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*Review*

# Orthodontically Induced Inflammatory Root Resorption Cellular Mechanisms and Preventive Protocols

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**Abstract**

Orthodontically induced inflammatory root resorption is a common adverse effect associated with orthodontic tooth movement and represents a significant clinical concern due to its potential to compromise root integrity and long-term dental stability. Although mild resorption is frequently observed and often considered an unavoidable consequence of treatment, severe forms may result in substantial root shortening and permanent structural damage. The biological basis of this phenomenon involves a complex interplay between mechanical forces, cellular responses, inflammatory mediators, and individual susceptibility factors. Mechanical loading applied during orthodontic treatment initiates a cascade of biological events within the periodontal ligament and surrounding alveolar bone. Cellular deformation, vascular alterations, and extracellular matrix remodeling stimulate the release of cytokines, chemokines, and growth factors that regulate bone remodeling and clastic cell activity. Key molecular pathways, including the receptor activator of nuclear factor kappa-B ligand (RANKL)/osteoprotegerin (OPG) system, play a central role in osteoclast and odontoclast differentiation, ultimately influencing the extent of root surface resorption. Emerging evidence has further highlighted the contribution of immune regulation, oxidative stress, hypoxia-related signaling, and regulated cell death pathways in the progression of resorptive lesions. The severity of root resorption varies considerably among individuals, reflecting the influence of multiple biological determinants. Genetic predisposition, root morphology, previous dental trauma, age-related tissue characteristics, and local periodontal conditions have all been associated with differences in susceptibility. These factors may alter the balance between tissue injury and repair, affecting the capacity of periodontal structures to withstand orthodontic forces. Preventive approaches focus on minimizing unnecessary tissue damage while maintaining efficient tooth movement. Contemporary strategies emphasize the application of controlled force systems, individualized treatment planning, periodic radiographic monitoring, and the use of adjunctive modalities aimed at supporting tissue preservation. Advances in imaging and molecular research continue to improve understanding of the mechanisms governing root resorption, providing opportunities for more targeted and biologically informed interventions that support both treatment efficiency and long-term dental health.

**Keywords:** *orthodontic tooth movement, inflammatory root resorption, mechanotransduction, osteoclastogenesis, cytokines, root preservation, orthodontics*

## Introduction

Orthodontic tooth movement is a biologically mediated process that relies on the application of controlled mechanical forces to induce remodeling of the periodontal ligament and surrounding alveolar bone. While this process is fundamental to successful orthodontic treatment, it is often accompanied by a variety of tissue responses, some of which may be undesirable. Among the most significant adverse effects is orthodontically induced inflammatory root resorption, a pathological phenomenon characterized by the loss of cementum and, in severe cases, dentin from the root surface. Although minor root resorption is considered a common and often clinically insignificant consequence of orthodontic treatment, extensive resorption can compromise root integrity, reduce tooth longevity, and negatively affect treatment outcomes (1).

Root resorption associated with orthodontic treatment is a multifactorial process involving a complex interaction between mechanical, cellular, molecular, and patient-related factors. The application of orthodontic forces generates compression and tension zones within the periodontal ligament, initiating a cascade of inflammatory events. These events include the release of cytokines, chemokines, growth factors, and other signaling molecules that regulate the recruitment and activation of osteoclasts and odontoclasts. Excessive or prolonged force application may disrupt the protective cementoblast layer covering the root surface, exposing mineralized tissues to clastic cell activity and facilitating resorption (2).

The pathophysiology of inflammatory root resorption shares several biological mechanisms with bone remodeling. Key molecular mediators such as receptor activator of nuclear factor kappa-B ligand (RANKL), osteoprotegerin (OPG), interleukins, tumor necrosis factor-α, and prostaglandins play central roles in regulating clastic cell differentiation and function. Recent advances in molecular biology have further highlighted the involvement of genetic predisposition, immune regulation, hypoxia-related pathways, and mechanotransduction signaling in determining individual susceptibility to root resorption during orthodontic treatment (3). These findings have contributed to a more comprehensive understanding of why some patients experience severe resorption despite receiving similar orthodontic forces and treatment protocols.

Several treatment-related variables have been associated with an increased risk of root resorption, including force magnitude, duration of force application, treatment length, type of tooth movement, and appliance mechanics. Intrusive movements, torque application, and prolonged treatment duration have frequently been linked to greater resorptive activity. In addition, anatomical characteristics such as root morphology, previous dental trauma, and systemic or genetic factors may influence the severity of tissue damage (4). Consequently, root resorption remains a significant clinical concern that necessitates careful treatment planning and monitoring throughout orthodontic therapy. In this review, we will discuss the cellular mechanisms underlying orthodontically induced inflammatory root resorption and examine current preventive protocols.

