Jordan Medical Journal

ORIGINAL ARTICLE

Effects of Cigarette Smoking on Host Defense Peptides in Saliva of Patients with Periodontitis

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Received: August 27, 2023

Accepted: Jun 23, 2024

DOI: https://doi.org/10.35516/jmj.v58i4.1663

Abstract

Background: smoking cigarettes is linked to numerous systemic and oral diseases, and affects both innate and adaptive immunity. Host defense peptides, often known as antimicrobial peptides (AMPs), are components of the innate immune system, classified as chemically active oligopeptides toxic to harmful microbes.

Aims: The goal of this research was to shed light on the effect of cigarette smoking on the production of host defense peptides in periodontitis patients.

Methods and Materials: Eighty-five participants were included in this study. Periodontal health assessed by: PLI, GI, BOP, PPD and CAL. Saliva samples were collected from all subjects. ELISA was carried out to estimate the levels of beta defensin and cathelicidin. Statistical analysis was performed using SPSS version 24. The ANOVA parametric test was used to determine and detect the differences between independent study groups, and the Tukey honestly significant difference (HSD)/post hoc test was performed to determine if there is any statistical connection between two data sets (intergroup comparison).

Results: The study findings showed both smokers and non smokers with periodontitis had considerably greater levels of beta defensin and cathelicidin than healthy controls. There was statistically significant reduction in beta defensin between smokers and non smokers ($p<0.001$); whereas no significant differences found between smokers and non smokers regarding cathelicidin level.

Conclusions: These findings suggested that, in the context of periodontal diseases, smoking can affect the protein levels of beta defensin but not the levels of cathelicidin.

Keywords: Cigarette smoking, antimicrobial peptides, saliva, innate immunity.

INTRODUCTION

Periodontal diseases have an array of inflammatory conditions that eventually cause tooth loss by affecting the gingiva, periodontal ligament and bone and being implicated in systemic inflammation [1]. Periodontitis is a chronic multifactorial inflammatory illness characterized by dysbiotic dental biofilms. Bacterial biofilm proliferation increases following altered homeostasis within the oral cavity [2], eventually, inflammation and disease arise as a result of the interaction between these microbiota and immune defense systems. The key molecular pathways in the pathogenesis of the periodontal diseases have been identified and are connected to activation of host-derived mediators that allow for apical down growth of the bacterial biofilm along the root surface, junctional epithelium loss, and fibers damage of the marginal periodontal ligament [3]. Although periodontal pathogens are the main factor responsible for the onset of periodontitis, the host immune response also influences disease progression and severity [4]. Host defense peptides (HDPs), which are also referred to as antimicrobial peptides (AMPs), are a class of peptides that possess multiple functions. Their primary biological function (in vivo) is to eradicate harmful microorganisms, including both Gram-negative and Gram-positive bacteria, fungi, and viral infections. AMPs are evolutionarily conserved molecules that participated in several aspects of innate immune system, found in all complex living organisms [5, 6]. Antimicrobial peptides (AMPs) are positively charged, amphipathic molecules that adopt an alpha helical conformation. They typically consist of 12-100 amino acid residues. Antimicrobial medications primarily eliminate bacteria by means of membrane interactions [7]. This mechanism of action varies from that of classical antibiotics, which are bacteriostatic drugs that target particular cellular processes, whereas AMPs are bactericidal with multiple potential targets apart from their membrane disruptions features [8].

Human beta defensin (HBD) participates in innate immune defense during health and regulates the interaction between the microbiota and epithelial surfaces during disease [9]. The primary barrier against oral bacteria, the oral epithelium, is continually exposed to over 500 bacterial species [10]. Yilmaz et al. [11] reported that patients with periodontitis had higher levels of salivary HBD in comparison to healthy controls. Cathelicidin is an important AMP in periodontal tissues protection from microbial insults in the oral cavity [12]. Cigarette smoking is regarded as one of the most significant environmental risk factors, and it has a direct impact on both the risk and prognosis of periodontitis [13]. It is generally accepted that smoking has several negative effects on periodontal tissues, including a chronic reduction in blood flow, changed neutrophil function, altered production of cytokines and growth factors, inhibition of fibroblast proliferation and adhesion, and reduced collagen and vascularity generation are some of these impacts [14, 15]. This study was conducted to identify possible influences of cigarette smoking on the release of host defense peptides (beta defensin and cathelicidin) in smoker and non-smoker periodontitis patients.

