OPEN ACCESS
[DOI]10.12016/j.issn.2096-1456.202660119

·综述·

牙根发育未完成恒牙正畸治疗的研究进展

耿瑞，　刘钧
口腔疾病防治全国重点实验室 国家口腔医学中心 国家口腔疾病临床医学研究中心 四川大学华西口腔医院正畸科，四川 成都（610041）

【摘要】 错𬌗畸形是儿童及青少年中最常见的口腔疾病之一。为避免错𬌗畸形进一步加重，恒牙列早期阶段实施正畸干预的临床需求不断增加。然而，此阶段部分牙齿牙根尚未发育完成，正畸力是否会干扰未成熟牙根的正常发育过程或增加牙根吸收风险，长期以来一直是临床实践中备受关注且存在争议的问题。基础研究显示，适度正畸力可激活牙周组织改建及成牙本质细胞功能，但未成熟牙根在根尖区存在更高的应力敏感性。有限元分析提示，不同牙根长度与形态条件下应力分布存在显著差异，短根及牙根形态异常牙齿更易在根尖区产生应力集中。临床研究未发现未成熟牙根在合理力值下出现显著发育停滞，但个体易感性及力学控制不当仍可能增加牙根吸收风险。对于阻生牙需要牵引及颅颌面发育异常的特殊患者，早期正畸干预具有现实必要性，故对治疗时机提出了更高要求。综合现有证据，牙根发育未完成并非正畸治疗的绝对禁忌，对于常规病例，可将恒牙牙根形成约2/3（Nolla 8期左右）作为启动治疗的参考；对于阻生牙或颅颌面发育异常等可能错失干预窗口的患者，不宜仅因根尖未闭合而延迟治疗，而应结合牙根发育阶段、阻生位置、牵引方向及预期获益进行个体化决策；一旦出现进行性牙根吸收或牙髓、牙周异常，应及时减力、暂停或调整治疗方案。未来研究应加强不同发育阶段的分层临床研究，并结合影像学与生物标志物探索牙根发育动态监测方法，进一步优化力值选择与风险评估策略。本综述围绕牙根发育未完成恒牙的正畸治疗，从生物学基础、力学机制及临床研究三个层面进行系统梳理，旨在为临床治疗时机选择及个体化治疗决策提供更多理论依据。

【关键词】 正畸治疗；　牙根发育；　牙根吸收；　未成熟牙根；　正畸治疗时机；　阻生牙牵引；　生物力学；　口腔医学
【中图分类号】 R78　【文献标志码】 A　【文章编号】 2096-1456（2026）10-1033-12
【引用著录格式】 耿瑞，刘钧. 牙根发育未完成恒牙正畸治疗的研究进展[J]. 口腔疾病防治，2026，34(10)：1033-1044. doi:10.12016/j.issn.2096-1456.202660119.

微信公众号

Research progress on the orthodontic treatment of permanent teeth with incomplete root development

GENG Rui, LIU Jun. State Key Laboratory of Oral Diseases & National Center for Stomatology & National Clinical Research Center for Oral Diseases & Department of Orthodontics, West China Hospital of Stomatology, Sichuan University, Chengdu 610041, China
Corresponding author: LIU Jun, Email:junliu@scu.edu.cn

【Abstract】 Malocclusion is one of the most common oral conditions in children and adolescents. To prevent its progression, the demand for earlier orthodontic intervention during the early permanent dentition has been increasing. However, at this stage, the roots of some teeth are not yet fully developed. Whether orthodontic force interferes with the normal development of immature roots or increases the risk of root resorption has long been a controversial issue in clinical practice. Basic research has demonstrated that appropriate orthodontic forces can stimulate periodontal tissue remodeling and activate odontoblastic activity, whereas immature roots exhibit greater stress sensitivity in the apical region. Finite element analyses have indicated that stress distribution varies significantly according to root length and morphol-

【收稿日期】2026-03-25；　【修回日期】2026-08-03
【基金项目】四川省科技计划资助（2024YFFK0201）
【作者简介】耿瑞，硕士研究生在读，Email：1518106728@qq.com
【通信作者】刘钧，教授，博士，Email：junliu@scu.edu.cn

ogy, with short roots and teeth with abnormal root morphology being more prone to stress concentration in the apical area. Clinical studies have generally not observed significant arrest of root development under controlled orthodontic forces; however, individual susceptibility and improper force management may still increase the risk of root resorption. For patients with special orthodontic needs, including impacted tooth traction and craniofacial developmental anomalies, early intervention is often clinically necessary, thereby placing higher demands on the determination of optimal treatment timing. Based on current evidence, incomplete root development should not be regarded as an absolute contraindication to orthodontic treatment. For routine cases, root formation of permanent teeth of approximately 2/3 (around Nolla stage 8) may serve as a reference point for initiating orthodontic treatment. For patients with impacted teeth or craniofacial developmental abnormalities who might otherwise miss the optimal window for intervention, treatment should not be delayed solely because of incomplete apical closure. Instead, individualized decisions should be made based on the stage of root development, the position of the impacted tooth, the direction of traction, and the anticipated benefits. If progressive root resorption or pulpal or periodontal abnormalities occur, orthodontic force should be promptly reduced, suspended, or otherwise adjusted. Future studies should focus on stratified clinical research across different stages of root development and integrate imaging techniques with biological markers to explore dynamic monitoring methods for root development, thereby further optimizing force selection and risk assessment strategies. This review systematically summarizes the biological basis, biomechanical characteristics, and clinical evidence regarding orthodontic treatment in teeth with incomplete root development, aiming to provide further theoretical support for treatment timing selection and individualized clinical decision-making.

【Key words】 orthodontic treatment; root development; root resorption; immature tooth root; orthodontic treatment timing; orthodontic traction of impacted teeth; biomechanics; stomatology

J Prev Treat Stomatol Dis, 2026, 34(10): 1033-1044.

错𬌗畸形主要表现为牙齿排列异常及上下颌关系失调，可对患者的颌面部形态、咀嚼与发音功能、颞下颌关节功能以及心理社会状态产生不同程度的影响[1-2]。由于错𬌗畸形多在生长发育阶段逐渐显现，临床上大量患者初诊时正处于替牙列期或恒牙列早期，这一时期既是颅颌面快速生长的重要窗口期，也是家长和患者迫切希望进行干预的阶段[3-4]。

然而，正畸治疗需求的提前与牙根发育尚未完成的现实情况之间，长期存在明显矛盾[5]。在替牙列期及恒牙列早期，部分恒牙牙根的根尖形态、牙根长度及根周组织结构尚未成熟[6]。正畸力是否会干扰牙根的正常发育、增加牙根吸收风险，一直是正畸学领域的核心争议之一[7]。受此影响，早期观点普遍持较保守态度[8]。近年来，随着基础研究和临床经验的不断积累，总体证据显示，在常规正畸力与常规疗程下，多数未成熟牙根仍可继续发育并达到接近正常的长度，但过早干预、过大矫治力或不当的大幅度牙移动可能干扰牙根继续发育并增加牙根吸收风险，因此临床上更强调把握时机与轻力原则[9-11]。然而，对于不同情况的牙齿在牙根发育到何种阶段时适合开始正畸治疗仍不明确：现有研究在研究对象、力学条件、牙根发育评价方法及随访时间等方面差异较大，且缺乏高质量证据对未成熟牙根在正畸力下的生物学反应进行系统阐明。

在缺乏明确官方临床指南的背景下，如何在满足早期治疗需求的同时最大限度降低对牙根发育的潜在不良影响，仍是临床实践中亟待回答的问题。本文拟从多学科、多层级证据出发，对正畸力作用于牙根发育未完成恒牙的影响及相关争议进行系统梳理，总结该领域的研究进展，以期为最佳临床治疗时机的判断及后续研究提供参考。

1　常用恒牙牙根发育分期方法及临床现状

恒牙牙根发育阶段的评估是制定正畸治疗时机和力学方案的重要基础。目前临床及研究中常用的恒牙牙根发育分期方法主要包括Demirjian分期、Moorrees分期及Nolla分期等[12-13]。其中，Nolla分期因操作简便、重复性较好、适用于常规影像学评估而被广泛应用于临床实践及相关研究中。Nolla分期将牙齿发育过程划分为0～10期，其中7～9期主要对应牙根形成与延长阶段，10期为根尖孔完全闭合[14]。

