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CONTEMPORARY STRATEGIES FOR DENTAL CARIES PREVENTION IN CHILDREN WITH DIABETIC POLYNEUROPATHY: AN EVIDENCE-INFORMED CLINICAL FRAMEWORK

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ABSTRACT

Background: Children and adolescents with diabetes may develop dental caries through interacting behavioral, salivary, microbiological and health-service factors. Diabetic peripheral polyneuropathy (DPN) represents a clinically important complication, but evidence linking pediatric DPN specifically to incident caries remains limited. Objective: To critically synthesize evidence concerning oral-health risks in pediatric diabetes and propose a coordinated, risk-adapted preventive framework for children with established DPN. Materials and Methods: A targeted narrative appraisal of pediatric diabetes guidelines, observational studies, systematic reviews and preventive dentistry recommendations available through November 2026 was undertaken. Published findings were distinguished from hypotheses concerning the additional contribution of DPN. Results: A 2025 systematic review included 16 pediatric type 1 diabetes studies (15 cross-sectional and one longitudinal) and indicated variability in cariogenic microbiota according to glycemic status. In a separate cohort of 80 children with type 1 diabetes, 59% had caries experience, 94% had gingivitis and 52% had reduced salivary buffering capacity. Neuropathy was not used to stratify these dental outcomes. A proposed multidisciplinary preventive pathway integrates lesion activity and caries risk, regular fluoride exposure, indicated sealants, individualized carbohydrate counseling, functional assistance where needed and scheduled reassessment. Conclusion: Established pediatric caries-prevention measures are applicable to children with diabetes and suspected or confirmed DPN; DPN-specific modifications should respond to documented functional limitations rather than an assumed direct neuropathic mechanism. Prospective comparative studies are necessary before efficacy claims can be made for the proposed framework [3–8,11–17].

Keywords: diabetic polyneuropathy; type 1 diabetes mellitus; children; dental caries; caries prevention; fluoride; salivary buffering; oral hygiene; multidisciplinary care; pediatric dentistry.

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INTRODUCTION

Dental caries remains a major preventable disease of childhood and arises from a dynamic imbalance between biofilm-mediated acid production and tooth remineralization. The World Health Organization has underscored the enormous global burden of oral disease, while contemporary caries biology recognizes the combined roles of fermentable carbohydrate exposure, oral biofilm, saliva, fluoride, individual susceptibility and socioeconomic conditions [1,2]. Because dental disease may compromise eating, sleep, school performance and well-being, oral prevention is an important component of comprehensive pediatric health care. In children living with diabetes mellitus, preventive planning must also take account of daily glucose monitoring, nutritional routines, the treatment of hypoglycemia and the demands of long-term self-management.

Diabetic peripheral polyneuropathy is a recognized complication of both type 1 and type 2 diabetes in young people. The SEARCH cohort documented age-adjusted peripheral neuropathy prevalences of 8.5% and 17.7% in youth-onset type 1 and type 2 diabetes, respectively, among participants evaluated in adolescence or young adulthood [5]. These estimates describe neurological complications, not dental outcomes. DPN must also be distinguished from autonomic neuropathy: impaired distal sensory function does not by itself demonstrate altered salivary secretion or oral sensory dysfunction. The term therefore identifies a subgroup potentially requiring coordinated health care but should not be treated as an established independent caries risk factor.

The aim of the present review is to clarify what published evidence supports about dental caries prevention in pediatric diabetes, identify the limits of transferring that evidence to children with DPN, and propose an original, clinically practicable prevention and research framework. The intended contribution is methodological integration rather than a new patient-level estimate of risk or intervention effectiveness.

