Journal of Medical and oral biosciences
ISSN (Online): 3007-9551
ISSN (Print): 3007-9543

JMOB
Open Access DOAJ

IRAQI Academic Scientific Journals

Type: Research article
Publish online: 30/ 05 /2026

Effect of Smoking on Oral Microbial Profile and Epithelial Cytological Changes: A Comparative Study

Mohammed A Mahdi [1*], Ahmed Mustafa Ahmed [2], Reyam Ibraheem daham [3]

[1*] College of Dentistry , Al-Iraqia University , Baghdad, Iraq,
Email: mohammed.a.mahdi@aliraqia.edu.iq
ORCID: https://orcid.org/0009-0009-7407-7549
[2] College of Dentistry, Al-Iraqia University , Baghdad, Iraq
Email: ahmed.mu.ahmed@aliraqia.edu.iq
https://orcid.org/0000-0003-3414-3205
[3] College of science, Mustansiriyah University,
Email: Reyam.I.D@uomustansiriyah.edu.iq ;
ORCID: https://orcid.org/0009-0004-3860-0703

OPEN ACCESS

ARTICLE INFO
Received:16 /04/2026
Revised: 30/ 04/ 2026
Accepted: 16 / 05 / 2026
Publish online: 30 / 05 / 2026
Plagiarism percentage at publication: 13%

* Corresponding Author: Mohammed A Mahdi
Email: mohammed.a.mahdi@aliraqia.edu.iq

CITATION

Mohammed A Mahdi, Ahmed Mustafa Ahmed, Reyam Ibraheem daham (2026). Effect of Smoking on Oral Microbial Profile and Epithelial Cytological Changes: A Comparative Study JMOB. 3 ;(2): 69-79. https://doi.org/10.58564/jmob.164

Abstract

Smoking is a major risk factor for many oral disease and changes such as the oral microbiome and the integrity of the epithelial cells. It is a major driver of microbial dysbiosis and causes early cytological changes which gives potential to inflammation, therefore increasing the risk for oral pathological conditions. This study intends to investigate the link between microbial dysbiosis and epithelial changes in regular smoker's subjects, and determine the smoking related oral diseases. Eighty participants were studied (40 smokers and 40 non-smokers). Analysis of salivary samples were performed for total bacterial counts, microbial composition, and diversity index. Buccal swabs were performed to collect the oral epithelial cells, and assessed cytologically for cell diameter, nuclear diameter, N/C ratio, inflammatory cell count, and atypia. Smokers had greater bacterial load (7.92 ± 0.48 log CFU/mL), proportion of pathogenic bacteria (61.8 ± 7.9%), and reduced diversity (2.08 ± 0.27). Cytological findings were consistent with both larger nuclear diameter (9.6 ± 1.3) and N/C ratio and higher inflammatory cell counts (38.5 ± 6.4 cells/HPF) in smokers compared with non-smokers (all $p < 0.001$). Smokers had cytological atypia in 35.0%, while non-smokers did not ($P < 0.01$). In Conclusion, Smoking induces significant microbial dysbiosis and early epithelial cytological changes in the oral cavity.

Keywords: *Smoking, oral microbiota, exfoliative cytology, nuclear–cytoplasmic ratio, inflammation, dysbiosis.*

Introduction

Smoking is one of the most serious global public health challenges and a well-known risk factor for systemic and oral diseases (1, 2). Tobacco smoke has many harmful and potentially carcinogenic constituents that can adversely affect oral tissues, resulting in diseases such as periodontal disease, oral mucosal lesions, and oral cancer. These adverse effects of tobacco are particularly important in the oral cavity, as it is the first site of exposure to tobacco (3). Along the decades, the effect of smoking on the oral microbiome have received more attention. The diversity and dynamism of the microbiome in the oral cavity are important factors in establishing and maintaining oral health (4, 5). This can be

