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GLP-1 Receptor Agonists And Osseointegration: Bone Health Implications For Implant Dentistry In The Era Of Widespread Weight-Loss Therapy

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Abstract:
Glucagon-like peptide-1 receptor agonists (GLP-1RAs) — including liraglutide, semaglutide, and tirzepatide — have shifted from niche antidiabetic agents to among the most widely prescribed medications worldwide, driven by their efficacy in chronic weight management. This expansion means that a rapidly growing share of candidates for dental implant rehabilitation now present with active or recent GLP-1RA use, yet no narrative review to date has examined the implications of these agents for osseointegration outside the context of type 2 diabetes mellitus (T2DM). This review synthesizes mechanistic, preclinical, and clinical evidence on GLP-1RA action on bone metabolism, highlighting a key preclinical finding that exenatide enhances peri-implant osseointegration via the LRP5/6/GSK-3β/β-catenin pathway in diabetic models, and a 2025 clinical review demonstrating reduced peri-implant marginal bone loss in diabetic patients using GLP-1RAs compared with insulin or metformin. It then contrasts this diabetic evidence base with a distinct and clinically important scenario: non-diabetic patients using GLP-1RAs for obesity, in whom randomized and cohort data show modest but measurable reductions in bone mineral density (BMD) proportional to weight loss magnitude, likely mediated by mechanical unloading rather than a direct pharmacological effect on bone. Drawing on the emerging orthopedic and musculoskeletal literature on perioperative GLP-1RA management, this review proposes practical considerations for implant dentists — including anamnestic screening, timing of surgery relative to the trajectory of weight loss, and interdisciplinary communication with prescribing physicians — and identifies the absence of direct clinical data on implant survival in non-diabetic GLP-1RA users as a priority for future research.

Keywords: *GLP-1 receptor agonists; dental implants; osseointegration; bone metabolism; semaglutide*

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I. Introduction

The global burden of obesity and type 2 diabetes mellitus (T2DM) has driven an unprecedented expansion in the clinical use of glucagon-like peptide-1 receptor agonists (GLP-1RAs). First developed for glycemic control, agents such as liraglutide (Saxenda®), semaglutide (Ozempic®, Wegovy®), and the dual GIP/GLP-1 agonist tirzepatide (Mounjaro®, Zepbound®) are now prescribed at scale for chronic weight management in patients without diabetes, producing sustained weight reductions in the range of 10-20% of body weight (Karam et al., 2025; Herrou et al., 2023). This shift has direct relevance to implant dentistry: a growing proportion of patients presenting for implant-supported rehabilitation — whether to replace teeth lost to periodontal disease, trauma, or age-related edentulism — will report current or recent GLP-1RA use, often for indications entirely unrelated to diabetes.

Bone metabolism is a recognized target of GLP-1 signaling, and a substantial preclinical literature describes osteoanabolic and anti-resorptive effects of GLP-1RAs mediated through Wnt/β-catenin signaling, mitochondrial biogenesis, and modulation of macrophage polarization (Meng et al., 2016; Pal et al., 2019; Zhu et al., 2025). Within dentistry, this evidence has begun to be translated specifically into periodontal and peri-implant contexts, but almost exclusively in patients with T2DM, where GLP-1RA use is intertwined with glycemic control (Ahmad et al., 2025). By contrast, the rapidly expanding population of non-diabetic patients using GLP-1RAs for weight loss presents a mechanistically distinct scenario: clinical trials in this group show measurable reductions in bone mineral density (BMD) that appear to track the magnitude of weight loss itself rather than a direct antiosteogenic drug effect (Hansen et al., 2024; Jensen et al., 2024). No narrative review to date has connected this evidence to the specific clinical question of dental implant osseointegration.

This review aims to (1) summarize the pharmacological mechanisms by which GLP-1RAs influence bone metabolism and osseointegration; (2) critically appraise the clinical evidence on bone density and fracture risk in both diabetic and non-diabetic GLP-1RA users; and (3) propose practical implications and research priorities for implant dentistry, drawing where necessary on the more developed orthopedic perioperative literature as an analogous clinical model.

