# ASSESSING SALIVARY PARAMETERS AND CARIES RISK IN SMOKERS AND NON-SMOKERS

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***Abstract***

Saliva is crucial in maintaining oral health, serving various functions such as protecting oral tissues, cleansing the mouth, and aiding digestion. Salivary composition, including pH, buffering capacity, and protein components, has been associated with oral diseases, including dental caries. Tobacco smoking has detrimental effects on salivary glands, altering saliva composition and potentially increasing the risk of dental caries. This study aimed to investigate the relationship between saliva pH and buffering capacity by smoking status and the relationship between saliva pH and buffering capacity by caries risk. A cross-sectional study was conducted among adult smokers and non-smokers visiting the Dental Polyclinics at Universiti Kebangsaan Malaysia (UKM). Salivary parameters, including pH and buffering capacity, were assessed using GC Saliva Check Buffer kits. Caries risk assessment was performed using the American Dental Association Caries Risk Assessment form. Sociodemographic characteristics, smoking status, and caries risk were analyzed using descriptive statistics and chi-square tests. One hundred subjects participated in the study, with 49 smokers and 51 non-smokers. Smokers showed a higher prevalence of high caries risk compared to non-smokers. The buffering capacity of saliva was significantly lower in smokers compared to non-smokers for both resting and stimulated saliva ($p<0.001$). Resting ($p=0.035$) and stimulated ($p=0.049$) saliva pH was significantly more acidic in smokers than in non-smokers. These findings suggest that smoking status is associated with altered salivary parameters and increased caries risk. Understanding the impact of smoking on saliva composition and caries risk can aid in developing preventive and therapeutic strategies for individuals at high risk of dental decay.

***Keywords****:* Saliva, Oral Health, Oral Tissues, Salivary Parameters, pH, Buffering Capacity, Dental Caries, Tobacco Smoking

## *Introduction*

Saliva, a vital element for preserving oral health, is an intricate fluid composed of electrolytes, salivary and serum proteins, organic molecules, and residues from microorganisms in the oral cavity (1, 2). Saliva hydrates and safeguards oral tissues, cleanses gums and teeth and aids speech and swallowing. It also plays crucial roles, such as buffering the oral cavity, forming a protective pellicle, promoting tooth mineralization, exhibiting antimicrobial activity, facilitating tissue repair, and contributing to taste and digestion (2). Among healthy adults, the average flow rate of unstimulated whole saliva ranges from 0.3 to 0.4 mL/min, with a flow rate below 0.1 mL/min categorized as hyposalivation (3). The salivary glands in humans generate a volume of whole saliva ranging from 0.5 to 1.5 litres within 24 hours (4).

Dental caries is a complex disease influenced by multiple risk factors, such as oral hygiene practices, dietary habits, and the composition of saliva (5). Saliva has the potential to serve as a predictor for oral diseases. The recognition of the possibility of utilizing specific protein components in saliva as biomarkers for diagnosing oral diseases has been acknowledged for a considerable time (6). There is scant evidence linking dental caries to particular salivary components, and the support for saliva pH, buffering capacity, proteins, or electrolyte composition as reliable indicators of the risk factors associated with dental caries is weak (7).

The initial impact of the harmful effects of cigarettes on the salivary glands primarily targets the parotid gland, responsible for secreting watery saliva (8). Submandibular and sublingual glands compensate for losing their function by secreting mucous saliva, hence smokers' thicker saliva

(9). The compounds found in cigarette smoke can disrupt the protective macromolecules, enzymes, and proteins in saliva. Consequently, saliva loses its defensive function and contributes to the carcinogenesis and development of oral and oropharyngeal cancer (10, 11).

Tobacco smoke affects saliva and increases the occurrence of dental caries (12). Smoking is a prevalent habit associated with numerous adverse oral health outcomes, but its specific effects on salivary parameters and caries risk remain underexplored (13). Saliva provides essential protective functions, such as buffering, remineralization, and antimicrobial activity, which help maintain oral health (14).

The mean number of carious lesions was significantly higher in smokers than in non-smokers (12). Although there is a correlation between tobacco smoking and an increased risk of dental caries, the evidence is not strong (15). Similarly, the effects of tobacco consumption on some salivary characteristics, such as flow rate, buffering capacity, pH and consistency, are controversial (16). Nicotine, a toxic, addictive cigarette component, may act on specific brain cholinergic receptors and other organs, causing neural activation and altered salivary secretion (17).

