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REVIEW ARTICLE

Laser phototherapy in the treatment of periodontal disease. A review

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Abstract Many studies in the literature address the effect of low-power lasers in the management of pathologies related to periodontal tissues. Due to the lack of standardized information and the absence of a consensus, this review presents the current status of laser phototherapy (LPT) in periodontics and discusses its benefits and limits in the treatment of periodontal disease. The literature was searched for reviews and original research articles relating to LPT and periodontal disease. The articles were selected using either electronic search engines or manual tracing of the references cited in key papers. The literature search retrieved references on wound and bone healing, analgesia, hypersensitivity, inflammatory process and antimicrobial photodynamic therapy. Each topic is individually addressed in this review. The current literature suggests that LPT is effective in modulating different periodontal disease aspects in vitro, in animals, and in simple clinical models. Further development of this therapy is now dependent on new clinical trials with more complex study designs.

Keywords Low-power laser · Photodynamic therapy · Phototherapy · Periodontal disease

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Introduction

The increasing use of lasers in dentistry and medicine reflects the great advances in this technology during recent decades. In periodontics, the most commonly used are high-power lasers. CO_2, Nd:YAG and Er:YAG lasers have been used for calculus removal, osseous surgery and soft-tissue management, such as gingivectomy, gingival curettage and melanin pigmentation removal [1].

The use of low-power lasers (commonly named laser phototherapy, LPT) in periodontics, on the other hand, has been claimed to be of benefit for modulating both periodontal disease aspects and therapy side effects. Several studies have shown that processes such as inflammation, soft tissue and bone healing, and side effects such as postoperative pain and posttreatment tooth hypersensitivity, can be positively influenced by LPT [2–10]. Besides that, the association of low-power lasers with photosensitizers, the so-called "antimicrobial photodynamic therapy" (aPDT), can also be used for reducing bacterial contamination of periodontal pockets [11–13].

LPT is represented by red or near-infrared light used in a low intensity range, with the consequent biological effects

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attributed to nonthermal cellular events [14]. LPT has been studied as adjuvant to traditional treatment of periodontal disease [8, 15–19].

Although there are different possibilities for using LPT in periodontics, many clinicians are still not familiar with this therapy. In addition, the controversial results observed in the literature are frequently related to the lack of standardization when reporting irradiation parameters and inappropriate specification of dosimetry (power, beam area, time, dose, contact or defocused irradiation mode) [20].

Therefore, a review of the literature seems to be highly necessary to put the available information together and to present its current status, as well as to contribute to an evidence-based use of low-power lasers in periodontics and to its demystification.

Materials and methods

In view of the importance of low-power lasers in periodontics, a literature search was conducted. Firstly, the electronic databases searched included Medline-PUBMED and ISI Web of Knowledge search engines. Reviews of the literature and original research articles were selected. A comprehensive second search was performed by hand searching of relevant references, such as the *Journal of Oral Laser Applications*, the *Proceedings of SPIE*, and Congress Proceedings of The World Federation for Laser Dentistry (WFLD, formerly International Society for Lasers in Dentistry).

LPT in the periodontal inflammatory process

The chronic periodontal inflammatory process leads to the destruction of the periodontal ligament, and subsequently, to loss of alveolar bone. The latter is primarily mediated by osteoclasts and is triggered by the proinflammatory molecule prostaglandin E_2 (PGE_2) [3]. Some studies have analyzed the inflammatory aspects of periodontal tissue and have shown that patients who have undergone conventional periodontal treatment in combination with LPT show better results [21, 22]. It has been reported that LPT is able to reduce gingival inflammation and metalloproteinase 8 (MMP-8) expression when applied after scaling and root planing [8, 21, 23], as well as to reduce inflammatory cells on histology [22]. Ozawa et al. [18] showed that LPT significantly inhibits the increase in plasminogen activity induced in human periodontal ligament cells in response to mechanical tensile force. Plasminogen activity is capable of activating latent collagenase, the enzyme responsible for cleaving collagen fibres. LPT also effectively inhibits PGE_2 synthesis [17, 24]. In human gingival fibroblast culture, LPT significantly inhibits PGE_2 production that is stimulated by lipopolysaccharide through a reduction on COX_2 gene expression in a dose-dependent manner [17]. A decrease in PGE_2 levels in cultures of primary human periodontal ligament cells has also been found after cell mechanical stretching [24].

