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SYSTEMIC HEALTH AND PHARMACOTHERAPY INTERPLAY IN MARGINAL BONE LOSS AROUND DENTAL IMPLANTS: A NARRATIVE REVIEW OF DIABETES, METABOLIC BONE DISEASE, SSRIS, PROTON PUMP INHIBITORS, AND LONG-TERM BISPHOSPHONATE THERAPY

Althaaf Khan *

Private Practice, South Africa.

* Corresponding Author

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ABSTRACT

Background: Long-term marginal bone loss (MBL) around osseointegrated dental implants is influenced not only by local biomechanical and biofilm-related factors but also by the systemic and pharmacological status of the host. Selective serotonin reuptake inhibitors (SSRIs), proton pump inhibitors (PPIs), poorly controlled diabetes mellitus, and long-term bisphosphonate therapy have each been implicated as modifiers of bone turnover, yet the strength, consistency, and clinical magnitude of these associations remain incompletely defined.

Objective: This narrative review synthesises the current evidence describing how these systemic conditions and medication classes alter bone remodelling and long-term peri-implant marginal bone stability, and evaluates the extent to which existing evidence is sufficient to guide clinical decision-making.

Methods: A narrative literature review was conducted using PubMed/MEDLINE, Scopus, and the Cochrane Library, supplemented by hand-searching of reference lists of relevant systematic reviews and meta-analyses published up to September 2026. Retrospective cohort studies, prospective cohort studies, systematic reviews, and meta-analyses addressing SSRIs, PPIs, diabetes mellitus, and bisphosphonates in relation to dental implant marginal bone loss or bone metabolism were included.

Results: SSRI and PPI use are each associated with modestly but consistently greater crestal and marginal bone loss around implants in retrospective cohorts, plausibly mediated through gut-derived serotonergic signalling and direct osteoclast/osteoblast inhibition, respectively. Poorly controlled diabetes mellitus (HbA1c > 8%) is associated with greater probing depth, bleeding on probing, and marginal bone loss compared with well-controlled diabetes or non-diabetic patients, whereas well-controlled diabetes shows outcomes comparable to systemically healthy patients. Long-term bisphosphonate therapy suppresses osteoclastic bone turnover and is associated with increased implant failure risk and greater marginal bone loss in meta-analyses, although the mechanistic link between jawbone turnover suppression and osteonecrosis remains contested. Across all four exposures, the underlying evidence base is dominated by retrospective, heterogeneous, and often underpowered cohorts.

Conclusion: Available evidence supports biologically plausible and clinically relevant associations between these systemic conditions/medications and long-term marginal bone loss, but causal inference is limited by study design. Prospective, adequately powered, multicentre human trials with standardised radiographic protocols and medication-exposure quantification are required to clarify the magnitude of risk and to inform evidence-based peri-implant maintenance protocols.

Keywords: Marginal Bone Loss; Dental Implants; Selective Serotonin Reuptake Inhibitors; Proton Pump Inhibitors; Diabetes Mellitus; Bisphosphonates

1 INTRODUCTION

Osseointegrated dental implants are among the most predictable rehabilitative treatments in modern dentistry, with reported long-term survival rates exceeding 90% at ten years ¹. Long-term implant success, however, is not defined by survival alone but by the maintenance of stable peri-implant marginal bone levels over time. Progressive marginal bone loss (MBL) beyond the initial physiological remodelling that follows implant loading is widely regarded as an early radiographic indicator of peri-implant disease and a precursor to late implant failure.

While local factors such as plaque accumulation, implant-abutment connection design, prosthetic emergence profile, and surgical technique are well-established determinants of MBL, a growing body of literature has drawn attention to the role of the host's systemic health status and concomitant pharmacotherapy. A 2018 systematic review and meta-analysis commissioned by the International Team for Implantology concluded that intake of proton pump inhibitors (PPIs) and selective serotonin reuptake inhibitors (SSRIs) was associated with an increased implant failure rate ¹, and a more recent scoping review has broadened this evidence base to include antihypertensives, anticoagulants, immunosuppressants, and nonsteroidal anti-inflammatory drugs ². In parallel, systemic conditions that directly perturb bone remodelling — most notably poorly controlled diabetes mellitus and metabolic bone diseases treated with long-term antiresorptive therapy — have been repeatedly linked to inferior peri-implant outcomes.

