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Glycemic Control and Dental Caries: Current Evidence and Biological Perspectives

Nuzulul Hikmah[1], Hafiedz Maulana[1]

[1]Department of Dentistry-Biomedical Sciences, Oral and Maxillofacial Pathology, Faculty of Dentistry, Jember University, Jember, Indonesia.

Corresponding Author: Nuzulul Hikmah

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ABSTRACT

Background: Diabetes mellitus is associated with several oral complications, including dental caries. Glycated hemoglobin (HbA1c) is a widely used marker of long-term glycemic control and may reflect the relationship between metabolic status and oral health. However, evidence linking glycemic control to dental caries remains unclear.

Objective: This review aimed to synthesize current evidence on the relationship between glycemic control and dental caries and to identify potential biological and clinical factors underlying variations in this relationship across populations.

Methods: PubMed and other relevant electronic databases were searched for studies published from January 2015 to December 2025. Search terms included combinations of "HbA1c," "glycemic control," "metabolic control," "diabetes," and "dental caries." Relevant studies examining glycemic status and caries-related outcomes were selected and descriptively synthesized as a narrative review.

Results: The reviewed evidence indicates a heterogeneous relationship between glycemic control and dental caries. Several studies reported that higher HbA1c levels were associated with greater caries experience or severity based on DMFT, DMFS, decayed teeth, ICDAS, caries activity, or root surface caries. Conversely, other studies reported nonsignificant or inconsistent associations. Differences in age, diabetes type and duration, oral hygiene, dietary habits, salivary characteristics, cariogenic microbial profiles, and caries assessment methods may contribute to these variations.

Conclusion: Poor glycemic control may be associated with greater dental caries experience, although the strength and consistency of this association vary across populations. HbA1c should be interpreted as one of several metabolic and biological factors that may contribute to caries susceptibility rather than as an independent determinant.

Keywords: Diabetes mellitus, Glycated hemoglobin A, Dental caries

INTRODUCTION

Diabetes mellitus is a complex metabolic disease characterized by elevated blood glucose levels resulting from insufficient insulin production, impaired insulin action, or both. Persistent hyperglycemia may lead to progressive damage to multiple organ systems and is associated with a wide range of systemic complications [1]. In addition to systemic complications, diabetes may influence oral health through metabolic and physiological alterations that affect the oral environment.

Glycated hemoglobin (HbA1c) is widely used to assess long-term glycemic status [2]. HbA1c is routinely used to evaluate glycemic control and treatment response and has been extensively applied in studies investigating the relationship between diabetes and oral health [3]. Consequently, HbA1c provides a clinically relevant measure for exploring whether differences in metabolic control are associated with variations in dental caries experience.

Dental caries is a multifactorial, biofilm-mediated disease characterized by progressive demineralization of dental hard tissues [4]. The potential relationship between glycemic control and dental caries has been investigated in both children and adults with type 1 and type 2 diabetes. Several studies have reported that poorer glycemic control or higher HbA1c concentrations are associated with greater caries experience or severity [5-9]. However, other studies have found no statistically significant association between HbA1c or metabolic control and caries-related outcomes [3,10-13]. These findings indicate that the relationship between glycemic control and dental caries remains unclear.

Several mechanisms may contribute to the potential association between glycemic control and dental caries. Poor metabolic control may be associated with alterations in salivary flow and composition, as well as changes in the oral microbial environment, which may increase susceptibility to dental caries [14-15]. However, the underlying mechanisms remain heterogeneous and are not yet fully understood. Therefore, this review aimed to synthesize current evidence on the relationship between glycemic control and dental caries and to explore potential factors underlying variations in this relationship across populations.