Nomura et al. [25] found that LPT significantly inhibits lipopolysaccharide-stimulated interleukin 1β (IL-1β) production in human gingival fibroblasts, and that this inhibitory effect is dependent on irradiation time. In an in vivo study, alterations in IL-1β concentrations in the periodontal pocket were not observed, although probing depth and both plaque and gingival indexes were reduced more on the laser side than on the placebo side [8]. Safavi et al. [26] evaluated the effect LPT on gene expression of IL-1β, interferon γ (IFN-γ) and growth factors (PDGF, TGF-β, bFGF) to provide an overview of the effect of LPT on their interactive role in the inflammation process. The findings of this study suggest an inhibitory effect of LPT irradiation on IL-1β and IFN-γ production and a stimulatory effect on PDGF and TGF-β. These alterations may be responsible for the antiinflammatory effects of LPT and its positive effects on wound healing.

An in vivo study conducted by Pejcic et al. [27] evaluated the effects of low-level laser irradiation treatment and conservative treatment on gingival inflammation. Authors reported a statistically significant improvement in gingival index and bleeding index after laser irradiation, especially over a longer period of time (3 and 6 months after irradiation). The results depended on the number of laser applications, and after the fifth application, a considerably antiinflammatory effect was achieved with the parameters considered. It was concluded that low-level laser irradiation (670 nm) associated with traditional periodontal therapy can be used as a successful physical adjuvant treatment, leading to better and longer-lasting therapeutic results.

The above-mentioned findings support the hypothesis that LPT can modulate the periodontal inflammatory process, especially through reducing PGE_2 release. The capacity of LPT to modulate inflammation seems not to be confined to a single mechanism, since different pathways in inflammatory modulation have been described. In summary, LPT influences the expression of COX_2 and IL-1β, as well as MMP-8, PDGF, TGF-β, bFGF and plasminogen. It is important to draw attention to the fact that LPT acts as a coadjuvant to traditional periodontal treatment, with no beneficial effect when used alone for this purpose [8, 10, 21–23].

LPT in wound healing

Periodontal wound healing is necessary when periodontitis and gingivitis, or trauma, have affected the composition and integrity of the periodontal structures. Therefore, several

processes, including inflammation and cellular migration, proliferation, and differentiation, are necessary for a successful repair [28, 29].

Several in vitro studies have shown that LPT at certain wavelengths may stimulate fibroblast proliferation when certain combinations of exposure parameters and power densities are used [6, 30–38]. At higher energy densities, no effect [31, 33] or even decreased proliferation has been reported [38, 39]. Therefore, Karu [40] suggested a "window-specificity" at certain wavelengths and energy densities, at which the positive effects of LPT can be expected.

The range of radiation doses at which stimulation of fibroblast proliferation has been observed is wide (0.45–60 J/cm^2). However, when considering studies using fibroblasts from the oral mucosa, gingival and periodontal ligament only, the range becomes narrow (0.45–7.9 J/cm^2; Table 1). For wavelengths between 660–692 nm and 780–786 nm, the radiation dose is restricted to 2 J/cm^2; for the range 809–830 nm, it is between 0.45 and 7.9 J/cm^2; and for 2940 nm, it is 3.37 J/cm^2 [6, 19, 30, 32, 34, 36–38].

Contradictory results can be found regarding the most efficient wavelength for promoting cell proliferation. However, stimulation of cell proliferation has been observed with both infrared lasers (λ=780, 809, 812, 830, 904 nm) [30, 33, 36, 37, 39] and red lasers [19, 30, 32–37, 39]. Observation of the parameters investigated up to now shows that not only the wavelength and the energy dose but also the power density are important for cell growth stimulation [41]. Azevedo et al. [34] tested two power densities (428.57 and 142.85 mW/cm^2) for the same dose (2 J/cm^2, λ=660 nm) and found that the lower power density caused higher stimulation.

