value between +1 (positive) and -1 (negative).

Results

The means of gingival recession in smokers and non-smokers were 1.64 ± 0.25 and 0.62 ± 0.2, respectively, with statistically significant differences ($P<0.001$).

Frequencies of gingival recession in smokers and non-smokers (mean score) according to age is presented in Figure 1. A significant association was noted between gingival recession and age in smokers (case group) and non-smokers (control group) (r =

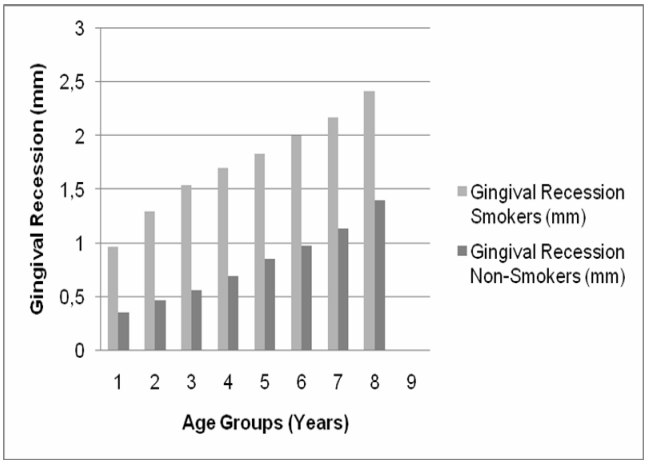

Figure 1. Gingival recession mean score according to age in non-smokers and smokers.

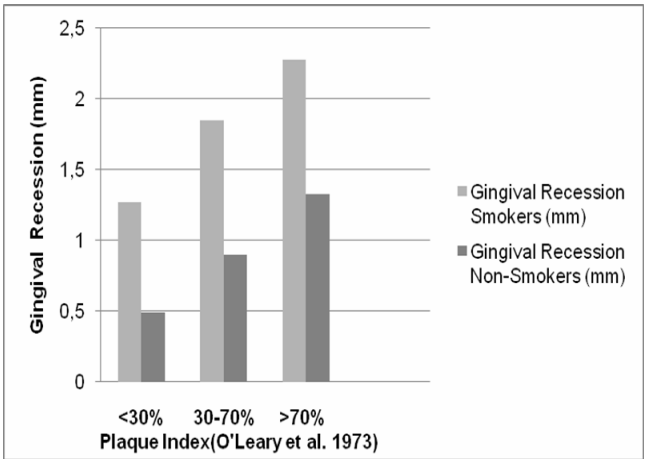

Figure 2. Gingival recession mean score according to plaque index (O'Leary et al, 1973) in non-smokers and smokers.

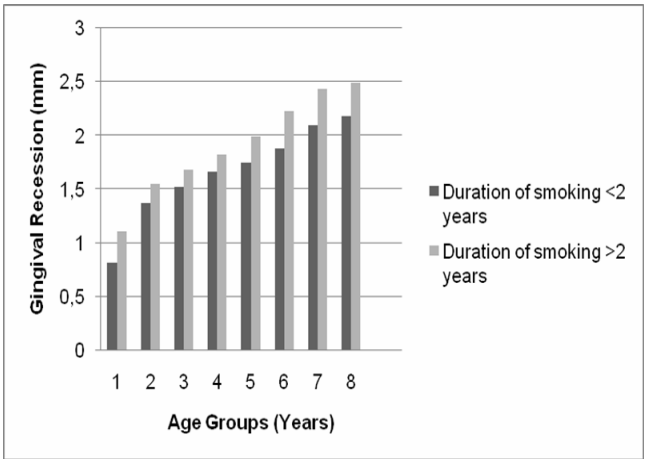

Figure 3. Gingival recession mean score according to age and duration of smoking in smokers.

