



(REVIEW ARTICLE)



LONG-TERM STABILITY AND BIOLOGICAL SIDE EFFECTS OF ORTHODONTIC TREATMENT: A NARRATIVE REVIEW OF RELAPSE MECHANICS, RETENTION STANDARDS, AND PATIENT-SPECIFIC EXTERNAL APICAL ROOT RESORPTION

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Global Journal of Research in Medicine and Dentistry, 2026, 05(04), 038–043

Publication history: Received on 20 August 2026; revised on 30 September 2026; accepted on 02 October 2026

Article DOI: <https://doi.org/10.58175/gjrmd.2026.5.4.0106>

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ABSTRACT

Orthodontic treatment reliably corrects malocclusion, yet two long-standing biological uncertainties continue to limit predictability of outcome: post-treatment relapse despite retention, and external apical root resorption (EARR) as an iatrogenic sequela of tooth movement. This narrative review synthesises the published literature on these two domains. For relapse and retention, the periodontal and gingival fibre apparatus, occlusal and neuromuscular equilibrium, and soft-tissue envelope are identified as the principal determinants of post-treatment stability, yet none of these factors, singly or combined, allow clinicians to predict which patients will relapse without lifelong retention. Fixed and removable retainer failure rates remain substantial over long follow-up periods, and stability appears largely independent of the complexity of the original malocclusion. For EARR, the literature confirms a multifactorial aetiology in which mechanical variables such as force magnitude, duration, and direction of movement interact with a substantial, heritable genetic component acting through interleukin-1, RANK/RANKL/osteoprotegerin, purinergic (P2X7R), and vitamin D receptor signalling pathways. Despite this, no validated pre-treatment screening protocol exists beyond conventional periapical or panoramic radiography, and clinicians remain unable to reliably identify high-risk patients before force application begins. Both domains share a common gap: biological variability at the periodontal, alveolar, and genetic level outpaces the predictive capacity of current clinical tools. Standardised long-term retention protocols and validated multifactorial risk-stratification models, potentially incorporating genetic markers alongside biomechanical and radiographic indices, represent priority areas for future research.

Keywords: Orthodontic Relapse; Retention; Root Resorption; External Apical Root Resorption; Periodontal Ligament; Genetic Polymorphism

1 INTRODUCTION

Fixed appliance and clear-aligner orthodontic therapy predictably corrects malocclusion in the majority of treated patients, restoring dental alignment, occlusal function, and facial aesthetics. Despite this predictability at the level of active treatment, two long-recognised biological uncertainties continue to constrain the profession's ability to guarantee a stable and unharmed long-term outcome: the tendency of corrected tooth positions to relapse once

appliances are removed¹, and the risk of external apical root resorption (EARR) as an iatrogenic biological cost of orthodontic force application².

Relapse and retention have been studied for over half a century, yet no consensus exists on the minimum duration, design, or intensity of retention required to guarantee permanent stability, and clinicians continue to observe unpredictable degrees of post-treatment movement even in apparently well-retained cases^{1,3}. Parallel to this, EARR remains one of the most extensively investigated yet incompletely explained complications of orthodontic care: although mechanical and biological risk factors have been repeatedly identified, no combination of clinical, radiographic, or genetic markers currently allows an orthodontist to reliably pre-screen a patient as "high-risk" before treatment begins².

This narrative review addresses these two persistent gaps in the evidence base. The first objective is to critically appraise the literature on relapse mechanics and retention standards, with particular attention to why long-term post-treatment stability remains poorly predicted by the complexity of the initial malocclusion, and why high relapse rates persist in the absence of lifelong retention. The second objective is to review the genetic, biological, and mechanical determinants of patient-specific EARR, and to evaluate the extent to which these factors can currently be translated into meaningful pre-treatment risk screening beyond routine radiographic monitoring.

2 MATERIALS AND METHODS

This article is a narrative review rather than a primary clinical study; accordingly, no human or animal subjects, patient data, or biological specimens were directly involved, and formal ethical approval and informed consent were not applicable to the preparation of this manuscript.

