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**Original Article**

# Biologically Informed Decision-Making Framework for Orthodontic Treatment in Reduced Periodontium

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## ABSTRACT

Orthodontic treatment in patients with reduced periodontium poses significant clinical challenges due to heightened risks of further periodontal breakdown, unpredictable tooth movement, and compromised long-term stability. Existing approaches often rely on empirical guidelines or clinician experience, lacking systematic integration of biological principles. This conceptual paper proposes a novel decision-making framework that translates periodontal biology, bone remodeling mechanisms, and mechanobiological responses into structured orthodontic planning for reduced-periodontium cases. The framework incorporates key constructs including periodontal support loss, alveolar bone biology, inflammatory burden, orthodontic force magnitude, tooth movement biomechanics, risk stratification, treatment sequencing, and biologic limits of tooth movement. It delineates a stepwise process: initial risk assessment based on periodontal status and inflammatory markers; modulation of force systems to respect mechanobiological thresholds; sequencing of periodontal stabilization prior to orthodontics; and adaptive monitoring to prevent exceeding biologic limits. This synthesis draws from established theories in periodontal biology and mechanobiology, offering a clinically actionable logic that bridges translational gaps. The framework's value lies in enhancing treatment predictability, minimizing iatrogenic risks, and optimizing outcomes in vulnerable patients, potentially informing future guidelines for interdisciplinary care in orthodontics and periodontology. By prioritizing biological fidelity over heuristic methods, it advances risk-based decision-making in complex cases.

**Keywords:** Reduced periodontium, Orthodontic decision framework, Bone remodeling, Mechanobiology, Risk stratification, Treatment sequencing, Biologic limits

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## Introduction

The integration of orthodontic therapy in patients with reduced periodontium represents one of the most persistent and complex clinical dilemmas in contemporary dental practice. Reduced periodontium most commonly the consequence of chronic or previously aggressive periodontitis is defined by irreversible loss of alveolar bone support, diminished periodontal ligament (PDL) volume, altered vascularity, and frequently concomitant gingival recession [1-4]. These structural and biological changes fundamentally transform the biomechanical environment in which orthodontic tooth movement occurs. Unlike the intact periodontium, where force-induced remodeling follows relatively predictable patterns, the compromised periodontal apparatus exhibits reduced adaptive capacity and heightened vulnerability to mechanical overload [5-7].

Paradoxically, patients with reduced periodontium often exhibit the greatest need for orthodontic intervention. Pathologic tooth migration, flaring, spacing, extrusion, and occlusal collapse are well-documented sequelae of periodontal breakdown, particularly in the anterior region [8-10]. These secondary malocclusions compromise not only esthetics but also function, phonetics, and long-term occlusal stability. Orthodontic correction, therefore, is frequently indispensable for comprehensive rehabilitation, facilitating plaque control, redistributing occlusal forces, and enabling prosthetic or restorative treatment [11, 12]. Yet, the same orthodontic forces required to reposition teeth may, if improperly calibrated, accelerate periodontal destruction, precipitating further attachment loss, root resorption, or even tooth loss [13]. This inherent tension between therapeutic necessity and biological risk—lies at the core of orthodontic decision-making in reduced-periodontium patients.

The clinical relevance of this challenge is magnified by demographic and epidemiologic trends. Periodontal disease prevalence increases markedly with age, affecting nearly half of adults to varying degrees, while moderate to severe forms disproportionately impact older populations [14, 15]. Concurrently, demand for adult orthodontic treatment has risen steadily over the past two decades, driven by advances in appliance technology, increased esthetic awareness, and expanded indications for interdisciplinary care [14]. As a result, orthodontists are increasingly confronted with patients whose periodontal histories complicate conventional treatment paradigms. Despite this reality, orthodontic education and clinical protocols have historically been rooted in assumptions derived from healthy periodontal conditions, limiting their applicability to compromised cases [16].

A central limitation of traditional orthodontic planning lies in its insufficient attention to the biological heterogeneity of the reduced periodontium. Standardized force magnitudes and biomechanical strategies, which may be well tolerated in intact alveolar bone, can generate excessive strain concentrations in teeth supported by diminished bone height and surface area [17]. Reduced periodontal support effectively increases the moment-to-force ratio acting on teeth, magnifying stress within the PDL and adjacent bone [18]. Moreover, inflammatory priming of periodontal tissues alters cellular responsiveness, biasing remodeling toward resorptive pathways even under relatively modest mechanical stimuli. These factors collectively undermine the validity of "one-size-fits-all" orthodontic mechanics and underscore the need for biologically adaptive approaches.

Current clinical management of orthodontic treatment in reduced-periodontium patients is largely guided by periodontal consensus statements and expert recommendations, which emphasize the importance of achieving periodontal health prior to orthodontic intervention [19, 20]. These guidelines advocate for non-surgical periodontal therapy, meticulous plaque control, and, where indicated, regenerative procedures, followed by reassessment before orthodontic forces are applied [21]. While such recommendations are indispensable for disease control, they remain largely procedural and stop short of addressing how biological parameters should inform orthodontic force systems, movement strategies, or treatment sequencing [22, 23]. For example, guidelines rarely specify how the degree of bone loss should translate into allowable force magnitudes, acceptable movement types, or safe displacement limits.

