Original Article

Comparative Analysis of Salivary Electrolyte Concentrations and Flow Rate in Chronic Smokers versus Non-Smokers

Allied Medical Research Journal
Vol:3 No.4: 81-88

Maham Maooz Ansari[1], Maryam Asim Khan[1], Hamna Khanani[1], Ayesha Yaqoob Kahout[2]

[1]*Ziauddin University, Karachi, Pakistan*
[2]*Bolan Medical College, Quetta, Pakistan*

ABSTRACT

Background: Tobacco smoking has well-documented adverse effects on oral health, yet the specific mechanisms by which smoking alters salivary electrolyte composition remain incompletely understood. Given saliva's critical role in maintaining oral homeostasis, investigating smoking-induced changes in salivary electrolytes may provide insights into disease susceptibility patterns observed in smoking populations. This study aimed to quantify and compare salivary electrolyte concentrations and flow rates between chronic smokers and non-smokers, while examining relationships between smoking duration and observed salivary changes.

Methods: We conducted a cross-sectional analysis involving 200 participants (100 smokers, 100 non-smokers) between ages 25-55 years. Both stimulated and unstimulated saliva samples underwent electrolyte analysis using ion-selective electrodes and spectrophotometric methods. Statistical evaluation employed independent t-tests, correlation analysis, and multiple regression modeling.

Results: Smokers demonstrated significantly elevated sodium ($p<0.001$), chloride ($p<0.001$), and calcium concentrations ($p<0.05$) relative to non-smokers. Conversely, potassium and phosphate concentrations were significantly decreased in the smoking group ($p<0.01$). Both stimulated and unstimulated salivary flow rates showed marked reductions in smokers ($p<0.001$). Strong positive correlations emerged between smoking duration and sodium/chloride concentrations.

Conclusion: Chronic tobacco use produces substantial alterations in salivary electrolyte composition and secretion rates, which may contribute to the heightened oral disease susceptibility observed in smoking populations. These findings suggest potential utility of salivary electrolyte profiles as biomarkers for smoking-related oral health risk assessment.

Keywords: *Biomarkers, Electrolytes, Oral Health, Saliva, Smoking, Tobacco.*

Received: July 29, 2025; Revised: September 15, 2025**; Accepted:** October 20, 2025

Corresponding Email: mahammaooz@gmail.com

DOI: https://doi.org/10.59564/amrj/03.04/012

INTRODUCTION

Tobacco smoking continues to represent a major global health challenge, affecting approximately 1.3 billion individuals worldwide. The oral cavity, being the primary site of tobacco exposure, bears a disproportionate burden of smoking-related pathology. Epidemiological evidence consistently demonstrates increased prevalence of periodontal disease, oral malignancies, impaired wound healing, and taste disturbances among smoking populations.[1,2]

Saliva functions as a complex biological fluid essential for oral health maintenance through multiple protective mechanisms including lubrication, pH buffering, antimicrobial activity, and digestive facilitation. The diagnostic potential of saliva has gained considerable attention due to its non-invasive collection and ability to reflect both local and systemic health status.[3,4] Among salivary components, electrolytes including sodium (Na+), potassium (K+), chloride (Cl-), calcium (Ca2+), and phosphate (PO43-) play fundamental roles in maintaining oral pH homeostasis, supporting

enamel remineralization processes, and preserving overall oral health.[5]

Previous investigations have suggested that tobacco use may substantially modify salivary composition and secretion patterns, potentially contributing to the elevated oral disease rates observed in smoking populations.[6,7] However, comprehensive electrolyte profiling in smoking-related salivary research remains limited, with insufficient exploration of dose-response relationships between smoking duration and compositional changes.

The pathophysiological mechanisms underlying smoking-induced salivary alterations appear multifactorial. Nicotine and related tobacco constituents can directly influence salivary gland function through sympathetic nervous system activation, resulting in altered secretion dynamics[8]. Furthermore, chronic tobacco smoke exposure may induce oxidative stress and inflammatory responses within salivary glands, potentially disrupting normal electrolyte transport mechanisms[9,10].