A structured literature search was conducted using PubMed/MEDLINE, Scopus, the Cochrane Library, and Google Scholar for articles published in English, with no lower date restriction, up to 2025. Search terms combined the concepts "orthodontic relapse", "orthodontic retention", "retainer failure", "post-treatment stability", "external apical root resorption", "orthodontically induced inflammatory root resorption", "root resorption genetics", and related synonyms, using Boolean operators and, where applicable, MeSH terms. Priority was given to systematic reviews, meta-analyses, and cohort or genetic-association studies with long-term follow-up; classic foundational studies on periodontal fibre biology and root resorption genetics were also retained where they remain the primary evidence source for a given claim. Reference lists of retrieved systematic reviews were hand-searched for additional eligible sources. Titles and abstracts were screened for relevance to the two review domains (relapse/retention and EARR), followed by full-text assessment of shortlisted articles. Findings were synthesised narratively rather than pooled quantitatively, consistent with the heterogeneous designs, outcome measures, and follow-up intervals of the included literature.

3 RESULTS AND DISCUSSION

3.1 Relapse Mechanics and Retention Standards

Orthodontic relapse is conventionally defined as the tendency of teeth to migrate back toward their pre-treatment positions following the removal of active appliances, and it remains one of the most consistently reported limitations of orthodontic treatment¹. In one of the field's most cited long-term cohorts, mandibular anterior alignment was found to change unpredictably over a 10- to 20-year postretention interval, with only a minority of cases remaining clinically acceptable without some form of continued retention³. Subsequent audits and cohort studies have reinforced this observation: relapse of overjet, overbite, arch form, and incisor irregularity can occur regardless of the retention protocol used, and no single pretreatment variable reliably predicts which individual patients will remain stable^{1,4}.

A central biological explanation for this unpredictability lies in the differential remodelling rates of the tissues altered during tooth movement. The principal fibres of the periodontal ligament reorganise relatively quickly following active movement, whereas the supracrestal (gingival) fibre network, and in particular the transseptal and free gingival fibres, remodel far more slowly and retain a form of tissue "memory" of the pretreatment tooth position for a substantially longer period^{5,6}. Experimental and histological work has shown that gingival connective tissue exerts a sustained, low-grade force on rotated or repositioned teeth long after orthodontic force has ceased, providing a plausible mechanistic basis for relapse of rotational corrections in particular^{6,7}. Because this soft-tissue remodelling process is not reliably altered by fixed appliance therapy itself, the neuromuscular and periodontal environment that originally contributed to the malocclusion is frequently left largely unchanged at the point of appliance removal^{8,9}.

Proffit's equilibrium theory offers a broader framework for this observation, proposing that tooth position reflects a balance between intraoral soft-tissue pressures (tongue, lips, cheeks), occlusal forces, and periodontal support, such that any orthodontic correction achieved without a corresponding permanent change in this soft-tissue and

neuromuscular equilibrium is inherently vulnerable to relapse once retentive forces are withdrawn⁹. This concept helps explain why relapse potential appears only loosely related to the initial severity or complexity of the malocclusion: a case that was mechanically straightforward to correct may nonetheless be highly unstable if the underlying soft-tissue and functional environment was never altered, while a more complex case corrected with attention to functional balance may prove comparatively stable^{10,4}.

These mechanistic uncertainties are reflected in the absence of a universally agreed retention standard. Contemporary opinion increasingly favours indefinite or lifelong retention, particularly for the mandibular labial segment, on the basis that the biological drive toward relapse, principally late mandibular growth, gingival fibre tension, and functional forces, does not resolve with time¹⁰. Yet even prolonged, well-maintained retention does not guarantee permanent stability: twenty-year follow-up of patients with permanently bonded mandibular canine-to-canine retainers has demonstrated that a meaningful proportion of retainers fail, fracture, or detach over time, with some degree of relapse observed even where retainers remained partially intact¹¹. Systematic review evidence corroborates this, reporting cumulative fixed retainer failure rates that increase substantially with each additional year of observation, most commonly due to bond failure or wire fracture rather than deliberate discontinuation^{12,13}. Removable retainer regimens are similarly vulnerable, since their effectiveness depends heavily on patient compliance, and audits of general dental practitioners have identified inconsistent understanding of retention protocols and follow-up responsibility once patients are discharged from specialist orthodontic care¹⁴.

The timing of relapse also argues against the assumption that stability is simply a function of a sufficiently long active treatment phase. Three-dimensional superimposition studies have demonstrated measurable dental movement within the first week after bracket removal, before any lapse in retainer wear would typically be expected, indicating that some component of relapse begins almost immediately as periodontal and occlusal forces re-equilibrate¹⁵. Comparable early instability has been documented following maxillary expansion for posterior crossbite correction, where systematic review data show that transverse relapse can occur even with structured retention protocols, reinforcing that the skeletal and dental changes achieved during treatment are not intrinsically self-maintaining¹⁶.

