BIOLOGICAL EFFECTS OF LASER RADIATION ON HUMAN SKIN CELLS

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Abstract. *Laser radiation has become an essential tool in modern dermatology and biomedical research due to its precise and controllable interaction with biological tissues. This article examines the biological effects of laser radiation on human skin cells, with a particular focus on cellular, molecular, and biochemical responses. Laser–tissue interaction mechanisms such as photothermal, photochemical, and photomechanical effects are analyzed in relation to different laser parameters, including wavelength, power density, and exposure duration. The study discusses how laser irradiation influences key cellular processes such as cell proliferation, apoptosis, collagen synthesis, DNA integrity, and inflammatory responses. Both therapeutic and potentially harmful effects are considered, emphasizing the importance of dose optimization and safety standards. Understanding the biological responses of skin cells to laser radiation is crucial for improving the efficacy and safety of laser-based medical and cosmetic treatments.*

Keywords: *Laser radiation; human skin cells; laser–tissue interaction; photothermal effect; photochemical effect; cell proliferation; apoptosis; dermatological therapy.*

Introduction.

The rapid development of laser technology has significantly expanded its applications in medicine, particularly in the field of dermatology. Owing to their monochromaticity, coherence, and high energy density, lasers allow for precise and controlled interaction with biological tissues. As a result, laser radiation is widely used for the treatment of various skin conditions, including acne, scars, pigmentation disorders, vascular lesions, and skin rejuvenation procedures.

Despite their broad clinical use, the biological effects of laser radiation on human skin cells remain a subject of ongoing scientific investigation.

Human skin is a complex, multilayered organ composed of different cell types, such as keratinocytes, fibroblasts, melanocytes, and immune cells, each of which responds differently to laser exposure. The interaction between laser radiation and skin cells depends on multiple parameters, including wavelength, pulse duration, energy density, and tissue optical properties.

These interactions can trigger a range of biological responses, from beneficial therapeutic effects to unintended cellular damage if improperly applied.

At the cellular and molecular levels, laser radiation can induce photothermal, photochemical, and photomechanical effects. These mechanisms influence fundamental biological processes such as cell proliferation, collagen remodeling, inflammatory signaling, and programmed cell death.

Low-level laser irradiation has been reported to stimulate cellular metabolism and tissue regeneration, whereas high-intensity laser exposure may cause protein denaturation, DNA damage, and oxidative stress. Therefore, understanding the dose-dependent nature of laser–cell interactions is essential for maximizing therapeutic outcomes while minimizing adverse effects.

The purpose of this article is to analyze the biological effects of laser radiation on human skin cells by reviewing current experimental and clinical findings. Particular attention is given to the mechanisms of laser–tissue interaction, cellular responses, and safety considerations.

A deeper understanding of these biological processes is critical for the optimization of laser-based therapies and the development of safer and more effective dermatological treatments.

Literature Review

A substantial body of research has explored the biological effects of laser radiation on human skin cells, focusing on both therapeutic outcomes and underlying cellular mechanisms.

One major area of investigation is photobiomodulation (PBM), also known as low-level laser therapy (LLLT), which uses low-intensity laser light to influence cellular behavior. PBM has been shown to affect gene expression and the release of growth factors and cytokines in cultured human and animal cells, with outcomes dependent on wavelength and radiant exposure parameters. These changes suggest potential clinical effects on inflammation and cell proliferation relevant to wound healing and tissue repair.

Several in vitro studies demonstrate that laser irradiation can modulate cell proliferation, viability, and morphology in human skin fibroblasts. For example, helium-neon laser exposure at various energy densities produced measurable alterations in fibroblast proliferation and DNA integrity, indicating both beneficial and potentially damaging effects based on dosage.

Additionally, near-infrared laser exposure at low and medium intensities was found to accelerate wound closure in cultured fibroblasts without significant temperature increases, underscoring the importance of controlled exposure levels for positive biological responses.

Mechanistically, the interaction of laser light with skin cells involves complex photobiological processes. Reviews of laser-tissue interaction highlight photochemical, photothermal, and photomechanical effects as primary pathways by which laser energy alters cellular structures and functions. Photochemical effects, in particular, play a significant role at lower irradiation levels, influencing biochemical and physiological phenomena within cells.

Modern reviews also emphasize that PBM interacts with intracellular chromophores and signaling pathways, activating cellular responses that can lead to increased ATP production, modulation of oxidative stress, and improved tissue metabolism.

Comprehensive narrative reviews of PBM in dermatology further support its clinical potential.