In the absence of biologically explicit guidance, clinical decision-making often defaults to experience-driven heuristics. Orthodontists may rely on intuition, anecdotal evidence, or conservative instincts when treating periodontally compromised patients, leading to substantial inter-clinician variability [24]. Some practitioners may adopt overly cautious approaches, applying minimal forces that prolong treatment duration and increase the risk of patient noncompliance. Others may underestimate biologic vulnerability, inadvertently applying force levels that exceed tissue tolerance, resulting in adverse outcomes [25]. Such variability undermines reproducibility, complicates interdisciplinary collaboration, and limits the ability to systematically evaluate treatment outcomes across settings.

The consequences of these limitations are reflected in clinical outcome data. Reports suggest that approximately 20–30% of orthodontically treated patients with reduced periodontium experience some degree of additional attachment loss during or after treatment, despite apparent disease control [26]. While not all such deterioration is attributable to orthodontic forces alone, these figures highlight deficiencies in current risk mitigation strategies. Moreover, complications such as increased mobility, gingival recession, and compromised esthetic outcomes remain significant concerns, particularly in the anterior region [14]. These challenges point to a fundamental gap between advances in periodontal biology and their translation into orthodontic decision-making.

Another critical shortcoming of existing models is the persistent disciplinary silo between orthodontics and periodontology. Although interdisciplinary care is widely advocated, collaboration often occurs sequentially rather than integratively, with limited shared decision logic [27, 28]. Advances in bone biology, mechanotransduction, and inflammatory signaling have dramatically expanded understanding of how tissues respond to mechanical forces, yet these insights are rarely operationalized into orthodontic planning frameworks [29, 30]. As a result, treatment remains reactive—responding to complications after they arise—rather than anticipatory and preventive.

Addressing these gaps requires a conceptual shift toward a biologically informed decision-making framework that explicitly integrates periodontal risk factors into orthodontic planning and execution. Such a framework must move beyond binary determinations of "disease present" or "disease controlled" and instead capture gradations of biological vulnerability [31-33]. Key determinants—including residual alveolar bone height, inflammatory burden, systemic modifiers, and tissue remodeling capacity—should collectively inform decisions regarding force magnitude, biomechanics, movement limits, and treatment sequencing [34]. By embedding biological intelligence into clinical logic, orthodontic care can be aligned more closely with tissue capacity rather than appliance capability.

From a theoretical standpoint, this approach is grounded in well-established principles of bone remodeling and mechanobiology. Frost's mechanostat theory posits that bone adapts to mechanical strain within defined thresholds, with excessive or insufficient strain leading to resorption or disuse atrophy, respectively [35, 36]. In the context of reduced periodontium, these thresholds are shifted downward due to diminished bone volume and compromised vascular supply, narrowing the window of adaptive response [37, 38]. Concurrently, chronic or residual inflammation—mediated by cytokines such as interleukin-1β, tumor necrosis factor-α, and prostaglandins—sensitizes tissues to mechanical stimuli, amplifying catabolic remodeling even at lower strain levels [39]. These interactions necessitate force modulation and strategic sequencing to avoid biologic overload.

Risk stratification models, long utilized in medicine and periodontology, offer a promising scaffold for orthodontic application [40]. By categorizing patients according to biologic risk profiles—incorporating severity of support loss, inflammatory status, and systemic conditions—clinicians can tailor orthodontic mechanics to individual tolerance levels. Similarly, treatment sequencing that prioritizes periodontal stabilization before orthodontic force application respects biologic healing timelines, while explicit recognition of biologic limits prevents movements that exceed alveolar housing capacity [41, 42]. Together, these principles form the foundation of a biologically coherent decision pathway.

The present paper proposes an original conceptual framework that synthesizes periodontal biology, mechanobiology, and orthodontic biomechanics into a unified clinical decision model for patients with reduced periodontium. Rather than offering prescriptive rules, the framework provides a structured logic through which biological variables are translated into treatment choices. By bridging theory and practice, it aims to enhance predictability, reduce iatrogenic risk, and promote meaningful interdisciplinary collaboration. Ultimately, this biologically informed approach seeks to reposition orthodontic treatment in reduced-periodontium patients from a high-risk endeavor to a controlled, evidence-aligned therapeutic process.

## Theoretical Background & Literature Review

### Periodontal Tissue Biology and Reduced Periodontium

The periodontium comprises the gingival tissues, periodontal ligament (PDL), cementum, and alveolar bone, functioning as a dynamic unit that supports tooth anchorage and absorbs occlusal forces [27, 43]. In health, the alveolar bone maintains equilibrium through continuous remodeling, balanced by osteoblastic apposition and osteoclastic resorption [29]. Reduced periodontium arises primarily from periodontitis, an inflammatory condition driven by microbial dysbiosis and host immune responses, leading to progressive attachment loss and alveolar bone resorption [31, 44]. Biologically, this involves elevated proinflammatory cytokines (e.g., IL-1, TNF-α) that activate osteoclastogenesis via RANKL pathways, resulting in net bone loss [34,44].

