



Association between glycemic control, nutrition, and periodontal outcomes in type 2 diabetes: a narrative review

Rutuja Khobragade¹, Vineetha Saminathan², Akshata Chikali³, Navin Kumar D⁴,
Laxmikant Late⁵, Twinkle Rani⁶, Ritik Kashwani^{7,*}

¹ Post Graduate Student, Department of Community Medicine, DMIHER Nagpur, India.

² Research Scholar, Department of Physiology, SRM Medical College Hospital and Research Centre, SRMIST, Kattankulathur-603203, Chengalpattu (Dt), Tamil Nadu, India.

³ Senior Resident, Department of Community Medicine, Jawaharlal Nehru Medical College, KLE Academy of Higher Education and Research (Deemed-to-be-University), Belagavi, Karnataka, India.

⁴ Assistant Professor, Department of Periodontology, Madha Dental College, Chennai, Dr. MGR Medical University, Guindy, Chennai, Tamil Nadu, India.

⁵ Department of Conservative Dentistry and Endodontics, Dr. D. Y. Patil Dental College and Hospital, Dr. D. Y. Patil Vidyapeeth, Pune, Maharashtra, India.

⁶ Intern, Kalinga Institute of Dental Sciences, KIIT deemed to be University, Bhubaneswar, Odisha, India.

⁷ Font Fusions Publication Pvt Ltd., Sector-22, Gautam Buddha Nagar, Uttar Pradesh, 201301, India.

*Corresponding author: Ritik Kashwani.

Director, Font Fusions Publication Pvt Ltd., Sector-22, Gautam

Buddha Nagar, Uttar Pradesh, 201301, India.

E-mail: director@fontfusionspublication.com

DOI: <https://doi.org/10.54448/ijn26407>

Received: 07-30-2026; Revised: 09-12-2026; Accepted: 09-25-2026; Published: 10-03-2026; IJN-id: e26407

Editor: Dr. Carla Maria Barreto da Silva de Sousa Rego, MD, MsD, Ph.D.

Abstract

Introduction: Type 2 diabetes mellitus (T2DM) and periodontitis are two prevalent chronic conditions that have been implicated in each other in a bidirectional manner, whereby hyperglycemia can predispose to periodontal disease, and periodontal inflammation can lead to hyperglycemia. Both conditions are linked via nutrition, which affects systemic inflammation, oxidative stress, and the host response in the periodontal tissues. **Objective:** The aim was to review the existing literature on the association between glycemic control, food intake and periodontal status in T2DM adults and to provide insight into the importance of integrated nutritional and periodontal care. **Methods:** PubMed/MEDLINE, Scopus, and Web of Science were queried for observational studies, interventional trials, systematic reviews, and mechanistic studies related to T2DM and glycated hemoglobin (HbA1c) levels, diet and micronutrient exposures, and periodontal parameters. **Results:** There is a consistent relationship between poor glycemic control (HbA1c $\geq$ 7%) and increased periodontal inflammation, pocket depth and attachment loss; severe periodontitis is associated with

poorer glycemic control; non-surgical periodontal therapy is associated with a short-term decrease of 0.3-0.4% in HbA1c. Mechanistic factors that are common to both include advanced glycation end product-receptor (AGE-RAGE) axis, oxidative stress, and pro-inflammatory cytokine signaling. Western type diets high in refined carbohydrates are associated with increased periodontal inflammation and poor metabolic profiles while low-glycemic diets with a higher omega-3 fatty acid, fiber and antioxidant vitamin (C, D, E) content are associated with decreased periodontal inflammation and better metabolic profiles., whereas Western-type diets high in refined carbohydrates have the opposite effect. **Conclusion:** There is a bi-directional relationship between glycemic control, nutrition and periodontal health in T2DM. Nutritional assessment and counselling together with diabetes and periodontal management is a plausible and low risk intervention to reduce morbidity, and there is limited high quality evidence.

Keywords: Type 2 Diabetes Mellitus. Periodontitis. Glycated Hemoglobin. Nutrition Therapy. Oxidative Stress. Fatty Acids. Omega-3.

