

# Root Resorption with Orthodontic Mechanics: Pertinent Areas Revisited

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## SUMMARY

Root resorption can occur at any time during orthodontic treatment and lead to a compromise in the prognosis of the tooth and the stability of the treatment results. Recent research has focused more on the cause and effect relationship as well as preventive or treatment options to combat this unwelcome event. Investigations have highlighted the genetic as well as molecular aspects of the process and enabled clinicians to determine which patients might be susceptible. A proper medical history, an assessment of predisposing factors, a radiographic evaluation for alterations in root morphology and careful planning and execution of orthodontic mechanics may reduce the incidence of root resorption. The current review is aimed at providing clinicians and academics with an insight into the process of root resorption, the methods of identification during its early stages and intervention at the right time to reduce its severity.

**Keywords:** Genetics, Orthodontics, Root resorption, Side effects, tooth movement biology.

**Abbreviations and acronyms:** PDL = periodontal ligament; IgE = immunoglobulin E; IOPAR = Progress periapical radiographs; CBCT = cone beam computed tomography; GCL = glutamate-cysteine ligase; CDC 42 = cell division cycle 42; TAGLN2 = transgelin-2; OPG = osteoprotegerin.

## INTRODUCTION

Root resorption, the unwanted but common sequela of orthodontic mechanotherapeutics, has been a concern to clinicians and patients since 1914, when it was first reported by Ottolengui.<sup>1</sup> The problem was investigated comprehensively by Ketcham, who published landmark articles in 1927.<sup>2,3</sup> Since that time, considerable additional research has been devoted to the issue and methods of control and prevention have been proposed. It has been shown that, among the many risk factors, applied orthodontic mechanics play a prominent role in root resorption.<sup>4</sup> Abbas and Hartsfield<sup>5</sup> reported an incidence of approximately one in 20 patients undergoing orthodontic treatment was susceptible to at least 5mm of root shortening. This information identifies root resorption as the second most common side effect of orthodontic treatment, following white spot lesions in tooth enamel.

It has been further shown that root resorption can appear during or after orthodontic treatment and compromise the stability of the treatment results and longevity of the tooth. Recent research has focused more on a cause and effect relationship as well as possible preventive or treatment options for this unwelcome event. Furthermore, research has highlighted the

genetic as well as molecular aspects of the process and helped clinicians determine who might be susceptible. The present review is aimed at providing clinicians and academics with an insight into the process of root resorption, the methods of detection during its early stages and the timing of possible intervention.

## PATHOPHYSIOLOGY

The process of root resorption is closely associated with injury and necrosis of the PDL. When heavy orthodontic forces are applied over a sustained period of time (weeks or months), necrosis (hyalinisation) of the compressed PDL may rapidly develop. The defensive leukocytes that migrate out of PDL capillaries include osteoclast progenitors that promptly coalesce to form multinucleated cells, capable of resorbing mineralised tissues (bone and tooth roots). External apical root resorption is initiated when the protective layer of cementoblasts, interfacing the hyalinised PDL, undergoes apoptosis and enables odontoclasts to resorb cementum and dentine. Initially, a protective layer of cementoid is removed which leaves a raw cemental surface open to odontoclastic attack.<sup>6–8</sup> Resorption is mostly observed radiographically in the apical region of the root because<sup>8</sup> the apical root third

is covered with cellular cementum, which relies on active cells and supporting vasculature, the loss of which renders the area vulnerable to trauma and cell injury-related reactions. It is reported that blood vessels occupy 47% of the PDL space in the apical region, compared with 4% at the cervical end of the root.<sup>9</sup> In addition, there is a decrease in the hardness and elastic modulus of the cementum, from the cervical to the apical region, making the apical areas prone to resorption.<sup>10</sup> Furthermore, the fulcrum of tooth movement (centre of rotation) is occlusal to the apical half of the root during tipping movements. This, along with the differences in the direction of the periodontal fibres likely results in increased trauma to the apical and middle thirds of the root.<sup>5</sup>