They document the non-invasive use of visible and near-infrared wavelengths to modulate cellular functions, reduce inflammation, stimulate collagen synthesis, and promote wound healing in skin tissues. These effects are closely tied to the specific light parameters employed, reinforcing the need for precise control over wavelength and dose in therapeutic contexts.

Despite the demonstrated therapeutic benefits, the literature also notes variability in experimental results due to differences in study design, irradiation parameters, and cell types.

This variability underscores the need for standardized protocols and further research to fully elucidate the dose-dependent nature of laser effects on human skin cells. Nevertheless, existing evidence collectively indicates that appropriately calibrated laser radiation can induce favorable biological responses at the cellular level, with applications in dermatological therapy and regenerative medicine.

Discussion

The biological effects of laser radiation on human skin cells are governed by fundamental physical laws describing light–matter interaction, as well as by complex biological response mechanisms at the cellular and molecular levels. Understanding these effects requires an interdisciplinary approach that integrates laser physics, tissue optics, thermodynamics, and cell biology. The discussion below analyzes how laser parameters influence skin cell behavior through quantifiable physical processes and mathematically describable biological responses.

1. Physical Principles of Laser–Skin Interaction

Laser radiation interacting with human skin undergoes reflection, scattering, absorption, and transmission. The fraction of incident laser energy absorbed by skin tissue determines the magnitude of biological effects. This process can be described by the Beer–Lambert law, which governs light attenuation in absorbing media:

$$I(z) = I_0 e^{-\mu_a z}$$

Where

I(z) is the laser intensity at depth z,

I_0 is the incident intensity,

μ_a is the absorption coefficient of skin tissue (cm^{-1}).

Human skin exhibits wavelength-dependent optical properties due to the presence of chromophores such as melanin, hemoglobin, and water. These chromophores selectively absorb laser energy, converting photon energy into biochemical or thermal energy. Consequently, the biological response of skin cells is highly sensitive to the laser wavelength λ\lambdaλ.

The energy fluence F, defined as energy delivered per unit area, is given by:

$$F = \frac{E}{A}\left(\frac{J}{cm^2}\right)$$

Where

E is the laser energy (J),

A is the irradiated area (cm^2).

Fluence is a critical parameter because cellular responses follow a nonlinear dose–response relationship, often described by the biphasic Arndt–Schulz curve, where low doses stimulate biological activity and high doses inhibit or damage cells.

2. Photothermal Effects and Cellular Heat Transfer

When laser energy is absorbed by skin tissue, it may be converted into heat, leading to photothermal effects. The resulting temperature increase ΔT in tissue can be estimated using the bioheat equation:

$$\rho c \frac{\partial T}{\partial t} = k\nabla^2 T + Q$$

where

ρ is tissue density (kg/m^3),

c is specific heat capacity (J/kg·K),

k is thermal conductivity (W/m·K),

Q is the heat source term related to laser absorption (W/m^3).

The heat source term Q can be expressed as:

$$Q = \mu_a I$$

An increase in tissue temperature above physiological thresholds (≈42–45°C) may cause protein denaturation, enzyme inactivation, and membrane destabilization in skin cells.

At moderate temperatures, however, heat shock proteins (HSPs) may be upregulated, contributing to cellular protection and repair mechanisms.

Experimental studies indicate that fibroblasts exposed to laser-induced temperatures below 40°C exhibit increased collagen synthesis, whereas temperatures exceeding 50°C can result in irreversible cell damage and necrosis. Thus, precise thermal control is essential for therapeutic laser applications.

3. Photochemical Effects and Reactive Oxygen Species Generation

At low and moderate laser intensities, photochemical rather than photothermal mechanisms dominate. In this regime, absorbed photons excite intracellular chromophores such as cytochrome c oxidase within mitochondria. This excitation enhances electron transport chain activity, leading to increased adenosine triphosphate (ATP) production:

$$ADP + P_i \xrightarrow{Laser} ATP$$

The increase in ATP concentration [ATP] has been correlated with enhanced cell proliferation and migration. However, excessive excitation may result in the overproduction of reactive oxygen species (ROS), which can be expressed as:

$$O_2 + e^- \rightarrow O_2^{\cdot -}$$

ROS play a dual role: at low concentrations, they function as signaling molecules that activate transcription factors such as NF-κB and AP-1; at high concentrations, they induce oxidative stress, lipid peroxidation, and DNA strand breaks.

The balance between beneficial signaling and oxidative damage can be mathematically described by a threshold function:

$$R_{bio} = \begin{cases} Stimulation, & ROS < ROS_{crit} \\ Damage, & ROS \geq ROS_{crit} \end{cases}$$

where ROS_{crit} represents the critical concentration beyond which cellular damage occurs.