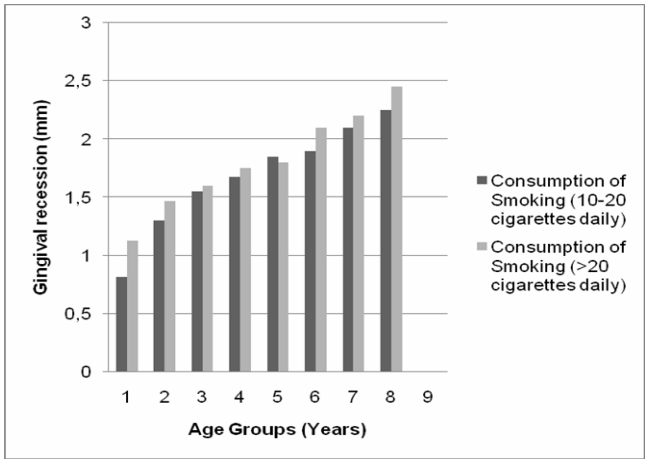

Figure 4. Gingival recession mean score according to age and consumption of cigarettes daily in smokers.

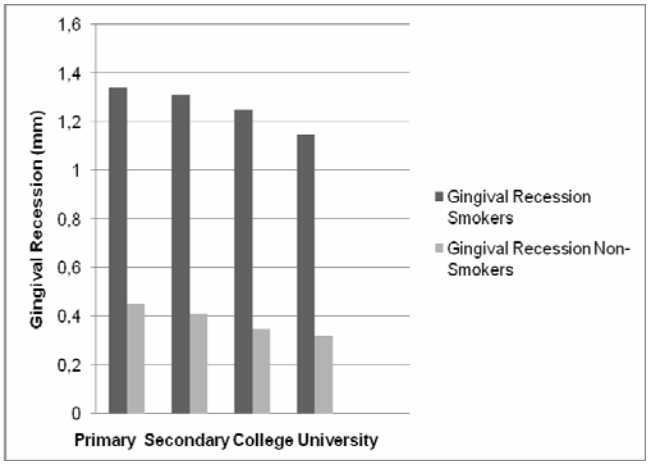

Figure 5. Gingival recession mean score according to educational status in non- smokers and smokers.

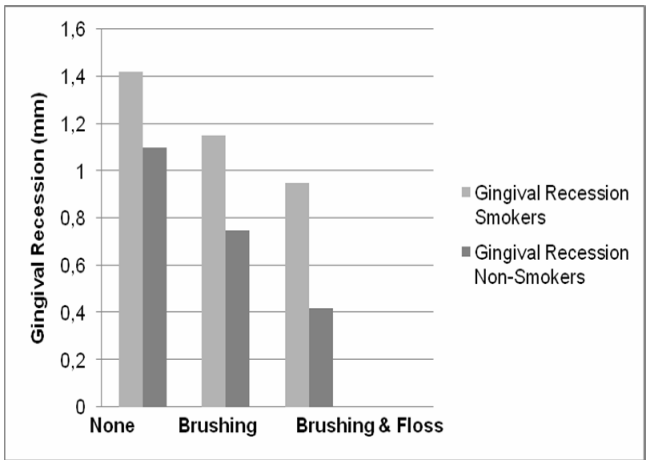

Figure 6. Gingival recession mean score according to methods of plaque control in non-smokers and smokers.

0.653, P < 0.001).

The frequency of gingival recession in relation to plaque index in the case and control groups is presented in Figure 2. The association of gingival recession and plaque index between the case and control groups was statistically significant (r = 0.312, P < 0.01) (Figure 3).

In the case group there was a significant association between gingival recession and duration of smoking (r = 0.481, P < 0.001), and gingival recession and daily consumption of cigarettes (r = 0.353, P < 0.001) (Figures 3 and 4).

A significant reverse association was noted between gingival recession and educational status (r = -0.248, P < 0.05), and between gingival recession and plaque control methods (r = -0.273, P < 0.05) (Figures 5 and 6).

Discussion

The present investigation was undertaken in order to evaluate the influence of tobacco smoke on gingival recession in a population of young adults.

In numerous cross-sectional studies, periodontal destruction has been associated with tobacco smoke. It has been suggested that smoking may also be a risk factor for gingival recession in adults with minimal periodontal destruction.[16]

The results of those studies revealed considerably more gingival recession in smokers compared to non-smokers after correcting for mean plaque index, gender, race and age in an analysis of covariance.