II. Theoretical Framework

Pharmacological mechanisms of GLP-1 receptor agonists on bone metabolism

The glucagon-like peptide-1 receptor (GLP-1R) is expressed on bone marrow stromal cells (BMSCs) and osteoclast precursors, but not directly on mature osteoblasts, indicating that GLP-1RA effects on bone formation are mediated indirectly through progenitor cell populations rather than through direct osteoblastic receptor activation (Meng et al., 2016). Activation of GLP-1R on BMSCs triggers PKA/β-catenin and PKA/PI3K/AKT/GSK3β signaling, promoting osteogenic differentiation while simultaneously inhibiting adipogenic differentiation — an effect consistent with a shift in the marrow niche away from adipocyte formation and toward osteoblastogenesis (Meng et al., 2016; Li et al., 2020). This Wnt/β-catenin-centered mechanism has been replicated across multiple GLP-1RA molecules and experimental models: exendin-4 downregulates histone deacetylase 1 (HDAC1) to activate Wnt3/β-catenin/Runx2 signaling in diabetic BMSCs (Deng et al., 2021); liraglutide activates PI3K/AKT, ERK1/2, and cAMP/PKA pathways converging on β-catenin in MC3T3-E1 preosteoblasts (Wu et al., 2017); and semaglutide activates the Wnt/LRP5/β-catenin axis to promote proliferation and osteogenic differentiation of bone-derived mesenchymal stem cells (Tian et al., 2025; Abo-Elenin et al., 2024).

Beyond Wnt signaling, GLP-1RAs exert osteoanabolic effects through at least two additional mechanisms. First, liraglutide upregulates AdipoR1 and PGC1α expression in osteoblasts via AMPK phosphorylation, enhancing mitochondrial biogenesis and respiration in a manner comparable in magnitude to parathyroid hormone (PTH 1-34) in ovariectomized rats (Pal et al., 2019). Second, GLP-1RAs modulate the local inflammatory and oxidative microenvironment of bone by shifting macrophage polarization from the pro-inflammatory M1 phenotype toward the reparative M2 phenotype, reducing TNF-α signaling and oxidative stress markers (HO-1, 4-HNE) while inhibiting osteoblast apoptosis (Zhu et al., 2025; Lu et al., 2022). Liraglutide additionally suppresses osteoclastogenesis in type 1 diabetic mice by downregulating Trem2 expression, independent of glycemic status (Yu et al., 2021). Collectively, this body of mechanistic evidence supports a model in which GLP-1RAs act as pleiotropic, indirect osteoanabolic agents — promoting bone formation, restraining resorption, and reducing local inflammation — largely independent of, though frequently studied alongside, their glycemic effects (Kitaura et al., 2021; Zhao et al., 2017).

Preclinical evidence directly relevant to peri-implant osseointegration

The mechanistic literature summarized above converges most directly on implant dentistry through a 2024 study comparing exenatide and insulin in a rat model of osseointegration under T2DM conditions. Chen et al. (2024) demonstrated that exenatide substantially outperformed insulin in promoting peri-implant osseointegration, with micro-CT and histological analysis confirming greater bone-to-implant contact and peri-implant bone microstructure in the exenatide group. Mechanistically, exenatide activated the LRP5/6/GSK-3β/β-catenin-dependent Wnt pathway in bone marrow mesenchymal stromal cells surrounding the implant site, while additionally suppressing Bmpr1a-mediated adipogenesis and Btrc-mediated inflammation — demonstrating that the osteogenic advantage of exenatide over insulin in this model was not simply a function of glycemic control, but reflected a direct, multi-pathway pro-osteogenic action localized to the implant interface (Chen et al., 2024).

Complementary evidence from periodontal regeneration models reinforces this picture. Co-therapy with stromal cell-derived factor-1 (SDF-1) and exendin-4 synergistically promoted proliferation, migration, and osteogenic differentiation of human periodontal ligament stem cells (PDLSCs) in vitro, and enhanced periodontal bone regeneration in a rat defect model in vivo, with greater recruitment of CXCR4+ and

CD90/CD34+ stromal cells to the healing site (Liang et al., 2021). Exendin-4 has also been shown to facilitate bone repair when combined with adipose-derived stem cell transplantation in a femoral defect model (Deng et al., 2019), and — in combination with the vitamin D analog eldecalcitol — to synergistically promote osteogenic differentiation of BMSCs via M2 macrophage polarization in diabetic osteoporosis (Lu et al., 2022). Taken together, these preclinical studies establish biological plausibility for a favorable effect of GLP-1RAs on the peri-implant bone-healing cascade, at least under conditions of impaired glucose metabolism.

Two distinct clinical populations, two distinct mechanisms

A critical distinction — and the central argument of this review — is that the favorable preclinical signal described above was generated almost entirely in models of T2DM, where GLP-1RA use corrects an underlying hyperglycemic, pro-inflammatory state known to impair osseointegration. The single clinical study identified that directly examines peri-implant outcomes, a retrospective cohort by Ahmad et al. (2025), found that diabetic patients with peri-implantitis treated with GLP-1RAs experienced significantly less clinical and radiographic peri-implant marginal bone loss than those treated with insulin or metformin ($p < .01$) — consistent with the mechanistic evidence and supporting a net-protective role of GLP-1RAs in diabetic peri-implant health.

