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ORIGINAL ARTICLE

Association of salivary flow rate and pH with salivary alkaline phosphatase in smokers and non-smokers with periodontitis

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ABSTRACT

Background: Changes in salivary flow rate, pH, and salivary alkaline phosphatase (S-ALP) may reflect periodontal disease activity in smokers.

Objective: To determine the association of salivary flow rate and salivary pH with salivary alkaline phosphatase in smokers and non-smokers with periodontitis.

Methods: This cross-sectional study was conducted at Azra Naheed Medical & Dental College from February to December 2025. A total of 200 adults (100 smokers with ≥5 pack-years and 100 non-smokers) aged 18-40 years with clinically diagnosed periodontitis were recruited by consecutive sampling. Unstimulated whole saliva was collected by passive drool for 5 minutes. Salivary pH was measured with a calibrated pH meter, and S-ALP was measured by spectrophotometry. Data was analyzed using SPSS version-25.

Results: There was a significant difference in salivary pH (5.64±0.514 vs. 7.02±0.12, p<0.001) and S-ALP (59.67±34.28 vs. 31.73±18.86, p<0.001) between smokers and non-smokers, respectively, while the difference in salivary flow rate was non-significant (Smokers:0.648±0.136 vs. Non-smokers:0.652±0.257 mL/min; p=0.880). There is a negative correlation of salivary flow rate (r=-0.651, p<0.001) and salivary pH (r=-0.450, p<0.001) with S-ALP among smokers; in non-smokers, only salivary flow rate (r=-0.560, p<0.001) showed a negative correlation with S-ALP. Among smokers, salivary pH showed a significant association with elevated S-ALP levels (p-value < 0.001), and among those with acidic salivary pH, 77.4% had elevated S-ALP levels compared with 22.6% with normal levels.

Conclusion: Smokers with periodontitis show significantly lower salivary pH and higher S-ALP than non-smokers. Since salivary pH is associated with elevated S-ALP among smokers, pH can serve as a useful marker for monitoring smoking-related periodontal biochemical changes.

Key Words: Periodontitis; Saliva; Alkaline Phosphatase; Hydrogen-Ion Concentration; Tobacco Smoking.

DOI: https://doi.org/10.53685/jshmdc.v7i1.443

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Received: 25.1.2026, 1st Revision: 25.5.2026, 2nd Revision: 1.6.2026, Accepted:10.6.26.

How to cite this article: Akram A, Amjad S, Jehangir HMM. Association of salivary flow rate and pH with salivary alkaline phosphatase in smokers and non-smokers with periodontitis: J Shalamar Med Dent Coll. 2026;7(1): 41-47.doi: https://doi.org/10.53685/jshmdc.v7i1.443

INTRODUCTION

Periodontitis is a chronic inflammatory disease associated with dental plaque and dental biofilm, usually starting as gingivitis. It is characterized by periodontal ligament and alveolar bone loss, leading to tooth loss and disability.[1] In addition to poor oral outcomes and inflammation, periodontitis can play a part in systemic conditions through various pathways involving low-grade inflammation and microbial translocation. Global Burden of Disease (GBD) has emphasized that oral diseases are highly prevalent all around the world, with severe periodontitis contributing to disability and long-term health issues.[2] Smoking promotes periodontal disruption through multiple mechanisms, such as vasoconstriction and reduced gingival blood supply, impaired neutrophil and macrophage function, impaired cytokine

functioning, oxidative stress, and delayed wound healing, leading to periodontal destruction and poor therapeutic response in smokers.[3] Previous studies have reported that there is a higher risk of periodontal disease and worse outcomes among smokers compared to non-smokers.[4-6]

The basic salivary properties, such as secretion rate and pH, play protective roles against microbes and chemical changes, including buffering action and oral homeostasis. Disruption of this balance may cause dysbiosis and inflammation, and these changes are significantly associated with smoking, which can alter salivary composition, pH, and the oral microbiome.[7] Studies comparing conventional cigarette smokers, e-cigarette users, and non-users have reported altered salivary characteristics, oral biofilm changes, and increased inflammatory activity in tobacco- or nicotine-exposed groups.[8,9]

