Influence of smoking on salivary interleukin-8 levels in chronic periodontitis

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ABSTRACTS

Background: smoking is the major environmental risk factor that has been associated with the pathogenesis and progression of periodontal diseases. Interleukin-8 (IL-8), has been associated with the immunopathology of periodontitis.

Objectives: To determine the influence of smoking on salivary Interleukin-8 level from smokers and non-smokers with periodontitis and periodontally healthy control subjects.

Materials and Methods: Un-stimulated saliva samples were collected of 90 participants: 30 smokers and 30 non-smokers with chronic periodontitis, as well as 30 periodontally healthy control subjects. The clinical parameters such as the pocket depth, clinical attachment loss, plaque index, and gingival index were measured. IL-8 level in the saliva was measured by Enzyme Linked Immunosorbent Assay (ELISA) kit.

Results: It was found that the mean value of salivary IL-8 levels was significantly higher in smokers (461.76 ± 329.66 ng/L) than in non-smokers periodontitis (257.83 ± 247.19 ng/L) and the controls (96.55 ± 62.35 ng/L) ($p < 0.001$). Moreover, salivary IL-8 levels were significantly higher in smokers compared with non-smokers periodontitis ($p< 0.001$).

Conclusion: Smoking subjects showed increased level of salivary IL-8 and a worse periodontal condition than non-smoking subjects. Our results suggest that smoking alters an immune response which may contribute to an increased susceptibility to periodontal disease among smokers

Keywords: Smoking; Chronic periodontitis; Saliva; Interleukine-8. **(Received: 15/8/2018; Accepted: 30/9/2018)**

INTRODUCTION

Saliva is a fluid, which provides a primary growth environment for oral flora of the oral cavity. Salivary secretions are protected in nature because they preserve the oral tissues in a physiological state. As the physic-chemical properties are changed, it affects the microorganisms which grow in the mouth. Therefore, the protective effect of saliva may be accomplished by means of secretion rate, buffering capacity, phosphate and calcium concentration (biochemical substances, antibacterial components, and different antioxidants).[(1)] Saliva with and without stimulation is readily accessible via a totally non-invasive collection method, which contains locally produced microbial and host response mediators. Subsequently, the use of saliva for periodontal diagnosis and monitor response to treatment has been the subject of considerable research activity, and proposed markers for disease including enzymes, proteins, immunoglobulins, cytokines, host cells, etc.[(2)]

Periodontitis is an array of inflammatory diseases affecting the supporting tissues of teeth and resulting in periodontal pocket formation and alveolar bone resorption, which might eventually lead to tooth loss. It is a chronic inflammatory disease associated with gram negative anaerobic bacteria present in the dental biofilm, leading to an excessive inflammatory response, which is influenced by several risk factors, such as stress and host-specific factors or habits, such as smoking.[(3)] A complex interaction between these bacteria and host defensive capacity results in periodontal tissue breakdown.[(4)] The presence of periodontopathogens, such as *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* is considered the predominant etiologic agents in periodontitis,[(5)] triggers the expression of proinflammatory cytokines, such as Interleukin-1(IL-1), Interleukin-6(IL-6), Interleukin-8 (IL-8), tumor necrosis factor-α (TNF-α) and matrix metalloproteinases (MMPs), which have been related with the immunopathology of periodontitis. These mediators may affect the activities of leukocytes, osteoblasts and osteoclasts and stimulate the tissue remodelling process systematically and locally.[(6)] Interleukin-8 (IL-8), is formerly known as neutrophil-activating peptide-1 (NAP-1), a member of the CXC chemokine family, plays a significant role in the recruitment and activation of