### CATEGORISING THE RESORPTION PROCESS

The earliest classification of root resorption considered the clinical severity of the process and was proposed by Brezniak and Wasserstein.<sup>11</sup> According to their description, a resorptive event may affect only the outer surface of the tooth root, which maintains a potential for full regeneration or remodelling (cemental or surface resorption). Alternatively, root resorption can reach into dentine and cause morphological alterations (dentinal resorption) or further, continue and lead to full resorption of hard tissues resulting in root shortening (circumferential root resorption). A pictorial representation of this phenomenon was provided and proposed as an index by Malmgren *et al.*<sup>12</sup>, which was further modified by Beck and Harris in 1994<sup>13</sup> (Figure 1). An additional classification with orthodontic relevance categorises the process as either internal (starting from the pulpal side) or external (which is further divided into apical as well as lateral root resorption). A repair process, initiated once the applied forces have been discontinued, lays down acellular cementum followed by the deposition of cellular cementum, the effects of which usually take at least 6-8 weeks to become radiographically evident.

### PREDISPOSING OR RISK FACTORS

A review of published literature has identified a number of risk factors, which predispose a patient to root resorption once subjected to orthodontic mechanics. These can be categorised as general or local factors.

### GENERAL FACTORS

#### Age at start of treatment

Even though most reports reveal poor correlation between the patient's age at the start of treatment and the incidence of root resorption, Sameshima and

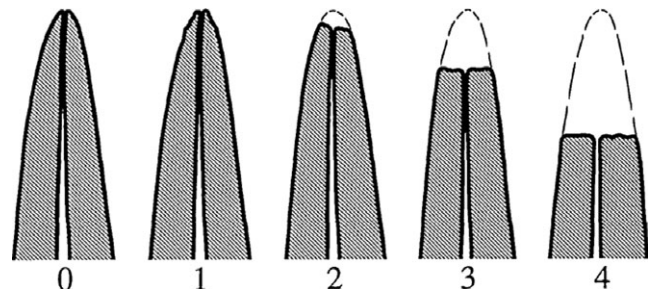


Fig. 1 Scoring system of Malmgren *et al.* (1982)<sup>9</sup> – Grade 0 – no resorption, Grade 1 mild resorption, root with normal length and only displaying an irregular contour, Grade 2 – moderate resorption, small area of root loss with apex exhibiting almost straight contour, Grade 3 – accentuated resorption, loss of almost one-third of root length, Grade 4 – extreme resorption, loss of more than one third root length. Grade 0 was added by Beck and Harris in their article for American Journal of Orthodontics and Dentofacial Orthopedics in the year 1994.<sup>10</sup> (Reprinted with permission from Elsevier)

Sinclair<sup>14</sup> reported an increased incidence in adults. This finding was confirmed by Ren *et al.*<sup>15</sup> through an animal study and Picanco *et al.*<sup>16</sup> through human research. The proposed reasons contributing to increased root resorption in adults were listed as a decreased periodontal vascularity and inelasticity, thicker cementum and its firm attachment in the apical third of the root in adults thereby increasing susceptibility.

### Gender

Though the majority of studies report little correlation between gender and the incidence of root resorption, Sameshima and Sinclair<sup>14</sup> reported a statistically insignificant increase in incidence in males. In addition, there are mixed reports indicating that females as well as males are prone to the process, which leaves clinicians in confusion. Recent literature supports the findings of Sameshima and Sinclair<sup>14</sup> while Geraldo de *et al.*<sup>17</sup> attributed the gender discrepancy to an altered root morphology and pipette-shaped central incisor roots in males which herald an increased tendency for root resorption. According to a thesis published by Kathryn and Begole<sup>18</sup>, females exhibit shorter root morphology than males in all ethnic groups.