临床上通常以影像学显示的牙根长度与最终根长的比例作为是否开始正畸治疗的判断依

据[15]。当前正畸临床中普遍认为，当恒牙牙根发育达到约2/3（对应Nolla 8期左右）时，可相对安全地开始正畸治疗。然而，该判断更多来源于临床经验的积累，而非基于高质量医学证据的总结[16]。截至目前，国内外尚缺乏针对牙根发育未完成恒牙正畸治疗时机的统一官方指南或明确标准[17-19]。国内仅有部分专家共识指出，在牙根发育未完成时过早施加正畸力，可能干扰牙根正常发育过程并增加牙根吸收的风险，因此建议在牙根基本发育完成后再开始正畸治疗[20]。现有文献多为回顾性研究或病例报告，并且在分期标准、研究对象等方面存在较大差异。当前已有的临床经验及指南在临床实践中具有一定指导意义，但尚不足以为不同临床情境下的治疗决策提供精细化依据。

2　特殊情况下的正畸需求

尽管“恒牙牙根发育至2/3后可开始正畸治疗”的经验性原则在多数常规病例中具有一定适用性，但在部分特殊临床情境下，该原则的局限性逐渐显现。

2.1　阻生牙牵引

对于需要进行阻生牙牵引的患者而言，相关恒牙在牙根尚未发育至2/3时即可能出现明确的正畸干预需求[21]。若过度强调等待牙根发育完成而采取保守策略，可能错失阻生牙萌出的最佳干预时机，进而增加后期治疗的难度，延长治疗周期，甚至影响最终的治疗效果[22-23]。因此，在此类病例中，单纯以牙根发育比例作为是否启动治疗的唯一判断标准，显然难以满足临床实际需求。

2.2　牙根长度的变异

对于因外伤、严重龋病或感染等原因导致牙髓坏死、牙根发育提前终止的恒牙，其牙根已失去继续发育的生物学潜力[24]。这类牙齿往往伴随牙根短小、根尖形态异常等结构特点，在正畸力作用下可能面临更高的牙根吸收风险[25-26]。目前，针对此类牙齿是否适合进行正畸治疗以及如何权衡潜在风险与治疗获益，学界尚未形成一致结论[27-28]。

此外，短根异常是一种牙根发育障碍，其人群患病率为0.6%~10%，表现为牙根短小圆钝、牙齿根冠比小于1.1，除牙根形态异常外，通常无其他临床症状[29]。该病约70%累及上颌切牙，常双侧对称出现，亦可涉及上颌前磨牙、下颌第二前磨牙等。短根异常病因尚不完全明确，但并非由牙根吸收所致，临床需注意鉴别[30]。正畸治疗中，短根异常患者发生牙根吸收的风险更高，应重视对其的诊断和制定更谨慎的治疗计划，对严重病例应视为正畸禁忌证[31-32]。

3　正畸力作用于牙根发育未完成恒牙的生物学基础

正畸力对牙根发育未完成恒牙的影响基本可从两个方面理解：未成熟牙根对正畸力刺激的反应及其在正畸力下发生牙根外吸收的风险，这两个生物学过程联系紧密、相互影响，涉及较多相同或相关的细胞和信号分子[33-34]。牙根形成依赖于赫特威氏上皮根鞘（Hertwig′s epithelial root sheath，HERS）与牙乳头间充质之间精确的上皮-间充质相互作用，当牙乳头最外层的间充质细胞与HERS内层细胞建立接触后，受HERS来源的信号诱导分化为成牙本质细胞。随后，这些成牙本质细胞在分泌根部牙本质的过程中，逐渐向牙乳头中心方向回缩，并在此过程中形成牙根结构[35]。因此，牙根的形成直接依赖于成牙本质细胞在HERS的诱导调控下有序生成根部牙本质；牙根外吸收的发生通常需要保护性结构受损（无细胞层、前期牙骨质、牙周膜完整性等）、局部炎症信号上调、募集并诱导单核细胞分化为破牙本质细胞等过程[36-37]。正畸力的介入会在牙根发育过程中叠加机械与炎症信号，从而干预其生长过程[38-39]。因此，阐明其细胞与分子基础，是理解临床风险边界的前提。

3.1　正畸力对HERS的影响

HERS不仅决定牙根形态，更是维持根尖发育潜能的关键结构，其结构完整性直接关系到未成熟牙根在外界刺激下的发育稳定性[40-41]。牙冠形成后，内外釉上皮层共同组成的HERS向根尖方向增殖延伸，指导牙根的形成。其通过分泌多种信号分子诱导牙乳头周边细胞分化为成牙本质细胞，形成根部牙本质基质[42]。随后HERS逐渐断裂退变，其残余形成Malassez上皮剩余；外层的牙囊细胞接触新生牙本质后分化为成牙骨质细胞，沉积牙骨质并建立牙周附着[43-44]。音猬因子（sonic hedgehog，Shh）、Wnt信号通路（Wnt signaling pathway，Wnt）、骨形态发生蛋白（bone morphogenetic protein，BMP）等信号通路在HERS与间充质之间的双向调控中发挥核心作用[45-46]。除经典发育通路外，外胚层发育不良蛋白A（ectodysplasin A，Eda）信号亦参与牙根形成的精细调控[47]。最新的基因敲除研究表明，Eda缺失可影响HERS的增

殖方向与空间构型，导致牙根形态异常，提示其在出生后牙根持续发育中具有重要作用[48]。

机械力对HERS的直接作用机制尚未完全阐明。但近期Zhao等[49]的动物实验结果显示，正畸力对HERS的影响具有明显的发育阶段依赖性。在HERS结构完整、根尖处于快速发育阶段时，施加短期、轻度正畸力，组织学改变较轻，HERS仍可维持相对连续性，根尖形态能够继续正常塑形。相反，在根形成晚期，当HERS已出现碎片化趋势时，施加相同强度的短期力更易导致根尖缩短及异常根尖形态，该阶段HERS对机械刺激更为敏感，发育潜能已下降。

另有研究发现，合适的正畸力可延迟压力侧HERS在新生牙本质表面的解离过程，从而暂时抑制牙骨质形成，减少成牙骨质细胞分化，进而在一定程度上防止牙根外吸收的发生，该过程主要通过压力侧根尖区域转化生长因子β1（transforming growth factor-β1，TGF-β1）的表达下调来实现[50]。TGF-β1是诱导HERS细胞发生上皮-间充质转化（epithelial-mesenchymal transition，EMT）的关键启动因子，其表达减少导致HERS细胞无法正常启动EMT程序[51-52]。与此同时，作为上皮细胞骨架标志物的角蛋白14（keratin 14，K14）在根尖区域维持连续性表达，证实HERS细胞仍保持紧密的上皮结构状态[53]。

但更应该注意的是，HERS的完整性极易受到机械力的干扰[54]。过大的正畸力可导致这一关键结构受损，使其无法继续行使引导牙根发育的功能，进而在临床上表现为牙根发育的停滞或形态异常[50]。

3.2　正畸力对成牙本质细胞的影响

早期研究提出，适度正畸力可能刺激成牙本质细胞活性，促进牙本质沉积，使未成熟牙根在正畸过程中表现出较强的抗吸收能力[55]。近年来的临床试验研究（Iranian Registry of Clinical Trials：IRCT20170928036471N1）进一步证实，短期轻度机械刺激可促进牙乳头细胞向成牙本质细胞分化，并加快根部牙本质形成，但最终根长仍趋于接近正常发育水平[56]。

在分子机制方面，Sheng等[57]通过三维培养模型及大鼠体内实验证实，Piezo型机械敏感离子通道蛋白1（Piezo type mechanosensitive ion channel component 1，Piezo1）的激活可促进牙乳头细胞分化为成牙本质细胞，并抑制家族序列相似蛋白83成员G（family with sequence similarity 83 member G，FAM83G）的表达，从而解除其对成牙本质分化的抑制作用，促进牙本质基质形成。Piezo1是一种机械门控阳离子通道，可将外界机械刺激转化为细胞内钙离子信号，从而激活下游转录调控网络[58-59]；FAM83G则是一种参与细胞增殖与分化调控的胞质蛋白，被认为是牙乳头细胞分化过程中的负调控因子[60]（ClinicalTrials.gov：NCT05316636）。该研究为解释未成熟牙根在适度正畸力下可能出现“加速性发育”提供了分子依据。然而，这种促进作用具有时间与剂量依赖性，过强或持续时间过长的机械刺激可能通过钙离子超载或激活核因子κB（nuclear factor-κB，NF-κB）炎症通路，转向炎症与牙根吸收等损伤通路[61]。