disturbed during a process referred to as dysbiosis, which results in a higher ratio of pathogenic microorganisms and a lower ratio of beneficial commensals (6). Several studies indicated that smoking causes significant alterations of oral microbiota compositions and densities with a propensity towards the development of a dysbiotic microbial community profile popularly associated with the potential for disease (7–9). Moreover to microbial alterations, smoking also leads to the structural and functional changes of oral epithelial cells (10, 11). Tobacco cause disruption of normal biological function via changes in the gene expression pattern, resulting in a nuclear enlargement, increase in nuclear–cytoplasmic (N/C) ratio, hyperchromasia, and other cytologic changes (12,13). These changes are early signs of epithelial stress and can occur before dysplasia or malignancy develops (14). Although, there are increasing evidence regarding the effects of smoking on oral microbiota and epithelial cells individually, there are few studies combining both the microbiological and cytological evaluations. Consequently, this study designed to investigate the link between microbial dysbiosis and epithelial changes in regular smoker's subjects. In addition, to determine the smoking related oral diseases.

Materials and Methods

Ethical Approval

The study protocol was approved by the Ethical Committee of Al-Iraqia University, approval number: [ESA&HER-29-04]. Written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

Study Design and Participants

The total number of subjects involved in the current study was s 80, in the age group (18–45 years). The subjects were comprised 40 smokers and 40 healthy participants. The inclusion criteria was the smokers individuals who had smoked cigarettes at least regularly for more than 1year and non-smokers had never smoked or used tobacco. Smokers were further categorized based on smoking duration (years) into short-term smokers (1–<5 years), moderate-duration smokers (5–10 years), and long-term smokers (>10 years). In addition, smokers were classified according to smoking intensity (cigarettes/day) into light smokers (<10 cigarettes/day) and heavy smokers (≥10 cigarettes/day). The exclusion criteria were subject with younger than 18 years or older than 40 years, and those with systemic diseases, active oral infections, nonsmokers advanced stages and grades periodontal diseases, alcohol abuse habit, or have received antibiotic therapy within three months before study entry.

Saliva Collection and Oral Epithelial Cell

Stimulated saliva samples were collected according to a protocol adapted from a previous study (15). Samples were collected in the morning between 9:00 AM and 11:00 AM to reduce variability. Participants were trained to avoid exercise, eating, drinking, and tooth brushing for at least one hour before sample collection. Approximately 5 mL of stimulated saliva was collected from each participant into sterile containers. Oral

epithelial cells were collected from the buccal mucosa using sterile cotton swabs under standardized conditions. Before swab collection, participants rinsed their mouths with normal saline to help remove debris and loosely attached dead cells.

Microbial Analysis

Ten-fold serial dilutions was used to dilute the saliva .Then, 0.1 ml was inoculated onto blood agar. The total microbial load was calculated. The biochemical tests and Gram stain were used to identify the isolated microbes. Microbial diversity was measured using Shannon index (16).

$$H' = -\sum_{i=1}^{S} p_i \ln p_i$$

H' Shannon diversity index
p_i *represents* proportion of the *i*th microbial species
S represents the total number of species identified

Cytological Analysis

The epithelial tissue smeared onto clean, labeled glass slides and fixed in 95% ethanol for 15–20 minutes to preserve cellular morphology. The slides were subsequently stained using the Papanicolaou (Pap) staining technique following standard protocols (17). The following cytomorphometric parameters were assessed: epithelial cell diameter (µm), nuclear diameter (µm), and nuclear–cytoplasmic (N/C) ratio. In addition, the number of inflammatory cells per high-power field (HPF) was recorded. Qualitative assessment included the evaluation of cytological atypia, such as nuclear enlargement, hyperchromasia, irregular nuclear borders, and binucleation.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 26.0 (18). Results are presented as mean ± standard deviation (SD). Comparisons between groups were performed using the independent t-test, while categorical variables were analyzed using the Chi-square test. Pearson's correlation coefficient was used to assess relationships between variables. A p-value <0.05 was considered statistically significant.

Results

A total of 80 participants were included, with 40 smokers and 40 non-smokers.

Demographic Data

There was a higher proportion of males in the smoker group (87.5%) than in non-smokers (82.5%), but the difference was not statistically significant (p =0.53). Females comprised 15·0% ($n^- = 78$) of the total sample (Table. 1). The largest age group was 26–35 years

(47.5%), followed by 18–25 years (28.8%), and 36–45 years (23.7%). Age distribution was not significantly different in smokers versus non-smokers (p = 0.69).