The mode of exposure may also play a role in optimizing the stimulation. Multiple exposures with both infrared and red lasers (λ=809, 830 and 685 nm) have been shown to cause a significantly higher proliferation of periodontal ligament and gingival fibroblasts than a single exposure with the same energy density [6, 19, 37]. Currently, the evidence available fails to show any increase in the procollagen content and in the synthesis of collagen by human gingival fibroblasts after low-power laser irradiation [33, 42]. As regards clinical evidence, the current literature is not as extensive as it is for in vitro evidence. Clinical evidence of the effect of LPT on periodontal wound healing can be found for irradiation after scaling and root planing, gingivoplasty and gingivectomy, showing significantly better healing in the sites treated with laser, despite the different methods of measurement used [8, 16, 21, 43, 44]. In a split-mouth study in 20 patients undergoing gingivectomy bilaterally, Amorim et al. [16] measured the probing depth of the gingival sulcus before and after surgery. They observed that the laser irradiated sides (4 J/cm^2, λ=685 nm) had probing depths significantly lower than the control sides at 21 and 28 days after surgery. Another study showed that sites receiving LPT (4 J/cm^2, λ=588 nm) had significantly faster surface epithelization than control sites 3, 7 and 15 days after surgery [43]. Additionally, complete wound healing was achieved faster in sites receiving LPT (within 18–21 days) than in control sites (within 19–24 days). On the other hand, for irradiation with the same energy density (4 J/cm^2) and 660 nm wavelength, Damante et al. found no significant differences when comparing irradiated and nonirradiated control sites for all histomorphometric parameters quantified. In contrast to the other above-mentioned studies, the irradiation was performed in punctal mode and used a lower output power (15 mW) [45].

After scaling and root planing, adjuvant LPT was shown to significantly reduce gingival index, probing depth and gingival crevicular fluid volume (GCF) [8]. Similar effects were observed by the same group in 2007 [21], and they also observed the importance of light coherence in the improvement in periodontal wound healing. A laser with longer coherence (HeNe laser) was clinically compared with another with shorter coherence (diode laser). For irradiations with the same power density of 100 mW/cm^2, both plaque and gingival index were significantly reduced on the side treated with the HeNe laser. These results appear to show that coherence is an important parameter in light stimulation in vivo, an effect not observed in vitro [46]. Thus, further studies are needed to elucidate the exact mechanism by which each parameter influences the effects of LPT in vivo.

The use of stem cells in future tissue engineering and regenerative medicine to replace conventional therapeutic modalities has been the subject of growing interest in different areas [47–49]. These cells have self-renewing properties and are able to differentiate into one or many different specialized cell types under controlled in vitro conditions [49]. LPT is expected to significantly influence stem cell proliferation leading to improved tissue healing. Recent in vitro studies submitting stem cells to LPT using low energy densities have shown promising results [48, 50]. However, in order to determine whether this therapy can contribute to an optimal attachment and functional improvement in the cells following implantation and also reduce tissue healing time, future studies are needed to evaluate its effect on new bone formation following stem cell implantation in injured tissues.

The literature review showed several positive results of the use of LPT in improving periodontal wound healing both in vitro and in vivo [6, 8, 16, 19, 21, 26, 32, 34, 37, 38, 43, 44, 51]. In particular, the evidence from in vivo studies indicates that LPT may be beneficial in enhancing periodontal healing after gingivectomy, scaling, root planing and intrabony defect surgery [8, 21, 43, 52].

Table 1 Laser irradiation conditions and results observed in vitro after LPT stimulation of fibroblast proliferation

