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The bidirectional relationship between diabetes mellitus and periodontal disease

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ABSTRACT

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia due to defects in insulin secretion, insulin action, or both. As a major global health concern, diabetes is associated with multiple systemic complications. In recent years, the bidirectional relationship between DM and periodontal diseases has gained increasing attention. This review aims to highlight the classification, pathophysiology, and complications of diabetes, while emphasizing the reciprocal interactions between DM and periodontal diseases. Moreover, the prevalence, pathogenesis, and clinical manifestations of periodontal diseases in diabetic individuals are discussed, along with dental treatment approaches tailored for these patients. The bilateral association between DM and periodontal disease highlights the necessity for clinicians to incorporate periodontal evaluations into the standard care of diabetic patients. Timely diagnosis, stringent glycemic management, and consistent periodontal treatment are critical measures to mitigate disease progression and enhance clinical results. Enhancing collaboration between medical and dental experts can improve patient education, facilitate preventative care, and ultimately preserve both systemic and periodontal health.

Keywords: Diabetes mellitus, gingivitis, periodontitis

INTRODUCTION

Diabetes mellitus (DM) is a chronic disease characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both, with a multifactorial etiology and lifelong duration.[1] There is evidence of a molecular link between periodontal disease and DM.[2] DM is known to increase the risk of periodontal disease, and several findings have been identified that explain this association.[3] In addition, periodontitis is known to significantly affect hemoglobin A1c (HbA1c) levels, and these patients tend to have poorer glycemic control. These two diseases show bidirectional relationships that trigger each other.[4] To reduce the incidence of DM-related diseases, periodontal diseases must be treated, and to mitigate periodontal diseases, DM must be well managed.[5] This review aimed to investigate the bidirectional relationship between DM and periodontal disease.

CLASSIFICATION OF DIABETES

There are two types of DM: Type 1 and Type 2. Type 1 DM (T1DM) is caused by the destruction of beta cells, resulting in insulin deficiency. Type 2 diabetes (T2DM) occurs when beta cells lose their ability to secrete insulin, typically due to insulin resistance.[6] (**Figure 1**). T2DM can be characterized by relative insulin deficiency, often accompanied by predominant insulin resistance, or by predominant insulin deficiency accompanied by insulin resistance.[7] According to a report prepared by the International Diabetes Federation (IDF), T2DM is the most common type of DM. According to the 9th edition of the IDF, approximately 90% of people of working age (20-64 years) with diabetes (diagnosed with or undiagnosed) in 2019 had T2DM. The number of people affected by DM is projected to rise to 417.3 million by 2030 and 486.1 million by 2045.[8] Only 10-15% of individuals with T1DM have a family history of the disease. It is present in 5% of siblings and 50% of monozygotic twins.[9] Evidence suggests that many genes play a role in insulin deficiency and beta-cell deficiency. Genome-wide studies have identified dozens of common genetic variants associated with an increased risk of T2DM.[10] Among the variants discovered thus far, the TCF7L2 gene has the most significant effect on susceptibility.[11] Women with a history of gestational diabetes mellitus (GDM) have an approximately 10-fold greater risk of developing T2DM than do those with standard glucose tolerance.[12]

DIAGNOSIS OF DIABETES

DM is diagnosed when fasting plasma glucose (FPG) is ≥126 mg/dl (7.0 mmol/L), random plasma glucose or 2-hour oral

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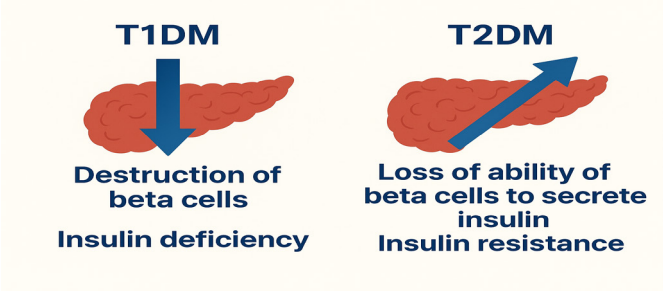

Figure 1. Type 1 and type 2 diabetes mellitus

This figure shows the key differences between type 1 and type 2 diabetes, highlighting autoimmune β-cell destruction in Type 1 and insulin resistance with relative insulin deficiency in type 2.T1DM: Type 1 diabetes mellitus, T2DM: Type 1 diabetes mellitus

glucose tolerance test (OGTT) values are≥200 mg/dl (11.1 mmol/L), or HbA1c is ≥6.5% (48 mmol/mol). FPG defines prediabetes as 100-125 mg/dl (5.6-6.9 mmol/L), 2-hour OGTT values as 140–199 mg/dl (7.8-11.0 mmol/L), or HbA1c values as 5.7-6.4% (39-47 mmol/mol).[13] The 10-year risk of diabetes diagnosis is 88% for individuals with an FPG of 126 mg/dl and an HbA1c percentage of 6.5%. In comparison, this rate is 55% for individuals with FPG levels of 126 mg/dl and an HbA1c percentage of 5.7-6.5%.[14] (Figure 2)