Graphical Abstract

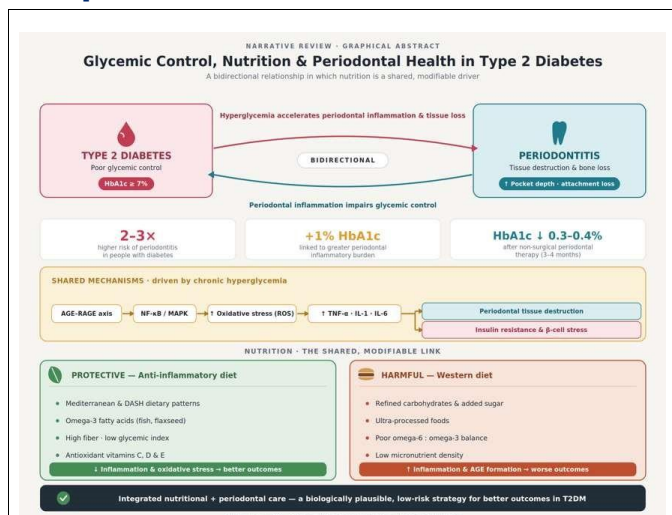


Figure 1. Bidirectional association and modifiable effect of nutrition on periodontal outcomes in type 2 diabetes mellitus (T2DM). Poor glycemic control (HbA1c ≥ 7%) and periodontal breakdown are mutually influential and each of them may contribute to the other through similar mechanisms, such as the activation of advanced glycation end product by its receptor (AGE-RAGE), the production of reactive oxygen species (ROS) and pro-inflammatory cytokines (interleukin-6 [IL-6], tumor necrosis factor-α [TNF-α] and C-reactive protein [CRP]). An anti-inflammatory (Mediterranean or DASH Pattern, dietary fibre, vitamins C, D, E, and omega-3 fatty acids) are associated with lower inflammatory markers and better glycemic and periodontal parameters, while western dietary pattern is directly associated with higher inflammatory markers and worse glycemic and periodontal parameters. The use of non-surgical periodontal therapy is correlated to around 0.3% to 0.4% decrease in HbA1c, and combined nutrological and periodontal therapy focuses on both diseases. Source: Own authorship.

Introduction

The prevalence of type 2 diabetes mellitus (T2DM) is increasing, particularly with increasing global obesity and population ageing, and is also one of the most common metabolic diseases globally [1]. Periodontal disease (PD), a chronic inflammatory disease of the supporting tissues of the teeth, is now known to be part of the diabetes associated complications, which include a variety of micro and macrovascular diseases caused by chronic hyperglycemia [2,3]. Periodontitis develops when there is a dysbiotic subgingival biofilm and it is maintained by a dysregulated host immune-inflammatory response that causes progressive destruction of the periodontal ligament and alveolar bone, and finally, tooth loss (if not treated) [3].

Now, it is known that there is a bidirectional relationship between diabetes and periodontitis. Poor glycemic control seems to be associated with higher prevalence, severity and duration of periodontitis, and,

poor glycemic control is associated with incident diabetes and diabetic complications [4,5]. This bidirectional connection has been proven in many observational studies and is supported by randomized controlled trials of periodontal interventions in which there was moderate improvement in metabolic parameters [5,6].

Both of these diseases are at the center of nutrition and in some cases, underemphasized. Food diets influence postprandial glycemia, systemic inflammation and oxidative stress and micronutrient availability for tissue repair and immune regulation which in turn affect metabolic control and periodontal tissue integrity [7,8]. Thus, the field of nutrology, which addresses the prevention and treatment of disease through nutrition, is well suited to provide input to integrated care models for the treatment of T2DM and periodontal disease [9].

The purpose of this narrative review is to summarize the current evidence of the association between glycemic control, nutrition and periodontal outcomes in adults with T2DM. It underscores the epidemiological connections, examines common biological pathways, assesses the role of dietary patterns and individual nutrients, and considers the clinical implications of an interdisciplinary approach of nutrition and periodontal health.

