

Original Article

Periodontal Phenotype and Orthodontic Treatment Outcomes: Evidence on Gingival Recession, Stability, and Risk

Hiroshi Tanaka^{1*}, Nur S. Ismail¹, Hana T. Desta¹

¹Department of Periodontics and Orthodontics, Graduate School of Dentistry, Osaka University, Osaka, Japan.

*E-mail ✉ h.tanaka@outlook.jp

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ABSTRACT

The periodontal phenotype, encompassing gingival thickness, keratinized tissue width, and alveolar bone morphology, plays a pivotal role in determining the outcomes of orthodontic treatment, particularly concerning gingival recession, post-treatment stability, and associated risks. This narrative review synthesizes evidence from peer-reviewed studies published between 2020 and 2025 to elucidate the interplay between periodontal phenotype and orthodontic interventions. Thin periodontal phenotypes are associated with heightened susceptibility to gingival recession during orthodontic tooth movement, with studies indicating increased incidence rates post-treatment compared to thick phenotypes. For instance, cross-sectional analyses reveal that thin gingival biotypes correlate with greater recession prevalence, especially in mandibular anterior regions, exacerbated by factors such as bleeding on probing and age. Longitudinal data suggest that orthodontic treatment with fixed appliances may elevate recession risk by up to 67% relative to untreated controls, though severity often remains mild. Stability of orthodontic outcomes is influenced by phenotype, as thicker gingiva and bone provide enhanced resistance to relapse and periodontal breakdown. Risk assessments highlight the importance of pre-treatment phenotype evaluation to mitigate adverse effects, with phenotype modification techniques, including soft tissue augmentation, demonstrating efficacy in preventing recession and improving long-term stability. Objectives of this review include delineating phenotype classifications, assessment methods, and evidence-based strategies for optimizing orthodontic outcomes in diverse phenotypes. By integrating multidisciplinary approaches, clinicians can minimize risks and enhance periodontal health, underscoring the need for tailored treatment protocols.

Keywords: Periodontal phenotype, Gingival recession, Orthodontic treatment, Treatment stability, Risk factors, Soft tissue augmentation

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Introduction

Orthodontic treatment aims to achieve functional occlusion, aesthetic harmony, and long-term stability while preserving periodontal health. However, tooth movement can impose biomechanical stresses on the periodontium, potentially leading to adverse outcomes such as gingival recession, alveolar bone loss, and relapse [1,2]. The concept of periodontal phenotype, which integrates gingival biotype (thick versus thin),

keratinized tissue dimensions, and underlying bone architecture, has emerged as a critical determinant of these outcomes [3-5]. Historically, gingival biotype was classified simplistically as thick or thin based on visual and probe transparency assessments, but recent advancements emphasize a multifaceted phenotype encompassing soft and hard tissue characteristics [6,7]. In patients undergoing orthodontic therapy [8-12], a thin periodontal phenotype is frequently linked to increased vulnerability to gingival recession, particularly in the labial aspects of mandibular incisors

where tooth proclination may exceed alveolar housing limits [13-15]. Evidence from prospective studies indicates that fixed appliance therapy can exacerbate recession in thin phenotypes, with incidence rates rising significantly post-retention [16]. Conversely, thick phenotypes offer greater resilience, reducing recession risk and supporting treatment stability [17,18]. Stability, defined as the maintenance of corrected tooth positions without relapse, is influenced by periodontal support; inadequate keratinized tissue or thin bone may compromise retention, leading to occlusal instability [19].

Risk factors associated with unfavorable outcomes include patient age, gender, skeletal patterns (e.g., hyperdivergent faces correlating with thin phenotypes), and pre-existing periodontal conditions [20-22]. Phenotype modification strategies, such as connective tissue grafting prior to orthodontics, have shown promise in augmenting soft tissue and preventing recession [23-25]. This review addresses the gap in synthesizing recent evidence (2020-2025) on how periodontal phenotype modulates orthodontic outcomes, focusing on gingival recession, stability, and risk.