SUBJECTS AND METHODS

Study design: This study was a case-control and cross-sectional study. It was carried out at the Department of Basic Sciences, College of Dentistry, University of

Baghdad, between November 2022 and February 2023. The study was authorized by the ethical committee at Dentistry College/ University of Baghdad (Project no 797823 in 9\2\2023).

Subjects

85 patients were enrolled in this study [The smoker group included 30 patients who had smoked cigarettes regularly for at least 10 cigarettes per day over the past two years [16], the non-smoker group include 30 patients with periodontitis and the remainder 25 subjects were assigned to the healthy control group. The inclusion criteria used in this study including the following: Good general health with no history of any systemic disease; patients had periodontal pockets that were equal to or more than 4mm and had at least 20 or more natural teeth with loss of attachment more than 4mm and participants had not had periodontal therapy during the previous six months. The exclusion criteria used in this study including the following: Systemic diseases which may affect the progression of periodontal disease; previous periodontal therapy for the last six months; the use of antibiotics and/or anti-inflammatory medication in the last three months.

Sample size

With the help of the website EPITOOLS (https://epitools.ausvet.com.au/case controls), the appropriate size of the sample was determined using a confidence interval of 95% and an error margin of 5%.

Oral Examination

Clinical examination was performed by a specialist dentist for every subject who was enrolled in the study. The periodontal status of all teeth was assessed using periodontal probe of William's graduation, the clinical periodontal parameters were included: Plaque index, gingival Index, bleeding on probing, probing pocket depth and clinical attachment level.

Saliva Sample collection

All participants were instructed not to eat or drink anything (except water) for at least 1 hour prior to saliva collection. participants were instructed to rinse their mouths repeatedly with sterile water and wait for 1-2 minutes for the water to flow out, then up to 2 ml of whole unstimulated saliva was collected into tubes. The collected saliva was centrifuged at 3000 rpm for 10 minutes, and the resultant supernatant was collected and stored at -20˚C in eppendorf tubes until further processing.

Measurement of Salivary Beta Defensin and Cathelicidin Levels

Beta defensin and cathelicidin concentrations in saliva were quantified using an ELISA according to the kit's instruction booklet (Shanghai, China).

Statistical analysis was performed using SPSS version 24. The ANOVA parametric test was used to determine and detect the differences between three or more independent groups, and the Tukey honestly significant difference (HSD)/post hoc test was performed to determine if there were any statistical connection between two data sets (intergroup comparison).

RESULTS

The current study included 60 periodontitis patients and 25 subjects with healthy periodontium as controls. The periodontitis patient groups included 30 males and 30 females, whereas the control group included 13 males and 12 females. The mean age of smoker periodontitis patients was 41.10± 12.89 years and 35.90± 8.86 for non-smoker periodontitis, whereas for healthy individuals the mean age was 34.27±9.79 years with no significant

differences (p>0.05) observed between them. Moreover, clinical periodontal parameters (PI, GI and BOP) were significantly higher in patient groups (smokers and non-smokers) as compared to the control group. On the other hand, there were no significant variations regarding all periodontal parameters between the smoker and non-smokers patient groups (p>0.05), as shown in (Table-1).

Table 1: Age and clinical parameters in the three study groups

Age (years) and clinical PD parameters	Study groups			*p*-value
	Smoker with periodontitis (n=30)	Non-smoker with periodontitis (n=30)	Healthy controls (n=25)	
Mean ± SD	41.10± 12.89^{aNS}	35.90±8.86^{bNS}	34.72±9.79^{cNS}	0.620
PI	1.89±0.69^{aNS}	1.30±0.55^{b*}	0.76±0.38^{c*}	0.000
GI	2.13± 0.62 ^{aNS}	1.86± 0.80^{b*}	0.60±0.10^{c*}	0.000
BOP	0.35± 0.04^{aNS}	0.36±0.02^{b*}	0.04± 0.0^{c*}	0.000
PPD	4.18±1.58 ^{aNS}	4.07± 1.52	-	0.830
CAL	4.76±2.95 ^{aNS}	4.66± 2.08	-	0.138

a: a comparison of smokers and nonsmokers with periodontitis; b: comparison of smoker with periodontitis and control groups; c: comparison of non-smoker with periodontitis and control groups

