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Association between Salivary Factors and Growth of Cariogenic Bacteria in Type-2 Diabetes Mellitus

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ABSTRACT

Background & objective: Dental caries is a chronic infectious disease involving the destruction of dental hard tissues by lactic acid, primarily initiated by *Streptococcus mutans* and progressed by *Lactobacilli*. In patients with Type-2 Diabetes Mellitus (T2DM), factors such as poor glycemic control and altered salivary properties are linked to increased caries risk. This study aimed to evaluate the association between salivary factors, glycemic status, and the growth of cariogenic bacteria in T2DM patients.

Methods: This cross-sectional analytical study included 108 T2DM patients (aged >30 years, diagnosed for ≥ 1 year) recruited from the Outpatient Department of Dentistry, National Healthcare Network (NHN), Wari under the Diabetic Association of Bangladesh, Dhaka. While exposure variables included salivary pH, glucose, flow rate, and saliva buffering capacity, outcome variables were the type and load of cariogenic bacteria (High load: ≥ 105 CFU/ml, and Low load: <105 CFU/ml). Uncontrolled diabetes was defined as HbA1c ≥ 6.5%. Salivary flow was categorized as normal flow rate (> 1 g/min), moderately low flow rate (0.7-1 g/min) and very low flow rate (< 0.7 g/min), and buffering capacity was determined by comparing the color of the test field with that of the sample as blue, green and yellow colors indicating high, moderate and low buffering capacity respectively.

Results: Neither age nor sex showed a significant association with bacterial load. However, elevated Fasting Blood Sugar (FBS) significantly increased the risk of a high bacterial load (OR: 6.4, 95% CI: 2.4–17.2, p < 0.001). Uncontrolled diabetes was associated with a nearly 12-fold higher risk of significant cariogenic bacterial load compared to controlled cases (OR: 11.7, 95% CI: 3.6–38.1, p < 0.001). Xerostomia also showed a strong correlation with high bacterial load (OR: 6.0, 95% CI: 2.1–16.8, p < 0.001). Furthermore, low salivary pH, elevated salivary glucose, very low salivary flow rate (found in 47.6% of high-load cases), and low-to-moderate buffering capacity (found in 90.5% of high-load cases) were all significantly associated with increased bacterial growth (p < 0.001).

Conclusion: Elevated FBS, uncontrolled glycemic status, and xerostomia are major drivers of high cariogenic bacterial loads in T2DM patients. Additionally, physiological changes in saliva-specifically reduced pH, increased glucose levels, low flow rates, and diminished buffering capacity-significantly promote the proliferation of *Streptococcus mutans* and *Lactobacilli*. These findings emphasize the importance of integrated oral health monitoring and strict glycemic management to mitigate caries risk in diabetic populations.

Keywords: Association, Salivary Factors, Growth, Cariogenic Bacteria, Type-2 Diabetes Mellitus

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) has emerged as a global epidemic, accounting for approximately 90–95% of all diabetes cases. According to the International Diabetes Federation (IDF), 415 million individuals were affected by diabetes worldwide in 2017. Global prevalence was estimated at 9.3% (463 million) in 2019, with projections suggesting an increase to 10.2% (578 million) by 2030 and 10.9% (700 million) by 2045. In Bangladesh, the diabetic population was 8.4 million in 2019, a figure expected to reach 10.1 million by 2030 and 15 million by 2045.[1] Sustained fasting blood glucose levels exceeding 125 mg/dL are diagnostic of the disease. Such hyperglycemia is frequently reflected in elevated salivary glucose levels, which have been proposed as a non-invasive biomarker for T2DM.[2] Among the various systemic complications of T2DM, dental caries represents a significant yet preventable oral health challenge. However, susceptibility to caries varies among diabetic patients, suggesting that the interplay of individual risk factors necessitates further clarification.