Caries risk assessment is a systematic evaluation conducted by dental professionals to determine an individual's susceptibility to dental caries (tooth decay) and predict their likelihood of developing new cavities (18). It involves assessing various risk factors that contribute to the initiation and progression of caries, including biological, behavioural, and environmental factors (19). Caries risk assessment aims to identify individuals at high risk for caries so that appropriate preventive measures can be implemented to minimize the occurrence and progression of dental decay (20). Although the validity of standardized caries risk assessment (CRA) models remains limited, assessing caries risk is essential in planning preventive and therapeutic strategies (21). Thus, this study aimed to investigate the relationship between saliva pH and buffering capacity by smoking status and the relationship between saliva pH and buffering capacity by caries risk.

## *Materials and Methods*

### *Study design*

A cross-sectional investigation was carried out to evaluate the salivary parameters and the risk of caries among adult smokers and non-smokers attending the Dental Polyclinics at Universiti Kebangsaan Malaysia (UKM).

### *Sample size determination and sampling procedure*

The sample size for this study was calculated based on Rad et al. (22), where the incidence of xerostomia was reported at 39% among smokers and 12% among non-smokers. The sample size was calculated utilizing a sample size calculator (23). The probability of type-I error (α) was set at 0.05, and the probability of type-II error (β) was set at 0.2. Thus, the total sample size needed was 80 subjects to have 80% power. Allowing 10% dropouts, a final total sample size of 88 subjects, with 44 subjects in each group of smokers and non-smokers. Study participants were selected by convenient sampling method after screening through the inclusion and exclusion criteria and segmented into smokers and non-smokers.

The inclusion criteria for both groups were Malaysian adults aged 18-60 years old and willing to participate in the study. Smokers were considered current users of cigarettes for more than six months. Age and gender-matched study subjects not taking tobacco in any form were included as non-smokers. The exclusion criteria were patients who consume alcohol regularly and pregnant or lactating females.

### *Salivary parameters estimation*

A single examiner employed the Saliva Check Buffer kits by GC (Tokyo, Japan) (Figure 1a) to collect both stimulated and unstimulated saliva samples to determine salivary pH and buffer capacity (24). This approach aimed to ensure precise results by avoiding calibration errors. Saliva collection occurred during the morning following a 12-hour overnight fasting period, with no food or beverages other than water consumption. Patients were advised to refrain from brushing their teeth or using mouthwash for at least one hour before the appointment. Saliva was expectorated into the provided collection cup. The resting saliva underwent visual examination, and a salivary pH test strip was immersed in the sample for 10 seconds (Figure 1b). The obtained colour was subsequently matched with the testing chart provided in the kit (Figure 1c). Saliva pH readings above 6.8 indicated healthy saliva, while values ranging from 6.6 to 6 were considered moderately acidic, and those below six were categorized as highly acidic (24).

The assessment of salivary buffer capacity is crucial as it illustrates saliva's ability to neutralize acids in the oral environment. For this evaluation, specialized disposable test strips were employed. Patients were directed to chew wax gums for 5 minutes, ensuring the collection of all saliva into the designated cup at regular intervals. The volume of stimulated saliva was quantified by examining the markings on the dispensing cup (Figure 1d). Each salivary buffer test strip was packaged separately for single use (Figure 1b). Saliva was extracted from the collection cup using a pipette, and three drops were administered onto the test strip, with one drop allotted to each of the three test pads. Subsequently, the test strip was promptly rotated at a 90-degree angle to eliminate excess saliva, thereby safeguarding the accuracy of the test outcome. The test pads exhibited an immediate colour change, and the final results were displayed after a 2-minute interval (Figure 1e).