Biochemical indicators can help us understand and diagnose active tissue turnover and inflammation, including S-ALP, which is involved in phosphate metabolism and is linked to bone turnover and remodeling. Clinical studies have reported higher S-ALP and crevicular-ALP levels in periodontitis and reductions following periodontal disease treatment, supporting their role as important biomarkers.[10-12]

Smoking is a well-established risk factor for periodontitis and alters the oral inflammatory response and salivary characteristics, which may influence the severity of periodontitis; however, there is limited data on its effects on salivary flow rate, pH, and S-ALP.[7] Most previous studies have evaluated these parameters independently, and compared healthy individuals with diseased patients, or assessed smokers and non-smokers without controlling for periodontal status. Previous studies have compared salivary and serum alkaline phosphatase levels among healthy individuals and patients with gingivitis or periodontitis, while others have assessed salivary pH, buffering capacity, and alkaline phosphatase levels in smokers and healthy non-smokers.[10,13] The present study addresses this research gap by combining assessments of salivary flow rate, pH, and S-ALP activity within a single clinical framework and analyzing the correlation between smoking status and periodontitis. Assessing and comparing these salivary parameters in smokers and non-smokers with periodontitis may help understand their relationship with smoking-related periodontitis and their diagnostic relevance.

The primary objective of this study was to determine the association of salivary flow rate and salivary pH with S-ALP levels among smokers and non-smokers with periodontitis. The secondary objective was to compare salivary flow rate, salivary pH, and S-ALP levels between smokers and non-smokers with periodontitis.

METHODS

This comparative cross-sectional study was conducted from February 2025 to December 2025 at the dental clinics of Azra Naheed Medical and Dental College, Lahore, Pakistan.

The sample size was calculated with a 95% confidence interval, 90% power of the test, and by taking the expected mean values of salivary pH in smokers and nonsmokers (healthy individuals) as 6.47±0.23 and 7.16±0.31. The calculated sample size was 8 (4 in each group). To improve precision and address possible dropouts, the final sample size was increased to 200 participants, with 100 smokers and 100 non-smokers.[13,14]

Both male and female adults aged 18 to 40 years were considered eligible for screening and were recruited through consecutive sampling. Patients were included if they had periodontitis diagnosed during clinical examination and fulfilled the smoking or non-smoking criteria of the study. Participants with diabetes mellitus, autoimmune salivary gland disorders, hepatic or renal disease, pregnancy or lactation, use of medications affecting salivary flow or inflammatory response, periodontal therapy or antibiotic use within the previous three months, and those unwilling to continue participation were excluded.

After obtaining written informed consent, patients presenting to the dental outpatient department during the study period were examined by a trained dental surgeon for periodontitis. Periodontitis was diagnosed when ≥4 teeth were involved, with at least one site showing probing depth ≥4 mm, clinical attachment loss ≥1 mm at the same site, and bleeding on probing at ≥2 sites.[10] Probing depth was measured at six sites per tooth, clinical attachment loss was recorded from the cemento-enamel junction to the base of the periodontal pocket, and bleeding on probing was recorded after probing. Patients fulfilling the diagnostic criteria for periodontitis were then assessed for smoking history. Smoking status was assessed using WHO Global Adult Tobacco Survey-based

