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Hormones and Its Effect on Orthodontic Tooth Movement

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Abstract

Orthodontic treatment relies on the application of controlled forces to teeth, which initiates a cascade of mechanical, biochemical, and cellular events within the periodontal ligament (PDL) and alveolar bone. These events lead to bone remodeling, a dynamic process coordinated by osteoblasts, osteoclasts, and osteocytes. Hormones act as critical regulators of these activities, influencing the rate, quality, and stability of orthodontic tooth movement (OTM). Some hormones accelerate bone resorption and tooth movement, while others inhibit these processes. This review synthesizes available evidence on the role of key endocrine and paracrine hormones—including sex hormones, prostaglandins, thyroid hormones, parathyroid hormone, calcitonin, vitamin D, and relaxin in orthodontic tooth movement.

Keywords: Hormones, Orthodontic tooth movement, Bone remodeling, Orthodontic forces

Introduction

Orthodontic treatment is widely employed to achieve functional occlusion, improved oral health, and enhanced facial esthetics. Tooth movement occurs when sustained forces are applied, disrupting the physiological equilibrium of the dentofacial complex. The resulting biological response involves bone remodeling around the teeth, where osteoclasts resorb bone on the compression side and osteoblasts deposit bone on the tension side.

The nature of orthodontic tooth movement is: -

- bio mechanic in nature
- based on the stress and strain on PDL environment,
- a result from forces delivered to the teeth via fixed or removable appliance.

Optimum orthodontic force is one which moves the teeth most rapidly in the desired direction, with the least possibility of damage to tissue and minimum patient discomfort. Schwarz defined it as the force leading to a change in tissue pressure, that approximated the capillary vessel and blood pressure and preventing their occlusion in the compressed PDL. Below the optimum level causes no reaction in PDL and forces exceeding optimal level would lead to tissue necrosis [41.] Oppenheim advocated the use of lightest force capable of bringing about the tooth movement[43.] Oppenheim and Schwarz stated the optimum force is equivalent to the capillary pulse pressure which is 20 – 25gm/sq.cm of tooth surface area [43.]

From clinical point of view, the Optimum Orthodontic force has the following characteristics [43]: -

- Minimal patient discomfort
- Produce rapid tooth movement
- The lag phase of tooth movement is minimal
- No marked mobility of the teeth being moved

From histological point of view, The Optimum Orthodontic force has the following characteristics [(43):] -

- The vitality to the tooth and supporting PDL is maintained
- Initiates maximum cellular response
- Produces direct or frontal resorption

When force is applied to a tooth, mechanical stress is loaded on the alveolar bone. Alveolar bone and the periodontal ligament (PDL) are compressed on one side, while on the opposite side, the PDL is stretched. The mechanical stress of the stretched PDL induces alveolar bone modeling (deposition), while the mechanical compression produces bone remodeling (resorption) [44.]

Several factors affect and modify the nature and amount of orthodontic tooth movement and they are: -

- Magnitude, direction and nature of the force
- Bone density
- Age of the person
- Systemic health
- Hormones
- And factors that influence the bone turnover.

Hormones are chemical messengers located inside of our body. Different hormones perform specific roles inside our body. Some of these hormones work quickly to start or stop a process, and some will continually work over the course for a long period of time to perform their necessary jobs.

On the other hand, the biology of orthodontic tooth movement comprises the study of cellular, biochemical and molecular phenomena occurring in the periodontal ligament and alveolar bone. The bone remodeling that occurs in the orthodontic movement is a dynamic process requiring coordinated cellular activities between osteoblasts, osteocytes and osteoclasts. All these activities are regulated by chemical mediators mainly hormones. Many authors have emphasized on the role of various endocrine and paracrine hormones on the orthodontic tooth movements through various literature review.

Discussion

The orthodontic tooth movement occurs by an inflammatory process involving osteoclasts, osteoblasts, neuropeptides, cytokines and alterations in innervation and local vascularization. Researchers have demonstrated that bone remodeling activity can be regulated by local factors such as the applied force, or by systemic factors such as drugs, hormones and vitamins.