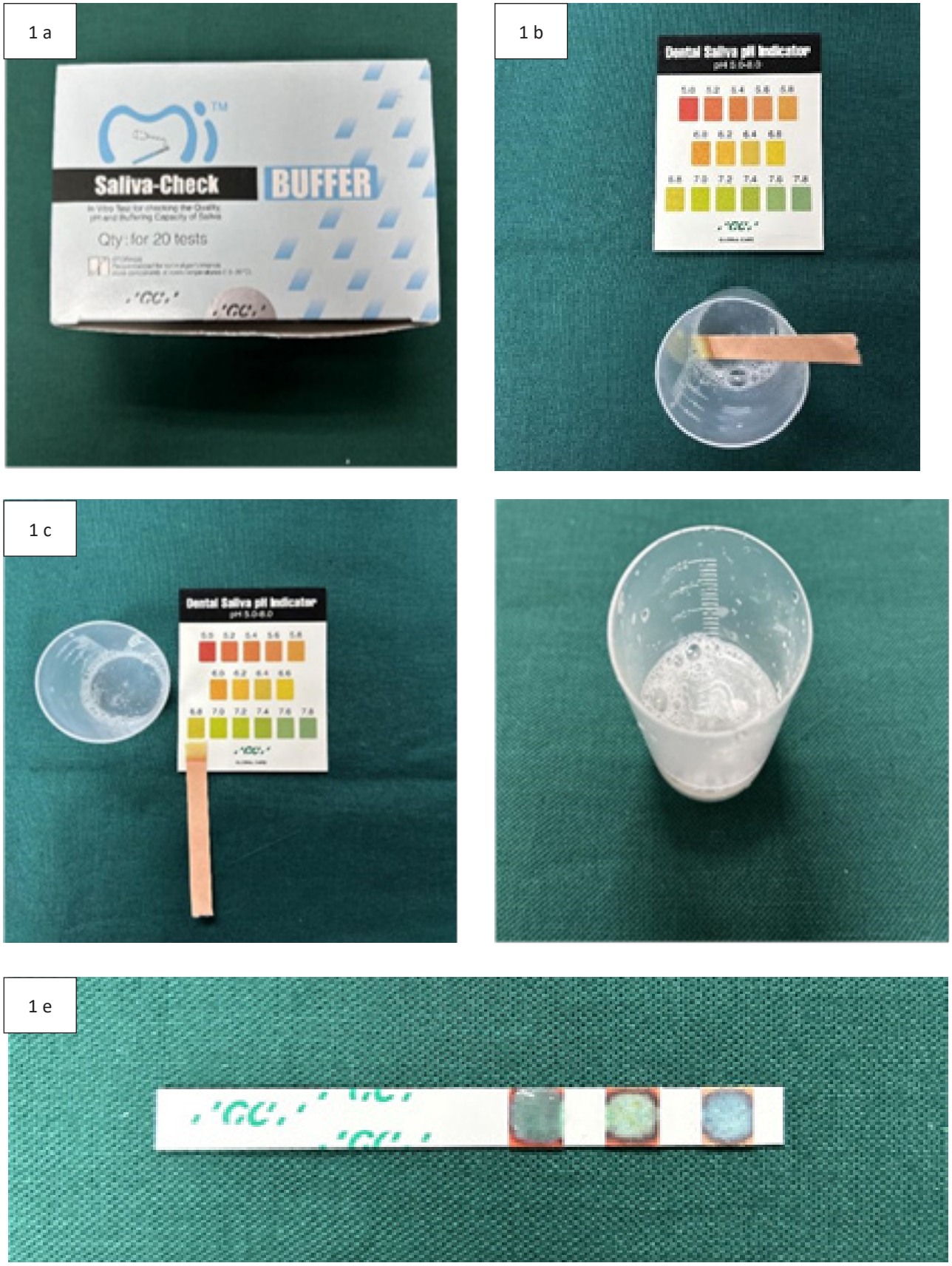


**Figure 1:** (a) The GC Saliva Check Buffer kit; (b) pH test strip in saliva; (c) saliva pH indicator; (d) collected saliva in a cup; (e) buffer test results.

Points were calculated based on the colours shown on the test pads of the buffer strip (Table 1). Results were interpreted based on the combined total points of the three test pads (Table 2). To conduct pH testing, a salivary pH test strip was inserted into the sample of stimulated saliva and left in place for 10 seconds. Buffering capacity in saliva refers to its ability to resist changes in pH when an acidic or alkaline substance is introduced. It is a crucial property that helps maintain the oral environment within a relatively stable pH range, vital for oral health.

