ORIGINAL ARTICLE

EXPERIMENTAL GINGIVITIS IN TYPE 1 DIABETIC PATIENTS: A CONTROLLED CLINICAL AND MICROBIOLOGICAL STUDY

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Abstract: *Background*: Type 1 diabetes mellitus (T1DM) is a chronic autoimmune disorder that alters the host's immune and inflammatory responses, potentially enhancing susceptibility to periodontal inflammation and disease. Experimental gingivitis models offer a controlled framework to evaluate host–microbiota interactions under plaque-induced challenges. Aim: This study aimed to evaluate and compare the clinical and microbiological responses to experimentally induced gingivitis between individuals with well-controlled type 1 diabetes and non-diabetic controls. *Materials and Methods*: A total of 155 volunteers (78 diabetics, 77 non-diabetics), aged 18–35 years, were enrolled in a 35-day controlled clinical trial. Following a 3-week period of no oral hygiene (Days 0–21) and a 2-week period of resumed hygiene (Days 21–35), clinical indices (Plaque Index—PI, Gingival Index—GI, and percentage of bleeding sites with GI ≥ 2) were recorded at six sites per tooth. Subgingival plaque samples were collected and analyzed for bacterial complexes using checkerboard DNA–DNA hybridization. *Results*: Both groups exhibited significant increases in PI and GI during plaque accumulation. However, diabetics developed an earlier and more pronounced inflammatory response ($p < 0.01$), with higher percentages of bleeding sites at Days 7 and 21. Red and orange bacterial complexes increased significantly during plaque accumulation, then decreased after oral hygiene reinstitution. *Conclusion*: Type 1 diabetic patients exhibited a hyperinflammatory response to bacterial plaque challenge compared with non-diabetic controls, despite similar plaque levels. These findings reinforce the critical importance of meticulous oral hygiene in diabetic individuals to mitigate periodontal risk.

Keywords: experimental gingivitis, type 1 diabetes mellitus, gingival inflammation, host response, subgingival microbiota

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1. Introduction

Periodontal diseases are chronic inflammatory conditions initiated by bacterial biofilms but significantly modified by the host's immune response. While plaque accumulation serves as the primary etiologic factor, the severity and rate of disease progression are largely dependent on host susceptibility. Among systemic conditions influencing periodontal health, diabetes mellitus has emerged as one of the most influential.

Type 1 diabetes mellitus (T1DM) is characterized by autoimmune destruction of pancreatic β-cells, resulting in insulin deficiency and hyperglycemia. The resulting metabolic imbalance contributes to oxidative stress, accumulation of advanced glycation end-products (AGEs), and upregulated pro-inflammatory cytokine expression (IL-1β, TNF-α, IL-6), which may amplify gingival inflammation.

Several epidemiological studies have demonstrated an increased prevalence and severity of periodontal diseases among diabetic individuals (Hugoson et al., 1989; Grossi et al., 1994). However, results have varied, particularly when considering differences in metabolic control, age, and study design. The experimental gingivitis model, introduced by Löe et al. (1965), provides a unique opportunity to observe the cause–effect relationship between plaque accumulation and gingival inflammation in a controlled manner.

The current study aimed to evaluate clinical and microbiological changes during experimental gingivitis in type 1 diabetic subjects compared with non-diabetic controls, using a standardized 35-day protocol. The study hypothesized that diabetic subjects would exhibit an earlier and more pronounced inflammatory response to a comparable bacterial challenge.

2. Materials and method

2.1 *Study design*

A controlled 35-day experimental gingivitis protocol was implemented, consisting of:

- Days 0–21: abstention from all oral hygiene (plaque accumulation phase)
- Days 21–35: reinstitution of optimal oral hygiene (healing phase)

All clinical and microbiological assessments were performed at baseline (Day 0), during the accumulation phase (Days 7, 14, 21), and after healing (Day 35).

2.2 *Participants*

A total of 155 volunteers (78 type 1 diabetics, 77 non-diabetic controls) were recruited.

Inclusion criteria: 18–35 years old, ≥ 24 teeth, probing depth < 4 mm, good systemic health, and for diabetics, HbA1c ≤ 8.5 %.

Exclusion criteria: smoking > 5 cigarettes/day, antibiotic use within 3 months, pregnancy, chronic medication influencing gingival status, or active caries.

Ethical approval was obtained from the Institutional Ethics Committee of UMF Craiova Nr.411/04.11.2025. Written informed consent was secured from all participants.