Cells	Reference	Wavelength (nm)	Laser	Dose (J/cm^2)	Power density	Irradiation time	Exposure	Proliferation
NIH/3T3 fibroblasts	[39]	632	HeNe	15	No info	No info	No info	Increase
				60	No info	No info	No info	Decrease
		780	Diode	7	No info	No info	No info	Increase
	[33]	904	GaAs diode	1 and 2 (3)	No info	No info	Two (6-h interval)	Increase
				2 and 3 (5)	No info	No info	Two (6-h interval)	No effect
BALB/c 3T3 clone A31 human fibroblasts	[31]	660	Argon dye	2.16	9 mW/cm^2	4 min	Single	Increase
				3.24	9 mW/cm^2	6 min	Single	No effect
Fibroblast cell culture isolated from CH3 mice	[35]	625	–	10	5 mW/cm^2	No info	Single	Increase
		635	—	10	5 mW/cm^2	No info	Single	Increase
		645	–	10	5 mW/cm^2	No info	Single	Increase
		655	–	10	5 mW/cm^2	No info	Single	Increase
		675	–	10	5 mW/cm^2	No info	Single	Increase
		810	Diode	10	5 mW/cm^2	No info	Single	Decrease
Fibroblasts from oral mucosa	[30]	812	GaAlAs	0.45	4.5 mW/cm^2	100 s	Single	Increase
Human gingival fibroblasts	[32]	670	–	2	1 W/cm^2	No info	Single	Increase
		780	–	2	5 W/cm^2	No info	Single	Increase
		692	–	2	3 W/cm^2	200 s	Single	Increase
		786	–	2	3 W/cm^2	40 s	Single	Increase
	[34]	660	GaAlAs diode	2	142.85 W/cm^2	14 s	Two (12-h interval)	Increase
				2	428.57 W/cm^2	4.8 s	Two (12-h interval)	Increase
	[36]	809	GaAlAs diode	1.9	No info	75 s	Single	Increase
				3.9	No info	150 s	Single	Increase
				7.9	No info	300 s	Single	Increase
	[37]	830	GaAlAs diode	3	84 mW (calculated 0.008 W/cm^2)	360 s	Single	No effect
				0.75		90 s	Three (24-h interval)	No effect
				1.5		180 s	Three (24-h interval)	Increase
				3		360 s	Three (24-h interval)	Increase
	[38]	2,940	Er:YAG	1.68	No info	No info	Single	No effect
				2.35	No info	No info	Single	No effect
				3.37	No info	No info	Single	Increase
				5	No info	No info	Single	Decrease
	[19]	685	Diode	2	No info	No info	Single	Increase
				2	No info	No info	Two (24-h interval)	Increase
Periodontal ligament fibroblasts	[6]	809	GaAlAs diode	1.9	No info	75 s	Single	Increase
				3.9	No info	150 s	Single	Increase
				7.9	No info	300 s	Single	Increase
				3.9	No info	150 s	Two	Increase
				3.9	No info	150 s	Three	Increase

LPT in bone healing

Regenerative periodontal therapy aims to predictably restore the tooth's supporting periodontal tissues, and should result in formation of a new connective tissue attachment and new alveolar bone [53]. Preclinical models have demonstrated periodontal regeneration following treatment with barrier membranes, different types of grafting materials or their combination [54–56]. Recently, it has been suggested that LPT may be indicated in combination with regenerative methods or even alone in order to stimulate bone repair in specific bone defects [52].

Some authors have investigated the effects of LPT on growth and differentiation of human osteoblast cells [57, 58]. Stein et al. [58] reported that LPT has a biostimulatory effect on human osteoblast-like cells during the first 72 h after irradiation. Histological studies using animal experimental models [59, 60] have also demonstrated that LPT can promote an increase in collagen fibres deposition, as well as in the amount of well-organized bone trabeculae after 30 days of induced-bone defect healing. It is suggested that LPT may accelerate the process of bone repair [59, 61–63] and/or cause increases in callus volume, especially in the early stages of haematoma absorption and bone remodelling [59].

The effects of LPT on the bone healing process in surgically created bone cavities were evaluated using a biochemical assay. The results indicated that LPT acts by affecting calcium transport during new bone formation [64]. These results corroborate the findings of Khadra et al. [65], in which bone defects showed significantly more calcium, phosphorus and protein after LPT in an experimental animal model.

For the treatment of deep intrabony defects, the use of barrier membranes and different types of grafting materials are usually indicated. In a study of the effect of LPT on the healing of bone defects associated with autologous bone grafts, bone remodelling was both quantitatively and qualitatively more evident in irradiated animals than in nonirradiated animals [9]. The association of matrix protein derivative with the LPT irradiation (during and after surgery) has shown a reduction in postoperative pain, which suggests that LPT may improve the effects of matrix protein derivative by reducing postoperative complications [44]. LPT biostimulation of bone tissue attachment to implant surfaces has also been reported. It has been shown that LPT influences the expression of osteoprotegerin, receptor activator of nuclear factor κB ligand and receptor activator of nuclear factor κB, and results in the expansion of bone cells metabolic activity [66, 67].