According to the findings of this research, there were statistically significant differences (p< 0.05) in the mean salivary levels of beta defensin and cathelicidin in the three different study groups, as shown in Table 2. However, the mean difference and intergroup comparisons of mean values of beta defensin and cathelicidin for all pairs of groups was observed in Table 3, indicated a significant decrease (p<0.05) in beta defensin level in smokers' group than non-smokers group. There were also significant differences between the non-smokers group and the control group, but not between the smokers' group and the control group (p>0.05). In addition, there were no statistically significant variations in cathelicidin levels between the smoking and nonsmoking groups (p>0.05), while there were statistically significant differences between the control group and each patient group (smokers and nonsmokers p<0.05).

Table 2: The difference in salivary mean levels of beta defensin and cathelicidin in studied groups

Host defense peptides	Study groups			ANOVA (*p*-value)
	smoker periodontitis (n=30)	non-smoker periodontitis (n=30)	Healthy control (n=25)	
Beta-defensin				
Range	(101-748)	(192-971)	(82-588)	0.000*
Mean± SD	335.7±149.8	536.1±222.1	296.7±153.1	
Cathelicidin				
Range	(18-49)	(16-59)	(8-41)	0.001*
Mean± SD	33.55±9.3	37.84±10.6	27.51±7.73	

Table 3: Intergroup comparisons of beta defensin and cathelicidin mean levels between all study groups.

Grouping	Mean difference	Tukey's HSD (*p*-value)
Beta-defensin		
Smoker vs. non-smoker	-200.2	0.000*
smoker vs. control	39.47	0.697NS
non-smoker vs. control	48.62	0.000*
Cathelicidin		
Smoker vs. non-smoker	-4.29	0.188NS
smoker vs. control	6.64	0.049*
non-smoker vs. control	10.33	0.000*

DISCUSSION

The oral epithelium functions as a barrier that prevents pathogens and other potentially hazardous elements, such as cigarette smoke, from invading; Antimicrobial peptides (AMPs) are commonly secreted in reaction to oral microorganisms or bacterial toxins, serving as a protective mechanism against the growing

colonization of pathogens. This renders them appropriate biomarkers for the identification of periodontal diseases [17,18]. According to our study inclusion criteria, it was expected that clinical measurements would differ between those with and without periodontitis. The periodontal conditions of patients in both groups with periodontitis (smokers and non-smokers) were similar, allowing the evaluation of smoking habit effect on AMP's regulation without the interference of different paeriodontal disease severity.

The clinical findings of the current research demonstrated that smoking has a considerable impact on the levels of salivary beta-defensin, but has no significant effect on the levels of cathelicidin. Reduced peptides levels were observed in smoker patients with periodontitis compared to the non-smokers with periodontitis. In fact, the mechanisms by which cigarette smoking exacerbates inflammation in chronic inflammatory diseases are not fully understood and the effect of smoking and its toxic products on AMP regulation should be futher explored. It is generally accepted that smoking inhibits the activation of a person's innate immune system in response to bacterial infection. Inadequate functioning of the innate immune system has been shown to potentially play an important role in smoking-related disorders. Our study findings consistent with results of Soldati et al, 2022 study which suggested that smoking has a negative impact on GCF HBD levels; reduced levels of GCF HBD were observed in diseased sites of smokers compared to the same sites of non-smokers [19]. In support of these findings, an in vitro study conducted by An et al., 2021 revealed that nicotine may function as an anti-inflammatory agent and has an inhibiting impact on the production of beta-defensin [20].

On the other hand; in 2014, Ertugrul and colleagues found that smoker patients with gingivitis or with generalized severe periodontitis displayed greater GCF levels of hBD 2 than non-smoker patients did. [21]. While a previous study found a significant decrease in hBD 1 and 2 gene expression in non-inflamed gingival biopsies of smokers compared to non-smokers [22]. Evidence suggests that smoking has been linked with alterations in the oral microbiome in periodontitis patients. In subgingival plaque samples, smokers had a larger number of periodontopathogenic microorganisms than nonsmokers [23]. Furthermore, smoking has been associated with cytotoxic and immunosuppressive effects on periodontal tissues. It reduced blood flow, the attachment and migration of fibroblast cells, the proliferation of oral tissue, as well as influenced neutrophil function. As a result of altered neutrophil functions in smokers, it is probable that neutrophils cannot migrate to inflammatory sites within the time limit needed or cannot function effectively. This may be the reason that AMPs are expressed at low levels, in response to pathogenic invasion and inflammation [24, 25].