### Ethnicity

Ethnic difference is considerable as evidenced by reports stating that Asians are less prone to root resorption compared with Caucasians and Hispanics.<sup>19</sup> Kathryn and Begole<sup>18</sup> conducted a detailed evaluation of pre-treatment root length of four ethnic group assessments. Caucasians and Hispanics were found to have larger relative root lengths compared with Asians and African Americans. The exception to this was the maxillary 2<sup>nd</sup> premolar which showed

greater root length values in Asians compared with Caucasians. The findings were interpreted as evidence that short roots (Asians, African Americans and females) were less likely to be affected by root resorption compared with roots that were longer or affected by altered morphology.

### Systemic diseases and medications

A close correlation exists between the immune system and the root resorptive process shown by an increased prevalence in patients with allergies and asthma. Elevated levels of IgE are present in patients suffering from asthma, atopy and allergy.<sup>20</sup> Asthma, in particular, results in an imbalance between T helper 1 and T helper 2 lymphocytes, the latter responsible for the pulmonary synthesis and release of inflammatory mediators, such as interleukins 4, 5, 6, 10 and 13.<sup>21</sup> The released cytokines attract inflammatory cells to the lung, which initiate further secretion of histamine, prostaglandins and leukotrienes. These signalling molecules enter the circulation and reach the periodontal ligament, where they are able to interact with target cells involved in tissue remodelling and tooth movement.<sup>22</sup> Moreover, the application of excessive orthodontic force in asthmatic patients, often results in tissue compression and necrosis, and subsequent clast cell activity leading to hard tissue resorption. In addition to asthma, diseases associated with low bone turn over, such as hypothyroidism, can lead to increased stress on tooth roots following applied orthodontic loads and lead to root resorption.<sup>23</sup>

### The genetic link revisited

Many studies have attempted to correlate root resorption with a patient's genetic characteristics and determined that individuals homozygous for IL-1 $\beta$  (+3953) allele 1 have a 5.6-fold increase in the risk of root resorption compared with those who are not homozygous. These individuals have reduced secretion of IL-1 $\beta$  leading to less catabolic bone remodelling response (resorption) and more damage to root structure.<sup>24,25</sup> Another candidate gene showing a close association with root resorption is TNFRSF11A which codes for the bone remodelling protein RANK. The reduced expression of any gene involved with bone remodelling will lead to stress concentration on tooth roots and likely precipitate root resorption.<sup>24,25</sup>

## LOCAL FACTORS

### Tooth shape and position

The majority of studies have suggested that central incisors are more susceptible to root resorption.<sup>13,26–28</sup>

However, two studies, indicated that lateral incisors were more affected<sup>19,29</sup> followed by molars and canines. The most commonly resorbed tooth in the mandibular arch is the canine followed by the lateral and central incisors<sup>19</sup>. Beck and Harris<sup>13</sup> reported more root resorption in the distal roots of molars as anchorage bends placed at the mesial aspect of the molars for bite opening caused the distal roots to be compressed in their bony sockets. A previous history of trauma<sup>30–32</sup> and pre-treatment root resorption<sup>11</sup> have been positively correlated with root resorption seen during orthodontic treatment. The relationship between root length and resorption also exhibited a positive correlation. The increase in dentine density following endodontic treatment produces resistance against the resorptive process when compared with vital teeth.<sup>28,33</sup> Interestingly, minimal resorption was observed in blunted apical forms<sup>19</sup> and the greatest resorption was encountered in pointed, tapered or dilacerated apical morphology.<sup>27</sup> This observation may be explained by pressure from an axial component of an applied orthodontic force is delivered maximally to the root apical region resulting in a localised ischaemic necrosis, which removes pre-cementum, cementoblasts and permits colonisation by dentinoclasts. Therefore, abnormal root shapes observed in pre-treatment diagnostic records should be considered with caution and carefully monitored throughout treatment for the development of iatrogenic damage.