questions.[15] Current cigarette smokers with cumulative exposure of ≥5 pack-years were enrolled in the smoker group, while participants who had never smoked tobacco were enrolled in the non-smoker group. Pack-years were calculated by multiplying the number of cigarette packs smoked per day by the number of years of smoking. No separate minimum smoking duration was applied beyond the cumulative pack-year criterion. Eligible participants were then enrolled in the respective smoker or non-smoker group. Patients diagnosed with periodontitis and found eligible according to the smoking criteria were instructed to report to the Physiology Department the next day at 8:00 am for saliva sample collection. Before sample collection, participants were advised to avoid food, drinks except water, alcohol, caffeine, smoking, and oral hygiene products for at least 2 hours. Unstimulated whole saliva was collected between 9:00 am and 12:00 pm using the passive drool method to minimize circadian variation. Participants were seated comfortably, allowed saliva to pool in the mouth for approximately 5 minutes, then allowed it to drip into a sterile container and later into a graduated cylinder. The saliva volume collected over 5 minutes was measured and expressed as mL/min. Salivary pH was measured immediately after collection using a calibrated digital pH meter (Hanna HI-8424), and the meter was calibrated to ensure accuracy. For S-ALP analysis, samples were centrifuged at approximately 3000 rpm for 5–15 minutes to obtain the supernatant. S-ALP levels were then measured by spectrophotometry using Shimadzu UV-1800. Quality control was maintained through instrument calibration and periodic duplicate checks. For categorical analysis, salivary parameters were categorized using predefined operational cut-off values. Unstimulated salivary flow rate was categorized as normal (≥0.30 mL/min), low/reduced (0.10–<0.30 mL/min), and very low (<0.10 mL/min) [15,16]; values <0.05 mL/min were considered to fall within the severe xerostomia range. Salivary pH was categorized as normal/near neutral (≥6.5), acidic (5.5–<6.5), and critically acidic/cariogenic (<5.5). S-ALP was categorized operationally as normal (≤25 U/L) and elevated (>25 U/L).[17] These categories were used only for categorical comparison between smokers and non-smokers.

Ethical Approval

Ethical approval was obtained from the Ethical Review Board (IRB#SU91-MBMSW-F22-031) of Azra Naheed Medical College, Lahore, Pakistan, on 1-12-2025. Written informed consent was obtained from all participants before recruitment, and confidentiality of participant data was maintained throughout the study

Statistical Analysis

Data were analyzed using SPSS version 25. Continuous variables, i.e., salivary flow rate, salivary pH, and S-ALP, were presented as mean±SD. Qualitative data, such as smoking status, were presented as frequencies and percentages. The Shapiro-Wilk test was performed to assess normality ($p>0.05$). An independent-samples t-test was applied to compare parameters between smokers and non-smokers. The correlation of salivary flow rate and salivary pH with S-ALP levels was assessed using the Pearson correlation test in smokers and non-smokers. Association of salivary flow rate and pH with S-ALP was determined by applying a chi-square test. A p-value ≤ 0.05 was considered statistically significant.

RESULTS

A total of 200 participants with periodontitis were analyzed (smokers =100 and non-smokers =100). The baseline characteristics of the study participants are shown in Table 1.

Unstimulated salivary flow rate was similar in smokers and non-smokers with no statistically significant difference ($p=0.880$). The salivary pH was significantly ($p<0.001$) lower (acidic) in smokers compared to non-smokers. Moreover, the S-ALP level was significantly ($p<0.001$) higher in smokers compared to non-smokers (Table 2).

Salivary parameters were categorized according to predefined operational cut-off values. Elevated S-ALP was observed in 85% of smokers compared with 56% of non-smokers, a statistically significant difference ($p<0.001$). Low salivary flow rate was more frequent among smokers than non-smokers (14.0% vs. 2.0%; $p=0.002$), although no participant in either group had a very low salivary flow rate. Salivary pH distribution also differed significantly between the two groups ($p<0.001$). Most smokers had acidic salivary pH, with 62% in the acidic range and 37% in the critical acidic/cariogenic range, whereas all non-smokers were in the normal/near neutral pH category (Table 3).

Table 1: Baseline characteristics of study participants

Variable	Non-smokers (n=100)	Smokers (n=100)	Total (n=200)
Age (mean±SD)	38.26±7.49	35.35±9.80	36.81±8.82
Male n (%)	52(52.0%)	65(65.0%)	117(58.5%)
Female n(%)	48(48.0%)	35(35.0%)	83(41.5%)

Table 2: Comparison of salivary flow rate, salivary pH, and S-ALP between smokers and non-smokers with periodontitis

Variable	Group	mean±SD	*p-value*
SFR (mL/min)	Smokers	0.65±0.26	0.880
	Non-Smokers	0.65±0.14	
Salivary pH	Smokers	5.65±0.51	<0.001*
	Non-Smokers	7.03±0.12	
S-ALP U/L)	Smokers	59.67±34.82	<0.001*
	Non-Smokers	31.73±18.86	

