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Review Article

A Review Study on the Efficiency of Low-Level Laser Therapy for Orthodontic Tooth Movement

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Abstract

For pain relief and improved healing, low-level laser therapy (LLLT) has been used in orthodontic operations. According to recent research, LLLT may speed up the differentiation and proliferation of cells linked to the process of bone remodelling and slow down the movement of orthodontic teeth. In order to determine the best procedure for accelerating tooth movement in orthodontic pathways, this paper analyses earlier research on the biological effects of LLLT on orthodontic tooth movement in animals and human patients.

Keywords: Low Level Laser Therapy; Orthodontic Tooth Movement; Osteoblasts; Osteoclasts; Light Accelerated Orthodontia; Photobiomodulation Therapy

Introduction

Achieving quick tooth movement and cutting down on treatment time are crucial for the success of orthodontic therapy. Young people who seek professional care frequently lament the lengthy orthodontic treatment schedule, which frequently lasts for two to three years. Previous studies have found that 8-30% of individuals forego therapy because it is painful or takes too long [1]. To solve this problem, a number of strategies were used, including the application of intermittent resonance vibrations, medication injections of vitamin D, prostaglandins, and osteocalcin around the alveolar sockets, and surgical intervention like cortication.

Although these techniques were efficient in hastening tooth movement, they were costly, invasive, and had a number of unfavourable side effects, including pain and discomfort after injections and the need for repeated treatments to achieve the desired results. In the past, photobiomodulation therapy-a less costly, non-invasive method employing low level laser light therapy-became more important in orthodontics. Due to the use of low intensity light or laser in the red to near-infrared spectrum of around 600-1000 nm [2], it is also known as Light Accelerated Orthodontics (LAO) Therapy or Low-Level Light Therapy (LLLT).

In order to lay the groundwork for clinical applications on human beings, this study examines LLLT at the cellular level and in animal models.

Laser characteristics

"Light amplification by stimulated emission of radiation" is what the abbreviation "laser" stands for [3]. Lasers have a monochromatic spectrum and a relatively small bandwidth [4]. They can be supplied in a form of intermittent or continuous emission. Gated pulsed and free running pulsed modes are subsets of interrupted mode [5].

There are two types of laser treatment for dentistry: high power and low level. High power lasers can cut both soft and hard tissue with an output power of 1 watt. Their energy density per square cm ranges from several hundred to several thousand watts. along order to lessen discomfort and enhance bone absorption along the length of the mid-palatal sutures during expansion, low level laser treatment (LLLT) is employed in orthodontics. As the first widely utilised commercial laser, He-Ne lasers with a wavelength of 632.8 nm have been employed in the majority of the primary investigations on LLLT [6]. The material that the laser is shone upon, the laser wave length and energy density, energy density in the 0.5-4 J/cm^2 range [7], maximum infrared laser penetration in bloodless tissues, and energy density in the 0.5-4 J/cm2 range are all related to the penetration of LLLT in a tissue. Factors including light intensity, power output, power density, total irradiation, and energy density are crucial in order to achieve the photo reactive characteristics of LLLT [8]. According to Kujawa., *et al*., LLLT increases acetyl cholin-

esterase and internal protein storage, which lengthens cellular life span [9].

Principle of LLLT

Photobiomodulation treatment is based on the Arndt-Schulz law [10], which states that low concentrations of any chemical or medication have a stimulating impact whereas greater amounts have an inhibitory effect. By having cytochrome-c oxidase absorb photons in the mitochondria, modest dosages of this treatment drive ATP synthesis and the production of negligible quantities of reactive oxygen species [11]. This anabolic effect quickens tooth movement by promoting the growth and differentiation of several cell lineages, such as osteoblast, osteoclast, fibroblasts, and PDL cells. Numerous studies have examined the efficiency of photobiomodulation treatment in relieving pain and speeding orthodontic tooth movement, but the findings have been inconsistent due to the distinct uniform features of each type of laser used and the laser settings [12].