Extensive research has investigated the determinants of dental caries in T2DM populations. An Indian study involving 400 patients revealed a root caries prevalence of 42%, with significant associations found between caries experience, advancing age, periodontal pocketing, and clinical attachment loss (> 3 mm).[3] Furthermore, comparative studies have shown that patients with poorly controlled T2DM exhibit significantly higher plaque indices, bleeding on probing, and altered mean buffering capacities compared to controlled diabetic and non-diabetic cohorts.[4]

Dental caries is a chronic infectious disease characterized by the demineralization of dental hard tissues. This destruction is mediated by lactic acid produced through the fermentation of dietary carbohydrates by bacteria residing in the oral biofilm.[5] The primary cariogenic pathogens include *Streptococcus mutans*, the principal initiator of decay, and *Lactobacilli*, which are typically more active during lesion progression.[6] While the link between high *Mutans streptococci* counts and caries experience is widely acknowledged, some literature suggests this association is not universal.[7,8] The pathogenicity of these facultative Gram-positive cocci stems from their ability to synthesize extracellular polysaccharides, with biofilm-forming strains of *S. mutans* being particularly linked to higher caries experience[7,9]

Although diabetic patients are predisposed to dental caries, the presence of cariogenic bacteria in salivary biofilms does not result in uniform disease susceptibility. This variation suggests that the factors predisposing individuals to caries are multifaceted and patient-specific. Poor glycemic control, diminished salivary buffering capacity, and an increased number of missing teeth have been linked to both root surface and coronal caries in T2DM patients.[10] The precise role of systemic markers (blood glucose, HbA1c) and local salivary parameters (glucose concentration, flow rate, and buffering capacity) in the development of caries within the T2DM population remains insufficiently understood. Despite these observations, many patients harbor preventable risk factors-specifically high bacterial loads and suboptimal glycemic status that require further investigation. Consequently, this study was designed to evaluate the association between salivary factors and the proliferation of cariogenic bacteria in T2DM patients by measuring salivary glucose, flow rate, and buffering capacity. Additionally, the study assessed the influence of fasting blood glucose (FBG) and glycated hemoglobin (HbA1c) on the oral bacterial load. Evaluating the association between these variables and the salivary bacterial load is essential for identifying high-risk individuals and developing targeted preventive strategies. The primary objective of this study was to determine the prevalence of high cariogenic bacterial loads and to elucidate the role of salivary and systemic factors in the growth of *Streptococcus mutans* and *Lactobacilli* among Type 2 diabetic patients.

METHODS

This cross-sectional analytical study was conducted at the Outpatient Department of Dentistry, National Healthcare Network (NHN), Wari under the Diabetic Association of Bangladesh, Dhaka over a one-year period from January to June 2022. The study protocol received formal approval from the Institutional Review Board of Primeasia University and the Ethical Review Committee of the concerned authorities. All procedures were performed in strict accordance with the World Medical Association Declaration of Helsinki (1964), as amended in 2013.

A total of 108 Type 2 diabetes mellitus (T2DM) patients aged >30 years with a confirmed diagnosis of T2DM for at least one year were consecutively included in the study . Patients with Type 1 diabetes, pregnancy, or total edentulism, active smokers, individuals with oral mucosal injuries or severe gingivitis, and those recently using antibiotics or antibacterial mouth rinses were excluded. Furthermore, patients experiencing xerostomia secondary to non-diabetic medications were excluded to maintain focus on diabetes-related salivary changes.

Clinical examinations and interviews were conducted by the investigator himself using a semi-structured questionnaire. Prior to sample collection, participants were provided with detailed instructions regarding fasting and oral hygiene. Patients were instructed to refrain from toothbrushing for at least one hour before the scheduled appointment. To prevent sample dilution, subjects rinsed their mouths with distilled water and waited five minutes before collection. Stimulated whole saliva was collected over a five-minute period using the paraffin pellet method, with patients seated upright and heads tilted slightly forward. Samples were collected in sterile tubes and were stored at -20^{O}C until analysis. Fasting blood samples were obtained immediately prior to saliva collection to synchronize glycemic data (FBG and HbA1c) with salivary data.

Fasting blood glucose (FBG) and glycosylated hemoglobin (HbA1c) data were extracted from standardized patient medical records. Concentrations were determined using the glucose oxidase (GOD-POD) method. Calculated by gravimetric analysis (weighing the collection tube before and after sampling). Rates were categorized as Normal (> 1 g/min), Moderately Low (0.7–1.0 g/min), and Very Low (< 0.7 g/min). Buffering capacity and bacterial load were assessed using a chair-side diagnostic kit (CRT Bacteria, Ivoclar Vivadent, Liechtenstein). Semi-quantitatively determined by colorimetric change (Blue: High; Green: Moderate; Yellow: Low). Quantification of the bacterial load (*Streptococcus mutans* and *Lactobacilli*) was based on the colony forming units per milliliter of saliva (CFU/ml) as low (< 105 CFU/ml) or high (≥ 105 CFU/ml). Then the demographic, clinical and salivary factors (salivary glucose, saliva flow rate and its buffering capacity) suspected to contribute to the growth of cariogenic bacteria in the saliva were compared between high and low bacterial load group patients to determine the factors that might be associated with high bacterial load in the saliva of T2DM patients.