When a force is applied to a tooth it results in a number of biophysical events such as Bone deformation, compression of periodontal ligament, tissue injury. These biophysical events lead to a biochemical reaction which bring about bone remodeling. Bone deformation and

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compression of periodontal ligament results in the release of extra cellular signaling molecules called first messengers. These first messengers include hormone such as PTH, local chemical mediators such as prostaglandin and neurotransmitter such as Substance P and vasoactive intestinal polypeptide (VIP).

The first messenger bind to receptors on the cell surface of target cells and initiate intra – cellular signaling. This leads to the formation of Second messenger which includes camp, cgmp, and calcium. This eventually leads to the initiation of bone forming cells (Osteoblasts) and bone resorbing cells (Osteoclasts).

Hormones are chemical substances that affect the activity of another part of the body (target site). Hormones serve as messengers, controlling and coordinating activities throughout the body. Upon reaching a target site, a hormone binds to a receptor, much like a key fit into a lock. Once the hormone locks into its receptor, it transmits a message that causes the target site to take a specific action. Hormone receptors may be within the nucleus or on the surface of the cell.

Effects of Hormones

- Stimulation or inhibition of growth
- mood swings / Emotional well being
- induction or suppression of apoptosis (programmed cell death)
- activation or inhibition of the immune system
- regulation of metabolism
- preparation of the body for mating, fighting, fleeing, and other activity
- preparation of the body for a new phase of life, such as puberty, parenting, and menopause control of the reproductive cycle
- hunger cravings
- Regulate the production and release of other hormones.
- Controls the internal environment of the body through homeostasis.

Sex Hormones

The association between the estrogen status and alveolar bone density in postmenopausal women were studied by the author Jeffrey B. Payne and they concluded that the E2, -sufficient women exhibited a gain in alveolar bone density, while the E2-deficient women exhibited loss in alveolar bone density.[7] To support this, various article was published in 2001, the author, Gustavo Hauber Gameiro et al, discussed the relation of certain systemic factors and orthodontic tooth movement. In this review article, they concluded that the estrogen and androgen decrease bone resorption and decreases tooth movement.[9]

In 2001, an investigation was conducted to find the association of estrogen and tooth movement and alveolar bone remodeling in rats by the author T. Yamashiro and T. Takano- Yamamoto. The study concluded that the increased rate of experimental tooth was due to decrease in estrogen levels.[10] This was followed by a study in 2002 by the author, Tanaka et al, where he evaluated the association between postmenopausal osteoporosis and tooth loss in rats and they concluded that the trabecular bone volume and trabecular number were lower due to reduced estrogen levels.[11]

In 2002, an investigation was done to determine the role of sex hormones in bone metabolism by N. Haruyama et al. In this study the role of estrogen and progesterone was determined during orthodontic tooth movement in rats and they concluded that the sex hormones are inversely proportional to orthodontic tooth movement. [12]

In 2007, S. G. Arslan et al evaluated the effects of estrogen deficiency on tooth movement in ovariectomized rats and they concluded that the amount and speed of movement was observed to be greater in ovariectomized rats due to decrease in estrogen levels.[18]

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According to X. Xu et al, estrogen binds with the receptors in periodontal tissue, regulates the remodeling of alveolar bones, promotes bone formation, and inhibits bone resorption. The estrogen levels are lower during menstruation and the luteal phase, and reaches the highest during ovulation. Orthodontic therapy should be planned according to the menstrual cycle since tooth movement, under the application of force, is faster during low estrogen levels.[21]

In 2011, Irin Sirisootorn et al**,** studied the amount of tooth movement and orthodontically induced root resorption in ovariectomized rats and they concluded that the tooth movement in the ovariectomized rats were more rapid due to decrease in estrogen levels[22].In 2012, Ssusilo et al, conducted a study that was aimed to evaluate the differences of orthodontic tooth movement during menstrual and ovulation cycle in five women and they concluded that when estrogen levels decline as in menstruation, tooth movement would increase, whereas when estrogen level increase as the time of ovulation, tooth movement would decrease[30]