The biological rationale for these associations converges on a common pathway: bone around an implant is maintained by a continuous cycle of osteoclastic resorption and osteoblastic formation, and any systemic or pharmacological perturbation of this coupled process has the potential to manifest clinically as accelerated crestal or marginal bone loss. SSRIs act on serotonergic signalling pathways that are now recognised to modulate osteoblast proliferation; PPIs interfere with gastric acid-dependent calcium absorption and appear to exert direct inhibitory effects on osteoclast and osteoblast function; poorly controlled hyperglycaemia impairs collagen cross-linking, microvascular perfusion, and immune-inflammatory regulation of bone; and long-term bisphosphonate therapy produces profound and sustained suppression of osteoclastic turnover, particularly in the metabolically active alveolar bone of the jaws.

Despite this plausible shared mechanism, the clinical literature addressing each of these four exposures is heterogeneous in design, largely retrospective, and frequently underpowered to detect clinically meaningful differences in MBL with confidence. The objective of this narrative review is therefore twofold: first, to synthesise and critically appraise the existing evidence describing how poorly controlled diabetes, metabolic bone disease, SSRIs, PPIs, and long-term bisphosphonate therapy alter bone turnover and long-term marginal bone loss around dental implants; and second, to identify the specific methodological gaps that must be addressed by future prospective, high-powered human trials before this evidence can be translated into definitive clinical guidance.

2 MATERIALS AND METHODS

This article was structured as a narrative literature review rather than a formal systematic review, reflecting the objective of providing a clinically oriented synthesis across four heterogeneous exposure categories rather than a quantitative pooled estimate for a single research question.

2.1 Search strategy

A literature search was performed using PubMed/MEDLINE, Scopus, and the Cochrane Library for articles published up to September 2026, without restriction on publication date at the lower bound. Search terms were combined using Boolean operators and included variations and combinations of: "dental implant", "marginal bone loss", "crestal bone loss", "osseointegration", "selective serotonin reuptake inhibitor", "SSRI", "proton pump inhibitor", "PPI", "diabetes mellitus", "glycaemic control", "HbA1c", "bisphosphonate", "antiresorptive", "osteoporosis", "medication-related osteonecrosis of the jaw", and "bone turnover". Reference lists of identified systematic reviews and meta-analyses were hand-searched for additional relevant primary studies.

2.2 Eligibility criteria

Eligible sources comprised retrospective and prospective cohort studies, case-control studies, systematic reviews, and meta-analyses published in English that reported implant survival, implant failure, marginal or crestal bone loss, or validated biochemical/imaging markers of bone turnover in relation to SSRI use, PPI use, diabetes mellitus status or glycaemic control, or bisphosphonate/antiresorptive therapy. Case reports, non-peer-reviewed sources, and studies not reporting extractable outcome data were excluded from quantitative description, although selected case-level or mechanistic sources were retained where they informed the discussion of pathophysiological mechanism.

2.3 Data synthesis

Given the heterogeneity of outcome definitions (crestal bone loss versus marginal bone loss versus implant failure), follow-up duration, and exposure quantification across the included body of literature, a narrative rather than meta-analytic synthesis was undertaken. Findings are presented by exposure category (SSRIs, PPIs, diabetes mellitus, and bisphosphonates/metabolic bone disease), with emphasis on the direction and approximate magnitude of effect, the proposed biological mechanism, and the methodological limitations of the underlying evidence.

2.4 Ethical considerations

As this article is a narrative review of previously published, publicly available literature and did not involve the collection of new patient data, formal ethical approval and informed consent were not required.