Materials and Methods

The authors designed this work as a narrative review. The following keywords were used in PubMed/MEDLINE, Scopus and Web of Science search: Type 2 Diabetes, Glycemic Control, Glycated Hemoglobin, HbA1c, Periodontitis, Periodontal Disease, Nutrition, Diet, Micronutrients, Omega-3 Fatty Acids, Vitamin C, Vitamin D, Antioxidants and Advanced Glycation End Products. The reference lists of retrieved articles were hand searched for further relevant articles.

Eligible studies were those that were observational (cross-sectional, case-control, or cohort design) and/or interventional trials, systematic reviews, and/or, mechanistic reports that examined at least two of the three domains of interest (glycemic control, nutrition, periodontal outcomes in the context of T2DM). More recent and methodologically adequate studies were selected, and seminal older studies, which were useful in the mechanistic story were kept. No protocol registration or a priori sample-size or statistical power calculation was possible as the review does not pool primary data; this is a methodological limitation, as is the absence of a formal risk-of-bias assessment and no protocol registration.

Results and Discussion

The bidirectional relationship between glycemic control and periodontal outcomes

A large body of evidence indicates that the degree of glycemic control is a key determinant of periodontal risk in people with T2DM. Individuals with diabetes have an estimated 2–3 times higher risk of developing periodontitis than those without diabetes, and this risk depends strongly on glycemic status [10]. HbA1c levels and the extent of diabetes complications are higher in people with T2DM who have periodontitis than in those who do not; likewise, among people without diabetes, HbA1c, fasting glucose, and diabetes risk are higher in those with periodontitis than in those without [11].

This association has been quantified using cross-sectional data. In a study of adults with T2DM, poor glycemic control (HbA1c $\geq 7\%$), a family history of diabetes, and C-reactive protein (CRP) were predictors of severe periodontitis, with each 1% increase in HbA1c corresponding to a significant increase in the periodontal inflamed surface area — an indicator of the total periodontal inflammatory burden [12]. Conversely, poor glycemic control was predicted by severe periodontitis, waist circumference, dietary imbalance, and a sedentary lifestyle, with the inflammatory burden of periodontitis being the strongest predictor. Beyond marginal periodontal tissues, glycemic control has also been related to apical (endodontic) periodontitis [13].

A therapeutic corollary of this relationship is that treating periodontitis may benefit metabolic control. In individuals with T2DM, non-surgical periodontal therapy (periodontal biofilm removal) has been shown to reduce HbA1c by approximately 0.3–0.4% (3–4 mmol/mol) at three to four months after treatment [14]. This effect size is small and not universal but is clinically meaningful and, in some analyses, comparable to adding a second glucose-lowering agent. Because of heterogeneity in baseline glycemic control, treatment intensity, and maintenance adherence, not all retrospective studies report a significant change in HbA1c. HbA1c therefore serves as a key biomarker linking the two diseases and a common endpoint in integrated care [15].

Shared biological mechanisms

Overlapping inflammatory and metabolic pathways drive the bidirectional relationship. The non-enzymatic glycation and oxidation products of proteins and lipids that are secreted into the plasma and tissues of hyperglycemic patients are advanced glycation end products (AGEs) and are proposed to be a critical

mechanism. The receptor for advanced glycation end products (RAGE) is the main cellular receptor of AGEs in the periodontium and it is expressed in gingival epithelium, endothelial cells, monocytes and macrophages [16].

The activation of AGE–RAGE axis triggers additional intracellular signaling pathways, including mitogen-activated protein kinase pathway and nuclear factor- κ B (NF- κ B) pathway leading to further increase in the generation of reactive oxygen species (ROS) and pro-inflammatory cytokines such as tumor necrosis factor- α (TNF- α), interleukin-1 (IL-1) and interleukin-6 (IL-6) [17]. This exaggerated and dysregulated inflammatory process leads to the loss of connective tissue and alveolar bone, slows down the healing process after periodontal surgery and contributes to a more chronic periodontal inflammatory state [18]. In early experimental studies, the gingival tissue destruction was directly attributed to oxidant stress and this effect was blocked by antioxidant pretreatment, which was suggested to explain the increased periodontal tissue loss in diabetes [19].