The objectives are threefold: (1) to define and classify periodontal phenotypes relevant to orthodontics; (2) to examine their impact on gingival recession and treatment stability based on contemporary studies; and (3) to identify risk factors and evidence-based management approaches [26-31]. By thematic organization, this narrative review provides clinicians with insights to integrate periodontal assessments into orthodontic planning, ultimately enhancing patient outcomes [32, 33].

Definition and Classification of Periodontal Phenotype

The periodontal phenotype represents the observable characteristics of the periodontium, influenced by genetic and environmental factors, and includes gingival thickness (GT), width of keratinized gingiva (KGW), and alveolar bone morphology [3]. Traditionally, gingival biotype was dichotomized into "thick" (flat, dense gingiva with broad KGW) and "thin" (scalloped, translucent gingiva with narrow KGW), but modern classifications adopt a more nuanced approach [6]. Recent studies advocate for a phenotype encompassing both soft and hard tissues, categorized as thin, medium, or thick based on GT thresholds: thin (<1 mm), thick (>1 mm) [7, 34].

In orthodontic contexts, phenotype classification is crucial as it predicts tissue response to tooth movement [35-37]. For example, a 2025 study classified

phenotypes using transgingival probing at multiple levels (sulcus base and 1-2 mm apical), revealing GT variations by arch location, with thicker phenotypes in maxillary posteriors [21]. Gender differences are noted, with females more likely to exhibit thin phenotypes (odds ratio 4.1-6.7) [18]. Facial patterns also correlate: hyperdivergent faces associate with thin phenotypes, hypodivergent with thick [22].

Alveolar bone thickness integrates into phenotype definitions, as thin labial bone (<1 mm) heightens dehiscence risk during orthodontics [4]. A comprehensive analysis from 2025 emphasized interactions between GT, KGW, and tooth morphology, showing moderate correlations ($r=0.4-0.6$) between GT and gingival angle, but weak negative links with crown length [18]. These classifications underscore the need for phenotype-specific orthodontic planning to avert complications.

Assessment Methods for Periodontal Phenotype

Accurate phenotype assessment is foundational for risk stratification in orthodontics. Methods range from non-invasive visual evaluations to invasive measurements, with recent studies validating their reliability [6,20, 23].

Visual assessment (VA) involves observing gingival contour and probe transparency through the gingiva (probe transparency method, PTM), where visibility indicates thin phenotype [20]. A 2025 comparative study of VA, PTM, and direct biometric ultrasonography in maxillary incisors found strong correlations ($r=0.62-0.76$) between methods, affirming VA and PTM as reliable for anterior regions [23]. PTM using color-coded probes showed no superiority over VA, with both aligning well with direct measurements [23].

Transgingival probing measures GT at standardized points, often under anesthesia [21]. A 2025 investigation compared single-point versus multi-level GT (mean GT from sulcus base to 2 mm apical), demonstrating good agreement ($\kappa=0.71-0.76$) for upper levels but moderate for apical ($\kappa=0.53$), highlighting vertical measurement variability [21]. Cone-beam computed tomography (CBCT) assesses bone morphology, revealing phenotype-related bone thickness differences [15].

Clinical parameters like KGW and papilla height complement assessments [18]. In a 2023 Bulgarian cohort, thick phenotypes predominated (thick flat > thick scalloped), with significant KGW variations by phenotype [38]. These methods enable pre-orthodontic phenotype identification, guiding interventions.

Impact of Periodontal Phenotype on Gingival Recession During Orthodontic Treatment

Gingival recession (GR), characterized by apical migration of the gingival margin, is a common sequela of orthodontic treatment, particularly in thin phenotypes [1,3, 13]. Evidence indicates thin gingiva (<1 mm) and narrow KGW predispose to recession, as orthodontic forces may induce dehiscence [4,16].

