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Effect of Low- level laser therapy (LLLT) on Orthodontic Tooth Movement - Cellular Level

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Abstract

Low-level laser therapy has been used to stimulate the orthodontic tooth movements (OTM). Low level laser therapy has biostimulatory effects. In the last decade, researchers have attempted to determine the affect of Low level laser therapy on the pathways and cells directly associated with orthodontic tooth movement. The results of studies on the rate of tooth movement are controversial. While the majority of published research outcomes indicate an increase in the rate of tooth movement after laser therapy compared to controls, but others reported no difference or even indicated the inhibitory effect of laser therapy on the rate of tooth movement. Most of the studies reported the effect of the LLLT on rate of orthodontic tooth movement but only few have dealt with the underlying mechanism of action of low- level laser therapy on cells involved in orthodontic tooth movement. The present paper discusses the effect of low level laser therapy on orthodontic tooth movement at cellular level.

Introduction

Orthodontic tooth movement occurs in the presence of a mechanical stimuli will cause changes in the microenvironment around the PDL due to alterations of blood flow, leading to the secretion of different inflammatory mediators such as cytokines, growth factors, neurotransmitters, colony-stimulating factors, and arachidonic acid metabolites. As a result of these secretions, remodeling of the bone occurs [1,2]. Bone remodeling involves resorption of bone on the pressure site and bone formation on the tension site [3]. Low level laser therapy has biostimulatory effects [4]. It stimulates the on-going biological process in tissue and has been found to be effective in modulating cell activity and production of endogenous molecules, which are also involved in orthodontic tooth movement [5-7].

In the last decade, researchers have attempted to determine the affect of LLLT on the pathways and cells directly associated with orthodontic tooth movement. The results of studies on the rate of tooth movement are controversial. While the majority of published research outcomes indicate an increase in the rate of tooth movement after laser therapy compared to controls [8-15], but others reported no difference [16-18] or even indicated the inhibitory effect of laser therapy on the rate of tooth movement [19]. Most of the studies reported the effect of the LLLT on rate of orthodontic tooth movement but only few have dealt with the underlying mechanism of action of LLLT on cells involved in orthodontic tooth movement.

How does LLLT work?

Effect of LLLT is photochemical not thermal. Response of a cell to LLLT occurs by absorption of light by photoacceptor molecule also termed as chromophores [20,21]. Cytochrome C oxidase is a key photoacceptor of light in the far-red to near-IR spectral range [22]. Cytochrome C oxidase is an integral membrane protein of mitochondria that contains four redox active metal centers. The Excitation of this molecule with light energy accelerates the rate of electron transfer [23] and in turn increases the capacity of mitochondria to generate ATP [20,24-26]. Increased ATP results in increased energy available for that cell's metabolic processes.

Low level laser therapy's effect on main cellular components involved in orthodontic tooth movement: In the last few decades, researchers have attempted to determine the affect of LLLT on the biological pathways involved in orthodontic tooth movement. Some authors believe that LLLT induces osteoblasts proliferation (in vivo studies, [27-28] and in vitro studies [29-38]. Which is responsible for the accelerated tooth movement. However, according to other researchers, bone resorption is the rate-limiting step in tooth movement [36]. Therefore, any procedure which has the potential to increase osteoclastic activity may increase the rate of orthodontic tooth movement. Recent studies highlight enhanced osteoclastic activity after low level laser therapy in vivo [39-44] and in vitro [45].

Low level laser therapy's effect on osteoclast factors: The control mechanism of bone turnover is the OPG/RANKL/RANK system which is also recognized as the final mediator of osteoclastogenesis [46,47]. Researchers have sought ways to determine which member(s) of the system is/are affected by LLLT.

LLLT Effects On RANK and RANKL

Activation and maintenance of osteoclastic activity is under control of binding of RANKL with RANK. When RANKL dock with RANK, preosteoclasts differentiate and become osteoclasts. Studies have observed greater number of RANK and RANKL positive cells in laser treated groups than in both the non-irradiated. [8,40,45]

LLLT Effects on OPG

Osteoprotregrin (OPG) competes with RANK for the binding of RANKL. OPG decreases the differentiation and activation of osteoclasts. Fujita et al. [8] found that the level of OPG expression between the laser and control group did not vary [8]. Kim's group reported a significant increase of the cytokine with LLLT application. But they also noted that magnitude of OPG upregulation was not as great as it was for RANK. Because the OPG to RANK ratio was skewed in favor of RANK, the team observed a net increase in osteoclastic differentiation and activation [40].

