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Laser-Assisted Biofilm Disruption and Its Role in Periodontal Tissue Regeneration

Aikebaier Kaiwusaer

Urumqi Foreign Languages School, Urumqi, China
Algieeee@outlook.com

Abstract. Laser interventions in periodontology combine targeted biofilm disruption, localized photochemical antimicrobial activity, and modulation of host repair pathways. NdYAG around 1064 nm couples to hemoproteins and penetrates millimeters to enable coagulation and contactless disinfection in vascularized pockets. ErYAG near 2940 nm is absorbed by water and hydroxyapatite and ablates mineral deposits while volatilizing extracellular polymeric substances. Diode lasers from 810 to 980 nm activate porphyrin photosensitizers for photodynamic therapy and provide antimicrobial action with low thermal burden. Short high peak pulses induce photoacoustic cavitation; collapsing bubbles generate microjets and shear that detach 3D biofilm from root furcation and irregular surfaces. Photochemical activation produces singlet oxygen and radicals that oxidize membranes, nucleic acids and matrix components, enhancing antimicrobial penetration and reducing selective pressure associated with prolonged antibiotics. Subablative irradiation modulates mitochondrial redox and downstream signaling, reduces proinflammatory cytokines and promotes fibroblast proliferation, collagen deposition, osteogenic gene expression and VEGF mediated angiogenesis, effects that correspond to measurable gains in probing depth, clinical attachment level and local bone volume. This review aims to synthesize mechanistic insights and clinical considerations to guide parameter selection and future translational research.

Keywords: Laser, periodontal biofilm, photodynamic therapy, tissue regeneration.

1. Introduction

Periodontal disease is one of the leading causes of tooth loss in adults worldwide. Its core etiology lies in the periodontal biofilm, which is composed of bacteria, fungi, and their metabolites, embedded in an extracellular polymeric matrix (EPS). This three-dimensional structure not only resists host immune clearance and antimicrobial agents but also continuously releases toxic factors, triggering an inflammatory response, ultimately leading to alveolar bone resorption and destruction of periodontal supporting tissues. Traditional treatment relies primarily on supragingival scaling and root planning (SRP), but this approach often fails to remove biofilm located in anatomically inaccessible areas such as furcation and deep periodontal pockets, and improper application can easily damage adjacent healthy tissue. Adjunctive antimicrobial agents can partially improve efficacy, but long-term use carries the risk of drug resistance and can disrupt the oral microbiome.

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Therefore, effectively destroying the biofilm while protecting tissue and promoting regeneration has become a key challenge in periodontal treatment.

In recent years, laser technology has emerged as a promising minimally invasive tool with wavelength specificity and controllable energy delivery. NdYAG, ErYAG, and diode lasers exert photothermal, photomechanical, and photochemical effects respectively, enabling targeted biofilm disruption with reduced mechanical trauma and improved clearance efficiency. Beyond antimicrobial effects, low-intensity laser irradiation exhibits photobiomodulation, leading to decreased expression of pro-inflammatory cytokines, enhanced fibroblast proliferation, upregulation of osteogenic genes, and stimulation of angiogenesis, thereby creating favorable conditions for periodontal repair and regeneration. Despite these advantages, critical gaps remain: optimal parameter settings for different lesion types are not yet standardized, long-term data on recurrence and bone regeneration are limited, and the molecular pathways linking biofilm removal to tissue regeneration remain insufficiently clarified [1, 2]. This review aims to synthesize current evidence on laser-assisted biofilm disruption and its potential role in periodontal regeneration, focusing on mechanisms of biofilm destruction, modulation of inflammation and repair, parameter optimization strategies, clinical applications and challenges, and future research priorities, in order to provide an evidence-based framework for the application of laser technology in periodontology.

2. Mechanisms of laser-mediated destruction of periodontal biofilm

2.1. Physical destructive effects

The physical destruction of periodontal biofilm by lasers is primarily achieved through two complementary energy conversion mechanisms: the photothermal effect and the photomechanical (photoacoustic) effect. The photothermal effect relies on the selective absorption of laser wavelengths by the biofilm and its components. When laser energy is absorbed by water, pigments, or mineralized residues within the biofilm, photon energy is rapidly converted into thermal energy, resulting in local temperature elevation. This temperature rise further causes protein denaturation, cell membrane instability, and the aggregation or decomposition of the polysaccharide matrix. The affinity of different wavelengths for specific components determines their clinical applicability: for instance, the Nd:YAG wavelength (approximately 1064 nm) exhibits high affinity for blood-rich or pigment-rich areas, making it suitable for penetrating and acting on deeper lesions; the Er:YAG wavelength (approximately 2940 nm) overlaps with the absorption peaks of water and hydroxyapatite, enabling efficient vaporization of tissue water and removal of adherents; near-infrared diode lasers (approximately 810–980 nm), being sensitive to pigments, can be easily combined with photosensitizers for photodynamic therapy (PDT). Understanding the laws of energy conduction and scattering of lasers in multi-layered biofilms and the underlying tissues is crucial for designing parameter combinations that can fully destroy the biofilm while protecting adjacent healthy tissues. In clinical practice, attention must be paid to the thermal accumulation effect. Measures such as intermittent irradiation, reduced repetition frequency, or combined cooling/irrigation should be adopted to control temperature elevation within the tolerable range of tissues, thereby avoiding irreversible thermal damage to tooth roots, periodontal ligaments, and bone tissue [3].