Understanding specific patterns of salivary electrolyte modification associated with tobacco use has several important implications. First, these changes may help explain the increased susceptibility of smokers to oral diseases such as dental caries and periodontal conditions. Second, salivary electrolyte profiles could serve as non-invasive biomarkers for evaluating smoking-related oral health risks. Third, this knowledge may inform the development of targeted preventive interventions for smoking populations. This study's primary objective was to compare salivary electrolyte concentrations and flow rates between chronic smokers and non-smoking controls. Secondary aims included examining correlations between smoking duration, daily cigarette consumption, and salivary parameters, as well as evaluating the potential clinical utility of salivary electrolytes as smoking-related biomarkers.

METHODOLOGY

This cross-sectional analytical investigation was conducted at Ziauddin Medical College and Bolan Medical College from January through August 2024. An equal number of participants were recruited from each college. The study was conducted in accordance with the principles of the Declaration of Helsinki, and all participants provided written informed consent prior to enrollment.

Sample Size Calculation

Sample size determination utilized G*Power 3.1.9.7 software, assuming a medium effect size (Cohen's d = 0.5) for between-group differences in mean salivary electrolyte concentrations, with $\alpha = 0.05$ and power = 0.80. The calculated minimum requirement was 64 participants per group. To accommodate potential dropouts and enable subgroup analyses, we recruited 100 participants for each group.

Inclusion and Exclusion Criteria

Smoker Inclusion Criteria:

- Age range 25-55 years
- Smoking history ≥10 cigarettes daily for ≥5 years
- Active smoking status (cigarette use within 24 hours of sample collection)
- Absence of systemic disease

Non-Smoker Inclusion Criteria:

- Age range 25-55 years
- Never-smoker status or smoking cessation >10 years prior
- Absence of systemic disease

Exclusion Criteria (Both Groups):

- Pregnancy or lactation
- Systemic conditions affecting salivary gland function (diabetes mellitus, Sjögren's syndrome, etc.)
- Medications influencing salivary flow (antidepressants, antihistamines, etc.)
- History of head and neck radiation therapy
- Alcohol consumption >14 units weekly
- Smokeless tobacco product use
- Active oral infections or inflammatory conditions

Participant Recruitment and Screening

Recruitment employed advertisements in local newspapers, university publications, and dental clinic postings. Initial screening utilized standardized telephone interviews. Qualified candidates underwent clinical examination and saliva collection procedures. Smoking history verification included the Fagerström Test for Cigarette Dependence and expired carbon

monoxide measurement using a CO monitor (Micro+ Smokerlyzer, Bedfont Scientific Ltd., UK).

Saliva Collection Protocol

Participants received instructions to avoid eating, drinking, smoking, or oral hygiene procedures for at least 2 hours before sample collection. All collections occurred between 9:00-11:00 AM to minimize circadian variation. A 10-minute relaxation period in a quiet room preceded sample collection.

- ***Unstimulated Saliva Collection:*** Following complete swallowing of existing saliva, participants remained seated quietly for 5 minutes. Saliva collection proceeded via passive drooling into pre-weighed sterile tubes for exactly 10 minutes. Volume determination employed gravimetric analysis (assuming saliva density = 1.0 g/mL).
- ***Stimulated Saliva Collection:*** After a 15-minute rest interval, stimulated saliva collection utilized 2g paraffin wax as stimulant. Participants chewed the wax for 30 seconds to initiate salivation, then expectorated into collection tubes for 10 minutes while continuing mastication. The initial 30 seconds of stimulated saliva was discarded.

Sample processing involved immediate ice placement and laboratory transport within 30 minutes. Samples underwent centrifugation at 3000 rpm for 10 minutes at 4°C to remove cellular debris, with supernatant storage at -80°C until analysis.

