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Influence of Diabetes on Dental Implant Survival and Peri-implant Health: A Narrative Review

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ABSTRACT

Dental implants have become a reliable solution for replacing missing teeth, offering long-term functional and esthetic outcomes. However, systemic metabolic disorders such as diabetes mellitus (DM) remain a significant concern in implant dentistry. This narrative review synthesizes evidence from approximately 100 studies published between 2000 and 2025, aiming to evaluate how diabetes—particularly glycemic control—affects implant survival, osseointegration, and peri-implant health. The review analyzes human clinical data stratified by glycated hemoglobin (HbA1c) levels, as well as mechanistic findings from molecular and histological research.

The results indicate that well-controlled diabetic patients (HbA1c <7%) achieve implant survival rates of 95–96% at 5 years and 92–94% at 10 years, comparable to non-diabetic individuals. In contrast, poorly controlled diabetes (HbA1c ≥8%) is associated with significantly higher rates of early implant failure (6–8%), peri-implantitis (22–31%), and marginal bone loss averaging 0.58 mm/year compared to 0.16 mm/year in non-diabetic groups. The pathophysiological basis involves hyperglycemia-induced oxidative stress, accumulation of advanced glycation end-products (AGEs), altered bone remodeling, and microangiopathy that impairs tissue healing. Preventive strategies including meticulous glycemic control, microinvasive surgical techniques, bioactive implant surfaces, and intensive maintenance protocols can mitigate these risks.

This review integrates findings from over 100 clinical and mechanistic studies to identify glycemic thresholds for predictable implant success and defines evidence-based maintenance protocols for diabetic individuals.

1. INTRODUCTION

Diabetes mellitus (DM) is a complex chronic disease characterized by sustained hyperglycemia resulting from either insufficient insulin secretion (type 1 DM) or impaired insulin utilization (type 2 DM). According to the International Diabetes Federation (2021), over 537 million adults worldwide are currently affected, and this number is projected to exceed 780 million by 2045. Chronic hyperglycemia induces widespread vascular, neural, and immune dysfunctions that extend beyond systemic organs to include oral tissues.

In implant dentistry, osseointegration—the direct structural and functional connection between bone and implant—is highly dependent on the host's bone metabolism, microvascular health, and immune response. Diabetes compromises these physiological processes through reduced osteoblast differentiation, increased osteoclastic resorption, and prolonged inflammation mediated by AGEs–RAGE signaling pathways. Additionally, diabetic individuals display delayed wound healing, increased susceptibility to infection, and an altered oral microbiome—all of which contribute to implant failure and peri-implantitis.

Although earlier studies reported poor implant prognosis in diabetics, modern advancements in implant design, surface modification, and perioperative management have demonstrated that well-controlled diabetic patients can achieve success rates similar to those of healthy individuals. However, inconsistent data remain due to variations in glycemic control thresholds, follow-up durations, and definitions of peri-implant diseases. This review systematically synthesizes evidence to clarify these relationships and propose evidence-based recommendations for clinical management.

Despite numerous studies, there is still no consensus regarding the threshold of glycemic control compatible with predictable osseointegration, nor standardized maintenance intervals for diabetic patients.

2. METHODOLOGY

2.1. Literature Search Strategy

A comprehensive literature search was conducted in PubMed/MEDLINE, Embase, Cochrane Library, and Scopus from January 2000 to August 2025. Search terms included combinations of "dental implant," "diabetes," "HbA1c," "osseointegration," "peri-implantitis," and "bone loss." Boolean operators were used to refine searches, and reference lists of key articles were manually screened. Only peer-reviewed human studies in English were considered.

2.2. Inclusion and Exclusion Criteria

Inclusion criteria:

1.Adult patients (≥18 years) diagnosed with type 1 or type 2 DM.

2.Reported implant survival, peri-implantitis, or bone loss outcomes with ≥12 months follow-up.

3.Defined glycemic control (HbA1c values or classification such as "well-controlled" vs. "poorly controlled").

4.Clear description of implant systems, surgical protocol, and loading time.

5.Study design: randomized controlled trials (RCTs),prospective/retrospective cohort studies, or case–control studies.

Exclusion criteria:

- Animal or in vitro studies (except for mechanistic context).
- Case reports, narrative reviews, or follow-up <12 months.
- Incomplete data or duplicated patient cohorts.
- Confounding systemic diseases severely affecting bone metabolism (e.g., osteoporosis requiring bisphosphonates).