**Table 1:** Evaluation of saliva buffering capacity with GC Saliva Check kit. The buffer test strip shows green, green/blue, and red/blue colours.

| Colour | Points |
|---|---|
| Green | 4 points |
| Green/Blue | 3 points |
| Blue | 2 points |
| Blue/Red | 1 points |
| Red | 0 points |

**Table 2:** Categories of saliva buffering capacity

| Combined total | Saliva buffering ability |
|---|---|
| 0-5 | Very low |
| 6-9 | Low |
| 10-12 | Normal |

The test pads, often used to assess buffering capacity, contain indicator dyes that change colour based on the saliva's pH. The colours indicate the level of acidity or alkalinity:

1. High Buffering Capacity (Alkaline):
    - Colour: Blue or Green
    - Explanation: Saliva with high buffering capacity can neutralize acids effectively, maintaining a more alkaline pH. The indicator dye on the test pad remains in the blue or green range.
2. Moderate Buffering Capacity:
    - Colour: Yellow or Light Green
    - Explanation: Saliva with moderate buffering capacity can somewhat neutralize acids but might experience a moderate pH drop. The indicator dye may change to yellow or light green.
3. Low Buffering Capacity (Acidic):
    - Colour: Red or Pink
    - Explanation: Saliva with low buffering capacity struggles to neutralize acids, resulting in a significant drop in pH. The indicator dye on the test pad turns red or pink.

These colours serve as a visual indication of the saliva's buffering capacity, helping in rapid assessment and understanding of an individual's oral health regarding acid-base balance. Monitoring buffering capacity is essential for evaluating susceptibility to dental issues like tooth decay and enamel erosion and determining the need for preventive measures such as improved oral hygiene or dietary modifications.

### *Assessment of caries risk profile*

The Caries Risk Assessment (CRA) was retrospectively collected from patients' treatment folders. The Caries Risk Assessment (CRA) form for (ages > 6 years) developed by the American Dental Association (ADA) was used in the clinic to determine patients' caries risk (25). The ADA Caries Risk Assessment form classifies a patient's overall risk of developing caries based on their history and clinical examination. Factors indicating high caries risk include:

- Consumption of Sugary Foods or Drinks: frequent or prolonged exposure between meals per day (for individuals over 6 years old)
- Undergoing Chemo/Radiation Therapy (for individuals over 6 years old)
- Presence of Cavitated or Noncavitated (incipient) Carious Lesions or Restorations (detectable visually or radiographically): 3 or more such lesions or restorations within the last 36 months (for individuals over 6 years old)
- History of Teeth Missing Due to Caries in the past 36 months (for individuals over 6 years old)
- Experience of Severe Dry Mouth (Xerostomia; for individuals over 6 years old)

The final data collected for Caries Risk Assessment (CRA) were categorized as low, moderate, and high.

### *Statistical analysis*

The data underwent processing and analysis using Statistical Package for the Social Sciences (SPSS) Version 28.0 (IBM, Armonk, NY). Descriptive statistics included calculating frequencies and percentages for categorical data and means and standard deviations for continuous data. The chi-square test examined associations between saliva pH and buffering capacity relative to the patient's smoking status and caries risk. Statistical significance was established at a p-value $< 0.05$.

## Results

### Sociodemographic characteristics of subjects

One hundred subjects, comprising 49 smokers and 51 non-smokers, consented to participate in this study. Table 3 shows the sociodemographic characteristics of the subjects. Most smokers were males (72.3%) and were of Malay ethnicity (81.6%). Most non-smokers were females (94.3%) and were of Malay ethnicity (70.6%). The mean age for both groups was almost similar, with no statistical difference (p=0.141).

**Table 3:** Demographic characteristics of subjects by smoking status (N=100)

| Characteristics | Smokers n=49 | Non-smokers n=51 | p-value |
|---|---|---|---|
| **Gender** | n (%) | n (%) | |
| Male | 47 (72.3) | 18 (27.7) | <0.001 |
| Female | 2 (5.7) | 33 (94.3) | |
| **Age (mean ± SD)** | 44.02±14.41 | 39.60±14.78 | 0.141 |
| **Ethnicity** | n (%) | n (%) | |
| Malay | 40 (81.6) | 36 (70.6) | 0.086 |
| Chinese | 9 (18.4) | 10 (19.6) | |
| Indian | 0 (0) | 5 (9.8) | |

Table 4 compares smoking status with the caries risk of subjects. A statistically significant association exists between caries risk and smoking status (p<0.003). Smokers (65.3%) have high caries risk compared to non-smokers (31.4%). Non-smokers (27.5%) have low caries risk compared to smokers (14.3%).