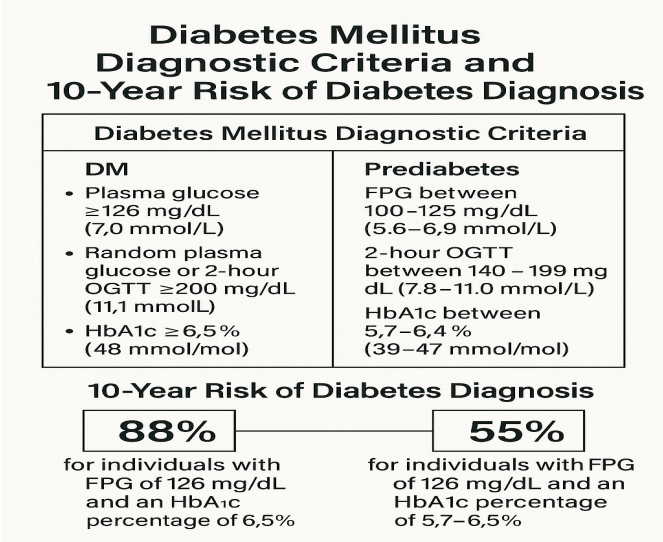

Figure 2. Diagnosis of diabetes

This figure summarizes plasma glucose and HbA1c thresholds for diabetes and prediabetes, along with estimated 10-year progression risks. OGTT: Oral glucose tolerance test, FPG: fasting plasma glucose, HbA1c: hemoglobin A1c

COMPLICATIONS AND PREVALENCE OF DIABETES

Diabetes is one of the fastest-growing global health emergencies of the 21st century. In 2021, approximately 537 million people had diabetes, and this number is expected to reach 643 million by 2030.[15] The prevalence of diabetes is 2% in rural areas and 4.6% in urban areas, which indicates that diabetes is more common in developed countries.[16] However, at the global and national levels, particularly in low- and middle-income countries, there are still significant challenges in understanding the burden of diabetes.[17] With the increase in the number of diabetic patients over time, both the sustainability of health care systems and the quality of life and life expectancy of diabetic patients are negatively affected.[18] Although diabetes is not often recognized as a primary cause of death, it is listed as a cause of death in 69% of death certificates for patients with diabetes (with a 30% rate as the primary cause) owing to the risk of developing heart failure.[19] However, evidence suggests that every 20 mg/dl increase in blood sugar increases the risk of heart failure by 23%, and that even prediabetic blood sugar levels increase this risk.[20] Studies have shown a positive association between diabetes and cardiovascular disease (CVD), chronic lower respiratory disease (CLRD), influenza and pneumonia, and kidney disease-related deaths.[21] Acute complications such as hypoglycemia and diabetic ketoacidosis, inadequate metabolic control can lead to poor growth and early onset of circulatory complications with T1DM.[15] In T2DM, ketoacidosis rarely occurs on its own. When ketoacidosis occurs, it is typically caused by stress from another condition, such as an infection.[6] Hyperglycemia caused by diabetes can lead to long-term damage and dysfunction in various tissues, particularly the eyes, kidneys, nerves, heart, and blood vessels.[22] In a study involving 20,837 patients with T2DM (excluding retinopathy), T2DM was noted to be a risk factor for cortical cataracts and posterior subcapsular cataracts. The findings of this study also emphasize the importance of regular eye examinations for patients with T2DM.[23] In addition, studies have indicated that depressive disorders are frequently observed in patients with T2DM.[24] In a study conducted by Kavak et al.,[25] anxiety was observed in addition to depression in patients with DM. Furthermore, the most common complaints reported by the diabetes patients who participated in this study were fatigue, weakness, and headache.[25] Hypertension and lipoprotein metabolism disorders are also common in individuals with diabetes.[6]

PERIODONTAL DISEASES

Gingivitis

Periodontal health, periodontal diseases, and conditions are further divided into three categories: gingivitis caused by dental biofilm, periodontal diseases not caused by dental biofilm, and periodontal health and gingival health (Table 1).[26] Plaque-induced gingivitis begins with the accumulation of microorganisms on teeth and is characterized by inflammation

