

E-ISSN: 2709-9369

P-ISSN: 2709-9350

www.multisubjectjournal.com

IJMT 2024; 6(11): 16-18

Received: 09-08-2024

Accepted: 14-09-2024

Mohannad M Hurry

The General Directorate of
Education in Babylon,
Ministry of Education,
Babylon, Iraq

Mohammed HH AL-Jassani

Physics Department, Faculty of
Sciences, University of
Kufa, Najaf, Iraq

Corresponding Author:

Mohannad M Hurry

The General Directorate of
Education in Babylon,
Ministry of Education,
Babylon, Iraq

Review: Tissue interactions with low-level laser

Mohannad M Hurry and Mohammed HH AL-Jassani

DOI: <https://doi.org/10.22271/multi.2024.v6.i11a.494>

Abstract

A laser is a device that uses photon emission to produce light through a process that involves optic amplification. discovered LLLT, a type of complementary therapy in the 1960s. Because LLLT uses low power densities to avoid heating tissue, it is often referred to as "cold laser therapy". Despite being around for almost 50 years, photobiomodulation—also referred to as low-level laser treatment, or LLLT—has not yet received mainstream recognition, mostly because the molecular, cellular, and tissular mechanisms of action are still unknown. Nonetheless, a great deal of information has been acquired in this field recently, which this review will outline.

Keywords: Laser-tissue interaction, photobiomodulation

Introduction

Cold Laser Therapy's History

Townes noticed that regular light could be manipulated to produce short waves, which led to the discovery of lasers in the early 1960s. However, the method for efficiently emitting light waves was developed by Arthur Schawlow. He understood that placing the atoms in a long, thin hollow with mirrors attached to either end would be the best way to allow the waves to radiate. By researching methods to "pump" atoms into higher energy states so they might generate light, Gordon Gould contributed to the advancement of the laser. Laser," which is an acronym for "Light Amplifier by Stimulated Emission of Radiation," was first used by Gould. The three researchers also investigated the advantages of employing different gasses and crystals to boost atoms' energy ^[1]. The utilization of low level lasers began with the development of the ruby laser in 1960 and the helium-neon laser in 1961. The first person to notice the advantages of laser therapy in a therapeutic context was Endre Mester. He noticed this when he used a Helium-Neon (HeNe) laser to treat hairless mice, which caused them to start growing hair. Mester treated patients with non-healing skin lesions following his discovery ^[2]

Light Sources

LLLT now uses a number of distinct light sources. These days, light-emitting diodes are widely used source of laser light, along with gallium arsenide semiconducting diode lasers. The increasing popularity of light-emitting diodes (LEDs) can be attributed to their ability to emit light across a far wider spectrum of wavelengths than any other laser. With the development of new technology, organic LEDs with an electroluminescent layer—which produces light when an electrical current passes through it— are the newest light source being studied ^[3].

Mechanisms and Laser Parameters

Light, oxygen, and photosensitizers are the three elements that are essential to a laser's operation ^[3].

Photosensitizers

To induce photochemical reactions, photosensitizers—also known by the term dyes can absorb, reflect, and transmit the energy of the laser's light into other molecules ^[4]. Various wavelengths of light are absorbed by these photosensitizers, which then transform the light into fluorescence and distinct atomic configurations. Reactive oxygen species are created as a result of this conversion process, which also causes an energy transfer between oxygen molecules ^[5]. In general, photosensitizers may be divided into two categories: porphyrin-based and non-porphyrin-based. Chlorins, phthalocyanines, and bacteriochlorin are examples of photosensitizers that are classified as porphyrin-based.

Non-porphyrin-based photosensitizers include cyanines, psoralens, anthracyclines, and hypericin^[4]. A photosensitizer must have several qualities to be deemed "ideal" in nature. According to^[6, 7], these qualities include a photosensitizer's capacity for being soluble in water, chemically stable, provide a large amount of oxygen, accumulate quickly in target tissues, and have a molar coefficient that ranges from (600 - 900 nm).

Dose

The amount of light energy focused on a certain unit of area over a predetermined amount of time is known as the laser's dosage or fluency. Joules per centimeter squared, is the unit of measurement that is employed. Laser's power output, expressed in watts, is taken into account when calculating the dosage. This value is then multiplied by the treatment time, expressed in seconds. The following formula provides a summary of this^[8]:

$$\text{Fluency} = (\text{Power (W)} \times \text{Time (s)}) \div \text{Area treated (cm}^2\text{)}$$

Density of Power

The intensity, sometimes called the density of light emitted by the laser, shows the output power concentrate. The area (cm²) of the target tissue is taken into consideration while calculating this power output. According to^[8], there is an inverse connection between the power density and a therapy area; the greater the treatment region, the lower the intensity.