The reverse direction of the relationship can be attributed to oxidative stress and the same AGE–RAGE mechanism that plays a role in systemic insulin resistance and β -cell dysfunction and causes metabolic dyshomeostasis in T2DM [20]. Low level systemic inflammatory mediators from periodontal infection may result in chronic systemic inflammation, and this is linked with increased insulin resistance and glycemic control [21]. Therefore, the excess ROS can be considered as an upstream node, and the antioxidant capacity, which is greatly modifiable by diet, as a common one [22].

Nutrition as a modifiable factor in periodontal and metabolic health

The role of nutrition in the diseases is exerted by several converging pathways: modulation of postprandial glycemia and insulin demand, control of systemic and local inflammation, antioxidant defense and tissue repair and immune function [23]. Considering the fact that dietary pattern is a potential target for glycemic control and periodontal outcomes improvement, it is logical to suggest that dietary pattern could be changed to obtain those benefits.

At the level of dietary patterns, a clear contrast emerges between two opposing profiles. Western-type diets raise circulating inflammatory markers (such as IL-6, TNF- α , and CRP) [24] and, after a meal, can drive greater postprandial hyperglycemia and AGE formation, which are associated with increased periodontal inflammation [25]. In contrast, anti-inflammatory diets such as the Mediterranean and

Dietary Approaches to Stop Hypertension (DASH) diets — low in sugar, high in fiber, rich in vegetables, and with a favorable omega-6:omega-3 ratio — are linked to lower periodontal inflammation and oxidative stress and to reduced cardiovascular and diabetes risk [26]. One frequently cited intervention achieved a significant reduction in gingival and periodontal inflammation by switching patients from a Western diet to one low in processed carbohydrates and high in omega-3 fatty acids, vitamin C, vitamin D, and fiber, suggesting host modulation rather than an effect on the biofilm [27].

In T2DM, low-glycemic, high-fiber diets can improve glycemic control while reducing the availability of substrates for AGE formation, addressing both metabolic and periodontal objectives at once [28].

Specific nutrients and micronutrients

Omega-3 polyunsaturated fatty acids (PUFA): Omega-3 fatty acid-based specialized pro-resolving lipid mediators are active anti-inflammatory compounds. Omega-3 supplementation with non-surgical periodontal therapy can be seen to have a beneficial effect on probing depth and clinical attachment level, but the data from the different clinical trials are inconsistent and the results are difficult to draw clear conclusions from because of inconsistency between different trials. The beneficial effects of omega-3 fatty acids on inflammatory milieu and lipid profile in diabetes are also beneficial [29].

Vitamin C (ascorbic acid): Vitamin C is needed for the synthesis of collagen and is a prominent antioxidant in the water-soluble part of the body. Deficiency leads to lessening of the integrity of connective tissue and less periodontal wound healing, and observational studies indicate a relationship between vitamin C consumption and periodontal disease. Diabetics are predisposed to oxidative stress and ascorbate status could be of special importance in this population [30].

Calcium and vitamin D: Vitamin D is an immunomodulator and in combination with calcium is vital to alveolar bone metabolism. Positive correlation has been established between better vitamin D status (25 hydroxy vitamin D in serum) and less periodontal disease and recovery after non-surgical periodontal therapy. Vitamin D also plays a role in insulin sensitivity and glucose homeostasis, which is another reason why it is similar to metabolic control [31].

Other antioxidant micronutrients: Antioxidants (vitamins A and E, minerals like zinc, magnesium, selenium) help maintain antioxidant protection, regulate immune function, and repair tissues; low intakes have been linked to increased periodontal disease, while foods high in antioxidants, minerals, and

dietary polyphenols have been associated with better periodontal and overall health. There are also emerging indications that probiotics and prebiotics have a beneficial effect on modulation of oral and gut microbiota to improve its state [32].

Of particular interest is the effect of micronutrient fortification or supplementation with individual micronutrients, which has yielded mixed results, and the effect of overall dietary quality, which has yielded the most consistent results [33]. Thus, the best nutrition tips are to follow an anti-inflammatory whole-food diet.