Salivary Microbial Findings

Salivary analysis showed a higher bacterial load in smokers (7.92 ± 0.48 log CFU/mL) compared to non-smokers (6.31 ± 0.42, p < 0.001). The proportion of pathogenic bacteria was also increased in smokers (61.8 ± 7.9%) versus non-smokers (30.4 ± 6.2%, p < 0.001). In contrast, commensal bacteria were lower in smokers (22.5 ± 4.8%) compared to non-smokers (47.9 ± 6.9%, p < 0.001) (Table.2).

Table. 1: Distribution of Patients According to Age and Gender

Variable	Category	Smokers (n=40)	Non-smokers (n=40)	Total (n=80)	χ^2	p-value
Gender	Male	35 (87.5%)	33 (82.5%)	68 (85.0%)	0.39	0.53
	Female	5 (12.5%)	7 (17.5%)	12 (15.0%)		
Age Group (years)	18–25	10 (25.0%)	13 (32.5%)	23 (28.8%)	0.74	0.69
	26–35	20 (50.0%)	18 (45.0%)	38 (47.5%)		
	36–45	10 (25.0%)	9 (22.5%)	19 (23.7%)		

Table. 2: Salivary Microbial Profile in Smokers and Non-smokers, Mean ± SD

Parameter	Smokers mean± SD (n = 40)	Smokers mean ± SD (n = 40)	p-value
Total bacterial count (log CFU/mL)	7.92 ± 0.48	6.31 ± 0.42	<0.001
Pathogenic bacteria (%)	61.8 ± 7.9	30.4 ± 6.2	<0.001
Commensal bacteria (%)	22.5 ± 4.8	47.9 ± 6.9	<0.001
Shannon diversity index	2.08 ± 0.27	3.36 ± 0.38	<0.001

Cytological Analysis

The cytologically evaluation of oral epithelial cells from smokers and non-smokers presented in (Table. 3). The mean epithelial cell diameter was significantly lower in smokers (48.2 ± 5.6 µm) compared to non-smokers (55.7 ± 4.9 µm, p < 0.001). In contrast, nuclear diameter was significantly increased in smokers (9.6 ± 1.3 µm) compared to non-smokers (7.2 ± 1.1 µm, p < 0.001). Accordingly, the nuclear–cytoplasmic (N/C) ratio was markedly higher in smokers (0.20 ± 0.03) than in non-smokers (0.13 ± 0.02, p < 0.001). Smokers also exhibited a significantly greater number of inflammatory cells (38.5 ± 6.4 cells/HPF) compared to non-smokers (16.8 ± 4.1 cells/HPF, p < 0.001). Cytological atypia was observed in 14 smokers (35.0%), whereas no atypical changes were detected

in non-smokers (0.0%, $p < 0.001$). Among qualitative findings in smokers were hyperchromasia of nuclei, irregularity of nuclear borders, and binucleation in some instances.

Correlation Analysis

Correlation analysis revealed that smoking variables, microbial parameters and cytological changes were significantly associated. The N/C ratio ($r = 0.68$, $p < 0.001$) and total bacterial load ($r = 0.66$, $p < 0.001$) showed strong positive correlations with smoking duration. There was also a positive correlation between bacterial load and inflammatory cells ($r = 0.69$, $p < 0.001$) (Table. 4).

Table. 3: Cytological analysis of oral epithelial cells in smokers and non-smokers, Mean ± SD

Parameter	**Smokers** mean± SD **(n = 40)**	**Non-smokers** mean± SD **(n = 40)**
Cell diameter (µm)	48.2 ± 5.6	55.7 ± 4.9
Nuclear diameter (µm)	9.6 ± 1.3	7.2 ± 1.1
Nuclear–cytoplasmic ratio	0.20 ± 0.03	0.13 ± 0.02
Inflammatory cells (cells/HPF)	38.5 ± 6.4	16.8 ± 4.1
Cytological atypia, n (%)	14 (35.0%)	0 (0.0%)

Smoking Intensity

When smokers were divided by smoking intensity heavy smokers (n = 22) demonstrated greater cytological and microbiotic change versus light smokers (n = 18). Heavy smokers had a significantly higher N/C ratio (0.23 ± 0.02) compared with light smokers (0.17 ± 0.03), $p < 0.001$. In heavy smokers (8.28 ± 0.39 log CFU/mL) vs. light smokers (7.45 ± 0.46 log CFU/mL, $p < 0.001$) the total bacterial count was similarly increased (table 5). The number of inflammatory cells was also significantly higher in the heavy smokers (52.7 ± 6.1 cells/HPF) vs the light smokers (40.3 ± 5.6 cells/HPF, $p < 0.001$). In addition, cytological atypia was more common among heavy smokers cases (10/22, 45.5%) than among light smokers (4/18 cases, 22.2%, $p = 0.04$).