Pinheiro and Gerbi [68] studied the photoengineering of bone repair processes and suggest that the effects of LPT on bone regeneration depends not only on the total dose of radiation, but also on the irradiation time and the irradiation mode. Studies have shown that LPT has a positive biomodulatory effect on the healing of bone defects [52, 54–56, 69], and that the positive effect of this therapy is more evident when the tissue is irradiated intraoperatively, directly to the surgical bed, and in the early stages of bone remodelling [52].

LPT in analgesia/pain reduction

Postoperative pain control is an essential part of periodontal surgery routine. Postoperative pain may be a consequence of surgical trauma and release of pain mediators [70–73], and is usually reported to be most intense during the first hours following surgery, just after cessation of local anaesthesia [74].

LPT has been suggested as an alternative method for postoperative pain control. Compared to oral analgesics and nonsteroidal antiinflammatory drugs, LPT can be advantageous because the therapeutic window for its antiinflammatory action overlaps with its ability to improve tissue repair [75]. The mechanism by which LPT reduces pain symptoms is still not clear, even though many studies have shown some physiological changes induced by the interaction of the light with different cells [2, 32, 33, 76]. Some authors describe a possible stabilization of nerve cell membranes, probably due to the more stable conformation of the lipid bilayers induced by LPT, and the associated integral proteins of the nerve cell membrane, which have already been reported in the literature [77]. The enhanced redox systems of the cell and an increase in ATP production have also been shown to restore neuronal membranes and decrease pain transmission [78].

Although some studies have indicated that LPT affects the neurophysiological processes in intact peripheral nerves in the absence of inflammation, the current literature is not conclusive [2]. Evidence of the effects of LPT on injured nerve fibres is considerably more consistent, revealing positive effects of this therapy [79, 80].

Recent reviews by Parker et al. [7] and Bjordal et al. [2] have shown that low-power lasers can significantly reduce pain. Bjordal et al. reviewed several basic and clinical studies in which LPT was used in acute pain following tissue injury. This systematic review concluded that LPT can modulate the inflammatory process in a dose-dependent manner and that it can be titrated to significantly reduce acute inflammatory pain in the clinical setting. The authors confirmed that, in acute pain, the optimal effects of LPT can be achieved when it is administered in higher energy densities during the first 72 h in order to reduce inflammation, followed by lower dosages to the target tissue during the following days with the aim of promoting

tissue repair. Well-designed randomized controlled trials have also shown an effect of LPT doses well below those expected to induce biological responses [81, 82]. Thus, further clinical trials with adequate doses are still necessary to estimate the magnitude of the effect of LPT in acute pain, since high doses would be expected to be effective for pain relief but to overdose stimulatory mechanisms. On the other hand, low doses are related to biomodulation but less effective for acute pain.

Antimicrobial photodynamic therapy

The presence of bacteria in the gingival sulcus and periodontal connective tissues is a determinant factor in the development of periodontitis [83]. In areas of difficult access such as furcations, invaginations and concavities, the use of manual curettes or ultrasound is not enough to ensure the eradication of periodontal pathogenic bacteria. Likewise, antibiotic-resistant strains may also damage the efficacy of conventional periodontal treatment [84]. Based on these facts, alternative methods are being studied with the aim of achieving a more efficient therapy such as antimicrobial phytochemicals and light-activated killing [85].

The development of laser technology and the discovery of its significant antimicrobial effects have introduced this treatment modality as a possible coadjuvant to the treatment of periodontitis. Differently from high-power lasers, low-power lasers do not increase tissue temperature [86]. Thus, when used alone, the same antimicrobial effect as that of high-power lasers in periodontitis active sites cannot be expected [87]. The antimicrobial effect of low-power lasers is achieved by association with extrinsic photosensitizers, which results in the production of highly reactive oxygen species [88] that cause damage to membranes, mitochondria and DNA, culminating in the death of the microorganisms [89–91]. This is the process of aPDT, and its use is being increasingly studied with the aim of complementing the microbial reduction achieved by conventional mechanical periodontal therapy.

