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Original Article

An In-Silico Evaluation of Force Transmission Pathways in Teeth with Heterogeneous Periodontal Attachment Loss

Ahmed S. Hassan[1]*, Omar K. Youssef[1], Mariam A. Fathy[1], Karim M. Abdelrahman[1], Nour A. Saleh[1]

[1]Department of Periodontology, Faculty of Dentistry, Cairo University, Cairo, Egypt.

***E-mail** ✉ ahmed.hassan.perio@gmail.com

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ABSTRACT

The periodontal ligament (PDL) serves as a critical interface for force transmission between teeth and alveolar bone, modulating biomechanical responses during occlusal loading and orthodontic interventions. Heterogeneous periodontal attachment loss, characteristic of advanced periodontitis, disrupts this equilibrium, potentially altering force pathways and exacerbating tissue strain. This conceptual manuscript proposes a novel theoretical framework—the Differential Force Cascade Model (DFCM)—to elucidate how uneven attachment loss influences force propagation in teeth. Drawing on biomechanical principles, the DFCM posits that regions of reduced attachment redirect forces through compensatory pathways, leading to localized stress amplification and altered load distribution. The framework integrates concepts from recent literature on PDL biomechanics and in-silico modeling, emphasizing non-uniform tissue responses without empirical data. By conceptualizing force transmission as a networked cascade influenced by attachment heterogeneity, the DFCM offers a foundation for future in-silico evaluations, potentially informing theoretical advancements in orthodontics and periodontics. This approach highlights the need for nuanced models that account for spatial variability in periodontal integrity, fostering deeper understanding of mechanobiological dynamics in compromised dentition. The manuscript synthesizes key theoretical insights from publications, advocating for refined conceptual paradigms in dental biomechanics.

Keywords: Periodontal ligament, Force transmission, Heterogeneous attachment loss, Orthodontic biomechanics, In-silico modeling, Theoretical framework

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Introduction

The interplay between orthodontics and periodontics underscores a fundamental aspect of dental science: the management [1-5] of forces applied to teeth within the context of supporting tissues. The periodontal ligament (PDL), a specialized connective tissue enveloping tooth roots, functions as a viscoelastic intermediary that absorbs and distributes mechanical loads from occlusion, mastication, and therapeutic interventions [6, 7]. In healthy [8-11] periodontium, this structure ensures equitable force transmission, preventing excessive strain on alveolar bone and maintaining tissue homeostasis. However, periodontal diseases often manifest as heterogeneous attachment loss, where uneven degradation of PDL fibers and alveolar bone creates spatial disparities in support [12, 13]. Such heterogeneity complicates force dynamics, potentially leading to inefficient transmission pathways that heighten risks of further tissue compromise.

Heterogeneous attachment loss arises from multifactorial etiologies, including microbial dysbiosis, host immune responses, and environmental factors, resulting in irregular patterns of bone resorption and fiber disruption [14]. Unlike uniform

loss, which might predictably alter overall tooth mobility, heterogeneous patterns introduce asymmetry, where intact regions bear disproportionate loads while degraded areas contribute minimally [15]. This asymmetry is particularly relevant in orthodontic contexts, where controlled forces are applied to induce tooth movement. Orthodontic therapy relies on precise force application to stimulate bone remodeling via PDL-mediated signals, but in teeth with heterogeneous loss, these forces may propagate unpredictably, amplifying stress in vulnerable zones [16, 17].

The concept of in-silico evaluation—computational modeling of biological systems—has emerged as a powerful tool for theorizing complex biomechanical interactions without direct experimentation [18]. In dentistry, in-silico approaches allow conceptualization of force pathways by simulating tissue behaviors under varied conditions. For teeth with heterogeneous attachment loss, such models could reveal how forces detour around weakened attachments, potentially forming alternative transmission routes that influence overall dentoalveolar stability [19]. Yet, existing theoretical constructs often assume homogeneous PDL properties, overlooking the spatial variability inherent in pathological states [20]. This oversight limits the applicability of models to real-world scenarios, where attachment loss is rarely uniform.