3 RESULTS AND DISCUSSION

3.1 Selective serotonin reuptake inhibitors and peri-implant bone turnover

Serotonin (5-hydroxytryptamine) has an established, dual-compartment role in skeletal homeostasis. Gut-derived serotonin acts on osteoblast progenitors via the Htr1b receptor to inhibit proliferation and thereby reduce bone formation, while centrally derived serotonin acts through hypothalamic pathways to promote bone formation via reduced sympathetic outflow⁴. Because SSRIs block the serotonin transporter systemically, including in bone marrow stromal cells and osteoblast/osteoclast lineages, their chronic use has the theoretical and empirically supported potential to shift this balance toward reduced bone formation and, secondarily, altered turnover.

A nationwide population-based cohort study in Korea found that both recent and former users of serotonin reuptake inhibitors had a significantly increased risk of subsequent low bone density compared with non-users, with a stronger association in recent users⁵, consistent with a reversible, exposure-dependent pharmacological effect rather than a fixed structural change. In the implant-specific literature, a retrospective study of 152 implants in 105 patients followed for a mean of 3.8 years found that SSRI use was associated with significantly greater marginal bone loss around osseointegrated implants³, and the 2018 ITI-commissioned systematic review similarly identified SSRIs as one of only two medication classes (alongside PPIs) with a consistent association with increased implant failure across the included studies¹.

Mechanistically, the balance of evidence suggests that the clinically observed effect represents a net negative influence of enhanced peripheral serotonergic tone on osteoblast-mediated bone formation, outweighing any potential benefit from central serotonergic effects, although in vitro data indicate that this relationship may be dose- and duration-dependent, with low concentrations potentially stimulating osteoblast and osteoclast proliferation and higher, sustained concentrations tending toward inhibition. This dose-response complexity, combined with substantial between-study heterogeneity in SSRI agent, dose, and duration of exposure, limits the precision with which the magnitude of MBL attributable to SSRI use can currently be estimated.

3.2 Proton pump inhibitors and peri-implant bone turnover

PPIs are among the most widely prescribed medications globally and have the most extensively documented association with peri-implant bone loss of the four exposures reviewed here. A retrospective cohort of 1,773 osseointegrated implants found that PPI use was associated with an increased risk of osseointegrated implant failure, attributed to a negative impact of PPIs on bone accrual⁶. A separate retrospective study of 1,480 implants in 635 patients reported mean crestal bone loss of 1.60 mm in PPI users versus 1.01 mm in non-users, a difference that persisted after adjustment for systemic confounders (1.87 mm versus 1.04 mm)⁷. Chrcanovic and colleagues similarly reported that PPI intake was associated with a significantly increased risk of dental implant failure in a large retrospective cohort⁸, and a retrospective study from Turkey found that PPI intake negatively affected osseointegration of dental implants⁹. More recently, fractal analysis of peri-implant trabecular bone microstructure in a matched case-control study demonstrated altered bone patterns consistent with negatively affected bone metabolism in long-term PPI users¹⁰.

This implant-specific evidence is consistent with the broader skeletal literature: large population-based cohort studies have linked PPI use with an increased risk of osteoporosis-related fractures ¹¹ and long-term PPI therapy with increased risk of hip fracture ¹², while a recent prospective study using high-resolution peripheral quantitative computed tomography found that regular long-term PPI intake was associated with measurable deterioration in peripheral bone mineral density, microarchitecture, and estimated bone strength in older patients ¹³. The proposed mechanism combines a reduction in gastric acid-dependent calcium absorption with a direct, acid-independent inhibitory effect of PPIs on osteoclast-mediated resorption of mineralised matrix, which — counterintuitively for an agent that inhibits resorption — appears in the implant setting to translate into impaired bone healing and remodelling around the implant-bone interface rather than a net gain in bone mass, since normal, coupled remodelling activity is required for successful long-term osseointegration and adaptation to functional load.