Clinical implications for integrated care

The relationship between glycemic control, nutrition, and periodontal disease lends support to an interprofessional, patient-centered approach to the care of a person with T2DM. Oral health professionals and periodontists have a strong advantage in providing diabetes care and screening for and treating periodontitis and in reinforcing dietary advice, as well as diabetes care and education, and physicians, nutrologists and dietitians who manage diabetes, have a strong advantage in providing diabetes care and education, screening and nutritional counseling for oral health, and diabetes care and education for metabolic and periodontal endpoints [34].

Based on current evidence, the following practical recommendations are proposed: low-glycemic and high fibre anti-inflammatory diet (Mediterranean or DASH diets); enough antioxidant vitamins (vegetables and fruits); limit refined carbohydrates and ultra-processed foods; and supply and maintain periodontal treatments as a component of diabetes treatment [35]. Oral examination can be used as an easy portal to the metabolic health and diet [36] as any deterioration in periodontal condition may be an early indicator of poor diet and metabolic risk.

Nutritional counseling should be tailored, culturally sensitive and multidisciplinary, as cultural context and socioeconomic circumstances have a significant impact on adherence to diets [37]. Table 1 summarizes representative studies underpinning the relationships discussed below, including their design and principal findings.

Table 1. Summary of representative studies on the relationships among glycemic control, nutrition, and periodontal outcomes in T2DM.

Study (first author, year)	Design / type	Focus	Key findings as discussed
Nascimento et al., 2018 [28]	Systematic review & meta-regression (prospective cohorts)	Diabetes as a risk factor for periodontitis	Diabetes is associated with an increased risk of incident periodontitis, supporting a directional link from hyperglycemia to periodontal disease.

Romano et al., 2021 [10]	Cross-sectional study (T2DM cohort)	Metabolic control and periodontal inflammatory burden	Bidirectional association: higher HbA1c relates to greater periodontal inflamed surface area, and periodontal inflammatory burden strongly predicts poor glycemic control.
Pérez-Losada et al. 2020 [11]	Cross-sectional study	Apical (endodontic) periodontitis and glycemic control	Glycemic control in T2DM is associated with apical periodontitis, extending the link beyond marginal periodontal tissues.
Simpson et al., 2022 [6]	Systematic review (Cochrane)	Periodontal therapy for glycemic control	Non-surgical periodontal therapy is associated with a short-term HbA1c reduction of ~0.3–0.4% in people with diabetes.
Kim et al., 2021 [12]	Clinical study	HbA1c change after periodontal therapy	Periodontal therapy is followed by measurable HbA1c change, the magnitude varying with baseline control and adherence.
Sanz et al., 2018 (EFP/IDF) [27]	Consensus report	Perio–diabetes evidence and clinical guidance	Consolidates evidence for the bidirectional link and recommends integrated periodontal–diabetes management.
Schmidt et al., 1996 [17]	Experimental study	AGE-induced oxidant stress in gingiva	AGEs induce gingival oxidant stress; antioxidant pretreatment is protective — an early mechanistic basis for accelerated periodontal breakdown in diabetes.
Katz et al., 2005 [15]	Clinical / tissue study	RAGE expression in gingiva	RAGE is expressed in gingival tissues of T2DM patients with chronic periodontitis, supporting local AGE–RAGE signaling.
Chapple & Matthews, 2007 [19]	Mechanistic review	ROS and antioxidants in periodontal destruction	Reactive oxygen species and impaired antioxidant defense contribute to periodontal tissue destruction.
Woelber et al., 2017 [20]	Randomized controlled pilot trial	Oral health–optimized (anti-inflammatory) diet	An anti-inflammatory, low-processed-carbohydrate diet reduced gingival and periodontal inflammation, indicating host modulation.
Bartha et al., 2022 [21]	Randomized controlled trial	Anti-inflammatory diet and periodontitis	Dietary modification reduced periodontal inflammation, supporting diet as a modifiable factor.
Stańdo-Retecka et al., 2023 [22]	Systematic review	Omega-3 adjunct to non-surgical therapy	Omega-3 supplementation as an adjunct may improve clinical periodontal parameters, though results are heterogeneous.
Machado et al., 2020 [24]	Systematic review & meta-analysis	Vitamin D and periodontitis	Better vitamin D status is associated with improved periodontal outcomes.