*SFR = Salivary flow rate. Independent-samples t-test applied. *p<0.05 considered statistically significant.*

Table 3: Stratified analysis of salivary flow rate, salivary pH, and S-ALP in smokers and non-smokers with periodontitis

Variable	Category	Smokers n (%)	Non-smokers n (%)	*P value*
S-ALP (u/L)	Normal (≤ 25)	15(15)	44(44)	<0.00*
	Elevated (> 25)	85(85)	56(56)	
SFR (mL/min)	Normal (≥ 0.30)	86(86)	98(98)	0.002*
	Low (0.10–<0.30)	14(14)	2(2)	
Salivary pH	Normal/ neutral (≥ 6.5)	1(1)	100(100)	<0.00*
	Acidic (5.5–<6.5)	62(62)	0(0)	
	Critical acidic /carcinogenic (<5.5)	37(37)	0(0)	

SFR= Salivary flow rate, S-ALP= Salivary Alkaline Phosphatase. **p<0.05 was considered statistically significant. Chi-square test was applied*

Among smokers, salivary flow rate ($p < 0.001$) and pH ($p < 0.001$) showed significant negative correlations with S-ALP. Among non-smokers, salivary flow rate showed a significant negative correlation with S-ALP ($p<0.001$), whereas pH showed no significant correlation with the flow rate ($p=0.99$) or S-ALP ($p=0.34$) (Table 4). Among smokers, salivary pH showed a significant association with elevated S-ALP, and it was elevated in 77.4% of participants with acidic pH $p<0.001$), while salivary flow rates showed no significant association with S-ALP. (Table-5)

Table 4: Correlation of salivary flow rate and salivary pH with S-ALP levels among smokers and non-smokers with periodontitis

Category	Variable Pair	*r*	*p-value*
Smokers	SFR - S-ALP	-0.651	<0.001*
	pH - S-ALP	-0.450	<0.001*
Non-Smokers	SFR - S-ALP	-0.560	<0.001*
	pH - S-ALP	-0.096	0.343

*SFR=Salivary flow rate, S-ALP=Salivary Alkaline Phosphatase. Pearson Correlation test applied. *p<0.05 considered statistically* significant

Table 5: Association of salivary flow rate and salivary pH with S-ALP in smokers with periodontitis

Variable	Category	S-ALP (u/L) ≤25 n (%)	S-ALP (u/L) >25 n (%)	*p-value*
SFR (mL/min)	Normal ≥ 0.30	15(17.4)	71(82.6)	0.09
	Low 0.10<0.30	0(0.0)	14(100)	
Salivary pH	Normal/ neutral ≥6.5	1(100)	0(0)	<0.001*
	Acidic 5.5–<6.5	14(22.6)	48(77.4)	
	Critically acidic /cariogenic <5.5	0 (0.0)	37(100)	

SFR=Salivary flow rate. S-ALP= Salivary Alkaline Phosphatase. *Chi-square test was applied. *p<0.05 was considered statistically significant*

DISCUSSION

This comparative cross-sectional study assessed the association of salivary flow rate and salivary pH with S-ALP levels among smokers and non-smokers with periodontitis. Unstimulated whole saliva was collected from 200 participants with periodontitis, including 100 smokers and 100 non-smokers, and salivary parameters were analyzed and compared.

In this study, the unstimulated salivary flow rate was similar between smokers and non-smokers with periodontitis. Recent literature has mixed findings, as several studies report an association between chronic tobacco smoking and reduced salivary flow and xerostomia, while some clinical studies report little difference.[18,19] Studies on nicotine intake, including conventional and e-cigarettes, reported that salivary flow can vary by product type, duration, and intensity of use, and sometimes report no significant reduction in baseline flow.[20,21] A possible reason could be that salivary flow may be less sensitive than biochemical markers for smoking-associated periodontitis, especially among participants in this study. In the current study, smokers showed significantly lower salivary pH than non-smokers, reflecting increased

salivary acidity ($p<0.001$). The previous literature similarly reports lower salivary pH and altered buffering capacity among smokers, and studies show that in tobacco-exposed groups, the oral microbiome changes, potentially promoting the selection of acid-tolerant pathogenic microbial communities that also promote inflammation.[22,23] Smoking-related reductions in buffering, increased oxidative stress, and shifts in the microbial environment, including altered microbial metabolism, can contribute to a sustained acidic environment in periodontitis, which may increase protease activity and tissue injury, thereby accelerating periodontal disease progression.[24,25]