3.3　牙根吸收的分子学机制

正畸相关的牙根外吸收是牙骨质与牙本质在破牙骨质细胞及破牙本质细胞介导下发生的病理性矿化组织吸收[62]。其发生通常需要保护性结构受损（包括前期牙骨质、牙周膜完整性等）、局部炎症信号——特别是核因子κB受体活化因子配体（receptor activator of nuclear factor- κB ligand，RANKL）/骨保护素（osteoprotegerin，OPG）比值上调及趋化因子增加、血液中的单核细胞被募集并黏附于暴露的矿化组织表面，最终分化为具有吸收功能的破牙骨质/破牙本质细胞，启动根吸收过程[63-64]。临床上牙根吸收通常不可逆，若在早期阶段及时停止加力，可能保留部分发育潜能；但当影像学已出现可见吸收时，往往提示HERS及牙乳头组织已受到损伤，牙根继续发育的可能性显著降低[49,65]。

近期研究发现，Piezo1在牙根吸收中亦发挥重要作用[66]。使用Piezo1激动剂Yoda1可上调C-X-C基序趋化因子配体12（C-X-C motif chemokine ligand 12，CXCL12）及其受体C-X-C基序趋化因子受体4（C-X-C motif chemokine receptor 4，CXCR4）的表达，增强淋巴细胞抗原6复合体位点C（lymphocyte antigen 6 complex locus C，Ly6C）高表达炎性单核细胞的募集，并促进其向M1型巨噬细胞极化，从而加重牙根吸收。通过腺相关病毒沉默Piezo1或使用CXCR4拮抗剂AMD3100阻断该信号轴，均可显著减少炎性细胞浸润并减轻吸收程度。进一步研究表明，白细胞介素（interleukin，IL）6是Piezo1活化的上游调控因子，其缺失可削弱相关炎症通路的激活[67]。

除Piezo1通路外，Tan等[68]发现，过大的机械力可诱导出现SRY盒转录因子9（SRY-box transcription factor 9，Sox9）和聚集蛋白聚糖（aggrecan，Acan）双阳性的间充质过渡态细胞亚群。该细胞群通过成纤维细胞生长因子2-成纤维细胞生长因子受体2-细胞外信号调节激酶（fibroblast growth factor 2 - fibroblast growth factor receptor 2 - extracellular signal-regulated kinase，FGF2 - FGFR2 - ERK）信号轴与表达基质金属蛋白酶14（matrix metalloproteinase 14，MMP14）的巨噬细胞相互作用，促进破骨细胞分化，导致炎性牙根吸收。局部应用负载小干扰RNA（small interfering RNA，siRNA）的明胶甲基丙烯酰化水凝胶（gelatin methacryloyl，GelMA）沉默Sox9表达，可在不影响牙齿移动的前提下显著减轻牙根吸收。

此外，一项临床试验发现，重力下可检测到龈沟液中S100钙结合蛋白A9（S100 calcium-binding protein A9，S100A9）、细胞外钙敏感受体（extracellular calcium-sensing receptor，CaSR）及II型细胞骨架角蛋白1（keratin，type II cytoskeletal 1，K2C1）等蛋白表达变化，提示力相关炎症、钙信号、破骨细胞生成及上皮组织损伤可能共同参与正畸相关炎性牙根吸收[69]（ClinicalTrials. gov：NCT04291443）。NOD样受体家族Pyrin结构域蛋白3（NOD-like receptor family pyrin domain containing 3，NLRP3）炎性小体是一种在促进破骨细胞活化及分化、促进正畸牙移动中发挥重要作用的炎症信号复合体[70]。最新研究发现，其介导的牙周膜细胞焦亡亦参与正畸相关牙根吸收。应用NLRP3抑制剂MCC950可减少破牙本质细胞数量，降低M1型巨噬细胞比例，在维持牙齿移动效率的同时减轻牙根吸收程度[71-72]。

基于上述研究结果，本文将不同大小正畸力作用下牙根发育未完成牙齿的主要生物学机制总结为图1，以直观展示适宜正畸力促进牙根发育及过大正畸力诱导牙根吸收的分子机制。

4　未发育完成/短根恒牙的力学特征

上述细胞与分子机制在正畸机械力作用下被激活，而不同力学分布模式决定了刺激的强度与空间位置，进而影响发育与吸收风险。不同的牙根形态和施力方向会显著影响牙齿受力时的应力分布特征，牙根长度、锥度及根尖形态和力的角度、方向的改变，会重塑牙齿-牙周支持系统之间的应力分布，从而影响牙齿移动模式及潜在风险。因此，从生物力学角度分析未成熟牙根的受力特征具有重要意义[73-74]。有限元分析（finite element analysis，FEA）因能够构建三维牙-骨复合体模型，实现不同牙根形态与不同方向、不同大小力的可控模拟，并精确量化牙体及牙周组织内的应力分布，成为研究正畸生物力学的重要工具[75-76]。

4.1　不同牙根形态造成的应力分布差异

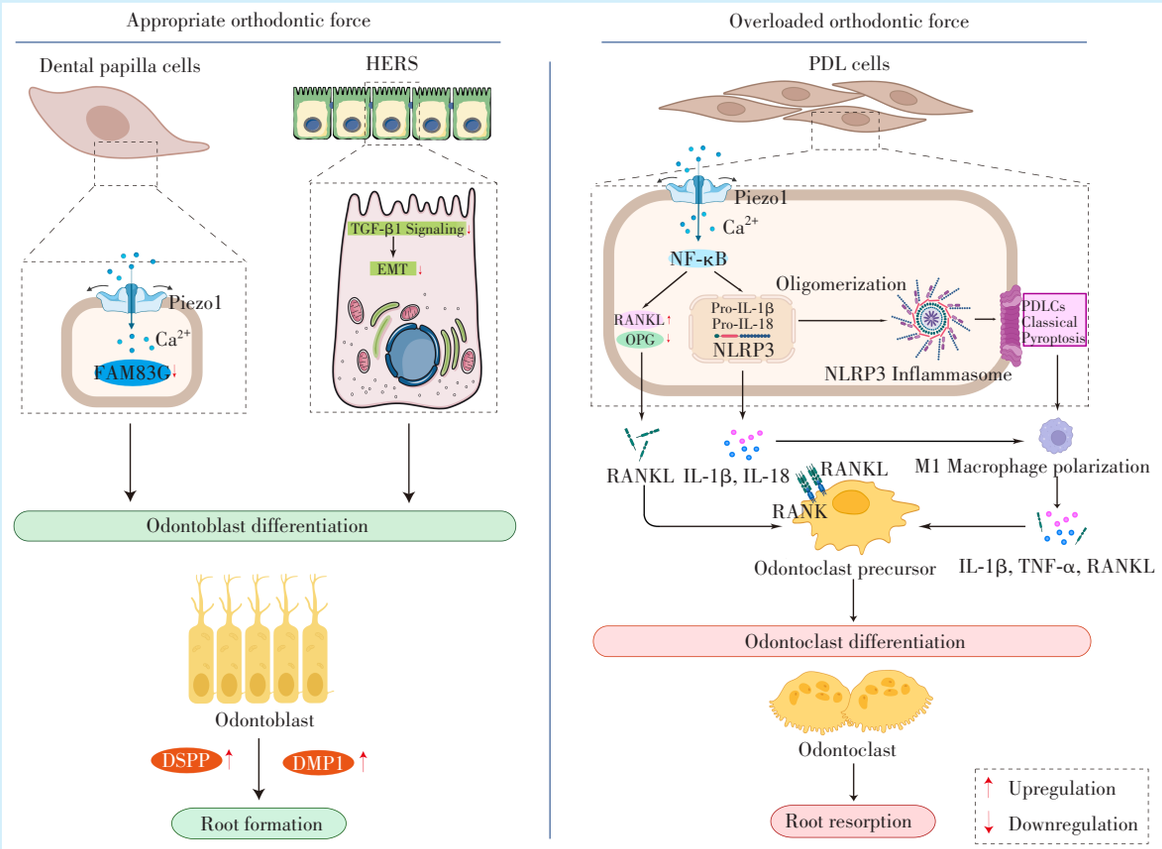
对于不同的牙根形态，Oyama等[77]采用三维有限元方法，构建了包括上颌中切牙、牙髓、牙周膜及牙槽骨在内的有限元模型，设计了五种牙根形态（正常、短根、钝状根尖、弯曲根尖、吸管状根尖）来分析根尖区的应力分布特征。结果显示正常牙根形态模型在根尖区未见明显应力集中；短根模型应力集中位于根中1/3；钝状根尖模型应力水平较低；弯曲根尖和吸管状根尖模型则在根尖区表现出显著的应力集中。

研究表明，牙根形态异常可在正畸力作用下导致根尖区应力集中，增加牙根吸收的风险。对于不同的牙根长度，另一项有限元研究发现，牙根缩短会改变支抗中心位置，使倾斜趋势加剧，并在牙周膜颈部及根尖区形成应力集中，最终导致较短牙根在施力后更易发生倾斜移动[78]。另有研究显示，短根牙齿在受到正畸力作用时产生的牙周膜应力更大，但总体应力值仍处于安全范围内（0.018~0.048 MPa）[79]。