LLLT Effects on other Osteoclast Factors

Dozens of cytokines, hormones, and peptides have been proven to play a role in bone turnover. A review of the literature yields a number of reports indicating how some of the factors involved in osteoclast regulation may be affected by LLLT [45-61].

LLLT and Transforming Growth Factor Beta 1

Transforming Growth Factor Beta 1 (TGF-β1) is integral in the differentiation and in maintaining the function of osteoclasts [68]. Research teams have discovered that sufficient expression of the polypeptide upregulates RANKL levels in the absence of osteoblasts, while excessive amounts of TGF-β1 in the presence of osteoblasts decreases RANKL upregulation [66-68]. Two research teams recently demonstrated increased levels of TGF-β1 after LLLT in the oral cavity[69,70].

LLLT and Cyclooxygenase 2 (Cox-2)

Cyclo-oxygenase is the rate-limiting enzyme in the conversion of arachidonic acid to prostaglandins, which is an essential component in osteoclast regulation. Several authors have shown that both Cox-2 and PGE-2 upregulate RANKL and inhibit OPG levels [71-73]. Matsumoto and colleagues demonstrated increased expression of Cox-2 after LLLT during bone repair in rats [20].

LLLT, fibronectin, PDL Collagen Remodeling

During orthodontic tooth movement the cells and components of the periodontal membrane matrix surrounding teeth undergo remodeling as mechanical forces induce biochemical changes in the microenvironment [74]. Fibronectin and collagen type 1 are important components of PDL organization. Fibronectin is synthesized in osteoblasts and fibroblasts. Fibroblast plays an important role in adhesion, growth, cell movement and differentiation during PDL reorganization [75]. It is particularly important in wound healing. Collagen type 1 is the main component in the PDL and is present in the high concentration in all fibers responsible for maintaining tooth position [76].

Increased expression of Fibronectin and turnover of collagen type 1 in LLLT groups compared to controls from day one of the experiment was found by Kim and his team of researchers. This may be due to the fact that Fibronectin induce the upregulation of RANKL which leads to the differentiation of osteoclast [77-78]. Fibronectin could, therefore, serve multiple purposes in PDL and bone turnover by assisting in phagocytic cells migration as well as increasing the presence of osteoclast like cells. The increased rate of collagen type 1 degradation and reorganization may also assist in minor increases in orthodontic tooth movement.

LLLT and Tissue Vascularity

Vascularization plays a key role in orthodontic tooth movement. Regardless of the type of bone resorption, whether frontal or undermining resorption, key cellular constituents arrive to the sites of bone resorption and deposition through the blood vessels. Along with nascent osteoclast, other phagocytic cells make their way through vessels and assist in not only bone but also tissue remodeling. Histological sections have revealed accelerated deposition of bony matrix as well as nascent vascularization on the laser experimental sides after seven days of healing when compared to controls [79,80]. Other researchers have shown increased vascularity after laser therapy in non osseous organs [81-83] as well as an increase in molecular factors related to vascular proliferation [84,85]. Additionally, some investigators have also found an upregulation of eNOS gene expression via PI3K signal pathway allowing for increased angiogenesis, endothelial migration, and revascularization [86]. The complete biochemical pathways through which LLLT influences osteoclast activity are not fully understood. Yet these researchers provide potential mechanisms whereby LLLT can influence osteoclast regulation by effecting enzymatic levels of TGF-β1, Cox-2, PGE-2, fibronectin, collagen turnover, and tissue vascularity preservation. These enzymes induce the expression or inhibition of members of the OPG/RANKL/RANK system and subsequently manipulate the differentiation, maturation, and maintenance of osteoclasts.

Conclusion

Orthodontic tooth movement is induced by mechanical stimuli and facilitated by the remodeling of the periodontal ligament and alveolar bone. The remodeling activities and the ultimately tooth displacement are the consequence of an inflammatory process. Vascular and cellular changes were the first events to be recognized. It is important for the clinicians to have knowledge of the effect of low level laser therapy on the cellular elements involved in orthodontic tooth movement. Orthodontically induced tooth movement associated with LLLT produced an increase in

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the vascularization, controls mechanism of bone turnover by regulating the OPG/RANKL/RANK system. Low level laser also regulates Transforming Growth Factor Beta 1, Cyclooxygenase 2 (Cox-2), fibronectin, PDL Collagen Remodeling. Further research is required to develop more solid scientific bases for the clinical use of LLLT and to describe the mechanism action of low power lasers as there are only a few studies in this field and different methodologies have been employed.

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