Commentary

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Periodontal Tissue's Dynamic Balance between Bone Formation and Resorption is Disrupted by Mechanical Force

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Description

Periodontal tissue remodeling is the foundation for Orthodontic Tooth Movement (OTM) during orthodontic treatment. Periodontal tissue's dynamic balance between bone formation and resorption is disrupted by mechanical force. The tension side of alveolar bone experiences bone formation, while the compression zone experiences bone resorption. The speed of OTM is significantly influenced by the rate of remodeling in periodontal tissue. Osteoclast-mediated bone resorption and osteoblast-mediated bone formation are two tightly coupled processes that are regulated by a variety of cytokines, chemokines and their receptors, as well as other inflammatory mediators. The process involves communication and regulation between varieties of cells. As a membrane-associated cytokine, receptor activator of NF-kappa (NK-B) ligand (RANKL) is essential for osteoclastogenesis. Bone resorption factors stimulate the expression of RANKL by osteoocytes, osteoblasts, and stromal cells. RANK, a receptor for RANKL, is expressed by osteoclast precursor cells. Osteoclastic differentiation is triggered when RANKL binds to RANK, which sets off a number of signal transduction pathways in the next step. Osteoprotegerin (OPG), a RANKL decoy receptor, is also produced by osteoblasts and stromal cells. Osteoclast differentiation and bone resorption can be inhibited by OPG by inhibiting the interaction between RANKL and RANK. Local RANKL gene transfection has the potential to significantly increase bone resorption, speed up bone remodeling, and promote OTM. Similarly, local OPG gene transfection can prevent OTM and bone resorption.

Autosomal Recessive Osteopetrosis

On the other hand, severe RANKL genetic defects may result in a particular form of autosomal recessive osteopetrosis (ARO) that is characterized by the failure of osteoclasts to resorb bone, resulting in increased bone mineral density. Due to the inability of osteoblasts to induce the formation of osteoclasts, OPG-deficient mice have been shown to have severe osteopetrosis, difficulty erupting teeth, and a complete absence of osteoclasts. The periodontal ligament is a connective tissue that is soft and fibrous and is buried between the cementum and the inner wall of the alveolar bone. Periodontal ligament tissue can be used to isolate periodontal ligament stem cells (PDLSCs).They are capable of self-renewal and differentiation across multiple lineages. PDLSCs are essential for maintaining periodontal homeostasis. PDLSCs can differentiate into a variety of cell lineages, including osteoblast-like cells, cementoblast-like cells, adipocytes, and fibroblast-like cells, under the right conditions for induction. PDLSCs have been shown to promote the formation of new alveolar bone tissue, periodontal ligament, and cementum from damaged periodontal tissue *in vivo*. PDLSCs have been identified as the most promising source of differentiation for alveolar bone regeneration. In conclusion, it is believed that PDLSCs have numerous potential applications in periodontal tissue regeneration engineering. During orthodontic tooth movement, PDLSCs also play a crucial role in remodeling the periodontal ligament and alveolar bone. Zhang created an OTM rat model and monitored the response of PDLSCs *in vivo*. The study found that PDLSCs could be reactivated during orthodontic treatment on both the compression and tension sides. After orthodontic treatment, PDLSCs also participate in the process of relapse. PDLSCs' osteogenic differentiation and proliferation could be aided by mechanical stress, according to *in vitro* studies. PDLSCs typically play a significant role in OTM, which may involve a wide range of mechanoreceptors and pathways, including the cytoskeleton, MAPK signal, TGF-/Smad, Wnt/-Catenin pathway, and RANKL/OPG axis. The primary component of the traditional Chinese medicine known as sinomenium acutum is Sinomenine (SIN), a natural alkaloid. Sinomenine is well-known for its obvious anti-inflammatory, immunosuppressive, antioxidant, antiarrhythmic, and anti-cancer properties. In the clinical treatment of rheumatoid diseases, it is frequently used. Additionally, some studies have demonstrated that sinomenine influences bone metabolism. It has been reported that sinomenine can regulate the Akt/RUNX2 signaling pathway to promote osteoblast differentiation while inhibiting osteoclast differentiation through the RANKL signaling pathways36. The expression of osteogenic markers like ALP, OCN, type I collagen (COL1A1), and osteopontin in MC3T3-E1 cells may be enhanced by sinomenine, according to reports. We hypothesized that sinomenine might promote osteogenic differentiation of PDLSCs and inhibit OTM and root resorption in

rats based on previous research. We are aware of no *in vivo* or *in vitro* studies on the impact of sinomenine on the osteogenesis of OTM and PDLSCs. As a result, the osteogenesis of PDLSCs and the effects of sinomenine on OTM and orthodontic induced root resorption (OIRR) in rats were the subjects of this study.

Clinical Effects of Sinomenine on Orthodontic Treatment

The clinical effects of sinomenine on orthodontic treatment and its use in periodontal tissue regeneration engineering will benefit greatly from this study's findings, as we anticipate. This study's animal experiment was carried out in accordance with the ARRIVE guidelines and was approved by Shandong University's Institutional Animal Ethics Committee (No. 20220104).In accordance with the guidelines established by the institutional ethics committee of Shandong University, fifty-four 6-week-old male Wistar rats weighing 180 15 g were purchased from SPF biotechnology company in Beijing, China. Under specific pathogen-free conditions, the rats were housed in pairs in standard plastic cages. They were raised in a room with a constant temperature of 21 °C, a relative humidity of 55 %, and a 12-hour light/dark cycle. Maintain adequate nutrition by feeding a laboratory powder diet and drinking plenty of water on an as-needed basis. Before the experiment began, each animal was adapted for a week. Each animal's general health and weight were tracked throughout the experiment. The rats were randomly divided into three groups, each with 18 rats:1) the normal saline control group; (2) a treatment group receiving a low dose of sinomenine (20 mg/kg);3) a treatment group receiving a high dose of sinomenine (40 mg/kg).To make the stock solution, dissolve sinomenine (Solarbio, Beijing, China) in 0.05% dimethyl sulfoxide. Before use, the stock solution was diluted with enough normal saline to reach the required concentrations. Sinomenine at doses of 20 mg/kg/d and 40 mg/kg/d was injected intraperitoneally into rats in the low-dose and high-dose groups, respectively. For 14 days; normal saline was injected intraperitoneally into rats in the control group. Bone metabolism can be controlled by Sinomenine. By regulating the Akt/Runx2 signaling pathway, previous studies demonstrated that sinomenine could promote osteogenic differentiation of MC3T3-E1 cells and inhibit osteoclastic differentiation of mesenchymal stem cells. Additionally, numerous studies have demonstrated the anti-inflammatory properties of sinomenine, which has long been used to treat rheumatoid arthritis and systemic lupus erythematous. It has been demonstrated that sinomenine can significantly inhibit the immune response of macrophages stimulated by LPS by downregulating inflammatory cytokines (TNF-, IL-1, and IL-6).The study of sinomenine's effect on OTM may be useful in the clinical treatment of sinomenine-treated orthodontic patients due to its widespread clinical application and its influence on bone metabolism.