In reduced periodontium, alveolar bone exhibits diminished density and height, often with dehiscences or fenestrations, altering load distribution [35,45]. The PDL, crucial for force transduction, thins and loses collagen fiber organization, impairing its shock-absorbing capacity [37]. These changes elevate susceptibility to further breakdown under mechanical stress, as the biologic threshold for adaptive remodeling is compromised [39, 46]. Clinically, this manifests as increased tooth mobility and potential for secondary occlusal trauma [40,47].

Bone Remodeling and Mechanobiology of Orthodontic Tooth Movement Orthodontic tooth movement (OTM) relies on alveolar bone remodeling, a mechanobiologically mediated process where applied forces induce strain in the PDL, triggering cellular responses [41]. According to mechanostat theory, optimal strains (1500-2500 microstrain) promote balanced resorption on compression sides and apposition on tension sides [24]. In reduced periodontium, however, baseline bone deficits lower this window, with strains easily exceeding pathologic thresholds (>4000 microstrain), leading to hyalinization and delayed movement [39, 48].

Mechanobiology involves PDL fibroblasts sensing forces via integrins and cytoskeletal elements, releasing mediators that recruit osteoclasts and osteoblasts [49]. In compromised tissues, persistent inflammation amplifies catabolism, potentially causing root resorption or bone defects [50]. Recent models emphasize fluid flow and piezoelectric effects in bone, but in reduced periodontium, these are disrupted by altered vascularity [51].

Biological Risk Factors Influencing Orthodontic Outcomes Key risks include severity of periodontal support loss, quantified by probing depths and bone levels, which correlate with increased OTM complications [52]. Inflammatory burden, marked by elevated cytokines, exacerbates tissue breakdown during force application [31]. Systemic factors like diabetes or smoking impair remodeling, while genetic polymorphisms in IL-1 genes heighten susceptibility [53, 54]. Biomechanical risks arise from force magnitude; light forces (20-50g) are advocated to minimize overload [55].

Gaps in Current Orthodontic Decision-Making Models Existing models focus on empirical protocols but neglect integrated biologic risk assessment [19]. Guidelines recommend sequencing but lack specificity on force modulation or limits [56]. Risk stratification is rudimentary, often ignoring mechanobiological thresholds, leading to variable outcomes [26]. A conceptual framework addressing these gaps is warranted.

Proposed Theoretical Framework The proposed framework is a conceptual decision-making tool that integrates periodontal biology, mechanobiology, and risk-based logic to guide orthodontic treatment in reduced-periodontium patients. It is visualized in **Figure 1** as a flowchart with three primary phases: Risk Assessment, Treatment Planning, and Execution/Monitoring. Each phase branches into decision nodes informed by biological constructs, ensuring translational application.

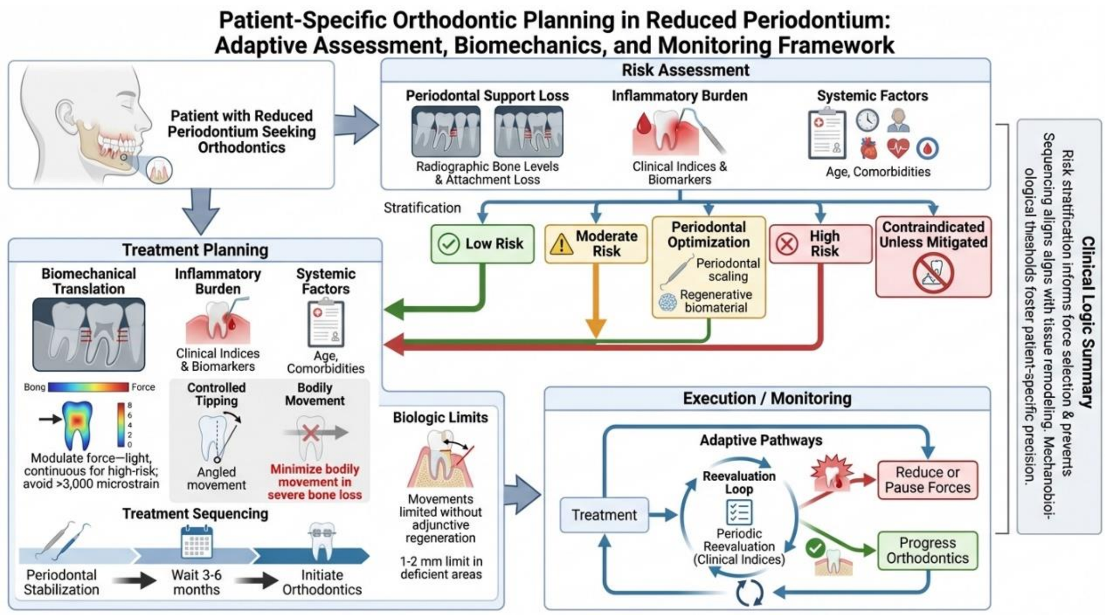


**Figure 1.** depicts a vertical flowchart starting with an entry point: "Patient with Reduced Periodontium Seeking Orthodontics."