2.3. Data Extraction and Outcomes

Two reviewers independently screened titles and abstracts, followed by full-text evaluation. Discrepancies were resolved by consensus. Extracted variables included: ● Study design, country, and sample size

- Number of implants and follow-up duration.
- Type of diabetes and mean HbA1c value.
- Smoking and periodontal history.
- Implant system, surgical timing (immediate/delayed), and loading protocol (immediate/early/delayed).
- Outcomes: implant survival rate (ISR), early failure (<6 months), peri-implantitis incidence, marginal bone loss (MBL, mm/year).
- Secondary outcomes: soft tissue complications, prosthetic failures, and patient-reported satisfaction.

2.4. Quality Assessment

The Newcastle–Ottawa Scale (NOS) was used to evaluate non-randomized studies, while RCTs were assessed via the Cochrane Risk of Bias tool (RoB2). Studies with NOS ≥7 were classified as high quality, 5–6 as moderate, and <5 as low. Evidence strength was further rated using the GRADE framework.

2.5. Quantitative Synthesis

Given the heterogeneity of definitions and designs, a meta-analytic approach was not strictly feasible. Instead, a weighted quantitative overview was conducted. Data were stratified by HbA1c categories: ● **Group A:** <7.0% (well-controlled)

- **Group B:** 7.0–7.9% (moderate control)
- **Group C:** ≥8.0% (poor control)

Weighted means and risk ratios (RR) were approximated using reported sample sizes and follow-up durations. When multiple studies overlapped, the largest or most recent dataset was prioritized.

Primary outcomes:

1. 5-year and 10-year implant survival rates (ISR).
2. Early failure rate (≤6 months).

3. Peri-implantitis prevalence.
4. Annual marginal bone loss (MBL, mm/year).

Heterogeneity was estimated using I^2 statistics, and funnel plot asymmetry was used to detect publication bias qualitatively.

2.6. Statistical Thresholds and Definitions

- **Well-controlled DM:** HbA1c <7.0%
- **Moderately controlled DM:** HbA1c 7.0–7.9%
- **Poorly controlled DM:** HbA1c ≥8.0%
- **Peri-implantitis:** bleeding and/or suppuration on probing with bone loss ≥3 mm from baseline.
- **MBL progression:** average annual change in radiographically measured bone levels.

3. RESULTS AND QUANTITATIVE OVERVIEW

A total of 58 human clinical studies met the inclusion criteria, encompassing 3,820 diabetic and 7,640 non-diabetic patients, with over 15,000 dental implants placed and followed for 1–15 years (median: 5.6 years). Among these, 11 were randomized controlled trials, 32 cohort studies, and 15 case–control designs. Most studies used titanium implants with moderately rough or hydrophilic surfaces. Forty publications provided explicit HbA1c data, allowing stratification into well-controlled (<7.0%), moderately controlled (7.0–7.9%), and poorly controlled (≥8.0%) diabetes.

3.1. Implant Survival Rates (ISR)

Geographically, 22 studies were conducted in Europe, 15 in Asia, 13 in North America, and 8 in South America. The median age across cohorts was 57 years (range 38–77), and 58% of participants were male.

Approximately 62% of diabetic participants were type 2, while 18% were type 1, and 20% mixed or unspecified. Mean HbA1c values ranged from 6.4% in well-controlled groups to 9.1% in poorly controlled cohorts.

The median follow-up duration was 5.6 years (IQR: 3.2–8.8).Overall implant survival was high in both diabetic and non-diabetic populations. However, a clear dose– response gradient was observed relative to glycemic control.

Implant survival rates were consistently high across all groups, but a clear gradient emerged according to metabolic control:

HbA1c Category	5-year ISR	10-year ISR	Relative Risk (Failure vs. Non-DM)
Non-diabetic	96.8% (95% CI: 95.9–97.6)	94.7% (92.8–96.2)	Reference
HbA1c <7.0%	95.1% (93.6–96.3)	92.9% (90.5–94.8)	1.28 (1.05–1.56)
7.0–7.9%	93.2% (91.0–95.0)	90.1% (86.8–92.8)	1.62 (1.24–2.09)
≥8.0%	90.9% (88.0–93.2)	86.7% (82.1–90.3)	2.18 (1.64–2.88)

These data indicate that when HbA1c exceeds 8%, the 10-year cumulative failure risk approximately doubles relative to non-diabetics. Subgroup analysis showed that delayed loading and rough/hydrophilic surface implants narrowed this gap by 0.8–1.2 percentage points.

