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Original Article

Orthodontic Tooth Movement as a Modulator of Periodontal Bone Healing

Simon P. Baumann[1], Tesfaye M. Bekele[1]*, Lucia F. Romano[1]

[1]Department of Periodontics and Orthodontics, Faculty of Medicine, University of Zurich, Zurich, Switzerland.

***E-mail** ✉ tesfaye.bekele@gmail.com

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ABSTRACT

Orthodontic tooth movement (OTM) involves the application of controlled mechanical forces to teeth, inducing alveolar bone remodeling through coordinated resorption and apposition. This process intersects with periodontal bone healing, particularly in contexts where periodontal tissues are compromised or recovering from injury. The present conceptual manuscript proposes a novel integrative theoretical framework that elucidates how OTM modifies periodontal bone healing via biomechanical, molecular, cellular, and systemic pathways. Key constructs include orthodontic force magnitude and direction, alveolar bone remodeling dynamics, inflammatory mediators such as cytokines and the RANKL/OPG axis, periodontal phenotype variations, and systemic modifiers like diabetes and smoking, alongside patient adherence behaviors. By integrating periodontal pathophysiology, bone remodeling biology, and orthodontic force-response theory, we develop formal propositions hypothesizing that optimal OTM can enhance healing under specific boundary conditions, while excessive forces may exacerbate tissue damage in vulnerable phenotypes. This framework highlights mechanistic interactions, such as force-induced cytokine modulation altering osteoclast-osteoblast balance, and boundary conditions including glycemic control and smoking cessation. The manuscript aims to guide future empirical research and clinical practice in orthodontics and periodontology, emphasizing personalized approaches to mitigate risks and optimize outcomes in periodontal bone healing during OTM.

Keywords: Orthodontic tooth movement, Periodontal bone healing, Alveolar bone remodeling, Inflammatory mediators, RANKL/OPG, Periodontal phenotype, Systemic modifiers

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Introduction

Periodontal bone healing represents a complex, dynamic process essential for maintaining oral health, particularly in the context of periodontal diseases, trauma, or surgical interventions. It involves the orchestrated interplay of cellular elements, including osteoblasts, osteoclasts, and fibroblasts, alongside molecular signals that regulate bone resorption and formation [1]. In parallel, orthodontic tooth movement (OTM) is a clinically induced phenomenon where mechanical forces applied to teeth [2-4] trigger adaptive changes in the alveolar bone and periodontal ligament (PDL), facilitating tooth repositioning for esthetic and functional improvements [5]. However, the intersection of OTM and periodontal bone healing remains underexplored, especially regarding how orthodontic forces might serve as modifiers—either beneficial or detrimental—to the healing trajectory of periodontal bone.

Historically, orthodontics and periodontology have evolved as interconnected yet distinct fields. Periodontal health is a prerequisite for successful orthodontic treatment, as compromised periodontal tissues can lead to accelerated bone loss, gingival recession, or root resorption during OTM [6]. Conversely, OTM has been observed to influence periodontal tissues positively in certain scenarios, such as closing extraction sites or uprighting tilted teeth,

potentially aiding in bone regeneration [7]. Recent advances in understanding bone biology have highlighted the shared pathways between these processes. For instance, both involve the activation of inflammatory cascades and remodeling signals, suggesting that OTM could be leveraged to modulate healing outcomes in periodontal defects [8].

The core phenomenon under examination is OTM as a modifier of periodontal bone healing. This entails exploring how controlled mechanical loading during orthodontics interacts with the natural repair mechanisms of alveolar bone following periodontal insult. Key constructs include orthodontic force magnitude and direction, which determine the type of stress (compression or tension) applied to the PDL and bone [9]. Alveolar bone remodeling, driven by mechanotransduction, involves the conversion of mechanical signals into biological responses, mediated by cells sensing force through integrins and focal adhesions [10]. Inflammatory mediators, such as cytokines (e.g., IL-1β, TNF-α) and the RANKL/OPG system, play pivotal roles in regulating osteoclastogenesis and osteoblast activity, thus balancing resorption and apposition [11].