This framework's clinical logic bridges biology to choices: risk stratification informs force selection, preventing overload in fragile tissues; sequencing aligns with remodeling timelines, optimizing outcomes [57]. It advances beyond static guidelines by embedding mechanobiological thresholds, fostering precise, patient-specific plans [16].

**Clinical Propositions**

Grounded in the proposed biologically informed decision framework, the following clinical propositions translate foundational periodontal and mechanobiological principles into explicit, testable expectations for orthodontic management in patients with reduced periodontium. Collectively, these propositions serve two functions: (a) they provide immediate guidance for risk-sensitive clinical reasoning, and (b) they delineate clear avenues for future empirical validation through prospective and mechanistic studies.

*Proposition 1: Integrated Biological Risk Stratification Will Outperform Periodontal Staging Alone In Predicting Orthodontic Complications.*

Risk stratification that simultaneously incorporates periodontal support loss, current inflammatory burden, and systemic host modifiers will predict orthodontic-related complications—such as progressive attachment loss, increased mobility, or treatment interruption—more accurately than reliance on periodontal staging or grading alone. In particular, patients classified as high risk (e.g., >50% alveolar bone loss combined with persistent bleeding on probing or elevated inflammatory markers) are expected to experience disproportionate tissue breakdown when exposed to conventional orthodontic force systems.

From a mechanobiological perspective, unmodified forces in such cases are likely to exceed reduced strain tolerance thresholds, amplifying catabolic remodeling pathways and exacerbating attachment loss [52]. By contrast, stratified decision-making enables preemptive force modulation, potentially maintaining applied forces below approximately 50 g, thereby attenuating overload-induced damage. This proposition positions biological risk stratification as a superior prognostic tool and a prerequisite for safe orthodontic intervention in compromised tissues.

*Proposition 2: Orthodontic force magnitudes calibrated to alveolar bone biology will enhance efficiency while preserving periodontal integrity*

Modulating orthodontic force magnitude in accordance with alveolar bone volume, density, and remodeling capacity will optimize tooth movement efficiency without compromising periodontal health in reduced-periodontium cases. Specifically, light continuous forces in the range of 20–50 g are expected to align with lowered strain thresholds (approximately 1500–3000 microstrain) characteristic of diminished periodontal support [55].

This biologically calibrated force application is hypothesized to favor adaptive remodeling over pathologic responses by minimizing hyalinization, preserving vascular perfusion within the periodontal ligament, and sustaining osteoclastic–osteoblastic coupling. Consequently, force calibration should enhance predictability of movement trajectories while reducing risks of root resorption and further attachment loss. This proposition challenges conventional force prescriptions by asserting that efficiency in compromised tissues derives from biological compatibility rather than force magnitude alone.

*Proposition 3: Sequenced periodontal stabilization prior to orthodontic force application will improve long-term treatment stability.*

Orthodontic treatment plans that prioritize a mandatory periodontal stabilization phase of at least 3–6 months before force application will demonstrate superior long-term stability compared with concurrent or immediate orthodontic intervention. This sequencing respects known timelines of bone remodeling and inflammatory resolution, allowing for downregulation of proinflammatory cytokines and partial restoration of tissue homeostasis [14].

By initiating orthodontic mechanics only after inflammatory burden is controlled, mechanical forces are applied to a biologically receptive environment, reducing the likelihood of relapse driven by unstable periodontal support. This proposition emphasizes that treatment sequencing is not merely procedural but biologically determinative, with direct implications for retention outcomes and maintenance of periodontal gains.

*Proposition 4: Controlled tipping biomechanics will yield safer outcomes than bodily movement in cases of severe periodontal support loss*

In patients with advanced periodontal support reduction, orthodontic mechanics that prioritize controlled tipping over bodily tooth movement will better preserve biologic limits and minimize adverse sequelae. Controlled tipping is expected to distribute stresses more evenly across a thinned periodontal ligament and reduced alveolar housing, thereby lowering peak stress concentrations that predispose to cortical plate perforation and root resorption [58].

This biomechanical strategy is particularly relevant in esthetically sensitive regions, where excessive or uncontrolled movement may precipitate gingival recession or compromise soft tissue contours. The proposition asserts that biomechanical choice is a biologically consequential decision and that conservative movement strategies can expand the safe envelope of orthodontic care in compromised patients.

*Proposition 5: Adaptive monitoring with biologic feedback will improve treatment success in complex reduced-periodontium cases*

Incorporating adaptive monitoring pathways—including periodic clinical reassessment and, where feasible, biologic indicators such as inflammatory cytokine profiles—will enable real-time treatment adjustments that prevent exceedance of tissue tolerance thresholds. When signs of recurrent inflammation emerge, proactive force reduction or temporary cessation is expected to restore remodeling balance and avert irreversible damage [57].