For the effective treatment of bacterial infectious diseases, it is paramount to have an adequate light source and a photosensitizer capable of binding to the targeted pathogen, so that photosensitization may occur in either subgingival or superficial oral tissues. Different light sources have been studied, such as light emitting diodes [92, 93], conventional light [94, 95] and low-power lasers [96]. There are several photosensitizers available for aPDT, but disinfection related to periodontopathogens generally indicates the use of cationic charged photosensitizers, such as toluidine blue, methylene blue and poly-L-lysine-chlorin-e6 conjugates [97, 98]. Plaque disclosure agents such as erythrosine and malachite green have also been addressed in in vitro studies as potential photosensitizers in aPDT [96, 99]. The interaction between the photosensitizer and microorganisms occurs within a few minutes, and this period (incubation or preirradiation time) must be respected before laser irradiation [88, 98].

Several in vitro studies have demonstrated that microorganisms related to periodontal disease such as *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans* (previously *Actinobacillus actinomycetemcomitans*), *Fusobacterium nucleatum*, *Prevotella intermedia* and *Streptococcus sanguis* are significantly suppressed by aPDT under different environmental conditions (pure cultures, pure or multispecies biofilms) [11, 13, 96, 100, 101]. Key virulence factors (lipopolysaccharide and proteases) present after the destruction of bacteria are also reduced by photosensitization [90, 102]. Moreover, some periodontal bacteria are capable of producing intrinsic photosensitizer (protoporphyrin IX) [85]. The so-called black-pigmented bacteria have been shown to be more susceptible to lethal photosensitization, even without the use of extrinsic photosensitizers [103–105].

More recently, in vivo studies have shown a decrease in bone loss and reduction of periodontal signs of redness and bleeding on probing after aPDT for the treatment of periodontitis [12, 106–108] and periimplantitis [109–112] in rat and dog models. These studies included healthy [12, 106–108], diabetic [113] and even immunosuppressed animals [114]. Clinical trials have shown the efficacy of this therapy as coadjuvant in the treatment of chronic [10, 115–117] and aggressive [118, 119] periodontitis and periimplantitis [120, 121], and even in treatment maintenance at follow-up session [122]. The photosensitization process is dependent on photosensitizer concentration, laser irradiation parameters and the microorganism species involved [11].

Antimicrobial PDT is considered a safe coadjuvant in nonsurgical treatment of periodontitis, as it has been proved to reduce the signs of inflammation and microbial infection without any harmful effects on adjacent periodontal tissues [12, 106, 123]. Its potential effect in improving wound healing has also been reported [98, 124]. The topical application of aPDT allows a local and specific action in the disease active site, with no effects on the microflora at other sites of the oral cavity, and reduced probability of side effects that are associated with the systemic administration of antimicrobial agents [11]. Additionally, the increasing bacterial resistance to antibiotics that frequently occurs in conventional therapy is highly unlikely to occur with aPDT, since it suppresses microorganisms in a short time by producing reactive oxygen species that interact with various cell structures and targets [89, 90]. Thus, repeated photosensitization of both antibiotic-resistant and antibiotic-susceptible bacteria seemed not to induce the selection of resistant strains [125].

Controlled clinical studies are still needed to better embed the use of aPDT in periodontists' clinical routine [126]. However, the in vitro and in vivo studies present in the literature indicate that aPDT may potentially become a successful antiinfectious procedure to be associated with conventional therapy in the management of periodontal disease [98, 127–129].

LPT in the treatment of dentin hypersensitivity

In periodontology, the removal of enamel or denudation of the root surface by loss of cementum and periodontal tissues following nonsurgical periodontal treatment are often associated with increased levels of dentin hypersensitivity (DH) [130–132]. Studies suggest that periodontal diseases play a role in the aetiology of this condition [133–135]. Numerous desensitizing agents have been clinically tested in an effort to find a means to alleviate the discomfort experienced by patients undergoing periodontal treatment [136]. The results have been variable and inconclusive due to the different methodologies employed, the variability in the subjective response and the influence of the placebo effect.