Taken together, the literature indicates that relapse is not primarily a failure of orthodontic mechanics but a reflection of an unresolved biological mismatch: active treatment reliably repositions teeth, but does not reliably or permanently alter the periodontal fibre architecture, occlusal function, or neuromuscular soft-tissue pressures that originally shaped the malocclusion^{8,9,17,18}. Because these tissue- and function-level variables are not routinely measured or altered during standard treatment planning, and because no validated pretreatment index currently integrates them into an individualised relapse-risk score, long-term stability remains, in practice, an empirical outcome observed only in retrospect rather than a variable that can be reliably predicted from the complexity of the original malocclusion^{1,4}.

3.2 Patient-Specific External Apical Root Resorption

External apical root resorption (EARR) is an inflammatory, osteoclast-mediated process resulting in permanent, irreversible loss of cementum and, in more severe cases, dentine at the root apex, and it is regarded as one of the most clinically significant iatrogenic risks of orthodontic tooth movement^{2,19}. Systematic review evidence confirms that some degree of radiographically detectable root shortening occurs in the great majority of orthodontically treated patients, with a smaller but clinically important subset developing severe resorption exceeding several millimetres of root length¹⁹.

Mechanically, the magnitude, duration, and direction of applied orthodontic force are consistently associated with the extent of EARR. Systematic review evidence indicates that comprehensive, longer-duration fixed appliance treatment increases both the incidence and severity of resorption relative to shorter or more limited treatment, and that heavier forces appear particularly harmful, whereas archwire sequencing, bracket prescription, and self-ligating versus conventional bracket design do not appear to meaningfully alter risk¹⁹. Root apex intrusion and torque movements, and apical displacement in general, have also been implicated as biomechanically higher-risk movements compared with tipping or bodily translation confined closer to the mid-root region^{20,21}. Comparative systematic review evidence suggests that appliance type itself, fixed labial appliances versus removable clear aligners, does not clearly alter the overall risk of EARR once treatment complexity and duration are accounted for, indicating that force delivery characteristics rather than appliance category per se are the more relevant mechanical determinant²².

At the cellular level, orthodontic force induces localised hyalinisation and sterile inflammation within the compressed periodontal ligament, recruiting osteoclast-like cells to the adjacent cementum and, once the protective cementoid and precementum layers are breached, to the underlying dentine^{20,23}. This cellular cascade is mediated by cytokine and bone-remodelling signalling pathways, including interleukin-1 (IL-1), receptor activator of nuclear factor- κ B (RANK) and its ligand RANKL, osteoprotegerin (OPG), and purinergic P2X7 receptor signalling, all of which regulate osteoclastogenesis and have been proposed as both mechanistic drivers and potential biomarkers of resorptive risk^{20,24,25}.

Critically, mechanical loading alone does not account for the substantial inter-individual variation observed clinically, since patients receiving comparable force systems for comparable durations frequently display markedly different degrees of root shortening^{26,27}. A genetic contribution to this variability was first substantiated in family-based heritability studies and subsequently confirmed in a retrospective twin cohort, which together indicate that a substantial proportion, by some estimates approaching or exceeding half, of the variance in EARR severity is attributable to genetic rather than purely mechanical or environmental factors^{28,29,27}.

Candidate-gene studies have since identified several polymorphisms associated with increased or decreased EARR susceptibility. Variation within the IL-1A and IL-1B genes has shown linkage and association with EARR severity in multiple independent cohorts, including analyses of endodontically treated teeth and their contralateral vital controls^{30,31,32}. Polymorphisms in genes encoding the P2X7 receptor and in the RANK/RANKL/OPG bone-remodelling axis have similarly been associated with differential resorption risk, implicating osteoclast regulatory pathways more broadly rather than a single isolated gene^{24,25}. Vitamin D receptor gene polymorphisms have also been associated with EARR in combination with specific clinical variables such as increased overjet, suggesting that genetic and mechanical risk factors may interact rather than act independently³³. Linkage analyses targeting additional candidate loci, including markers on chromosome 18, have yielded more equivocal findings, underscoring that EARR is genetically complex rather than attributable to a single Mendelian locus³⁴.