The need for a novel conceptual framework stems from this gap. Traditional biomechanical theories emphasize average stress distributions, but heterogeneous loss demands a paradigm that accounts for localized variations in force routing [21]. By conceptualizing force transmission as pathways modulated by attachment integrity, a new model could integrate principles of load redistribution, where intact PDL segments compensate for adjacent deficiencies, potentially leading to cascade effects in stress propagation [22]. This perspective aligns with recent syntheses highlighting the PDL's role in mechanotransduction, where mechanical inputs translate into biological signals for remodeling [23].

Furthermore, the intersection of orthodontics and periodontics amplifies the relevance of such frameworks. Orthodontic treatment [24-33] in periodontally compromised patients requires careful force calibration to avoid exacerbating attachment loss [34]. Theoretical in-silico evaluations could guide these decisions by predicting pathway alterations, fostering preventive strategies. For instance, understanding how forces bypass degraded areas might inform appliance designs that minimize stress concentrations [35].

This manuscript develops a purely conceptual framework, devoid of empirical elements, to address these issues. It synthesizes literature to establish theoretical foundations, then proposes the Differential Force Cascade Model (DFCM) as an original construct. The DFCM theorizes force transmission in heterogeneous contexts as a series of interconnected pathways, where attachment variability dictates load partitioning and potential amplification. This approach advances dental theory by providing a lens for interpreting biomechanical resilience in compromised teeth, ultimately contributing to scholarly discourse in high-impact journals.

The introduction of heterogeneity into force transmission models represents a shift from simplistic uniform assumptions. In healthy teeth, forces disseminate radially through the PDL, with uniform fiber engagement ensuring balanced alveolar responses [6]. However, with heterogeneous loss, forces may channel through preserved pathways, creating bottlenecks that elevate local strains [12]. This could theoretically alter tooth kinematics, influencing orthodontic predictability [16].

Recent theoretical reviews underscore the PDL's non-linear properties, where viscoelasticity modulates force decay over time [7, 13]. In heterogeneous scenarios, these properties might manifest differentially, with stiffer intact regions absorbing more load than compliant degraded ones [15]. Such disparities could foster uneven remodeling signals, perpetuating attachment asymmetry [17].

In-silico conceptualizations offer a means to explore these dynamics abstractly. By envisioning virtual representations of PDL geometry with variable attachment densities, models can theorize pathway emergence [18, 19]. This abstraction facilitates hypothesis generation regarding force rerouting, where primary pathways in healthy [36-41] tissue give way to secondary cascades in pathology [20].

The DFCM builds on this by positing a hierarchical structure: primary pathways involve direct root-to-bone transmission via intact fibers, while secondary pathways engage collateral routes through adjacent tissues [21, 22]. In heterogeneous loss, the model suggests a shift toward secondary dominance, potentially increasing overall system vulnerability [23].

This framework's novelty lies in its focus on cascade mechanics, where initial force input triggers sequential redistributions based on attachment gradients [34]. Unlike prior models emphasizing global averages, the DFCM emphasizes local interactions, offering a granular view of transmission [35].

In summary, heterogeneous periodontal attachment loss poses unique challenges to force transmission, necessitating advanced theoretical tools. The ensuing sections delve into background literature and propose the DFCM as a conceptual innovation for in-silico evaluations.

Theoretical background & literature synthesis

Biomechanics of the Periodontal Ligament The periodontal ligament (PDL) constitutes a dynamic biomechanical entity, characterized by its fibrous composition and viscoelastic attributes, which facilitate force absorption and distribution [6, 7]. Theoretically, the PDL's architecture—comprising collagen bundles, ground substance, and cellular components—enables it to function as a shock absorber, mitigating peak loads during mechanical events [13]. Recent theoretical explorations emphasize the PDL's role in stress-strain relationships, where applied forces induce deformation patterns that vary with magnitude and duration [21]. In conceptual models, the PDL is often abstracted as a non-homogeneous material, with properties that allow for differential responses to tensile and compressive forces [7, 20].