A 2025 cross-sectional study of 96 post-orthodontic patients found GR more prevalent in thin phenotypes, linked to bleeding on probing (>30%) and age, but not incisor protrusion [1]. Prospective controlled research reported 67% higher GR incidence in fixed appliance-treated patients versus controls at 1-year post-treatment, predominantly mild (≤ 1 mm) in canines and premolars [3]. Thin phenotypes exhibited greater severity, with recession areas up to 1.85 mm² in canines [14].

Longitudinal comparisons of fixed orthodontic therapy (FOT) and Invisalign in thin versus thick phenotypes showed significant FMPS, GI, FMBS, and PPD reductions over 6 months, but GR persisted more in thin groups [5]. Pre-orthodontic soft tissue augmentation protocols, like connective tissue grafting (CTG), prevented GR, with 69% root coverage versus 22% in untreated [7,25].

In skeletal Class III malocclusion, combined periodontal-orthodontic-orthognathic treatment improved phenotypes, increasing GT and KGW by 1.5-2 mm, reducing recession [15]. These findings affirm thin phenotypes amplify GR risk, necessitating preventive measures.

Role of Periodontal Phenotype in Orthodontic Stability

Orthodontic stability relies on periodontal integrity, with phenotype influencing relapse susceptibility [2,17]. Thick phenotypes provide robust support, resisting occlusal forces and maintaining alignment [18,19].

A 2025 review highlighted biomechanical strategies for periodontal-compromised patients, noting thick phenotypes correlate with lower relapse rates due to enhanced bone and gingival resilience [2]. In post-retention evaluations, GR incidence rose from 11% post-treatment to 67% at 5+ years, but thick phenotypes showed less progression [14]. Phenotype modification via surgically facilitated orthodontics (SFOT) with bone grafting accelerated movement and stabilized outcomes, yielding thick KGW and no recession at 3-year follow-up [2].

In Class III cases, phenotype enhancement increased labial bone thickness and height, supporting stability [10]. Thin phenotypes, associated with hyperdivergent patterns, exhibited higher instability risks, with increased probing depths predicting relapse [14,22]. Multidisciplinary approaches, integrating periodontal therapies, optimize stability in vulnerable phenotypes [2].

Risk Factors Associated with Thin Periodontal Phenotype in Orthodontics

Thin periodontal phenotypes are widely recognized as a significant risk factor in orthodontic treatment, predisposing patients to gingival recession (GR), alveolar bone dehiscence, compromised post-treatment stability, and accelerated progression of periodontal disease [4,24]. Understanding the determinants and amplifiers of thin phenotypes is essential for risk stratification and individualized treatment planning.

Demographic and Anatomical Factors

Gender and age are consistently implicated as determinants of thin phenotypes. Epidemiological studies indicate that females are more likely to exhibit thin gingival biotypes, potentially due to differences in gingival connective tissue composition, collagen density, and hormonal influences on vascularization and tissue remodeling [1,18,22]. Age-related changes in the periodontium, including reduced fibroblast activity, diminished collagen synthesis, and gradual alveolar bone loss, may further exacerbate vulnerability in thin phenotypes. Skeletal morphology also plays a role: hyperdivergent facial patterns are frequently associated with thin gingival tissue and alveolar bone, increasing the odds of recession during orthodontic tooth movement [22]. Pre-treatment periodontal status—including baseline keratinized gingival width (KGW), existing recession, and inflammatory markers—strongly influences risk, as compromised tissue is less resilient to mechanical and inflammatory challenges [1,18].

Gingival Thickness and Morphology

Gingival thickness (GT) remains the most robust predictor of risk. Studies demonstrate that $GT \leq 1$ mm significantly increases the likelihood of GR, with reported odds ranging from 4- to 6.7-fold compared to thicker biotypes [1,18]. Scalloped gingival morphologies, often observed in thin phenotypes, are more susceptible to trauma, inflammation, and surgical insult, whereas thick, flat phenotypes confer protective

advantages and maintain structural integrity under orthodontic forces [38]. Analogous evidence from implantology indicates that thin phenotypes indirectly elevate peri-implantitis risk, reinforcing the principle that tissue thinness reduces resistance to iatrogenic and inflammatory insults [24].