In the present study, smokers had significantly higher S-ALP levels than non-smokers ($p<0.001$), indicating increased tissue turnover and inflammation. Previous studies support S-ALP as a biomarker in periodontitis, with higher S-ALP levels in active disease and reductions after periodontal therapy, indicating sensitivity to disease activity.[10,11,26] Smoking increases the inflammatory response and tissue breakdown during periodontitis, thereby increasing the secretion or leakage of S-ALP into saliva and crevicular fluid.[12,27] Tobacco-related oxidative stress and immune responses may intensify tissue degradation and alveolar bone remodeling, resulting in higher S-ALP levels despite similar saliva flow.[25]

Among smokers with periodontitis, salivary pH showed a significant negative association with S-ALP levels, whereas this association was not significant among non-smokers. This suggests that smoking may strengthen the relationship between acidic salivary changes and periodontal biochemical activity. Recent studies have concluded that combining parameters such as pH with biomarkers improves the discrimination between active periodontal diseases and inflammatory activity.[14,26] A more acidic oral environment indicates higher levels of acidic microbial activity, which may co-occur with periodontal inflammation, thereby elevating S-ALP.[26] The lack of correlation in non-smokers may be due to less pH variability and unrestricted blood supply for repair.[4,5] Salivary biochemical activity is linked to both smoking and pH, and indicates multi-parameter salivary profiling in the future.[14, 27] Acidic saliva is a proximal indicator of periodontal inflammation, while smoking has an additional destructive role through immune and oxidative mechanisms.[7,8]

The differential correlation pattern between smokers and non-smokers may be explained by the additional effect of tobacco exposure on the oral and periodontal environment. In smokers, reduced salivary pH may be due to smoking-related impairment of buffering capability, microbial dysbiosis, oxidative stress, and microvascular dysfunction, which can increase periodontal breakdown and S-ALP release into saliva.[25,27] This may also explain why salivary pH reported a significant inverse correlation with S-ALP in smokers. In contrast, non-smokers showed no significant correlation between pH and S-ALP, possibly because their salivary pH remained relatively stable, making pH less effective at modulating S-ALP activity. In this group, S-ALP may have been more closely related to periodontal tissue turnover and the dilutional effect of salivary flow rather than to acidity.[11,27] Therefore, smoking strengthens the link between acidic salivary changes and periodontal breakdown, whereas in non-smokers, S-ALP activity may be driven mainly by local periodontal inflammation independent of salivary pH.

CONCLUSION

In patients with periodontitis, salivary flow rate and salivary pH were significantly associated with S-ALP levels in smokers. Smokers showed significantly lower salivary pH and higher S-ALP levels than non-smokers, indicating greater smoking-related salivary acidity and periodontal biochemical activity. Assessment of salivary flow rate, pH, and S-ALP can help support clinical diagnosis and monitoring of smokers with periodontitis by indicating salivary acidity, reduced salivary protective function, and increased periodontal tissue breakdown.

Limitations and future recommendations

This single-center study with a modest sample size measured salivary parameters at a single time point, potentially influenced by hydration, diet, and recent oral activity. Additionally, detailed periodontal parameters and radiographic bone loss were not assessed. Future multicenter longitudinal studies with larger samples, objective smoking verification, and comprehensive periodontal evaluation are recommended to validate these findings.