4.2　不同移动方向下的力学特征

除牙根形态外，不同的移动类型也会产生显著不同的应力分布格局[80-81]。Rodrigues等[79]利用三维有限元模型模拟了上颌中切牙在健康状态及根尖外吸收（缩短3 mm）条件下，分别施加0.6 N压入、伸长、倾斜及平移四种正畸力，观察到在不同正畸运动方式中，平移运动产生的von Mises应力值最高，提示其存在更高的牙根吸收风险；Moga等[82]则模拟了压入、伸长、旋转、倾斜和平移5种移动方式，得出的结论是旋转和平移是产生应力最大的移动方式。然而，成熟的临床经验及专家共识普遍认为压低移动与牙根吸收风险关系更为密切，进行压低时需格外警惕[20,83]。

上述差异可能源于评价指标、模型参数及力学条件设定的不同。有限元分析的结论多以von Mises应力等材料力学指标作为评价终点，而牙根吸收的发生不仅涉及牙周膜局部压应力集中，更与个体易感性、血供受限及多种复杂的生物学反



Ca^{2+}: calcium ions; DMP1: dentin matrix protein 1; DSPP: dentin sialophosphoprotein; EMT: epithelial - mesenchymal transition; FAM83G: family with sequence similarity 83 member G; HERS: Hertwig's epithelial root sheath; IL: interleukin; NLRP3: NOD-like receptor family pyrin domain containing 3; NF-κB: nuclear factor-κB; OPG: osteoprotegerin; PDL: periodontal ligament; PDLC: periodontal ligament cell; Piezo1: piezo-type mechanosensitive ion channel component 1; RANK: receptor activator of nuclear factor-κB; RANKL: receptor activator of nuclear factor-κB ligand; TGF-β1: transforming growth factor-β1; TNF-α: tumor necrosis factor-α. The figure was created with Affinity for China, version 3.2.1; Serif (Europe) Ltd., Nottingham, UK

Figure 1　Schematic diagram of the biological mechanisms underlying root development and resorption in permanent teeth with incomplete root development under different orthodontic force magnitudes

图1　牙根发育未完成恒牙在不同正畸力作用下牙根发育及根吸收的生物学机制示意图

应相关[84-85]。此外,压低移动虽未必产生最高的整体等效应力,但其应力主要集中于根尖区域,在牙根发育未完成情况下可能导致更高的生物学敏感性[86-87]。因此,不同研究结果之间并非直接矛盾,而是反映了力学评价指标与临床生物学风险之间的差异。

此外,对于临床中常见的倒置阻生中切牙,往往需要更精细地调整施力角度,以获得更理想的牙根发育效果[88]。Yang等[89]的研究结论建议,当倒置角度 > 20°时,牵引力方向宜控制在相对于牙冠长轴100°~110°范围内;角度较小时可适当调整至110°~120°。

5　牙根发育未完成恒牙正畸治疗的临床证据

5.1　常规正畸治疗

在常规临床情境下,经验性共识认为当恒牙牙根发育达约2/3时即可开始正畸治疗,对应Nolla分期约8期。该阶段牙根已形成一定长度,根尖尚未闭合,具有较好的继续发育潜力,同时牙周支持组织对正畸力的适应能力亦相对较强,已有较多临床研究支持在此阶段启动治疗的安全性。

Wan等[90]进行的一项回顾性研究显示,对牙根发育未完成恒牙施加常规正畸力,未观察到牙根发育受阻等不良结局,治疗后牙根长度可随生长发育继续增加,而牙根发育完成组则出现根长或根体积下降。Dodeja等[91]利用锥形束计算机体

层摄影（cone-beam computed tomography，CBCT）分别对接受了快速扩弓和固定矫治的牙根发育未完成恒牙进行了三维评估，结果显示未成熟牙根在治疗后均达到了正常根长。另有随机对照研究对牙根发育未完成的前磨牙施加正畸力后进行随访，结果显示受力牙齿最终均可达到理想根长，部分病例在受力阶段甚至表现出牙根发育速率加快的趋势[56]。上述研究提示，在适当控制力值的前提下，正畸力并不必然干扰牙根的正常发育进程。

5.2　阻生牙牵引

除常规正畸治疗外，阻生牙牵引同样是临床上常见的情境，对阻生牙进行早期干预不仅有利于改善萌出路径、促进牙根发育，也可减少对邻牙的不利影响[92-93]（ClinicalTrials.gov：NCT05629312[93]）。郭苏莹等[94]对40例牙长轴与对侧同名牙成角≤90°的单侧埋伏上切牙进行正畸牵引治疗后证实，在对侧同名牙牙根发育未完成时对埋伏上切牙进行牵引，更有利于其牙根生长和牙槽骨的改建。

而对于倒置阻生的上颌中切牙，Wang等[21]的研究提示，相较于在对侧同名非阻生牙牙根基本发育完成时开始牵引的患者，在对侧牙齿处于Nolla 7~8期时进行阻生前牙牵引，更利于获得理想的根长与根生长量。此外，Maruya等[95]报告了3例在牙根形成1/2、1/3甚至仅1/4时即开始牵引治疗的病例，最终均可在保持牙髓活力的情况下完成排齐与功能重建。

关于上颌尖牙阻生，多数证据同样显示牵引更适合在生长发育期、牙根未完全形成阶段实施[96]。此时牙周组织改建能力较强，牙根仍具发育潜力，理论上更有利于生理性萌出[97-98]。同时，应充分认识到相关并发症风险，包括邻牙骨丧失、根吸收及牙龈退缩等[99-101]。临床实施过程中需合理设计牵引方向与力值，密切影像学监测，并加强医患沟通及多学科协作，以降低不良事件发生率[102]。

5.3　特殊疾病患者

颅骨锁骨发育不全(cleidocranial dysplasia, CCD)是一种由Runt相关转录因子2（runt-related transcription factor 2，RUNX2）基因突变导致的常染色体显性遗传性骨骼疾病，主要表现为成骨细胞分化障碍，临床上以颅缝闭合延迟、锁骨发育不良及多发性牙异常构成的典型“三联征”[103-104]。在此类患者中，第二前磨牙常发生错位或萌出异常，单纯正畸辅助萌出成功率通常不理想[105]。Chen等[106]开展的回顾性研究分析了影响牵引成功率的因素，结果显示不同Nolla分期之间成功率差异显著，其中Nolla 9~10期失败率明显高于Nolla 7~8期。该结果与Zhao等[49]的动物实验结果一致，即过晚启动牵引可能反而不利于萌出成功，在牙根发育尚未接近完成时介入可能更具优势。

6　总结与展望

综上所述，尽管现行教材与部分指南在表述上仍趋于谨慎，但近年来积累的大量临床证据显示：在轻力的前提下对牙根发育未完成恒牙启动正畸治疗总体是安全可行的。本文将不同情况下对牙根发育未完成牙齿进行正畸治疗的决策建议整理为图2。

本领域虽已取得诸多进展，但仍存在明显局限。首先，动物模型中牙根发育节律与人类存在差异，所施加的力值及时间尺度难以完全等比换算；其次，人类研究多为回顾性或小样本观察性研究，缺乏高质量、长期随访的前瞻性数据。由于伦理限制，难以开展严格随机对照试验，但仍可通过多中心前瞻性队列研究、标准化影像评估、数字化三维测量技术等方式，提高证据质量。此外，目前关于牙根发育的评价多集中于根长变化，对根尖形态、牙本质厚度、血供状态及功能恢复的综合评估仍较不足；不同发育阶段力值推荐尚缺乏量化阈值；个体差异（遗传背景、炎症状态、系统性疾病）对风险的调节作用亦未被充分纳入分析。

未来研究可从以下几个方向深入推进：①建立更精细的发育分期与风险分层模型，将影像学、分子标志物及力学参数整合；②借助三维影像与人工智能技术，实现牙根体积与形态的动态监测；③开展多中心长期随访研究，评估早期干预对牙根远期稳定性与功能的影响；④继续探索靶向调控炎症反应或细胞焦亡相关的辅助治疗策略，以降低正畸相关牙根吸收风险。

综上所述，恒牙牙根发育未完成阶段启动正畸治疗并非绝对禁忌，而是一项需要在生物学活性、力学条件与临床需求之间寻求平衡的治疗决策。现有研究总体提示，与牙根发育完成的牙齿相比，牙根发育未完成的恒牙在正畸治疗过程中发生明显牙根吸收的倾向较低，可能具有相对更强的抵御牙根吸收的能力。随着基础研究与临床证据的不断积累，未来有望实现更加精准、安全、个体化的治疗策略，为儿童青少年患者提供兼顾

