



Structural Changes in Primary Teeth of Diabetic Children: Composition and Ultrastructure: A Systematic Review

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Abstract Background: Type 1 diabetes mellitus (T1DM) in children is associated with distinct oral health complications, including alterations in primary tooth structure and composition. These changes may affect enamel and dentin integrity, caries risk and periodontal status. This systematic review aimed to evaluate the structural, compositional and ultrastructural changes in primary teeth of diabetic children. **Methods:** Following PRISMA guidelines, PubMed/MEDLINE, Science Direct, Google Scholar and Cochrane Library were searched for studies up to July 2024. Observational, case-control, cohort and cross-sectional studies assessing primary teeth of T1DM children with clinical, chemical or imaging techniques (SEM, EDX) were included. Quality was assessed using the Newcastle-Ottawa Scale. **Results:** Ten studies met the inclusion criteria. SEM/EDX analyses showed significant enamel abnormalities in diabetic children, including prism defects and an irregular aprismatic layer. Calcium and phosphorus content were consistently lower (Ca: 33.8% vs. 37.6% in controls, $p = 0.017$; P: 16.8% vs. 18.9%, $p < 0.001$). Caries outcomes varied: some studies reported higher DMFT scores in diabetics (e.g., 1.43 vs. 0.56, $p < 0.05$), while others found no difference or lower scores. Periodontal indices were consistently worse in diabetics, with significantly higher gingival indices ($p < 0.01$) and plaque accumulation. Altered tooth eruption patterns (earlier by ~1.6 years) and reduced salivary buffering capacity were also observed. **Conclusions:** Children with T1DM exhibit clear alterations in the structure and composition of primary teeth, including enamel hypoplasia, reduced mineralization and higher susceptibility to periodontal disease. Variability in caries outcomes suggests an influence of metabolic control, oral hygiene and dietary factors. Early preventive dental care and interdisciplinary management are recommended.

Key Words Type 1 Diabetes Mellitus, Primary Teeth, Enamel Hypoplasia, Ultrastructure, Pediatric Oral Health, Dental Caries, Scanning Electron Microscopy (SEM)

INTRODUCTION

Diabetes, particularly in children, induces significant structural and compositional changes in primary teeth, which can adversely affect dental health. It can directly impact oral health, quality of life and psychological well-being. Diabetes mellitus, particularly Type 1 diabetes (T1DM), is associated with several oral health complications, including a higher

prevalence of dental caries, periodontal disease and variations in dental morphology, all resulting from hyperglycemia and its systemic effects.

One of the most prominent and well-documented issues is the increased incidence of dental caries among children with T1DM. Studies have consistently demonstrated that diabetic children exhibit higher rates of caries compared to

their non-diabetic peers. The underlying causes include factors such as reduced salivary flow, altered salivary composition and the presence of elevated glucose levels in saliva, which facilitate the growth of cariogenic bacteria like *Streptococcus mutans* [1]. The hyperglycemic environment hampers the repair mechanisms of dental tissues, making these children more susceptible to dental decay [2]. Additionally, nutritional factors and dietary patterns, which are often skewed in diabetic children, can exacerbate caries risk [3].

The structural integrity of primary teeth is likewise compromised in diabetic children. Research indicates that the morphology of enamel and dentin undergoes significant changes due to the metabolic disturbances associated with diabetes. For instance, diabetic children frequently exhibit enamel hypoplasia—a condition characterized by the underdevelopment of enamel that leads to thinner enamel and increased susceptibility to caries [4,5]. Furthermore, studies suggest that diabetes may influence the chronological eruption pattern of teeth, accelerating the eruption of primary teeth in younger age groups while potentially delaying it in older children, reflecting disruptions in normal dental development processes [6]. T1DM is also linked with periodontal complications from an early age [4]. Overall, given the dual threat posed by diabetes-related systemic conditions and the resulting structural changes in dental tissues, it is critical that healthcare providers adopt a comprehensive approach to managing the oral health of diabetic children. The objective of this systematic review was to evaluate the structural, compositional and ultrastructural changes in the primary teeth of children with T1DM.

METHODS

Protocol and Registration

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines.

Eligibility Criteria

Studies were considered eligible if they investigated children diagnosed with diabetes mellitus (either type I or type II) who have primary dentition, with a focus on identifying structural changes, compositional differences and ultrastructural alterations in primary teeth. The outcomes were assessed using imaging techniques, such as scanning electron microscopy, along with various chemical analysis methods. Eligible study designs included observational studies, case-control studies, cohort studies and cross-sectional studies. Only publications in English and studies published up to July 2024 were included. Studies that were reviews, case reports, editorials or animal studies were excluded from this review.