Table. 4: Correlation between Smoking Parameters, Microbial Profile, and Cytological Changes

Variables	**r**	**p-value**
Smoking duration vs N/C ratio	0.68	<0.001
Smoking duration vs bacterial load	0.66	<0.001
Bacterial load vs inflammatory cells	0.69	<0.001

Table. 5: Comparison of Parameters According to Smoking Intensity, Mean ± SD

Parameter	**Light smokers mean± SD (<10/day, n = 18)**	**Heavy smokers mean± SD (≥10/day, n = 22)**	**p-value**
N/C ratio	0.17 ± 0.03	0.23 ± 0.02	<0.001
Bacterial count (log CFU/mL)	7.45 ± 0.46	8.28 ± 0.39	<0.001
Inflammatory cells (cells/HPF)	40.3 ± 5.6	52.7 ± 6.1	<0.001
Cytological atypia, n (%)	4 (22.2%)	10 (45.5%)	0.04

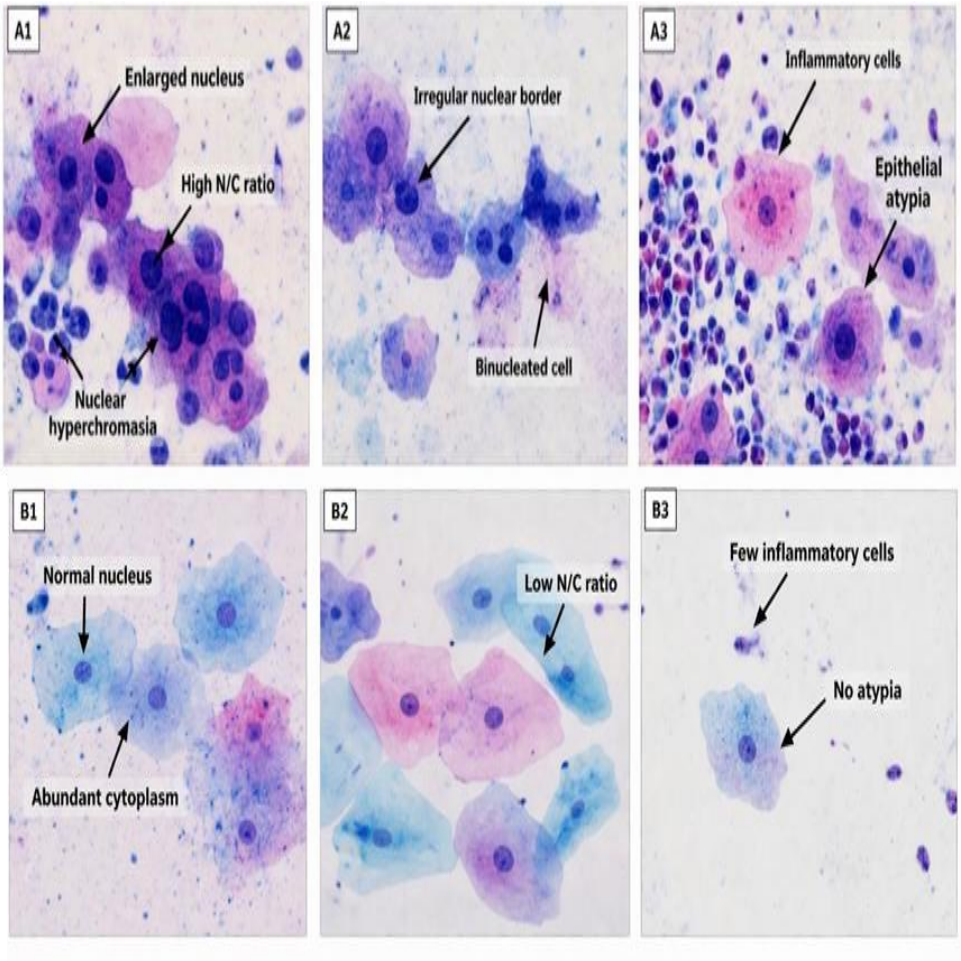

Figure. 1: The oral epithelium in smokers and non-smokers. Smokers (A1-A3) exhibit nuclear enlargement, increased N/C ratio, hyperchromasia, and inflammatory infiltration, whereas non-smokers (B1-B3) show normal cellular morphology with low inflammatory background (×40).