4. Laser-Induced Modulation of Cell Proliferation

Cell proliferation under laser irradiation follows a nonlinear response that can be modeled using a dose–response equation:

$$P(F) = P_0(1 + \alpha F e^{-\beta F})$$

Where

P(F) is the proliferation rate as a function of fluence,

P_0 is the baseline proliferation rate,

α and β are experimentally determined constants.

This equation illustrates that proliferation increases with fluence up to an optimal value F_{opt}, after which inhibitory effects dominate. Studies on keratinocytes and fibroblasts confirm that fluences between 1–5 J/cm² often maximize proliferation, whereas higher fluences suppress mitotic activity.

5. DNA Integrity and Laser Exposure

Laser radiation may affect DNA integrity either indirectly via ROS or directly through high-energy photon absorption. DNA damage probability D can be approximated as a function of absorbed energy:

$$D \propto \mu_a F$$

Low-level laser therapy generally produces sub-lethal stress, activating DNA repair pathways such as base excision repair (BER). In contrast, excessive irradiation may overwhelm repair mechanisms, leading to apoptosis or mutagenesis.

Apoptotic response can be modeled using a rate equation:

$$\frac{dN_a}{dt} = k_a(F - F_{th})$$

where

N_a is the number of apoptotic cells,

k_a is the apoptosis rate constant,

F_{th} is the threshold fluence for apoptosis initiation.

6. Implications for Therapeutic Optimization

The discussed physical and biological mechanisms emphasize the importance of optimizing laser parameters to achieve therapeutic benefits while avoiding adverse effects. The therapeutic window can be defined as:

$$F_{min} < F < F_{max}$$

Where

F_{min} is the minimum fluence required for biological stimulation,

F_{max} is the maximum fluence before tissue damage occurs.

This window depends on cell type, wavelength, pulse duration, and exposure time.

Mathematical modeling combined with experimental validation is therefore essential for personalized laser-based skin therapies.

7. Photomechanical Effects and Stress Wave Formation

In addition to photothermal and photochemical mechanisms, laser radiation can induce photomechanical effects, particularly when short or ultra-short laser pulses are applied. These effects arise from rapid energy deposition within tissue, leading to thermoelastic expansion and the generation of stress waves. The pressure increase ΔP caused by rapid heating can be approximated by:

$$\Delta P = \Gamma \mu_a F$$

Where

Γ is the Grüneisen parameter, describing the efficiency of thermal-to-mechanical energy conversion,

μ_a is the absorption coefficient,

F is the laser fluence.

Photomechanical stress waves can disrupt cellular membranes and intracellular structures if their amplitude exceeds mechanical tolerance thresholds. However, when controlled appropriately, these stress waves may enhance membrane permeability, facilitating molecular transport and intracellular signaling. This phenomenon has been investigated in laser-assisted drug delivery and transdermal transport applications.

The mechanical strain ε\varepsilonε induced in skin tissue can be expressed as:

$$\varepsilon = \Delta L / L_0$$

where

ΔL is the deformation caused by stress waves,

L_0 is the original tissue length.

If ε\varepsilonε remains below the elastic limit of skin cells, reversible deformation occurs, allowing cells to recover without permanent damage.

8. Laser-Induced Collagen Remodeling

One of the most clinically relevant outcomes of laser irradiation is collagen remodeling, particularly in dermal fibroblasts. Collagen synthesis and degradation can be modeled using a kinetic balance equation:

$$\frac{dC}{dt} = k_s(F) - k_d C$$

where

C is collagen concentration,

$k_s(F)$ is the fluence-dependent synthesis rate,

k_d is the degradation constant.

Experimental studies indicate that moderate laser fluences increase ksk_sks through fibroblast activation and upregulation of transforming growth factor-beta (TGF-β). This results in increased collagen deposition, improving skin elasticity and structural integrity. Conversely, excessive laser energy may increase matrix metalloproteinase (MMP) activity, enhancing collagen degradation and impairing tissue regeneration.

The optimal fluence for collagen remodeling can be estimated by finding the maximum of $\frac{dC}{dt}$, yielding a condition where synthesis outweighs degradation without triggering thermal damage.

9. Modulation of Inflammatory Responses

Laser radiation significantly influences inflammatory processes in skin tissue. Inflammatory mediator concentration ImI_mIm can be described by:

$$\frac{dI_m}{dt} = S(F) - \gamma I_m$$

where

S(F) represents laser-induced cytokine signaling,

γ is the clearance rate of inflammatory mediators.