Laboratory Analysis

Electrolyte Analysis

- **Sodium and Potassium:** Ion-selective electrode methodology using automated analyzer (Roche Cobas 6000, Roche Diagnostics, Switzerland)
- **Chloride:** Colorimetric analysis using mercuric thiocyanate (Roche Cobas 6000)
- **Calcium:** Colorimetric analysis using o-cresolphthalein complexone (Roche Cobas 6000)
- **Phosphate:** Colorimetric analysis using ammonium molybdate (Roche Cobas 6000)

Quality Control

All determinations were performed in duplicate with mean values used for statistical analysis. Internal quality control samples were included in each analytical batch, maintaining inter-assay and intra-assay coefficients of variation below 5%.

Statistical Analysis

Data analysis employed SPSS version 28.0 (IBM Corp., Armonk, NY). Distribution normality was assessed using Shapiro-Wilk tests and Q-Q plots. Descriptive statistics presented normally distributed data as means ± standard deviations and non-normally distributed data as medians (interquartile ranges). Independent t-tests compared means between groups for normally distributed data, while Mann-Whitney U tests addressed non-normally distributed data. Pearson or Spearman correlation coefficients assessed variable relationships. Multiple linear regression analysis identified independent predictors of salivary electrolyte concentrations. Statistical significance was set at $p<0.05$.

RESULTS

Participant Characteristics

The study enrolled 200 participants equally distributed between smokers and non-smokers. Demographic and clinical characteristics for both groups are presented in Table 1. Groups showed comparable age and gender distributions. Smokers reported mean smoking duration of 18.7 ± 8.2 years with average daily consumption of 16.4 ± 6.8 cigarettes. The mean Fagerström score of 5.8 ± 2.1 indicated moderate to high nicotine dependence levels.

Salivary Flow Rates

Both unstimulated and stimulated salivary flow rates exhibited significant reductions in smokers compared to non-smokers (Table-2). The reduction proved more pronounced for unstimulated saliva, with smokers demonstrating a 42% decrease in flow rate relative to non-smokers. Stimulated salivary flow rate decreased by 28% in the smoking group.

Table 1. Demographic and Clinical Characteristics of Study Participants

Characteristic	Smokers (n=100)	Non-smokers (n=100)	p-value
Age (years)	38.5 ± 9.2	37.8 ± 8.7	0.572
Gender (Male/Female)	62/38	58/42	0.574
BMI (kg/m²)	24.8 ± 3.4	24.2 ± 3.1	0.189
Smoking duration (years)	18.7 ± 8.2	-	-
Cigarettes per day	16.4 ± 6.8	-	-
Fagerström score	5.8 ± 2.1	-	-
Expired CO (ppm)	28.4 ± 12.6	3.2 ± 1.8	<0.001

Data presented as mean ± standard deviation or frequency. BMI = Body Mass Index; CO = Carbon monoxide; ppm = parts per million

Table 2. Comparison of Salivary Flow Rates between Smokers and Non-smokers

Flow Rate Parameter	Smokers (n=100)	Non-smokers (n=100)	Mean Difference (95% CI)	p-value
Unstimulated flow rate (mL/min)	0.31 ± 0.15	0.53 ± 0.22	-0.22 (-0.27 to -0.17)	<0.001
Stimulated flow rate (mL/min)	1.24 ± 0.48	1.72 ± 0.56	-0.48 (-0.62 to -0.34)	<0.001

Data presented as mean ± standard deviation. CI = Confidence interval

Salivary Electrolyte Concentrations

Significant differences emerged in all measured electrolyte concentrations between smokers and non-smokers (Table-3). Smokers exhibited elevated concentrations of sodium, chloride, and calcium, while potassium and phosphate levels were decreased compared to non-smoking controls.