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neutrophil during inflammation.[7] IL-8 is predominantly produced by gingival endothelial cells, fibroblasts, keratinocytes and macrophages in response to periodontal bacterial and bacterial components. IL-8 is a potent pro-inflammatory cytokine that regulates alveolar bone resorption during tooth crusade by acting early in the inflammatory response,[7,8] thus directly contributing to the progression of the periodontal lesion.[8,9] The expression of IL-8 mRNA and protein have been detected to be increased in chronically inflamed periodontal tissue, as well as gingival crevicular fluid of patients with periodontitis.[10,11] It is recognized that overproduction and excessive IL-8 mediated chemotactic and activation having effects on neutrophils in the inflamed gingiva may contribute to the periodontal tissue impairment.[12]

Smoking is the major risk factor, which is closely related with extent and severity of periodontal diseases.[13] Cross-sectional studies have consistently shown that smokers are two to six times more likely to have severe periodontitis than non-smokers.[14] In addition, clinical studies have recognized that severe periodontal disease with increased bone loss, greater periodontal attachment loss, more gingival recession and periodontal pocket formation are more frequent in smoking patients with periodontitis compared to non-smokers.[15,16] It is well established that smoking alters the host response, including changes vascular function, neutrophil/monocyte activities, adhesion molecule expression, cytokine and inflammatory mediator release, as well as antibody production.[17,18] These changes likely contribute to the negative effect of smoking on the reparative and regenerative potential of the periodontium. Various studies have stated the possible role of IL-8 in GCF as a biomarker for periodontal disease but less literature is available on its role in saliva.

Based on these findings, it seems reasonable to speculate that IL-8 may influence the initiation and progression of periodontitis. Thus, the aim of present study was to assess the influence of smoking on periodontal health by estimating the IL-8 level in saliva of smokers and nonsmokers with chronic periodontitis. The IL-8 levels in saliva among individuals with healthy periodontium, smokers and non-smokers with periodontitis were also compared and correlated with clinical findings.

MATERIALS AND METHODS

Study population:

The protocol for the present case-controlled study was approved by the Committee of ethics at College of dentistry/ Hawler Medical University. Written informed consent was obtained from all participants before conducting the study.

Ninety male participants, aged between 25-50 years old were selected by random sampling from patients attending outpatient clinics of the Department of Periodontitics at College of Dentistry, Hawler Medical University from February to August 2017. Subjects were categorized into three groups: Group-I comprised of 30 smokers with chronic periodontitis; Group-II comprised of 30 non-smokers with chronic periodontitis; and Group-III comprised of 30 non-periodontitis and non-smoking controls.

Periodontitis diagnosis was based on the classification of American Academy of Periodontology.[19] Patients with at least 30% of sites with loss of attachment were classified as having generalized chronic periodontitis and radiographic evidence of bone loss. Subjects must not have less than 20 standing teeth in their mouth. The periodontal patients group included individuals with probing pocket depth (PPD) ≥ 5 mm and clinical attachment level (CAL) ≥ 3 mm (> 30% affected sites). Furthermore, current smokers who smoked ≥ 10 cigarettes per day for not less than 2 years and who fulfilled the criteria of chronic periodontitis, were enrolled in the study. The control group consisted of individuals without a history of periodontal disease and attachment loss, as well as probing pocket depth (PPD) ≤ 3 mm.

All the participants were systemically healthy; had no medical history or clinical evidence of any acute or chronic diseases; had no intraoral inflammatory and non- inflammatory lesions; had no history of antibiotic and anti-inflammatory drug treatment within the previous 6 months; had no history of scaling and root planning for at least 6 months prior to sampling and recording.

Saliva sampling and analysis:

Unstimulated whole expectorated saliva (3 mL) was collected from each subject between 8:00 and 11:00 A.M. (at least 2 hours after a meal) before doing periodontal examination according to a modification in the method described by Navazesh.[20] Subjects were requested to rinse their mouth with distilled water thoroughly to remove exfoliated cells and food debris, which they

expectorated at least 3 mL of un-stimulated whole saliva into a 5mL sterile tubes while seated in an upright position. Collected samples were placed immediately on ice pack, then transported to the laboratory and centrifuged at 3500 rpm for 10 minutes. The supernatant structure was kept frozen at -40 ºC as aliquots till analysis. Salivary IL-8 levels were measured with an ELISA kit using Human Interleukine-8(IL-8) provided by Expert chem SERV (Catalog # DRE10290) according to manufacturer's instructions. The standard range was 50ng/L- 1000 ng/L.