2.3 *Clinical assessments*

Description of Clinical Indices Used

The Plaque Index (PI) used in this study followed the Silness and Löe criteria, evaluating the thickness of dental plaque at the

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cervical third of the tooth. Scores ranged from 0 (no plaque) to 3 (abundance of soft deposits visible to the naked eye).

The Gingival Index (GI) was recorded according to the Löe and Silness system, assessing gingival color, edema, and bleeding tendency after gentle probing. GI values ranged from 0 (normal gingiva) to 3 (severe inflammation with a tendency to spontaneous bleeding).

Additionally, the percentage of gingival sites with GI ≥ 2 was calculated at each time point to quantify the extent of clinically significant bleeding inflammation.

These indices were recorded at six sites per tooth, using standardized probing techniques with a UNC-15 periodontal probe.

PI and GI were measured at six sites per tooth using calibrated probes (UNC-15). Inter-examiner reliability ($\kappa > 0.85$) was established before the study. The percentage of sites with GI ≥ 2 was used to quantify the extent of bleeding inflammation.

2.4 *Microbiological sampling*

Subgingival samples were collected from distolingual sites of first molars in each quadrant using sterile Gracey curettes. Samples were pooled and analyzed for 40 bacterial species by checkerboard DNA–DNA hybridization, focusing on:

- Red complex (Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia)
- Orange complex (Fusobacterium nucleatum, Prevotella intermedia)
- Blue complex (Actinomyces naeslundii, Streptococcus mitis)

2.5 *Statistical analysis*

Data were analyzed using SPSS v27. Continuous variables were expressed as mean ± SD. Within-group changes were evaluated using paired t-tests; between-group differences were tested with unpaired t-tests and Mann–Whitney U tests. The significance level was set at $p < 0.05$.

3. Results

Table 1 presents the demographic characteristics of the study population. The diabetic and control groups were well balanced with respect to age, sex distribution, and number of teeth present, with no significant differences between groups. As expected, HbA1c levels were significantly higher in the diabetic group ($p < 0.001$), confirming distinct metabolic profiles between the cohorts

Table 1. Demographic characteristics.

Variable	Diabetics (n = 78)	Controls (n = 77)	p-value
Age (years, mean ± SD)	26.4 ± 4.8	25.9 ± 5.2	0.57
Sex (M/F)	32/46	31/46	0.94
HbA1c (%)	8.1 ± 0.7	5.3 ± 0.4	< 0.001
Teeth present	27.2 ± 1.3	27.4 ± 1.2	0.63

No significant baseline differences were found between the groups except for HbA1c levels.

Table 2 illustrates the evolution of plaque accumulation and gingival inflammation over the 35-day study period. Both groups

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exhibited sharp increases in PI and GI during the no-hygiene phase (Days 0–21). However, diabetic participants consistently demonstrated higher GI values and a greater percentage of bleeding sites from Day 7 onward ($p < 0.05$). After reintroduction of oral hygiene (Day 35), all indices returned near baseline levels.

Table 2. Mean Plaque Index (PI) and Gingival Index (GI).

Day	Group	PI (mean ± SD)	GI (mean ± SD)	% GI ≥ 2 ± SD
0	Diabetics	0.18 ± 0.10	0.25 ± 0.11	1.5 ± 1.0
	Controls	0.17 ± 0.08	0.22 ± 0.09	1.1 ± 0.8
7	Diabetics	0.85 ± 0.23	0.90 ± 0.20	12.5 ± 4.3
	Controls	0.80 ± 0.20	0.78 ± 0.22	8.3 ± 3.6
14	Diabetics	1.40 ± 0.27	1.30 ± 0.25	27.8 ± 8.5
	Controls	1.32 ± 0.29	1.10 ± 0.24	19.2 ± 6.7
21	Diabetics	2.05 ± 0.21	1.55 ± 0.28	41.0 ± 9.8
	Controls	1.92 ± 0.25	1.28 ± 0.23	26.2 ± 7.9
35	Diabetics	0.20 ± 0.10	0.30 ± 0.12	1.9 ± 0.9
	Controls	0.18 ± 0.09	0.25 ± 0.10	1.3 ± 0.8

Both groups showed significant PI and GI increases during plaque accumulation ($p < 0.001$). The diabetics demonstrated higher GI and bleeding percentages from Day 7 onward ($p < 0.05$).