According to Wang Bin et al in 2014, where they evaluated the role of estrogen on orthodontic tooth movement in Female patients. They concluded that the tooth movement was increased during the menstrual phase due to decrease in estrogen levels.[32] In 2015, a study was conducted to evaluate the effect of female and male steroid sex hormones on tooth movement in rats by N. Haruyama et al and concluded that the orthodontic tooth movement was accelerated in in experimental groups due to the decrease in estrogen, progesterone and testosterone.[33]

The articles that support “sex hormone deficiency can cause accelerated tooth movement” was done in 2016 and 2017 by Mackie et al and Dr. Khalid Ashraf et al where they discussed the effect of estrogen on orthodontic tooth movement in rats and concluded that the absence of estrogen and progesterone can significantly accelerate orthodontic tooth movement [35,37]

The recent article that was published in 2024 by Peruga et al, concluded that by quantifying changes in tooth mobility within the alveoli during the menstrual cycle in women undergoing orthodontic treatment, it was possible to determine that female sex hormones influence the effectiveness of orthodontic treatment [45].

Various studies were conducted to investigate the effects of sex hormones on orthodontic tooth movement. The studies have confirmed that increase levels of sex hormones result in decrease orthodontic tooth movement. Therefore, this methodology can be used in clinical practice to accelerate orthodontic tooth movement during menstrual phase as the levels of sex hormones were declined during this period.

Prostaglandin

In 1982 a study was conducted by Yamasaki et al to find the relation of prostaglandin and orthodontic tooth movement in monkey and they concluded the rate of orthodontic tooth movement doubles in area with higher amount of prostaglandin.[2] This was followed by a study in 1984 by Yamasaki et al where he demonstrated the effect of prostaglandin in clinical cases for orthodontic tooth movement and they also supported that the PG hormone can accelerated the orthodontic tooth movement.[3]

In 1995, an investigation was done to determine the role of prostaglandin E2 (PGE2) on the rate of tooth movement and the amount of root resorption in rats by Leiker et al and this study showed that the injection of exogenous PGE 2 enhances the amount of orthodontic tooth movement.[6]

In 2004, a study was done to evaluate the relation of prostaglandin and orthodontic tooth movement in rats by

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Chung – Fu Chao et al and they concluded that an increase in osteoclastic activity and increased orthodontic tooth movement on the site of Prostaglandin injection.[13] In 2014, an author, Kale Et Al, evaluated the effects of prostaglandinE2 and 1, 25-dihydroxycholecalciferol (1, 25-DHCC) on orthodontic tooth movement in rats. And they again proved that both PGE2 and 1, 25-DHCC enhanced the amount of tooth movement significantly when compared with the control group.[14]

In 2012, an investigation was done to find the effects of prostaglandin E2 (PGE2) administered by different methods on orthodontic tooth movement and bone in rabbits by Cağlaroğlu and Erdem and he concluded that the submucosal and intraligamentous PGE2 administration significantly increases orthodontic tooth movement and bone metabolism, but the intraligamentous route is found to be more effective.[26] In 2015, a study done by Massoud Selfi et al to investigate the effect of Thyroid Hormone, Prostaglandin E2, and Calcium Gluconate on orthodontic tooth movement and root resorption in rats and they also concluded that the combination of thyroxine and prostaglandin E2, would decrease the root resorption and increase the rate of orthodontic tooth movement in rats.[34]

A recent study done by Illahi et al[46] in 2024 on healthy human and animal subjects undergoing active orthodontic treatment with fixed appliances have been studied to evaluate the effects of biologic agents on tooth movement. Prostaglandins, whether applied locally or systemically, have been used to accelerate the process. The results showed that prostaglandins can significantly increase the rate of tooth movement. Two main methods have proven effective: first, local injections of PGE2 at doses between 0.1g and 1.0g; second, oral administration at an optimal dosage of 10–25g per kilogram of body weight per day. From the above-mentioned studies; it's clear that the increased levels of prostaglandin hormone can result in accelerated orthodontic tooth movement.