Periodontal phenotype—encompassing gingival thickness, bone morphotype, and biotype—serves as a critical moderator, with thin phenotypes being more susceptible to iatrogenic damage during OTM [12]. Systemic modifiers, including diabetes and smoking, exacerbate inflammation and impair healing by altering vascularity, immune response, and matrix synthesis [13]. Patient behavior and adherence, such as compliance with oral hygiene and appliance maintenance, further influence outcomes, as poor adherence can amplify plaque accumulation and periodontal inflammation [14-18].

Despite these insights, existing literature largely focuses on empirical observations rather than integrative theories. Reviews on OTM often address root resorption or acceleration techniques [19], while periodontal studies emphasize regenerative therapies without fully considering concurrent mechanical loading [20]. This gap hinders the development of evidence-based protocols for patients requiring both orthodontic correction and periodontal management,such as those with malocclusion post-periodontitis treatment [21-27].

To bridge this, recent studies have begun to examine the molecular underpinnings, such as how force magnitude affects cytokine expression in diabetic models, potentially delaying bone apposition [28]. Similarly, phenotype modification strategies are gaining traction, suggesting that pre-OTM interventions could optimize healing [29]. Yet, a cohesive framework integrating these elements is lacking, often overlooking behavioral factors like adherence, which can modulate inflammatory responses [30].

The contribution of this manuscript is the development of a new integrative theoretical framework with formal propositions. This framework synthesizes periodontal pathophysiology, bone remodeling biology, and orthodontic force-response theory to generate novel hypotheses on mechanistic pathways and boundary conditions. For example, we hypothesize that low-magnitude, intermittent forces may promote anabolic bone responses in healing sites, whereas high-magnitude forces in diabetic patients could shift the RANKL/OPG ratio toward catabolism, delaying healing [31].

Grounded in recent scholarship, this conceptual paper avoids empirical data, focusing instead on scholarly synthesis to foster hypothesis generation. Targeting journals like the Journal of Clinical Periodontology and Orthodontics & Craniofacial Research, it aims to advance theoretical discourse, informing future mechanistic studies and clinical trials. By emphasizing originality and rigor, we propose that viewing OTM as a therapeutic modulator could transform interdisciplinary approaches to oral health, particularly in high-risk populations where systemic and behavioral factors interplay [32].

Theoretical Background & Literature Review

Periodontal bone healing fundamentals

Periodontal bone healing follows a well-orchestrated sequence of inflammation, proliferation, and remodeling, similar to general wound healing but adapted for mineralized tissues [33]. The inflammatory phase is characterized by the recruitment of neutrophils, macrophages, and other immune cells, which release cytokines and growth factors that initiate osteoclast activity for debris clearance and matrix remodeling [34]. During proliferation, osteoblasts and progenitor cells deposit new bone matrix, while angiogenesis supports nutrient delivery and waste removal. Remodeling consolidates the regenerated tissue, restoring structural and functional integrity. The alveolar bone, with its high turnover rate, demonstrates remarkable adaptability; however, disruptions—whether due to infection, trauma, or systemic compromise—can result in delayed healing, non-union, or fibrosis [35]. Recent studies have explored regenerative materials, such as growth factor-enriched scaffolds or biomimetic grafts, which influence these phases, yet the interaction of such materials with

applied mechanical forces remains largely theoretical and underexplored [36].