This proposition anticipates that dynamic, feedback-informed decision-making will enhance overall treatment success rates in complex cases by shifting care from reactive correction to anticipatory modulation. Adaptive monitoring thus functions as the operational backbone of the framework, reinforcing its departure from static, one-time planning models.

## Discussion

The conceptual framework advanced in this manuscript addresses a persistent translational gap in orthodontic management of patients with reduced periodontium by explicitly embedding biological determinants into clinical decision-making. Traditional orthodontic approaches in such patients have often relied on experiential judgment or static guideline interpretation, frequently overlooking the dynamic interplay between mechanobiology, inflammation, and compromised tissue architecture [54]. By contrast, the present framework integrates periodontal biology, bone remodeling theory, and orthodontic biomechanics into a coherent, executable logic that aligns with contemporary understandings in both periodontology and orthodontics [16].

A central premise of the framework is that reduced periodontium fundamentally alters the mechanostat set points governing bone adaptation. Loss of alveolar support narrows the window between adaptive and destructive strain, rendering conventional force systems potentially injurious [24]. Concurrently, elevated inflammatory burden—mediated by cytokines such as interleukin-1β and tumor necrosis factor-α—biases remodeling toward catabolism, further reducing tissue tolerance [34]. The proposed decision pathways operationalize these insights by linking risk assessment directly to force selection, biomechanics, and sequencing, thereby minimizing the likelihood of biologic overload and unintended tissue loss [40].

Importantly, the framework extends beyond existing periodontal staging and grading systems, which, while valuable for disease characterization, offer limited guidance for orthodontic mechanics [31]. By introducing mechanobiological thresholds and biologically grounded movement limits, the model provides a novel interdisciplinary bridge between diagnosis and execution. This integration supports a precision-oriented paradigm akin to personalized medicine, wherein treatment intensity and strategy are tailored to individual biologic capacity rather than averaged norms [56].

From a theoretical standpoint, the framework adapts Frost's mechanostat theory to compromised periodontal environments, proposing that strain thresholds are not fixed but context-dependent [24]. It also draws on emerging insights from periodontal ligament mechanobiology, wherein force transduction via integrins and cytoskeletal pathways regulates cellular responses—processes that may be impaired under conditions of vascular disruption and inflammation. By articulating these relationships explicitly, the framework addresses a key gap in current literature, namely the absence of biologically specified force limits and decision rules in orthodontic guidelines [26].

While conceptual in nature, the framework generates falsifiable propositions and offers immediate heuristic value for clinicians managing high-risk patients. By shifting orthodontic planning from reactive adjustment to anticipatory, biologically informed design, it holds potential to reduce the documented incidence of additional attachment loss reported in compromised cases. Ultimately, this model reframes orthodontic care in reduced periodontium as a biologically constrained, dynamically monitored process, advancing both theoretical clarity and clinical prudence in interdisciplinary practice.

## Implications for Practice

Adoption of this framework has direct implications for orthodontic and periodontal practice, emphasizing interdisciplinary synergy to optimize outcomes in reduced-periodontium cases. Clinicians can employ risk stratification at intake to categorize patients, guiding conservative force systems and sequencing to mitigate iatrogenic harm [22]. For example, in moderate-risk scenarios, integrating light forces with tipping mechanics may expedite alignment while preserving attachment levels [58].

Practically, the framework encourages routine biologic monitoring, such as assessing inflammatory markers, to inform adaptive adjustments during treatment [31]. This could reduce chair time and complications, enhancing patient satisfaction in adult orthodontics. Educational curricula should incorporate these principles, training practitioners in mechanobiology to bridge silos between specialties [19]. Ultimately, by prioritizing biologic fidelity, the model supports safer, more effective care, potentially informing updated guidelines for managing periodontally compromised patients.

**Limitations**

As a purely conceptual paper, this framework lacks empirical validation, relying on synthesized literature rather than original data. Assumptions about biologic thresholds, such as strain limits, may vary individually, necessitating prospective studies for refinement [39]. The model does not address all variables, like microbial influences or advanced regenerative techniques, and its applicability to extreme cases remains theoretical. Future research should test the propositions to confirm translational efficacy.