Table 1. Classification of periodontal diseases and conditions

Main categories	Subcategories
Periodontal health, gingival diseases and conditions	Periodontal and gingival health
	Gingivitis-dental biofilm-induced
	Gingival diseases-non-dental biofilm-induced
Periodontitis	Necrotizing periodontal diseases
	Periodontitis
	Periodontitis as a manifestation of systemic disease
Other conditions affecting the periodontium	Systemic diseases or conditions affecting the periodontal supporting tissues
	Periodontal abscesses and endodontic-periodontal lesions
	Mucogingival deformities and conditions
	Traumatic occlusal forces
	Tooth and prosthesis-related factors

of the gingiva.[27] Clinically, gingivitis can be accompanied by knife-edge gingival loss, along with papillary blunting, edema, redness of the gingiva, bleeding on probing, and discomfort. Patients may experience complaints such as bleeding in the gingiva, pain, bad breath, and difficulty eating.[28] The primary approach in the treatment of plaque-induced gingivitis is the removal of plaque-retentive factors and the cleaning of dental calculus. Additionally, providing patients with oral hygiene education is essential.[29] Non-plaque-induced gingival diseases may sometimes be early indicators of systemic conditions, whereas in other cases, they remain as localized pathological changes limited to the gingival tissue. The etiology of these diseases includes genetic/developmental disorders, infections, inflammatory and immune diseases, reactive processes, neoplasms, endocrine/metabolic disorders, traumatic lesions, and pigmentation.[30]

Periodontitis

Periodontitis is a chronic, multifactorial inflammatory disease characterized by progressive destruction of the tissues supporting the teeth. According to the classification, periodontitis can be divided into three categories: Necrotizing periodontal disease, periodontitis, and periodontitis, all of which are signs of systemic disease (Table 1).[26] The staging of periodontitis is based on parameters such as severity, complexity, extent, and distribution of the disease (Table 2).[31] The grading of periodontitis is based on primary criteria and grade modifiers. Grade modifiers include risk factors such as smoking status and the presence or absence of diabetes (Table 3).[32]

THE BIDIRECTIONAL RELATIONSHIP BETWEEN DIABETES AND PERIODONTAL DISEASE

It is well known that DM increases the risk of periodontal disease.[3] DM and periodontal disease exhibit overlapping metabolic and inflammatory mechanisms that underlie their widely recognized two-way association.[33] Ongoing Tumor Necrosis Factor (TNF) activity leads to cytokine imbalance, creating a pathway by which bacteria can trigger a more severe inflammatory reaction in individuals with diabetes.[34] Although diabetic plasma produced regular chemotactic activity when stimulated with endotoxin, Polymorphonuclear leukocyte (PMN) chemotactic responses showed no association with gingival inflammation or attachment loss in either diabetic or non-diabetic individuals. These findings indicate a marked reduction in PMN chemotactic function in diabetics with advanced periodontitis and suggest that this dysfunction may play a role in the greater severity of periodontal disease in diabetic patients.[35] The immune alterations in gingival crevicular fluid (GCF) and saliva of T1DM patients-marked by increases in interleukin-1 beta (IL-1β), interleukin-6 (IL-6), interleukin-8 (IL-8), and interleukin-17A (IL-17A) alongside decreases in interleukin-2 (IL-2), interleukin-4 (IL-4), interleukin-12 p70 (IL-12p70), and interferon gamma-induced protein 10 (IP-10)-indicate a substantial pro-inflammatory shift that may enhance periodontal inflammation. The close correspondence between salivary and GCF cytokine profiles highlights saliva's potential as a non-invasive biomarker source, with salivary IL-8 emerging as a particularly promising indicator of periodontal status in T1DM.[36] Studies conducted on both humans and animals have shown that both types of diabetes increase inflammation in periodontal tissues, inhibit new bone formation, and increase receptor activator of nuclear factor kappa-B ligand (ReRANKL) levels.[37] In addition, alterations in bone metabolism markers, including receptor activator of nuclear factor kappa-B Ligand (RANKL) and osteoprotegerin (OPG), play a role in the development of periodontal complications in individuals with T1DM.[38] The receptor for receptor for advanced glycation end Products (RAGE) was initially identified as a signaling receptor that recognizes advanced glycation end products (AGEs)-molecules formed by the non-enzymatic glycation and

Table 2. Periodontitis is classified into stages determined by its severity-based on the amount of interdental clinical attachment loss, radiographic bone destruction, and tooth loss-as well as by the complexity of the condition and the pattern of its extent and distribution.