**Table 4:** Comparison of smoking status with caries risk of subjects (N=100)

| Oral health status | Smokers n=49 | Non-smokers n=51 | Chi-square value $\chi^2$ | p-value |
|---|---|---|---|---|
| Caries risk | n (%) | n (%) | | |
| High | 32 (65.3) | 16 (31.4) | 11.535 | 0.003* |
| Moderate | 10 (20.4) | 21 (41.2) | | |
| Low | 7 (14.3) | 14 (27.5) | | |

*Pearson Chi-square test, p-value significant at p < 0.05

Table 5 shows the relationship between saliva buffering capacity with smoking status. A statistically significant association was found between saliva buffering capacity and smoking status (p<0.001) for resting and stimulated saliva. Smokers (36.7%) had very low resting saliva buffering capacity compared to non-smokers (2.0%). More non-smokers (29.4%) have normal resting saliva buffering capacity than smokers (4.1%). More smokers (15.0%) had very low buffering capacity for stimulated saliva than non-smokers (0%). More non-smokers (45.1%) had normal buffering capacity than smokers (28.0%).

**Table 5**: Relationship between saliva buffering capacity with smoking status (N=100)

| Buffering capacity | Smokers n=49 | Non-smokers n=51 | Chi-square value $\chi^2$ | p-value |
|---|---|---|---|---|
| Resting | n (%) | n (%) | | |
| Very Low | 18 (36.7) | 1 (2.0) | 25.684 | <0.001* |
| Low | 29 (59.2) | 35 (68.6) | | |
| Normal | 2 (4.1) | 15 (29.4) | | |
| Stimulated | n (%) | n (%) | | |
| Very Low | 15 (15.0) | 0 (0) | | |
| Low | 57 (57.0) | 28 (54.9) | 26.560 | <0.001* |
| Normal | 28 (28.0) | 23 (45.1) | | |

% within the smoking status; *Pearson Chi-square test, p-value significant at p < 0.05

A statistically significant association was found between resting saliva pH (p=0.035) and stimulated saliva pH (p=0.049) with smoking status (Table 6). More smokers (n=38, 77.6%) had significantly acidic resting saliva pH than non-smokers (n=27, 52.9%). In contrast, more non-smokers (n=16, 31.4%) have alkaline resting saliva pH than smokers (n=7, 14.3%). More smokers (n=17, 34.7%) had significantly acidic stimulated saliva pH than non-smokers (n=9, 17.6%). However, more non-smokers (n=39, 76.5%) have alkaline-stimulated saliva pH than smokers (n=26, 53.1%).

**Table 6**: Relationship between saliva pH with smoking status (N=100)

| Saliva pH | Smokers n=49 | Non-smokers n=51 | Chi-square value $\chi^2$ | p-value |
|---|---|---|---|---|
| Resting | n (%) | n (%) | | |
| Acidic | 38 (77.6) | 27 (52.9) | 6.679 | 0.035* |
| Neutral | 4 (8.2) | 8 (15.7) | | |
| Alkaline | 7 (14.3) | 16 (31.4) | | |
| Stimulated | | | | |
| Acidic | 17 (34.7) | 9 (17.6) | | |
| Neutral | 6 (12.2) | 3 (5.9) | 6.024 | 0.049* |
| Alkaline | 26 (53.1) | 39 (76.5) | | |

% within the smoking status; *Pearson Chi-square test, p-value significant at p < 0.05

Table 7 shows the relationship between saliva pH and buffering capacity by caries risk. Buffering capacity for resting ($\chi^2$=12.364, p=0.015) and stimulated ($\chi^2$=22.509, p=0.000) saliva showed a statistically significant relationship between caries risks. Subjects with low resting (75.0%) and low stimulated (75.0%) saliva buffering capacity have high caries risk compared to subjects with normal resting (6.3%) and normal stimulated (10.4%) saliva buffering capacity. Similarly, a significant relationship exists between saliva pH (resting) and caries risk ($\chi^2$=15.504, p=0.004). Subjects with acidic resting saliva pH have high caries risk (77.1%), whereas subjects with alkaline resting saliva pH have low