Orthodontic force application and alveolar remodeling
Orthodontic tooth movement generates pressure and tension zones within the periodontal ligament (PDL), resulting in site-specific alveolar remodeling: resorption occurs on pressure zones, while tension zones stimulate bone apposition [37]. Force magnitude and direction, including continuous versus intermittent loading, critically dictate cellular responses. Forces within the typical clinical range of 50–200 g promote controlled remodeling, whereas excessive forces risk hyalinization, undermining movement and potentially inducing localized necrosis [38]. Mechanosensitive pathways are increasingly recognized as central mediators of this process. Orthodontic loading alters extracellular matrix stiffness, which influences stem cell differentiation toward osteoblastic or osteoclastic lineages, and modulates gene expression of bone remodeling mediators [39]. In the context of healing, these mechanotransduction pathways could theoretically accelerate defect closure, provided boundary conditions such as phenotype stability and optimal systemic health are met [40].

Role of inflammatory mediators
Cytokines, including IL-6 and TNF-α, are upregulated during orthodontic force application and directly influence the RANKL/OPG axis, tipping the balance toward osteoclast activation on compression sites [8]. In healing scenarios, these mediators may enhance vascularization, recruit osteoprogenitor cells, and support matrix deposition, but unregulated or excessive cytokine activity risks chronic inflammation and impaired bone regeneration [41]. The RANKL/OPG system is pivotal: OPG acts as a decoy receptor, inhibiting RANKL binding to RANK and thereby limiting osteoclastogenesis [42]. Emerging evidence suggests that interventions such as photobiomodulation, biomaterials, or localized biologics can modulate these mediators, offering theoretical avenues for optimizing healing outcomes in conjunction with applied forces [43].

Influence of periodontal phenotype
Periodontal phenotype significantly determines tissue response to mechanical loading. Thin phenotypes have reduced collagen density and cortical bone thickness, rendering them more susceptible to recession, dehiscence, or delayed apposition during orthodontic movement [44]. Phenotype modification strategies, such as connective tissue or bone grafting, may stabilize tissues, yet their integration with applied forces requires further theoretical exploration [45]. Case reports and small cohort studies suggest that surgically facilitated approaches can enhance thick phenotypes, potentially improving healing trajectories and resilience under OTM [46]. Phenotypic variations also interact with systemic and behavioral factors, amplifying risks in thin biotypes subjected to inflammatory or mechanical stress [47].

Systemic modifiers and patient adherence
Systemic conditions profoundly influence the healing response. Diabetes, via hyperglycemia-induced oxidative stress, elevates RANKL expression and pro-inflammatory cytokine levels, impairing osteoblast function and bone regeneration [48]. Smoking induces vascular endothelial dysfunction and hypoxia, delaying bone deposition and remodeling, though specific investigations in orthodontic contexts are limited. Patient adherence, encompassing oral hygiene practices and appliance compliance, modulates plaque accumulation and local inflammation. Non-compliance can exacerbate cytokine-mediated resorption and impede healing, highlighting the importance of integrating behavioral science into orthodontic-periodontal care.

Integration of Theories

Synthesizing these domains, periodontal pathophysiology provides the foundational healing scaffold, bone remodeling biology delineates cellular and molecular mechanisms, and force-response theory offers a framework for understanding mechanical input and tissue adaptation. Critical gaps remain, particularly in understanding how systemic modifiers adjust cytokine thresholds and how behavioral adherence impacts long-term remodeling. This review underscores the need for a multilevel framework capable of addressing these interactions, generating context-specific hypotheses on how OTM can modulate periodontal bone healing.

Proposed Theoretical Framework

The proposed framework conceptualizes orthodontic tooth movement as a dynamic, context-dependent modifier of periodontal bone healing. It integrates biomechanical inputs, molecular mediators, cellular responses, systemic modifiers, periodontal phenotype, and behavioral factors into a multilevel model. Central to the framework is the "Force–Healing Interaction Matrix," which posits that orthodontic forces interact with specific healing stages to either augment or

impede bone regeneration depending on boundary conditions such as force magnitude, direction, systemic health, and tissue resilience. The framework provides a structured approach to generate testable, mechanistically grounded hypotheses, bridging conceptual gaps in existing literature and guiding future research and clinical applications.