## Review

The development of inflammatory root resorption during orthodontic treatment is closely linked to the biological response of periodontal tissues to mechanical loading. Contemporary evidence suggests that the process is not merely a consequence of excessive force application but rather the result of a complex interaction between mechanotransduction pathways, inflammatory mediators, and clastic cell activity. Mechanical stress within the periodontal ligament stimulates the release of cytokines and signaling molecules that promote osteoclast and odontoclast differentiation through pathways such as RANK/RANKL/OPG. When protective cementoblast layers are disrupted, these cells gain access to the root surface and initiate resorptive activity (5).

Individual susceptibility also appears to play a significant role in determining the extent of tissue


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damage. Variations in immune responses, genetic factors, root morphology, and local tissue characteristics may explain why patients exposed to similar orthodontic mechanics experience different degrees of resorption. Recent studies have highlighted the contribution of hypoxia-related signaling, immune modulation, and emerging pathways such as ferroptosis in regulating inflammatory responses associated with root surface breakdown (6). These findings indicate that root resorption is a multifactorial phenomenon rather than a purely mechanical complication.

### *Mechanotransduction and Inflammatory Signaling*

Mechanotransduction represents the biological process through which orthodontic forces are converted into cellular and molecular responses within the periodontal ligament, alveolar bone, and root surface tissues. The application of mechanical stress produces alterations in extracellular matrix organization, vascular perfusion, and cellular deformation, initiating a cascade of signaling events that regulate tissue remodeling. Cells residing within the periodontal ligament, including fibroblasts, osteoblasts, cementoblasts, and immune cells, function as mechanosensors capable of detecting changes in their microenvironment and responding through the release of inflammatory mediators. These responses are essential for tooth movement but may also contribute to pathological root surface changes when tissue homeostasis is disrupted (7).

Compression of the periodontal ligament induces localized ischemia and hypoxic conditions, stimulating the production of pro-inflammatory cytokines such as interleukin-1β, interleukin-6, and tumor necrosis factor-α. These mediators influence the expression of receptor activator of RANKL, a key regulator of clastic cell differentiation and activation. Elevated RANKL expression promotes the formation of osteoclasts and odontoclasts capable of resorbing mineralized tissues. The balance between RANKL and its decoy receptor OPG is therefore critical in determining whether tissue remodeling remains physiological or progresses toward excessive resorptive activity. Evidence indicates that prolonged or concentrated force application may shift this balance toward increased clastic activity, thereby increasing susceptibility to root surface damage (8).

Immune regulation has emerged as an important component of force-induced tissue responses. Orthodontic loading triggers recruitment of macrophages, T lymphocytes, dendritic cells, and other immune cell populations that participate in the sterile inflammatory reaction associated with tooth movement. These cells contribute to cytokine production and interact with resident periodontal cells, creating a dynamic signaling network that influences both bone remodeling and root resorption. Recent investigations have identified immune checkpoint molecules and immunomodulatory pathways as potential regulators of inflammatory responses within periodontal tissues, suggesting that the severity of tissue damage may be influenced by mechanisms extending beyond conventional inflammatory mediators (9).

Growing attention has also been directed toward molecular pathways associated with oxidative stress and regulated cell death. Cytokine-mediated signaling, reactive oxygen species production, and intracellular stress responses appear to influence the behavior of periodontal and clastic cells under orthodontic force. Research examining ferroptosis-related mechanisms has proposed that iron-dependent lipid peroxidation may contribute to tissue injury and inflammatory amplification within the periodontal environment. These observations expand the current understanding of mechanotransduction by linking mechanical stimuli to broader cellular stress pathways that may participate in the development and progression of root resorption during orthodontic treatment (10).

### *Biological Determinants of Root Resorption Susceptibility*

Considerable variation exists in the severity of root resorption observed among orthodontic patients, even when similar treatment mechanics and force systems are employed. This variability highlights the influence of biological determinants that govern


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individual tissue responses to orthodontic loading. Genetic predisposition has long been regarded as a major contributor to susceptibility. Polymorphisms affecting inflammatory mediators, cytokines, and bone remodeling pathways may alter the balance between tissue resorption and repair, thereby influencing the extent of root surface damage. Variations in genes associated with interleukin signaling, tumor necrosis factor activity, and osteoclast differentiation have been linked to increased resorptive responses, suggesting that inherited biological characteristics can modify cellular behavior during orthodontic treatment (11).

Root morphology also plays a substantial role in determining vulnerability to resorption. Teeth with pipette-shaped, pointed, dilacerated, or unusually short roots frequently demonstrate greater susceptibility than teeth with normal root anatomy. Structural characteristics influence stress distribution along the root surface and may create localized regions where mechanical loading is concentrated. Apical regions are particularly susceptible because of their reduced periodontal ligament area and limited capacity to dissipate orthodontic forces. Histological observations have shown that areas exposed to concentrated stress are more likely to exhibit cementoblast disruption and subsequent clastic cell attachment (12).