### What do orthodontic mechanics do to tooth roots?

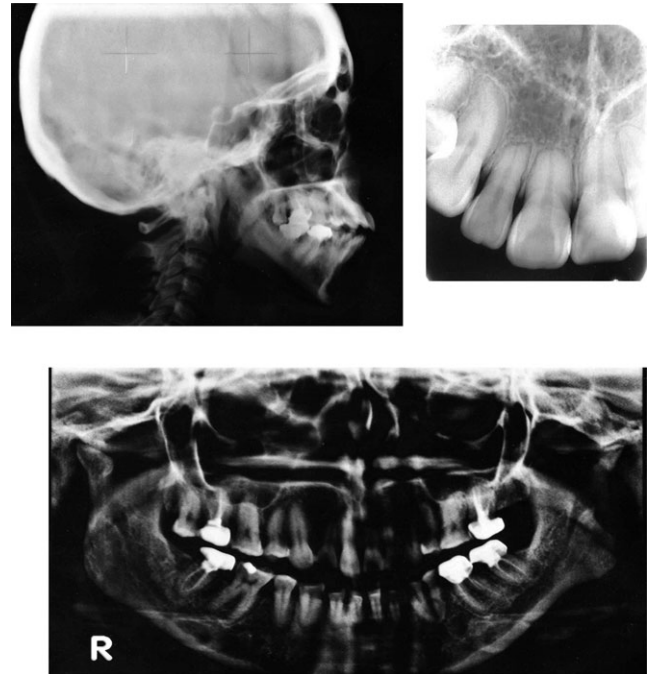
The detrimental nature of fixed appliances compared with removable appliance therapy on tooth roots has been previously examined and assessed<sup>34</sup>. While Beck and Harris<sup>13</sup> found no statistically significant difference in the resorption rate between Begg Light-Wire mechanics and Edgewise (Tweed) techniques, McNab *et al.*<sup>35</sup> reported a higher incidence as well as increased root resorption in patients treated with the Light-Wire appliance. It was determined that the incidence root resorption increased 3.72 times when extractions were undertaken as part of Light-Wire appliance therapy. Alexander<sup>29</sup> considered the presence of jiggling movements or round-tripping movements during mechanotherapy as likely causes of increased resorption. Of the various tooth movements, intrusion and torqueing have been most commonly associated with root damage and are evident and required in Class II division 2 correction. The displacement of the root apex horizontally or by torqueing has been proven to result in root resorption.<sup>19,36,37</sup> The highest incidence of root resorption has been reported to occur when 3 – 4.5 mm of torqueing movement was performed.<sup>19</sup> The relationship between the length of treatment time and root

resorption has been positively correlated by almost all studies<sup>11,19,36</sup>. Ahu Acar *et al.*<sup>38</sup> evaluated the effect of the type of force applied, whether continuous or interrupted, on the pattern of resorption and observed less severe apical blunting and smaller resorption affected areas when the applied force was intermittent.

## IDENTIFYING MID-TREATMENT RESORPTION

Progress periapical radiographs (IOPAR) still form the major investigative method used to identify mid-treatment root resorption. Multiple grading systems and scoring criteria exist for assessing the resorptive process. Contemporary digital imaging tools such as cone beam computed tomography (CBCT) with reduced radiation dose and high accuracy, have been critical for the purpose of identification.<sup>4</sup> Durack *et al.*<sup>39</sup> compared IOAPR and CBCT for the detection of resorption craters and concluded that IOPAR carries inherent limitations and shortcomings. A detailed study by Sherrard *et al.*<sup>40</sup> concluded that IOPAR evaluation could lead to an underestimation of root length by an average of 2.6 mm while a CBCT discrepancy is of the order of 0.3 mm, making it the assessment tool of choice. The main problems of CBCT usage are the associated increased radiation dose, cost and ethical issues. Panoramic radiographs which offer less radiation exposure, less patient and operator time and better patient co-operation, forms the least reliable method confirmed by the difficulty in identifying precise root morphology.<sup>23</sup> Moreover, the midline structures are often obscured in panoramic radiographs which makes resorption difficult to assess in central and lateral incisors. A comparative evaluation of the root resorption process occurring in maxillary central incisors as identified through IOPAR and orthopantamograph is provided in Figure 2.