Category	Feature	Stage I	Stage II	Stage III	Stage IV
Severity	Interdental CAL at site of greatest loss	1-2 mm	3-4 mm	≥5 mm	≥5 mm
	Radiographic bone loss	Coronal third (<15%)	Coronal third (15-33%)	Extending to mid-third of root and beyond	Extending to mid-third of root and beyond
	Tooth loss	No tooth loss due periodontitis	No tooth loss due periodontitis	Tooth loss due to periodontitis of≤4 teeth	Tooth loss due to periodontitis of≥5 teeth
Complexity	Local			In addition to Stage II complexity:	In addition to stage III complexity:
	Probing depth	≤4 mm	≤5 mm	≥6 mm	
	Bone loss	Mostly horizontal	Mostly horizontal	Vertical bone loss ≥3 mm	
	Additional findings			Furcation Class II/III moderate ridge defect	Need for complex rehabilitation due to: masticatory dysfunction secondar occlusal trauma (tooth mobility degree ≥2) severe ridge defect bite collapse, drifting, flaring <20 remaining teeth (10 opposing pairs)
Extent & distribution	Add to stage description	Localized (<30%), generalized, or molar/ incisor pattern	Localized (<30%), generalized, or molar/ incisor pattern	Localized (<30%), generalized, or molar/ incisor pattern	Localized (<30%), generalized, or molar/incisor pattern

CAL: Clinical attachment loss,

Table 3. Periodontitis is graded according to its biological characteristics, taking into account the current or potential risk of rapid progression, the expected response to therapy, and its impact on systemic health.

Category	Subcategory	Feature	Grade A: slow progression	Grade B: moderate progression	Grade C: rapid progression
Primary criteria	Direct evidence of progression	Longitudinal data (radiographic bone loss or CAL)	Evidence of no loss over 5 years	<2 mm over 5 years	≥2 mm over 5 years
	Indirect evidence of progression	% bone loss/age	<0.25	0.25 to 1.0	>1.0
		Case phenotype	Heavy biofilm deposits with low levels of destruction	Destruction commensurate with biofilm deposits	Destruction exceeds expectation given biofilm specific clinical patterns suggestive of periods of rapid progression and/or early onset disease (e.g., molar/incisor patterns; lack of expected response to standard bacterial control therapies)
Grade modifiers	Risk factors	Smoking	Non-smoker	Smoker <10 cigarettes/day	Smoker ≥10 cigarettes/day
		Diabetes	Normoglycemic / no diabetes	HbA1c <7.0% in diabetic patients	HbA1c ≥7.0% in diabetic patients

HbA1c: Hemoglobin A1c, CAL: Clinical attachment level

oxidation of proteins and lipids, which accumulate in diabetic conditions and at sites of inflammation.[39] AGE–RAGE axis is likely an essential pathway of tissue destruction and impaired repair in diabetes-associated periodontitis.[40] Also, there is a clear link between reactive oxygen species (ROS) and the development of T2DM. ROS-induced oxidative stress impairs insulin secretion and action, leading to hyperglycemia, while high glucose further increases ROS production through various signaling pathways.[41] The reduced salivary total antioxidant capacity observed in diabetic individuals with periodontitis reflects a compromised oxidative balance associated with greater inflammatory burden. This inverse relationship suggests that total antioxidant capacity may serve as a valuable indicator of periodontal disease severity in both healthy and diabetic populations.[42] Salvi et al.[43] included nine nondiabetic individuals with pocket depths <4 mm and no attachment loss, as well as nine patients with T1DM who had well- or moderately controlled diabetes. In this study, subgingival plaque collected from each region on the first, 21st, and 35th days was analyzed. Although both groups responded to experimental plaque accumulation with gingival inflammation, patients with T1DM exhibited an earlier and more severe response to similar bacterial stimuli. Kıran et al.[44] conducted a study in which T2DM patients with gingivitis or mild periodontitis underwent periodontal treatment, including scaling and root planing (SRP), without systemic antibiotics. In contrast, a control group with similar conditions received no treatment. Three months after treatment, the prevalence of gingival bleeding, which had been 55%, decreased to 24%, representing a 50% reduction, and HbA1c levels decreased from 7.3% to 6.5%. A study has shown that high glycemic levels cause more severe gingivitis. The correlation coefficient between increases in DM and T2DM was 0.1456054, and a one-unit increase in glycemic level was associated with an approximate 0.00124% increase in gingivitis levels.[45] Periodontal diseases may be a significant risk factor for various systemic diseases.[46] Preliminary evidence suggests that periodontal therapy may have systemic benefits in individuals with diabetes. Based on these findings, the adjunctive use of systemic antibiotics alongside mechanical periodontal treatment may be beneficial, particularly in cases involving gram-negative periodontal pathogens.[47] Furthermore, few existing studies suggest that periodontal therapy may be effective in improving metabolic control by reducing HbA1c and fasting blood glucose (FBG) concentrations in diabetic patients. However, the significance of this improvement is questionable, and further research is needed.[48] The general conclusion that can be drawn is that glycemic control has a positive effect on periodontal status and that periodontal infection management contributes positively to metabolic control in patients with DM. Furthermore, owing to the common pathogenesis of both diseases, the presence of one disease increases the likelihood of early diagnosis of the other disease (Table 4), (Table 5).[49]