Orthodontic Mechanics as Risk Amplifiers

Orthodontic appliance type and biomechanics significantly modulate risk in thin phenotypes. Fixed appliances, particularly those with continuous archwires, are associated with higher incidences of GR, especially after the retention phase [3,14]. Lingual retainers, if improperly adapted or non-passive, can induce unintended tooth movement, exacerbating soft tissue trauma and promoting recession [16]. A 2025 case series reported multiple instances of lower anterior GR linked to faulty lingual retainers, which were successfully mitigated through connective tissue grafting (CTG), underscoring the importance of appliance precision and maintenance in high-risk phenotypes [16].

Periodontal Health Markers and Inflammatory Burden

Pre-existing periodontal health further influences outcomes. Elevated bleeding on probing (BOP) scores, increased plaque accumulation, and chronic gingival inflammation amplify the susceptibility of thin phenotypes to recession during orthodontic therapy [1,38]. Cross-sectional prevalence studies reveal that thin, scalloped gingival phenotypes predominate among patients with gingivitis, whereas thick, flat phenotypes are more commonly observed in periodontally healthy populations, suggesting that inherent tissue thickness confers resilience against inflammatory insults [38].

Risk Mitigation Strategies

Effective mitigation requires systematic phenotype assessment, including transgingival probing, ultrasonography, or CBCT-based measurement of gingival and alveolar dimensions. Prophylactic interventions—such as soft tissue augmentation, CTG, or SFOT—can enhance gingival thickness and keratinized tissue, reducing susceptibility to GR and improving post-treatment stability [5,6]. Orthodontic mechanics should be adapted according to tissue phenotype, with controlled force application, careful retainer design, and ongoing periodontal monitoring to minimize trauma. Patient education on oral hygiene

and plaque control further complements mechanical and surgical strategies, fostering a biologically favorable environment for safe tooth movement.

Collectively, these risk factors highlight the multifactorial nature of thin periodontal phenotypes in orthodontics, where demographic, anatomical, mechanical, and inflammatory determinants converge to influence clinical outcomes [39-46]. Early identification and proactive management of high-risk patients are essential for minimizing adverse sequelae and optimizing both periodontal and orthodontic stability.

Strategies for Phenotype Modification in Orthodontic Patients

Periodontal phenotype modification is increasingly recognized as a critical adjunct to orthodontic treatment, particularly for patients with thin biotypes who are at heightened risk of gingival recession (GR), alveolar bone dehiscence, and post-treatment instability [2,5,25]. By enhancing soft and hard tissue dimensions before or during orthodontic therapy, these strategies aim to improve the biological envelope, optimize force distribution, and preserve periodontal integrity throughout treatment [47, 50].

Soft Tissue Grafting

Soft tissue grafting, including connective tissue grafts (CTGs) and free gingival grafts, remains the cornerstone of phenotype enhancement. These procedures increase keratinized gingival width (KGW) and gingival thickness, providing mechanical and biological support against orthodontic forces. A 2022 randomized controlled trial evaluated pre-orthodontic CTG, demonstrating an anticipated gain in KGW of approximately 1.6 mm. Notably, post-grafting stability was preserved during orthodontic movement, with 69% of sites achieving complete recession coverage [6,7]. The procedure not only augments tissue volume but also enhances vascularization and fibroblast activity, which are essential for sustaining tissue health under biomechanical stress.

Surgically Facilitated Orthodontic Therapy (SFOT)

Surgically facilitated orthodontic therapy (SFOT), often combined with allografts and platelet-rich fibrin (PRF), has emerged as an effective strategy for modifying thin phenotypes while accelerating orthodontic movement. Clinical reports indicate that SFOT can safely achieve desired tooth repositioning in

as little as six months, with stable prevention of gingival recession observed at three-year follow-up [2]. Mechanistically, selective decortication and bone augmentation stimulate regional acceleratory phenomena, enhancing osteoclastic and osteoblastic activity, improving alveolar bone support, and reinforcing the overlying gingiva.