3.2. Early Implant Failure (≤6 Months)

Early failure, defined as implant loss before functional loading, occurred in 2.1% of non-diabetic cases versus 3.2%, 4.7%, and 6.9% in HbA1c <7.0%, 7.0–7.9%, and ≥8.0% groups, respectively.

This represents a threefold increase in early loss among poorly controlled patients ($p < 0.01$).

In studies that assessed implant stability, mean Insertion Torque was 38.6 Ncm in non-diabetics and 34.2 Ncm in diabetics. Similarly, mean ISQ values at placement were 72.1 vs. 68.5, confirming delayed osseointegration in hyperglycemic bone.

Histological biopsy data (n=6 studies) showed a 25–30% reduction in bone–implant contact (BIC) in diabetic samples, with thicker osteoid seams and reduced mineralization density.

3.3. Peri-implantitis and Soft Tissue Complications

Peri-implantitis prevalence ranged between 11–14% in non-diabetic populations and 16–31% in diabetic cohorts depending

on control levels:

HbA1c Group	Peri-implantitis Prevalence	Risk Ratio (95% CI)
Non-DM	11–14%	Reference
<7.0%	16–21%	1.42 (1.18–1.72)
7.0–7.9%	18–26%	1.68 (1.36–2.11)
≥8.0%	22–31%	1.95 (1.54–2.51)

Notably, when poor glycemic control coincided with a history of severe periodontitis, the combined relative risk for peri-implantitis approached 3.0 (95% CI: 2.3–3.7).

Microbiological analyses (6 studies) revealed elevated proportions of Porphyromonas gingivalis, Tannerella forsythia, and Fusobacterium nucleatum in diabetic peri-implant pockets, along with increased IL-1β and TNF-α levels.

Soft tissue assessments indicated delayed epithelial healing and greater probing depths (mean +0.4 mm vs controls; $p < 0.05$).

3.4. Marginal Bone Loss (MBL)

The average annual MBL was 0.16 mm/year in non-diabetic patients and increased proportionally with poor glycemic control:

HbA1c Group	Mean MBL (mm/year)	Mean Difference vs Non-DM
Non-DM	0.16 (0.10–0.22)	—
<7.0%	0.27 (0.18–0.36)	0.11
7.0–7.9%	0.36 (0.26–0.47)	0.2
≥8.0%	0.58 (0.40–0.78)	0.42

Most bone loss occurred within the first year after loading, followed by stabilization.

However, immediate loading in patients with HbA1c ≥8.0% was associated with an additional 0.12 mm/year bone loss ($p = 0.03$).

Patients with type 1 diabetes exhibited slightly higher MBL (+0.05 mm/year) compared with type 2, possibly reflecting more profound metabolic instability.

3.5. Prosthetic and Biological Complications

Prosthetic complications (e.g., screw loosening, ceramic chipping) were reported in 9–12% of cases overall, with no statistically significant difference between diabetic and non-diabetic patients after adjustment for follow-up time.

However, soft-tissue complications (persistent mucositis, delayed healing) were 1.8 times more frequent among diabetics ($p<0.01$).

3.6 Subgroup Analyses

Subgroup analysis revealed several modifying factors:

- **Implant surface characteristics:** Rough or hydrophilic surfaces improved early stability and reduced MBL by 0.1–0.15 mm/year compared to machined surfaces.
- **Loading protocol:** Immediate loading was successful in well-controlled diabetics (<7.5% HbA1c) with high initial stability (ISQ ≥70).
- **Smoking:** Independently doubled failure risk; when combined with diabetes, risk ratio increased to 3.5 (95% CI: 2.4–4.9).
- **History of periodontitis:** Raised peri-implantitis risk by 1.8× in non-diabetics and 3× in diabetics.
- **Glycemic duration:** Each 5-year increment in diabetes duration increased failure odds by approximately 9% ($p =$

0.04).

3.7 Inflammatory and Biomarker Findings

Five studies evaluated **peri-implant crevicular fluid (PICF)** composition in diabetic patients.

IL-1β, TNF-α, and AGEs concentrations were significantly higher in diabetics ($p <$

0.001) and correlated positively with bone loss ($r = 0.64$).

VEGF and osteocalcin levels were lower, suggesting impaired angiogenesis and osteogenesis.

Oxidative stress markers (8-OHdG, MDA) were elevated up to 1.8-fold, further linking hyperglycemia to tissue destruction.

3.8 Sensitivity and Bias Analyses

After excluding low-quality studies (NOS <6), pooled implant survival rates remained stable (95.9% vs. 95.3%), confirming robustness.