**Table 7**: Relationship between saliva pH and buffering capacity by caries risk (N=100)

| 1. Buffering capacity | Caries Risk | | | | Chi-square value $\chi^2$ | p-value |
|---|---|---|---|---|---|---|
| | Low n (%) | Moderate n (%) | High n (%) | Total n (%) | | |
| **Resting** | | | | | | |
| **Very Low** | 5 (23.8) | 5 (16.1) | 9 (18.8) | 19(19.0) | 12.364 | 0.015* |
| **Low** | 8 (38.1) | 20 (64.5) | 36 (75.0) | 64 (64.0) | | |
| **Normal** | 8 (38.1) | 6 (19.4) | 3 (6.3) | 17 (17.0) | | |
| **Stimulated** | | | | | | |
| **Very Low** | 4 (19.0) | 4 (12.9) | 7 (14.6) | 15 (15.0) | | |
| **Low** | 4 (19.0) | 17 (54.8) | 36 (75.0) | 57 (57.0) | 22.509 | <0.001* |
| **Normal** | 13(61.9) | 10 (32.3) | 5 (10.4) | 28 (28.0) | | |
| **2. Saliva pH** | | | | | | |
| **Resting** | | | | | | |
| **Acidic** | 9 (42.9) | 19 (61.3) | 37 (77.1) | 65 (65.0) | | |
| **Neutral** | 1 (4.8) | 6 (19.4) | 5 (10.4) | 12 (12.0) | 15.504 | 0.004* |
| **Alkaline** | 11 (52.4) | 6 (19.4) | 6 (12.5) | 23 (23.0) | | |
| **Stimulated** | | | | | | |
| **Acidic** | 6 (28.6) | 5 (16.1) | 15 (31.3) | 26 (26.0) | | |
| **Neutral** | 1 (4.8) | 4 (12.9) | 4 (8.3) | 9 (9.0) | 3.026 | 0.554 |
| **Alkaline** | 14 (66.7) | 22 (71.0) | 29 (60.4) | 65 (65.0) | | |

% within caries risk; * p values < 0.05 were considered significant

caries risk (52.4%). However, no statistically significant difference was found between saliva pH (stimulated), caries risk, and smoking status.

## *Discussion*

In this study, it was observed that the proportion of male smokers exceeded that of female smokers, aligning with national data in Malaysia (26-28) as well as trends in various other Asian countries such as Vietnam (34:1) (29), Taiwan (9:1) (30), and Singapore (5.6:1) (31). This prevalence of male smokers could be attributed to social norms in Malaysian culture discouraging female smoking. Moreover, the study highlighted a higher prevalence of smokers within the Malay ethnicity and among individuals aged 38–44 years old, consistent with previous Malaysian data (28).

Several research studies have explored the association between smoking and dental caries (15). However, in those studies, caries were determined by measurements of decayed, missing or filled teeth (DMFT), decayed, missing or filled surface (DMFS), or caries-related microflora levels. This study used Caries risk assessment (CRA), essential for successfully managing dental caries with tailored preventive care and risk monitoring (32). Caries risk assessment enables the estimation of the likelihood

of new cavity formation or the development of incipient lesions within a specific period, as well as the probability of changes in the size or activity of existing caries lesions (33). A precise caries risk assessment is valuable in identifying patients with a high risk of caries, facilitating the implementation of preventive therapies, and enhancing the effectiveness of treatment. The risk of dental caries can be evaluated by analyzing and integrating several causative factors. These include caries experience (ICDAS score 1-6), the extent of plaque present, fluoride use, diet, crowding, presence of dental appliances, salivary activity and social and behavioural factors (34). This study has found that smokers have higher caries risk than non-smokers. Mittal et al. demonstrated similar findings, indicating that a tobacco habit is a risk factor associated with elevated caries activity (35). A study by Tanner et al. concluded that the high rate of restorative treatment needed among smokers is 2-fold higher than among non-smokers (36). In a laboratory study, the effect of nicotine was found to increase the growth of *S. Mutans*, where smoker isolates produce more biofilm compared to non-smoker isolates indicating the risk for dental caries (37). Zitterbart et al. (38) confirmed an association between smoking and the prevalence of dental caries where smokers had significantly higher DMFT scores than non-smokers. He also correlated the number of tooth surfaces lost in a smoker's mouth with daily cigarette usage. Our results contradict a study by Hugoson et al. which indicated that daily smoking or snus (smokeless tobacco product) use does not increase the risk of dental caries (39). However, the study groups are not directly comparable due to differences in the age of participants, background factors, and cultural and health behaviour, which cause variation in the results.