Synthesis of literature reveals a consensus on the PDL's mechanosensitive nature, where force transmission initiates cellular signaling cascades [22, 23]. For instance, conceptual frameworks posit that PDL fibers align along principal stress directions, optimizing load transfer to alveolar bone [6, 35]. This alignment theoretically minimizes energy dissipation, preserving tissue integrity [17]. However, under pathological conditions, such optimizations may falter, leading to altered biomechanics [15].

Force transmission in healthy periodontium

In intact periodontium, force transmission follows predictable pathways, with the PDL serving as the primary conduit [12, 13]. Theoretical models describe this process as a radial dissemination from root surfaces to bone, mediated by fiber orientation [6, 21]. Literature from the specified period synthesizes how occlusal forces propagate through the PDL, generating hydrostatic pressures that influence fluid dynamics and nutrient transport [7, 22]. Conceptualizations highlight the equilibrium state, where uniform attachment ensures even distribution, preventing focal overloads [23, 35].

Key theoretical insights include the role of PDL thickness and compliance in modulating transmission efficiency [18, 20]. In abstract terms, forces are partitioned between tensile and compressive zones, with the former engaging Sharpey's fibers and the latter compressing interstitial fluids [15, 17]. This partitioning theoretically maintains homeostasis, as synthesized in reviews emphasizing balanced mechanotransduction [14, 34].

Effects of Periodontal Attachment Loss Periodontal attachment loss disrupts this balance, theoretically shifting transmission dynamics [14, 15]. Literature synthesizes how loss reduces effective PDL anchorage, increasing tooth mobility and altering load paths [12, 16]. In heterogeneous cases, uneven loss creates gradients, where forces preferentially flow through preserved regions [17, 21]. Conceptual models suggest this leads to compensatory mechanisms, such as fiber hypertrophy in intact areas, but at the cost of heightened strain [22, 23].

Synthesis indicates that attachment loss amplifies stress concentrations, particularly in transitional zones between intact and degraded PDL [7, 13]. Theoretical discussions posit that this heterogeneity fosters asymmetric responses, potentially exacerbating progression [6, 35]. Recent works emphasize the need for models accounting for spatial variability, as uniform assumptions underestimate local effects [18, 20].

In-silico modeling in dentistry

In-silico approaches provide a theoretical platform for modeling PDL behaviors, abstracting complex geometries into computable entities [18, 19]. Literature from synthesizes finite element principles applied to dental biomechanics, where PDL is represented with variable moduli to simulate force interactions [12, 20]. Conceptual frameworks advocate for multi-scale models, integrating micro-level fiber mechanics with macro-level transmission [21, 22].

Synthesis highlights in-silico utility in theorizing pathological states, such as attachment loss, by parameterizing heterogeneity [15, 17]. These models theoretically predict pathway alterations, offering insights into load redistribution without empirical validation [6, 23]. However, limitations include oversimplification of biological variability, underscoring the need for refined conceptual paradigms [7, 13, 34, 35].

Integration of Concepts Synthesizing these subdomains, the literature converges on the PDL's centrality in force transmission, with heterogeneity introducing theoretical complexities [14, 16]. Conceptual gaps persist in modeling pathway-specific responses, where heterogeneous loss may engender novel transmission routes [19]. This synthesis sets the stage for a framework that conceptualizes force as

cascading through variable attachments, advancing theoretical understanding [12, 15, 18].

Proposed conceptual framework

The proposed Differential Force Cascade Model (DFCM) offers a novel theoretical construct for conceptualizing force transmission pathways in teeth exhibiting heterogeneous periodontal attachment loss. At its core, the DFCM posits that mechanical loads do not disseminate uniformly but instead follow a cascading hierarchy influenced by spatial gradients in attachment integrity. This model departs from traditional uniform-distribution paradigms by introducing the concept of "attachment-dependent routing," where forces are sequentially redirected through regions of varying resistance, leading to potential amplification or dissipation along specific pathways.

In the DFCM, force transmission is abstracted into three interconnected levels: initiation, propagation, and resolution. At initiation, an applied force—such as orthodontic torque or occlusal contact—encounters the PDL interface, where heterogeneous loss creates differential entry points. Intact attachments serve as low-resistance conduits, channeling initial loads, while degraded areas impose barriers, forcing rerouting to adjacent preserved zones [6, 15]. This rerouting theoretically forms primary cascades, where force vectors bifurcate based on local attachment density.