Table 3 shows the relative proportions of major bacterial complexes at baseline, peak inflammation (Day 21), and recovery (Day 35). Both diabetics and controls showed significant increases in red and orange complexes during plaque accumulation, followed by reductions after oral hygiene reinstitution. Blue complex species decreased during inflammation and rebounded after hygiene, reflecting shifts between pathogenic and health-associated microbiota.

Table 3. Relative proportion of bacterial complexes.

Complex	Baseline (%)	Day 21 (%)	Day 35 (%)	Change ($p < 0.05$)
Red – Diabetics	5.2 ± 1.1	17.5 ± 3.2	6.0 ± 1.4	↑ Day 0–21; ↓ 21–35
Red – Controls	4.9 ± 1.0	14.2 ± 2.8	5.1 ± 1.3	↑ Day 0–21; ↓ 21–35
Orange – Diabetics	12.3 ± 2.4	24.1 ± 4.2	14.5 ± 3.0	↑ Day 0–21; ↓ 21–35
Blue – Both	22.1 ± 3.8	14.2 ± 2.5	23.8 ± 3.9	↓ Day 0–21; ↑ 21–35

The bacterial composition shifted toward pathogenic complexes during the plaque accumulation phase, followed by a return to health-associated flora after oral hygiene reinstitution.

Figure 1 illustrates the progressive increase in PI and GI during the 21-day plaque accumulation period, followed by a return toward baseline after oral hygiene was resumed. Diabetic subjects showed an earlier and more pronounced rise in GI relative to controls, indicating enhanced inflammatory susceptibility.

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Figure 2 depicts the percentage of gingival sites with bleeding (GI ≥ 2). The diabetic group exhibited significantly higher bleeding proportions at Days 7, 14, and 21 ($p < 0.01$), supporting the observation of a hyperinflammatory response even in the presence of similar plaque levels.

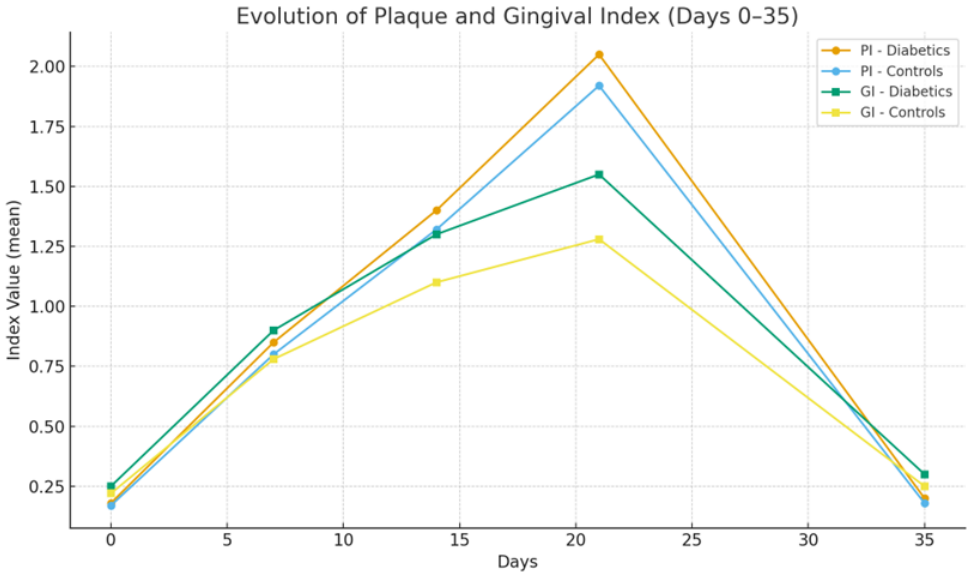

Figure 1. Evolution of Plaque and Gingival Index (Days 0–35).