Until now, there have been no reliable biomarkers identified for the chair-side estimation of the dental resorptive process. The search for biochemical markers was promoted by the discovery of dentine specific proteins in gingival crevicular fluid, as the by-products of root resorption.<sup>41</sup> The main problem associated with this detection method was the need for high-level laboratory instrumentation to perform ELISA, Western-blot or electrophoresis (SDS-PAGE). Rody Jr *et al.* through their recent publications<sup>42,43</sup> carried out liquid chromatographic mass spectrometry and immunoassay to quantify root resorption in the deciduous dentition (not orthodontically treated) and suggested strong upregulation of glutamate-cysteine ligase (GCL), which is a rate-limiting enzyme that catalyses the formation of the cellular antioxidant, glutathione. The upregulation of epidermal growth



**Fig. 2** Root resorption process of same patient as identified through orthopantamograph and intra oral periapical radiograph. This patient was treated three times by different orthodontists for closing the openbite and was unsuccessful in all three times. There was a failure in diagnosis at the initial point by not identifying the vertical maxillary excess and mandibular retrognathism leading to poor treatment planning and execution. Lateral cephalogram showed the evidence of unsuccessful treatment. Please note the limitation observed in the orthopantamograph in identifying midline structures and making the resorption process in the central incisors obscure. IOPAR is considered effective tool in comparison to orthopantamograph in identifying this phenomenon.

factor receptor pathway substrate-8-like protein 2 and the down regulation of two exosome-related proteins, cell division cycle 42 (CDC 42) and tagalin-2 (TAGLN2), were identified. Immunoassay revealed a statistically significant difference in the levels of IL-1 $\beta$ , osteoprotegerin (OPG) and MMP-9, leading to a conclusion that IL-1 RA was downregulated in patients with root resorption. However, translating this research data into a useful clinical approach for identification and diagnosis still requires additional investigation.

Once root resorption has been identified, it is recommended that orthodontic mechanics be discontinued for at least 6 months. During this rest period, it is anticipated that root resorption craters will be repaired. Intermittant forces are tissue friendly in comparison with continuously-applied forces as the former allow repair during the rest period.

## THE REPAIR PROCESS

Upon cessation of an orthodontic force, active root resorption will stop and a partial, functional or anatomic repair process commences.<sup>44,45</sup> Partial repair

occurs when exposed dentine is incompletely covered by new cementum leaving an exposed area. Functional repair occurs when the exposed dentine has been completely covered by a thin layer of repair cementum but without the re-establishment of original contour. Anatomic repair, as the most preferred, is characterised by the restoration of the root surface to its original contour. It has been reported by Owmann-Moll and Kuroi<sup>44</sup> and Cheng *et al.*<sup>45</sup> that it takes at least 8 weeks of rest for anatomic repair to occur while the partial and functional repair processes predominate during the first 4-6 weeks. Owmann-Moll *et al.*<sup>46</sup> in 1995 clearly delineated the observed percentage of repair process as 17 to 31% during the first 4 weeks (partial repair), 33-40% during 5-8 weeks (functional repair) and 12% after 8 weeks (anatomic repair). Cheng *et al.*<sup>45</sup> reported that 8 weeks of rest produced anatomic repair in 4 examined teeth irrespective of the amount of applied force or stress or the amount of orthodontic damage induced. It was concluded that a minimum of 4 weeks rest is essential for the initiation of the repair process.