MATERIALS & METHODS

A literature search was conducted using PubMed and other relevant electronic databases to identify studies published between January 2015 and December 2025. The search strategy combined the following keywords: “HbA1c,” “glycemic control,” “metabolic control,” “diabetes,” and “dental caries.” Inclusion criteria were: 1) original research articles published between 2015 and 2025; 2) studies involving individuals with type 1 or type 2 diabetes mellitus; 3) studies evaluating glycemic status using HbA1c or other glycemic parameters; 4) studies reporting dental caries outcomes, including DMFT, DMFS, ICDAS, caries activity, or root surface caries; and 5) experimental studies investigating biological mechanisms related to caries susceptibility, including salivary alterations, enamel mineralization, and oral microbiota. Exclusion criteria included: 1) duplicate publications; 2) studies unrelated to diabetes, glycemic control, or dental caries; and 3) articles without accessible full text or sufficient relevant information. The findings were descriptively synthesized using a narrative approach, focusing on clinical evidence linking glycemic control to dental caries and the potential biological mechanisms underlying this relationship.

RESULT

PubMed and other relevant electronic databases were searched for studies published from January 2015 to December 2025 using predefined keywords. The eligible studies were evaluated descriptively and integrated into a narrative synthesis. Among the included studies, 21 indicated an association between glycemic control and dental caries (Table 1), while seven reported a statistically nonsignificant relationship between the two variables, as shown in Table 2. Table 1 suggest that poorer glycemic control may be associated with greater caries experience or severity. However, the outcomes assessed varied substantially among studies, including decayed missing filled teeth/decayed missing filled surfaces (DMFT/DMFS), decayed teeth, caries intensity, International Caries Detection and Assessment System Committee (ICDAS) based assessment, coronal caries, root surface caries, and

broader caries-risk measures. In contrast, several studies in Table 2 did not demonstrate a statistically significant relationship between glycemic control and dental caries. An oral hygiene, dietary habits, salivary characteristics, and microbial factors have been identified as potential determinants that may modify the relationship between glycemic control and caries.

Table 1. Principal findings of studies supporting an association between glycemic control and dental caries

Reference	Study population	Caries-related outcome	HbA1c / glycemic control	Principal finding
Carneiro et al., 2015 [5]	87 children and adolescents with T1DM	DMFT	≤8; 8–10; >10%	Caries experience increased across the higher HbA1c categories, with the highest mean DMFT reported in the group with HbA1c >10%.
Lai et al., 2017 [6]	68 diabetic and 136 non-diabetic children	Caries prevalence	≤7.5%; >7.5%	Caries prevalence was higher among children with poorer glycemic control.
Yonekura et al., 2017 [16]	108 patients with T2DM	DT and DMF index	HbA1c	HbA1c was significantly associated with the number of decayed teeth, whereas the association with the DMF index was not statistically significant.
Almusawi et al., 2018 [7]	100 patients with T2DM	Caries risk assessment	<7%; 7–8%; >8%	The proportion of participants classified as having high or extreme caries risk was greater among those with HbA1c >8%, suggesting poorer glycemic control was associated with greater caries risk.
Coelho et al., 2018 [14]	60 T1DM patients and 60 non-diabetic individuals	ICDAS codes	Non-diabetic vs poorly controlled diabetic (>7.5%)	Poorly controlled diabetic participants showed higher frequencies of several ICDAS caries categories than non-diabetic participants, indicating greater caries experience/severity.
Díaz Rosas et al., 2018 [17]	60 pediatric patients with diabetes	DMFT	Mean HbA1c 9.01%	HbA1c showed a statistically significant correlation with DMFT, supporting an association between poorer metabolic control and caries experience.
Vidya et al., 2018 [18]	87 children with T1DM	DMFS	≤6.99% vs ≥7%	Uncontrolled diabetes was associated with significantly higher DMFS than controlled diabetes (1.471 vs 0.158; p=0.043).
Majbauddin et al., 2019 [8]	91 patients with T2DM	DT and DMFT	HbA1c ≤8 vs >8%	HbA1c was significantly associated with both the number of decayed teeth and DMFT in regression analyses, supporting a relationship between poorer glycemic control and caries experience.

Cont. Table 1. Principal findings of studies supporting an association between glycemic control and dental caries

Reference	Study population	Caries-related outcome	HbA1c / glycemic control	Principal finding
Pappa et al., 2019 [19]	150 children and adolescents	DMFT	Healthy; <7.5%; ≥7.5%	Mean DMFT was substantially higher in poorly controlled diabetes than in well-controlled diabetes and healthy controls.
Schmolinsky et al., 2019 [20]	3,731 participants from a population-based cohort	DMFS	No diabetes; ≤7%; >7%	DMFS was higher in participants with diabetes than in non-diabetic participants, although the difference between well- and poorly controlled diabetes was not pronounced.