The advent of dental lasers has raised another possibility for the treatment for DH, and has become the object of research interest in recent decades. The effect of LPT on DH depends on many variables, such as the type of equipment used (low- or high-power lasers), wavelength and selection of parameters. However, contradictory results have been reported in the literature due to the lack of information related to the irradiation protocol used and the subjectivity of the evaluation of DH. The effect of high-power lasers in the treatment of DH has already been described. CO_2, Nd:YAG and Er:YAG are suitable lasers for the management of DH [5]. Their mechanism of action is related to the increase in tooth surface temperature. Following their use, it is possible to notice the complete or partial closure of dentinal tubules after recrystallization of dentinal surface.

Several mechanisms are proposed to explain the decrease in pain after LPT in DH. The positive effects are mainly attributed to the formation of tertiary dentin and the reduction in sensory nerve activity [137]. Although information on the neurophysiological mechanism is not yet conclusive, it is postulated that LPT mediates an analgesic effect related to the depolarization of C-fibre afferents. This interference in the polarity of cell membranes by increasing the amplitude of its potential action is capable of blocking the transmission of pain stimuli in hypersensitive dentin. Histological studies have shown that the formation of hard tissue is enhanced as a reaction of the dental pulp to laser irradiation [138, 139]. Matsui et al. [140] showed that hydroxyl generated by laser irradiation activates cell-signalling molecules such as G-protein, thereby promoting the formation of hard tissue by human dental pulp cells.

The promising results observed in in vitro studies stimulated the evaluation of LPT for the treatment of DH in vivo. Wakabayashi et al. [141] showed that a low-power diode laser was effective in 61 out of 66 patients. The results of Gerschman et al. [142] were also satisfactory and corroborated those of Wakabayashi et al. [141]. Groth [4] reported that the results with the low-power laser were significantly better, and established an irradiation protocol of three sessions with an interval of 72 hours between them. As these studies were performed with low-power lasers in the infrared spectrum, Ladalardo et al. [143] studied the influence of different wavelengths on pain reduction, and observed that the 660 nm red diode laser was more effective than the 830 nm infrared diode laser. Marsilio et al. [144] observed positive clinical results using a low-power laser in the red spectrum, with 86.53% and 88.88% pain reduction for 3 and 5 J/cm^2, respectively. Corona et al. [145] compared this same wavelength with fluoride varnish frequently used in the treatment of DH, and obtained better results with LPT. Also, it should be born in mind that the evaluation of treatments for DH is not a simple procedure due to the interference of the placebo effect, which is easily observed in clinical trials [146].

However, in spite of being speculative, the mechanisms proposed for the effects of LPT on DH reduction require serious consideration and new experiments may elucidate the cellular events involved in this process. Still, the positive results and the lack of serious side effects reported in the literature allow the statement that low-power lasers are an effective method for the treatment of DH, being a predictable, reliable and simple approach.

Final considerations

LPT has been increasingly studied due to its clinical effects including analgesia, modulation of inflammation and biomodulation. Although a few studies mention the possible placebo effect of LPT [146], several in vitro and in vivo studies have shown its efficacy when used as coadjuvant in traditional periodontal treatment. The clinical applications of LPT include periodontal inflammation modulation, improvement in wound and bone healing processes, control of postoperative pain and posttreatment tooth hypersensitivity, and microbial reduction when associated with an extrinsic photosensitizer. Despite the clinical observations, additional clinical trials are still needed to better elucidate these events and to provide the clinical outcomes of LPT in periodontics with a solid scientific basis.

The optimal laser parameters and the irradiation protocols are not fully described in the literature. The main

factors hindering their precise definition are the lack of standardization in the reporting of laser parameters and the great variety of research methodologies used. However, considering the high number of positive reports currently available in the literature, new in vitro investigations should no longer seek to confirm the effectiveness of LPT, but to determine its precise mechanism and to identify the ideal set of parameters and protocols for different clinical situations.

Although LPT seems to enhance clinical outcomes in periodontology, it is only reasonable to perform it in a decontaminated and calculus-free periodontium. This means that conventional periodontal treatment should not be discarded. LPT must be considered an adjuvant therapy, which may have additional benefit for a previously well-treated periodontal tissue.

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