*HbA1c, glycated hemoglobin; AGE, advanced glycation end product; RAGE, receptor for advanced glycation end products; ROS, reactive oxygen species; EFP/IDF, European Federation of Periodontology/International Diabetes Federation.

Limitations and future directions

The current evidence-based is flawed in several respects. The majority of nutrition and periodontal/glycemic data is derived from cross sectional and observational studies, which are unable to demonstrate causality and are confounded by other factors, including smoking, obesity and socioeconomic status [38]. There is limited research on dietary patterns or specific nutrients in patients with T2DM and periodontitis, and most studies are small and have a variety of designs, which makes synthesis difficult via meta-analysis. Likewise, the reported modest and variable reduction in HbA1c with periodontal treatment is similar to the variability seen from one person to the next and one clinic to the next [39].

Randomized controlled trials using well-designed diet and nutrient profile interventions as adjuncts to periodontal treatment in individuals with T2DM, with clearly defined periodontal and glycemic outcomes are suggested to be performed in the future. The metabolomic, microbiome and inflammatory markers in the mechanistic studies will help to understand the specific nutrients influence on AGE–RAGE and oxidative-stress pathway in periodontal and metabolic tissue. This type of research would contribute to the evidence supporting the integrated approach to nutrological and periodontal care which is proposed in this synthesis.

Conclusion

Glycemic control, nutrition, and periodontal health are bidirectionally connected in type 2 diabetes. Poor glycemic control aggravates periodontal inflammation and tissue destruction, severe periodontitis compromises metabolic control, and nutrition is a common, modifiable determinant acting through inflammation, oxidative stress, and the AGE–RAGE axis. Anti-inflammatory dietary patterns emphasizing fiber, omega-3 fatty acids, and antioxidant micronutrients are associated with more favorable periodontal and metabolic profiles, whereas Western-type diets have the opposite effect. Integrating nutritional assessment and dietary counseling into diabetes and periodontal care is a biologically plausible, low-risk strategy that may improve outcomes. Robust interventional evidence is still needed to define optimal nutritional interventions and to quantify their effects on hard clinical endpoints.

CRedit

Author contributions: All authors contributed to the conception of the review, the literature search and analysis, and the drafting and critical revision of the manuscript, and all approved the final version and agree to be accountable for the work.

Acknowledgment

The authors thank their respective institutions for their support.

Ethical Approval

Not applicable. This is a narrative review of previously published literature and did not involve new studies on human participants or animals.

Informed Consent

It was applicable.

Funding

This review received no external funding from any agency in the public, commercial, or not-for-profit sectors.

Data Sharing Statement

Data sharing is not applicable to this article, as no new data were created or analyzed. This narrative review is based entirely on previously published studies, all of which are cited in the reference list and are available through their original publishers.

Study Registration

Not applicable. As a narrative (non-systematic) review, this work was not eligible for PROSPERO registration and no protocol was registered.

Publication statement

All authors have agreed to the publication of this article.

Conflict of Interest

The authors declare no competing interests.

Similarity Check

It was applied by Ithenticate®.

Application of Artificial Intelligence (AI)

Not applicable.

Peer Review Process

It was performed.

About The License©

The author(s) 2026. The text of this article is open access and licensed under a Creative Commons Attribution 4.0 International License.