Combined Periodontal–Orthodontic–Orthognathic Approaches

In complex skeletal malocclusions, such as Class III cases, combined periodontal-orthodontic-orthognathic interventions have demonstrated success in achieving durable thick phenotypes. Bone grafting, guided tissue regeneration, and strategic orthodontic movements work synergistically to augment alveolar bone and gingival dimensions, producing stable soft and hard tissue envelopes that support long-term orthodontic outcomes [15]. These integrated approaches highlight the importance of interdisciplinary planning, particularly in high-risk phenotypes or severe malocclusions.

Biologic and Mechanobiologic Adjuncts

Additional biologic adjuncts, including selective decortication, piezosurgical corticotomies, and the use of growth factor–enriched matrices, further support tissue regeneration and phenotype modification [2]. These techniques enhance local cellular activity, including fibroblast proliferation, angiogenesis, and osteogenesis, thereby reinforcing both gingival and alveolar structures against mechanical stress. When combined with orthodontic forces, these interventions can reduce the risk of iatrogenic recession, improve soft tissue coverage, and enhance post-treatment stability [7,16].

Collectively, these phenotype modification strategies underscore a proactive approach to orthodontic care, emphasizing risk mitigation, biologically guided treatment, and long-term periodontal preservation. By tailoring interventions to individual phenotypes, clinicians can achieve safer tooth movement, minimize adverse sequelae such as gingival recession or root exposure, and optimize esthetic and functional outcomes.

Discussion

The interplay between periodontal phenotype and orthodontic treatment outcomes represents a multifaceted domain where soft and hard tissue characteristics profoundly influence susceptibility to

gingival recession, post-treatment stability, and overall risk profiles. Synthesizing the thematic elements from the preceding sections, it is evident that thin periodontal phenotypes—characterized by gingival thickness (GT) less than 1 mm, narrow keratinized tissue width (KTW) below 2 mm, and thin labial bone—predispose patients to adverse effects during and after orthodontic interventions [1-3, 51-60]. This vulnerability stems from biomechanical stresses imposed by tooth movement, which can exacerbate pre-existing dehiscences or fenestrations, leading to apical migration of the gingival margin [4, 52, 54]. Conversely, thick phenotypes confer protective benefits, enhancing tissue resilience and mitigating recession progression [5, 53, 55].

A critical examination of the evidence reveals consistent associations between thin phenotypes and elevated gingival recession incidence. For instance, cross-sectional analyses of post-orthodontic cohorts demonstrate higher recession prevalence in thin biotypes, particularly in mandibular incisors, where factors such as age and bleeding on probing amplify risks [51]. Longitudinal data further corroborate this, showing recession rates escalating from 11% at treatment completion to over 67% five years post-retention in adolescents with fixed appliances and lingual retainers, with thin phenotypes and vertical skeletal patterns (e.g., high mandibular plane angle) as key predictors [52]. These findings align with narrative reviews emphasizing excessive incisor proclination in thin labial bone as a precipitating factor for recession, underscoring the need for phenotype-tailored movement limits [53]. However, not all studies attribute recession solely to orthodontics; some indicate no direct causal link, instead highlighting periodontal health markers like bleeding on probing ($\geq 30\%$) and age as primary influencers, with orthodontic movement exerting minimal independent effect [51]. This discrepancy may arise from methodological variations, such as retrospective designs lacking baseline recession measurements or heterogeneous phenotype assessment protocols.

Phenotype assessment methods, while diverse, exhibit limitations that impact their clinical utility and the interpretability of study outcomes. Indirect techniques like probe transparency and visual inspection offer practicality but suffer from moderate reliability, especially in posterior regions [6, 54]. Direct methods, including transgingival probing and cone-beam computed tomography (CBCT) for bone morphotype, provide greater accuracy but are invasive or radiation-intensive, reserving their use for high-risk cases [7, 54]. Recent evidence advocates for multimodal approaches,

integrating clinical parameters (e.g., KTW, papilla height) with imaging to refine classifications [13, 55]. Gender and craniofacial morphology add layers of complexity; females and hyperdivergent faces are more prone to thin phenotypes, potentially confounding associations with recession [18,22, 53]. These demographic factors warrant consideration in risk stratification, as they may interact with orthodontic mechanics to heighten instability.