LITERATURE REVIEW

The relationship between diabetes and dental caries is not uniform across studies. In a 2025 systematic review and meta-analysis, Ta and colleagues identified 16 eligible studies comparing the caries-associated microbiota of children with type 1 diabetes and their peers; 15 studies were cross-sectional and only one was longitudinal. Poorer glycemic control was associated with increases in selected cariogenic streptococcal and lactobacilli taxa, but evidence that these patterns produce consistently greater caries incidence was less secure [7]. Another meta-analysis published in 2024 reported differences in plaque indices but inconsistent differences across several caries-related endpoints [9]. Together, the findings caution against treating type 1 diabetes, glycated hemoglobin or the presence of neuropathy as sufficient standalone predictors of dental disease.

A focused pediatric study provides concrete information for clinical planning. Gunasekaran and colleagues examined 80 children and adolescents with type 1 diabetes; 47 (59%) had at least one decayed, missing or filled tooth, 75 (94%) had gingivitis and 42 (52%) had reduced salivary buffering capacity [6]. In that cohort, measured HbA1c did not predict

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caries experience or salivary characteristics, whereas more frequent brushing and interdental cleaning were associated with lower caries experience. These cross-sectional associations cannot establish that a change in hygiene behavior will produce a specific magnitude of future risk reduction; nevertheless, they reinforce the need to assess individual exposures and protective practices. A 2025 case-control study with 40 children with type 1 diabetes and 40 age- and sex-matched controls reported higher caries occurrence and plaque-related findings in the diabetic group, but its retrospective single-center design limits generalizability [8].

Evidence directly connecting neuropathy to oral disease is largely extrapolated from older populations. In the adult DCCT/EDIC cohort, diabetic peripheral neuropathy was associated with subsequent tooth-loss outcomes (adjusted odds ratio 1.88; 95% confidence interval 1.24–2.87) [10]. Tooth loss in adults, however, incorporates periodontal, restorative and lifetime risk pathways and must not be reinterpreted as evidence that neuropathy causes dental caries in children. Established pediatric diabetes guidelines address screening for neurological complications and medical follow-up, including autonomic manifestations, but do not provide a validated DPN-specific caries-prevention program [3,4]. A cautious response is therefore to modify oral-health support only when functional difficulties, altered routines or additional risk factors are directly observed.

Evidence-based pediatric dentistry supports clinical risk assessment, twice-daily age-appropriate fluoridated toothbrushing, indicated professional fluoride applications, fissure sealants for susceptible molars and nonrestorative treatment of suitable lesions [11–15]. Dietary counseling should address the frequency and form of fermentable carbohydrates while respecting the non-negotiable need to treat hypoglycemia promptly according to the diabetes care plan [16]. Recommendations for children with special health care needs also emphasize accessibility, assistance with home hygiene and communication between medical and dental professionals [17,18]. These established principles offer a defensible foundation for a DPN-adapted pathway, but their comparative effectiveness specifically in pediatric DPN remains to be tested.

MATERIALS AND METHODS

This article was designed as a narrative evidence review and original clinical-methodological synthesis, not as a prospective clinical investigation, retrospective patient cohort, registered systematic review or meta-analysis. Sources were identified through targeted searches of PubMed-indexed literature and official resources of the World Health Organization, the International Society for Pediatric and Adolescent Diabetes (ISPAD), the American Academy of Pediatric Dentistry (AAPD) and the American Dental Association (ADA). Search concepts included “diabetic peripheral neuropathy”, “children”, “type 1 diabetes”, “type 2 diabetes”, “dental caries”, “oral health”, “salivary flow”, “fluoride varnish”, “sealants”, “hypoglycemia” and “prevention”. Publications and guidance available through 14 November 2026 were considered; emphasis was placed on pediatric studies, authoritative recommendations and sources specifically addressing the neuropathy–oral-health relationship. Eighteen traceable references were selected for the final synthesis [1–18].

