Kaohsiung Journal of Medical Sciences (2018) xx, 1–8

Available online at www.sciencedirect.com

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journal homepage: http://www.kjms-online.com

Review Article

Orthodontic tooth movement: The biology and clinical implications

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Received 28 October 2017; accepted 12 January 2018

KEYWORDS
Orthodontic tooth movement;
Periodontal ligament;
Bone remodeling;
Accelerated tooth movement

Abstract Orthodontic tooth movement relies on coordinated tissue resorption and formation in the surrounding bone and periodontal ligament. Tooth loading causes local hypoxia and fluid flow, initiating an aseptic inflammatory cascade culminating in osteoclast resorption in areas of compression and osteoblast deposition in areas of tension. Compression and tension are associated with particular signaling factors, establishing local gradients to regulate remodeling of the bone and periodontal ligament for tooth displacement. Key regulators of inflammation and tissue turnover include secreted factors like RANK ligand and osteoprotegerin, transcription factors such as RUNX2 and hypoxia-inducible factor, cytokines, prostaglandins, tissue necrosis factors, and proteases, among others. Inflammation occurred during tooth movement needs to be well controlled, as dysregulated inflammation leads to tissue destruction manifested in orthodontic-induced root resorption and periodontal disease. Understanding the biology has profound clinical implications especially in the area of accelerating orthodontic tooth movement. Surgical, pharmacological, and physical interventions are being tested to move teeth faster to reduce treatment times and time-dependent adverse outcomes. Future developments in acceleratory technology and custom appliances will allow orthodontic tooth movement to occur more efficiently and safely.

Conflicts of interest: All authors declare no conflicts of interests.

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https://doi.org/10.1016/j.kjms.2018.01.007

Please cite this article in press as: Li Y, et al., Orthodontic tooth movement: The biology and clinical implications, Kaohsiung Journal of Medical Sciences (2018), https://doi.org/10.1016/j.kjms.2018.01.007

Introduction

Orthodontics is a special discipline dedicated to the investigation and practice of moving teeth through the bone. Moving teeth through the dentoalveolar complex is a synergistic sequence of physical phenomenon and biological tissue remodeling. The physical behavior of tooth movement due to orthodontic force relies on Newton's Laws. The tooth biological system reacts to variation in force magnitude, time of application and directionality through receptor cells and signaling cascades that ultimately produce bone remodeling and orthodontic tooth movement (OTM). This review focuses on the biology of tooth movement and its implication in clinical orthodontics.

Periodontium: the tooth supporting complex

Periodontium is the investing and supporting attachment of the teeth to alveolar bone. It includes both the soft tissues of periodontal ligament (PDL) and gingiva as well as the hard tissues of cementum and alveolar bone (Fig. 1).

The ability of teeth to move through the bone relies on the PDL, which attaches the tooth to the adjacent bone. The PDL is a dense fibrous connective tissue structure that consists of collagenous fiber bundles, cells, neural and vascular components and tissue fluids. Its primary function is to support the teeth in their sockets while allowing teeth to withstand considerable chewing forces. On average, the PDL occupies a space about 0.2 mm wide. Depending on its location along the root, PDL width can range from 0.15 to 0.38 mm, with its thinnest part located in the middle third of the root. PDL space also decreases progressively with age [1]. Most PDL space is taken up by bundles of collagen fibers (mainly Type I) that are embedded in the intercellular substance. The terminal portion of the fibers that insert in the cementum and alveolar bone is termed Sharpey's fibers. These fibers can be divided into the principal fibers, the accessory fibers and the oxytalan (elastic) fibers. According to their orientation and location along the tooth, the principal fibers can be further categorized into the transseptal fiber (or interdental ligament) and alveolodental ligament (Fig. 1). Transseptal fibers extend interproximally connecting the cementum of adjacent teeth to maintain tooth alignment, and the alveolodental ligament group of fibers helps teeth withstand compression forces during mastication. In addition to principal fibers, accessory fibers run from alveolar bone to cementum in different planes, more tangentially to prevent rotation of the tooth. Besides PDL fibers, paradental cells of different functions reside in the PDL space, including: 1) synthetic cells like fibroblasts which make up 50–60% of total PDL cellularity, osteoblasts, and cementoblasts; 2) resorptive cells such as osteoclasts, fibroblasts, cementoclasts; 3) progenitor cells including undifferentiated mesenchymal cells; 4) defense cells such as macrophages, mast cells and lymphocytes; and 5) epithelial cells, i.e. remnants of the epithelial root sheath of Hertwig [1]. Together, these various cells participate in the homeostasis of the periodontium. Finally, the PDL space is filled with tissue fluid known as interstitial fluid that is ultimately derived from the vascular system. This fluid-filled chamber allows the PDL space to evenly distribute forces loaded onto teeth, serving as a shock absorber.