Table-3. Comparison of Salivary Electrolyte Concentrations between Smokers and Non-smokers

Electrolyte	Smokers (n=100)	Non-smokers (n=100)	Mean Difference (95% CI)	p-value	Effect Size (Cohen's d)
Unstimulated Saliva					
Sodium (mmol/L)	28.6 ± 12.4	18.2 ± 8.9	10.4 (7.6 to 13.2)	<0.001	0.95
Potassium (mmol/L)	16.8 ± 6.2	22.4 ± 7.8	-5.6 (-7.6 to -3.6)	<0.001	0.79
Chloride (mmol/L)	32.4 ± 14.8	21.6 ± 10.2	10.8 (7.4 to 14.2)	<0.001	0.83
Calcium (mmol/L)	2.18 ± 0.84	1.86 ± 0.68	0.32 (0.08 to 0.56)	0.009	0.42
Phosphate (mmol/L)	8.4 ± 3.2	10.8 ± 3.8	-2.4 (-3.4 to -1.4)	<0.001	0.68
Stimulated Saliva					
Sodium (mmol/L)	22.4 ± 9.8	15.6 ± 7.2	6.8 (4.5 to 9.1)	<0.001	0.78
Potassium (mmol/L)	18.2 ± 5.4	24.8 ± 6.8	-6.6 (-8.4 to -4.8)	<0.001	1.07
Chloride (mmol/L)	26.8 ± 11.2	18.4 ± 8.6	8.4 (5.8 to 11.0)	<0.001	0.83
Calcium (mmol/L)	1.94 ± 0.72	1.68 ± 0.58	0.26 (0.06 to 0.46)	0.012	0.40
Phosphate (mmol/L)	7.2 ± 2.8	9.6 ± 3.4	-2.4 (-3.2 to -1.6)	<0.001	0.78

Correlation Analysis

Strong positive correlations were identified between smoking duration and sodium concentrations in both unstimulated (r = 0.72, p<0.001) and stimulated saliva (r = 0.68, p<0.001). Similarly, chloride concentrations demonstrated significant positive correlations with smoking duration (unstimulated: r = 0.69, p<0.001; stimulated: r = 0.64, p<0.001). Negative correlations were observed between smoking duration and potassium concentrations (unstimulated: r = -0.58, p<0.001; stimulated: r = -0.62, p<0.001).

Table-5. Multiple Regression Analysis for Salivary Sodium Concentration (Unstimulated)

Predictor Variable	β coefficient	Standard Error	t-value	p-value	95% CI
Smoking status	6.82	1.24	5.50	<0.001	4.38 to 9.26
Smoking duration†	0.48	0.08	6.00	<0.001	0.32 to 0.64
Age	0.12	0.08	1.50	0.135	-0.04 to 0.28
Gender (Male)	2.14	1.18	1.81	0.071	-0.19 to 4.47
BMI	-0.18	0.22	-0.82	0.414	-0.61 to 0.25

R^2 = 0.68, F = 42.8, p<0.001. †Among smokers only. CI = Confidence interval

Multiple Regression Analysis

Multiple regression analysis was conducted to identify independent predictors of salivary electrolyte concentrations. The models incorporated age, gender, BMI, smoking status, smoking duration, and cigarettes per day as predictor variables.

Table-4. Correlation between Smoking Parameters and Salivary Electrolytes (Smokers Group Only)

Parameter	Smoking Duration	Cigarettes per Day	Fagerström Score
Unstimulated Saliva			
Sodium	0.72***	0.45***	0.38***
Potassium	-0.58***	-0.32**	-0.28**
Chloride	0.69***	0.41***	0.35**
Calcium	0.28**	0.19	0.22*
Phosphate	-0.42***	-0.26**	-0.31**
Stimulated Saliva			
Sodium	0.68***	0.43***	0.36**
Potassium	-0.62***	-0.35**	-0.29**
Chloride	0.64***	0.39***	0.33**
Calcium	0.24*	0.16	0.19
Phosphate	-0.48***	-0.28**	-0.33**

**Correlation coefficients (Pearson's r). *p<0.05, **p<0.01, **p<0.001*

DISCUSSION

This investigation demonstrates substantial alterations in salivary electrolyte composition and flow rates among chronic smokers compared to non-smoking controls. The findings reveal significant changes in all measured electrolytes, which may have important implications for understanding oral disease susceptibility patterns observed in smoking populations. The reduction in stimulated salivary flow rate suggests that smoking also compromises the functional reserve capacity of salivary glands. The observed reductions in both unstimulated and stimulated salivary flow rates among smokers align with previous research findings[11,12].