References

1. GBD 2021 Diabetes Collaborators. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. *Lancet*. 2023;402(10397):203-234.
2. Loe H. Periodontal disease. The sixth complication of diabetes mellitus. *Diabetes Care*. 1993;16(1):329-334.
3. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases. *Nat Rev Dis Primers*. 2017;3:17038.
4. Preshaw PM, Bissett SM. Periodontitis and diabetes. *Br Dent J*. 2019;227(7):577-584.
5. Nirankari K, et al. Global dietary patterns and their impact on oral health: insights for dentistry. *Dent Dent Pract J*. 2024;6(2):180063.
6. Simpson TC, Clarkson JE, Worthington HV, MacDonald L, Weldon JC, Needleman I, et al. Treatment of periodontitis for glycaemic control in people with diabetes mellitus. *Cochrane Database Syst Rev*. 2022;4(4):CD004714.
7. Martinon P, Fraticelli L, Giboreau A, Dussart C, Bourgeois D, Carrouel F. Nutrition as a key modifiable factor for periodontitis and main chronic diseases. *J Clin Med*. 2021;10(2):197.
8. Woelber JP, Tennert C. Diet and periodontal diseases. *Monogr Oral Sci*. 2020;28:125-133.
9. Nogueira-de-Almeida CA, Ribas Filho D, et al. Nutrology and the integrated management of chronic disease: principles and practice. *Int J Nutrology*. 2023;16(1):1-10.
10. Romano F, Perotto S, Mohamed SEO, Bernardi S, Giraudi M, Caropreso P, et al. Bidirectional association between metabolic control in type-2 diabetes mellitus and periodontitis inflammatory burden: a cross-sectional study in an Italian population. *J Clin Med*. 2021;10(8):1787.
11. Pérez-Losada FL, Estrugo-Devesa A, Castellanos-Cosano L, Segura-Egea JJ, López-López J, Velasco-Ortega E. Apical Periodontitis and Diabetes Mellitus Type 2: A Systematic Review and Meta-Analysis. *J Clin Med*. 2020 Feb 17;9(2):540.
12. Kim EK, Lee SG, Choi YH, Won KC, Moon JS, Merchant AT, et al. HbA1c changes in patients with diabetes following periodontal therapy. *J Periodontal Implant Sci*. 2021;51(2):133-143.
13. Du Y, Xiao H, Luo R, Li G, Ren Y. The role of HbA1c in the bidirectional relationship between periodontitis and diabetes and related interventions: a narrative review. *Front Nutr*. 2025;12:1606223.
14. Vlassara H, Uribarri J. Advanced glycation end products (AGE) and diabetes: cause, effect, or both? *Curr Diab Rep*. 2014;14(1):453.
15. Katz J, Bhattacharyya I, Farkhondeh-Kish F, Perez FM, Caudle RM, Heft MW. Expression of the receptor of advanced glycation end products in gingival tissues of type 2 diabetes patients with chronic periodontal disease. *J Clin Periodontol*. 2005;32(1):40-44.
16. Nayyar A, Sood A, Chaudhry S, Kaur H, Khanna C, Pawar P. Titanium implant corrosion and peri-implant disease: current concepts and future directions. *Oral Sphere J Dent Health Sci*.

2026;2(1):47-51.