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Evidence was organized into four analytical domains: pediatric neuropathy and its screening context; diabetes-related dental and salivary outcomes; effectiveness and indications of established caries-prevention measures; and practical adaptations for children with confirmed DPN. Direct evidence was defined as studies with pediatric participants and dental outcomes; indirect evidence comprised adult neuropathy studies or general pediatric preventive dentistry guidance. Published proportions were reproduced with their original denominators and outcome labels. No pooled risk ratios, comparative effectiveness estimates, new statistical tests or synthetic patient outcomes were created. Figures 1 and 2 display estimates from different published cohorts without implying a causal or mathematical relationship between them. Tables and the proposed pathway are decision-support syntheses, not validated clinical scoring instruments.

The proposed model was constructed as an iterative clinical process beginning with a combined medical and dental history, followed by individual risk assessment, tailored preventive action, longitudinal lesion monitoring and interprofessional review. Suggested indicators for future evaluation were stated prospectively rather than presented as observed results. Because no participants were recruited and no identifiable patient-level data were obtained, preparation of this literature-based review did not require human-subject consent or research ethics approval. Any subsequent pediatric cohort or intervention trial would require ethics review and parental permission and age-appropriate assent.

RESULTS

The synthesized findings support an evidence hierarchy in which diabetes-associated oral-health abnormalities and established prevention practices can be discussed with moderate confidence, but an independent causal contribution of pediatric DPN remains undetermined. The available pediatric studies commonly assess caries experience, plaque, gingival status, salivary variables and glycemic indices; they seldom stratify dental outcomes by confirmed neuropathy and may not use uniform lesion-activity criteria. Table 1 therefore distinguishes sources capable of informing prevention from those that should not be interpreted as testing DPN-specific effectiveness.

Table 1. Selected published evidence and limits of applicability to children with diabetic polyneuropathy

Evidence source	Reported finding	Interpretation for pediatric DPN
Gunasekaran et al. (2022), n=80 children with T1D [6]	59% with caries experience; 94% gingivitis; 52% reduced saliva buffer	Pediatric dental burden; neuropathy subgroup not reported
Ta et al. (2025), 16 pediatric T1D studies [7]	15 cross-sectional; one longitudinal; microbiota associated with glycemic status	Limited longitudinal evidence; no DPN-specific preventive efficacy

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Evidence source	Reported finding	Interpretation for pediatric DPN
Wang et al. (2025), 40 T1D vs 40 controls [8]	Higher caries occurrence and plaque measures in T1D sample	Small retrospective study; no causal conclusion
SEARCH cohort, youth-onset diabetes [5]	Age-adjusted DPN prevalence 8.5% (T1D), 17.7% (T2D)	Neurological epidemiology only; caries not measured
Adult DCCT/EDIC participants [10]	DPN associated with tooth loss (OR 1.88; 95% CI 1.24–2.87)	Adult tooth loss, not pediatric caries causation

The numerical estimates most directly related to pediatric oral health are shown in Figure 1. The three variables are different clinical outcomes within the same 80-participant cohort and are not mutually exclusive; percentages must not be added together. The original investigation additionally found that HbA1c was not significantly associated with caries experience in this sample [6].