The results of the present study showed that low levels of beta defensin and cathelicidin were encountered in healthy controls as compared to patients groups. These findings were similar to previous studies' findings [26, 27]. On the contrary, some results indicated that chronic periodontitis patients expressed less HBDs than healthy controls [28]. The location and expression of AMPs can be affected by periodontal infection and inflammation [29]. The relationship between AMP levels and periodontal disease remains controversial in the scientific literature.

Cathelicidin is a crucial AMP associated with the defense mechanism of periodontal tissues against microbial invasion [30].

Compared to healthy gingival tissue, its production was greater in inflamed gingival tissue [31]. In the current research, a comparison of smokers and non-smokers in relation to the cathlicidin level revealed no statistically significant differences; this result coincides with Lopes et al (2021) findings [32]. In a previous study of (Türkoğlu et al, 2016) observed that non-smoker patients with chronic periodontitis exhibited a greater level of cathelicidin in their gingival crevicular fluid (GCF) compared to smoker patients with periodontitis [33]. Additionally, it was proven by Takeuchi et al. in 2012 that salivary cathelicidin concentrations have been associated with a breakdown of periodontal tissue in individuals who have chronic periodontitis [34]. Moreover, Turkoglu et al. (2009) discovered that individuals who suffered from chronic periodontitis had considerably greater levels of LL-37 in the gingival crevicular fluid than periodontally healthy patients had [33]. Further investigations are suggested to determine the precise association between the severity of periodontitis and the level of this peptide in smokers and nonsmokers considering the conflicts reported in the findings of clinical studies. The sample size in this study was relatively small, and only two categories of antimicrobial peptides (AMPs) were studied, despite the fact that other types also have an antimicrobial function that needs to be clarified.

CONCLUSION

Based on the present data, it might be concluded that salivary cathelicidin levels are not significantly influenced by smoking in the context of periodontal disease. The present study reveals that smoking suppresses beta defensin levels in smokers with chronic periodontitis. This finding might support the effect of smoking on the pathogenesis of periodontal disease. Further studies with larger sample sizes are needed to evaluate the effect of smoking on AMPs in chronic periodontitis patients with varying degrees of the disease severity. Also, studies investigating the effect of smoking on the expression of cathelicidin and beta defensin in gingival tissues obtained from patients with periodontitis would lead to better understanding of the effect of smoking on periodontal disease pathogenesis.

ACKNOWLEDGEMENTS

Thanks to all participants in the present study.

CONFLICT OF INTERESTS

The authors declare no competing interests.

REFERENCES

Kinane DF, Stathopoulou PG, and Papapanou PN. Periodontal diseases. Nature reviews Disease primers. 2017; 3:1-14.

2. Al-Obaidi MJ, Al-Ghurabi BH. Potential Role of NLRP3 Inflammasome Activation in the Pathogenesis of Periodontitis Patients with Type 2 Diabetes Mellitus. J. Med. Chem. Sci., 2023; 6: 522-531.

3. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. Journal of periodontology. 2018; 89: S159-S172.

4. Al-Ghurabi BH. The Role of Soluble TLR-2 in the Immunopathogenesis of Gingivitis. International Medical Journal. 2021; 28:37 – 39.

5. Zasloff M. Antimicrobial peptides of multicellular organisms. Nature 415. 2002; 389–395

6. Phoenix DA, Dennison R, Harris F. Antimicrobial

Peptides: Their History, Evolution, and Functional Promiscuity, First Edition, Wiley. 2013;1-18.

