Low Intensity Light Therapy: Exploring the Role of Redox Mechanisms

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Abstract:

Low-intensity light therapy (LILT) appears to be working through newly recognized photoacceptor systems. The mitochondrial electron transport chain has been shown to be photosensitive to red and near-infrared (NIR) light. Although the underlying mechanisms have not yet been clearly elucidated, mitochondrial photostimulation has been shown to increase ATP production and cause transient increases in reactive oxygen species (ROS). In some cells, this process appears to participate in reduction/oxidation (redox) signaling. Redox mechanisms are known to be involved in cellular homeostasis and proliferative control. In plants, photostimulation of the analogous photosynthetic electron transport chain leads to redox signaling known to be integral to cellular function. In gene therapy research, ultraviolet lasers are being used to photostimulate cells through a process that also appears to involve redox signaling. It seems that visible and near visible low-intensity light can be used to modulate cellular physiology in some nonphotosynthetic cells, acting through existing redox mechanisms of cellular physiology. In this manner, LILT may act to promote proliferation and/or cellular homeostasis. Understanding the role of redox state and signaling in LILT may be useful in guiding future therapies, particularly in conditions associated with pro-oxidant conditions.