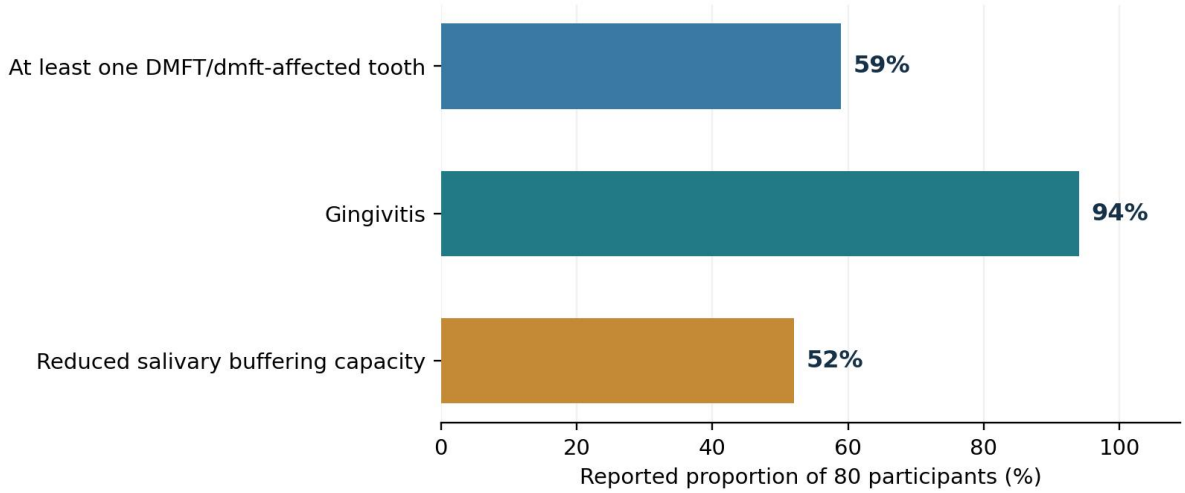


Figure 1. Selected oral-health findings reported in 80 children and adolescents with type 1 diabetes by Gunasekaran et al. [6]. Percentages are rounded as in the source, represent different outcomes and do not identify DPN-specific findings.

The available pediatric and transitional-age neuropathy evidence clarifies why special-needs care planning may be relevant while also exposing a major knowledge gap. Figure 2 reproduces published age-adjusted peripheral neuropathy prevalence in the SEARCH cohort; the type 1 and type 2 diabetes groups differed in characteristics and risk profiles, and neither bar represents caries prevalence or preventive treatment response [5]. These findings justify clinician awareness of neuropathy, not the automatic assumption that oral tactile deficits or salivary dysfunction are present.

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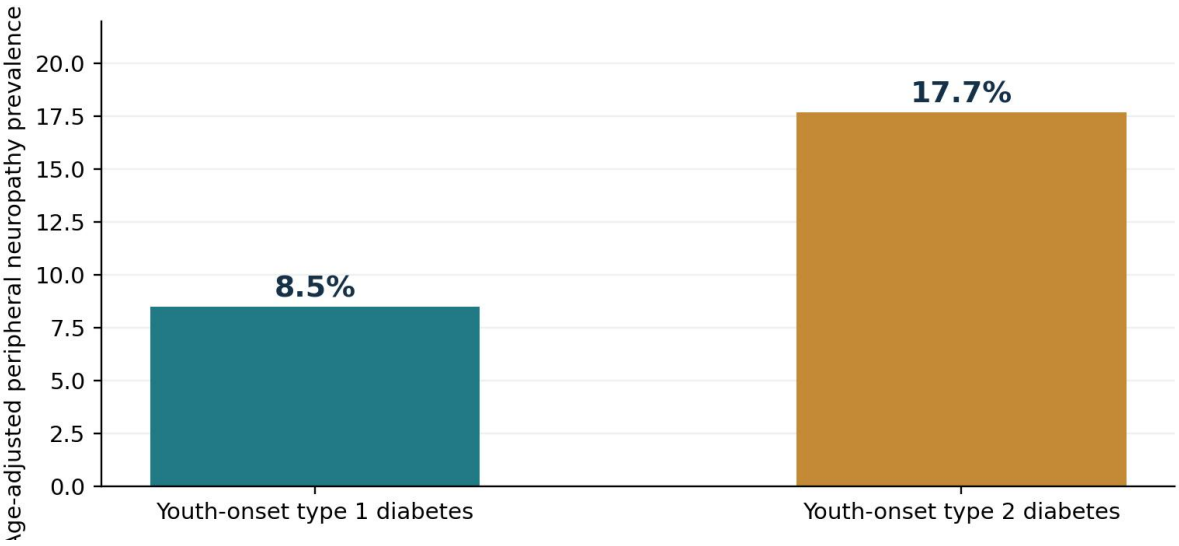


Figure 2. Peripheral neuropathy prevalence in participants with youth-onset diabetes assessed during adolescence or young adulthood in the SEARCH study [5]. Age-adjusted estimates; not dental outcomes and not a randomized comparison.