The 42% reduction in unstimulated flow rate is particularly concerning, as resting salivary flow is critical for maintaining baseline oral hygiene and pH homeostasis. Several pathophysiological mechanisms may explain this reduction, including nicotine-induced sympathetic nervous system activation leading to vasoconstriction and diminished glandular secretion[13]. Additionally, chronic tobacco smoke exposure may cause structural damage to salivary glands, including acinar cell atrophy and fibrotic changes[14]. This impairment may be particularly problematic during meals, when increased salivary flow is needed for proper digestion and oral clearance. The decreased flow rates observed in smokers may contribute to their increased risk of dental caries, periodontal disease, and oral candidiasis[15].

The elevation of sodium and chloride concentrations in smokers' saliva represents a significant departure from normal ionic homeostasis. These changes may reflect altered membrane permeability and transport mechanisms in salivary gland cells. Nicotine has been demonstrated to affect sodium-potassium ATPase activity and ion channel function, which could explain the observed electrolyte imbalances[16,17]. The strong positive correlation between smoking duration and sodium/chloride concentrations suggests a dose-dependent relationship, indicating that prolonged tobacco exposure results in more severe alterations in salivary composition.

The decreased potassium concentration in smokers' saliva is particularly noteworthy, as potassium plays a crucial role in maintaining cellular function and membrane potential. This reduction may contribute to impaired oral tissue repair and increased susceptibility to infections. The observed correlation between smoking duration and potassium reduction suggests that this effect becomes more pronounced with extended tobacco exposure. Elevated calcium concentrations in smokers' saliva may initially appear beneficial, as calcium is essential for enamel remineralization. However, the concurrent reduction in phosphate levels may actually impair the remineralization process, as both calcium and phosphate are required for optimal hydroxyapatite formation[18]. The imbalance between these minerals may contribute to the increased prevalence of dental caries observed in smokers.

Clinical Implications

The alterations in salivary electrolyte composition observed in this study have several important clinical implications. The ionic imbalances may contribute to the increased prevalence of oral diseases in smokers. The elevated sodium and chloride concentrations, combined with reduced flow rates, may create an environment that favors pathogenic bacterial growth and biofilm development[19].

The reduced potassium and phosphate concentrations may impair the natural protective mechanisms of saliva, including its buffering capacity and remineralization potential. This could explain why smokers often experience more severe forms of periodontal disease and dental caries despite similar oral hygiene practices compared to non-smokers[20].

The strong correlations between smoking duration and electrolyte changes suggest that these parameters could potentially serve as biomarkers for assessing the cumulative effects of tobacco exposure on oral health. This information could be valuable for developing personalized preventive strategies and monitoring the effectiveness of smoking cessation interventions.

Mechanistic Considerations

The mechanisms underlying the observed changes in salivary electrolyte composition are likely multifactorial. Direct effects of nicotine on ion transport mechanisms in salivary glands may play a primary role. Nicotine has been shown to affect various ion channels and transporters, including sodium-potassium ATPase, calcium channels, and chloride channels[21,22]. These effects could directly alter the ionic composition of saliva by disrupting normal electrolyte transport processes.

Chronic inflammation induced by tobacco smoke exposure may also contribute to the observed changes. Inflammatory mediators can affect glandular function and alter the permeability of ductal epithelium, potentially leading to changes

in electrolyte concentrations[23]. The presence of reactive oxygen species in tobacco smoke may cause oxidative damage to salivary gland cells, further compromising their function.

Autonomic nervous system dysfunction associated with chronic smoking may represent another important mechanism. Smoking affects both sympathetic and parasympathetic nervous system function, which could alter the neural control of salivary gland secretion and modify the composition of saliva[24].