The repair of root resorption craters occurs by the deposition of cellular or acellular cementum. Vardimon *et al.*<sup>47</sup> described the process with the help of expansion studies as non-functional retarded acellular repair and functional rapid cellular repair. Accordingly, the first increment of repair tissue was characterised by the apposition of acellular cementum overlying the bottom of a resorption crater. This layer had sparse Sharpey fibre attachments. Subsequently, the increments deposited were characterised by mixed fibrillar cellular cementum, comprising extrinsic (Sharpey fibres) and intrinsic fibres. This occurs as a result of a differential healing process. Acellular cementum forms very slowly and occurs during initial rest phases. Later, when healing becomes faster, the cementocytes are trapped within their lacunae as complete mineralisation of the increments takes place. Owmann-Moll *et al.*<sup>44,46</sup> and Cheng *et al.*<sup>45</sup> supported this hypothesis and suggested acellular cementum predominated in tooth repair following shorter rest periods (2-3 weeks) compared with teeth subjected to longer resting time periods (6-7 weeks).

### AUGMENTING THE REPAIR PROCESS WITH ADJUNCT APPROACHES

Research has demonstrated possible ways of reducing the rate of root resorption during mechanotherapy which have included drugs, hormones, and the application of low-intensity pulsed ultrasound.<sup>23,48</sup> The drugs which have been administered included bisphosphonates as potent bone resorption inhibitors. The

anti-inflammatory properties of tetracyclines and NSAIDs have also been noted to reduce root resorption. Hormones which have a positive effect on resorption include corticosteroids and L-thyroxine due to its dual activation of parathormone and bone remodelling resulting in less stress on the root apex.<sup>23</sup> These research data are obtained from either animal experiments or by incidental observation of patients consuming these drugs. This creates uncertainty related to the clinical applicability of pharmacological programmes solely for the purpose of suppressing the root resorptive process. Considering the unfavourable effects that pharmacology might have in other systems, a rest period of at least 8 weeks is considered the best option, if mid-treatment root resorption is diagnosed.

### CONCLUSIONS

It is well known that orthodontic therapy is associated with root shortening. A proper medical history, an assessment of predisposing factors, a radiographic evaluation for alterations in root morphology and careful planning and execution of orthodontic mechanics may reduce the incidence of root resorption to an extent. A mid-treatment radiographic evaluation with IOPAR can identify teeth at risk and can indicate the need for an adequate rest period so that functional or anatomic repair might be promoted. Further treatment in affected patients should be performed with caution and with the appropriate application of very light forces while avoiding movements definitively associated with resorption, such as intrusion. Mavragani *et al.*<sup>49</sup> in 2002 suggested early-age treatment, when roots were incompletely developed and therefore showed more root length at the end of treatment due to continuing development. This may be considered when planning orthodontic treatment in young children but lacks practicality.

### REFERENCES

- Ottolengui R. The physiological and pathological resorption of tooth roots. *Dent Items Interest* 1914;36:322-362.
- Ketcham AH. A preliminary report of an investigation of apical root resorption of permanent teeth. *Int J Orthod* 1927;13:115-127.
- Ketcham AH. A radiographic study of orthodontic tooth movement: A preliminary report. *J Am Dent Assoc* 1927;14:1577-1596.
- Krishnan V. Critical issues concerning root resorption: A contemporary review. *World. J Orthod* 2005;6:30-40.
- Abass KS, Hartsfield JK. Orthodontics and External Apical Root Resorption. *Semin Orthod* 2007;13:246-56.
- Brudvik P, Rygh P. The initial phase of orthodontic root resorption incident to local compression of the periodontal ligament. *Eur J Orthod* 1993;15:249-263.