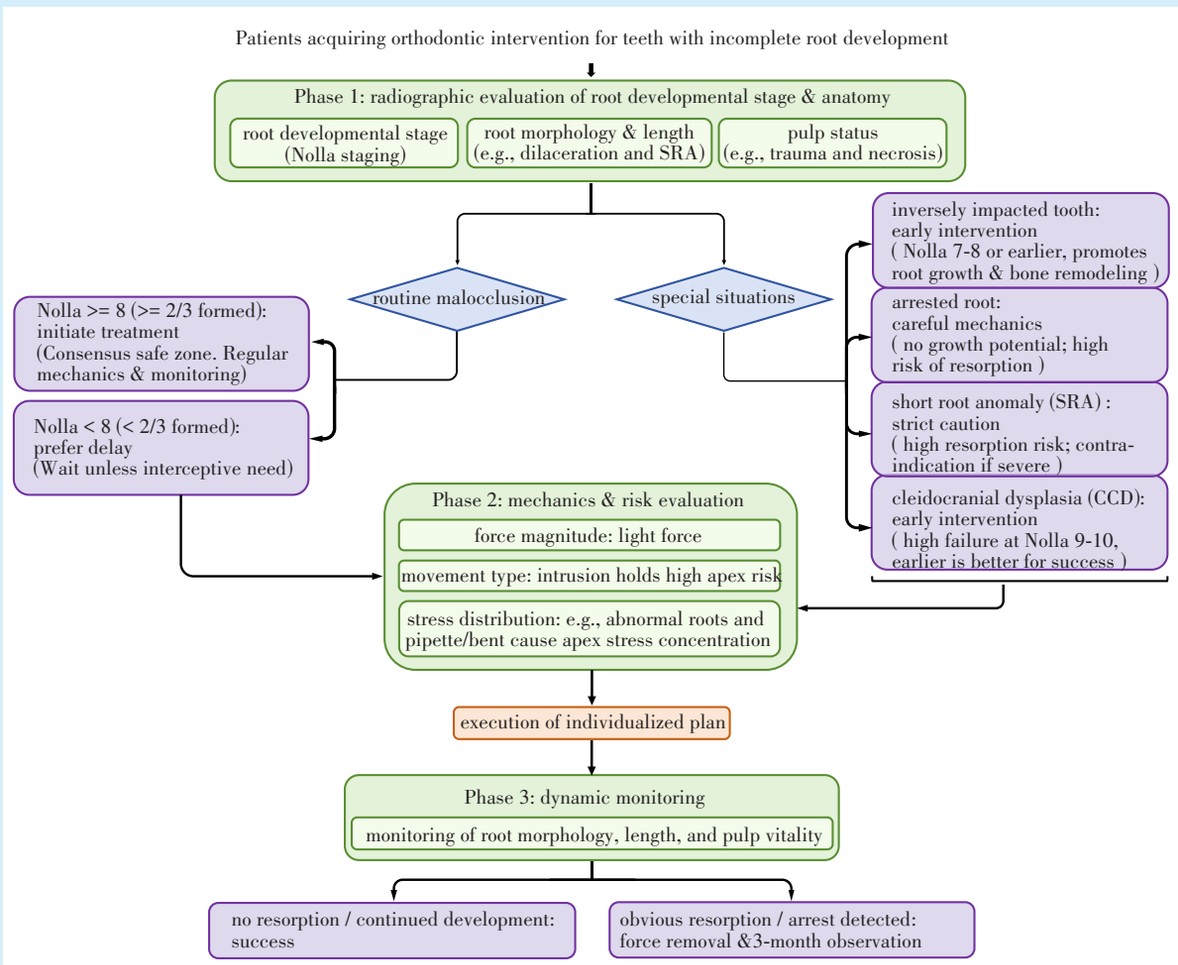


Figure 2　Clinical decision-making pathway for the orthodontic treatment of permanent teeth with incomplete root development

图2　牙根发育未完成恒牙正畸治疗的临床决策路径

发育潜能与长期稳定性的正畸治疗方案。

【Author contributions】 Geng R conducted the literature search and wrote the article. Liu J conceptualized and reviewed the article. All authors read and approved the final manuscript as submitted.

【Ethics statement】 The authors have nothing to report.

【Funding】 This study was supported by the grant from Sichuan Science and Technology Program (No. 2024YFFK0201). The funder had no role in the study design, data collection and analysis, decision to publish, or preparation of the manuscript.

【Competing interests】 All authors have completed the ICMJE uniform disclosure form at www.icmje.org/disclosure-of-interest/ and declare: support from the Sichuan Science and Technology Program (No. 2024YFFK0201) for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the previous three years; no other relationships or activities that could appear to have influenced the submitted work.

【Data availability statement】 Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

【Generative AI statement】 Figure 1 in this manuscript was created with Affinity for China 3.2.1, version 3.2.1; Serif (Europe) Ltd., Nottingham, UK. The authors reviewed and edited the output and take full responsibility for the figures. No other generative AI technology was used in the creation of this manuscript.

参考文献

[1] Atasever İşler AA, Hezenci Y, Bulut M. Prevalence of orthodontic malocclusion in children aged 10-12: an epidemiological study[J]. BMC Oral Health, 2025, 25(1): 249. doi: 10.1186/s12903-025-05650-x.

[2] Zhou C, Duan P, He H, et al. Expert consensus on pediatric orthodontic therapies of malocclusions in children[J]. Int J Oral Sci, 2024, 16(1): 32. doi: 10.1038/s41368-024-00299-8.

[3] Dinu S, Igna A, Petrescu EL, et al. Timing of orthodontic intervention for pediatric class II malocclusion: a systematic review on early *vs*. late treatment outcomes[J]. Children, 2025, 12(11): 1533. doi: 10.3390/children12111533.

[4] Najjar H, Almansour T, Aljaiban Y, et al. Growth-related changes in the upper and lower jaw: clinical implications for treatment timing[J]. Int J Community Med Public Health, 2025, 12(11): 5319-

5324. doi: 10.18203/2394-6040.ijcmph20253721.

[5] Wu M, Li Y, Wang Q, et al. Evaluation of the effects on lower central incisors intrusion and lower first molar extrusion at different mixed dentition stages using clear aligners for deep overbite treatment: a retrospective study[J]. Clin Oral Investig, 2025, 29(11): 527. doi: 10.1007/s00784-025-06621-w.

[6] Kumari S, Sharma A, Singh A, et al. Use of endodontic procedures in young permanent teeth[J]. J Pharm Bioallied Sci, 2024, 16 (Suppl 1): S797-S799. doi: 10.4103/jpbs.jpbs_1018_23.

[7] Roscoe MG, Meira JB, Cattaneo PM. Association of orthodontic force system and root resorption: a systematic review[J]. Am J Orthod Dentofacial Orthop, 2015, 147(5): 610-626. doi: 10.1016/j.ajodo.2014.12.026.

[8] Weltman B, Vig KW, Fields HW, et al. Root resorption associated with orthodontic tooth movement: a systematic review[J]. Am J Orthod Dentofacial Orthop, 2010, 137(4): 462-476. doi: 10.1016/j.ajodo.2009.06.021.

[9] Wu J, Dong Z, Lin P, et al. Beyond virality: evaluating evidence-based content delivery in social media-based pediatric orthodontic education[J]. BMC Oral Health, 2025, 25(1): 1603. doi: 10.1186/s12903-025-06998-w.

[10] Madiraju GS, Almugla YM, Mohan R, et al. An epidemiological study on early orthodontic treatment need among eastern Saudi Arabian children in the mixed dentition stage[J]. Sci Rep, 2024, 14 (1): 4084. doi: 10.1038/s41598-024-54381-6.

[11] Türker G, Öztürk T, Çoban G, et al. Effects of alternate rapid maxillary expansions and constrictions procedures on root development compared to rapid maxillary expansion during orthodontic face mask treatment[J]. BMC Oral Health, 2025, 25(1): 860. doi: 10.1186/s12903-025-06270-1.

[12] Haghi Ashtiani G, Davies JA, Liversidge HM. Symmetry in the developmental stages of permanent human teeth: a comparative study between maxilla and mandible[J]. Eur Arch Paediatr Dent, 2025, 26(3): 483-491. doi: 10.1007/s40368-024-00996-2.

[13] Al-Juhani A, Binshalhoub A, Showail S, et al. Comparative analysis of dental age estimation: a systematic review and meta-analysis assessing gender-specific accuracy of the demirjian and nolla methods across different age groups[J]. Cureus, 2024, 16(12): e75031. doi: 10.7759/cureus.75031.