7. Lei J, Sun L, Huang S, Zhu C, Li P, He J, et al. The antimicrobial peptides and their potential clinical applications. Am. J. Transl. Res. 2019;11: 3919–3931.
8. Zhang QY, Yan ZB, Meng YM, Hong XY, Shao G, Ma JJ, et al. Antimicrobial peptides: Mechanism of action, activity and clinical potential. Military Medical Research.2021; 8: 1-25.
9. Zhu C, Tan H, Cheng T, Shen H, Shao J, Guo Y, et al. Human β-defensin 3 inhibits antibiotic-resistant Staphylococcus biofilm formation. journal of surgical research. 2013; 183: 204-213.
10. Shaker ZF, Al-Ghurabi BH. Study the role of proinflammatory and anti- inflammatory cytokines in Iraqi chronic periodontitis patients. Journal of baghdad college of dentistry. 2012; 24: 164-169.
11. Yilmaz D, Topcu AO, Akcay EU, Altındis M, Gursoy UK. Salivary human beta-defensins and cathelicidin levels in relation to periodontitis and type 2 diabetes mellitus. Acta Odontologica Scandinavica,2020; 78: 327-331.
12. Puklo M, Guentsch A, Hiemstra PS, Eick S, Potempa J. Analysis of neutrophil-derived antimicrobial peptides in gingival crevicular fluid suggests importance of cathelicidin LL-37 in the innate immune response against periodontogenic bacteria. Oral microbiology and immunology, 2008; 23: 328-335.
13. Miki O, Hanioka T. Destructive effects of smoking on molecular and genetic factors of periodontal disease. Tob Induc Dis, 2010; 8: 4.
14. Ryder MI. The influence of smoking on host responses in periodontal infections. Periodontology 2000, 2007; 43: 267-277.
15. Johnson GK, Guthmiller JM. The impact of cigarette smoking on periodontal disease and treatment. Periodontol 2000. 2007; 44:178-194.
16. Sulsky, Sandra I et al. Evaluating the association between menthol cigarette use and the likelihood of being a former versus current smoker. Regulatory toxicology and pharmacology: RTPvol. 2014; 70: 231-41.
17. Gorr SU. Antimicrobial peptides of the oral cavity. Periodontol.2000. 2009; 51: 152–180.
18. Gorr SU, Abdolhosseini M. Antimicrobial peptides and periodontal disease. J. Clin.Periodontol.2011; 38:126–141.
19. Soldati KR, Gutierrez LS, Anovazzi G, Scarel-Caminaga RM, Zandim-Barcelos DL. Impact of smoking on protein levels of beta-defensins in periodontal disease. Braz Dent J. 2022; 33:79-86.
20. An N, Holl J, Wang X, Rausch MA, Andrukhov O, Rausch-Fan X. Potential Suppressive Effect of Nicotine on the Inflammatory Response in Oral Epithelial Cells: An In Vitro Study. International Journal of Environmental Research and Public Health; 2021;18:483.
21. Ertugrul AS, Sahin H, Dikilitas A, Alpaslan NZ, Bozoglan A, Tekin Y. Gingival crevicular fluid levels of human beta-defensin-2 and cathelicidin in smoker and nonsmoker patients: a cross-sectional study. J. Periodontal. Res.2014 ;49:282–289.
22. Wolgin M, Liodakis S, Pries AR, Zakrzewicz A, Kielbassa AM. HBD-1 and hBD-2 expression in HaCaT keratinocytes stimulated with nicotine. Arch Oral Biol. Jun;2012; 57:814-9.
23. Shchipkova AY, Nagaraja HN, Kumar PS. Subgingival Microbial Profiles of Smokers with Periodontitis. J Dent Res.2010;89:1247–1253.
24. Yin L, Chino T, Horst OV, Hacker BM, Clark, EA, Dale BA, et al. Differential and coordinated expression of defensins and cytokines by gingival epithelial cells and dendritic cells in response to oral bacteria. BMC Immunol.2010 ;11:37–37.
25. Türkoğlu O, Eren G, Emingil G, Azarsız E, Kutukculer N, Atilla G. Does smoking affect gingival crevicular fluid LL-37 levels following non-surgical periodontal treatment in chronic periodontitis? Arch Oral Biol.2016; 61:98-105.
26. Pereira AL, Franco GC, Cortelli SC, Aquino DR, Costa FO, Raslan SA, Cortelli JR. Influence of periodontal status and periodontopathogens on levels of oral human β-defensin-2 in saliva. J Periodontol. 2013;84:1445-53.