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**References**

1. Gkantidis N, Christou P, Topouzelis N. The orthodontic-periodontic interrelationship in integrated treatment challenges: a systematic review. J Oral Rehabil. 2010;37(5):377-90. doi:10.1111/j.1365-2842.2010.02068.x
2. Huyen NT, Nghi PH, Phuong ĐTL, Trang TTT, Huyen LT. Public debt and prosperity nexus in Asian countries: nonlinearity and threshold analysis. J Organ Behav Res. 2023;8(1):74-91. https://doi.org/10.51847/tw5g65dco8
3. Bahrawi SAH, Ali EARFE. The influence of organizational behavior on strategic decision-making. Asian J Indiv Organ Behav. 2023;3:25-35. https://doi.org/10.51847/cb7NzhSkVg
4. Ncube M, Sibanda M, Matenda FR. The influence of AI and the pandemic on BRICS nations: South Africa's economic performance during crisis. Ann Organ Cult Leadersh Extern Engagem J. 2023;4:17-24. https://doi.org/10.51847/lrMvYTE3OF
5. Oruba Z, Gibas-Stanek M, Pihut M, Cześnikiewicz-Guzik M, Stós W. Orthodontic treatment in patients with periodontitis - a narrative literature review. Aust Dent J. 2023;68(4):238-46. doi:10.1111/adj.12974 Estomatología Univalle
6. Tam LT, An HTT, Linh TK, Nhung LTH, Ha TNV, Huy PQ, et al. The impact of COVID-19 on value co-creation activities: a study of economics students in Vietnam. Ann Organ Cult Leadersh Extern Engagem J. 2023;4:25-34. https://doi.org/10.51847/QeaHrAoLoL
7. FigueroaValverde L, Rosas-Nexticapa M, AlvarezRamirez M, Lopez-Ramos M, MateuArmand V, Lopez-Gutierrez T. Prostate cancer trends in southern Mexico: statistical data from 1978 to 2020. Asian J Curr Res Clin Cancer. 2023;3(2):1-7. https://doi.org/10.51847/Vd5RkEeAhh
8. Wishney M. Potential risks of orthodontic therapy: a critical review and conceptual framework. Aust Dent J. 2017;62 Suppl 1:86-96. doi:10.1111/adj.12486
9. Okoro C, Adebayo F, Bello A. Enhanced clinical decision-making in early prostate cancer recurrence using [18F]Fluciclovine PET/CT. Asian J Curr Res Clin Cancer. 2023;3(2):36-50. https://doi.org/10.51847/Nuh7wPRHUj
10. Fitero A, Negruț N, Cseppento DCN, MirelaTit D, Negru PA, Bustea C, et al. Inequities in antiviral therapy for diabetic individuals affected by COVID-19. Ann Pharm Pract Pharmacother. 2023;3:9-20. https://doi.org/10.51847/BAIbQWifek
11. Eke PI, Thornton-Evans G, Wei L, et al. Periodontitis in US adults: National Health and Nutrition Examination Survey 2009-2014. J Am Dent Assoc. 2018;149(7):576-88.e6. doi:10.1016/j.adaj.2018.04.023
12. Owusu A, Bonnaire K, Hailemeskel B. Lovastatin in the context of organ transplantation: comprehensive review and survey findings. Ann Pharm Pract Pharmacother. 2024;4:1-7. https://doi.org/10.51847/VKrex2ILgt
13. Moga RA, Buru SM, Olteanu CD. Assessment of the maximum amount of orthodontic force for PDL in intact and reduced periodontium (part I). Int J Environ Res Public Health. 2023;20(3):1889. doi:10.3390/ijerph20031889
14. Deandra FA, Sulijaya B, Sudjatmika DA, Harsas NA. Selection of bone graft material and proper timing of periodontal surgery for orthodontic patients: a systematic review. Heliyon. 2024;10(1):e24201. doi:10.1016/j.heliyon.2024.e24201
15. Cissé C, Konaré MA, Samaké M, Togola I. Exploring the anti-inflammatory potential of