Suzuki et al., 2019 [21]	1,897 patients with T2DM	Decayed teeth	<6.5%; 6.5–7.9%; ≥8%	The proportion with decayed teeth increased progressively with higher HbA1c categories, reaching the highest prevalence in those with HbA1c ≥8%.
Iscan et al., 2020 [3]	50 children with T1DM and 50 healthy controls	ICDAS II	Diabetes vs healthy	Children with diabetes had a greater proportion of moderate and advanced carious lesions than healthy controls.
Pachoński et al., 2020 [22]	50 children with T1DM	DMFT	Control; ≤7.5%; >7.5%	Mean DMFT was highest among poorly controlled participants (5.80), compared with well-controlled participants (3.44) and controls (3.88).
Rahiotis et al., 2021 [23]	23 adults with T2DM and 18 controls	DMFT	<7%; >7%	DMFT increased from controls to well-controlled and poorly controlled diabetes groups, with the highest value observed in poorly controlled diabetes.
Ferizi et al., 2022 [15]	80 children with T1DM, aged 10–15 years	DMFT; plaque; gingival index; cariogenic bacteria	<7.5% vs >7.5%	Poorly controlled diabetes was associated with significantly higher DMFT, plaque and gingival indices, and greater *S. mutans* and *Lactobacillus*counts (all p<0.001); salivary flow did not differ significantly.
Mohan et al., 2022 [9]	341 adults with T2DM	Decayed teeth / DMFT	7.0–7.9% vs ≥8%	Participants with HbA1c ≥8% had a significantly higher proportion of decayed teeth (48.2% vs 28.3%) and a 2.65-fold higher adjusted risk of decayed teeth after accounting for dietary and oral-hygiene factors.

Cont. Table 1. Principal findings of studies supporting an association between glycemic control and dental caries

Reference	**Study population**	**Caries-related outcome**	**HbA1c / glycemic control**	**Principal finding**
Varughese et al., 2022 [24]	150 patients with T2DM	DMFT; PUFA; coronal and root caries; RCI	≤8% vs >8%	HbA1c positively correlated with DMFT (r=0.458) and PUFA (r=0.522); poorly controlled diabetes was also associated with greater severity of coronal and root caries.
Alkahtani et al., 2024 [25]	30 T2DM and 30 non-diabetic participants	Salivary metabolites and caries-related biomarkers	HbA1c measured	T2DM was characterized by altered salivary metabolites, including increased lactate and formate, which are relevant to cariogenic processes.
Heydari et al., 2024 [26]	1,011 adults ≥60 years; 324 with diabetes	DMFT; decayed teeth	Controlled vs uncontrolled DM	Mean DMFT was almost identical between diabetic and non-diabetic participants, but among those with diabetes, decayed teeth were significantly associated with uncontrolled diabetes (adjusted OR 1.24; p=0.006).
Curto et al., 2025 [27]	260 children with T1DM, aged 6–12 years	DMF-T and decayed/filled teeth	<7% vs >7%	Poor metabolic control was associated with significantly higher numbers of decayed teeth and DMF-T (3.14 vs 2.46), supporting an association between poorer glycemic control and caries experience.

Note: HbA1c: glycated hemoglobin, DMFT: decayed missing filled teeth, T1DM: type 1 diabetes mellitus, T2DM: type 2 diabetes mellitus, DT: decayed teeth, CAMBRA: caries management by risk assessment, ICDAS: international caries detection and assessment system committee, DMFS: decayed missing filled surfaces, PUFA: pulpal involvement, ulceration, fistula and abscess, RCI: root caries index.

Table 2. Principal findings of studies showing limited or no significant association between glycemic control and dental caries

Reference	Study population	Caries-related outcome	HbA1c / glycemic control	Principal finding
Kogawa et al., 2016 [28]	32 controlled T2DM, 31 poorly controlled T2DM, and 37 non-diabetic subjects	DMFT components	<7%; ≥7%	No statistically significant difference in the assessed DMFT components was observed according to glycemic control.