Information Sources and Search Strategy

A comprehensive search was performed using electronic databases including PubMed/MEDLINE, Science Direct,

Google Scholar and Cochrane Library. The search strategy combined Medical Subject Headings (MeSH) and free-text terms: ("Diabetes Mellitus"[Mesh] OR "diabetes") AND ("Primary Teeth" OR "Deciduous Teeth") AND ("structural" OR "composition" OR "ultrastructure" OR "enamel" OR "dentin"). Furthermore, the reference lists of eligible studies and relevant review articles were manually screened to identify additional pertinent articles.

Study Selection

All retrieved records were imported into a reference management software where duplicates were removed. Two independent reviewers initially screened titles and abstracts to identify studies that potentially met the eligibility criteria. Following this, full-text articles were obtained and assessed for inclusion by the same reviewers. Any discrepancies in study selection were resolved through consensus discussion among reviewers. The PRISMA flowchart (Figure 1) documents the selection process, showing initial identification of 844 records, with 10 studies meeting final inclusion criteria after exclusions.

Data Extraction

A standardized data extraction form was developed and piloted to ensure consistency in capturing relevant information. Data were independently extracted by two reviewers and included details regarding study characteristics (such as authors, year of publication, country and study design), participant demographics (including age, type of diabetes and duration of disease) and methodological aspects (for example, sample preparation, imaging modalities and analytical techniques). In addition, key findings related to structural, compositional and ultrastructural changes in primary teeth were recorded, along with reported limitations and potential sources of bias.

Quality Assessment

The methodological quality and risk of bias in the included studies were assessed using an appropriate quality appraisal tool, such as the Newcastle-Ottawa Scale for observational studies. Each study was evaluated independently by two reviewers. Discrepancies in the quality assessment were resolved through discussion, ensuring that the overall quality of evidence was thoroughly synthesized to inform the strength of the conclusions drawn from the review.

Data Synthesis and Analysis

Due to the heterogeneity in study designs, methodologies and reported outcomes, a narrative synthesis was deemed the most appropriate approach for data analysis. The extracted data were organized into tables and summarized in the text, highlighting common findings as well as inconsistencies among the studies.

RESULTS

This systematic review included 10 studies investigating structural, compositional and ultrastructural changes in