Overall, these studies show that diabetic individuals exhibit a stronger inflammatory response and may benefit from periodontal and metabolic outcomes after treatment. However, differences in study design and short follow-up periods limit the strength of these conclusions, highlighting the need for more robust research.

STUDIES CONDUCTED ON DM PATIENTS WITH PERIODONTITIS

Researches Not Including Periodontal Treatment in Patients with DM and Periodontitis

Wu et al.[50] reported in their meta-analysis that the prevalence of T2DM was significantly greater in patients with periodontitis. Compared with patients with T2DM, those with T2DM had 0.61 mm deeper periodontal pockets, 0.89 mm greater attachment loss, approximately two more teeth lost, and significantly worse periodontal status. Cohort studies have shown that the risk of developing periodontitis in T2DM patients may increase by 34%, and in cases of severe periodontitis, this risk may increase by 53%. Abidin et al.[51] conducted a systematic meta-analysis including 11 studies, using data from Kuwait, New York, Belgium, Lithuania, India, Iran, Turkiye, Malaysia, Hong Kong, and Serbia. The sample sizes ranged from 48 to 365 participants, with patient ages ranging from 4 to 18 years. The participants were assessed for

plaque index, gingival index, and clinical attachment loss, and children and adolescents with diabetes had worse periodontal status than their nondiabetic peers did.[51] Lappin et al.[52] conducted a study involving 19 healthy volunteers, 23 patients with periodontitis, 28 patients with T1DM, and 34 patients with both T1DM and periodontitis. The study revealed that plasma IL-8 levels were greater in patients with diabetes and periodontitis than in those with periodontitis alone. Additionally, plasma IL-8 levels were significantly correlated with both clinical probing depth and attachment loss. Tsai et al.[53] conducted a study involving 4,343 individuals aged 45–90 years from the US population. They reported that the prevalence of severe periodontitis (two sites with 6 mm attachment loss and at least one site with a probing pocket depth of 5 mm) was significantly greater in patients with uncontrolled diabetes (FBG≥126 mg/dl) and demonstrated that the likelihood of severe periodontitis was greater in individuals with diabetes. A systematic review and meta-analysis of 10 studies conducted by Abariga et al.[54] examined the relationship between periodontitis and GDM in pregnant women. The results revealed that periodontitis increased the risk of GDM by 66%.54 However, two case-control studies involving children with T1DM reported opposite findings. Children with T1DM were observed to have better oral health than healthy children. Compared with age-matched T1DM control groups, children with T1DM had better oral health, as indicated by lower rates of dental caries and improved gingival status.[55] In a systematic review and meta-analysis of 15 studies by Stöhre et al.,[56] patients with and without DM were compared based on HbA1c and FBG levels. As a result, a bidirectional relationship between periodontal disease and DM has been identified. In a systematic review of 28 studies from 14 countries involving 27,894 patients, Poudel et al.[57] reported that individuals with diabetes have inadequate oral health knowledge, poor oral health behaviors, and fewer dental visits. These people rarely receive oral health education and dental referrals from their care providers. Oral health education provided by diabetes care providers, along with referrals to dentists when necessary, can improve patients' oral health behaviors (**Table 4**), (**Table 5**).[57]

Overall, these studies consistently show that diabetes- across T1DM, T2DM, and GDM- is associated with worse periodontal outcomes and heightened inflammatory profiles, supporting the bidirectional relationship between metabolic control and periodontal health. However, differences in age groups, study designs, diagnostic criteria, and population heterogeneity limit direct comparison. Additionally, several studies rely on cross-sectional data, small samples, or region-specific cohorts, making it difficult to draw causal conclusions. More standardized and longitudinal research is needed to clarify these associations.