3.3 Diabetes mellitus and glycaemic control

Diabetes mellitus, particularly when poorly controlled, is one of the most extensively studied systemic modifiers of peri-implant bone stability. A retrospective study of 929 implants in 204 patients found no statistically significant association between diabetes per se and implant failure or peri-implantitis, but noted that the single poorly controlled diabetic patient included experienced multiple implant failures, underscoring the importance of glycaemic threshold rather than diabetes diagnosis alone ¹⁴. This threshold effect has been reproduced across the broader literature: a 2024 systematic review concluded that overall implant survival in diabetic patients is generally comparable to non-diabetic patients (96–97% at one year, 87–96% at five years), but that peri-implant health metrics — marginal bone loss and probing depth in particular — were adversely affected specifically in patients with poorly controlled diabetes (HbA1c > 8%), with a dose-response relationship between rising HbA1c and worsening peri-implant outcomes ¹⁵.

A systematic review and meta-analysis examining the association between diabetes and dental implant complications similarly concluded that diabetic patients, and particularly those with suboptimal glycaemic control, demonstrate significantly greater marginal bone loss and higher rates of peri-implantitis than non-diabetic patients ¹⁶, a finding echoed by an overview of systematic reviews focused specifically on implant survival and marginal bone loss in diabetic populations ¹⁷. The biological plausibility of this glycaemic threshold effect is well established: chronic hyperglycaemia promotes the accumulation of advanced glycation end-products within the collagenous bone matrix, impairs osteoblast differentiation and function, compromises microvascular perfusion necessary for bone healing, and dysregulates the local immune-inflammatory response to bacterial biofilm, collectively favouring a shift toward net bone resorption at the peri-implant interface once glycaemic control deteriorates beyond a critical threshold.

Clinically, this body of evidence supports a nuanced message: diabetes mellitus itself is not a contraindication to implant therapy, but achieving and maintaining good glycaemic control (generally HbA1c ≤ 7–8%) prior to and following implant placement appears to be a modifiable determinant of long-term marginal bone stability, reinforcing the rationale for pre-implant metabolic optimisation and closer peri-implant maintenance intervals in patients with suboptimal glycaemic control.

3.4 Metabolic bone disease and long-term bisphosphonate therapy

Bisphosphonates remain the most widely prescribed class of antiresorptive agents for osteoporosis and other metabolic bone diseases, acting by binding to hydroxyapatite at sites of active bone turnover and inducing osteoclast apoptosis, thereby profoundly and durably suppressing osteoclastic resorption. A systematic review and meta-analysis of 33 publications investigating bisphosphonate use in relation to dental implant outcomes found that bisphosphonate therapy was associated with increased implant failure and greater marginal bone loss compared with non-users ¹⁸, consistent with the mechanistic expectation that suppressed osteoclastic turnover impairs the coupled remodelling response required for implant osseointegration and long-term crestal bone adaptation.

The relationship between bisphosphonate-induced turnover suppression and the jaws specifically remains an area of active mechanistic debate. It has long been proposed that the alveolar processes of the jaws possess a substantially higher basal bone turnover rate than the axial and appendicular skeleton, rendering them disproportionately vulnerable to bisphosphonate-induced over-suppression and, in its most severe form, medication-related osteonecrosis of the jaw (MRONJ). However, a scintigraphic study comparing mandibular, maxillary, and femoral bone turnover in bisphosphonate-treated and untreated patients found that mandibular turnover was, if anything, similar to that of the femur and significantly lower than that of the maxilla, and that turnover in none of the examined skeletal sites was significantly altered by bisphosphonate administration — findings that are difficult to reconcile with the simple "over-suppression" hypothesis and suggest that the pathogenesis of MRONJ, and by extension of bisphosphonate-associated marginal bone loss, is likely multifactorial, involving impaired angiogenesis, altered immune surveillance, and local infective or traumatic triggers in addition to turnover suppression ¹⁹.

By contrast with bisphosphonate-specific data, the broader relationship between osteoporosis as a diagnosis (independent of antiresorptive treatment status) and implant outcomes appears less consistently detrimental. A 2025

systematic review and meta-analysis of fourteen studies found no significant difference in pooled implant survival between osteoporotic and non-osteoporotic patients, concluding that osteoporosis alone does not appear to compromise dental implant outcomes when treatment is individualised and carefully planned ²⁰. This distinction is clinically important: it is the pharmacological suppression of bone turnover by long-term antiresorptive therapy, rather than the underlying metabolic bone disease per se, that appears to carry the principal risk for impaired peri-implant bone remodelling and for the rare but serious complication of MRONJ, particularly with intravenous and long-duration oral regimens ²¹.