[14] Nolla CM. The development of the permanent teeth[J]. J Dent Child, 1960, 27: 254-266.

[15] Declerck E, EzEldeen M, Wyatt J, et al. Application of orthodontic force in autotransplanted teeth: a longitudinal study[J]. Clin Oral Investig, 2025, 29(1): 69. doi: 10.1007/s00784-024-06106-2.

[16] Seo W, Park S, Lee E, et al. Panoramic assessment of root development in immature maxillary incisors after treatment with prefabricated functional appliances[J]. Children, 2025, 12(10): 1416. doi: 10.3390/children12101416.

[17] 房兵, 金作林, 白玉兴, 等. 儿童和青少年早期错畸形诊治策略的专家共识[J]. 上海口腔医学, 2021, 30(5): 449-455. doi: 10.19439/j.sjos.2021.05.001.
Fang B, Jin ZL, Bai YX, et al. Experts consensus on diagnostic and therapeutic strategies for malocclusions at early developing stage[J]. Shanghai J Stomatol, 2021, 30(5): 449-455. doi: 10.19439/j.sjos.2021.05.001.

[18] Hamidaddin MA. Optimal treatment timing in orthodontics: a scoping review[J]. Eur J Dent, 2024, 18(1): 86-96. doi: 10.1055/s-0043-1768974.

[19] Kirschneck C, Proff P, Lux C. Ideal treatment timing of orthodontic anomalies-a German clinical S3 practice guideline[J]. J Orofac Orthop, 2022, 83(4): 225-232. doi: 10.1007/s00056-022-00409-3.

[20] 李煌, 武秀萍, 黄兰, 等. 正畸根尖区牙根外吸收的风险因素及其临床处理建议的专家共识[J]. 华西口腔医学杂志, 2022, 40 (6): 629-637. doi: 10.7518/hxkq.2022.06.002.
Li H, Wu XP, Huang L, et al. External apical root resorption in orthodontic tooth movement: the risk factors and clinical suggestions from experts' consensus[J]. West Chin J Stomatol, 2022, 40 (6): 629-637. doi: 10.7518/hxkq.2022.06.002.

[21] Wang Y, Chen X, Zhou D, et al. Appropriate timing of treatment contributes to better root development of impacted anterior teeth in children[J]. Am J Orthod Dentofacial Orthop, 2024, 165(1): 18-26. doi: 10.1016/j.ajodo.2023.06.022.

[22] Yanoshita M, Hirose N, Onishi A, et al. Orthodontic treatment for a patient with root resorption of all four maxillary incisors due to bilaterally impacted canines[J]. Case Rep Dent, 2022, 2022: 5628030. doi: 10.1155/2022/5628030.

[23] Yang L, Dong Z, Cheng Y, et al. Early orthodontic traction does not compromise root development in inversely impacted maxillary central incisors: a three-year cone-beam computed tomography evaluation[J]. Clin Oral Investig, 2026, 30(6): 271. doi: 10.1007/s00784-026-06962-0.

[24] Zhou Y, Bi M, Yang H, et al. Epidemiological and imaging characteristics of impacted maxillary incisors and effect on root development[J]. Sci Rep, 2025, 15(1): 13049. doi: 10.1038/s41598-025-96920-9.

[25] Park YS, Andrew Choi EH, Jamal H Alwaznah F, et al. Is apex position a determining factor in the orthodontic traction duration of unilateral impacted maxillary canines? A cone-beam computed tomography-based 3-dimensional study[J]. Am J Orthod Dentofacial Orthop, 2026, 169(2): 168-176. doi: 10.1016/j.ajodo.2025.09.015.

[26] Dawood HM, Kroeger A, Chavda V, et al. Under pressure-mechanisms and risk factors for orthodontically induced inflammatory root resorption: a systematic review[J]. Eur J Orthod, 2023, 45 (5): 612-626. doi: 10.1093/ejo/cjad011.

[27] Tarkan H, Gürbüz OÖ. The impact of orthodontic traction on outcomes in impacted canine management: a quantitative analysis[J]. Angle Orthod, 2026, 96(2): 181-191. doi: 10.2319/032125-227.1.

[28] Liu Z, Ouyang Y, Lou Y, et al. Orthodontically induced root resorption in endodontically treated and vital teeth: a cone beam computer tomographic study[J]. Prog Orthod, 2025, 26(1): 8. doi: 10.1186/s40510-025-00553-7.

[29] Lind V. Short root anomaly[J]. Scand J Dent Res, 1972, 80(2): 85-93. doi: 10.1111/j.1600-0722.1972.tb00268.x.

[30] Yu M, Jiang Z, Wang Y, et al. Molecular mechanisms for short root anomaly[J]. Oral Dis, 2021, 27(2): 142-150. doi: 10.1111/

odi.13266.

[31] Marechal C, Thierens LAM, De Pauw GAM. Combined orthodontic-orthognathic management of a patient with generalized short root anomaly and anterior open bite[J]. Am J Orthod Dentofacial Orthop, 2023, 164(1): 131-142. doi: 10.1016/j.ajodo.2023.05.004.

[32] Sameshima GT, Iglesias-Linares A. Orthodontic root resorption[J]. J World Fed Orthod, 2021, 10(4): 135-143. doi: 10.1016/j.ejwf.2021.09.003.

[33] Nakai Y, Praneetpong N, Ono W, et al. Mechanisms of osteoclastogenesis in orthodontic tooth movement and orthodontically induced tooth root resorption[J]. J Bone Metab, 2023, 30(4): 297-310. doi: 10.11005/jbm.2023.30.4.297.

[34] Zheng X, Huang H, Zhou Z, et al. Axin1 regulates tooth root development by inhibiting AKT1-mTORC1 activation and Shh translation in Hertwig's epithelial root sheath[J]. Development, 2024, 151(21): dev202899. doi: 10.1242/dev.202899.

[35] Rao P, Jing J, Fan Y, et al. Spatiotemporal cellular dynamics and molecular regulation of tooth root ontogeny[J]. Int J Oral Sci, 2023, 15(1): 50. doi: 10.1038/s41368-023-00258-9.

[36] Patel S, Saberi N, Pimental T, et al. Present status and future directions: root resorption[J]. Int Endod J, 2022, 55(Suppl 4): 892-921. doi: 10.1111/iej.13715.

[37] Wang J, Yu M, Liu Y, et al. Three-dimensional visualization of Hertwig's epithelial root sheath during tooth root development with the miniTESOS tissue clearing method[J]. Am J Transl Res, 2024, 16(9): 4403-4421. doi: 10.62347/PEIQ4730.

[38] Ying Q, Jiang Y, Sun C, et al. AGEs impair osteogenesis in orthodontic force-induced periodontal ligament stem cells through the KDM6B/Wnt self-reinforcing loop[J]. Stem Cell Res Ther, 2024, 15(1): 431. doi: 10.1186/s13287-024-04058-8.

[39] Chen L, Yu H, Li Z, et al. Force-induced Caspase-1-dependent pyroptosis regulates orthodontic tooth movement[J]. Int J Oral Sci, 2024, 16(1): 3. doi: 10.1038/s41368-023-00268-7.

[40] Bi F, Chen T, Guo J, et al. A single-cell transcriptomic atlas of epithelial cell heterogeneity during the crown-to-root transition in the mouse molar[J]. Int J Mol Sci, 2026, 27(3): 1162. doi: 10.3390/ijms27031162.

[41] Ruangchan C, Ngamphiw C, Krasaesin A, et al. Genetic variants in KCTD1 are associated with isolated dental anomalies[J]. Int J Mol Sci, 2024, 25(10): 5179. doi: 10.3390/ijms25105179.

[42] Li J, Parada C, Chai Y. Cellular and molecular mechanisms of tooth root development[J]. Development, 2017, 144(3): 374-384. doi: 10.1242/dev.137216.

[43] Liu C, Yuan H, Huang J, et al. Bmp9 modulates cell proliferation and intercellular junctions in HERS during tooth root development [J]. Genes Dis, 2025, 13(1): 101777. doi: 10.1016/j.gendis.2025.101777.

[44] Jeong JK, Kim TH, Choi H, et al. Impaired breakdown of Herwig's epithelial root sheath disturbs tooth root development[J]. Dev Dyn, 2024, 253(4): 423-434. doi: 10.1002/dvdy.667.

[45] Sun K, Yu M, Wang J, et al. A Wnt10a-Notch signaling axis controls Hertwig's epithelial root sheath cell behaviors during root furcation patterning[J]. Int J Oral Sci, 2024, 16(1): 25. doi: 10.1038/s41368-024-00288-x.