Studies Involving Periodontal Treatment in Patients with DM and Periodontitis

Kaur et al.[58] evaluated the impact of nonsurgical periodontal treatment in 100 T2DM patients with chronic periodontitis, grouped by glycemic control. Significant improvements in periodontal parameters and a 10.8% reduction in HbA1c were observed at 3 and 6 months, respectively. However, patients with poor baseline glycemic control showed less clinical improvement than nondiabetic individuals did. Another systematic review examined 35 studies involving 3.249 participants in periodontal treatment or control groups. The HbA1c levels of participants who were diagnosed with T2DM and periodontitis and who underwent periodontal treatment were 0.43% lower than those of the control group after 3 to 4 months.[59] Elnour et al.'s[60] meta-analysis, which included 1469 patients and 11 studies, revealed a slight association between periodontal treatment and glycemic control in patients with uncontrolled diabetes. Periodontitis treatment was shown to reduce HbA1c in diabetic patients with poor glycemic control. In a systematic review and meta-analysis of nine studies by Baeza et al.,[61] SRP in patients with periodontitis was determined to be effective in improving metabolic control and reducing systemic inflammation in those with T2DM. In a randomized controlled trial, the effects of non-surgical periodontal therapy on oral hygiene behaviors, periodontal status, and oral health-related outcomes in T2DM patients were evaluated. The participants were randomly assigned to an intervention group (n=35) or a control group (n=33). After one month, the intervention group showed greater improvements in probing depth and clinical attachment level. The treatment had a positive impact on oral health-related quality of life and self-care behaviors.[62] Bian et al.[63] randomly assigned 72 patients with T2DM and moderate-to-severe periodontitis into four groups: combined SRP with curettage, SRP alone, curettage alone, and cleaning. Clinical parameters and inflammatory markers [Tumor Necrosis Factor-alpha (TNF-α) and (hs-CRP)] were measured before and after treatment. After 3 months, the combined group showed the greatest improvement across all periodontal indices, and all groups exhibited substantial reductions in inflammatory markers, with TNF-α and high-sensitivity C-reactive protein (hs-CRP) levels lowest in the combined treatment group (**Table 4**), (**Table 5**).[63]

Overall, these studies demonstrate that nonsurgical periodontal therapy can improve periodontal parameters and modestly enhance glycemic control in patients with T2DM, although the magnitude of HbA1c reduction varies. Combined or intensive treatments appear to yield greater clinical and inflammatory benefits. However, differences in treatment protocols, follow-up durations, baseline glycemic control, and sample sizes limit the comparability of results. Most studies also lack long-term data, making it difficult to determine the sustained metabolic impact of periodontal therapy.