Low level laser therapy's (LLLT) biostimulatory effects

The laser may be utilised in a variety of biomedical applications, however it is most frequently employed with optical tools like fluoroscopy and high resolution coherence tomography. It triggers light-induced chemical processes like photobiostimulation or photobiomodulation in receptive cells and tissues. The photothermal reaction that occurs when the laser's light energy interacts with target tissues causes photoablation, which leads to the destruction of the tissue. While the low-level laser is utilised to have photochemical impacts on the tissues, the high-power laser initiates photomechanical interaction. Red and infrared light are used in low-level laser therapy (LLLT) to improve soft tissue wound healing and to lessen pain and inflammation [14].

The cellular mechanism of LLLT

Red and infrared light wavelengths that activate the electron receptor chain in mitochondrial membranes are what cause the majority of LLLT's biological effects. Figure 1 lists the primary mechanisms [15] through which it functions.

The effects of the LLLT particularly on osteoblasts, osteoclasts, and fibroblasts are more of interest in regards to orthodontic tooth movement.

Effect of LLLT on Various studies have been conducted to show the effects of low level laser therapy on various periodontal cells and are discussed in the given table (Table 1).

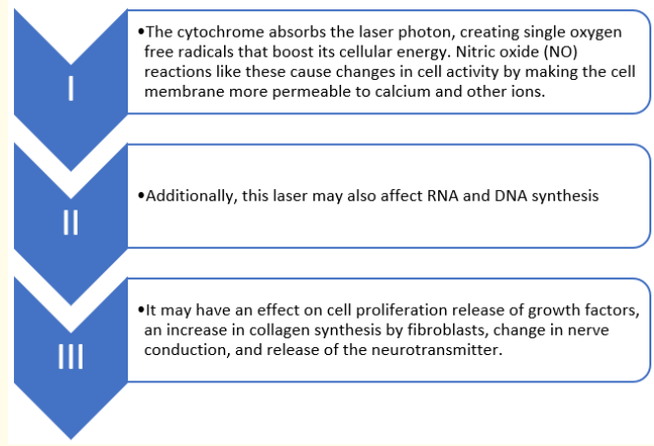

Figure 1: The cellular mechanism of LLLT.

Cell type	Studies conducted by	Effect of LLLT
Osteoblast	Dörtbudak., *et al.* [16]. and Grassi., *et al*. [17]	Examined the pulsed diode soft laser's (LLLT) in vitro biostimulatory effects on osteoblasts. They discovered that LLLT caused G2/M arrest on the cell cycle, enhanced bone matrix synthesis, and boosted DNA replication and proliferation. Further stimulating the rate of development and differentiation of human osteoblast-like cells, LLLT also increased the osteogenic capacity of growth-induced cells.
Osteoclasts	Aihara., *et al.* [18] Fujita., *et al.* [19] Xu., *et al.* [20]	showed that LLLT enhanced osteoclast development and activation via RANK expression by using a Ga-Al-As semiconductor laser on rat osteoclast precursor cells *in vitro.* Observed that LLLT in vitro promoted the differentiation and activation of osteoclasts due to increased c-fms gene expression and RANK/RANKL. They have shown that LLLT indirectly inhibited osteoclast differentiation by downregulating the RANKL: OPG mRNA ratio in osteoblasts and promoting proliferation and differentiation of osteoblasts, thus contributing to bone remodeling.
Fibroblast	Soudry., *et al.* [21]	It has been shown that LLLT *in vitro* increases the proliferation and expression of HGF, bFGF, IGF-1, and IGFBP3 in HGF.

Table 1: Effect of LLLT on various periodontal cells.