[46] Jeong JK, Choi H, Kim TH, et al. Notum regulates tooth root morphogenesis by modulating Wnt signaling[J]. J Dent Res, 2026, 105(7): 938-948. doi: 10.1177/00220345251383829.

[47] Yang R, Mei Y, Jiang Y, et al. Ectodysplasin a (EDA) signaling: from skin appendage to multiple diseases[J]. Int J Mol Sci, 2022, 23(16): 8911. doi: 10.3390/ijms23168911.

[48] Fons Romero JM, Star H, Lav R, et al. The impact of the eda pathway on tooth root development[J]. J Dent Res, 2017, 96(11): 1290-1297. doi: 10.1177/0022034517725692.

[49] Zhao L, Matsumoto Y, Iseki S, et al. Effects of short-term orthodontic force application on the root at different developmental stages in rat maxillary molars[J]. Am J Orthod Dentofacial Orthop, 2023, 163(4): 531-539.e2. doi: 10.1016/j.ajodo.2022.04.016.

[50] Lou Y, Matsumoto Y, Iseki S, et al. Difference in apical resorption activity during rat molar root formation in response to mechanical force[J]. Eur J Orthod, 2023, 45(2): 208-216. doi: 10.1093/ejo/cjac071.

[51] Itaya S, Oka K, Ogata K, et al. Hertwig's epithelial root sheath cells contribute to formation of periodontal ligament through epithelial-mesenchymal transition by TGF-β[J]. Biomed Res, 2017, 38(1): 61-69. doi: 10.2220/biomedres.38.61.

[52] Chen J, Chen G, Yan Z, et al. TGF-β1 and FGF2 stimulate the epithelial-mesenchymal transition of HERS cells through a MEK-dependent mechanism[J]. J Cell Physiol, 2014, 229(11): 1647-1659. doi: 10.1002/jcp.24610.

[53] Nakasone N, Yoshie H. Occlusion regulates tooth-root elongation during root development in rat molars[J]. Eur J Oral Sci, 2011, 119(6): 418-426. doi: 10.1111/j.1600-0722.2011.00856.x.

[54] Yao EH, Du JH, Jiang XQ. Tooth root development and homeostasis during eruptive and post-eruptive movement[J]. Chin J Dent Res, 2024, 27(4): 273-289. doi: 10.3290/j.cjdr.b5860254.

[55] Kong X, Cao M, Ye R, et al. Orthodontic force accelerates dentine mineralization during tooth development in juvenile rats[J]. Tohoku J Exp Med, 2010, 221(4): 265-270. doi: 10.1620/tjem.221.265.

[56] Dalaie K, Badiee M, Behnaz M, et al. Effect of orthodontic forces on root length of immature mandibular second premolars: a split-mouth randomized clinical trial[J]. Dental Press J Orthod, 2021, 26(5): e2119355. doi: 10.1590/2177-6709.26.5.e2119355.oar.

[57] Sheng X, Li J, Ma H, et al. Piezo1 regulates odontogenesis *via* a FAM83G-mediated mechanism in dental papilla cells *in vitro* and *in vivo*[J]. Biomolecules, 2025, 15(3): 316. doi: 10.3390/biom15030316.

[58] Lin Y, Ren J, McGrath C. Mechanosensitive Piezo1 and Piezo2 ion channels in craniofacial development and dentistry: recent advances and prospects[J]. Front Physiol, 2022, 13: 1039714. doi: 10.3389/fphys.2022.1039714.

[59] Coste B, Mathur J, Schmidt M, et al. Piezo1 and Piezo2 are essential components of distinct mechanically activated cation channels [J]. Science, 2010, 330(6000): 55-60. doi: 10.1126/science.1193270.

[60] Teti L, Pérez-Idarraga A, Salcedo-Miranda M, et al. Randomised clinical trial on the effect of intermittent vibrational force application during orthodontic treatment with aligners on root resorption [J]. Orthod Craniofac Res, 2025, 28(6): 1015-1026. doi: 10.1111/ocr.70028.

[61] Deng J, Zhang YN, Bai RS, et al. Mechanosensor YAP mediates bone remodeling *via* NF-κB p65 induced osteoclastogenesis during orthodontic tooth movement[J]. Prog Orthod, 2025, 26(1): 2. doi: 10.1186/s40510-024-00548-w.

[62] Rostami G, Hadagalu Revana Siddappa R, Kishen A. Signaling pathways in root resorption: linking inflammation, odontoclastogenesis, and tissue remodeling[J]. J Endod, 2026, 52(1): 47-61. doi: 10.1016/j.joen.2025.08.018.

[63] Zhou J, Guo L, Yang Y, et al. Mechanical force regulates root resorption in rats through RANKL and OPG[J]. BMC Oral Health, 2022, 22(1): 290. doi: 10.1186/s12903-022-02327-7.

[64] Jia L, Zhang L, Wang J, et al. Chronic intermittent hypobaric hypoxia attenuates root resorption following delayed tooth replantation by upregulating OPG/RANKL signaling pathway[J]. BMC Oral Health, 2026, 26(1): 737. doi: 10.1186/s12903-026-08135-7.

[65] Zheng Q, Ma L, Wu Y, et al. Automatic 3-dimensional quantification of orthodontically induced root resorption in cone-beam computed tomography images based on deep learning[J]. Am J Orthod Dentofacial Orthop, 2025, 167(2): 188-201. doi: 10.1016/j.ajodo.2024.09.009.

[66] Dong X, Zhu Y, Li B, et al. Tooth movement modulation and root protection *via* Piezo1-mediated production of superoxide dismutase 3[J]. J Periodontal Res, 2026, 61(5): 572-585. doi: 10.1111/jre.70056.

[67] Zhang ZH, Zhu R, Liu Y, et al. IL6-dependent PIEZO1 activation promotes M1-mediated orthodontic root resorption *via* CXCL12/CXCR4[J]. J Dent Res, 2025, 104(7): 763-773. doi: 10.1177/00220345251316472.

[68] Tan M, He M, Zong M, et al. Identification of a force-induced $Sox9^{+}Acan^{+}$ transitional subpopulation linked to FGF2-FGFR2-ERK signaling in orthodontic bone remodeling[J]. Adv Sci, 2026, 13(18): e19330. doi: 10.1002/advs.202519330.

[69] Mohammed-Salih HS, Al-lami HA, Saloom HF, et al. Detection of orthodontically induced inflammatory root resorption-associated biomarkers from the gingival crevicular fluid by proteomics analysis: a randomized-controlled clinical trial[J]. 3 Biotech, 2023, 13(5): 157. doi: 10.1007/s13205-023-03572-5.

[70] 王佳佳, 张疆戬, 曾福磊. NLRP3炎症小体调节正畸牙移动过程中组织改建的研究进展[J]. 口腔疾病防治, 2025, 33(2): 169-176. doi: 10.12016/j.issn.2096-1456.202440017.
Wang JJ, Zhang JT, Zeng FL. Research progress on NLRP3 inflammasome-mediated regulation of tissue remodeling during orthodontic tooth movement[J]. J Prev Treat Stomatol Dis, 2025, 33(2): 169-176. doi: 10.12016/j.issn.2096-1456.202440017.

[71] Li X, Men X, Ji L, et al. NLRP3-mediated periodontal ligament cell pyroptosis promotes root resorption[J]. J Clin Periodontol, 2024, 51(4): 474-486. doi: 10.1111/jcpe.13914.

[72] Li X, Liu X, Zhou J, et al. Human dental follicle stem cell-derived exosomes reduce root resorption by inhibiting periodontal ligament cell pyroptosis [J]. Stem Cell Res Ther, 2025, 16(1): 79. doi: 10.1186/s13287-025-04216-6.

[73] Wood-Bailey AP, Smith C, Fitton LC, et al. Biomechanical impact of tooth root morphology to inform dental implant design[J]. Arch Oral Biol, 2026, 181: 106455. doi: 10.1016/j.archoralbio.2025.106455.

[74] Moga RA, Olteanu CD, Delean AG. Orthodontically induced external root resorption: a finite element analysis[J]. J Clin Med, 2026, 15(7): 2503. doi: 10.3390/jcm15072503.

[75] Cattaneo PM, Cornelis MA. Orthodontic tooth movement studied by finite element analysis: an update. what can we learn from these simulations [J]. Curr Osteoporos Rep, 2021, 19(2): 175-181. doi: 10.1007/s11914-021-00664-0.

[76] Wang S, Peng X, Tian Z, et al. Canine distal movement impact on teeth and aligner deformation in clear aligner treatment: a 3D finite element study[J]. J World Fed Orthod, 2026, 15(3): 233-240. doi: 10.1016/j.ejwf.2025.12.004.