Table 4. Clinical and animal studies

Authors (year)	n/population	Age	Case definition	Study design	Intervention / control	Follow-up	Results	Conclusion
Naguib et al.[34] (2004)	Diabetic vs. non-diabetic mice	Adult mice	Experimental diabetes+bacterial stimulus	Animal model	No intervention	3 days	The persistent expression of TNF-alpha, the chemokines MCP-1, and MIP-2	Cytokine dysregulation associated with prolonged TNF expression enables bacteria to induce a more damaging inflammatory response in diabetic individuals.
Mendes-Frias et al.[36] (2025)	Adults with T1DM and metabolically healthy controls (510.000 individuals)	Adults	Assesses GCF	Observational case-control study	No intervention	Baseline	Higher IL-6 and IL-8 concentrations and strong correlations with key pro-inflammatory cytokines	Salivary IL-8 emerged as the most promising biomarker for distinguishing periodontal status in T1DM patients.
Pendyala et al.[42] (2013)	T2DM patients and healthy subjects with and without periodontal disease (n=120)	40–60 years	Measured salivary total antioxidant capacity	Case-control	No intervention	Baseline	The mean salivary total antioxidant capacity was lowest in diabetic patients with periodontitis.	Total antioxidant capacity is inversely correlated with inflammation severity and can serve as a valuable marker of periodontitis in both healthy and diabetic patients.
Salvi et al.[43] (2005)	T1DM and non-diabetics (n=18)	16-35 years	T1DM experimental gingivitis	A controlled clinical and microbiological study	21-day plaque accumulation	Days 0, 21, and 35	The mean DNA probe counts of the red and orange complex species increased significantly between days 0 and 21, and decreased significantly between days 21 and 35 in both groups.	Both diabetics and non-diabetics react to experimental plaque accumulation with gingival inflammation. Type 1 diabetics, however, develop an earlier and higher inflammatory response to a comparable bacterial challenge.
Kıran et al.[44] (2005)	T2DM adults (N=44)	31–79 years	T2DM+chronic periodontitis	Randomized clinical trial	The treatment group received NSPT, whereas the control group received no periodontal treatment.	Baseline, 1, and 3 months	HbA1c levels in the treatment group decreased significantly, whereas the control group showed a slight but insignificant increase for this parameter.	NSPT is associated with improved glycemic control in patients with T2DM.
Lappin et al.[52] (2015)	Adults with T1DM (N:104) 19 healthy volunteers, 23 patients with periodontitis, 28 patients with T1DM, and 34 patients with T1DM and periodontitis.	Adults	T1DM susceptibility	Cross-sectional	No intervention	Baseline	Patients with diabetes and periodontitis had higher plasma levels of IL-8 than patients with periodontitis alone.	Elevated plasma IL-8 levels may contribute to cross-susceptibility between periodontitis and T1DM. *P. gingivalis* LPS and AGE in combination caused significantly greater expression of IL-6, IL-8, and CXCL5 from THP-1 monocytes and OKF6/TERT-2 cells than LPS alone.
Tsai et al.[53] (2002)	US adults with T2DM (N:4343)	45–90 years	T2DM±severe PD	Cross-sectional epidemiologic	No intervention	Baseline	Individuals with poorly controlled DM had a significantly higher prevalence of severe periodontitis than those without DM.	An association between poorly controlled T2DM and severe periodontitis.
AlMutairi et al.[55] (2020)	Saudi children with T1DM (N:40)	12-15 years	T1DM oral/periodontal health	Cross-sectional	No intervention	Baseline	The differences were statistically significant only in the oral health domain.	Children with T1DM had better oral health, as assessed by dental caries and gingival status, compared with their age-matched controls.
Kaur et al.[58] (2015)	Patients with type 2 diabetes and chronic periodontitis (n:100)	45-60 years	T2DM+PD, stratified by HbA1c	Prospective interventional	NSPT (n:50), no prevention (n:50)	Baseline, 3, and 6 months.	Improvements in the gingival index and bleeding on probing were greater in the nondiabetic participants and in the treated patients with reasonable glycemic control than in those with poor glycemic control.	NSPT improved glycemic control and periodontal health in patients with T2DM. however, patients with poor baseline glycemic control showed less clinical improvement than those without diabetes or those with reasonable glycemic control.
Hsu et al.[62] (2021)	Adults with T2DM	35-65 years	T2DM+ periodontitis	RCT	NSPT±CHW support	1, 3, and 6 months	The patients in the experimental group were more likely to change their oral self-care behaviors than patients in the control group.	The NSPT with CHW strategy had a positive effect on 1-month periodontal treatment outcomes, long-term OHQoL and oral self-care behaviors in patients with T2DM.
Bian et al.[63] (2021)	Patients with T2DM complicated with moderate-to-severe chronic periodontitis (n=72)	35-72 years	Moderate–severe periodontitis	RCT	Root planing and periodontal curettage combined group (n=18), root planing group (n=18), periodontal curettage group (n=18) and cleansing group (n=18).	3 months	TNF-α and hs-CRP in the combined group were lower than those in the root planing group, periodontal curettage group. Group and cleansing group, and those in the root planing group and periodontal curettage group were significantly lower than those in the cleansing group	The combination therapy of periodontal curettage and root planing exerted beneficial effects on moderate-to-severe chronic periodontitis in patients with type 2 diabetes mellitus, which holds the potential to maintain blood glucose levels and improve patients' quality of life.

TNF-alpha: Tumor necrosis factor-alpha, MCP-1: Monocyte chemoattractant protein-1, MCP-2: Monocyte chemoattractant protein-2, DM: Diabetes mellitus, T1DM: Type 1 diabetes mellitus, T2DM: Type 2 Diabetes Mellitus, IL-6: Interlökin 6, IL-8: Interlökin 8, SRP: Scaling and root planning, HbA1c: Hemoglobin A1c, NSPT: Nonsurgical periodontal treatment, *P. gingivalis: Porphyromonas gingivalis*, LPS: lipopolysaccharide, AGE: Advanced glycation end products, CXCL5: C-X-C motif chemokine ligand 5, THP-1: Human acute monocytic leukemia cell line, OKF6/TERT-2: Oral keratinocyte field 6/ telomerase reverse transcriptase-2, PD: Probing depth, CHW: Chlorhexidine glukonat, and hs-CRP: High-sensitivity C-reactive protein.