Cont. Table 2. Principal findings of studies showing limited or no significant association between glycemic control and dental caries

Reference	Study population	Caries-related outcome	HbA1c / glycemic control	Principal finding
Kamran et al., 2019 [10]	100 children with T1DM and 100 healthy controls	Mean DMF score	6–7%; 7–8%; ≥8%	DMF scores were similar across HbA1c categories, indicating no clear gradient between glycemic control and caries experience in this population.
Gunasekaran et al., 2022 [29]	80 children/adolescents with T1DM	Caries experience	Mean HbA1c 7.7±0.9%	HbA1c was not associated with caries experience, despite 59% of participants having ≥1 DMF tooth. Toothbrushing and interdental flossing were associated with lower caries experience.
ElBshari et al., 2024 [11]	42 children/adolescents with poorly controlled diabetes and 57 controls	DMFT and DMFS	HbA1c ≥7.5%	No significant differences were found in DMFT or DMFS between poorly controlled diabetic participants and controls, suggesting that poor glycemic control alone may not be a dominant determinant of caries in this population.
Firouzi et al., 2024 [12]	20 patients with T2DM	DMFT	<7% vs >7%	Mean DMFT was higher in uncontrolled diabetes (13.55 vs 10.22), but the difference was not statistically significant (p=0.137); HbA1c was not significantly correlated with dental caries (p=0.20).
Alkahtani and Baysan, 2025 [30]	91 T2DM and 91 non-diabetic adults	ICDAS; root caries severity	HbA1c measured; controlled vs uncontrolled	T2DM participants had significantly higher overall ICDAS scores and more severe lesions than controls. However, differences in ICDAS severity between controlled and uncontrolled diabetes were not statistically significant; HbA1c showed only a weak correlation with salivary spinnbarkeit.
Akgun et al., 2025 [13]	30 children with T1DM and 30 healthy controls	DMFS; PUFA	≤7%; >7%	In the T1DM group, correlation analyses between HbA1c levels and oral health indices (DMFS/dfs, PUFA/pufa) revealed no statistically significant (P > .05)

Note: HbA1c: glycated hemoglobin, DMFT: decayed missing filled teeth, T1DM: type 1 diabetes mellitus, T2DM: type 2 diabetes mellitus, ICDAS: international caries detection and assessment system committee, DMFS: decayed missing filled surfaces, PUFA: pulpal involvement, ulceration, fistula and abscess.

DISCUSSION

The present review indicates that glycemic control is potentially related to dental caries, but the strength and statistical significance of this relationship vary across populations and study settings. Several studies have reported greater caries experience, severity, or caries-related risk among individuals with poorer glycemic control or higher HbA1c concentrations, whereas other studies have not identified a significant association.

The association observed in several studies may be explained by the effects of chronic hyperglycemia on the oral environment. Higher HbA1c concentrations represent greater cumulative exposure to elevated blood glucose over several months [31] and may therefore reflect a metabolic state that can influence oral physiological conditions. Studies included in this review reported reductions in salivary flow among individuals with poorer metabolic control (Table 1). Reduced salivary flow may diminish oral clearance and buffering capacity, potentially favoring the retention of fermentable substrates and acids and thereby increasing susceptibility to caries [32]. Coelho et al. (2018) also reported lower unstimulated salivary flow among individuals with type 1 diabetes together with higher bacterial loads in saliva and dental biofilm [14].

Alterations in saliva may extend beyond salivary flow. Saliva provides calcium, phosphate, fluoride, and other components that contribute to the maintenance of tooth mineral equilibrium [33]. Previous studies have reported alterations in salivary characteristics among individuals with diabetes mellitus, including reduced salivary flow (hyposalivation) and changes in salivary pH and urea concentration [34]. Consequently, metabolic alterations associated with diabetes may potentially influence the oral environment through changes in salivary composition. Previous studies have indicated that type 1 diabetes mellitus may influence salivary composition, while hyperglycemia can contribute to gingival inflammation and compromise periodontal health. These alterations may also disrupt tooth mineralization, potentially increasing susceptibility to dental caries in children [35].