Data were processed using the Statistical Package for Social Sciences (SPSS) version 25.0. Descriptive statistics summarized the demographic and clinical profiles. Categorical variables were compared between high and low bacterial load groups using the Chi-square (χ^2) or Fisher's Exact probability test, while continuous variables were analyzed using the unpaired t-test. The level of significance was set at 5% and p-value < 0.05 was considered statistically significant.

RESULTS

The study cohort comprised 108 patients with T2DM, with a mean age of 45.1 ± 9.1 years (range: 31–65 years). Over 40% of the participants were aged ≤ 40 years, while 29.6% each fell into the 41–50 and >50 age brackets. Female participants slightly outnumbered males (55.6% vs. 44.4%) (Table I). Regarding their diabetic history, the majority (77.8%) had a disease duration of ≤ 10 years and the rest > 10 years. While raised fasting blood sugar

(FBS ≥ 7 mmol/L) was found in 89% of the patients, uncontrolled diabetes (HbA1c > 7%) was observed in > 70% of the cases. Xerostomia was reported in more than half (51.9%) of the patients (Table I).

Over 60% of the patients had low salivary pH (< 6.2) and raised salivary glucose (> 1 mg/dL). The level of salivary glucose ranged between 0.30 mg/dL and 4.9 mg/dL with a median value of 1.2 mg/dL. Forty (37%) patients had very low salivary flow, 28(26%) had moderate flow and 40(37%) had normal flow. Over half (51.9%) of the patients exhibited low salivary buffering capacity, 18.5% moderate buffering capacity and 29.6% high buffering capacity (Table II). Microbiological analysis identified *Streptococcus mutans* and *Lactobacilli* as the predominant cariogenic species. A significant majority of the patients (77.8%) exhibited a high bacterial load (≥105 CFU/ml), while 22.2% maintained a low load (<105 CFU/ml). (Table III).

Patients with high bacterial load in saliva were comparatively old than those with low bacterial load (p = 0.436). A male predominance was observed in patients with high bacterial load, although the difference was not statistically significant (p=0.214). While age and sex did not show statistically significant associations with microbial density (p=0.436 and p=0.214), several clinical and salivary factors were strongly correlated with high bacterial loads (Table 1). (Tab IV).

Longer duration of diabetes (> 10 years) was considerably higher (23.8%) in patients with high bacterial load than that in patients with low bacterial load (16.7%). Raised FBS was significantly higher (82.1%) in patients with high bacterial load than that in patients with low bacterial load (41.7%) with risk of having high bacterial load in patients with raised FBS was 6.4(95% CI of OR = 2.4 – 17.2) times greater than in patients with normal FBS (p < 0.001). Uncontrolled diabetes demonstrated their significant presence in the former cohort (70.2%) than that in the latter cohort (16.7%) with risk of developing high bacterial load in patients with uncontrolled diabetes is nearly 12-fold (95% CI of OR = 3.6 – 38.1) higher than in patients with controlled diabetes (p < 0.001). Two-thirds of the patients with high bacterial load complained of xerostomia compared to 25% of those with low bacterial load (p < 0.001). The risk of significant bacterial load in patients with xerostomia was 6.0(95% CI of OR = 2.1 – 16.8) times more than that in patients without xerostomia (p < 0.001) (Table V).

Furthermore, salivary dynamics played a critical role; low salivary pH more often tends to be associated with high bacterial load than that with low bacterial load (p < 0.001). Raised salivary glucose (1 mg/dL) was also significantly associated with high bacterial load (p < 0.001). Nearly half (47.6%) of the patients with high bacterial load had very low salivary flow rate compared to none with low bacterial load (p < 0.001). Majority (90.5%) of the patients with high bacterial load had low (66.7%) and moderate (23.8%) saliva buffering capacity as opposed to none with low bacterial load (p < 0.001) (Table VI).