3.5 Synthesis and the case for prospective, high-powered human trials

Taken together, the evidence reviewed here demonstrates a biologically coherent pattern: each of the four exposures acts, through a distinct mechanism, on the coupled osteoblast-osteoclast remodelling cycle that governs long-term marginal bone stability around dental implants, and each is associated in the clinical literature with a modest but consistently directional increase in marginal or crestal bone loss and, in some analyses, implant failure risk ^{1, 2}. Nonetheless, several recurring methodological limitations temper the strength of causal inference that can currently be drawn.

First, the overwhelming majority of the underlying studies are retrospective cohort or case-control designs, which are inherently vulnerable to confounding by indication (for example, patients prescribed PPIs or SSRIs may differ systematically in comorbidity burden, smoking status, or oral hygiene behaviour from non-users) and to inconsistent or incomplete documentation of medication dose, duration, and adherence. Second, outcome definitions vary considerably across studies — marginal bone loss, crestal bone loss, exposed implant threads, and implant failure are not interchangeable outcomes, and radiographic measurement protocols (periapical versus panoramic imaging, reference points, follow-up intervals) are rarely standardised, limiting comparability and precluding robust meta-analytic pooling in several of the exposure categories reviewed. Third, sample sizes in the implant-specific literature are frequently modest, and few studies are powered a priori to detect the relatively small absolute differences in marginal bone loss (often in the order of 0.2–0.8 mm) that are reported between exposed and unexposed groups, raising the possibility of both type I and type II error across the body of evidence as a whole. Fourth, dose-response and duration-response relationships — which are biologically expected for pharmacologically mediated effects on bone turnover — have rarely been formally modelled in the dental implant literature, despite being partially explored in the broader skeletal fracture literature for PPIs and bisphosphonates.

Addressing these gaps will require prospective, multicentre cohort studies and, where ethically and practically feasible, randomised or quasi-randomised comparative designs that: (i) recruit sufficiently large and adequately powered samples stratified by medication class, dose, and duration of exposure; (ii) apply standardised, calibrated radiographic protocols for marginal bone level measurement at predefined time points; (iii) capture glycaemic control as a continuous, longitudinally updated variable rather than a binary diabetes diagnosis; (iv) incorporate biochemical markers of bone turnover (such as serum C-terminal telopeptide and procollagen type I N-terminal propeptide) alongside radiographic outcomes to strengthen mechanistic inference; and (v) extend follow-up well beyond the one- to three-year windows that characterise much of the existing literature, given that both SSRI/PPI effects and antiresorptive-related complications may manifest, or continue to evolve, only after several years of continuous exposure. Only with this level of methodological rigour can the modest but consistent associations identified in the current retrospective literature be confirmed as causal, quantified with clinically actionable precision, and translated into evidence-based, medication-specific peri-implant maintenance and risk-stratification protocols.

4 CONCLUSION

Selective serotonin reuptake inhibitors, proton pump inhibitors, poorly controlled diabetes mellitus, and long-term bisphosphonate therapy each exert a biologically plausible and clinically observable negative influence on the bone remodelling processes that sustain long-term marginal bone stability around dental implants. Across all four exposures, the magnitude of the reported effect is generally modest at the individual-patient level but consistent in direction, and each is underpinned predominantly by retrospective, heterogeneous, and comparatively underpowered clinical evidence. These findings support a cautious but proactive clinical approach — including pre-implant metabolic and pharmacological history-taking, individualised risk counselling, and closer peri-implant maintenance intervals for patients within these exposure categories — while underscoring that prospective, adequately powered, methodologically standardised human trials remain essential to convert the current associative evidence base into definitive, medication- and disease-specific clinical guidance.

STATEMENTS ON COMPLIANCE WITH ETHICAL STANDARDS

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