Regarding treatment stability, thick phenotypes appear to bolster long-term alignment by resisting relapse and periodontal breakdown [2,17, 56]. Evidence from best evidence consensus suggests that phenotype modification therapies (PhMT), such as connective tissue grafts (CTG) or corticotomy-assisted orthodontic therapy (CAOT) with bone augmentation, can convert thin to thick phenotypes, thereby enhancing stability [56, 57]. For example, CAOT combined with particulate grafting accelerates movement, reduces treatment duration, and minimizes relapse as measured by mandibular irregularity indices over 10 years [57]. In Class III malocclusions requiring orthognathic surgery, phenotype enhancement via combined interventions increases labial bone thickness and KTW, supporting occlusal stability [15, 58]. Nonetheless, the evidence base is constrained by a paucity of long-term prospective studies; most data derive from short-term follow-ups (<5 years), limiting insights into sustained stability [56]. Moreover, while PhMT expands safe movement envelopes—particularly for decompensation in thin phenotypes—it introduces procedural complexities, costs, and risks like surgical complications or root damage [56].

Risk factors extend beyond phenotype to encompass orthodontic modalities and patient behaviors [61-66]. Fixed appliances correlate with higher recession incidence compared to aligners, though both may induce mild recession in thin biotypes [3,5, 59]. Lingual retainers, if maladapted, can provoke localized recession, necessitating vigilant monitoring [16, 52]. Pre-existing periodontal conditions, such as plaque accumulation or gingivitis, exacerbate risks, with thin scalloped phenotypes predominant in inflamed states [38, 55]. Modification strategies mitigate these; soft tissue augmentation pre-orthodontics prevents recession in vulnerable sites, achieving up to 1 mm GT gains and wider KTW [6,25, 60]. Hard tissue PhMT, like surgically facilitated orthodontic therapy (SFOT), further safeguards against dehiscence, though its benefits are most pronounced in interdisciplinary contexts [2, 57]. Despite these advances, gaps persist: few studies address ethnic variations, with some populations (e.g., Asians) exhibiting higher thin

phenotype prevalence [56]. Additionally, the absence of randomized controlled trials (RCTs) comparing PhMT versus no intervention in orthodontic patients hampers causal inferences, with reliance on consensus statements and retrospectives introducing bias [56, 57]. Clinically, these insights advocate for a paradigm shift toward proactive phenotype evaluation and modification. Pre-treatment protocols should incorporate comprehensive assessments to identify at-risk patients—those with thin GT (<1 mm), narrow KTW (<2 mm), or thin bone (<1 mm)—and integrate periodontal consultations for augmentation where indicated [28,30]. For instance, in cases of planned labial expansion or proclination, PhMT can avert recession and enhance esthetics, though timing is crucial: pre-orthodontic grafting stabilizes tissues, while intra-treatment CAOT expedites progress [57, 60]. Post-treatment, frequent periodontal surveillance is essential, particularly in thin phenotypes, to detect early recession and ensure stability [51, 52]. This multidisciplinary approach not only minimizes risks but also optimizes outcomes, aligning with evidence-based guidelines from bodies like the American Academy of Periodontology (AAP) [56, 57].

Limitations across the reviewed literature merit acknowledgment. Many studies employ cross-sectional or retrospective designs, precluding causality and potentially overestimating associations due to recall bias [1, 51, 59]. Heterogeneity in phenotype definitions—ranging from binary (thin/thick) to tripartite (thin/medium/thick)—and assessment tools complicates meta-analyses [3, 6, 53]. Sample sizes are often modest, with underrepresentation of diverse demographics, limiting generalizability [18, 22]. Furthermore, while 2020-2025 publications reflect contemporary advancements, the recency restricts long-term data on PhMT durability [56]. Future research should prioritize RCTs with standardized phenotypes, longitudinal tracking beyond 5 years, and cost-effectiveness analyses to refine protocols.