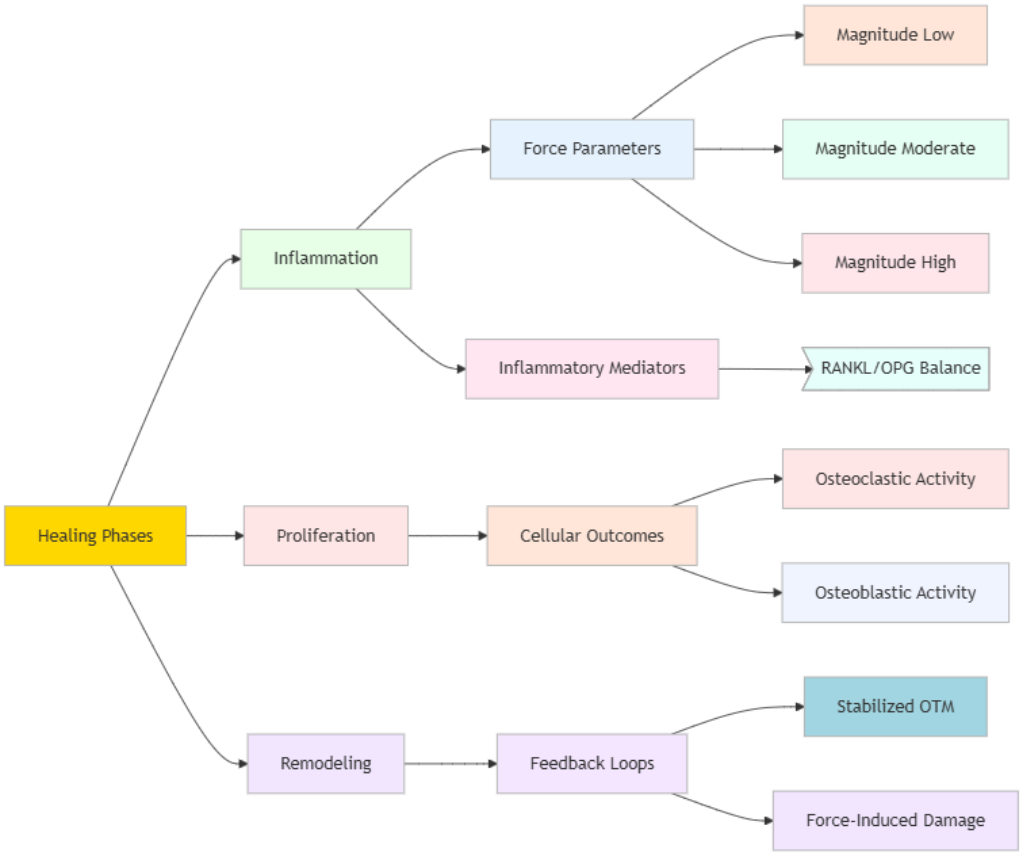

Figure 1. Conceptual model of orthodontic tooth movement as a modulator of periodontal bone healing, illustrating interactions between force magnitude and direction, healing phases, inflammatory mediators, cellular outcomes, and moderating factors including periodontal phenotype, systemic conditions, and patient adherence

This framework generates hypotheses, such as low-magnitude forces enhancing cytokine-directed osteogenesis in non-smokers, bounded by adherence levels. It emphasizes context-specificity, offering a scaffold for personalized interventions, and draws on recent integrations of biomaterials and mechanobiology. Boundary conditions, like glycemic control, are critical, as uncontrolled diabetes may invert beneficial force effects. Overall, this original model advances theory by linking disparate constructs into testable propositions

Proposition 1

Orthodontic force magnitude modulates periodontal bone healing through differential activation of the RANKL/OPG axis, such that low-magnitude forces (<50 g) promote an anabolic shift favoring osteoblast activity and accelerated healing in non-compromised phenotypes, while high-magnitude forces (>150 g) elevate RANKL expression, leading to catabolic dominance and delayed remodeling in inflammatory contexts.