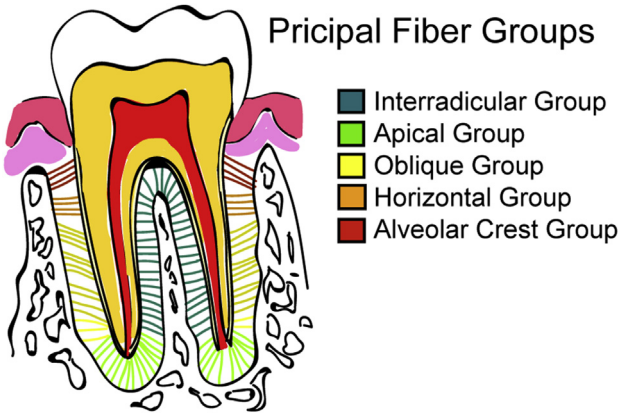

Figure 1. Components of the periodontium. Different types of principal fiber groups are indicated with different colors. Red: pulp. Yellow: dentin. White crown: enamel. Pink: Gingiva. Black outline: alveolar bone.

The alveolar bone is a mineralized connective tissue that consists of mineralized tissue (60 w%), organic matrix (25 w %) and water (15 w%) [2]. While the majority of alveolar bone is trabecular, a plate of compact bone called the lamina dura lies adjacent to the PDL space. PDL fibers anchor to the alveolar bone by piercing through the lamina dura, while the other ends connect to the cementum (Fig. 1). Multiple cell types, namely osteoblasts, osteoclasts and osteocytes, play critical roles in the homeostasis and function of the alveolar bone. In addition, macrophages, endothelial cells and adipocytes can also be found within the alveolar bone. Osteoblasts are mononucleated and specialized "bone forming" cells. Both osteoblasts and fibroblasts can synthesize Type I collagen matrix. Osteoblasts differ from fibroblasts because they can express Runx2 (aka. Cbfa1), a master switch for osteoblast differentiation from mesenchymal progenitor cells [3]. The number of osteoblasts decreases with age, leading to an imbalance of bone deposition and resorption [4]. Osteocytes are derived from osteoblasts that are embedded in mineralized bone during bone apposition. During this process, minerals such as hydroxyapatite, calcium carbonate and calcium phosphate get deposited around the osteocyte, forming lacuna, the space that an osteocyte occupies during its entire lifespan. Lacunae are connected via narrow channels known as cannaliculi, where dendrites of osteocytes contact and communicate via gap junctions. While the "bone forming" osteoblasts and osteocytes arise from the mesenchymal cell lineage, the "bone resorbing" osteoclasts originate from a different progenitor population, the hematopoietic/monocyte lineage, and are formed by the fusion of multiple monocytes becoming "multinucleated". Osteoclasts are characterized by their high expression of Tartrate Resistant Acid Phosphatase (TRAP), Cathepsin K, Chloride channel 7 (ClCN7), and Osteoprotegerin (OPG). Cathepsin K is a protease capable of catabolizing bone matrix proteins such as elastin, collagen and gelatin. ClCN7 shuffles chloride ions through the cell membrane, thereby maintaining osteoclast neutrality. OPG (aka osteoclastogenesis inhibitory factor or tumor necrosis factor receptor superfamily member 11B) is an osteoblast expressed decoy receptor for the receptor activator of

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