7. Brudvik P, Rygh P. Root resorption beneath the main hyalinized zone. *Eur J Orthod* 1994;16:249–263.
8. Brudvik P, Rygh P. Multi-nucleated cells remove the main hyalinized tissue and start resorption of adjacent root surfaces. *Eur J Orthod* 1994;16:265–273.
9. Blaushild N, Michaeli Y, Steigman S. Histomorphometric study of the periodontal vasculature of the rat incisor. *J Dent Res* 1992;71:1908–12.
10. Chutimanutskul W, Ali Darendeliler M, Shen G, Petocz P, Swain M. Changes in the physical properties of human premolar cementum after application of 4 weeks of controlled orthodontic forces. *Eur J Orthod* 2006;28:313–18.
11. Brezniak N, Wasserstein A. Orthodontically induced inflammatory root resorption –part II: Clinical aspects. *Angle Orthod* 2002;72:180–184.
12. Malmgren O, Goldson L, Hill C, Orwin A, Petrini L, Lundberg M. Root resorption after orthodontic treatment of traumatized teeth. *Am J Orthod* 1982;82:487–91.
13. Beck BW, Harris EF. Apical root resorption in orthodontically treated subjects – analysis of edgewise and light wire mechanics. *Am J Orthod Dentofac Orthop* 1994;105:350–361.
14. Sameshima GT, Sinclair PM. Predicting and preventing root resorption – Part I - Diagnostic factors. *Am J Orthod Dentofac Orthop* 2001;119:505–510.
15. Ren Y, Maltha JC, Liem RS, Stokroos I, Kuijpers-Jagtman AM. Age-dependent external root resorption during tooth movement in rats. *Acta Odontol Scand* 2008;66:93–98.
16. Picano GV, de Freitas KMS, Cancado RH, Valarelli FP, Picano PRB, Feijao CP. Predisposing factors to severe external root resorption associated to orthodontic treatment. *Dental press J Orthod* 2013;18.
17. de Antonio Geraldo O, Alberto C, Jose Luiz Cintra Martins-Ortiz J, Fernanda M, Solange de Oliveria Braga F. Analysis of predictors of root resorption in orthodontic treatment. *J Dent Oral Hyg* 2011;3:46–52.
18. Kathryn E, Begole E. Prevalence of short dental roots in four ethnic groups in an orthodontic population. University of Illinois at Chicago 2011, Thesis, Publication number-1492989
19. Sameshima GT, Sinclair PM (2001) Predicting and preventing root resorption – Part II – Treatment factors. *Am J Orthod Dentofac Orthop* 2001;119:511–515.
20. Davidovitch Z, Krishnan V. Adverse effects of Orthodontics: A report of two cases. *World J Orthod* 2008;9:268 (Online only)
21. Robinson DS, Hamid Q, Ying S, *et al.* Predominant TH2-like bronchoalveolar T-lymphocyte population in atopic asthma. *N Engl J Med* 1992;326:298–304.
22. Meikle MC, Heath JK, Atkinson SJ, Hembry RM, Reynolds JJ. Molecular biology of tooth movement. In: Norton LA, Burstone CJ, eds. Boca Raton, Florida: CRC Press, 1989: 71–86.
23. Abubara A. Biomechanical aspects of external root resorption in orthodontic therapy. *Med Oral Patol Oral Cir Bucal* 2007;12.
24. Al-Qawasmi RA, Hartsfield JK Jr, Everett ET, Flury L, *et al.* Genetic predisposition to external apical root resorption. *Am J Orthod Dentofac Orthop* 2003;123:242–52.
25. Al-Qawasmi RA, Hartsfield JK, Everett ET, Weaver MR, *et al.* Root resorption associated with orthodontic force in IL-1Beta knockout mouse. *J Musculoskelet Neuronal Interact* 2004;4:383–85.
26. Janson GR, De Luca Canto G, Martins DR, Henriques JF, De Freitas MR. A radiographic comparison of apical root resorption after orthodontic treatment with 3 different fixed appliance techniques. *Am J Orthod Dentofac Orthop* 1999;118:262–273.
27. L' Abee EM, Saderink GCH. Apical root resorption during Begg treatment. *J Clin Orthod* 1985;19:60 – 61.
28. Remington DN, Joondeph DR, Artun J, Riedel RA, Chapko MK. Long term evaluation of root resorption occurring during orthodontic treatment. *Am J Orthod Dentofac Orthop* 1989;96:43–46.