Clinical Parameters:

Clinical measurements and radiographic examination of all participants were performed by a single experienced examiner after the collection of saliva sample. The Gingival index (GI),[21] plaque index (PI),[22] Probing pocket depth (PPD), Clinical attachment level (CAL) using UNC 15 probe were measured at six sites for all the present teeth except third molar.

Statistical analysis:

The descriptive data were expressed as mean and standard deviation (Mean ± SD). Comparison of clinical parameters of the three groups was analyzed using T-test and analysis of variance by one-way ANOVA test. Comparison of salivary IL-8 level among three groups was analyzed by one-way ANOVA test. Pair-wise comparison was performed by Tukey's multiple comparison tests to determine the difference of salivary IL-8 level between the groups. Possible correlations between salivary IL-8 levels and clinical periodontal parameters were assessed by the Spearman's Correlation Coefficients. Statistical analyses were performed with SPSS version 22 (SPSS, Chicago, IL, USA), and *P*-value<0.05 were considered statistically significant.

RESULTS

The mean age of the participants was 39.10 ± 4.71 years in Group I; 38.83 ± 3.29 years in Group II; and 38.40 ± 4.64 years in Group III with no significant differences among the three study groups ($p > 0.05$).

All clinical periodontal parameters were significantly higher in the patients groups as compared to the healthy control. On the other hand, the mean PPD, CAL, and PI were significantly higher in the smokers group compared to non-smokers ($P < 0.001$), while the mean GI score among the smokers (1.30 ± 0.28) was significantly less than that of non-smokers (2.51 ± 0.39) ($P < 0.001$) (Table 1).

The mean values of salivary IL-8 levels in smoker and non-smoker periodontitis groups were higher than in the control group (Figure 1). Salivary IL-8 level was significantly higher in smokers, followed in descending order by non-smokers and the controls with the mean values of (461.76 ± 329.66, 257.83 ± 247.19, and 96.55 ± 62.35 ng/L) respectively. With respect to salivary IL-8 levels, a pair- wise comparison between the groups also showed a highly statistically significant results ($p< 0.01$) (Table 2). By Spearman correlation coefficient analysis, in smokers and non-smokers with chronic periodontitis and control groups, salivary IL-8 levels did not show any significant correlation with clinical parameters of periodontal disease (PPD, CAL, PI, and GI) (all $p > 0.05$, data not shown).

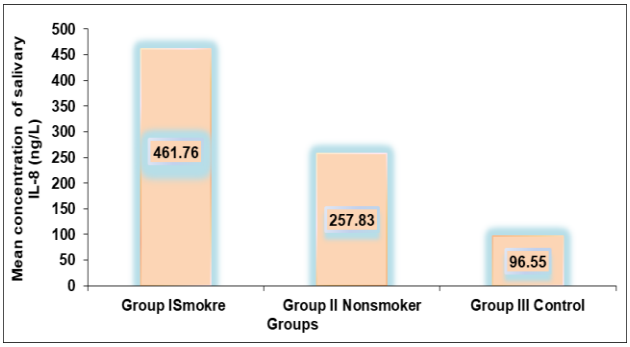

Figure 1: Mean levels of IL-8 in saliva of smoker and non-smoker periodontitis groups and control group

DISCUSSION

Smoking is one of the most important risk factors for periodontal disease. It has been shown that various components of tobacco smoke, nicotine act on the periodontal tissues causing destruction of the supporting tissues.[23] IL-8 is a multifunctional cytokine that plays a role in immune and inflammatory activities like recruitment and activation of neutrophils.[9] Thus, this study attempted to estimate and compare the level of IL-8 in saliva of smokers and non-smokers with chronic periodontitis and correlate these levels with the severity of periodontal diseases.