The proposed risk appraisal expands ordinary caries assessment to include the practical impact of diabetic complications without turning DPN into a universal high-risk classification. The clinician should record active noncavitated and cavitated lesions, previous restorations, hygiene quality, plaque, frequency of sugar exposure, fluoride history, saliva-related symptoms and accessibility of follow-up. Neuropathy-specific questions concern independently documented difficulties with dexterity, pain, sensation, fatigue or self-care rather than hypothetical oral neuropathic changes. Table 2 translates those dimensions into actionable clinical questions.

Table 2. Risk and function domains for integrated pediatric dental assessment

Assessment domain	Suggested examination or question	Implication for preventive planning
Caries activity and prior burden	Visual assessment with appropriate drying; record new and active lesions, fillings, plaque	Primary determinant of individualized recall and lesion-specific care
Diet and hypoglycemia	Review snacking pattern, sweetened medicines, essential rescue-carbohydrate use	Coordinate routine diet counseling without compromising urgent hypoglycemia treatment
Fluoride and toothbrushing	Review age-appropriate fluoride toothpaste, brushing assistance, professional varnish	Reinforce proven protective factors; verify caregiver support
Salivary / mucosal symptoms	Ask about dry mouth, medications, hydration and mucosal discomfort; test when indicated	Investigate reversible causes, not presumed autonomic neuropathy

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Assessment domain	Suggested examination or question	Implication for preventive planning
DPN and functional capacity	Confirm medical diagnosis; ask about hand dexterity, sensory pain and ability to self-brush	Offer adaptive aids or assisted brushing only when documented need exists
Health-system continuity	Dental access, missed visits, endocrinology communication, school support	Coordinate recall and written prevention plan

Prevention should begin with established age-appropriate recommendations rather than a novel untested drug or device. For most children, twice-daily assisted or independent brushing with fluoridated toothpaste, plaque control, modification of nonessential frequent sugar exposures and routine dental surveillance remain the core of care [11,12]. A dental professional may select professionally applied fluoride varnish, pit-and-fissure sealants and nonrestorative arrest measures according to lesion status, age, susceptibility and local regulatory guidance [13–15]. If a child with DPN cannot maintain adequate home hygiene because of demonstrated functional limitations, adapted handles, caregiver-assisted brushing or structured coaching may be reasonable pragmatic modifications; their DPN-specific impact must be prospectively established. Table 3 separates the established interventions from proposed adaptations.

Table 3. Proposed risk-adapted preventive management (evidence versus adaptation)

Clinical scenario	Evidence-informed action	Evidence status / safeguard
Low disease activity, reliable home care	Twice-daily fluoridated brushing; age-appropriate diet guidance; regular review	Established pediatric recommendations [11,12]
Active early lesions or high caries risk	Individualized fluoride varnish, sealants on susceptible molars, nonoperative lesion care	Established lesion-specific preventive guidance [12–15]
Frequent medically necessary hypoglycemia rescue	Treat hypoglycemia immediately; plan routine exposures with diabetes team; resume oral hygiene as feasible	Essential medical treatment takes priority; dental tactics are adjunctive [16]
Confirmed DPN with self-care difficulty	Adapt toothbrush grip / caregiver assistance; shorter instructions; documented functional review	Proposed individualized support, not proven DPN-specific effect [17]
Dry mouth, pain or complex comorbidity	Evaluate underlying cause; coordinate endocrine and dental review; consider specialist referral	Assessment required; do not attribute xerostomia to DPN by default [3,4]

The resulting five-stage framework is shown in Figure 3. It is intentionally iterative: a newly detected active lesion, increased sugar frequency, altered medication or emerging

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functional limitation may necessitate a revised plan. For medically complex children, the dental history and plan should be communicated to the diabetes team and family without changing insulin treatment or interfering with rapid correction of hypoglycemia. The pathway is a conceptual implementation aid rather than a tested diagnostic rule.