[77] Oyama K, Motoyoshi M, Hirabayashi M, et al. Effects of root morphology on stress distribution at the root apex[J]. Eur J Orthod, 2007, 29(2): 113-117. doi: 10.1093/ejo/cjl043.

[78] Dong Y, Wang Y, Yang Z, et al. Biomechanical effects of root/cortical bone relation on tooth movement during premolar-extraction space closure with clear aligners: a finite element study[J]. Front Bioeng Biotechnol, 2025, 13: 1717813. doi: 10.3389/fbioe.2025.1717813.

[79] Rodrigues Fonseca Tavares A, Aurelio de Carvalho M, Cardoso Lazari-Carvalho P, et al. Teeth with external apical root resorption under orthodontic movement: an in silico analysis on stress and displacement[J]. J Orofac Orthop, 2025. doi: 10.1007/s00056-025-00620-y.

[80] Moradinejad M, Mazhari M, Mazhary M, et al. Effects of fixed orthodontic lingual retainers on PDL stress, root resorption risk, and tooth displacement[J]. Sci Rep, 2025, 15(1): 20559. doi: 10.1038/s41598-025-06004-x.

[81] Knop L, Gandini LG Jr, Shintcovsk RL, et al. Scientific use of the finite element method in orthodontics[J]. Dental Press J Orthod, 2015, 20(2): 119-125. doi: 10.1590/2176-9451.20.2.119-125.sar.

[82] Moga RA, Olteanu CD, Delean AG. Trabecular bone component assessment under orthodontic loads and movements during periodontal breakdown - a finite elements analysis[J]. Dent J, 2024, 12(6): 190. doi: 10.3390/dj12060190.

[83] Antonarakis GS, Zekeridou A, Kiliaridis S, et al. Periodontal considerations during orthodontic intrusion and extrusion in healthy and reduced periodontium[J]. Periodontol 2000, 2024. doi: 10.1111/prd.12578.

[84] Roscoe MG, Cattaneo PM, Dalstra M, et al. Orthodontically induced root resorption: a critical analysis of finite element studies' input and output[J]. Am J Orthod Dentofacial Orthop, 2021, 159(6): 779-789. doi: 10.1016/j.ajodo.2020.02.023.

[85] Demir A, Akgün Kantar A, Dindaroğlu F. Do different orthodontic archwires alter the biomechanical effects of open coil springs on dentoalveolar structures? A finite element analysis[J]. Am J Or-

thod Dentofacial Orthop, 2026, 169(6): 786-797. doi: 10.1016/j.ajodo.2026.01.018.

[86] Rudolph DJ, Willes PMG, Sameshima GT. A finite element model of apical force distribution from orthodontic tooth movement[J]. Angle Orthod, 2001, 71(2): 127 - 131. doi: 10.1043/0003-3219(2001)071<0127:AFEMOA>2.0.CO;2.

[87] Zheng W, Lu X, Chen G, et al. The osteoclastic activity in apical distal region of molar mesial roots affects orthodontic tooth movement and root resorption in rats[J]. Int J Oral Sci, 2024, 16(1): 19. doi: 10.1038/s41368-024-00284-1.

[88] Chen L, Mo S. Cone-beam CT evaluation of orthodontic treatment outcomes for multiple impacted maxillary anterior teeth[J]. BMC Oral Health, 2025, 26(1): 58. doi: 10.1186/s12903-025-07317-z.

[89] Yang Q, Yang L, Li N, et al. Effect of force direction and impaction angulation during dilaceration impacted central incisor traction: a finite element analysis[J]. BMC Oral Health, 2024, 24(1): 823. doi: 10.1186/s12903-024-04601-2.

[90] Wan J, Zhou S, Wang J, et al. Three-dimensional analysis of root changes after orthodontic treatment for patients at different stages of root development[J]. Am J Orthod Dentofacial Orthop, 2023, 163(1): 60-67. doi: 10.1016/j.ajodo.2021.08.025.

[91] Dodeja T, Alsulaiman AA, Will LA, et al. Orthodontic forces interrupt root formation in immature teeth: myth or fact a pilot study [J]. Turk J Orthod, 2025, 38(1): 12-19. doi: 10.4274/TurkJOrthod.2025.2024.142.

[92] Xu S, Zheng B, Li X, et al. Untreated, impacted canine leading to root resorption of the central incisor: a case report of orthodontic treatment with canine substitution[J]. Cureus, 2026, 18(3): e106124. doi: 10.7759/cureus.106124.

[93] Willems G, Butaye C, Raes M, et al. Early prevention of maxillary canine impaction: a randomized clinical trial [J]. Eur J Orthod, 2023, 45(4): 359-369. doi: 10.1093/ejo/cjad014.

[94] 郭苏莹, 陆史俊, 丁元凤. 埋伏上切牙正畸牵引的临床分析[J]. 口腔疾病防治, 2024, 32(4): 273-279. doi: 10.12016/j.issn.2096-1456.2024.04.005.
Guo SY, Lu SJ, Ding YF. Clinical analysis of orthodontic traction of impacted upper incisors[J]. J Prev Treat Stomatol Dis, 2024, 32(4): 273-279. doi: 10.12016/j.issn.2096-1456.2024.04.005.

[95] Maruya Y, Hino R, Tadano M, et al. Orthodontic management of severe inversely impacted maxillary central incisors: a case series [J]. Front Oral Health, 2024, 5: 1474190. doi: 10.3389/froh.2024.1474190.

[96] Choi YK, Kim SH, Kim YI, et al. Prevalence and characteristics of impacted teeth in Korean orthodontic patients at ten university dental hospitals[J]. Korean J Orthod, 2025, 55(3): 234-241. doi: 10.4041/kjod24.213.

[97] Zogakis IP, Anagnostou C, Ioannidou I, et al. Radiographic evaluation of impacted and transmigrant canines: prevalence and sex-based differences in an orthodontic cohort[J]. Dent J, 2025, 13(9): 386. doi: 10.3390/dj13090386.

[98] Pinho T, Carvalho JP. Three-dimensional comparison of CBCT and intraoral scans for assessing orthodontic traction of impacted canines with clear aligners[J]. Dent J, 2025, 13(7): 286. doi: 10.3390/dj13070286.

[99] Mattos PM, Gonçalves FM, Basso IB, et al. Periodontal parameters in orthodontically tractioned teeth: a systematic review and meta-analysis[J]. Korean J Orthod, 2022, 52(6): 420-431. doi: 10.4041/kjod22.123.

[100] Kim J, Jung S, Lee KJ, et al. Forced eruption in impacted teeth: analysis of failed cases and outcome of re-operation[J]. BMC Oral Health, 2024, 24(1): 254. doi: 10.1186/s12903-024-03963-x.

[101] Simić S, Nikolić P, Stanišić Zindović J, et al. Root resorptions on adjacent teeth associated with impacted maxillary canines[J]. Diagnostics, 2022, 12(2): 380. doi: 10.3390/diagnostics12020380.

[102] Lee YH, Chang NY, Chae JM. Impact of orthodontic forced eruption timing on root development of impacted maxillary canines: a linear and volumetric analysis using cone-beam computed tomography images[J]. Angle Orthod, 2025, 95(5): 490-496. doi: 10.2319/070424-531.1.

[103] Liu DD, Zhang CY, Liu Y, et al. RUNX2 regulates osteoblast differentiation *via* the BMP4 signaling pathway [J]. J Dent Res, 2022, 101(10): 1227-1237. doi: 10.1177/00220345221093518.

[104] Ogawa S, Higuchi S, Yoshimoto Y, et al. Functional impact of pathogenic mutations in the Runt homology domain of mouse Runx2 on skeletal and dental phenotypes in cleidocranial dysplasia[J]. J Bone Miner Res, 2026, 41(8): 874-889. doi: 10.1093/jbmr/zjaf201.

[105] Shi Y, Ren J, Wang K, et al. Exploring the complexities of cleidocranial dysplasia: dental anomalies and treatment interventions[J]. Am J Orthod Dentofacial Orthop, 2025, 167(3): 319-330.e2. doi: 10.1016/j.ajodo.2024.10.014.

[106] Chen JL, Yu LN, Ran QC, et al. Impacted second premolars in cleidocranial dysplasia: three-dimensional position and morphology characteristics and factors affecting the success rate of closed eruption[J]. Am J Orthod Dentofacial Orthop, 2026, 169(2): 177-188. doi: 10.1016/j.ajodo.2025.09.016.

（编辑　罗燕鸿）

Open Access
This article is licensed under a Creative Commons Attribution 4.0 International License.
Copyright © 2026 by Editorial Department of Journal of Prevention and Treatment for Stomatological Diseases

官网