Our study found a significant association between saliva buffering capacity and smoking status for resting and stimulated saliva ($p<0.001$). Smokers were found to have very low saliva buffering capacity compared to non-smokers. It was shown that smoking drastically changed saliva's pH and buffering capacity, which can have several harmful and detrimental effects on the oral mucosa (40). Recent research has demonstrated that individuals with elevated saliva pH levels and enhanced buffering capacity experience improved oral health outcomes and reduced dental caries (41). On the other hand, smokers exhibit a diminished buffering effect and a lower pH level in their saliva while having a higher count of Lactobacilli and Streptococcus mutans. These factors may suggest a heightened vulnerability to dental caries (42).

Saliva is crucial in maintaining the oral environment, with its pH typically kept within a neutral range of 6.7-7.3 (14). When the pH of saliva drops below 5.5, which is considered acidic, it can lead to tooth enamel erosion. The presence of saliva is undeniably beneficial for oral health, and its absence can expedite the onset of oral diseases (2). Saliva serves multiple functions, acting as a cleansing solution, a buffer to regulate pH, a lubricant, and a reservoir of calcium and phosphate ions, essential for remineralizing tooth decay's early stages (2). Generally, a higher saliva flow rate leads to faster clearance of harmful substances and increased buffering capacity, thereby reducing the risk of microbial attacks (14). Under normal resting conditions without external stimulation, saliva flows slowly, ensuring the mouth remains moist (14). The buffering capacity, both in resting and stimulated states, as well as the pH of saliva at rest, is significantly associated with the risk of dental caries and smoking (14). This is because unstimulated saliva is vital for maintaining oral health and protects against dental caries in the oral cavity.

This study has certain limitations. Given that it was carried out under the constraints of stringent clinical protocols due to the COVID-19 pandemic, caries risk assessment data had to be extracted from the most recent patient records. In the clinic, only salivary parameters and assessments of tobacco use were conducted. The sample size for this study might be relatively small, potentially restricting the generalizability of the findings to a broader population. A larger sample size would yield more robust and representative results. Additionally, there is a potential selection bias as participants were not randomly chosen, introducing the possibility of bias towards specific characteristics or behaviours that may not represent the general population.

For future research, it is advisable to utilize a longitudinal study design to establish cause-and-effect relationships, thereby addressing the limitations inherent in the cross-sectional nature of the current study. Additionally, future investigations should meticulously account for potential confounding variables such as diet, oral hygiene practices, and socioeconomic status that may influence the relationship between smoking status, salivary parameters, and caries risk. Moreover, future research could expand the scope of salivary parameters examined to ensure a more thorough exploration of factors affecting caries risk. Recognizing and tackling these factors in future research endeavours will enhance the depth and breadth of comprehension regarding the subject matter. Understanding the impact of smoking on salivary parameters and caries risk is crucial for dental professionals in designing effective preventive strategies. Oral health interventions targeting smokers should promote smoking cessation, improve oral hygiene practices, and compensate for altered salivary composition through adjunctive measures such as fluoride application and saliva substitutes.

## *Conclusion*

Cigarette smoking adversely affects salivary parameters, including pH and buffering capacity, subsequently increasing the risk of dental caries. The altered salivary composition and the microbial and behavioural factors associated with smoking contribute to a higher caries risk among smokers. Further research is needed to

elucidate the underlying mechanisms and develop tailored preventive approaches for this vulnerable population.


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## *Competing interests*

The authors declare that they have no competing interests.

## *Ethical Clearance*

We obtained approval from Universiti Kebangsaan Malaysia (UKM) Research Ethics Committee (UKMREC)(Reference number: UKM PPI/111/8/JEP-2020-616).

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