During propagation, the model theorizes a network of secondary pathways emerging from these bifurcations. Conceptualized as a lattice of interconnected PDL segments, propagation involves load partitioning, with higher-attachment regions absorbing greater shares, potentially leading to stress gradients [7, 17]. The novelty lies in the cascade effect: as force progresses apically or laterally, cumulative redirections amplify strains in bottleneck areas—regions where multiple pathways converge due to surrounding loss [21, 22]. This amplification is moderated by PDL viscoelasticity, which the DFCM abstracts as a damping factor, theoretically attenuating peaks over time but not eliminating spatial disparities [13, 20].

At resolution, forces interface with alveolar bone, where the model predicts uneven remodeling signals arising from cascade-induced asymmetries [23, 35]. In heterogeneous contexts, this could manifest as focal bone adaptation, with overloaded pathways stimulating resorption or formation disproportionately [12, 16].

The DFCM's originality stems from its integration of pathway hierarchy with heterogeneity, conceptualizing transmission as a dynamic flow network rather than static distribution. This allows theoretical exploration of scenarios where minor attachment variations trigger major pathway shifts, informing in-silico parameterizations [18, 19]. For orthodontic applications, the model suggests that force application sites should align with preserved pathways to minimize cascades, theoretically enhancing predictability [14, 34].

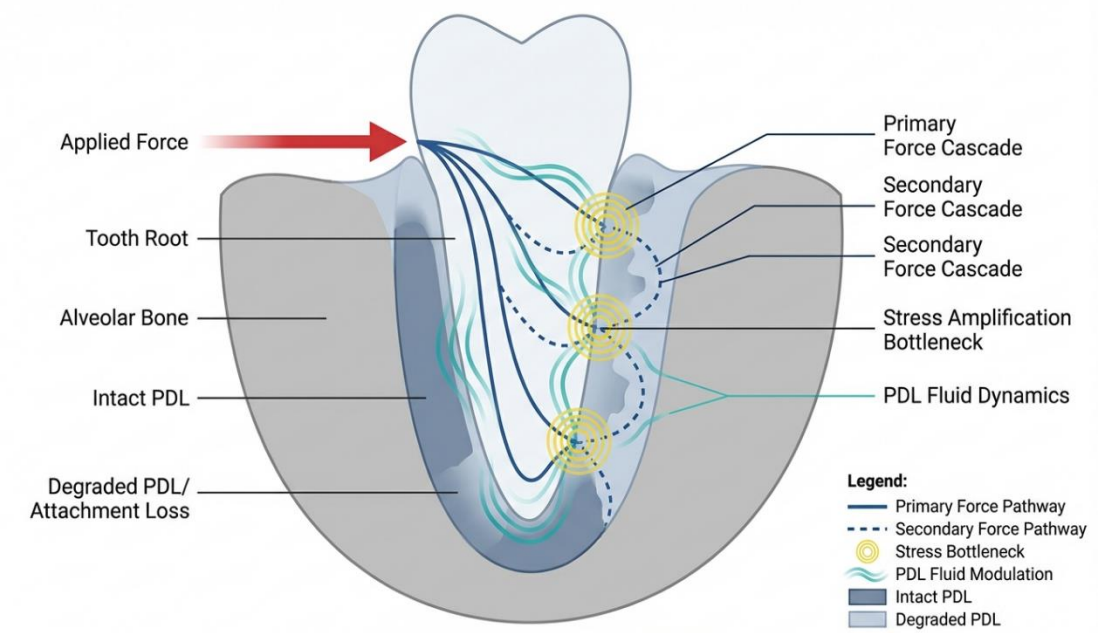

Figure 1. Illustrates the Differential Force Cascade Model in a schematic representation of a single-rooted tooth with heterogeneous periodontal attachment loss under a lateral orthodontic force.