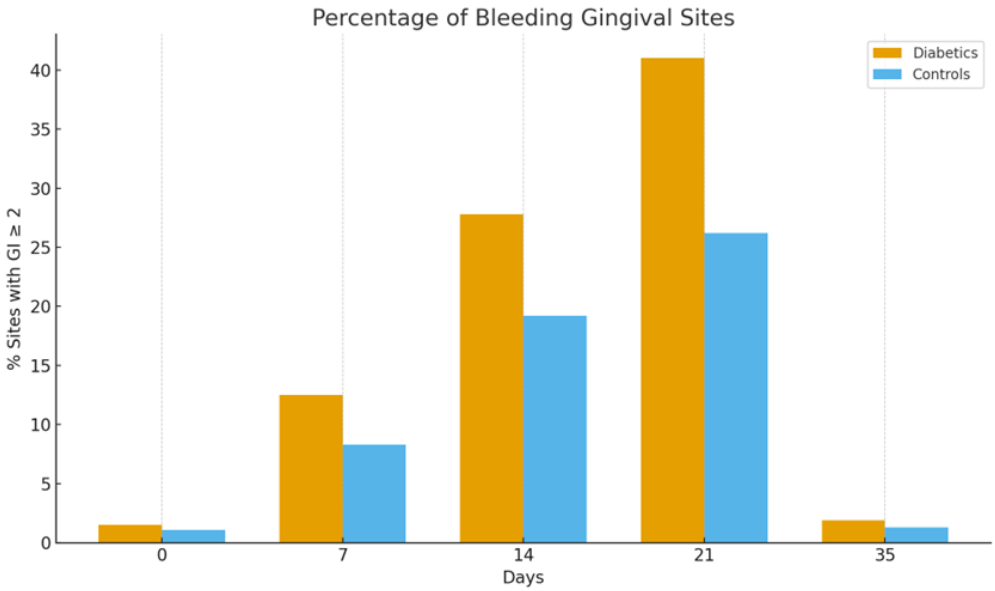

Figure 2. Percentage of Bleeding Gingival Sites.

Figure 3 shows the temporal changes in bacterial complexes within the diabetic group. During plaque accumulation, red and orange complexes increased substantially, consistent with a pathogenic shift. After reinstitution of hygiene, these levels decreased, while blue

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complex bacteria—associated with periodontal health—recovered.

Figure 4 presents a radar chart comparing key inflammatory parameters at Day 21 between diabetics and controls. Diabetic subjects displayed consistently higher values across all indices, corroborating their amplified inflammatory response to plaque challenge.

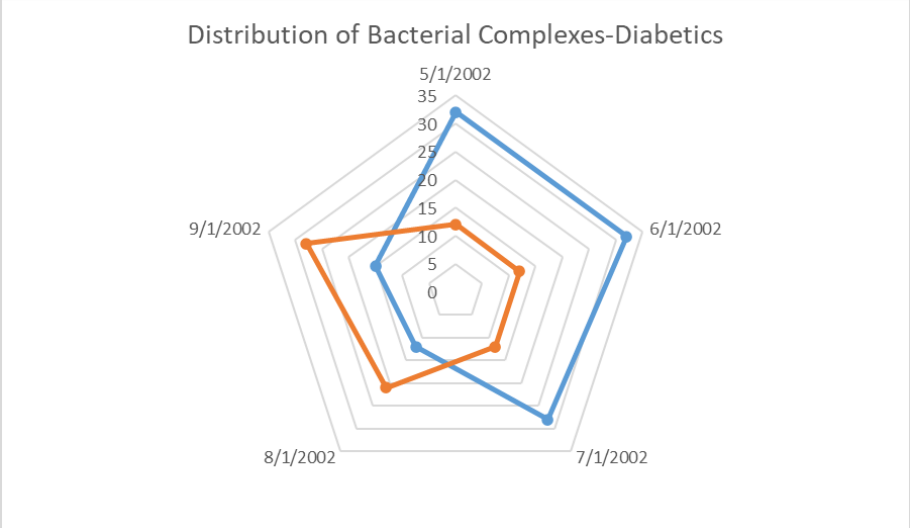

Figure 3. Distribution of Bacterial Complexes – Diabetic Group.

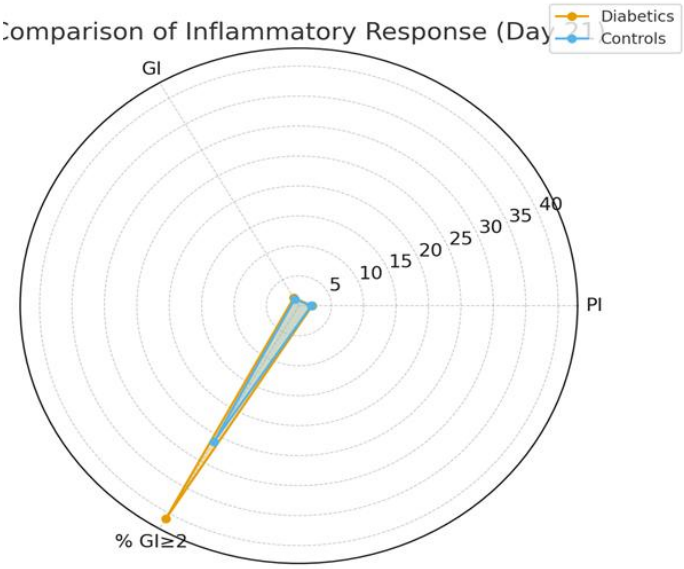

Figure 4. Radar Chart: Comparative Inflammatory Response at Day 21.