REFERENCES

1. Shafique R, Hassan H, Shakoor A, Aziz S, Aslam K, Najfi NZ. Perception and misconceptions of patients about dental scaling treatment: a cross-sectional study. J Oral Health Oral Epidemiol. 2024; 13(3): 122-126. doi:10.34172/johoe.2310.1595

2. Bernabe E, Marcenes W, Abdulkader RS, Abreu LG, Afzal S, Alhalaiqa FN, et al. Trends in the global, regional, and national burden of oral conditions from 1990 to 2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet. 2025; 405(10482): 897-910. doi: 10.1016/S0140-736(24)02811-3
3. Zhang Y, Shen Z, Yang J, Ren J, Zhang C, Tan L, et al. Single-cell spatial atlas of smoking-induced changes in human gingival tissues. Int J Oral Sci. 2025; 17(1): 60-73. doi:10.1038/s41368-025-00385-5
4. Leite FRM, Nascimento GG, Scheutz F, Lopez R. Effect of smoking on periodontitis: a systematic review and meta-regression. Am J Prev Med. 2018; 54(6): 831-841. doi:10.1016/j.amepre.2018.02.014.
5. Alwithanani N. Periodontal disease and smoking: systematic review. J Pharm Bioallied Sci. 2023; 15(Suppl-1): S64-71. doi:10.4103/jpbs.jpbs_516_22.
6. Battancs E, Gheorghita D, Nyiraty S, Lengyel C, Eördegh G, Baráth Z, et al. Periodontal disease in diabetes mellitus: a case-control study in smokers and non-smokers. Diabetes Ther. 2020; 11(11): 2715-28. doi: 10.1007/s13300-020-00933-8.
7. Boynes SG, Sofiyeva N, Saw T, Nieto V, Palomo L. Assessment of salivary matrix metalloproteinase-8 and activated salivary matrix metalloproteinase-8 in periodontitis patients: a systematic review and meta-analysis. Front Oral Health. 2025; 6: 1444399. doi:10.3389/froh.2025.1444399
8. Mohd Hasan NW, Baharin B, Mohd N, Abdul Rahman M, Hassan N. Comparative effects of e-cigarette smoking on periodontal status, salivary pH, and cotinine levels. BMC Oral Health. 2024; 24(1): 861. doi:10.1186/s12903-024-04650-7
9. Thomas SC, Xu F, Pushalkar S, Lin Z, Thakor N, Vardhan M, et al. Electronic cigarette use promotes a unique periodontal microbiome. MBio. 2022; 13(1): e00075-22. doi:10.1128/mBio.00075-22
10. Koppolu P, Sirisha S, Mishra A, Deshpande K, Lingam AS, Alotaibi DH, et al. Alkaline phosphatase and acid phosphatase levels in saliva and serum of patients with healthy periodontium, gingivitis, and periodontitis before and after scaling with root planing: a clinico-biochemical study. Saudi J Biol Sci. 2021; 28(1): 380-385. doi:10.1016/j.sjbs.2020.10.016
11. Rasaei N, Ghadiri A, Peighan M, Rekabi A, Atashkar N. Evaluation of alkaline phosphatase in gingival crevicular fluid and saliva of patients with periodontitis and healthy individuals. J Family Med Prim Care. 2022; 11(11): 6983. doi:10.4103/jfmpc.jfmpc_477_22
12. Noh JW, Jang JH, Yoon HS, Kim KB, Heo MH, Jang HE, et al. Evaluation of salivary biomarkers of periodontal disease based on smoking status: a systematic review. Int J Environ Res Public Health. 2022; 19(21): 14619. doi :10.3390/ijerph192114619
13. Ahmadi-Motamayel F, Falsafi P, Goodarzi MT, Poorolajal J. Comparison of salivary pH, buffering capacity and alkaline phosphatase in smokers and healthy non-smokers: retrospective cohort study. Sultan Qaboos Univ Med J. 2016; 16(3): e317-21. doi:10.18295/squmj.2016.16.03.009
14. Buktare K, Parikh S, Shah J. Comparative evaluation of salivary flow rate, pH, and alkaline phosphatase in tobacco users and healthy individuals: a biomarker study. J Int Oral Health. 2024;16(6):479-86. doi: 10.4103/jioh.jioh_122_24