The results of the present study showed a significantly greater frequency of gingival recession in smokers compared to non-smokers, which is consistent with similar studies; however, some of those have suggested a negative impact on gingival recession and periodontal health from tobacco smoke.[21,22,23,24,25]

The frequency of gingival recession increased with age in both smokers and non-smokers in the present study. The significant association between gingival recession and age in the present study is consistent with the results of other studies.[26,27,28,,29] Only in one study age did not result in a significant contribution to gingival recession.[30]

In the present study plaque index was at a higher level in all ages and smoking groups compared to non-smoking groups. Previous studies have reported lower plaque indexes in all the smoking groups, indicating a higher standard of oral hygiene in their population samples.[15,25,30]

Regarding the duration of smoking, a significant association was noted between gingival recession and duration of smoking in the present study. This finding is consistent with other observations.[28-30] In another study a significantly greater frequency of diseased sites and a significantly greater bone height reduction were observed in heavy smokers as compared to light

smokers, indicating a dose-dependent relationship.[25]

The effect of smoking appears in middle age and heavy exposure is being associated with a greater frequency of gingival recession and periodontal diseases compared to light exposure.[25]

In the present study smoking was defined as the habit at the time of investigation regardless of possible changes over time. In addition, its range of variation was rather narrow: most subjects reported a consumption of over 20 cigarettes daily and smoking for over 2 years. Duration indicates the period from the beginning of the habit to the time of investigation and does not take into account the possible intervals of no smoking.

Smokers with a smoking duration of 2 years or more showed a greater frequency of gingival recession than those who had smoked for less than 2 years according to age. Similarly, smokers with a consumption of 20 cigarettes daily or more showed greater frequency of gingival recession than those who smoked less than 20 cigarettes per day. These findings are consistent with that the results of similar studies.[28,29] Furthermore, these findings are correlated with age.

In the present study the educational status of smokers and non-smokers was associated with gingival recession frequency, i.e. more educated cases had less gingival recession, especially in smokers, which might be attributed to the fact that more educated subjects have realized the value and importance of preventive dentistry and oral hygiene procedures, adopting proper habits and standards of oral hygiene, and following regular dental checkups. Similar observations have been recorded in other studies, indicating that the level of education is the most important contributor to buccal gingival recession.[27,30-,33]

As mentioned above the use of both toothbrush and dental floss decreased gingival recession more effectively than the use of just one method or no method, both in non-smokers and smokers. This finding is consistent with the results of another study which showed that dental floss is the most effective tool for removal of dental plaque from interproximal surfaces of teeth.[30,34,35]

The present observations clearly indicate an association between chronic smoking and gingival recession and show a causative role for smoking in periodontal disease. The role of smoking as a causative factor in gingival recession could be possibly explained via alterations in the immune response and local changes such as decreasing gingival circulation. Alterations in the immune response such as altered neutrophil chemotaxis, phagocytosis, and an increase in the production of PGE_2 by monocytes in response to LPS are also contributing factors. The exact changes in the immunologic mechanisms which are involved in the rapid issue destruction seen in smokers are currently unclear. However, the effects of smoking on periodontal disease progression are reversible with smoking cessation.[31]

The response of microcirculation to plaque accumulation appears to be altered in smokers when compared with non-smokers. With the development of inflammation, increases in gingival blood vessels were less prominent in smokers than in non-smokers.[13,17]

Longer studies with more participants are needed in order to further investigate the detrimental effects of smoking on periodontal tissues.

Conclusion

The mean of gingival recession in smokers was greater than that in non-smokers and the difference was statistically significant. Significant positive associations were noted between gingival recession and age, and between gingival recession and plaque index between smokers and non-smokers. In smokers significant associations were observed between gingival recession and duration of smoking, and between gingival recession and the number of cigarettes smoked daily. A significant reverse association was noted between gingival recession and educational status, and between gingival recession and plaque control methods.

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