Discussion

The present study demonstrated that smoking is associated with significant alterations in salivary microbial composition and oral epithelial cytology. Smokers had more bacterial load, a higher proportion of pathogenic bacteria, a less diverse microbial

community, and significant cytological changes, including increased N/C ratio and inflammatory cell infiltration. These results indicate that tobacco exposure has microbiological and cellular effects in the oral cavity. Smokers have been reported to have a markedly higher total bacterial count. These results are compatible with previous study done by Brook,(2011)(19). It showed that smoking facilitates colonisation by pathogenic microorganisms and leads to deviation of the normal oral flora (19). Additionally, the results of the current study showed a significantly higher bacterial load was seen for the smoker group compared to non-smokers (7.92 ± 0.48 log CFU/mL vs. 6.31 ± 0.42, $p < 0.001$), increased pathogenic bacteria and reduced commensals was also found in smokers. These results agree with results of previous study (20). Al-Marzooq *et al.,* (2022) showed that smokers possess a specific oral environment with increased bacterial load and decreased diversity (20). The noted loss of microbial diversity in smokers is likely significant 2.08 ± 0.27 vs 3.36 ± 0.38, $p < 0.001$), indicative of ecological imbalance (dysbiosis). These results are consistent with those of Shapiro *et al.,* (2022), who found smoking significantly changes the oral microbial ecosystem, highly favoring anaerobic and pathogenic species. Dysbiosis leads to chronic inflammation and higher susceptibility to oral diseases (8). Cytopathological evaluation showed that smokers had marked epithelial changes characterized by increased nucleus size, N/C ratio and cytological atypia. These results are in agreement with Brima *et al.,* (2017) and Parmar *et al.,* (2020). They mentioned that tobacco exposure causes nuclear enlargement, hyperchromasia, and raised N/C ratio (21,22). The increased N/C ratio was (0.20 ± 0.03 and 0.13 ± 0.02 in smokers and in non-smokers respectively ($p < 0.001$). This results indicated the heightened cellular metabolic activity and early dysplastic changes.

The higher inflammatory cell count in smokers is also consistent with prior studies which reported that cellular responses to tobacco smoke induces chronic inflammatory response which can lead damaging effect on the epithelium leading to a progressive altering of oral epithelium directed towards carcinogenesis (23–25). In the current study, there was a higher mean number of inflammatory cell infiltration in smokers (38.5 ± 6.4 cells/HPF), when compared with non-smokers (16.8 ± 4.1 cells/HPF; $p < 0.001$), which concurs with the role of inflammation as an important mediator for changes to oral health due to tobacco consumption.

The smoking, dysbiotic-microbiome and epithelial transitions were confirmed using population- based correlational analysis. The strong correlation between the smoking duration and N/C ratio ($r = 0.68$, $p < 0.001$) suggests tobacco exposure-related epithelial cells morphology defects accumulate over time. Similarly, the association between duration of smoking and bacterial load ($r = 0.66$, $p < 0.001$) is consistent with the theory that showed the augment microbial colonization due to chronic smoking.

The correlation between bacterial load with the inflammatory cell count was strong ($r = 0.69$, $p < 0.001$), the result of the current study indicated that changes in the oral microbial population have contributed to the observed inflammatory response in the oral mucosa. This conclusion is consistent with Hajishengallis, (2014) & zhoa *et al.,* (2023) suggestions that disrupted microbial populations acted as drivers of chronic inflammation (26,27).