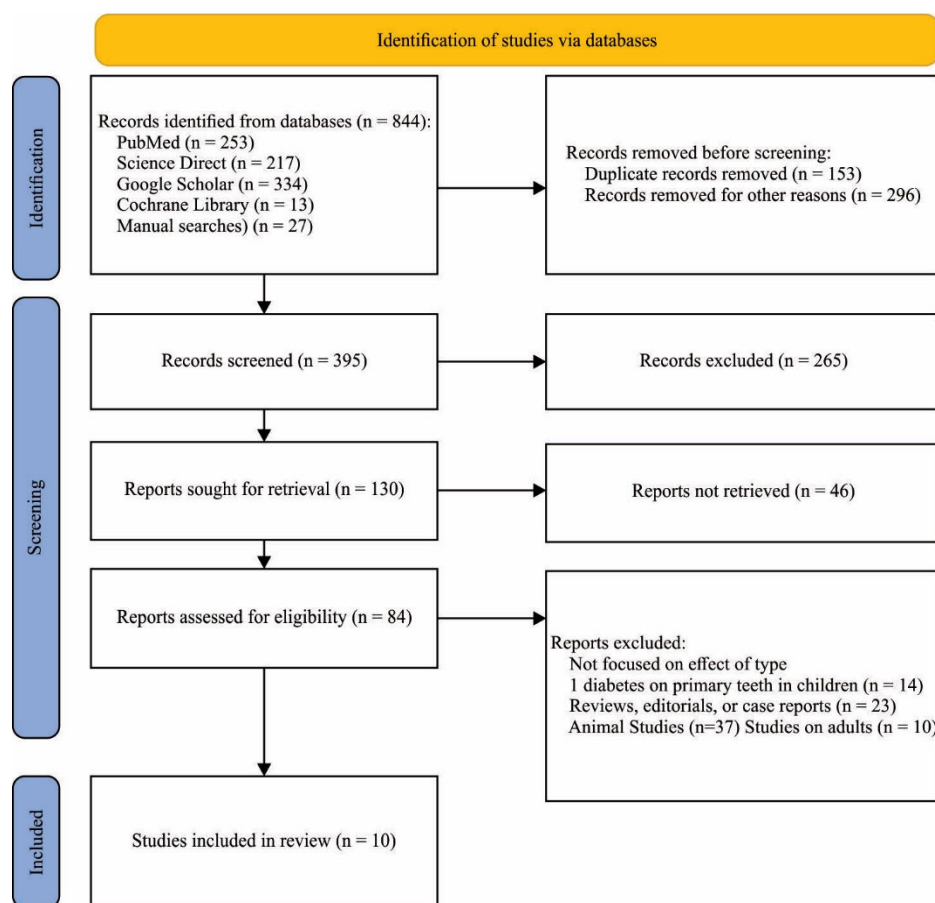


Figure 1: PRISMA Flowchart Showing the Study Selection Process

Table 1: Newcastle-Ottawa Quality Assessment of Included Studies

Study (Author, Year)	Selection (0-4)	Comparability (0-2)	Outcome/Exposure (0-3)	Total Score (0-9)	Quality Rating
Kulkarni <i>et al.</i> [10]	4	1	2	7	Good
Orbak <i>et al.</i> [11]	3	1	2	6	Fair
Rafatjou <i>et al.</i> [12]	4	2	3	9	Good
Syed <i>et al.</i> [13]	4	2	3	9	Good
Sadeghi <i>et al.</i> [14]	3	1	2	6	Fair
Sarmamy <i>et al.</i> [9]	3	1	2	6	Fair
Assiri <i>et al.</i> [15]	4	2	3	9	Good
López del Valle [16]	3	1	2	6	Fair
Ismail <i>et al.</i> [17]	4	2	3	9	Good
Ferizi <i>et al.</i> [18]	4	2	3	9	Good

primary teeth of children with diabetes mellitus. The studies encompassed diverse geographical regions including Saudi Arabia, Turkey, Iran, Iraq, India, Kosovo, Puerto Rico and Hong Kong, employing various methodologies such as scanning electron microscopy (SEM), energy-dispersive X-ray spectroscopy (EDX), clinical examinations and salivary analyses. The quality assessment using the Newcastle-Ottawa Scale indicated that most included studies were of good quality (scores 7-9), with a few fair-quality studies (scores 6). This suggests that while methodological variability exists, the overall evidence base was acceptable for synthesis. The results are shown in Table 1.

The study by Syed *et al.* [10] conducted SEM and EDX analyses on 50 primary teeth from diabetic children and 50 from healthy controls. Results revealed significant

ultrastructural abnormalities in diabetic enamel, including loosely packed prisms and an uneven aprismatic layer. EDX showed lower calcium (33.75% vs. control, $p = 0.017$) and phosphorus (16.76% vs. control, $p = 0.0001$) content in diabetic enamel. Orbak *et al.* [8] similarly observed accelerated dental development in diabetic children before age 10, followed by delayed eruption patterns, along with significantly higher gingival ($p < 0.001$) and calculus indices ($p < 0.001$) compared to controls (Table 2).

Regarding caries prevalence, findings were inconsistent. Rafatjou *et al.* [9] found no significant difference in DMFT scores ($p = 0.158$) but higher dmft in controls ($p = 0.008$), while Ferizi *et al.* [16] reported that children with poor metabolic control ($HbA1c > 7.5\%$) had significantly higher DMFT scores and plaque accumulation. Conversely,