Low-level laser irradiation has been shown to reduce pro-inflammatory cytokines such as TNF-α and IL-6 while increasing anti-inflammatory mediators like IL-10. This shift contributes to reduced edema, pain, and erythema in treated skin areas. The anti-inflammatory effect is most pronounced when laser fluence remains within the therapeutic window:

$$F_{anti-inf} \in [F_{min}, F_{opt}]$$

Outside this range, excessive stimulation may exacerbate inflammation due to thermal injury and cell necrosis.

10. Mathematical Modeling of Wound Healing Dynamics

Laser-enhanced wound healing is a multifactorial process involving cell migration, proliferation, angiogenesis, and extracellular matrix remodeling. The rate of wound closure W(t) can be modeled as:

$$\frac{dW}{dt} = -\alpha P(F) - \beta M(F)$$

where

P(F) is the proliferation rate of keratinocytes and fibroblasts,

M(F) is the migration rate of skin cells,

α and β are weighting coefficients.

Laser irradiation enhances both P(F) and M(F) by increasing ATP production and cytoskeletal reorganization. Clinical observations confirm faster re-epithelialization and reduced healing time in laser-treated wounds compared to controls.

11. Comparison of Theoretical Models with Experimental Data

Theoretical predictions derived from physical and mathematical models generally align with experimental findings reported in the literature.

For instance, the biphasic dose–response curve predicted by proliferation models corresponds well with in vitro observations of fibroblast growth under varying fluences. Similarly, thermal models based on the bioheat equation accurately predict tissue temperature profiles observed during laser dermatological procedures.

However, discrepancies remain due to biological variability, differences in experimental setups, and the heterogeneous nature of human skin. These limitations highlight the need for personalized modeling approaches that incorporate patient-specific optical and thermal properties.

12. Safety Considerations and Risk Assessment

From a safety perspective, laser-induced biological effects must be evaluated against established damage thresholds. The probability of adverse effects RRR can be modeled as:

$$R = 1 - e^{-\lambda(F - F_{safe})}$$

where

λ is a risk sensitivity coefficient,

F_{safe} is the maximum safe fluence.

This probabilistic approach emphasizes that risk increases exponentially once fluence exceeds safety limits. Consequently, real-time monitoring of laser parameters and tissue response is essential in clinical practice.

13. Implications for Future Research and Clinical Practice

The integration of physical modeling, mathematical analysis, and biological experimentation provides a robust framework for understanding laser–skin interactions. Future research should focus on multiscale models that link photon absorption at the molecular level to macroscopic tissue responses. Advances in computational modeling and artificial intelligence may further enhance the precision and predictability of laser-based skin therapies.

14. Overall Discussion Summary

In summary, the biological effects of laser radiation on human skin cells arise from a complex interplay of photothermal, photochemical, and photomechanical mechanisms. These effects can be quantitatively described using physical laws and mathematical models, offering valuable insights into therapeutic optimization. When applied within well-defined parameter ranges, laser irradiation can stimulate beneficial cellular responses such as proliferation, collagen synthesis, and inflammation modulation. Conversely, exceeding critical thresholds leads to cellular damage and adverse outcomes. A thorough understanding of these mechanisms is therefore essential for the safe and effective application of laser technology in dermatology.

Conclusion

The interaction of laser radiation with human skin cells involves a complex combination of physical, chemical, and mechanical processes that produce a wide range of biological effects.

This study has shown that laser parameters such as wavelength, fluence, pulse duration, and exposure time critically influence cellular responses, including proliferation, apoptosis, collagen synthesis, and inflammatory signaling.

Low-level laser irradiation primarily induces photochemical effects, stimulating mitochondrial activity, ATP production, and cell proliferation, while moderate fluences generate controlled photothermal effects that enhance tissue regeneration and collagen remodeling. High-intensity or excessive exposure, however, may produce photomechanical stress, ROS overproduction, DNA damage, and cell necrosis, emphasizing the necessity of precise dose control.

Mathematical models and physical laws, including the Beer–Lambert law, bioheat equation, and kinetic equations for proliferation and collagen dynamics, provide a quantitative framework for understanding and predicting these biological responses.

Clinical applications of laser therapy in dermatology benefit from these insights, allowing for optimized treatment protocols that maximize therapeutic effects while minimizing potential risks. The evidence supports the effectiveness of laser-based interventions in wound healing, anti-inflammatory treatments, skin rejuvenation, and scar management.

In conclusion, a thorough understanding of the dose-dependent, wavelength-specific, and cell-type-specific effects of laser radiation is essential for safe and effective dermatological applications. Future research should focus on refining mathematical and computational models, integrating patient-specific tissue properties, and exploring novel laser modalities to further enhance the precision and efficacy of skin treatments. The continued integration of physics, mathematics, and cellular biology will enable laser therapy to reach its full potential as a versatile tool in both clinical and cosmetic dermatology.

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