27. Yılmaz D, Güncü GN, Könönen E, Barış E, Çağlayan F, Gursoy UK. Overexpressions of hBD-2, hBD-3, and hCAP18/LL-37 in gingiva of diabetics with periodontitis. Immunobiology.2015;220:1219-26.
28. Brancatisano FL, Maisetta G, Barsotti F, Esin S, Miceli M, Gabriele M, et al. Reduced human beta defensin 3 in individuals with periodontal disease. J Dent Res. 2011; 90:241-5.
29. Gursoy UK, Könönen E. Understanding the roles of gingival beta-defensins. J Oral Microbiol.2012;4:15127.
30. Puklo M, Guentsch A, Hiemstra PS, Eick S, Potempa J. Analysis of neutrophil-derived antimicrobial peptides in gingival crevicular fluid suggests importance of cathelicidin LL-37 in the innate immune response against periodontogenic bacteria. Oral Microbiol Immunol.2008;23:328-35.
31. Hosokawa I, Hosokawa Y, Komatsuzawa H, Goncalves RB, Karimbux N, Napimoga MH, et al. Innate immune peptide LL-37 displays distinct expression pattern from beta-defensins in inflamed gingival tissue. Clin Exp Immunol.2006;146:218-25.
32. Lopes AB, Colombo NH, Assem NZ, Nuernberg MAA, Garcia VG, Duque C, et al. LL-37 and hBD-2 in the gingival crevicular fluid of smokers and nonsmokers with periodontitis. Rev Odontol UNESP.2021;50:e20210056.
33. Turkoglu O, Emingil G, Kutukculer N, Atilla G. Gingival crevicular fluid levels of cathelicidin LL-37 and interleukin-18 in patients with chronic periodontitis. J Periodontol. 2009; 80:969–976.
34. Takeuchi Y, Nagasawa T, Katagiri S, Kitagawara S, Kobayashi H, Koyanagi T,etal.Salivary levels of antibacterial peptide(LL-37/hCAP-18)and cotinine in patients with chronic periodontitis. J. Periodontol.2012; 83:766–762.

تأثير تدخين السجائر على الببتيدات المناعية في لعاب المرضى المصابين بالنساغ المزمن

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Received August 27, 2023

Accepted: Jun 23, 2024

DOI:
https://doi.org/10.35516/jmj.v58i4.1663

الملخص

الخلفية والأهداف : تدخين السكائر مرتبط بالعديد من الامراض (جهازية وفموية) ويؤثر على الجهاز المناعي الغير متخصص والمتخصص. الببتيدات الدفاعية؛ غالبا تعرف بالببتيدات المضادة للميكروبات وهي من مكونات الجهاز المناعي الغير متخصص وتعتبر من مجموعة الاوليكوببتيدات الفعالة كيميائيا وهي سامة للمكيروبات الضارة.

ويهدف هذا البحث هو تسليط الضوء على تاثير التدخين على انتاج الببتيدات الدفاعية عند مرضى النساغ المزمن.

منهجية الدراسة : خمسة وثمانون شخص مشترك في هذه الدراسة، الحالة الصحية للثة قيمت بواسطة: مؤشر اللويحة السنية، مؤشر اللثة، مؤشر النزيف اللثوي، مؤشر عمق الجيوب اللثوية ومؤشر فقدان الانسجة الرابطة. تم جمع عينات اللعاب من جميع الأشخاص. تقنية ايلايزا استخدمت لتقدير مستوى الديفينسين نوع بيتا والكاثليسدين، التحليل الاحصائي تم باستخدام برنامج SPSS 24 واستخدم اختبار انوفا لكشف الاختلافات بين مجاميع الدراسة بالإضافة لاختبار توكي لتحديد وجود أي ارتباط احصائي بين مجاميع الدراسة.

النتائج: وجدت الدراسة ارتفاع مستوى الببتيدات المناعية عند مجاميع المدخنين وغير المدخنين مقارنة بمجموعة الاصحاء، هناك انخفاض معنوي في مستوى الديفينسين نوع بيتا بين المدخنين وغير المدخنين ($p<0.001$) بينما لايوجد أي فرق معنوي بين المدخنين وغير المدخنين بالنسبة لمستوى الكاثليسدين.

الاستنتاجات : هذه النتائج ترجح انه التدخين له تأثير على مستوى البروتين ديفنسين نوع بيتا لكن لايؤثر على مستوى الكاثليسدين.

الكلمات الدالة: تدخين السكائر، الببتيدات المضادة للميكروبات، اللعاب، المناعة الغير متخصصة.