In summary, the evidence underscores periodontal phenotype as a cornerstone of orthodontic success, with thin variants heightening recession and instability risks, amenable to modification for improved prognoses. By bridging periodontal and orthodontic disciplines, clinicians can foster resilient outcomes, though ongoing research is imperative to address evidentiary gaps.

Conclusions and Future Directions

This narrative review highlights the critical influence of periodontal phenotype on orthodontic treatment outcomes, emphasizing its role in determining

susceptibility to gingival recession, alveolar bone remodeling, and post-treatment stability. Thin periodontal phenotypes—characterized by reduced gingival thickness, minimal keratinized tissue, and slender alveolar bone—are consistently associated with heightened vulnerability to soft tissue dehiscence, gingival recession, and potential root exposure during orthodontic tooth movement (OTM). Conversely, thick phenotypes, with robust gingival dimensions and thicker alveolar support, confer protective advantages, buffering against inflammatory and mechanical challenges, and contributing to improved post-treatment stability. Through comprehensive examination of recent peer-reviewed evidence (2020–2025), this review successfully delineates phenotype classifications, assessment modalities, impacts on recession and stability, associated risks, and modification strategies, thereby providing a cohesive framework for understanding how periodontal morphology shapes orthodontic outcomes.

Clinically, these insights underscore the necessity of pre-treatment periodontal phenotype evaluation to inform individualized treatment planning. Assessment modalities, ranging from visual inspection and transgingival probing to advanced imaging techniques such as cone-beam computed tomography (CBCT) and high-frequency ultrasonography, enable accurate quantification of gingival and alveolar dimensions, facilitating risk stratification. Based on phenotype characterization, tailored interventions—including soft tissue grafting, autogenous or allogenic hard tissue augmentation, and minimally invasive surgical techniques—can mitigate the adverse effects of thin phenotypes, preserve alveolar bone integrity, and optimize both esthetic and functional results. Integrating such interventions into routine orthodontic protocols enhances long-term periodontal health while maintaining the biomechanical objectives of OTM.

Looking forward, future research should prioritize large-scale, multicenter, prospective randomized controlled trials (RCTs) to establish robust causal relationships between periodontal phenotype modification therapy (PhMT) and reductions in gingival recession, root resorption, and relapse. Incorporating state-of-the-art imaging technologies, such as CBCT, ultrasound, and optical coherence tomography, will allow precise, reproducible quantification of periodontal dimensions and remodeling dynamics over time. Additionally, investigations into the influence of genetic polymorphisms, epigenetic regulation, environmental factors, and ethnic variability on periodontal phenotype expression may reveal novel determinants of individual

susceptibility, enabling truly personalized orthodontic care.

Further exploration should assess how different orthodontic appliances—fixed brackets versus clear aligners—interact with diverse phenotypes, evaluating both the biomechanical and inflammatory implications for tissue remodeling. Comparative studies on force application strategies, duration, and magnitude in thin versus thick phenotypes may provide practical guidelines to minimize iatrogenic recession and maximize treatment stability. Economic evaluations of PhMT interventions, including cost-effectiveness analyses of pre-treatment soft/hard tissue augmentation versus long-term management of post-orthodontic complications, will inform accessible and sustainable care pathways for diverse populations.

Finally, integrating these advances into interdisciplinary treatment guidelines will foster closer collaboration between periodontists, orthodontists, and general dentists. Standardized protocols for phenotype assessment, modification, and monitoring will enhance patient safety, optimize periodontal–orthodontic outcomes, and facilitate evidence-based decision-making. Collectively, this approach promises to advance the precision, predictability, and long-term stability of orthodontic therapy, ultimately improving oral health outcomes across varied patient populations.

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