Mechanism of action of lasers in orthodontic tooth movement

- When orthodontic pressures are applied to teeth, osteoclasts and osteoblasts are increased on the compressed side and the traction side, respectively, causing tooth movement. In physiologic remodelling, resorption and deposition happen evenly, and the bone mass stays the same. Because osteoblasts are in charge of causing the osteoclastic cells to proliferate and differentiate, the pace of remodelling is linked to osteoblast activity. TNF (tumour necrosis factor) and RANKL [22] (receptor activator of nuclear KB ligand) are two examples of these factors. Low-level laser therapy can speed up tooth movement during orthodontic treatment by causing the periodontal ligament's RANKL to rise. Pre-osteoclast-like cells were examined by Aihara., *et al*. [23] to determine the level of RANK following radiation *in vitro*. When compared to the control group, the laser treatment group had greater levels of RANK and RANKL expressed by RT-PCR and immunohistochemical staining. RANK/RANKL levels were also assessed by Kim., *et al*. [24] utilising 2 immunohistochemistry analyses. From the start of the trial, they were aware that RANKL was present in both the laser therapy and control groups, but from the start to the finish, RANKL levels were higher in the laser group.
- The PDL area contains large quantities of type I collagen fibres, and tooth movement requires an increase in fibre turnover. The mesenchyme of the PDL is covered with fibronectin, which promotes fibrolast proliferation and differentiation as well as enhanced synthesis of type I collagen and fibronectin during OTM. GaAlAs low level laser treatment with an output power of 96 mw and a wave length of 808 nm has been demonstrated to raise the levels of fibronectin and type I collagen in response to mechanical stress. When early irradiations were used to treat rat calvarial cells, it was discovered that bone nodule development happened more frequently than when later treatments were used. According to Nakyamada., *et al*. [25] fibronectin has a significant role in bone and PDL turnover because it may stimulate the up-regulation of RANKL, which results in osteoclastic differentiation.
- On the traction side, low-level laser treatment can promote osteogenesis and boost bone density [26].
- Osteoclastic progenitors are encouraged to multiply and their ability to differentiate into adult osteoclasts is influenced by the macrophage-colony stimulating factor (M- CSF). Low-level laser treatment may stimulate osteoclastogenesis, which may cause tooth displacement, and M- CSF on the squeezed side. Photoreceptors of the mitochondrial respiration chain stimulate cells in a way that causes them to respond. OTM relies heavily on vascularization since both frontal and undermine resorptions need blood vessel supply. Garavello., *et al*. [27] used a low power intra cutaneous laser for 14 days after drilling a hole in the rat's tibia. Seven days after radiation, histologic samples from the laser group exhibited more osseous matrix deposition than those from the control group. Additional results show enhanced vascularity following laser treatment in non-osseous tissue as well as increased molecular elements linked to vascular proliferation.

Animal studies of LLLT

In several animal experiments, the effects of laser light on the movement of orthodontic teeth have been investigated. Kawasaki [28] and Shimizu reported the results of the first study ever carried out to determine the impact of LLLT on animal tooth movement in 2000. In rats, they used a closed coil spring to provide a mesial strain of 10 g, and for three minutes, they used a Ga-Al-As diode laser with a wavelength of 830 nm and a power of 100 mW. The irradiation procedure was developed based on earlier research that showed the laser had osteogenic effects on rats during maxillary expansion [28] and throughout the process of bone repair in tooth extraction sockets. The study also found a substantial increase in bone production on the tension side and an increase in the quantity of osteoclasts on the pressure side, as well as a 1.3-fold increase in tooth movement in the irradiation group. Except for one study that concentrated on maxillary incisors, most research on rats between the ages of 6 and 12 weeks has focused on the upper first molars. The majority of these research made use of Ga-Al-As diode lasers that emitted infrared light at wavelengths between 780 and 830 nm.

A recent study by Duan., *et al*. [29] sought to examine the variations in tooth movement rates while employing continuous wave lasers as opposed to pulsed wave lasers.

Later, the study by Kim., *et al*. [30] on beagle dogs using pulsed wave laser showed an accelerated influence on the pace of tooth movement. The examinations by Marquezans., *et al*. [31] revealed that there were no significant variations in the pace of tooth movement versus the control group, in contrast to most studies that reported an increased speed of tooth movement in rats by laser irradiation. The use of higher orthodontic pressures and the use of laser on older aged (70-120 day old) rats were the two key variations in these experiments. This demonstrated that the effects of laser radiation can vary based on age and intensity.

Goulart., *et al*. [32] split-mouth double-blind research in dogs examined the impact of laser treatment on orthodontic movement speed in relation to irradiation dose (5.25 J/cm2 and 35.0 J/cm2 irradiation groups). Orthodontic mobility was shown to be accelerated in the 5.25 J/cm2 dose group while being slowed down in the 35.0 J/cm2 group. According to this postulation, it may be possible to modify the mode of laser therapy in order to control the speed of tooth movement.

