

## **Protein conformational modulation by photons: a mechanism for laser treatment effects.**

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### **Abstract**

Responsiveness to low-level laser treatment (LLTT) at a wavelength of 450-910 nm has established it as an effective treatment of medical, veterinary and dental chronic pain, chronic inflammation conditions (arthritis and macular degeneration), wound repair, and lymphoedema, yet the mechanisms underlying the effectiveness of LLLT remain unclear. However, there is now sufficient evidence from recent research to propose an integrated model of LLLT action. The hypothesis presented in this paper is that external applications of photons (through laser at an appropriate dose) modulates the nervous system through an integrated mechanism. This stimulated mechanism involves protein-to-protein interaction, where two or more proteins bind together to facilitate molecular processes, including modification of proteins by members of SUMO (small ubiquitin-related modifier proteins) and also protein phosphorylation and tyrosination. SUMO has been shown to have a role in multiple nuclear and perinuclear targets, including ion channels, and in the maintenance of telomeres and the post-translational modification of genes. The consequence of laser application in treatment, therefore, can be seen as influencing the transmission of neural information via an integrated and rapid modulation of ion channels, achieved through both direct action on photo-acceptors (such as cytochrome c-oxidase) and through indirect modulation via enzymes, including tyrosine hydroxylase (TH), tyrosine kinases and tyrosine kinase receptors. This exogenous action then facilitates an existing photonic biomodulation mechanism within the body, and initiates ion channel modulation both in the periphery and the central nervous system (CNS). Evidence indicates that the ion channel modulation functions predominately through the potassium channels, including two pore leak channels (K2P), which act as signal integrators from the periphery to the cortex. Photonic action also transforms SUMOylation processes at the cell membrane, nucleus and telomeres via signalling processes from the mitochondria (which is the main target of laser absorption) to these targets. Under the hypothesis, these observed biological effects would play a part in the bystander effect, the abscopal effect, and other systemic effects observed with the application of low level laser (LLLTT). The implications of the hypothesis are important in that they point to mechanisms that can account for the effectiveness of laser in the treatment and prevention of inflammatory diseases, chronic pain and neurodegenerative disorders.

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## **Intracellular signaling cascades following light irradiation**

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### **ABSTRACT**

Low- level light therapy (LLLT) using red to near- infrared (NIR) (630–1000 nm) light has gained attention in recent years as a therapy in ophthalmology, neurology, dermatology, dentology, and regenerative medicine. Advancement in the basic science fields of photobiology has propelled LLLT into the therapeutic revolution. The potential mechanisms on LLLT- induced biological effects have been investigated by numerous researchers throughout the world. This article reviews the current intracellular signaling cascades in photobiology and photomedicine under the influence of red to NIR light on mammalian cells. Specifically, mitochondrial retrograde signaling initiated by cytochrome c oxidase photomodulation is discussed in detail in the treatment of indications using LLLT, such as vitiligo management, retinal protection, and tumor therapy. The pathways through activating receptor tyrosine kinases are also highlighted in LLLT- induced neuroprotection, wound healing, and skeletal muscle regeneration. The understanding of the LLLT- induced biological reactions in cellular and subcellular levels is crucial for the advancement of LLLT in treatment of diseases.