In the present study, smokers with chronic periodontitis had significantly increased PPD,

Table 1: Comparison of the three groups with respect to clinical periodontal parameters.

Clinical parameters§	Group I	Group II	Group III	P-value
PPD	5.82± 0.75	5.25 ± 0.27	1.97± 0.63	0.000*
CAL	4.93± 1.07	3.90± 0.73	0.00± 0.00	0.000*
PI	1.86± 0.42	1.27± 0.33	0.37± 0.15	0.000*
GI	1.30 ± 0.28	2.51± 0.39	0.79± 0.27	0.000**

§ values expressed as mean ± SD
PPD=probing pocket depth, CAL= Clinical attachment level, PI=Plaque index, GI= gingival index
Group I= Smoker, Group II= Nonsmoker, Group III= Control
*** Statistical difference at P< 0.05 by ANOVA test**
**** Statistical difference at P< 0.05 by Kruskal-Wallis test**

Table 2: Comparison of the three groups with respect to salivary IL-8 levels

Study groups	Salivary IL-8 Mean ± SD	Comparison	Mean differences Mean ± SD	P-value*
Group I: Smoker	461.76 ± 329.66	I vs II	209.6 ± 364.91	0.004
Group II: Nonsmoker	257.83 ± 247.19	I vs III	-209.6 ± 155.32	0.000
Group III: Control	96.55 ± 62.35	II vs III	-364.91 ± 155.32	0.030

***Significance at P< 0.05 by HSD (honest significant difference)**

CAL, and PI, compared to non-smokers (P < 0.001). However, the gingival index (GI) was significantly higher in non-smokers with chronic periodontitis than in smokers (P < 0.001). Previous studies [24,25] showed similar results. Jenifer et al.,[26] found a positive relationship between periodontal disease and smoking and have also reported a positive correlation with greater probing depth, attachment loss and plaque index in smokers. This supports the concept that smokers are generally presented with reduced gingival inflammation and bleeding on probing, compared to non-smokers, because smoking has a strong, chronic, dose-dependent suppressive effect on gingival inflammation and bleeding on probing.[27] Results significantly showed increased IL-8 levels in the saliva of smokers with periodontitis, compared to non-smokers with periodontitis and healthy controls (P< 0.001). Comparing between smoker with periodontitis and control group, salivary IL-8 level increased significantly and this relationship was highly significant ($P < 0.001$) the same results were obtained when non-smokers with chronic periodontitis and control group were compared. Again, there was significant increase in salivary IL-8 level ($P < 0.001$). Comparing smoker to non-smoker periodontitis groups, it was found that salivary IL-8 level increased significantly ($P < 0.001$). Our results demonstrated that smoking significantly influences salivary IL-8 levels in periodontitis patients.

Studies evaluating the effects of smoking on the expression of IL-8 level in periodontitis patients, have suggested that smoking increases the IL-8 expression in GCF.[25,28] It has also been reported that following periodontal therapy IL-8 levels decreased significantly in non-smokers, while in smokers it increased steadily and became significantly higher than in non-smokers.[28] It is primarily attributed to the fact that tobacco smoke activates more cells of the periodontium to express IL-8, thereby resulting in a local accumulation of polymorphonuclear cells (PMN's).[29] The results of our study explain the hypothesis that an exaggerated response by neutrophils occurs in response to increased IL-8 expression in smokers, suggesting an effect of smoking on the inflammatory responses against oral pathogens. Interestingly, increased GCF production of inflammatory molecules, such as IL-1β, IL-6, and IL-8, and suppression of anti-inflammatory molecules, such as IL-4, were discussed to imply increased periodontal destruction among smokers.[30] Moreover, Johnson et al.,[31] reported that nicotine can directly modify the production of cytokines and inflammatory mediators, causing increased IL-1, and IL-8 production by gingival keratinocytes. Nicotine also has a deleterious effect on the gingival and periodontal fibroblasts leading to further periodontal progression and periodontal tissue destruction.[32] A study on vitro by Lizheng et al.,[32] demonstrated that nicotine

up-regulates the production of IL-1β and IL-8 via the α7nAChR/NF-κB pathway in human periodontal ligament cells (PDL), which could be inhibited by pretreatment with PDTC or α-BTX, suggested that α7nAChR/NF-κB pathway might play a key role in the up-regulation of these cytokines in smoking-associated periodontitis.[32] Altogether, these studies underline the important role of smoking by assessing potential markers for periodontitis.