Sericanthe chevalieri and Ceiba pentandra as natural antitussives for children. Spec J Pharmacogn Phytochem Biotechnol. 2024;4:49-58. https://doi.org/10.51847/0OVczhRtw1
16. Maltha JC, van Hulst M, Breuning KH, Kuijpers-Jagtman AM. Mechanobiology of orthodontic tooth movement: an update. J World Fed Orthod. 2023;12(4):156-60. doi:10.1016/j.ejwf.2023.05.001
17. Staufer K, Landmesser H. Effects of crowding in the lower anterior segment--a risk evaluation depending upon the degree of crowding. J Orofac Orthop. 2004;65(1):13-25. doi:10.1007/s00056-004-0207-4
18. Deshmukh J, Deshmukh S, Deshmukh P, Deshmukh R. Merge to emerge - an interdisciplinary approach for management of periodontally compromised orthodontically treated patient. J Indian Soc Periodontol. 2017;21(1):71-75. doi:10.4103/jisp.jisp_107_16
19. Barbosa-Liz DM, Marques LS, Siqueira JTT, Borges R, Oliveira PS, Malo J. Overview of systematic reviews on periodontal-orthodontic interactions: A comprehensive literature analysis. Orthod Craniofac Res. 2024;27(2):193-202. doi:10.1111/ocr.12720
20. Silva-Hormazábal M, Alsina Á. Exploring the impact of integrated education on medical sciences: a comprehensive review. Ann Pharm Educ Saf Public Health Advocacy. 2023;3:30-6. https://doi.org/10.51847/h9MdCIGsUf
21. Luchian I, Tatarciuc D, Curca FR, Jelea M, Tomescu D, Cojocaru A, et al. The influence of orthodontic treatment on periodontal health between challenge and synergy: A narrative review. Dent J (Basel). 2024;12(4):112. doi:10.3390/dj12040112
22. Hsu YT, Huang NC, Wong A, Chang KC, Lin GH, Wang CW, et al. Periodontal risk assessment based on dental and gingival morphology: A comparative analysis of African versus Asian American cohorts. Clin Adv Periodontics. 2020;10(4):224-30. doi:10.1002/cap.10117
23. Alhossan A, Al Aloola N, Basoodan M, Alkathiri M, Alshahrani R, Mansy W, et al. Assessment of community pharmacy services and preparedness in Saudi Arabia during the COVID-19 pandemic: A cross-sectional study. Ann Pharm Educ Saf Public Health Advocacy. 2024;4:43-9. https://doi.org/10.51847/C52qAb0bZW
24. Frost HM. Bone “mass” and the “mechanostat”: a proposal. Anat Rec. 1987;219(1):1-9. doi:10.1002/ar.1092190104
25. Cardoso EM, Reis C, Manzanares-Céspedes MC. Chronic periodontitis, inflammatory cytokines, and interrelationship with other chronic diseases. Postgrad Med. 2018;130(1):98–104. doi:10.1080/00325481.2018.1396876.
26. Cadenas de Llano-Pérula M, Rooijers V, Verdonck A, Fudalej PS, Baan F, Willems G, et al. Risk factors for gingival recessions after orthodontic treatment: a systematic review. Eur J Orthod. 2023;45(5):528-44. doi: 10.1093/ejo/cjad026
27. Nanci A, Bosshardt DD. Structure of periodontal tissues in health and disease. Periodontol 2000. 2006;40:11-28. doi: 10.1111/j.1600-0757.2005.00141.x
28. Singh N, Pradhan P, Giri R, Satapathy D. Ki-67 expression as a complementary marker for histopathological grading of astrocytic tumors. Arch Int J Cancer Allied Sci. 2023;3(2):5-10. https://doi.org/10.51847/u7xvxvsgKS
29. Tyrovola JB. The "Mechanostat Theory" of Frost and the OPG/RANKL/RANK system. J Cell Biochem. 2015;116(12):2724-9. doi: 10.1002/jcb.25265
30. Pardo-Zamora F, Castellano-Rioja G. Liquid biopsy in oral cancer diagnosis: a narrative review of emerging diagnostic tools. Arch Int J Cancer Allied Sci. 2024;4(1):1-6. https://doi.org/10.51847/CcaLqtzvoN
31. Neurath N, Kesting M. Cytokines in gingivitis and periodontitis: from pathogenesis to therapeutic targets. Front Immunol. 2024;15:1435054. doi:10.3389/fimmu.2024.1435054.
32. Kajanova J, Badrov A. Medical students’ perspectives on trust in medical AI: a quantitative comparative study. Asian J Ethics Health Med. 2024;4:44-57. https://doi.org/10.51847/36mpdZ9AZ8
33. Lembo L, Barra M, Iriti A. Building trust in the application of machine learning algorithms for rare disease diagnosis. Asian J Ethics Health Med. 2023;3:26-39. https://doi.org/10.51847/Mo7NXmiBnA
34. Garlet GP. Destructive and protective roles of cytokines in periodontitis: a re-appraisal from host defense and tissue destruction viewpoints. J Dent Res. 2010;89(12):1349-63. doi: 10.1177/0022034510376402
35. Antonarakis GS, Buser D, Katsaros C. Periodontal considerations during orthodontic intrusion and extrusion in healthy and reduced periodontium. Periodontol 2000. 2024. doi: 10.1111/prd.12578