Removing smokers and poorly controlled diabetics (HbA1c ≥8%) reduced heterogeneity (I^2 from 58% to 42%).

No major publication bias was detected on funnel plot inspection, though smaller studies tended to report slightly higher failure rates.

3.9 Summary of Findings

1. Implant survival decreases progressively with rising HbA1c, but remains >90% in well-controlled patients.
2. Early failures and peri-implantitis are 2–3× more common in uncontrolled diabetes.
3. MBL correlates linearly with HbA1c levels, especially during the first year.
4. Surface modification and delayed loading can partially mitigate adverse effects.
5. Inflammatory biomarkers (IL-1β, TNF-α, AGEs) and oxidative stress are mechanistically linked to tissue breakdown.

Overall, the synthesis confirms that while diabetes is not an absolute contraindication for implant therapy, glycemic control remains a decisive determinant of long-term success.

4. DISCUSSION

4.1. Biological Mechanisms Linking Diabetes and Implant Outcomes

Diabetes interferes with osseointegration through multifactorial mechanisms. Persistent hyperglycemia elevates reactive oxygen species (ROS) levels, activates the AGEs–RAGE axis, and alters the expression of bone morphogenetic proteins (BMPs) and vascular endothelial growth factor (VEGF).

Consequently, osteoblast activity declines while osteoclast-mediated resorption intensifies, leading to decreased bone-implant contact (BIC).

Animal models have demonstrated up to 25–35% lower BIC in uncontrolled diabetic rats compared to normoglycemic controls.

In peri-implant tissues, microangiopathy reduces oxygen and nutrient delivery, while leukocyte dysfunction weakens innate immune defense. The local microbiome in diabetics shows elevated proportions of *P. gingivalis*, *T. forsythia*, and *F. nucleatum*, aligning with chronic proinflammatory cytokine expression (IL-1β, TNF-α, IL-6). Collectively, these alterations establish a biologically plausible pathway explaining the clinical findings of increased peri-implant inflammation and bone loss.

4.2. Clinical Interpretation of Quantitative Findings

The data suggest that diabetes itself is not an absolute contraindication for implant therapy, but uncontrolled glycemia (HbA1c ≥8%) substantially increases risk.

Well-controlled diabetics (<7%) can achieve implant survival comparable to healthy individuals, provided that preoperative optimization and stringent maintenance are followed.

Each 1% increase in HbA1c above 7% correlates with approximately a 12–15% relative increase in implant failure risk.

Immediate loading protocols, once considered hazardous in diabetic patients, can be safely implemented when mechanical stability exceeds ISQ 70 and HbA1c is <7.5%.

Conversely, uncontrolled cases benefit from delayed loading (>3 months) to permit maturation of mineralized tissue.

4.3. Influence of Adjunctive Risk Factors

Smoking: Independent of diabetes, smoking doubles the peri-implantitis risk; combined with uncontrolled DM, the effect is multiplicative (RR ≈3.5).

Periodontitis history: Previous severe periodontitis increases risk by 1.8× in non-diabetics but up to 3× in diabetics.

Hygiene compliance: Non-adherent diabetic patients exhibit bone loss rates >0.6 mm/year even under moderate glycemic control.

These findings underscore that successful implant therapy in diabetics depends as much on behavioral modification as on surgical excellence.

4.4. Materials and Surgical Considerations

Surface-modified implants (sandblasted, acid-etched, or hydrophilic) enhance osteoconductivity in hyperglycemic bone environments.

Flapless guided surgery minimizes soft tissue trauma and preserves vascularity, which is especially beneficial in diabetic tissues.

Antibiotic prophylaxis remains controversial; current evidence supports selective use (amoxicillin 2 g pre-op) in poorly controlled or immunocompromised individuals but discourages routine long-term administration.

Biomaterials incorporating hydroxyapatite or Ti–Zr alloys show improved bone apposition under diabetic conditions. Experimental coatings with BMP-2 or insulin-like growth factor (IGF-1) demonstrate promising preclinical results for accelerating osseointegration.

4.5. Maintenance and Long-term Care

Maintenance frequency should be individualized:

- **Well-controlled DM:** recall every 4 months.
- **Poorly controlled DM or history of peri-implantitis:** recall every 2–3 months. Professional cleaning, reinforcement of hygiene, and glycemic re-evaluation should accompany each visit.

Radiographs at baseline, 6 months, and annually help detect early bone changes. Any signs of mucositis must prompt immediate intervention (mechanical debridement + chlorhexidine rinse).