Working Mechanisms

LLLT adheres to the Arndt-Schultz law, which states that cellular activity is activated by weak stimuli, inhibited by moderate stimuli, and destroyed by intense/high stimuli. Through a mechanism called photobiomodulation, LLLT is thought to trigger photochemical reactions within the cells^[2]. Very low radiation intensities make up LLLT, which promotes the repair of injured cells^[9]. Tissues are affected by LLLT because of a phenomenon where light reflection causes changes in the refractive indices of tissues and air. The law of Snellius may be used to summarize this^[2];

$$\frac{\sin \theta_1}{\sin \theta_2} = \frac{n_2}{n_1}$$

The angles of normal air surface to light are represented by θ_1 , normal tissue surface to laser beam by θ_2 , air refraction index by n_1 , and tissue refraction index by n_2 . According to this computation, light has "scattering behavior" in tissues, affecting the volume distribution and light intensity in certain tissues^[2]. In addition to visible (380–700 nm) and infrared (700–1000 nm) radiation, LLLT also uses fluence, which is the amount of energy per unit area of a specific size. For fluence, a unit of measuring is J/cm². The laser specifications also include the power density, which is measured in Watts per centimeter squared^[10]. The red and near-infrared light wavelengths that are often employed in LLLT vary from 600 to 1070 nm^[2].

Laser-Tissue Interaction

Melanin and hemoglobin absorb wavelength bands shorter than 600 nm, making the 600–1070 nm range the most efficient for penetrating tissue. Wavelengths between 600

and 700 nm are typically utilized to treat superficial illnesses, whereas wavelengths between 780 and 950 nm are used to reach deeper tissue. It has recently been recognized that LLLT affects the mitochondria of cells, causing a rise in reactive oxygen species, a drop in adenosine triphosphate (ATP), and a stimulation of transcription factors^[2]. Additionally, lasers have a significant impact on cells' endoplasmic reticulum (ER)^[11]. According to^[2], photodynamic therapy (PDT), which uses soft laser therapy *in vitro* to promote tissue healing, affects mitochondria and causes an increase in cell migration and proliferation as well as a modification of growth factors and cytokines and an increase in tissue oxygenation^[12]. Additional research has revealed that certain cells respond to LLLT in different ways. According to one study, keratinocyte cells are more sensitive to laser irradiation than fibroblast cells. Upon laser treatment, fibroblasts exhibited a greater baseline expression of catalase, but keratinocytes exhibited a much higher amount of reactive oxygen species^[13]. Additionally, LLLT has demonstrated the capacity to promote the motility and proliferation of epithelial cells, hence speeding up the healing process of wounds. Chromophores absorb a photon of light when cells are administered to a laser. An electron will move from a low-energy orbit to a high-energy orbit when the photon is absorbed. Numerous cellular functions are activated by this electron migration^[2]. Photodynamic treatment (PDT) is a therapeutic method that may be achieved by combining LLLT with a photosensitizer. Many studies have been carried out to see how adding a photosensitizer to LLLT may affect cells *in vitro* and how this PDT may cause cancer cells to undergo apoptosis. Programmed apoptosis happens when cells fail to maintain proper chromosomal structure throughout the proliferation process and subsequently perish, whereas unprogrammed apoptosis happens when an external effect is present (e.g., LLLT or PDT given to cells)^[14]. According to^[15], the primary mechanism of PDT-induced cell death is the generation of ROS through two paths: Type I, which generates free radicals and radical ions, and Type II, which results in the production of a singlet oxygen as a result of energy transference from the photosensitizer to a triplet oxygen. According to^[16], the production of these ROS causes cyto-damage to cancerous cells^[17, 18]. Additionally, studies have demonstrated that the combination of pheophorbide and laser inhibits the formation of colonies by prostate cancer cells^[19]. By focusing on vitamin A nuclear receptors, a chlorin-based photosensitizer has demonstrated an amazing capacity to cause toxicity in triple-negative breast cancerous cells^[20]. Additional research has confirmed that a 680 nm laser and ZnPcSmix may cause cells with cancer death in human lung and colon cells by localizing in the mitochondria and lysosomes^[21]. Further study results show that 5 J/cm² is the optimal dose at 680 nm wavelength^[22].