36. Bate GB, Adeleye AO, Ijanu EM, Olalere EO, Amoo AO, Asaju CI, et al. Quality assessment of wastewater: physicochemical and bacteriological evidence from Dutse abattoir, North-West Nigeria. World J Environ Biosci. 2023;12(3):58-66. https://doi.org/10.51847/5xxrD8Fbka
37. de Jong T, Bakker AD, Everts V, Smit TH. The intricate anatomy of the periodontal ligament and its development: lessons for periodontal regeneration. J Periodontal Res. 2017;52(6):965–74. doi:10.1111/jre.12477.
38. Nagdalian A, Askerova A, Blinov A, Shariati MA. Evaluation of the toxicity of copper oxide nanoparticles toward pea seeds. World J Environ Biosci. 2024;13(3):23-30. https://doi.org/10.51847/A2gMbUMBUD
39. Milne TJ, Ichim I, Patel B, McNaughton A, Meikle MC. Induction of osteopenia during experimental tooth movement in the rat: alveolar bone remodelling and the mechanostat theory. Eur J Orthod. 2009;31(3):221–31. doi:10.1093/ejo/cjp032.
40. Johnson TM, Bice RW, Gilbert WA. Orthodontic treatment of periodontally compromised teeth after laser periodontal therapy: a case report. Photobiomodul Photomed Laser Surg. 2021;39(8):528–34. doi:10.1089/photob.2020.4910.
41. Isola G, Matarese G, Cordasco G, Perillo L, Ramaglia L. Mechanobiology of the tooth movement during the orthodontic treatment: a literature review. Minerva Stomatol. 2016;65(5):299-327
42. Nguyen Ha M, Le Thanh T, Pham Thi Thanh V. Factors affecting retail customers' satisfaction when using M-banking services: case study at Sacombank - Hanoi branch. J Organ Behav Res. 2024;9(1):48-63. https://doi.org/10.51847/YrZHHIko2r
43. Cantile T, Lombardi S, Quaraniello M, Riccitiello F, Leuci S, Riccitiello A. Studying the knowledge and behavior of parents in dealing with children's dental injuries. Ann Dent Spec. 2024;12(2):1-5. https://doi.org/10.51847/FYF9lXJwPt
44. Jamal BT. Does sphincter pharyngoplasty improve speech hypernasality in cleft patients with velopharyngeal incompetence? Ann Dent Spec. 2023;11(3):9-13. https://doi.org/10.51847/2OnbthAhZO
45. Sakhnenkova TI, Abdul-Kadyrova LR, Akhilgova ZA, Brovikova AA, Markov OO, Saribekyan AA, et al. Morphological and biochemical analysis of 3D scaffold based on biocompatible polymer for tissue engineering. J Adv Pharm Educ Res. 2023;13(3):29-33. https://doi.org/10.51847/v8o0GbXJdN
46. Levochkina ED, Belyaev NG, Tkach AI, Menadzhiev AS, Volkova MN, Akifeva NM, et al. Data analysis of autoimmune bioindicators in the context of predicting cardiomyocyte damage. J Adv Pharm Educ Res. 2024;14(3):62-9. https://doi.org/10.51847/ilO1LTBQLt
47. Zharashueva EB, Mirzayeva AK, Iakovleva AN, Dyshnieva NA, Dudurgova AT, Utovka OV, et al. Sanitary and hygienic assessment of the dressing material modified with silver nanoparticles. J Adv Pharm Educ Res. 2024;14(3):9-14. https://doi.org/10.51847/J10C8y3UWQ
48. Bisri DY, Hallis IK, Saputra TA, Bisri T. Brain relaxation score on craniotomy brain tumour removal with adjuvant thiopental and dexmedetomidine: a case report. J Adv Pharm Educ Res. 2023;13(3):73-8. https://doi.org/10.51847/CTKVDUSitR
49. Henneman S, Von den Hoff JW, Maltha JC. Mechanobiology of tooth movement. Eur J Orthod. 2008;30(3):299-306. doi: 10.1093/ejo/cjn020
50. Xu H, Guan J, Jin Z, Yin C, Wu S, Sun W, Zhang H, Yan B. Mechanical force modulates macrophage proliferation via Piezo1-AKT-Cyclin D1 axis. FASEB J. 2022;36(8):e22423. doi:10.1096/fj.202200314R.
51. Kartashev VP, Xingyuan S, Medvedev IN, Tkacheva ES, Vorobyeva NV. Physiological changes in the erythrocytes of an aging organism experiencing physical. J Biochem Technol. 2023;14(1):50-6. https://doi.org/10.51847/GGLSMMHC5s
52. Martin C, Celis B, Ambrosio N, Bollain J, Antonoglou GN, Figuero E, et al. Effect of orthodontic therapy in periodontitis and non-periodontitis patients: a systematic review with meta-analysis. J Clin Periodontol. 2022;49 Suppl 24:72-101. doi: 10.1111/jcpe.13487
53. Van Dyke TE, Sheilesh D. Risk factors for periodontitis. J Int Acad Periodontol. 2005;7(1):3-7
54. Vardhan NRH, Ladani RK, Reddy AR. Integrated bioinformatics approach unveils genes linked to metastasis in head and neck squamous cell carcinoma. J Biochem Technol. 2023;14(4):31-44. https://doi.org/10.51847/oaS45jOPi7
55. Theodorou CI, Kuijpers-Jagtman AM, Bronkhorst EM, Wagener FADTG. Optimal force magnitude for bodily orthodontic tooth movement with fixed

appliances: a systematic review. Am J Orthod Dentofacial Orthop. 2019;156(5):582-92. doi: 10.1016/j.ajodo.2019.05.011

56. Chackartchi T, Polak D, Stabholz A, Chaushu S. Orthodontic treatment in periodontitis patients. Periodontol 2000. 2025. doi: 10.1111/prd.12634
57. Wen X, Jin Y, Zhao Z. Exploring the mechanical and biological interplay in the periodontal ligament. Int J Oral Sci. 2025;17(1):23. doi: 10.1038/s41368-025-00354-y
58. Kojima Y, Fukui H. Numerical simulations of canine retraction with T-loop springs based on the updated moment-to-force ratio. Eur J Orthod. 2012;34(1):10-8. doi: 10.1093/ejo/cjq164