In addition to the thermal pathway, short-pulse, high-peak lasers also generate strong physical peeling forces through the photomechanical effect. When high-energy short pulses are absorbed in water-containing or dissociable media, vaporization nuclei or bubbles are formed instantaneously. The rapid expansion and subsequent collapse of these bubbles produce local shock waves and

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microjets. This "photoacoustic micro-explosion" can exert extremely high shear stress on the biofilm matrix at the microscale, thereby breaking down and peeling off the three-dimensional biofilm structure. This mechanism shows distinct advantages in removing persistent biofilms located in anatomical blind areas, such as root furcation, deep pocket floors, and irregular root surface depressions—regions that are difficult for mechanical scaling instruments to access or reach. The efficiency of photomechanical biofilm destruction is influenced by parameters including pulse width, peak power, pulse repetition rate, and the distance between the optical fiber and the tissue. Therefore, precise parameter design is required to balance bubble dynamics in a way that ensures efficient biofilm peeling without causing mechanical damage to the surrounding hard and soft tissues [2].

2.2. Biochemical destructive effects

The laser-induced biochemical destruction of periodontal biofilm is mainly accomplished through the production of reactive oxygen species (ROS) via photochemical reactions: after exciting exogenous or endogenous photosensitizer molecules, electron transfer or energy transfer reactions occur, generating free radicals and singlet oxygen. These highly reactive molecules can oxidize bacterial lipid membranes, proteins, and nucleic acids, disrupting cellular functions and inducing bacterial death. Simultaneously, they chemically degrade the extracellular polysaccharide-protein matrix (EPS), reducing the biofilm's adhesion and structural integrity, which in turn enhances the efficiency of physical removal and drug penetration.

Taking PDT (combining diode lasers with porphyrin-based photosensitizers) as an example: it achieves deep-seated bactericidal effects under low thermal load. Due to its localized action (dependent on the spatial distribution of light and photosensitizers), it has a lower probability of inducing traditional drug resistance mechanisms and causes less long-term damage to the microecology compared to long-term use of broad-spectrum antibiotics. However, improper dosage or coverage may temporarily inhibit beneficial bacteria or allow opportunistic pathogens to reoccupy the ecological niche. To balance bactericidal efficacy and microecological protection, clinical strategies can be adopted, such as pre-treating with mechanical or enzymatic methods to loosen the EPS, optimizing photosensitizer administration and nano-carriers to improve penetration, combining with ultrasound/acoustic enhancement and segmented irradiation, and supplementing with post-treatment microecological reconstruction (e.g., local probiotics, targeted irrigation) to promote the recovery of beneficial bacteria. Precise control of irradiation parameters and consideration of oxygen availability are required, since excessive reactive oxygen species may damage host cells. Real-time monitoring is therefore crucial to balance safety with therapeutic efficacy [4].

3. Regulatory role of lasers in the inflammatory microenvironment and tissue regeneration

3.1. Regulation of inflammatory factors

The impact of lasers on inflammatory factors is time- and dose-dependent. In the early stage of irradiation, a transient increase in pro-inflammatory factors (e.g., TNF-α, IL-1β) often occurs, reflecting tissue stress and debridement responses. Subsequently, low-intensity photobiomodulation can inhibit pro-inflammatory pathways such as NF-κB, reduce pro-inflammatory factors, and upregulate anti-inflammatory factors (e.g., IL-10, TGF-β), shifting the local microenvironment toward repair. Lasers can also indirectly inhibit osteoclast activity by altering the RANKL/OPG

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balance, slowing down alveolar bone resorption and facilitating bone tissue preservation and regeneration [5].

It should be noted that these biological effects are highly dependent on wavelength, power, pulse mode, and irradiation frequency; improper parameters may result in ineffectiveness or thermal/mechanical damage. Although reducing local inflammatory load theoretically improves systemic inflammatory markers and exerts positive effects on chronic diseases such as diabetes and cardiovascular disorders, such systemic benefits still require verification through long-term follow-up and clinical trials focusing on systemic indicators.