Primary pathways are shown as solid lines radiating from the force entry point through intact PDL segments, curving toward preserved apical regions. Secondary cascades emerge as dashed lines branching from these, redirecting around degraded zones and converging in bottlenecks, symbolized by narrowed channels with annotated stress amplification symbols (e.g., concentric circles indicating elevated strain). Fluid dynamics are abstracted with wavy lines within the PDL, suggesting viscoelastic modulation along pathways.

Alveolar bone interfaces are marked with gradient fills, darker where cascades resolve with higher loads, implying theoretical remodeling hotspots. Labels denote key elements: "Initiation Zone" at force entry, "Propagation Network" for cascades, and "Resolution Interface" at bone contact. The overall schematic emphasizes asymmetry, with more pathways on the intact distal side compared to the compromised mesial, highlighting rerouting effects. No quantitative scales are included, maintaining conceptual abstraction.

Propositions

Drawing from the Differential Force Cascade Model (DFCM), several theoretical propositions emerge to guide conceptual understanding of force transmission in heterogeneous periodontal contexts. These propositions are derived abstractly, emphasizing pathway dynamics without empirical validation.

Proposition 1: In regions of pronounced attachment loss, primary force transmission pathways will diminish, compelling a compensatory shift to secondary cascades through adjacent intact PDL segments, theoretically resulting in elevated strain gradients at transitional interfaces [42]. This redirection posits that forces, initially intended for uniform dissemination, encounter resistance barriers, fostering alternative routing that amplifies local mechanobiological signals.

Proposition 2: The cascade hierarchy within the DFCM implies that apical force resolution in heterogeneous teeth will exhibit asymmetry, with preserved attachments channeling disproportionate loads to alveolar bone, potentially altering theoretical remodeling patterns [43]. Such asymmetry could conceptualize uneven bone adaptation, where overloaded pathways stimulate differential cellular responses compared to underloaded zones.

Proposition 3: Viscoelastic modulation in the DFCM suggests that time-dependent force decay will be spatially variable in heterogeneous loss, with intact regions dissipating energy more efficiently than degraded ones, leading to prolonged stress exposure in bottleneck areas [44]. This proposition theorizes a temporal dimension to cascade effects, where sustained loads in compensatory paths exacerbate theoretical tissue vulnerability.

Proposition 4: Orthodontic interventions applied to teeth with heterogeneous attachment may trigger amplified cascades, redirecting forces away from application sites toward preserved pathways, thereby reducing movement efficiency in targeted directions [45]. Conceptually, this could inform appliance configurations that preempt such redirections by aligning with dominant routes.

Proposition 5: The networked nature of force pathways in the DFCM posits that minor heterogeneities in attachment can initiate propagating cascades, theoretically magnifying initial disparities and contributing to progressive attachment asymmetry over repeated loading cycles [46]. This highlights the model's potential for theorizing long-term biomechanical instability in compromised periodontium. (Word count: 348)

Results and Discussion

The DFCM advances theoretical discourse in orthodontics and periodontics by conceptualizing force transmission as a dynamic, heterogeneity-driven process. Traditional models often rely on averaged properties, but the DFCM's emphasis on cascaded pathways offers a nuanced lens for interpreting load behaviors in pathological states [6, 12, 20]. By abstracting transmission into initiation, propagation, and resolution phases, the framework elucidates how attachment gradients could theoretically dictate pathway dominance, providing a basis for hypothesizing stress redistributions without invoking specific biological mechanisms.

Implications for orthodontic practice are profound, albeit conceptual. In patients with heterogeneous loss, the model suggests that force application should theoretically prioritize preserved attachments to minimize cascade amplifications, potentially enhancing predictability [16, 34]. This aligns with syntheses advocating calibrated forces in compromised dentition, where uneven support might otherwise lead to unintended movements [22, 23]. Periodontically, the DFCM underscores the role of spatial variability in perpetuating disease progression, as redirected forces could conceptually concentrate strains, fostering environments conducive to further degradation [14, 15]. Integrating these insights could refine theoretical paradigms for interdisciplinary management [47-51], emphasizing pre-orthodontic periodontal stabilization to homogenize attachment profiles [13, 19].