Table 2: Summary of Included Studies

Study	Country	Study Design	Sample (Diabetic vs. Control)	Methods	Key Findings
Kulkarni <i>et al.</i> [10]	India	Cross-sectional	80 T1DM vs. 80 healthy	CPQ questionnaire, DMFT, PI, GI	Higher gingival inflammation (GI) in T1DM ($p = 0.014$), - Lower DMFT in T1DM ($p \leq 0.001$), Poorer OHRQoL in T1DM children ($p \leq 0.05$)
Orbak <i>et al.</i> [11]	Turkey	Case-control	50 T1DM vs. 50 healthy	DMFT, PI, GI, CI, SEM (eruption)	Accelerated dental development in T1DM before age 10, Higher GI ($p < 0.001$) and CI ($p < 0.001$) in T1DM, More caries in controls ($p < 0.05$)
Rafatjou <i>et al.</i> [12]	Iran	Case-control	80 T1DM vs. 80 healthy	DMFT, dmft, PI, GI	Higher GI in T1DM ($p = 0.001$), No difference in DMFT ($p = 0.158$) or PI ($p = 0.373$), Higher dmft in controls ($p = 0.008$)
Syed <i>et al.</i> [13]	Saudi Arabia	Observational (SEM/EDX)	50 T1DM vs. 50 healthy	SEM, EDX (Ca/P mass percentage)	Ultrastructural enamel defects (loose prisms, perforations) in T1DM, Lower Ca ($p = 0.017$) and P ($p = 0.0001$) in T1DM enamel, Abnormal aprismatic layer in T1DM
Sadeghi <i>et al.</i> [14]	Iran	Cross-sectional	50 T1DM vs. 50 healthy	DMFT, PI, GI, PPD, CI	Higher PI ($p < 0.05$) and DMFT ($p < 0.05$) in T1DM, GI higher only in 13-18-year-olds ($p < 0.01$), No link between HbA1c and periodontal indices ($p < 0.09$)
Sarmamy <i>et al.</i> [15]	Iraq	Case-control	66 T1DM vs. 66 healthy	DMFT/dmft, DI, GI, CI	Earlier tooth eruption in T1DM (by 1.6 years), Lower dmft/DMFT in T1DM ($p < 0.05$), Higher GI and CI in T1DM ($p < 0.05$)
Assiri <i>et al.</i> [16]	Saudi Arabia	Case-control	40/40	DMFT/dmft, salivary pH, flow rate, buffering capacity, bacterial counts	Lower dmft in diabetic children; reduced buffering capacity; no difference in salivary flow/pH or bacterial counts
López del Valle [17]	Puerto Rico	Case-control	25/25	DMFT, plaque/gingival indices, bacterial counts	Higher DMFT, plaque and gingival bleeding in diabetic children; no difference in bacterial counts
Ismail <i>et al.</i> [18]	Hong Kong	Comparative	32/32	DMFT/dmft, plaque/gingival indices	Higher plaque in diabetic children; no difference in DMFT/dmft or gingival bleeding
Ferizi <i>et al.</i> [19]	Kosovo	Case-control	80 (34 good/ 46 poor HbA1c)	DMFT, plaque/gingival indices, salivary flow, bacterial counts	Poor metabolic control linked to higher DMFT, plaque, gingivitis and cariogenic bacteria

Assiri *et al.* [13] found lower dmft scores in diabetic children despite reduced salivary buffering capacity.

Periodontal health consistently showed deterioration in diabetic children across studies. Sadeghi *et al.* [11] reported higher plaque ($p < 0.05$) and DMFT ($p < 0.05$) indices in diabetic children aged 12-18 years, while Ismail *et al.* [15] noted significantly increased plaque accumulation without differences in caries or gingival bleeding. López del Valle [14] observed higher DMFT (1.43 vs. 0.56), plaque and gingival bleeding (23.9 vs. 4.2 sites) in diabetic children despite similar bacterial counts.

Salivary changes were noted in multiple studies. Assiri *et al.* [13] reported reduced buffering capacity in diabetic children, while Ferizi *et al.* [16] found higher cariogenic bacteria counts in poorly controlled diabetics. However, Kulkarni *et al.* [7] emphasized that maternal education and dental visit frequency significantly influenced oral health-related quality of life more than diabetes status alone.

DISCUSSION

This systematic review explored the structural, compositional and ultrastructural changes in the primary teeth of children with T1DM, drawing on evidence from ten studies conducted across various geographical regions. The findings collectively demonstrate that diabetes exerts a notable impact on the dental health of children, particularly affecting the integrity of enamel and dentin, caries prevalence, periodontal status and tooth eruption patterns.

One of the most consistent findings across multiple studies was the presence of enamel defects in diabetic children. Syed *et al.* [10] utilized SEM and EDX analyses to reveal significant ultrastructural abnormalities in diabetic enamel, including loosely packed prisms, surface perforations and an uneven aprismatic layer. Additionally, this study reported significantly lower calcium and phosphorus content in diabetic enamel compared to healthy controls. These findings align with earlier animal studies [17] and reinforce the hypothesis that metabolic disturbances in diabetes disrupt normal enamel formation and mineralization processes during tooth development.

The review also highlights considerable variation in caries prevalence among diabetic children. While studies like Rafatjou *et al.* [9] and Sarmamy *et al.* [12] reported lower or comparable DMFT and dmft scores in diabetic children relative to controls, other studies such as Ferizi *et al.* [16] and López del Valle [14] identified higher DMFT scores in diabetic children, particularly among those with poor metabolic control. Notably, Assiri *et al.* [13] found reduced dmft scores in diabetic children despite diminished salivary buffering capacity, suggesting that factors beyond diabetes status such as oral hygiene practices, dietary habits and maternal education may influence caries risk, as also emphasized by Kulkarni *et al.* [7]. Enamel hypoplasia, which refers to underdeveloped enamel, is associated with fluctuations in metabolic control during tooth development and is noted to have a higher incidence in children born to diabetic mothers. Evidence indicates that such structural deficiencies can lead to thinner enamel layers, making teeth

more susceptible to caries and other dental pathologies [17]. The role of dental enamel integrity, particularly in the context of diabetes, highlights the need for targeted monitoring and preventive care strategies to mitigate risks associated with compromised enamel formation. A systematic review outlined that primary teeth are more likely to experience carious lesions compared to permanent teeth due to developmental systemic issues associated with diabetes [18].