Thyroid Hormone

According to Seunghye Kim et al, there was a possible relationship between thyroid hormone and the rate of orthodontic tooth movement. He discussed a case of a 11-year-old girl, who showed sudden increase in orthodontic tooth movement of the impacted canine at certain points, which coincided with the hyperthyroid period. It shows a possible relationship between thyroid hormone and the rate of orthodontic tooth movement.[20] In 2014, a study was done to investigate the effect of Thyroid Hormone, Prostaglandin E2, and Calcium Gluconate on Orthodontic Tooth Movement and Root Resorption in Rats by Massoud Selfi et al where he concluded that the combination of thyroxine and prostaglandin E2, would decrease the root resorption and increases the rate of orthodontic tooth movement in rats.[34]

According to the study of Shirazi et al, thyroid hormone administration in rats not only increased the speed of tooth movement, but also reduced the extent of root resorption.[9] Loberg & Engstrom, also reported a protective effect of thyroxin on root resorptive lesions, induced by the application of orthodontic forces.[9] Vasquez et al showed that animals treated with thyroid hormones (intra-peritoneal or oral) had significantly less force- induced root resorptive lesions compared with a control group.[9] In 2021, Berry et al,[47] conducted a systematic review on the effects of thyroid hormones on orthodontic tooth movement. They concluded that the impact of thyroxine administration on tooth movement and orthodontically induced inflammatory root resorption in animal models remains unclear.

The authors suggested that the application of thyroid hormones may have a protective effect on the root

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surface either during orthodontic treatment and accelerates orthodontic tooth movement.

Parathyroid Hormone

In 1972, a study was done find the role of parathyroid hormone and cortisone acetate during orthodontic tooth movement in cats by Zee Davidovitch et al and they concluded that the parathyroid group had significantly higher orthodontic tooth movement.[1] In 2013 , another study was done by FAN LI et al, to investigate the effect of parathyroid hormone on orthodontic tooth movement in rats and they concluded that the tooth movements were significantly increased when the parathyroid hormone increases.[29]

In 2014, N. Watted et al**,** discussed the role of PTH on orthodontic tooth movement. Orthognathic surgery, increased tooth movement and can be used for postsurgical orthodontic tooth movement. In this article it was suggested that the increase parathyroid hormone (PTH) and calcium metabolism was the major cause. This study concludes that increased facility of orthodontic tooth movement immediately post-surgery while no association was found with surgery-related altered levels of PTH and calcium.[31]

In 2021, Sultan et al.[48] conducted a systematic review and found that, although most studies suggest parathyroid hormone (PTH) may have a favorable effect on orthodontic tooth movement (OTM), many of these studies had several sources of bias. To better determine the efficacy of intermittent PTH administration in enhancing the rate and stability of OTM, well-designed animal studies with minimal bias and long-term clinical trials are needed.

Authors suggest that the increased levels of PTH can results in accelerated orthodontic tooth movement. These results indicate that the orthodontist should be attentive to patients treated with increased levels of PTH, for example in cases with osteoporosis.

Calcitonin

In 2016, Dr. Sourabh Jindal et al, discussed the role of hormones in orthodontic tooth movement. In this review article the role of growth hormone, thyroid hormones, PTH, Estrogen, Vitamin D, Calcitonin were evaluated. Estrogen inhibits cytokine production which is the prime factors for bone resorption and therefore reduces tooth movement. PTH stimulates bone resorption by increasing directly the number of osteoclasts. Locally injected PTH induces local bone resorption. Thyroid hormone increases the rate of bone remodeling and bone resorption and increases tooth movement and decreases tooth resorption. Vitamin D increases the rate of bone remodelling and increases bone resorption and increases tooth movement.[36]

In 2020, Ali et al.[49] conducted a study on the effects of calcitonin on post-orthodontic relapse in rats. They found that the relapse ratio was significantly reduced in the three-dose calcitonin group (28%) compared to the single-dose group (34%) and the control group (46%). This reduction was accompanied by a non-significant increase in the number of osteoblasts and bone area, as well as a non-significant decrease in the number of osteoclasts in the three-dose group. However, immunohistochemical expression of RANK, RANKL, and OPG showed no statistically significant differences at the end of the relapse period