29. Alexander SA. Levels of root resorption associated with continuous arch and sectional arch mechanics. *Am J Orthod Dentofac Orthop* 1996;110:321–324.
30. Mirabella A, Artun J. Risk factors for apical root resorption of maxillary anterior teeth in adult orthodontic patients. *Am J Orthod Dentofac Orthop* 1995;108:48–55.
31. Newman WG. Possible etiologic factors in external root resorption. *Am J Orthod* 1975;67:539–552.
32. Brin I, Becker A, Zilberman Y. Resorbed lateral incisors adjacent to impacted canines have normal crown size. *Am J Orthod Dentofac Orthop* 1993;104:60–66.
33. Spurrier SW, Hall SH, Joondeph DR, Shapiro PA, Riedel RA. A comparison of apical root resorption during orthodontic treatment in endodontically treated teeth and vital teeth. *Am J Orthod Dentofac Orthop* 1990;97:130–134.
34. Linge BO, Linge L. Apical root resorption in upper anterior teeth. *Eur J Orthod* 1983;5:173–183.
35. McNab S, Battistutta D, Taverne A, Symons AL. External apical root resorption following orthodontic treatment. *Angle Orthod* 2000;77:227–232.
36. Baumirind S, Korn EL, Boyd RL. Apical root resorption in orthodontically treated adults. *Am J Orthod Dentofac Orthop* 1996;110:311–320.
37. Parker RJ, Harris EF. Directions of orthodontic tooth movements associated with apical root resorption of the maxillary central incisor. *Am J Orthod Dentofac Orthop* 1998;114:677–683.
38. Acar A, Canyurek V, Kocaga M, Ervedi N. Continuous Vs discontinuous force application and root resorption. *The Angle Orthodontist* 1999;69:159–163.
39. Durack C, Patel S, Davies J, Wilson R, Manocci F. Diagnostic accuracy of small volume cone beam computed tomography and intra oral periapical radiography for the detection of simulated external inflammatory root resorption. *Int Endod J* 2011;44:136–47.
40. Sherrard JF, Rossouw EP, Benson BW, Carrillo R, Buschang PH. Accurac and reliability of tooth and root lengths measured on cone-beam computed tomographs. *Am J Orthod Dentofac Orthop* 2010;137:S100–108.
41. Mah J, Prasad N. Dentine phosphoproteins in gingival crevicular fluid during root resorption. *Eur J Orthod* 2004;26:25–30.
42. Rody WJ Jr, Wjijemgunasinghe M, Holliday LS, McHugh KP, Wallet SM. Immunoassay analysis of proteins in gingival crevicular fluid samples from resorbing teeth. *Angle Orthod* 2016;86:187–92.
43. Rody WJ Jr, Holliday LS, McHugh KP, Wallet SMSpicer VKrokhin O. Mass spectrometry analysis of gingival crevicular fluid in the presence of external root resorption. *Am J Orthod Dentofac Orthop* 2014;145:787–98.
44. Owmann-Moll P, Kurol J. The early reparative process of orthodontically induced root resorption in adolescents – location and type of tissue. *Eur J Orthod* 1998;20:727–32.
45. Cheng LL, Turk T, Elekdag-Turk S, Jones AS, Yu Y, Darendeliler MA. repair of root resorption 4 and 8 weeks after application of continuous and heavy forces on premolars for 4 weeks: a histologic study. *Am J Orthod Dentofac Orthop* 2010;138:727–34.

46. Owmann-Moll P, Kuroi J, Lundgren D. Repair of orthodontically induced root resorption in adolescents. *The Angle Orthod* 1995;65:403–8.
47. Vardimon AD, Graber TM, Pitaru S. Repair process of external root resorption subsequent to palatal expansion treatment. *Am J Orthod Dentofac Orthop* 1993;103:120–30.
48. El-Bialy T, El-Shamy I, Graber TM. Repair of orthodontically induced root resorption by ultrasound in humans. *Am J Orthod Dentofac Orthop* 2004;126:186–93.
49. Mavragani M, Boe OE, Wisth PL, Selvig KA. Changes in root length during orthodontic treatment: advantages for immature teeth. *Eur J Orthod* 2002;24:91–97.

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