Clinical studies of LLLT

According to the findings of animal experiments, an adequate quantity of energy would speed up tooth movement, while an excessive amount would have an inhibitory impact and an inadequate amount would have no effect. The appropriate type and wavelength of laser for biostimulation, a clear LLLT application protocol, a difference in tooth movement rate, and compliance with the requirements for a randomised controlled trial (RCT)/split-mouse design with control and/or placebo group have all been demonstrated in human studies. The first study to look at how LLLT affects human orthodontic tooth movement was done by Cruz., *et al*. [33]. Ga-Al-As diode laser with a wavelength of 780 nm was used in the split-mouth investigation, and a total of 2.0 J (5 J/cm2, irradiated 10 times for 10 s) of radiation was applied. The investigation was done in relation to the animal experiment by Luger., *et al*. [34] which sought to find the ideal laser dosage. The study found that the experimental group's rate of tooth movement increased by 34%.

Thereafter, other studies were conducted to look at how lasers affected human participants' tooth movement. The majority of research used a Ga-Al-As diode laser that emits infrared light with a wave length of 780-810 nm, and they carried out split-mouth tests with 150 g of canine retraction force after removing the upper first premolar. Although every study showed that using a laser increased the speed of tooth movement, there didn't seem to be a noticeable difference between the laser group and the control group in the study carried out by Limpanichkul., *et al*. [35]. According to the scientists, this outcome resulted from insufficient laser dosage, which did not speed up tooth movement. The "18.4 J" of energy that Limpanichkul., *et al*. [35] employed was determined to be higher than the "2.0-8.0 J" range of energy levels other research had utilised to establish an improved tooth movement in human participants after taking the findings of other investigations into account.

The Youssef., *et al*. [36] investigation discovered that when an 8.0 J Ga-Al-As diode laser was irradiated, tooth movement occurred twice as quickly while causing 70% less discomfort.

Using 80g of orthodontic force to retract the maxillary lateral incisor, Genc., *et al*. [37] evaluated the rate of orthodontic tooth movement and the amount of nitric oxide in the gingival crevicular fluid (GCF). In this work, orthodontic tooth movement was significantly accelerated by 20-40% using a 2.0 J (0.71 J/cm2, 10 times of irradiation, for 10 s each) laser. The nitric oxide level of GCF did not, however, alter statistically significantly throughout orthodontic treatment, according to their research.

Combining the results of the earlier clinical trials, it was discovered that applying a continuous wave of 5-20 J/cm2, 2.0-8.0 J with the laser's tip in contact with the gingival surface accelerated the pace of orthodontic tooth movement.

Discussion

In both animal experiments and clinical trials involving human participants, the impact of laser light lithography (LLLT) in accelerating tooth movement is still debatable. Several additional papers have shown a zero impact or even an inhibitory effect, despite the fact that the bulk of the data have shown favourable benefits. The wavelength, power, spectrum area, dosage, application frequency, and exposure period of the laser all affect the effects in different ways.

According to the Arndt-Schulz rule, tiny dosages of each drug stimulate, whereas moderate levels inhibit, and excessive doses kill. Setting the ideal laser dose towards the therapeutic window is crucial since an insufficient dosage will have no impact while an excessive dosage may hinder tooth movement.

There are still a number of problems with the use of LLLTs, such as the high cost of the laser equipment, the length of time required to apply the laser, and the necessity for educated human resources. To establish a "gold standard" for the clinical use of LLLT, further clinical trials are required.

Conclusion

By raising the levels of RANKL in the PDL and M-CSF, low level laser therapy (LLLT) can quicken the pace of tooth movement during orthodontic treatment. On the traction side, they can also promote osteogenesis and boost bone density. Additionally, it has been observed that LLLT increases acetyl cholinesterase and internal protein storage, both of which contribute to longer cellular lifespan.

Understanding the biostimulation brought on by LLLT will enable regulation of tooth movement rate as well as more general therapeutic implications in orthodontics.

Future Scope

Although the literature points to the benefits of LLLT using a diode laser for speeding OTM and decreasing dental discomfort associated with OTM, more research on a larger sample size is required to better understand the processes generated by laser treatment during OTM. To evaluate the amounts of acetyl cholinesterase and internal protein storage observed during LLLT, more research must be done.

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