Table I. Distribution of patients by demographic and clinical profile (n = 108)

Demographic & Clinical Profile	Frequency	Percentage
Demographic Characteristics		
Age* (years)		
≤ 40	44	40.8
41-50	32	29.6
> 50	32	29.6
Sex		
Male	48	44.4
Female	60	55.6
Profile of Diabetes		
Duration of diabetes (years)		
≤ 10	84	77.8
> 10	24	22.2
FBS (mmol/L)		
< 7 (Normal)	12	11.1
≥ 7 (Raised)	96	88.9
HbA1c (%)		
< 6.5 (Controlled diabetes)	32	29.6
≥ 6.5 (Uncontrolled diabetes)	76	70.4
Presence of xerostomia	56	51.9

***Mean age** = 45.1 ± 9.1 yrs; **range** = (31 – 65) yrs.

Table II. Distribution of patients by their salivary factor (n = 108)

Salivary Factors	Frequency	Percentage
Salivary pH		
< 6.2	68	63.0
≥ 6.2	40	37.0
Salivary glucose* (mg/dL)		
≤ 1	40	37.0
> 1	68	63.0
Saliva flow rate (g/min)		
Very Low (< 0.7g/min)	40	37.0
Moderate (< 0.7-1.0 g/min)	28	26.0
Normal (> 1.0 g/min)	40	37.0
Saliva buffering capacity		
Low	56	51.9
Moderate	20	18.5
High	32	29.6

***Median salivary glucose** = 1.2(range: 0.3 - 4.9) mg/dL.

Table III. Distribution of patients by cariogenic bacterial load in saliva (n=108)

Cariogenic bacterial load in saliva	Frequency	Percentage
***Streptococcus mutans* (CFU/ml)**		
<105 CFU/ml	24	22.2
≥105 CFU/ml	84	77.8
***Lactobacilli* (CFU/ml)**		
<105 CFU/ml	24	22.2
≥105 CFU/ml	84	77.8

Table IV: Comparison of demographic characteristics by bacterial load (n=108)

Demographic characteristics	Bacterial load		p-value
	High (n = 24)	Low (n = 84)	
Age (yrs)			
≤ 40	32(38.1)	12(50.0)	
41 – 50	24(28.6)	8(33.3)	
> 50	28(33.3)	4(16.7)	
Mean ± SD#	45.5 ± 9.7	43.8 ± 6.3	0.436
Sex*			
Male	40(47.6)	8(33.3)	0.214
Female	44(52.4)	16(66.7)	

Figures in the parentheses indicate corresponding %; ***Chi-squared Test** (χ^2) was done to analyze the data. #Data were analyze using **Unpaired t-Test** and were presented as **mean ± SD.**

Table V: Association of bacterial load with different parameters of diabetes

Profile of diabetes*	Bacterial load		OR (95% CI of OR)	p-value
	High (n = 84)	Low (n = 24)		
Duration of diabetes (yrs)				
> 10	20(23.8)	4(16.7)	1.5(0.5 – 5.1)	0.458
≤ 10	64(76.2)	20(83.3)		
FBS (mmol/L)				
≥ 7 (Raised)	69(82.1)	10(41.7)	6.4(2.4 – 17.2)	< 0.001
< 7 (Normal)	15(17.9)	14(58.3)		
HbA1c (%)				
≥ 6.5 (Uncontrolled)	59(70.2)	4(16.7)	11.8(3.6 – 38.1)	< 0.001
< 6.5 (Controlled)	25(29.8)	20(83.3)		
Presence of xerostomia	56(66.7)	6(25.0)	6.0(2.1 – 16.8)	< 0.001

***Chi-squared Test** (χ^2) was done to analyze the data; figures in the parentheses indicate corresponding %;

Table VI: Association between bacterial load and different parameters of diabetes

Salivary factors*	Bacterial load		p-value
	High (n = 24)	Low (n = 84)	
Salivary pH			
< 6.2	67(79.8)	8(33.3)	< 0.001
≥ 6.2	17(20.2)	16(66.7)	
Salivary glucose (mg/dL)			
> 1	64(76.2)	4(16.7)	< 0.001
≤ 1	20(23.8)	20(83.3)	
Saliva flow rate (g/min)			
Very Low	40(47.6)	0(0.0)	< 0.001
Moderate	24(28.6)	4(16.7)	
Normal	20(23.8)	20(83.3)	
Saliva buffering capacity			
Low	56(66.7)	0(0.0)	< 0.001
Moderate	20(23.8)	0(0.0)	
High	8(9.5)	24(100.0)	

***Chi-squared Test** (χ^2) was done to analyze the data; figures in the parentheses indicate corresponding %;

DISCUSSION

In the present study, 30% of the T2DM patients were over 50 years of age, with a mean age of 45.1 ± 9.1 years. A significant majority of these patients (70%) exhibited uncontrolled diabetes, and over half