17. Schmidt AM, Weidman E, Lalla E, Yan SD, Hori O, Cao R, et al. Advanced glycation endproducts (AGEs) induce oxidant stress in the gingiva: a potential mechanism underlying accelerated periodontal disease associated with diabetes. *J Periodontol Res.* 1996;31(7):508-515.
18. BR P, R Y, Kumar P, Patil K, Chandavarkar V, Sagarkar AR. Diabetes and dental woes: a study on burden of oral diseases in type 2 diabetes mellitus patients in Mysuru. *Oral Sphere J Dent Health Sci.* 2025;1(2):48-55.
19. Chapple ILC, Matthews JB. The role of reactive oxygen and antioxidant species in periodontal tissue destruction. *Periodontol* 2000. 2007;43:160-232.
20. Woelber JP, Bremer K, Vach K, König D, Hellwig E, Ratka-Krüger P, et al. An oral health optimized diet can reduce gingival and periodontal inflammation in humans - a randomized controlled pilot study. *BMC Oral Health.* 2017;17(1):28.
21. Bartha V, Exner L, Schweikert D, Woelber JP, Vach K, Meyer AL, et al. Effect of the Mediterranean diet on gingivitis: A randomized controlled trial. *J Clin Periodontol.* 2022 Feb;49(2):111-122.
22. Stańdo-Retecka M, Piatek P, Namiecinska M, Bonikowski R, Lewkowicz P, Lewkowicz N. Clinical and microbiological outcomes of subgingival instrumentation supplemented with high-dose omega-3 polyunsaturated fatty acids in periodontal treatment - a randomized clinical trial. *BMC Oral Health.* 2023 May 13;23(1):290.
23. Kaur A, Sood A, Chaudhry S, Khichy A, Arora R. Nutrigenomics: a narrative review of diet's influence on periodontal health. *Oral Sphere J Dent Health Sci.* 2025;1(4):259-262.
24. Machado V, Lobo S, Proença L, Mendes JJ, Botelho J. Vitamin D and periodontitis: a systematic review and meta-analysis. *Nutrients.* 2020;12(8):2177.
25. Dommisch H, Kuzmanova D, Jönsson D, Grant M, Chapple I. Effect of micronutrient malnutrition on periodontal disease and periodontal therapy. *Periodontol* 2000. 2018;78(1):129-153.
26. Gheisary Z, Mahmood R, Harri Shivanantham A, Liu J, Lieffers JRL, Papagerakis P, et al. The clinical, microbiological, and immunological effects of probiotic supplementation on prevention and treatment of periodontal diseases: a systematic review and meta-analysis. *Nutrients.* 2022;14(5):1036.
27. Sanz M, Ceriello A, Buysschaert M, Chapple I, Demmer RT, Graziani F, et al. Scientific evidence on the links between periodontal diseases and diabetes: consensus report of the joint EFP/IDF workshop. *J Clin Periodontol.* 2018;45(2):138-149.
28. Nascimento GG, Leite FRM, Vestergaard P, Scheutz F, López R. Does diabetes increase the risk of periodontitis? A systematic review and meta-regression analysis of longitudinal prospective studies. *Acta Diabetol.* 2018;55(7):653-667.
29. Lamster IB, Pagan M. Periodontal disease and the metabolic syndrome. *Int Dent J.* 2017;67(2):67-77.
30. Woelber JP, Gärtner M, Breuninger L, Anderson A, König D, Hellwig E, et al. The influence of an anti-inflammatory diet on gingivitis. A randomized controlled trial. *J Clin Periodontol.* 2019;46(4):481-490.
31. Moszura J, Gawlak-Socka S, Pęksa J, Bielecka-Kowalska N, Kłosek S. The role of vitamin D3 in periodontal health: implications for bone metabolism, immune modulation and inflammation control. *Nutrients.* 2026;18(4):577.
32. Vavetsi K, Nomikos T, Vassilopoulos S, Bobetsis YA. Effect of nutritional antioxidants on periodontal disease and periodontal therapy. *Dent J (Basel).* 2025;13(12):570.
33. Rucklidge JJ, Bruton A, Welsh A, Ast H, Johnstone JM. Annual Research Review: micronutrients and their role in the treatment of paediatric mental illness. *J Child Psychol Psychiatry.* 2025;66(4):477-497.
34. Mamun AA, Awual MR. Integrating Periodontal Care Into Diabetes Management: A Narrative Perspective for Bangladesh With Global Relevance. *Health Sci Rep.* 2025 Nov 5;8(11):e71465.
35. Caprara G. Mediterranean-type dietary pattern and physical activity: the winning combination to counteract the rising burden of non-communicable diseases (NCDs). *Nutrients.* 2021;13(2):429.
36. Balice G, Paolantonio M, Murmura G, Serroni M, Di Gregorio S, Femminella B. The influence of diet and physical activity on periodontal health: a narrative review. *Dent J (Basel).* 2025;13(5):200.
37. [Caselli LB, Maim RL, Chinarelo LM, Castro FPL de. Major considerations and outcomes of periodontal diseases and infective endocarditis: a concise systematic review. *MedNEXT J Med Health Sci [Internet].* 2025 Jun. 6 [cited 2026

Oct. 4];6(S3). Available from:
<https://mednext.zotarellifilhoscientificworks.com/index.php/mednext/article/view/459>

- 38.** Alexa I, Dumitrescu R, Alexa VT, Popescu S, Bolchis V, Floare L, Balean O, Galuscan A, Jumanca D. Cross-sectional comparative assessment of periodontal status in diabetic and non-diabetic individuals within a Romanian cohort. J Clin Med. 2025;14(22):8199.
- 39.** Păunică I, Giurgiu M, Dumitriu AS, Păunică S, Pantea Stoian AM, Martu MA, Serafinceanu C. The bidirectional relationship between periodontal disease and diabetes mellitus - a review. Diagnostics (Basel). 2023;13(4):681.