The condition of periodontal and pulpal tissues before treatment further affects biological responsiveness. Previous dental trauma, even when clinically asymptomatic, may compromise cementum integrity and alter the reparative capacity of periodontal tissues. Similarly, pre-existing inflammatory conditions can modify local cytokine profiles and enhance clastic activity following orthodontic force application. Age-related changes in tissue metabolism have also been investigated, with evidence suggesting differences in cellular turnover, vascularity, and regenerative potential between younger and older individuals. These factors may influence the capacity of root surfaces to recover from mechanical injury and regulate the progression of resorptive lesions (13).

Current research increasingly emphasizes the role of immune regulation and tissue homeostasis in determining susceptibility. Cellular communication within the periodontal ligament involves complex interactions between fibroblasts, immune cells, osteoblasts, cementoblasts, and extracellular matrix components. Mechanical stress alters this microenvironment and may trigger disproportionate inflammatory responses in susceptible individuals. Emerging concepts propose that susceptibility reflects the ability of periodontal tissues to maintain biological equilibrium under sustained orthodontic loading. Disruptions in signaling networks responsible for cellular adaptation, repair, and inflammatory control may facilitate prolonged clastic activity and increase the likelihood of clinically significant root resorption, even in the presence of carefully controlled treatment protocols (14).

### *Contemporary Strategies for Prevention and Tissue Preservation*

Efforts to preserve root structure during orthodontic treatment are increasingly guided by a deeper understanding of the biological events associated with tissue remodeling. Prevention begins with the careful selection of force magnitude and duration, as excessive mechanical loading is strongly associated with increased inflammatory activity and clastic cell recruitment. Contemporary orthodontic protocols favor light, controlled forces that facilitate tooth movement while minimizing hyalinization, vascular compromise, and damage to the cementoblastic layer. Intermittent force application and appropriate treatment intervals have also been proposed to allow periods of tissue recovery, supporting reparative processes on the root surface and reducing cumulative resorptive activity (15).

Advances in diagnostic imaging have enhanced the ability to detect early structural changes before substantial root loss occurs. Cone-beam computed tomography provides greater sensitivity than conventional radiography for identifying small resorptive defects and evaluating three-dimensional root morphology. This technology has improved risk assessment in patients presenting with predisposing anatomical features, previous dental


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trauma, or a history of root resorption. Periodic radiographic monitoring throughout treatment enables clinicians to identify progressive changes and modify biomechanics when necessary, limiting further tissue damage and preserving long-term tooth stability (16).

Interest has grown in adjunctive therapeutic approaches designed to influence biological pathways involved in resorption and repair. Low-intensity pulsed ultrasound has attracted attention because of its potential to stimulate cellular regeneration, enhance cementum repair, and modulate inflammatory responses within periodontal tissues. Experimental and clinical investigations suggest that ultrasounds may influence osteogenic and cementogenic activity while reducing the extent of clastic tissue destruction. Although current evidence remains heterogeneous, the biological rationale supporting its application has encouraged continued exploration of noninvasive interventions aimed at preserving root integrity during orthodontic treatment (17).

Non-pharmacological adjuncts, including photobiomodulation and other physical stimulation techniques, have also been evaluated for their capacity to regulate cellular metabolism and inflammatory signaling. These modalities are believed to enhance mitochondrial activity, promote tissue repair, and influence the expression of mediators involved in bone remodeling. Their incorporation into orthodontic practice reflects a broader shift toward biologically informed treatment strategies that prioritize tissue preservation alongside efficient tooth movement. As knowledge of molecular signaling pathways continues to expand, preventive approaches are increasingly focused on maintaining periodontal homeostasis and supporting physiological repair mechanisms throughout active orthodontic therapy (18).

## Conclusion

Orthodontically induced inflammatory root resorption remains a complex biological consequence of orthodontic tooth movement involving intricate interactions between mechanical forces, inflammatory mediators, and cellular signaling pathways. Individual susceptibility is influenced by genetic, anatomical, and immunological factors that modulate tissue responses to orthodontic loading. Advances in understanding mechanotransduction and molecular regulation have provided valuable insights into the pathogenesis of root resorption. Careful treatment planning, risk assessment, and the application of evidence-based preventive strategies are essential for preserving root integrity while achieving optimal orthodontic outcomes.

## Disclosure

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### *Data availability*

All data is available within the manuscript.

### *Author contribution*

All authors contributed to conceptualizing, data drafting, collection and final writing of the manuscript.

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