In contrast to our results, some studies have shown significantly lowered IL-8 level among smokers with chronic periodontitis in blood,[33] in GCF,[34] and salivary,[35] whereas other studies have reported no changes on the GCF levels of IL-1β,IL-4,IL-6 and IL-8 among smokers and non-smokers.[36] These studies have suggested that reduction in chemokines in smokers could contribute to the phenomena of impaired neutrophil chemotaxis and migration in the periodontium in spite of the presence of leucocytosis.[37] However, the increased level of salivary IL-8 in our study confirmed several findings in medical literature on positive association between smoking and increased expression of pro-inflammatory and chemokine molecules particularly IL-8. However, in the present study no correlation was found between clinical periodontal parameters PPD, CAL, PI, GI and salivary IL-8 levels.

CONCLUSION

In the present clinical cross-sectional study, smoking subjects showed increased level of salivary IL-8 and a worse periodontal condition than non-smoking subjects. Our results suggest that smoking alters an immune response which may contribute to an increased susceptibility to periodontal disease among smokers

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الخلاصة:

خلفية وهدف البحث: ان التدخين يعد احد العوامل الرئيسيه المساهمه في التسبب و تطور مرض الأنسجه ما حول الفم .(interleukin-8) يكون مشاركا في عملية تقدم و تطور حاله المرض من الناحيه المناعيه لمرض الأنسجه ما حول الفم.تحديد تأثير التدخين على مستوى ال (interleukin-8) اللعابي على مرضى(مدخنين و غير مدخنين) يعانون من مرض الأنسجه ما حول الفم و مقارنتها مغ أشخاص لا يشكون من مرض الأنسجه ما حول الفم و اعتبارهم عينه سيطرة(control).

المواد والطرق: عينات من اللعاب الغير محفز تم جمعه من 90 مشارك تم تصنيفهم على ثلاث مجاميع : 30 مدخن و 30 غير مدخن و 30 سريريا على صحه تامه في ما يخص الأنسجه ما حول الفم.المعايير السريريه مثل عمق الجيب اللثوي, مستوى فقدان الالتماس, فهرس البلاك و فهرس اللثه تم قياسهم. مستوى ال IL-8 في اللعاب تم قياسه بأستعمال عدة فحص انزيم الأرتباط المناعي (ELISA).

النتائج: من خلال هذه التجربه لقد وجد ان معدل القيمه لل (interleukin-8) اللعابي كان كبير جدا في مجموعه المدخنين الذين يعانون من من مرض الأنسجه ما حول الفم (461.76 ± 329.66 ng/L) بالمقارنه مع غير المدخنين الذين يعانون من من مرض الأنسجه ما حول الفم (257.83 ± 247.19 ng/L) وفي مجموعه ال (control) (96.55 ± 62.35 ng/L) ($p < 0.001$). اما مستويات ال (interleukin-8) اللعابي في اللأشخاص الذين يعانون المرض فقد كانت كبيره جدا في مجموعه المدخنين عنده مقارنتها مع غير المدخنين ($p< 0.001$) .

الأستنتاج: الأشخاص المدخنين اظهرو مستويات مرتفعه لل (interleukin-8) اللعابي ترافها تزايد في سوء حاله الأنسجه ما حول الفم بالمقارنه مع غير المدخنين. نتائجنا تقترح ان التدخين قد يغير في استجابه الجهاز المناعي و الذي قد يساهم في زيادة سوء حاله مرض الأنسجه ما حول الفم بين المدخنين الذين يعانون من مرض الأنسجه ما حول الفم .