Vitamin D

In 1951, a study was done to investigate the effect of local application of Vitamin D3 on tooth movement in rats by T. Takano-Yamamoto et al and they concluded that the 1, 25- (OH)2D3 showed an increase in osteoclastic activity hence greater tooth movement. [5]

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According to Kale et al and Hussain et al, 1, 25-DHCC enhanced the amount of tooth movement significantly. In 2013, Ghada Nimeri et al discussed the role of hormones in orthodontic tooth movement. In this review article, PTH, vitamin D and relaxin were evaluated. PTH stimulates bone resorption by increasing directly the number of osteoclasts. Locally injected PTH induces local bone resorption. The role of relaxin is known in the remodeling of soft tissue rather than remodeling of bone. It has been shown that it increases collagen in the tension site and decreases it in compression site during orthodontic movement. Therefore PTH, Vitamin D and shows promising results whereas relax in does not accelerate tooth movement, but increase the tooth mobility.[25]

In a 1996 study by Baran and colleagues, rats treated with vitamin D presented more bone formation on the pressure side of the periodontal ligament after application of orthodontic forces. [9] In 2004**,** Kawakam and Takano-Yamamoto, also observed an increase in the mineral appositional rate on alveolar bone, after the application of orthodontic forces in rats. They suggested that local application of vitamin D could intensify the re-establishment of supporting tissue, especially alveolar bone, after orthodontic treatment. [9]

In 2024, a review article by Brijesh Kuar Singh[50] suggested that the majority of studies found locally injected calcitriol to be clinically effective and cost-efficient in reducing overall orthodontic treatment time. It was also observed that calcitriol initially increased osteoclastic activity, followed by a rise in osteoblastic activity. These findings were supported by both laboratory-based studies and questionnaire-based assessments related to Vitamin D.

Some studies have shown that the Vitamin D can increase the orthodontic tooth movement. However, other studies have shown that Vitamin D inhibits orthodontic tooth movement. Therefore, more studies have to be conducted for a better clarification. [9]

Relaxin

In 2004, a study was conducted to evaluate the role of human relaxin on orthodontic tooth movement by ZI JUN IUL et al and they concluded that the administration of human relaxin may accelerate the early stages of orthodontic tooth movement in rats.[14] However, a study conducted by Monica S. Madan et al, studied the role of relaxin on orthodontic tooth movement in rats and they concluded that the human relaxin does not accelerate orthodontic tooth movement in rats. [19]

According to Ghada Nimeri et al, PTH stimulates bone resorption by increasing directly the number of osteoclasts. Locally injected PTH induces local bone resorption. The role of relaxin is known in the remodeling of soft tissue rather than remodeling of bone. It has been shown that it increases collagen in the tension site and decreases it in compression site during orthodontic movement. Therefore PTH, Vitamin D and shows promising results whereas relaxin does not accelerate tooth movement, but increase the tooth mobility. [25]

Some studies have shown that the relaxin can increase the orthodontic tooth movement. However, other studies have shown that relaxin inhibits orthodontic tooth movement. Therefore, more studies have to be conducted for a better clarification.

Conclusion

For a long period of time patients have been seeking for effective and convenient orthodontic treatment. After years of research, it can be concluded that the fluctuation in hormone levels can affect the orthodontic tooth movement. Certain hormones such as estrogen, progesterone decline during the menstrual phase and

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hence activation of orthodontic tooth movement during this period can accelerate the orthodontic tooth movement. However, special care should be taken by the orthodontists that the declining levels of estrogen and progesterone can lead to osteoporosis and this is mainly seen after menopause.

Orthodontists have long been observed that teeth move at different rates, and that individuals have different responses to treatment. Some of these differences are caused by changes in bone remodeling induced by drugs and systemic factors. Therefore, clinicians should pay careful attention to the medications being used by their patients and medical history, so that the best therapeutic strategy – including force control and appointment intervals – can be selected for each case.

Abbreviations

OTM- Orthodontic Tooth Movement

PDL – Periodontal Ligament

PTH- Parathyroid Hormone

1, 25-DHCC -1, 25-dihydroxycholecalciferol

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