Periodontal health outcomes were more consistently reported across studies, with diabetic children exhibiting higher plaque indices, gingival inflammation and calculus formation compared to non-diabetic peers. Sadeghi *et al.* [11] and Orbak *et al.* [8] both found significantly higher gingival and plaque indices in diabetic children, with Orbak *et al.* also reporting increased calculus accumulation. Similarly, López del Valle [14] observed more extensive plaque and gingival bleeding sites in diabetic children. These findings suggest that the systemic inflammatory effects of diabetes contribute to early periodontal disease manifestations, independent of bacterial load, as several studies including López del Valle [14] and Assiri *et al.* [13] reported no significant differences in bacterial counts between diabetic and healthy children. The interrelationship between diabetes and periodontal health underlines the importance of interdisciplinary approaches in managing these children's health, combining medical and dental care strategies to improve overall outcomes.

Alterations in tooth eruption patterns were also identified. Orbak *et al.* [8] and Sarmamy *et al.* [12] noted accelerated dental development and earlier tooth eruption in diabetic children before age 10, with delayed eruption patterns in older diabetic children. This disruption in normal eruption sequences likely reflects the influence of systemic metabolic imbalances on dental and skeletal development in diabetic children.

Salivary alterations, while less consistently reported, were also a notable finding in this review. Assiri *et al.* [13] identified reduced salivary buffering capacity in diabetic children, while Ferizi *et al.* [16] reported higher levels of cariogenic bacteria in those with poor glycemic control. These changes, combined with altered enamel composition and periodontal susceptibility, likely exacerbate the risk of dental complications in diabetic children. The conflicting results regarding caries prevalence across studies likely reflect multifactorial influences. Differences in metabolic control are a key determinant, as poor glycemic regulation has been linked to higher DMFT scores [16], whereas well-controlled patients may show similar or even lower caries experience compared to healthy peers [9,13]. In addition, oral hygiene practices, dietary patterns and socioeconomic factors, such as maternal education and frequency of dental visits, strongly modulate caries risk [7]. These contextual variables may explain why some studies reported lower or comparable DMFT in diabetic children, while others identified significantly higher rates.

This review has several limitations. First, only a limited number of head-to-head studies were available and some had

relatively small sample sizes, which may reduce the strength of the evidence. Second, substantial heterogeneity existed across the included studies in terms of diagnostic methods-ranging from SEM and EDX to clinical indices-which limits direct comparability of findings. Third, most studies were cross-sectional and lacked long-term follow-up, making it difficult to assess the progression and persistence of dental changes over time. Finally, some studies included older children with mixed dentition, which may have introduced variability. Overall, the findings of this review underscore the significant impact of T1DM on the dental health of children, particularly affecting enamel quality, caries susceptibility, periodontal status and eruption patterns. The variability in caries outcomes across studies suggests that factors such as glycemic control, oral hygiene behaviors, dietary habits and healthcare access play a crucial role in modulating oral health risks in diabetic children. These results highlight the importance of integrating regular dental care and preventive interventions into the comprehensive management of pediatric diabetes to minimize the long-term impact of oral health complications. The clinical implications of these findings are important for pediatric dentistry and diabetes care. Early and regular dental screening should be integrated into the management of children with T1DM, with an emphasis on preventive strategies such as topical fluoride applications, calcium/phosphate-based remineralization agents and dietary counseling. Closer collaboration between pediatric dentists, endocrinologists and caregivers is essential to detect early enamel changes, manage periodontal inflammation and minimize long-term oral complications. Education of parents and children about oral hygiene practices tailored to diabetic patients may further reduce the burden of disease.

CONCLUSIONS

This systematic review demonstrates that children with T1DM show distinct structural and compositional alterations in primary teeth, including enamel hypoplasia, reduced calcium and phosphorus content and characteristic ultrastructural defects. These changes increase vulnerability to periodontal disease and may influence caries risk, which varies depending on glycemic control, oral hygiene and dietary factors. Early, proactive and interdisciplinary dental management-including preventive care and regular monitoring-should be integrated into diabetes care to preserve oral function and improve quality of life. Further longitudinal and mechanistic studies are warranted to clarify long-term outcomes and guide targeted preventive strategies.

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