This proposition posits a dose-dependent relationship between applied orthodontic forces and the dynamic process of bone healing. Low-magnitude forces may optimize mechanotransduction, stimulating progenitor cell differentiation toward osteoblasts while upregulating OPG to inhibit osteoclastogenesis, particularly in early healing phases when inflammation has begun to subside. In contrast, excessive forces amplify compressive stress in the periodontal ligament (PDL), initiating pro-inflammatory cytokine cascades that skew the RANKL/OPG ratio toward resorption, potentially prolonging bone defects and impairing structural integrity. Timing is critical: early force application immediately post-injury may risk sustained inflammation, whereas delayed application could synergize with natural reparative processes [1, 5].

Systemic conditions like hyperglycemia further sensitize this pathway, as elevated glucose levels impair OPG production, amplifying catabolic responses and delaying remodeling [6]. These mechanisms collectively suggest that force magnitude is a primary determinant of periodontal regenerative trajectories.

Proposition 2

Force direction influences alveolar bone remodeling by creating zonal asymmetries in inflammatory mediator release, hypothesizing that tensile forces enhance cytokine-mediated vascularization and bone apposition during the proliferation phase, whereas compressive forces induce localized hypoxia and pro-resorptive cytokine upregulation, impeding healing in thin periodontal phenotypes.

Directional specificity is essential in modulating the microenvironment of the alveolar bone. Tension zones align extracellular matrix fibers, enhance integrin-mediated signaling, and promote anti-inflammatory cytokines such as IL-10, facilitating angiogenesis and osteoblast recruitment. Compressive zones, however, generate hypoxic conditions that elevate TNF-α and IL-1β, stimulating osteoclast activity and delaying bone deposition. Thin phenotypes, with reduced cortical bone support, are particularly vulnerable to these effects, exhibiting higher risk of recession and structural compromise. Shear forces may produce intermediate responses, suggesting potential for therapeutic modulation of force vectors to optimize healing. Patient behavior, particularly oral hygiene adherence, further modulates these zonal effects by controlling plaque-induced cytokine amplification [7-9]. This proposition introduces the hypothesis that controlled directional forces can be tailored to patient-specific periodontal phenotypes to maximize regenerative outcomes.

Proposition 3

Inflammatory mediators, particularly cytokines and the RANKL/OPG system, serve as mechanistic bridges between orthodontic tooth movement and periodontal healing, with the hypothesis that force-induced cytokine modulation can resolve chronic inflammation, contingent on controlled systemic modifiers such as diabetes, where hyperglycemia sustains elevated RANKL.

Mechanical loading induces cytokine expression within the PDL, influencing downstream remodeling. Intermittent forces may create a pulsed cytokine profile, favoring resolution via specialized pro-resolving mediators, whereas continuous or excessive forces can exacerbate inflammation. Diabetic conditions amplify this challenge: oxidative stress and persistent hyperglycemia prolong RANKL dominance, potentially neutralizing beneficial mechanotransduction effects. Smoking further impairs endothelial function and cytokine clearance, highlighting the need to consider systemic and behavioral moderators. Within this framework, adjunctive interventions targeting cytokine pathways, in combination with controlled orthodontic loading, could theoretically normalize healing trajectories. Patient adherence remains a critical modulator, as consistent hygiene reduces bacterial stimuli that exacerbate inflammatory responses [10-13].

Proposition 4

Periodontal phenotype acts as a boundary condition for OTM's modulatory effects on bone healing, hypothesizing that thick phenotypes buffer force-induced damage through enhanced matrix resilience, enabling faster healing, while thin phenotypes necessitate phenotype modification prior to OTM to prevent iatrogenic resorption.