The oral microbiota may represent another pathway linking metabolic status with caries susceptibility. Dental caries is driven by microbial biofilm activity and the production of acids from fermentable carbohydrates [36]. In diabetic populations, increased bacterial loads have been reported in association with poorer metabolic control and greater caries susceptibility [14-15]. However, microbial changes should not be interpreted as a direct consequence of HbA1c alone. Dietary exposure, oral hygiene, salivary flow, medication use, and other host factors can alter the oral microbial environment [37-38]. Therefore, the observed relationship between HbA1c and caries may reflect interactions among systemic metabolic status, saliva, microbiota, and behavioral factors.

Some studies did not identify statistically significant associations between glycemic control or HbA1c and caries-related outcomes (Table 2). These findings indicate that elevated HbA1c alone may not be sufficient to explain differences in caries experience. In particular, oral hygiene may exert a substantial influence. Previous studies have indicated that preventive oral hygiene practices, particularly regular toothbrushing and dental floss use, may influence the association between systemic metabolic status and dental caries [39-40].

Dietary factors may similarly influence this relationship. Dental caries is influenced by the frequency and amount of fermentable carbohydrate exposure [41], and dietary practices may differ substantially among individuals with similar HbA1c levels [42]. Previous studies have demonstrated alterations in the composition and functional profiles of the supragingival microbiome associated with the transfer of circulating glucose and fructose from the bloodstream into the oral cavity via saliva. These

findings suggest that the migration of circulating saccharides into saliva may contribute to changes in the oral microbial environment and potentially play a role in the pathogenesis of dental caries in patients with type 2 diabetes mellitus [42]. Thus, two individuals with comparable HbA1c values may have different caries experiences because of differences in sugar consumption, oral hygiene, and salivary composition.

Another factor to consider is the variability in caries assessment methods, study populations, and research designs, particularly the predominance of cross-sectional studies. The studies included in this review employed diverse caries-related outcomes, such as DMFT, DMFS, the number of decayed teeth, caries intensity, ICDAS, root surface caries, and broader caries-risk indicators. Furthermore, the study populations varied, encompassing children and adolescents with type 1 diabetes, adults with type 2 diabetes, and older individuals. These populations also differed in diabetes duration, glycemic status, oral hygiene practices, dietary habits, and access to dental care. Such methodological and population-related heterogeneity may contribute to the inconsistent findings across studies. Therefore, longitudinal studies incorporating repeated assessments of glycemic control and standardized caries evaluation methods are needed to better elucidate the temporal relationship between metabolic control and dental caries development.

From a biological perspective, the available evidence supports a multifactorial model in which glycemic control may influence caries susceptibility. Wang et al. (2025) described several potential pathways through which hyperglycemia may increase the risk of dental caries. First, elevated blood glucose levels may impair salivary gland function, resulting in reduced salivary flow and diminished buffering capacity. Second, hyperglycemia may interfere with enamel mineralization, potentially compromising enamel structure and increasing its vulnerability to carious lesions. Third, children with diabetes may have greater exposure to carbohydrate-rich foods and sugary beverages, thereby providing additional fermentable substrates for cariogenic microorganisms. In addition, diabetes-related alterations in immune function and periodontal tissue repair may contribute to gingival inflammation and changes in microbial pathogenicity [34]. However, these mechanisms require further investigation before specific metabolic pathways can be considered causal.

CONCLUSION

Current evidence suggests that poor glycemic control is frequently associated with greater dental caries experience in individuals with diabetes. However, variations in age, diabetes type and duration, oral hygiene practices, dietary habits, salivary flow and composition, cariogenic microbial profiles, and methods used to assess glycemic control and dental caries may contribute to the heterogeneity of findings across studies. From a biological perspective, hyperglycemia may influence the oral environment through alterations in salivary gland function, enamel mineralization, the availability of fermentable substrates for cariogenic microorganisms, and immune responses. Nevertheless, the available evidence does not establish HbA1c as an independent determinant of dental caries. Further longitudinal and mechanistic studies using standardized caries assessment methods and comprehensive adjustment for potential confounding factors are warranted to clarify the relationship between glycemic control and dental caries.

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