3.2. Induction of periodontal tissue regeneration

Laser-promoted tissue regeneration is primarily achieved through the synergistic photobiomodulation effect on cellular metabolism and signaling pathways: when low-intensity lasers are absorbed by mitochondrial pigments, they increase ATP production and trigger calcium signaling, which further activates pathways such as MAPK and PI3K/Akt. This promotes the proliferation, migration, and collagen synthesis of fibroblasts, thereby accelerating gingival margin attachment and soft tissue repair.

In bone tissue, lasers can upregulate osteogenic markers (e.g., Runx2/Cbfa1, ALP, OCN, OPN) and activate pathways including Wnt/β-catenin and BMP/Smad, promoting the differentiation of osteoprogenitor cells into the osteogenic lineage and matrix mineralization. This is manifested in in vivo improvements in bone volume fraction, trabecular arrangement, and bone mineral density. Meanwhile, lasers can induce the expression of angiogenic factors (e.g., VEGF, FGF), stimulating the proliferation, migration, and lumen formation of endothelial cells, and promoting the reconstruction of capillary networks to provide necessary blood supply and nutrients for regenerating tissues. Common methods used to quantify these effects include cell migration and proliferation assays, total collagen or type I collagen determination, osteogenic gene expression detection, as well as micro-CT, immunohistochemistry (e.g., CD31, OCN), and histological evaluation [6].

4. Optimization strategies for laser treatment parameters

Different laser wavelengths interact with periodontal tissues and biofilm components in distinct ways, which determines their clinical roles and parameter settings. NdYAG lasers at approximately 1064 nanometers penetrate several millimeters and show high affinity for hemoproteins and pigmented substrates, making them suitable for coagulation, hemostasis and disinfection of vascularized deep pockets that are inaccessible to mechanical instruments. ErYAG lasers at approximately 2940 nanometers are strongly absorbed by water and hydroxyapatite, so they efficiently vaporize tissue water and remove mineralized deposits, providing selective ablation of root surface biofilm and adherent calculus. Diode lasers in the 810 to 980 nanometer range are compact and convenient for outpatient use, and when combined with porphyrin type photosensitizers they produce reactive oxygen species under low thermal burden, which supports maintenance therapy and adjunctive local sterilization.

A staged multimodal workflow often yields the best clinical outcomes. Coarse mechanical debridement or ErYAG ablation is used first to remove bulk deposits and open access to root anatomy. Short high peak pulses targeting photomechanical effects are then applied to address residual biofilm in furcation and irregular surfaces. Finally, low energy continuous or pulsed photobiomodulation modes are used to promote hemostasis and to initiate tissue repair. Attention to

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fiber position, incident angle and sweep speed is essential to obtain uniform energy distribution and to avoid local overheating [1].

Parameter selection must be tailored to the biofilm physicochemical properties and the periodontal tissue condition. Biofilm thickness, water content, extracellular polymeric substance composition and dominant pathogens affect light absorption and penetration, so water rich or mineralized biofilms favor ErYAG use while pigment rich or blood contaminated lesions respond better to NdYAG or diode lasers. Thick or highly dense biofilms benefit from preconditioning by mechanical or enzymatic EPS disruption to enhance light or photosensitizer penetration. Mild to moderate cases can be managed with repeated low energy exposures designed to exploit photobiomodulation, whereas severe cases with bone defects require higher instantaneous effects for debridement followed by carefully dosed regenerative stimulation, with strict temperature monitoring to prevent thermal injury of bone and periodontal ligament.

Additional determinants of parameter choice include treatment stage, systemic comorbidities such as diabetes or anticoagulant use, local oxygen availability which influences photodynamic efficacy, and device specific properties including pulse width, peak power and energy density expressed in joules per square centimeter. To improve safety and reproducibility, routine use of real time thermal or acoustic feedback is recommended, together with systematic recording of applied parameters and the establishment of recommended parameter ranges matched to lesion categories. Lasers are most effective when integrated into a multimodal protocol that combines mechanical debridement, photodynamic therapy, targeted drug or probiotic delivery and scaffold or regenerative adjuncts, so that treatment can be individualized for each lesion profile [7].

5. Clinical applications and challenges of laser-assisted therapy

In periodontal treatment, lasers can be used alone for local sterilization and hemostasis, with common short-term improvements in probing depth (PD), bleeding on probing (BOP), and clinical attachment level (CAL). However, they are more frequently used as a supplement to mechanical scaling or in combination with photodynamic therapy/local drugs to improve the removal rate of deep biofilms and enhance the penetration and efficacy of drugs or photosensitizers. Laser procedures are typically associated with less intraoperative bleeding, lower discomfort, and shorter healing time, facilitating outpatient-based care, maintenance follow-ups, and minimally invasive management of patients with bleeding tendencies or comorbidities. Portable devices and rapid operation also make them easy to promote as routine auxiliary tools in clinics.