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suffered from xerostomia accompanied by impaired saliva buffering capacity. The primary bacteria isolated were *Streptococcus mutans* and *Lactobacilli*. Notably, over three-quarters (77.8%) of the participants presented with a high bacterial load (≥ 105 CFU/ml). Xerostomia was significantly more prevalent in the high bacterial load group (66.7%) compared to the low bacterial load group (25%). Specifically, patients with xerostomia faced a 6.0-fold (95% CI = 2.1–16.8) higher risk of significant bacterial colonization ($p < 0.001$). These findings align with Khovidhunkit et al.[11] who reported a higher prevalence of xerostomia (62% vs. 36%) and hyposalivation (46% vs. 28%) in T2DM patients compared to non-diabetic controls. Similarly, Sandberg and associates[12] observed that T2DM patients experience xerostomia to a significantly greater degree than non-diabetics.

Reduced salivary function was a hallmark of this cohort, as 37% of patients exhibited a very low salivary flow rate (< 0.7 g/min). Research by Bernardi et al.[13] suggests that salivary flow is diminished in T2DM patients regardless of metabolic control. Furthermore, Puttaswamy and colleagues[14] noted both low flow rates and reduced buffering capacity in T2DM subjects. Our study also identified a strong association between low salivary pH and high bacterial load ($p < 0.001$). While Elkafri and peers[15] observed significantly lower salivary pH in patients with FBG > 200 mg/dL, Wang et al.[16] conversely found higher salivary pH in diabetic groups, though buffering capacity remained similar to controls.

Regarding bacterial composition, Almusawi[17] found that while 78% of T2DM patients had high *Streptococcus mutans* counts, only 42% had high *Lactobacilli* counts. In contrast, our study found both species to be equally dominant. Uncontrolled diabetes was strikingly more common in the high bacterial load group (70.2%) than in the low load group (16.7%). Patients with uncontrolled diabetes (HbA1c ≥ 6.5%) had an 11.7-fold (95% CI=3.6–38.1) higher risk of high bacterial colonization ($p < 0.001$). This mirrors findings by Al-Obaidaa et al.[18] regarding bacterial prevalence in subgingival pockets. While some literature on the diabetes-caries link remains contradictory-with Lai et al.[19] finding significant differences in cariogenic bacteria and Hintao et al.[6,7] finding none-our data supports a strong correlation. Hintao et al.[6,7] specifically linked *Streptococcus mutans* and *Lactobacilli* to root caries, while coronal caries was associated primarily with *Lactobacilli*. Additionally, Kampoo et al.[20] observed that T2DM patients in Thailand with active caries had significantly higher *Lactobacillus* counts. Goodson et al.[21] hypothesized that hyperglycemia-induced salivary glucose leads to oral acidification, perturbing the microbiome and increasing the risk of caries and gingivitis.

Our analysis revealed significant correlations between *Streptococcus mutans* counts and salivary factors, including flow rate, buffering capacity, and glucose levels. Interestingly, demographics (age/sex) and diabetes duration were not associated with high *Lactobacilli* counts. Bernardi et al.[13] noted that salivary glucose remains elevated in diabetics regardless of glycemic control, and Puttaswamy et al.[14] identified salivary glucose as a key influencer of periodontal status. The reduction in buffering capacity caused by hyposalivation creates an environment conducive to the proliferation of *Streptococci mutans* and *Lactobacillus*.[11] By categorizing glycemic control, the study established a clear link between HbA1c levels and bacterial load. Consistent with this finding, numerous studies demonstrated that poor glycemic status significantly enhances the risk of dental caries by promoting elevated counts of these cariogenic pathogens.

CONCLUSION

In Conclusion, salivary factors including flow rate, buffering capacity, and glucose levels-are pivotal in the multiplication of cariogenic bacteria. Hyposalivation and poor glycemic control create a synergistic effect that promotes bacterial growth in T2DM patients. Clinical xerostomia, low salivary pH,

and elevated salivary glucose are also strongly associated with increased bacterial colonization. Furthermore, a very low salivary flow rate and diminished buffering capacity create a favorable environment for bacterial growth. Given the high prevalence of uncontrolled diabetes in our population, these findings underscore the need for integrated strategies that combine routine glycemic monitoring with professional oral health care to mitigate the risk of dental caries.

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