Limitations and Future Directions

Several limitations should be considered when interpreting these results. The cross-sectional design prevents establishment of causal relationships between smoking and salivary changes. Longitudinal studies would be valuable for better understanding the temporal relationship between tobacco exposure and salivary alterations.

The study focused on a specific age range (25-55 years) and may not be generalizable to younger or older populations. Future research should investigate whether similar changes occur in different age groups and whether there are age-related differences in susceptibility to smoking-induced salivary alterations.

The study did not evaluate the reversibility of these changes following smoking cessation. Understanding whether salivary electrolyte imbalances normalize after quitting smoking would have important implications for counseling patients and developing treatment strategies.

Future research should also investigate the relationship between salivary electrolyte changes and specific oral health outcomes, such as caries risk, periodontal disease severity, and oral cancer development. Additionally, studies examining the effects of different tobacco products (e.g., e-cigarettes, smokeless tobacco) on salivary composition would provide valuable insights into the relative risks associated with various forms of tobacco use.

CONCLUSION

This study demonstrates that chronic smoking induces significant alterations in salivary electrolyte composition and flow rates. The observed changes, including elevated sodium and chloride concentrations, reduced potassium and phosphate levels, and decreased flow rates, may contribute to the increased susceptibility of smokers to oral diseases. The strong correlations between smoking duration and electrolyte changes suggest that these parameters could serve as useful biomarkers for assessing smoking-related oral health risks. These findings support the importance of smoking cessation for maintaining optimal oral health and suggest that monitoring salivary electrolyte composition may be valuable for developing personalized preventive strategies for smoking populations.

Acknowledgments

None.

Author Contributions

Maham Maooz Ansari contributed to the conceptualization and study design. **Maryam Asim Khan** was responsible for data collection and analysis. **Hamna Khanani** handled data interpretation and manuscript drafting, while **Ayesha Yaqoob Kahout** conducted the literature review and manuscript revision. All authors reviewed and approved the final version of the manuscript.

Ethical Approval

This study is approved by the Ethics Review Committee of Bolan Medical College, Quetta, Pakistan (ERC/BMC/2024/045).

Grant Support and Funding Disclosure

None.

Conflict of Interests

None.

REFERENCES

1. 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. Dental and Medical Problems. 2024;61(1):129-44.
2. Kurniawan AV, Amtha R, Gunardi I, Heriandi A, Sari EF. The Impact of Electronic and Conventional Cigarette Use towards Saliva Profile and Oral Microbiota in Adolescents. Asian Pacific Journal of Cancer Prevention. 2025 Jan 1;26(1):309-18.
3. Hasan NW, Baharin B, Mohd N, Rahman MA, Hassan N. Comparative effects of e-cigarette smoking on periodontal status, salivary pH, and cotinine levels. BMC Oral Health. 2024 Jul 29;24(1):861.
4. Erdemir EO, Erdemir A. The detection of salivary minerals in smokers and non-smokers with chronic periodontitis by the inductively coupled plasma-