This evidence base does not extend to the rapidly growing population of non-diabetic patients using GLP-1RAs primarily for weight management, in whom a different mechanism appears to predominate. Substantial, rapid weight loss — whether achieved through caloric restriction, bariatric surgery, or pharmacotherapy — is independently associated with accelerated bone turnover and BMD loss, largely attributable to reduced mechanical loading on the skeleton (Kim & Cho, 2026). In this population, GLP-1RA-associated BMD changes appear to reflect this weight-loss-mediated unloading effect superimposed on — and potentially partially offset by — the direct osteoanabolic signaling described in Section 2.1. The clinical net effect in non-diabetic patients is therefore less favorable and less predictable than the diabetic evidence alone would suggest, and is addressed in detail in Section 3.

III. Clinical Evidence On Bone Density, Fracture Risk, And Peri-Implant Outcomes

The most direct clinical evidence on GLP-1RA effects on bone in non-diabetic individuals comes from a randomized, placebo-controlled phase 2 trial by Hansen et al. (2024), conducted in 64 adults with increased fracture risk (T-score < -1.0 or recent low-energy fracture) but without diabetes. After 52 weeks of once-weekly semaglutide 1.0 mg, there was no significant change in the bone formation marker P1NP relative to placebo, but the bone resorption marker CTX was significantly higher in the semaglutide group (estimated treatment difference 166.4 ng/L, $p = .021$), and areal BMD was significantly lower at the lumbar spine ($p = .007$) and total hip ($p = .001$), with no significant difference at the femoral neck. Body weight was correspondingly lower by 6.8 kg in the semaglutide group ($p < .001$), consistent with a weight-loss-mediated bone effect rather than a direct antiosteogenic drug action (Hansen et al., 2024).

A parallel finding emerged from a secondary analysis of a randomized trial comparing exercise, liraglutide, and their combination following an initial low-calorie diet in adults with obesity (Jensen et al., 2024). Liraglutide monotherapy reduced hip and spine BMD significantly more than exercise alone, despite similar or greater weight loss, whereas the combination of exercise and liraglutide preserved BMD at a level statistically indistinguishable from placebo — suggesting that mechanical loading through exercise can substantially offset the BMD decline associated with pharmacologically induced weight loss (Jensen et al., 2024). Two recent narrative reviews reach a convergent conclusion: preclinical data support a direct osteoanabolic action of GLP-1RAs, but human data in non-diabetic populations show, at best, a neutral-to-modestly-negative effect on BMD and bone turnover markers, without clear evidence of increased fragility fracture risk at doses approved for obesity treatment (Karam et al., 2025; Anastasilakis et al., 2025). A recent clinical case report further illustrates the practical dimension of this concern, describing a postmenopausal woman who lost approximately 15% of body weight on semaglutide over one year and subsequently experienced two falls, prompting the authors to recommend earlier-than-standard monitoring of BMD and bone remodeling markers in similar patients (Ambrogini, 2026).

Indirect but instructive evidence comes from the orthopedic and musculoskeletal literature, where GLP-1RA use is increasingly encountered in the perioperative setting. A meta-analysis of GLP-1RA use in spinal fusion surgery — a bone-healing model with clear parallels to osseointegration — found that GLP-1RA use was associated with significantly reduced pseudarthrosis at 6 and 12 months (RR = 0.63 and 0.64, respectively), though this benefit did not persist at 24 months and was accompanied by a significantly increased risk of acute kidney injury (RR = 1.30) (Zahed et al., 2025). Two 2025-2026 orthopedic reviews likewise describe a heterogeneous picture: GLP-1RAs appear to promote osteoblastogenesis and restrain osteoclast activity mechanistically, and may aid perioperative glycemic and weight optimization, yet their net effect on fracture risk and bone healing in clinical practice remains incompletely defined, and perioperative management

must additionally account for delayed gastric emptying and its anesthetic implications (Restrepo Mejia et al., 2026; Gatto et al., 2025). No equivalent clinical dataset yet exists for dental implant osseointegration specifically in non-diabetic GLP-1RA users, which we identify as the principal evidence gap addressed in Section 4.