Beyond cytokine and bone-remodelling genetics, systemic bone metabolism itself appears relevant: reduced systemic bone mineral density has been associated with increased susceptibility to EARR, plausibly reflecting shared regulatory pathways between skeletal bone turnover and cementum/dentine resorption at the root surface³⁵. Adjunctive and modifying biological factors, including systemic conditions and pharmacological exposures, have also been examined as potential effect-modifiers of resorption risk during active treatment, although the overall evidence base for specific adjunctive interventions remains limited and heterogeneous³⁶.

Despite this expanding mechanistic and genetic literature, no validated composite risk-screening tool currently exists that reliably identifies, before treatment begins, which individual patients will develop clinically significant EARR. Root morphology, pre-existing idiopathic resorption, and other radiographically visible features can flag some at-risk individuals, but none of these markers, alone or in combination with known clinical variables such as overjet or treatment duration, is sufficient on its own to predict EARR reliably at the individual patient level^{26,27}. Genetic testing for IL-1, RANK/RANKL/OPG, or P2X7R polymorphisms remains confined to the research setting and has not been translated into routine pre-treatment screening protocols^{31,24}. As a result, current clinical practice continues to rely almost exclusively on baseline periapical or panoramic radiography, supplemented by interim radiographic review during treatment, to monitor for resorption after it has already begun rather than to pre-screen for susceptibility before force application, leaving the precise genetic, biological, and mechanical thresholds that trigger severe EARR in a given patient largely unresolved^{2,19}.

3.3 Convergent Themes and Clinical Implications

Although relapse/retention and EARR are typically discussed as separate clinical problems, they share a common underlying pattern: in both domains, the biological response of periodontal, gingival, and dental hard tissues to orthodontic force is more variable between individuals than current clinical prediction tools can account for^{1,2}. In relapse, this variability manifests as an unpredictable soft-tissue and neuromuscular "memory" that undermines mechanically successful corrections^{6,9}; in EARR, it manifests as an individualised, partly genetically determined inflammatory and resorptive response to an otherwise standard mechanical stimulus^{28,27}. In neither case does the complexity or type of the original malocclusion, nor the technical quality of tooth movement achieved, reliably forecast the biological outcome that follows^{4,26}.

From a clinical standpoint, this suggests that both problems are best managed through a strategy of monitoring and mitigation rather than pretreatment elimination of risk. For relapse, this means defaulting toward long-term or indefinite retention protocols, particularly of the mandibular labial segment, with realistic counselling of patients that retention is a permanent maintenance requirement rather than a finite postoperative phase, alongside routine review of retainer integrity given the substantial failure rates reported over multi-year follow-up^{10,12,13}. For EARR, this means continued reliance on interim radiographic monitoring during active treatment, with force reduction, a treatment pause, or a change in mechanics considered where early resorption is detected, since these remain the only interventions with reasonable supporting evidence once treatment is underway^{19,36}.

The priority for future research in both domains is the development and prospective validation of composite risk-stratification tools that move beyond single-variable prediction. For retention, this would ideally integrate soft-tissue, functional, and occlusal indices alongside traditional cephalometric and model-based measures to generate an individualised relapse-risk profile at the point of debond^{9,10}. For EARR, this would involve prospective validation of genetic panels (IL-1, RANK/RANKL/OPG, P2X7R, and vitamin D receptor variants) in combination with established

clinical and radiographic predictors, tested against clinically meaningful resorption thresholds rather than statistical association alone^{31,24,33}. Until such tools are validated, long-term stability and root integrity will remain, in large part, individually variable biological outcomes rather than fully predictable consequences of treatment planning.

4 CONCLUSION

Long-term orthodontic stability and biological safety continue to be limited less by mechanical or technical shortcomings than by an incomplete understanding of individual patient biology. Relapse reflects the slow and incomplete remodelling of periodontal and gingival fibre architecture and the persistence of pretreatment soft-tissue and neuromuscular pressures, such that stability cannot currently be reliably predicted from the complexity of the original malocclusion, and high relapse rates persist wherever lifelong retention is not maintained. EARR reflects a comparably multifactorial interaction between mechanical loading and a substantial, polygenic biological susceptibility acting through interleukin-1, RANK/RANKL/OPG, and related bone-remodelling pathways, the precise thresholds of which remain incompletely defined at the individual patient level. In both domains, current clinical practice depends on monitoring and reactive management, indefinite retention on one hand and radiographic surveillance on the other, rather than validated pretreatment risk prediction. Integrating biomechanical, soft-tissue, and genetic risk markers into prospectively validated, individualised screening tools represents the most promising direction for narrowing these long-standing gaps in orthodontic evidence.

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