15. Villa A, Connell CL, Abati S. Diagnosis and management of xerostomia and hyposalivation. Ther Clin Risk Manag. 2015;11:45–51. doi:10.2147/TCRM.S76282
16. Navazesh M, Kumar SK. Measuring salivary flow: challenges and opportunities. J Am Dent Assoc. 2008; 139: 35S-40S. doi:10.14219/jada.archive.2008.0353
17. Patel RM, Varma S, Suragimath G, Zope S. Estimation and comparison of salivary calcium, phosphorus and alkaline phosphatase levels in patients with periodontal disease. J Clin Diagn Res. 2016; 10(10): ZC136–ZC139. doi:10.7860/JCDR/2016/22817.8707
18. Sreeramareddy CT, Acharya K, Ramakrishna Reddy N. Single, dual, and poly use of tobacco products, and associated factors among adults in 18 Global Adult Tobacco Survey countries during 2015–2021. Int J Ment Health Addict. 2025; 23: 4118-33. doi:10.1007/s11469-024-01340-x
19. Zięba S, Zalewska A, Żukowski P, Maciejczyk M. Can smoking alter salivary homeostasis? A systematic review on the effects of traditional and electronic cigarettes on qualitative and quantitative saliva parameters. Dent Med Probl. 2024; 61(1): 129-44. doi:10.17219/dmp/172084
20. Atagün ÖS, Baltacıoğlu E, Alver A, Yücesan FB, Yuva P, Kehribar MA, et al. Effects of smoking on local and systemic oxidative stress markers in individuals with periodontitis. Northwest Med J. 2024;4(4): 195–205. doi:10.54307/2024.NWMJ.71
21. Alqahtani SM, Gokhale ST, Elagib MFA, Shrivastava D, Nagate RR, Alshmrani BAM, et al. Assessment and correlation of salivary Ca, Mg, and pH in smokers and non-smokers with generalized chronic periodontitis. Medicina (Kaunas). 2023; 59(4): 765. doi:10.3390/medicina59040765.
22. Wang X, Mi Q, Yang J, Guan Y, Zeng W, Xiang H, et al. Effect of electronic cigarette and tobacco smoking on the human saliva microbial community. Braz J Microbiol. 2022; 53(2): 991-1000. doi: 10.1007/s42770-022-00721-5
23. Shapiro H, Goldenberg K, Ratiner K, Elinav E. Smoking-induced microbial dysbiosis in health and disease. Clin Sci (Lond). 2022; 136(18):1371-87. doi: 10.1042/CS20220175
24. Basic A, Dahlén G. Microbial metabolites in the pathogenesis of periodontal diseases: a narrative review. Front Oral Health. 2023; 4:1210200. doi:10.3389/froh.2023.1210200
25. Sczepanik FSC, Grossi ML, Casati M, Goldberg M, Glogauer M, Fine N, et al. Periodontitis is an inflammatory disease of oxidative stress: we should treat it that way. Periodontol 2000. 2020; 84(1): 45-68. doi:10.1111/prd.12342
26. Nayak DS, Dave A, Manek PV, Jambhekar SV, Shetty RR, Syed NK, et al. Evaluating alkaline phosphatase level and inflammation parameters in smokers with chronic periodontitis. Adv Hum Biol. 2026; 16(1): 161-7. doi:10.4103/aihb.aihb_174_25
27. Silva H. Tobacco use and periodontal disease: the role of microvascular dysfunction. Biology (Basel). 2021; 10(5): 441. doi: 10.3390/biology10050441

AUTHORS' CONTRIBUTION:

AA: Conception of the study, data acquisition, analysis, manuscript drafting, final approval
SA: Conception of the study, data collection, analysis & interpretation, critical review
HMMJ: Data collection, data analysis, manuscript drafting and final review

All Authors agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

CONFLICT OF INTEREST:
All authors declared no conflict of interest.

GRANT SUPPORT AND FINANCIAL DISCLOSURE:
No specific grant was taken for this research from any funding agency in the public, commercial or not-for-profit sectors.

DATA SHARING STATEMENT:
The data are available from the corresponding author upon reasonable request.

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