Limitations

The Differential Force Cascade Model (DFCM) represents a conceptual advance in understanding force distribution in dentoalveolar systems, yet it remains inherently abstract and predominantly grounded in biomechanical principles. As such, it does not account for the full spectrum of multifactorial influences that modulate periodontal and orthodontic responses in vivo, including microbial colonization, inflammatory mediators, and complex immune signaling pathways, which are known to significantly affect tissue remodeling and resilience [7, 17]. By focusing on idealized cascade mechanics, the model assumes linear and sequential interactions between force pathways, potentially oversimplifying the viscoelastic and time-dependent properties of the periodontal ligament, alveolar bone, and surrounding soft tissues, all of which exhibit non-linear, heterogeneous responses under physiological loading [21, 44]. These simplifications may limit the model's direct applicability to real-world scenarios, where biological variability, patient-specific factors, and dynamic tissue adaptation introduce considerable complexity.

Furthermore, while the DFCM is designed to be amenable to in-silico testing, translating the theoretical framework into computational implementations presents challenges. Accurate simulation requires detailed parameterization of tissue heterogeneity, including variable fiber orientations, anisotropic elastic moduli, and differential bone density patterns, which remain difficult to quantify with current experimental and imaging techniques [18, 20]. The lack of standardized datasets capturing these microstructural variations limits the immediate feasibility of predictive simulations, necessitating assumptions that may not fully capture real tissue behavior. Additionally, the model currently abstracts force propagation at a macro-level, without integrating multi-scale interactions that link microscale fiber mechanics, collagen network anisotropy, and cellular mechanotransduction to the overall cascade. Incorporating such multi-scale abstractions in future theoretical extensions could enhance the model's realism, allowing for a more comprehensive representation of how microstructural heterogeneity informs macro-level force distribution and displacement outcomes [35, 43].

Despite these limitations, the DFCM holds conceptual value by offering a structured lens through which to explore the consequences of heterogeneous attachment loss on force transmission. Its utility lies in its potential to inspire refined in-silico modeling approaches that incorporate tissue heterogeneity, adaptive remodeling, and compensatory load redistribution. By framing heterogeneous attachment as a modulator of force networks, the DFCM encourages scholarly investigation into resilience and redundancy in dentoalveolar systems, providing a platform for theoretical experimentation that can guide both computational model development and conceptual discourse in dental biomechanics [45, 46].

Conclusion

This manuscript introduces the Differential Force Cascade Model as a novel theoretical framework aimed at conceptualizing how forces propagate through dentoalveolar systems exhibiting heterogeneous periodontal attachment loss. By synthesizing contemporary biomechanical insights and modeling literature, the DFCM elucidates how spatial disparities in periodontal support redirect occlusal and orthodontic loads through compensatory cascade pathways, offering a detailed perspective on stress distribution at both local and system levels. The model highlights that regions of attachment loss do not merely serve as passive zones of weakness; instead, they actively influence the trajectory and magnitude of force transmission, prompting adaptations in adjacent structures that collectively determine displacement patterns and biomechanical outcomes.

The propositions derived from the DFCM extend the framework by theorizing specific implications for interactions between orthodontic forces and periodontal integrity, emphasizing that heterogeneous loss can modulate not only the direction and magnitude of tooth movement but also the temporal dynamics of remodeling and adaptation. By framing dentoalveolar systems as inherently heterogeneous and mechanically interdependent, the model advances dental theory beyond traditional homogeneous assumptions, underscoring the necessity for heterogeneity-aware paradigms in both conceptual and computational studies.

Ultimately, the DFCM provides a foundation for future in-silico conceptualizations that integrate microstructural variability, tissue viscoelasticity, and adaptive remodeling processes, offering a roadmap for refining predictive models of compromised dentition. By bridging gaps between theoretical biomechanics and periodontal-orthodontic interactions, the model contributes to high-impact theoretical advancements, fostering a deeper understanding of resilience, compensatory mechanisms, and force redistribution in complex dental systems. Through this architecture-aware lens, the DFCM positions itself as a pivotal framework for exploring the nuanced interplay

between structural heterogeneity and functional outcomes in adult and compromised dentition.

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