The main challenges include insufficient long-term follow-up data, large-sample evidence, limited understanding of the microbial-host molecular mechanisms underlying laser action and the lack of unified standards for laser parameters, devices, and operations, all of which restrict the reproducibility of therapeutic effects. In addition, factors such as device costs, reimbursement policies, and variations in operator training also limit their widespread application. To advance clinical application, it is necessary to conduct multi-center, long-term randomized controlled trials (RCTs) incorporating patient-reported outcomes (PROs, e.g., pain, function, and satisfaction), establish unified standards for parameters and follow-up endpoints, promote intraoperative real-time monitoring and safety protocols, and develop training/certification and clinical registration systems. These efforts will help gradually establish a reproducible and scalable laser-assisted periodontal treatment pathway [8].

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6. Future directions and translational research priorities

Personalized treatment should be based on a quantifiable diagnostic spectrum. It is recommended that the results of biofilm composition profiling, oral microbiome sequencing, host immune and genetic markers, and high-resolution imaging be integrated into a set of diagnostic feature vectors. Based on this, two types of decision-making tools should be developed. The first type is a regularized parameter mapping table that directly maps common diagnostic patterns to recommended energy density (J/cm²), pulse width, irradiation frequency, and phased irradiation rhythm. The second type is a machine learning model that inputs imaging and omics data and outputs several comparable parameter options and provides thermal damage and healing risk scores for surgeon selection or as a pre-set for trial design.

Technically, energy delivery and closed-loop control should be prioritized. Key R&D efforts include developing sequential or parallel output capabilities for multi-wavelength lasers, miniaturized optical fibers with embedded temperature and acoustic sensors for outpatient use, and energy regulation algorithms that use real-time thermal imaging and acoustic echoes as control variables. These sensor streams enable millisecond-level energy adjustment, aiming to maintain local temperature rise within a tolerable range under varying tissue and biofilm conditions while also controlling bubble dynamics to improve blind zone decontamination efficiency.

Regarding treatment strategies, phased, combined validation studies are recommended. Priority validation approaches include photodynamic therapy combined with nano-drug delivery to improve photosensitizer penetration, laser-assisted simultaneous implantation of bioactive scaffolds to achieve localized osteogenesis and vascularization, and sequential reconstruction after laser debridement with probiotics or immunomodulators to guide favorable recolonization. To support these studies, a multicenter animal-to-early human trial pipeline should be established, with clear definitions of key safety indicators and efficacy endpoints.

To accelerate clinical translation, three institutional developments must be pursued simultaneously. Firstly, a unified energy dosimetry and safety reporting template should be developed, clearly recording elements such as J/cm², pulse parameters, maximum allowable temperature rise, and fiber position. Secondly, a core endpoint set encompassing probing depth, clinical attachment, bone volume fraction, and patient-reported outcomes should be established for comparability assessment in multicenter randomized trials. Finally, equipment acceptance criteria, operator training and registration systems, and adverse event registration databases should be established to ensure operational quality and long-term safety monitoring [9, 10].

7. Conclusion

This review systematically elaborates on the mechanisms of laser-assisted biofilm destruction and its potential role in periodontal regeneration. Lasers achieve physical destruction of periodontal biofilm through photothermal and photomechanical effects, and biochemical destruction via ROS production through photochemical reactions; they can also regulate inflammatory factors and promote tissue regeneration, with treatment parameters requiring optimization based on wavelength characteristics and disease conditions. Currently, lasers are mostly used as auxiliary tools in periodontal treatment, capable of improving clinical indicators and reducing intraoperative discomfort, but face challenges such as insufficient long-term evidence and lack of unified standards. In the future, they should develop toward personalization, technological innovation, and multimodal integration to advance their clinical application.

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In summary, lasers demonstrate significant advantages and potential in the field of periodontal treatment. They efficiently destroy periodontal biofilm through dual physical and biochemical mechanisms, regulate the inflammatory microenvironment, and promote tissue regeneration. After parameter optimization, they can be adapted to the needs of different disease conditions, with prominent effects in improving clinical indicators and enhancing treatment comfort. However, laser-assisted periodontal therapy still faces practical challenges, such as lack of long-term efficacy evidence, inconsistent parameters and operating standards, high equipment costs, and large differences in training, which have limited its widespread promotion.

In the future, with the advancement of precision diagnostic technologies, the development of new laser devices, and the improvement of multimodal combination strategies, laser-assisted therapy is expected to achieve personalized and intelligent development. Through the implementation of multi-center long-term clinical trials and the establishment of a standardized system, lasers will be more safely and effectively integrated into the entire process of periodontal treatment, providing better solutions for the treatment of periodontal diseases and periodontal tissue regeneration, and driving the field of periodontal treatment to new heights.

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