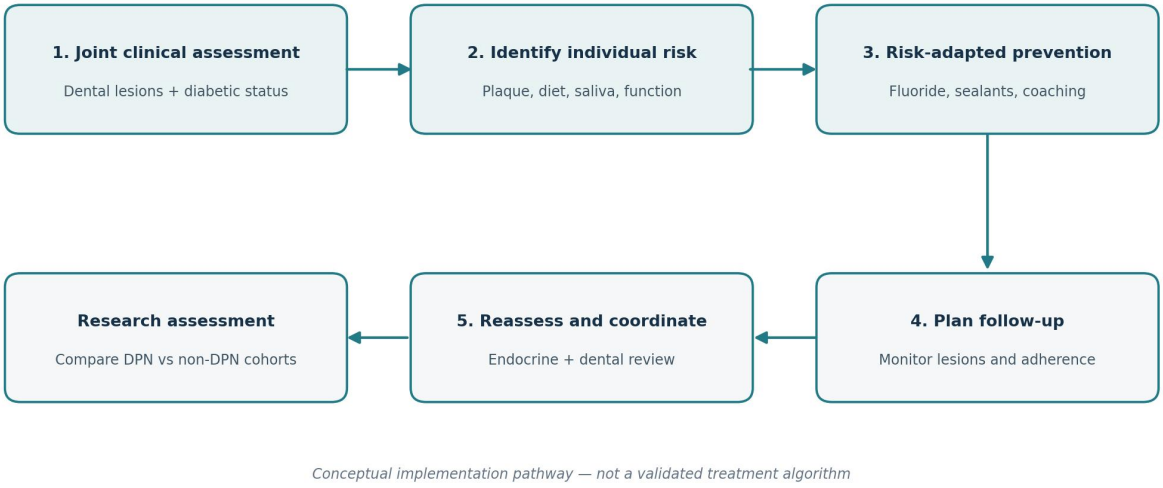


Figure 3. Author-proposed coordinated dental caries-prevention pathway for children with diabetes and documented polyneuropathy. Conceptual model based on pediatric guidance [3,4,11–18]; its effectiveness has not been clinically validated.

For future clinical validation, the most informative design would enroll children with diabetes and objectively documented DPN alongside a comparable pediatric diabetes group without DPN. Neuropathy status should be confirmed using the treating specialist's appropriate criteria rather than inferred from diabetes duration or symptoms alone. Assessment should separate type 1 and type 2 diabetes or adjust for relevant differences, record baseline caries activity and glycemic context, and use prespecified follow-up. Table 4 proposes outcome definitions that would permit a rigorous comparison without assuming the treatment benefit in advance.

Table 4. Proposed outcome and follow-up matrix for a prospective validation study

Outcome	Suggested operational measure	Interpretation / safeguard
Primary dental outcome	New active cavitated lesions or caries increment over 12 months	Requires standardized, calibrated dental examination
Secondary lesion outcome	Arrest or progression of initially noncavitated lesions	Specify lesion scoring and examiner reliability
Protective behaviors	Supervised brushing logs, fluoride receipt, missed appointments	Proxy behaviors are not equivalent to reduced caries incidence

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Outcome	Suggested operational measure	Interpretation / safeguard
Risk modifiers	Diet frequency, HbA1c, diabetes type/duration, reported dryness, medications	Confounders; no single factor establishes causation
DPN phenotype	Clinician-documented DPN category and functional limitation	Distinguish peripheral from autonomic manifestations
Care acceptability / safety	Child/caregiver burden, hypoglycemia safety, adverse events, quality of life	Prevention must not obstruct medically necessary care

DISCUSSION

The current evidence base supports prevention-oriented oral care in children with diabetes but does not yet establish a distinct mechanism or treatment advantage for those with DPN. This distinction is essential when interpreting the literature. Associations among glycemic status, cariogenic microbiota, plaque and caries may reflect differing sampling strategies, oral hygiene, dietary patterns and access to care [6–9]. In the 80-participant pediatric sample, high frequencies of gingivitis and reduced buffering were observed, but an independent relationship between HbA1c and caries was not demonstrated [6]. Such heterogeneity argues for clinically assessed disease activity and modifiable exposures rather than automatic classification of all children with diabetes as having equivalent caries risk.