The N/C ratio, bacterial load, inflammatory cell count and frequency of atypical cells were significantly higher in heavy smokers compared to light smokers. The dose–response relationship is consistent with other studies. Vellappally *et al.,* (2027) reported that the severity of changes in oral tissues was correlated with the degree and duration of

smoking (28). The increased incidence of cytological atypia was higher in heavy smokers (45.5%) than in light smokers (22.2%) suggesting that some degree of epithelial dysplasia occurs with progressive tobacco exposure as seen in the current study.
The results of the current study showed that the utilization of non-invasive oral swab cytology offered a pragmatic and ethically acceptable approach to assess potential epithelial changes among healthy individuals. This strategy could enable the detection of subclinical changes before there is any need for invasive procedures in study participants. This study has some limitations including; the relatively small sample size and restricted age range which may limit the generalisation of the findings. Additionally, the cross-sectional design precludes establishing a causal relationship between smoking, microbial dysbiosis, and epithelial cytological alterations.

CONCLUSION

In conclusion, smokers have an altered diversity of the oral microbiome as compared to non-smokers, higher bacterial load and changes were noted at epithelial cytology level with an increased N/C ratio and inflammatory infiltration seen upon cytological examination. These effects are dose-dependent, and also correlate with smoking duration. The findings suggest that combined salivary microbial analysis and exfoliative cytology may be useful non-invasive approaches for detecting early smoking-related oral alterations before the development of clinically apparent lesions.

Declarations

Acknowledgment

The authors sincerely acknowledge the National Health Factory for its support through the provision of critical equipment and technical resources

Competing interests statement

The authors declare that they have no competing interests.

Ethics statement

The author approved that this research follows the journal's Attached Ethic Approval guidelines as appeared on the journal's author guidelines page.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author contributions

MAM contributed to the study conception and design, data analysis, microbiological analysis, interpretation of results, and manuscript writing. **AMA** contributed to data collection, cytological analysis, and manuscript revision. **RID** contributed to sample collection, statistical analysis, interpretation of results, and critical revision of the manuscript. All authors read and approved of the final manuscript.

References

1. Agnihotri R, Gaur S. Implications of tobacco smoking on the oral health of older adults. Geriatr Gerontol Int . 2014 ; 14(3):526–540 . doi:10.1111/ggi.12285

2. Sakthisankaran SM, Sakthipriya D, Swamivelmanickam M. Health Risks Associated with Tobacco Consumption in Humans: An Overview. J Drug Deliv Ther . 2024; 14(5):163–173 . doi:10.22270/jddt.v14i5.6523

3. Ford PJ, Rich AM. Tobacco Use and Oral Health. Addiction . 2021 ; 116(12):3531–3540 . doi:10.1111/add.15513

4. Faran Ali SM, Tanwir F. Oral microbial habitat a dynamic entity. J Oral Biol Craniofacial Res . 2012 ; 2(3):181–187 . doi:10.1016/j.jobcr.2012.07.001

5. Li X, Liu Y, Yang X, Li C, Song Z. The Oral Microbiota: Community Composition, Influencing Factors, Pathogenesis, and Interventions. Front Microbiol . 2022 ; 13:895537 . doi:10.3389/fmicb.2022.895537

6. Belizário JE, Napolitano M. Human microbiomes and their roles in dysbiosis, common diseases, and novel therapeutic approaches. Front Microbiol. 2015; 6(OCT):151578 . doi:10.3389/fmicb.2015.01050

7. Huang C, Shi G. Smoking and microbiome in oral, airway, gut and some systemic diseases. J Transl Med . 2019 ; 17(1):225 . doi:10.1186/s12967-019-1971-7

8. Shapiro H, Goldenberg K, Ratiner K, Elinav E. Smoking-induced microbial dysbiosis in health and disease. Clin Sci . 2022 ; 136(18):1371–1387 . doi:10.1042/CS20220175

9. Prince Y. The oral microbiome and its association with chronic and systemic disease in a South African population. Cape Peninsula University of Technology . Preprint posted online 2021 . https://etd.cput.ac.za/handle/20.500.11838/3380

10. Michcik A, Cichorek M, Daca A, et al. Tobacco smoking alters the number of oral epithelial cells with apoptotic features. Folia Histochem Cytobiol . 2014 ; 52(1):60–68 . doi:10.5603/FHC.2014.0007

11. Dilovar Rustamovna K. the Effect of Tobacco Smoking on the Organs and Tissues of the Oral Cavity. World Bull Public Heal . 2023 ; 19:216–220 . https://www.scholarexpress.net

12. Prasad K. Utility of Exfoliative (SCRAPE) Cytology In Early Diagnosis of Oral Malignancies and In Identification of Risk Factors In the Population of North Karnataka, Mainly Hubli and Dharwad. Rajiv Gandhi University of Health Sciences (India) . Preprint posted online 2014.