4. Discussion

The present controlled experimental gingivitis study showed that young adults with type 1 diabetes mellitus (T1DM), despite good-to-moderate metabolic control, develop an earlier and more pronounced gingival

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inflammatory response to standardized plaque accumulation than non-diabetic controls, even in the presence of comparable plaque levels. Diabetic subjects exhibited higher Gingival Index (GI) values and a significantly greater percentage of bleeding sites from Day 7 onwards, while Plaque Index (PI) followed a similar trajectory in both groups. These findings support the concept that diabetes primarily acts as a modifier of the host response, rather than changing the quantity of bacterial challenge.

Our results are in line with previous experimental gingivitis models in T1DM. Salvi et al. (2005) reported that, under controlled plaque accumulation, diabetic patients developed more pronounced gingival inflammation and bleeding compared with non-diabetic subjects, confirming an exaggerated inflammatory response to a similar biofilm challenge. Likewise, Giannobile et al. (2010) observed elevated levels of pro-inflammatory biomarkers in gingival crevicular fluid during experimental gingivitis in T1DM patients compared with systemically healthy controls, further supporting the notion of a hyper-reactive periodontal inflammatory phenotype in diabetes. The present study corroborates these observations at the clinical level, showing that, from early time points, diabetic individuals cross the threshold into clinically evident inflammation and bleeding more rapidly than matched controls.

The clinical pattern observed here also aligns with epidemiological and clinical evidence indicating a higher prevalence and severity of periodontal disease in diabetes. Several recent reviews and umbrella analyses have reinforced the bidirectional relationship between periodontal disease and diabetes, highlighting diabetes as a major risk factor for gingivitis and periodontitis, and periodontal inflammation as a contributor to poorer glycemic control (Costa et al., 2023; Păunică et al., 2023; Di Domenico et al., 2023; "An Umbrella Review of the Association Between Periodontal Disease and Diabetes Mellitus," 2024; "Periodontitis: an often-neglected complication of diabetes," 2024). These works converge on the idea that even in relatively young populations, diabetes predisposes to an exaggerated periodontal inflammatory response, which is consistent with the present findings in 18–35-year-old participants.

From a microbiological perspective, both diabetics and controls in our study showed a shift toward a more pathogenic biofilm during plaque accumulation, with significant increases in red and orange complexes and a concomitant decline in health-associated blue complex species. After oral hygiene reinstitution, red and orange complexes decreased, while blue complex bacteria rebounded toward baseline.

This dynamic is comparable to earlier observations in experimental gingivitis models that demonstrated a transition from gram-positive, health-associated flora toward gram-negative anaerobes as plaque matures (Ximénez-Fyvie et al., 2000; Salvi et al., 2005). The key point is that, although the microbiological trends were broadly similar in diabetics and controls, the clinical inflammatory expression was consistently greater in T1DM subjects, strengthening the argument that the host response is the principal differentiating factor.

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Recent microbiome studies in children and adolescents with T1DM also support a pattern of dysbiosis associated with diabetes. Selway et al. (2023), Carelli et al. (2023), and Chakraborty et al. (2021) reported alterations in oral microbial communities and early markers of periodontal disease in young individuals with T1DM, often linked to glycemic control and inflammatory status. These studies indicate that T1DM may prime the oral environment for earlier and more pronounced inflammatory responses to plaque. Our data extend these findings to an adult population under standardized plaque challenge and suggest that, even with controlled experimental conditions and exclusion of overt periodontitis, diabetes still amplifies gingival inflammation.

The biological mechanisms underlying this hyperinflammatory gingival response in T1DM are multifactorial. Chronic hyperglycemia promotes formation of advanced glycation end products (AGEs) and engagement of the AGE–RAGE axis on endothelial cells and monocytes, enhancing NF-κB activation and upregulating pro-inflammatory cytokines such as IL-1β, TNF-α, and IL-6. Neutrophil chemotaxis and phagocytosis are frequently impaired, while oxidative stress is increased, and collagen turnover and repair are delayed. These mechanisms have been well documented in both experimental and clinical studies (Sereti et al., 2021; "Diabetes mellitus promotes susceptibility to periodontitis—novel insights," 2023; Duda-Sobczak et al., 2018). In the context of the present study, they offer a plausible explanation for why, under comparable plaque conditions, diabetic subjects progressed more rapidly to higher GI scores and higher proportions of bleeding sites than their non-diabetic counterparts.