atomic emission spectrophotometry technique. Journal of periodontology. 2006 Jun;77(6):990-5.
5. Tjahajawati S, Rafisa A, Lestari EA. The effect of smoking on salivary calcium levels, calcium intake, and bleeding on probing in female. International journal of dentistry. 2021;2021(1):2221112.
6. Alqahtani SM, Gokhale ST, Elagib MF, Shrivastava D, Nagate RR, Alshmrani BA, Alburade AM, Alqahtani FM, Nagarajappa AK, Natoli V, Srivastava KC. Assessment and correlation of salivary Ca, Mg, and pH in smokers and non-smokers with generalized chronic periodontitis. Medicina. 2023 Apr 14;59(4):765.
7. Rad M, Kakoie S, Brojeni FN, Pourdamghan N. Effect of long-term smoking on whole-mouth salivary flow rate and oral health. Journal of dental research, dental clinics, dental prospects. 2010 Dec 21;4(4):110.
8. Yiu AJ, Kalejaiye A, Amdur RL, Hesham HT, Bandyopadhyay BC. Association of serum electrolytes and smoking with salivary gland stone formation. International journal of oral and maxillofacial surgery. 2016 Jun 1;45(6):764-8.
9. Singh M, Ingle NA, Kaur N, Yadav P, Ingle E. Effect of long-term smoking on salivary flow rate and salivary pH. Journal of Indian Association of Public Health Dentistry. 2015 Jan 1;13(1):11-3.
10. Humphrey SP, Williamson RT. A review of saliva: normal composition, flow, and function. J Prosthet Dent. 2001;85(2):162-169.
11. Nigar S, Hassan S, Maqsood A, Ahmed N, Al-Askar M, Mokeem SA, Alqutub MN, Aldahian N, AlSagob EI, AlMubarak AA, Vohra F. An assessment of unstimulated salivary flow rate, IgA and clinical oral dryness among active and passive smokers. International journal of occupational medicine and environmental health. 2022 Jan 1;35(1):39.
12. Dawes C, Pedersen AM, Villa A, et al. The functions of human saliva: a review sponsored by the World Workshop on Oral Medicine VI. Arch Oral Biol. 2015;60(6):863-874.
13. Jessie K, Pang WW, Haji Z, Rahim A, Hashim OH. Proteomic analysis of whole human saliva detects enhanced expression of interleukin-1 receptor antagonist, thioredoxin and lipocalin-1 in cigarette smokers compared to non-smokers. International journal of molecular sciences. 2010 Nov 9;11(11):4488-505.
14. Dolatabadi A, Noori F, Raee A. Effect of conventional cigarettes and e-cigarettes on salivary biomarkers: A systematic review. Journal of Advanced Periodontology & Implant Dentistry. 2024 Apr 22;16(1):44.
15. Tomar SL, Asma S. Smoking-attributable periodontitis in the United States: findings from NHANES III. Journal of periodontology. 2000 May;71(5):743-51.
16. Nagler R, Lischinsky S, Diamond E, Drigues N, Klein I, Reznick AZ. Effect of cigarette smoke on salivary proteins and enzyme activities. Archives of Biochemistry and Biophysics. 2000 Jul 15;379(2):229-36.
17. Kamal NM, Shams NS. The impact of tobacco smoking and electronic cigarette vaping on salivary biomarkers. A comparative study. The Saudi Dental Journal. 2022 Jul 1;34(5):404-9.
18. Larsen MJ. Enamel/saliva-inorganic chemical reactions. Textbook of cariology. 1986.
19. Fiyaz M, Ramesh A, Ramalingam K, Thomas B, Shetty S, Prakash P. Association of salivary calcium, phosphate, pH and flow rate on oral health: A study on 90 subjects. Journal of Indian Society of Periodontology. 2013 Jul 1;17(4):454-60.
20. Bergström J, Eliasson S, Dock J. A 10-year prospective study of tobacco smoking and periodontal health. Journal of periodontology. 2000 Aug;71(8):1338-47.
21. Benowitz NL. Pharmacology of nicotine: addiction, smoking-induced disease, and therapeutics. Annual review of pharmacology and toxicology. 2009 Feb 10;49(1):57-71.
22. Saputri D, Nasution AI, Surbakti MR, Gani BA. The correlation between pH and flow rate of salivary smokers related to nicotine levels labelled on cigarettes. Dental Journal. 2017 Jun;50(2):61-5.
23. Johnson GK, Hill M. Cigarette smoking and the periodontal patient. J Periodontol. 2004;75(2):196-209.
24. Krüger K, Dischereit G, Seimetz M, et al. Time course of cigarette smoke-induced changes of systemic inflammation and muscle structure. Am J Physiol Lung Cell Mol Physiol. 2015;309(2):L119-L128.
25. Kambalyal P, Kambalyal P, Hungund S. Comparison of salivary calcium level in smokers and non-smokers with chronic periodontitis, aggressive periodontitis, and healthy controls. Journal of International Society of Preventive and Community Dentistry. 2015 Dec 1;5(Suppl 2):S68-73.