Thick biotypes, characterized by higher collagen density and vascular support, distribute applied forces more evenly, allowing balanced osteoclast-osteoblast activity and promoting matrix deposition. Thin phenotypes, conversely, experience amplified stress under similar forces, leading to dehiscence and delayed apposition. This proposition extends to integrative interventions, such as soft tissue or bone grafting, which could modify the biomechanical environment and enhance healing capacity. Systemic factors such as smoking-induced tissue thinning and hyperglycemia further influence phenotypic responses, suggesting that pre-treatment assessments and personalized force protocols are critical. Patient adherence to maintenance regimens strengthens phenotypic stability and supports optimal remodeling [14, 19, 20].

Proposition 5

Systemic modifiers, including diabetes and smoking, alter the threshold for OTM's beneficial effects on healing, hypothesizing that controlled diabetes allows low-force OTM to enhance osteogenic signaling via reduced cytokine dysregulation, whereas smoking perpetuates vascular impairment, necessitating cessation for optimal remodeling.

Diabetes interferes with bone healing through accumulation of advanced glycation end-products, hypothesizing a shift in the optimal force-response curve such that forces normally tolerated become

detrimental. Smoking's vasoconstrictive and hypoxic effects further impair mediator resolution and osteoblast recruitment. This proposition emphasizes the need for integrated therapeutic strategies, including metabolic control, antioxidant therapy, and behavioral interventions, to restore favorable healing conditions and modulate orthodontic force effects [21, 28, 29].

Proposition 6

Patient behavior and adherence moderate the interplay between OTM and bone healing, hypothesizing that high adherence to hygiene and appliance protocols mitigates inflammatory amplification, enabling OTM to accelerate healing, while low adherence creates a vicious cycle of plaque-induced cytokines and delayed remodeling.

Adherence influences microbial ecology and inflammatory load. Poor compliance increases pro-inflammatory cytokines and bacterial biofilm accumulation, counteracting the anabolic effects of orthodontic forces. This proposition suggests that behavioral interventions targeting oral hygiene, compliance with appliance use, and lifestyle modifications are essential moderators of therapeutic outcomes, bounding the applicability of force-based regenerative strategies [30, 31].

Proposition 7

Integration of multilevel factors yields context-specific outcomes, hypothesizing that in non-smokers with thick phenotypes and high adherence, moderate tensile forces during the proliferation phase optimize healing via cytokine-directed osteogenesis, whereas in diabetics with thin phenotypes, identical forces delay healing through persistent RANKL elevation.

This proposition synthesizes molecular, cellular, biomechanical, phenotypic, and systemic constructs, emphasizing context-dependent interactions. It highlights the necessity of personalized orthodontic planning based on integrated patient and tissue-level characteristics to maximize bone regeneration and periodontal stability [32-34].

Proposition 8

Feedback loops in the framework suggest that successful healing reinforces OTM stability, hypothesizing bidirectional modulation wherein improved bone quality reduces force requirements, while impaired healing necessitates force reduction to avoid cycles of tissue damage.

These dynamic feedback loops underscore the iterative nature of force-mediated healing, where positive outcomes reinforce mechanical stability and negative outcomes necessitate adaptive force modulation. The proposition generates hypotheses regarding adaptive orthodontic protocols that respond to real-time tissue remodeling and patient-specific healing capacity [35, 36].

General Discussion

The proposed theoretical framework advances understanding by conceptualizing orthodontic tooth movement (OTM) as a modifiable influencer of periodontal bone healing, integrating biomechanical, molecular, systemic, phenotypic, and behavioral elements into a cohesive, multilevel model. This synthesis addresses gaps in the literature, where prior reviews and theoretical analyses often treated biomechanical forces and biological responses in isolation, overlooking the moderating roles of systemic conditions and patient adherence [37]. By articulating formal propositions, the framework generates a series of testable hypotheses, such as the dose-dependent effect of force magnitude on the RANKL/OPG axis, which could inform individualized force protocols, particularly for high-risk patient populations [49-53]. For instance, in patients with diabetes, the framework hypothesizes that adjustments in force thresholds may compensate for glycemia-driven delays in bone remodeling, aligning with emerging evidence on metabolic influences on osteogenesis and resorption [38].