13. Voltaggio L, Cimino-Mathews A, Bishop JA, et al. Current concepts in the diagnosis and pathobiology of intraepithelial neoplasia: A review by organ system. CA Cancer J Clin . 2016 ; 66(5):408–436 . doi:10.3322/caac.21350

14. Tilakaratne WM, Jayasooriya PR, Jayasuriya NS, De Silva RK. Oral epithelial dysplasia: Causes, quantification, prognosis, and management challenges. Periodontol 2000 . 2019 ; 80(1):126–147 . doi:10.1111/prd.12259

15. Ebersole JL, Schuster JL, Stevens J, et al. Patterns of salivary analytes provide diagnostic capacity for distinguishing chronic adult periodontitis from health. J Clin Immunol . 2013 ; 33(1):271–279 . doi:10.1007/s10875-012-9771-3

16. Shannon CE. A Mathematical Theory of Communication. Bell Syst Tech J . 1948 ; 27(3):379–423 . doi:10.1002/j.1538-7305.1948.tb01338.x

17. Drury R. Theory and Practice of Histological Techniques. Vol 36. Elsevier health sciences ; 1983 .

18. IBM Corpoperation. IBM SPSS Statistics for Windows. Version 25.0. IBM corp Armonk, NY; 2011.

19. Brook I. The impact of smoking on oral and nasopharyngeal bacterial flora. J Dent Res . 2011 ; 90(6):704–710 . doi:10.1177/0022034510391794

20. Al-Marzooq F, Al Kawas S, Rahman B, et al. Supragingival microbiome alternations as a consequence of smoking different tobacco types and its relation to dental caries. Sci Rep . 2022 ; 12(1):2861 . doi:10.1038/s41598-022-06907-z

21. Brima EI. Detection of cytological changes in oral mucosa among users of smokeless tobacco (shamma) in the KSA. Diagn Cytopathol . 2017 ; 45(8):693–699 . doi:10.1002/dc.23745

22. Parmar ND, Master N, Gupta DS. Study of Effect of Smoking on Cytomorphometry of Buccal Mucosal Cells among Smokers in South Gujarat Region. Natl J Clin Anat . 2020 ; 9(3):90–96 . doi:10.4103/NJCA.NJCA_33_20

23. Prasetyaningtyas N, Jatiatmaja NA, Radithia D, et al. The Response of the Tongue Epithelial on Cigarette Smoke Exposure as a Risk Factor for Oral Cancer Development. Eur J Dent . 2021 ; 15(2):320–324 . doi:10.1055/s-0040-1721312

24. Khowal S, Wajid S. Role of Smoking-Mediated molecular events in the genesis of oral cancers. Toxicol Mech Methods . 2019 ; 29(9):665–685 . doi:10.1080/15376516.2019.1646372

25. Fenercioglu AK, Uzun H, Unal DO. The Convergent Immunopathogenesis of Cigarette Smoke Exposure: From Oxidative Stress to Epigenetic Reprogramming in Chronic Disease. Int J Mol Sci . 2026 ; 27(1):187 . doi:10.3390/ijms27010187

26. Hajishengallis G. The inflammophilic character of the periodontitis-associated microbiota. Mol Oral Microbiol . 2014 ; 29(6):248–257 . doi:10.1111/omi.12065

27. Zhao M, Chu J, Feng S, et al. Immunological mechanisms of inflammatory diseases caused by gut microbiota dysbiosis: A review. Biomed Pharmacother . 2023 ; 164:114985 . doi:10.1016/j.biopha.2023.114985

28. Vellappally S, Fiala Z, Smejkalová J, Jacob V, Somanathan R. Smoking related systemic and oral diseases. Acta Medica (Hradec Kralove). 2007; 50(3):161–166 . doi:10.14712/18059694.2017.76