Table 5. Systematic review and meta-analyses

Authors (year)	n/population	Age (years)	Included studies (n)	Case definition	Intervention/ control	Follow-up	Results	Conclusion
Zainal Abidin et al.[51] (2021)	Diabetic and non-diabetic patients	4-18 years	11 studies	T1DM/T2DM periodontal status	N/A	N/A	PI, GI, CAL and PPD were significantly higher in DM children than in non-DM children.	Showed significant differences in PI, GI, PD and CAL between the two groups, favouring non-DM children.
Vlachou et al.[38] (2024)	T1DM clinical studies	Adults	22 studies	T1DM with periodontal disease	N/A	N/A	Molecular mechanisms underlying this association involve oxidative stress and dysregulated host immune responses, mediated by inflammatory cytokines such as IL-6, IL-8, and MMPs.	Preventive measures to manage periodontal complications in T1DM patients may improve overall health outcomes.
Corbella et al.[48] (2013)	Adults with DM & periodontitis	Adults	15 studies	T1DM/T2DM + periodontitis	NSPT vs control	3-4 and 6 months.	A reduction of -0.38% after 3-4 months and of -0.31% after 6 months of follow-up was found for HbA1c, favoring the treatment group.	NSPT improves metabolic control in patients with both periodontitis and diabetes.
Wu et al.[50] (2020)	Adults (n:53)	Adults	53 studies	T2DM & periodontitis	N/A	N/A	T2DM could elevate the risk of developing periodontitis by 34%.	Both dentists and physicians need to be aware of the strong connection between periodontitis and T2DM.
Abariga & Whitcomb[54] (2016)	Pregnant women	Adults	10 studies	Periodontitis & GDM	N/A	N/A	More than 2-fold increased odds of GDM among women with periodontitis	Periodontitis is associated with a statistically significant increased risk for GDM compared to women without periodontitis.
Stöhr et al.[56] (2021)	DM with periodontitis	Adults	15 studies	Periodontitis-DM	N/A	N/A	A positive bidirectional association between periodontal disease and diabetes mellitus	Underline the need for screening of patients with periodontitis regarding diabetes mellitus and vice versa.
Simpson et al.[59] (2022)	Adults with T1DM and T2DM (N:3249)	Adults	35 RCTs	Diabetes+ periodontitis	NSPT vs minimal care	3–12 months	Treating periodontitis leads to an HbA1c reduction of about 0.43% at 3–4 months, 0.30% at 6 months, and 0.50% at 12 months.	Periodontal treatment using subgingival instrumentation improves glycemic control in people with both periodontitis and diabetes by a clinically significant amount when compared to no treatment or usual care.
Elnour & Mirghani[60] (2023)	Adults with DM (N: 1469)	Adults	11 studies	T1DM/T2DM with periodontitis	Surgical therapy+NSPT	3-12 months	Periodontitis treatment improved the HbA1c among patients with diabetes and poor glycemic control.	Screening for this common disease is essential in diabetes holistic care.
Baeza et al.[61] (2020)	Adults with T2D and periodontitis	Adults	9 studies	T2DM+periodontitis	Nonsurgical therapy± antimicrobials	3-6 months	SRP was effective in reducing HbA1c	SRP has an impact on metabolic control and the reduction of systemic inflammation in patients with T2D.

DM: Diabetes mellitus, T1DM: Type 1 diabetes mellitus, T2DM: Type 2 diabetes mellitus, PI: Plaque index, GI: Gingival index, CAL: clinical attachment loss, PPD: Periodontal probing depth, IL-6: Interlökin 6, IL-8: Interlökin 8, MMP: Matrix metalloproteinases, HbA1c: Hemoglobin A1c, NSPT: Nonsurgical periodontal treatment, GDM: Gestational diabetes mellitus, SRP: Scaling and root planning.

CONCLUSION

There is a well-established reciprocal association between DM and periodontal disease, with a substantial impact on both the course of the disease and the patient's general health. Research indicates that while active periodontal disease might worsen systemic inflammation and have a detrimental impact on metabolic outcomes, poor glycemic control increases periodontal inflammation. Improving the long-term prognosis for those affected requires an understanding of this interplay. Clinicians should emphasize the importance of rigorous glycemic control and integrate routine periodontal assessments into the management of patients with diabetes to ensure the highest possible standard of care. Frequent periodontal therapy, patient education on oral hygiene, and close coordination between dental and medical practitioners can all help reduce problems and improve quality of life. Therefore, limiting the advancement of the disease and promoting systemic and periodontal health requires a coordinated, interdisciplinary approach.

ETHICAL DECLARATIONS

Peer Review Process

This review was externally peer-reviewed.

Conflict of Interest

The authors declare no conflicts of interest.

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