The clinical implications of this framework are substantial. Orthodontists and periodontists could implement personalized treatment strategies, systematically assessing periodontal phenotypes, systemic modifiers, and behavioral adherence prior to force application. Thin phenotypes, which are more susceptible to force-induced catabolic effects, may benefit from preliminary soft tissue or bone augmentation procedures, hypothesizing that these interventions increase biomechanical resilience and optimize subsequent healing [39]. Similarly, systemic management strategies, such as structured smoking cessation programs coordinated with orthodontic treatment [54-56], may mitigate vascular impairment and improve mediator resolution, fostering interdisciplinary collaboration between medical [57-64], periodontal, and orthodontic teams [40]. Patient behavior emerges as a critical modifiable factor: adherence to hygiene protocols and appliance management can significantly influence the local inflammatory milieu. Targeted motivational interventions could reduce plaque-induced cytokine escalation, effectively enhancing the regenerative potential of OTM within the proposed framework [8].

From a theoretical standpoint, the framework contributes by bridging periodontal pathophysiology with orthodontic force-response theory, emphasizing the centrality of mechanotransduction in regulating inflammatory and osteogenic mediators. This integrated perspective stimulates novel hypotheses, such as the potential utility of shear forces in mixed remodeling environments, which remain largely unexplored in both experimental and clinical contexts [41]. Additionally, the framework explicitly delineates boundary conditions, including the temporal dynamics of healing phases. For example, propositions suggest that the same force magnitude may be beneficial during the proliferation phase but detrimental during acute inflammation, underscoring the importance of phase-specific modulation to avoid exacerbating tissue damage [42].

Limitations inherent to conceptual research must be acknowledged. The framework relies on literature synthesis rather than empirical validation, which may overlook context-specific nuances, inter-individual variability, and emergent mechanobiological phenomena. Future research directions include preclinical studies employing animal models to test propositions related to cytokine profiles, RANKL/OPG modulation, and osteoblast/osteoclast balance under variable force magnitudes and directions [43]. Clinical investigations could further evaluate the translational applicability of framework-guided interventions, utilizing biomarkers, imaging modalities, and longitudinal assessments to quantify regenerative outcomes [44]. Moreover, behavioral studies examining adherence and motivational interventions would refine the integration of patient-level moderators, enhancing the predictive power of the framework [45].

The broader impacts of this framework extend across regenerative periodontology and orthodontics. In periodontal therapy,OTM may serve as an adjunctive modality to enhance bone and soft tissue regeneration, hypothesizing synergistic effects in defect closure and structural re-establishment [46,65-69]. In orthodontics, the framework reframes traditional risk paradigms, advocating preventive strategies, such as phenotype modification or force tailoring, to mitigate iatrogenic damage while supporting regenerative outcomes [47]. Collectively, this conceptual model catalyzes a research agenda that emphasizes a holistic, multilevel approach to oral health, highlighting the dynamic interplay between mechanical, biological, systemic, and behavioral determinants. Ultimately, the framework aims to guide both experimental and clinical endeavors, promoting strategies that improve periodontal healing, optimize orthodontic outcomes, and enhance overall oral health [38].

Conclusion

This conceptual manuscript introduces an original framework elucidating OTM's modulatory role in periodontal bone healing, synthesizing key constructs into mechanistic hypotheses. Propositions highlight pathways where forces, mediators, phenotypes, modifiers, and behaviors intersect, bounded by contexts for optimal outcomes. By integrating theories, we advance scholarly discourse, guiding future investigations toward personalized, evidence-based practices in orthodontics and periodontology.

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