Our findings are also consistent with the growing body of evidence that periodontal therapy can contribute to modest improvements in glycemic control. Several recent umbrella and systematic reviews reported that non-surgical periodontal treatment is associated with small but clinically relevant reductions in HbA1c levels in patients with diabetes (Di Domenico et al., 2023; "Effect of Periodontal Treatment in Patients with Periodontitis and Diabetes Mellitus," 2023; "The role of periodontal treatment on the reduction of hemoglobin A1c," 2025). In this context, the hyperinflammatory periodontal response observed in our T1DM group further supports the inclusion of periodontitis as a complication of diabetes and underscores the importance of periodontal care within comprehensive diabetes management.

Compared with cross-sectional and observational studies that often include broad age ranges and variable metabolic control, the strengths of the present investigation include a relatively homogeneous, young adult population, strict inclusion/exclusion criteria, and a well-controlled experimental gingivitis protocol with standardized plaque accumulation and healing phases. The use of validated clinical indices, calibrated examiners, and a microbiological assessment focusing on recognized bacterial complexes provides a robust framework for evaluating both clinical and microbial responses.

However, several limitations should be acknowledged. First, the study focused on young adults with relatively good-to-moderate metabolic control (HbA1c ≤ 8.5%),

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and the results may not be generalizable to older individuals, those with long-standing diabetes, or poorly controlled glycemia, where the inflammatory response may be even more pronounced. Second, the observation period was limited to 35 days, which captures short-term gingival changes but does not address long-term progression to periodontitis. Third, subgingival plaque sampling was restricted to specific molar sites and pooled for analysis, which may underestimate site-specific variability in microbial composition. Finally, no biochemical or molecular markers (e.g., cytokines, matrix metalloproteinases, oxidative stress markers) were collected, limiting the ability to directly correlate clinical findings with underlying immune and inflammatory mechanisms.

Future research should build on these findings by incorporating longitudinal designs that track periodontal and metabolic outcomes over longer periods, including patients with varying degrees of glycemic control and diabetes duration. Adding gingival crevicular fluid and serum biomarkers, as suggested by previous work (Giannobile et al., 2010; Sereti et al., 2021), would allow a deeper understanding of the molecular pathways linking T1DM and periodontal inflammation. Moreover, studies integrating detailed microbiome profiling and host-response analyses could help identify specific microbial–host signatures that characterize high-risk diabetic phenotypes.

From a clinical standpoint, the present results reinforce several key messages. First, even in young adults with T1DM and without established periodontitis, gingival tissues respond more aggressively to plaque accumulation, highlighting the necessity of meticulous daily oral hygiene and regular professional prophylaxis. Second, early identification and management of gingival inflammation in diabetic patients may help prevent progression to destructive periodontal disease and may contribute indirectly to better metabolic control. Third, the data support closer interprofessional collaboration between diabetologists and dental professionals, with routine periodontal screening and preventive counseling integrated into diabetes care pathways. Within the limitations of this study, it can therefore be concluded that T1DM significantly enhances susceptibility to plaque-induced gingival inflammation, confirming diabetes as a potent modifier of the periodontal host response. These findings complement and extend existing evidence, strengthening the rationale for recognizing periodontal disease as a relevant complication of diabetes and for prioritizing periodontal prevention and treatment in this population.

5. Conclusions

Within the limitations of this study, it can be concluded that both diabetic and non-diabetic individuals respond to bacterial plaque accumulation with gingival inflammation. However, patients with type 1 diabetes exhibit an earlier onset and greater severity of gingival inflammation, despite comparable plaque levels. The findings highlight diabetes as a potent modifier of host response, emphasizing the need for rigorous preventive and therapeutic periodontal care in this population.

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Author contributions

Authors read and approved the final manuscript. All authors have equally contributed to this work.

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Conflict of interest statement

The authors declare no conflicts of interest concerning this study.

Data availability statement

Will be provided on request.

Ethics statement

This study was approved by the Ethics Committee of the University of Medicine and Pharmacy of Craiova (approval data no. 411/04.11.2025).

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