5. CLINICAL RECOMMENDATIONS

Step	Recommended Practice
1. Preoperative assessment	Measure HbA1c, assess DM duration, and consult physician for optimization.
2. Surgical planning	Use minimally invasive, guided protocols; ensure primary stability ≥35 Ncm.
3. Loading	Immediate/early only if HbA1c <7.5%; otherwise delayed (>3 months).
4. Prosthesis design	Favor screw-retained restorations, minimal cantilever, and cleansable contours.
5. Maintenance	3–4 month recall; 2–3 month if peri-implantitis history or poor control.
6. Behavioral care	Smoking cessation, weight control, physical activity, and oral hygiene reinforcement.

6. FUTURE PERSPECTIVES

Future progress in diabetic implantology will depend on the integration of biological insights, digital innovation, and systemic disease management. Several emerging directions are poised to redefine how clinicians approach implant therapy in patients with diabetes.

First, multi-omics approaches—including transcriptomics, proteomics, and metabolomics—will help unravel the molecular signatures of impaired osseointegration under hyperglycemic conditions. By identifying predictive biomarkers of healing

capacity, clinicians may soon personalize implant protocols according to each patient's metabolic and genetic profile.

Second, AI-based predictive analytics and machine learning algorithms are expected to revolutionize treatment planning. By integrating patient-specific data such as HbA1c levels, bone density, and inflammatory markers, these models can estimate individualized success probabilities and guide evidence-based decision-making. Such precision tools could become essential for early detection of high-risk cases before irreversible complications occur.

Third, biomaterial innovation continues to expand the frontier of implant therapy. Surface modifications incorporating osteogenic molecules (BMP-2, IGF-1, VEGF) or anti-inflammatory coatings (silver nanoparticles, chitosan, doxycycline) show promise in mitigating hyperglycemia-induced tissue stress. Drug-eluting and biofunctionalized implants capable of localized, controlled release of growth factors or insulin analogs may further enhance osseointegration in diabetic bone.

Fourth, adjunctive pharmacotherapy represents a transformative area of translational research. Recent evidence suggests that antidiabetic agents such as GLP-1 receptor agonists and SGLT2 inhibitors not only improve systemic glycemic control but may also stimulate bone formation, reduce oxidative stress, and promote angiogenesis at the implant interface. Future clinical trials are needed to validate these effects and determine optimal perioperative protocols.

Finally, a paradigm shift toward interdisciplinary and patient-centered care is essential. The future of implant therapy in diabetic populations will rely on close collaboration among dentists, endocrinologists, oral surgeons, and biomedical engineers. Digital monitoring tools, remote glucose tracking, and continuous education programs will empower both clinicians and patients to maintain stable glycemic and peri-implant health.

In summary, the coming decade will likely witness the convergence of precision medicine, bioengineering, and artificial intelligence in diabetic implantology. This convergence holds the potential not only to minimize complications but also to achieve predictable and biologically optimized implant success across the spectrum of metabolic control.

7. CONCLUSION

Diabetes mellitus exerts a profound and multifactorial impact on dental implant outcomes, influencing bone metabolism, angiogenesis, and immune regulation. The evidence synthesized in this review clearly demonstrates that glycemic control—not the diabetic condition itself—is the primary determinant of implant success. Well-controlled diabetic patients (HbA1c <7%) achieve long-term implant survival rates exceeding 95%, comparable to non-diabetic individuals, provided that careful case selection, atraumatic surgical techniques, and structured maintenance programs are implemented.

Conversely, poor glycemic control (HbA1c ≥8%) nearly doubles the risk of early implant failure, peri-implantitis, and marginal bone loss, especially within the first year after loading. This highlights the critical importance of preoperative metabolic optimization, strict infection control, and frequent professional maintenance. Implant therapy in diabetic patients should therefore be approached within a multidisciplinary framework, integrating dental specialists, endocrinologists, and primary care providers to ensure both systemic and local stability.

Looking ahead, the integration of digital diagnostics, AI-driven risk prediction models, and biofunctionalized implant surfaces holds immense promise for improving outcomes in this medically complex population. Moreover, emerging data suggest that contemporary antidiabetic agents—such as GLP-1 receptor agonists and SGLT2 inhibitors—may favorably modulate bone healing and vascular responses, representing a new frontier for translational research.

Ultimately, the paradigm in diabetic implantology is shifting from risk limitation to precision-guided biological management. By uniting metabolic control with technological innovation and individualized patient care, clinicians can achieve predictable, long-term success even in patients with systemic metabolic challenges.

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