Neuropathy introduces additional interpretative complexity. The SEARCH results demonstrate that neuropathy can occur in people with youth-onset diabetes, especially in the type 2 diabetes cohort, whereas adult EDIC findings associate neuropathy with tooth loss [5,10]. Neither observation establishes that pediatric peripheral nerve disease independently changes enamel mineralization, salivary composition or cariogenic biofilm. Salivary dysfunction could be influenced by autonomic processes, medication, hydration or glycemia, but peripheral neuropathy should not be used as a substitute for direct investigation of these mechanisms. Likewise, difficulty performing dental self-care should be assessed functionally and not simply inferred from a neurological diagnosis.

The preventive priorities identified here are intentionally conservative. Fluoride exposure, indicated fissure sealants, nonrestorative lesion management, appropriate diet counseling and sustained caregiver engagement all have a broader pediatric evidence base [11–15]. The novelty of the proposed framework lies in structuring communication between pediatric dentistry, endocrinology and families and in requiring DPN-specific adaptation to be justified by measured need. This approach also protects against potentially harmful messages surrounding hypoglycemia: a child should never delay or avoid prescribed rapid-acting carbohydrate treatment to protect the teeth. Instead, the dental team should help

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reduce discretionary exposures and support hygiene after the acute episode, with instructions aligned to the child's diabetes plan [16].

Several limitations require explicit acknowledgement. The present review is narrative and not exhaustive; no independent dual-reviewer screening, formal risk-of-bias synthesis or pooled-effect calculation was performed. The key pediatric oral-health study was cross-sectional, the 2025 microbiota review identified predominantly cross-sectional evidence, and adult tooth-loss data are not directly transportable to children with DPN [6,7,10]. Estimates in Figures 1 and 2 arise from different cohorts and different outcomes and cannot be joined into a synthetic predictive model. Regional prevalence, current patient care at the author's institution and the effectiveness of any proposed intervention in Uzbekistan were not evaluated. The framework should therefore be read as a hypothesis for structured service improvement and prospective study, not as an established therapeutic protocol.

Future research should prioritize multicenter prospective cohorts, standardized clinical neuropathy assessment, calibrated caries examinations and the careful measurement of salivary variables where clinically indicated. Comparators must account for diabetes duration, type, age, glycemic status, fluoride exposure, socioeconomic conditions and oral-hygiene assistance. Pragmatic intervention studies could test whether coordinated dental appointments, caregiver-assisted brushing or tailored counseling improve adherence and lesion outcomes in the subset of children whose DPN produces measurable self-care barriers. Until such evidence is available, standard pediatric prevention remains the appropriate foundation for clinical care.

CONCLUSION

Dental caries prevention in children with diabetic polyneuropathy should be grounded in established pediatric oral-health measures and individualized assessment rather than assumptions about neuropathy-related caries mechanisms. Current studies suggest that children with diabetes may experience substantial plaque, gingival and selected salivary abnormalities; however, the direct and independent contribution of pediatric DPN to new caries development remains unproven. A coordinated pathway incorporating lesion activity, fluoride, sealants, dietary counseling, observed self-care capacity, safe hypoglycemia management and joint pediatric dental–endocrine follow-up is clinically reasonable. Its superiority over routine risk-based care must be evaluated in prospective pediatric studies before it can be claimed as an effective DPN-specific prevention strategy.

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