References

1. Weart S. Solid state insurrection: How the science of substance made American physics matter. Am Assoc Phys Teach; 2019.
2. Chung H, Dai T, Sharma SK, Huang Y-Y, Carroll JD, Hamblin MR. The nuts and bolts of low-level laser (light) therapy. Ann Biomed Eng. 2012;40(2):516-533.
3. Agostinis P, Berg K, Cengel KA, Fuchs J, Girotti AW, Gollnick SO, *et al.* Photodynamic therapy of cancer: an

- update. *CA Cancer J Clin.* 2011;61(4):250-281.
4. Wainwright M. Photodynamic therapy—from dyestuffs to high-tech clinical practice. *Rev Prog Color Relat Top.* 2004;34(1):95-109.
 5. Dougherty TJ, Gomer CJ, Henderson BW, Jori G, Kessel D, Korbelik M, *et al.* Photodynamic therapy. *J Natl Cancer Inst.* 1998;90(12):889-905.
 6. Yano S, Hiruma M, Saito K, Abe H, Matsumoto Y. Current states and future views in photodynamic therapy. *J Photochem Photobiol C Photochem Rev.* 2011;12(1):46-67.
 7. Mehraban N, Freeman HS. Developments in PDT sensitizers for increased selectivity and singlet oxygen production. *Materials (Basel).* 2015;8(7):4421-4456.
 8. Tuner J, Hode L. Laser therapy: clinical practice and scientific background: a guide for research scientists, doctors, dentists, veterinarians and other interested parties within the medical field. Prima books; 2002.
 9. Bae C-S, Lim H-S, Park J-S, Ahn S-Y, Kim Y-H. Effect of Ga-As laser on the regeneration of injured sciatic nerves in the rat. *In vivo (Brooklyn).* 2004;18(4):489-495.
 10. Bresler S. Introduction to molecular biology. Elsevier; 2012.
 11. Dewaele M, Verschuuren E, Van Pelt J, Depuydt E, Demeyere K, Keyaerts M, *et al.* Autophagy pathways activated in response to PDT contribute to cell resistance against ROS damage. *J Cell Mol Med.* 2011;15(6):1402-1414.
 12. Bartos A, Karwowski M, Gac S, Suchorska WM, Korbelik M. Pre-conditioning with near infrared photobiomodulation reduces inflammatory cytokines and markers of oxidative stress in cochlear hair cells. *J Biophotonics.* 2016;9(11-12):1125-1135.
 13. Engel KW, Khan I, Arany PR. Cell lineage responses to photobiomodulation therapy. *J Biophotonics.* 2016;9(11-12):1148-1156.
 14. Kroemer G, Galluzzi L, Vandenabeele P, *et al.* Classification of cell death: recommendations of the Nomenclature Committee on Cell Death 2009. *Cell Death Differ.* 2009;16(1):3-11.
 15. Kim MM, Ghogare AA, Greer A, Zhu TC. On the *in vivo* photochemical rate parameters for PDT reactive oxygen species modeling. *Phys Med Biol.* 2017;62(5)
 16. Robertson CA, Evans DH, Abrahamse H. Photodynamic therapy (PDT): a short review on cellular mechanisms and cancer research applications for PDT. *J Photochem Photobiol B Biol.* 2009;96(1):1-8.
 17. Xu DD, Cho WC, Wu P, Lam HM, Leung AW. Photo-activated pheophorbide a inhibits the growth of prostate cancer cells. *Laser Phys.* 2011;21:1670-1674.
 18. Lin H-Y, Hsiao M, Yang J-S, Hsu Y-C, Lin J-T, Tsai F-J, *et al.* Demethoxycurcumin induces autophagic and apoptotic responses on breast cancer cells in photodynamic therapy. *J Funct Foods.* 2015;12:439-449.
 19. Gheewala T, Skwor T, Munirathinam G. Photodynamic therapy using pheophorbide and 670 nm LEDs exhibits anti-cancer effects in-vitro in androgen dependent prostate cancer. *Photodiagnosis Photodyn Ther.* 2018;21:130-137.
 20. Isaac-Lam MF, Mee AD. Photodynamic activity of vitamin-chlorin conjugates at nanomolar concentrations against triple-negative breast cancer cells. *ACS Omega.* 2019;4(2):2907-2920.
 21. Manoto SL, Sekhejane PR, Houreld NN, Abrahamse H. Localization and phototoxic effect of zinc sulfophthalocyanine photosensitizer in human colon (DLD-1) and lung (A549) carcinoma cells (*in vitro*). *Photodiagnosis Photodyn Ther.* 2012;9(1):52-59.
 22. Manoto SL, Houreld NN, Abrahamse H. Phototoxic effect of photodynamic therapy on lung cancer cells grown as a monolayer and three-dimensional multicellular spheroids. *Lasers Surg Med.* 2013;45(3):186-194.