IV. Implications For Implant Dentistry

Extrapolating from orthopedic perioperative evidence

In the absence of direct implant-specific data in non-diabetic GLP-1RA users, the orthopedic perioperative literature offers the closest available clinical analogy. Two features of that literature are directly transferable to implant planning. First, the magnitude of BMD decline appears proportional to the magnitude and rate of weight loss, suggesting that patients in an active, rapid weight-loss phase — typically the first 6-12 months of GLP-1RA therapy — may represent a period of relatively greater skeletal vulnerability than patients who have reached a stable target weight (Kim & Cho, 2026; Hansen et al., 2024). Second, combined mechanical loading (through resistance exercise) appears to substantially mitigate BMD loss even during continued pharmacotherapy (Jensen et al., 2024), suggesting that skeletal health during GLP-1RA therapy is modifiable rather than fixed, an important consideration when counseling patients preparing for implant treatment.

Practical clinical considerations

Pending direct clinical trials, several pragmatic measures can reasonably be incorporated into implant treatment planning. Anamnesis should specifically capture GLP-1RA use, its indication (T2DM versus weight management), duration of therapy, and — where relevant — the magnitude and trajectory of recent weight loss, since these factors differentiate patients likely to benefit from a favorable pharmacological bone effect (diabetic patients with improved glycemic control) from those in whom mechanical unloading may be the dominant influence (non-diabetic patients with substantial recent weight loss). For patients in an active, rapid weight-loss phase, a conservative approach favoring delayed rather than immediate loading protocols may be prudent, extrapolating from the general principle — well established in the bariatric surgery literature — that periods of accelerated bone turnover warrant additional healing time before functional loading is introduced. In patients with additional risk factors for compromised bone quality (postmenopausal status, long-term GLP-1RA use, prior fragility fracture), consultation with the prescribing physician regarding baseline or interval BMD assessment may be warranted before extensive implant rehabilitation, consistent with the monitoring approach proposed in the general endocrinology literature (Kim & Cho, 2026; Ambrogini, 2026). Delayed gastric emptying associated with GLP-1RAs, though outside the bone-specific scope of this review, also carries recognized implications for perioperative sedation and anesthesia planning and should be addressed during informed consent (Restrepo Mejia et al., 2026).

A public health perspective

The expansion of GLP-1RA prescribing is not confined to private, higher-income settings. As access to these medications broadens within Brazil's public and supplementary health systems, implant dentists working within the Sistema Único de Saúde (SUS) and municipal health networks are increasingly likely to encounter patients on GLP-1RA therapy, frequently without a clear channel of communication with the prescribing physician. Given the divergent implications of diabetic versus weight-loss indications described in Sections 2.3 and 3, closer interdisciplinary communication between medical and dental teams — potentially formalized through shared referral or consultation protocols within public oral health services — represents a low-cost, high-value opportunity to reduce the risk of unrecognized skeletal vulnerability in this expanding patient population.

Research gaps and future directions

This review identifies a clear and specific gap: no published clinical study has examined implant survival, marginal bone loss, or osseointegration outcomes in non-diabetic patients using GLP-1RAs for weight management. Prospective cohort studies stratifying patients by GLP-1RA indication, duration of therapy, and weight-loss trajectory are needed to determine whether the favorable diabetic signal (Ahmad et al., 2025; Chen et al., 2024) or the unloading-mediated BMD decline seen in non-diabetic trials (Hansen et al., 2024; Jensen et al., 2024) predominates in real-world implant outcomes. Such studies would ideally incorporate bone turnover markers and standardized radiographic bone-level assessment, mirroring the methodology already established in the periodontal literature (Ahmad et al., 2025), and should examine whether adjunctive resistance exercise — shown to mitigate BMD loss in the general weight-loss literature (Jensen et al., 2024) — similarly protects peri-implant bone quality.

V. Conclusion

GLP-1 receptor agonists exert genuine, mechanistically well-supported osteoanabolic and anti-inflammatory effects on bone, with direct preclinical evidence of enhanced peri-implant osseointegration and clinical evidence of reduced peri-implant bone loss in patients with type 2 diabetes. However, this favorable evidence base cannot be extrapolated uncritically to the much larger and rapidly growing population of non-diabetic patients using these agents for weight management, in whom randomized clinical data show measurable BMD reduction proportional to weight loss itself. As GLP-1RA use becomes a routine feature of the medical history taken before implant treatment, implant dentists should distinguish between these two clinical scenarios, incorporate pragmatic screening and timing considerations pending direct evidence, and advocate for the prospective clinical research needed to define implant outcomes in this expanding and clinically heterogeneous population.

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