EFFECT OF VISIBLE SPECTRUM OF ELECTROMAGNETIC RADIATION (VISIBLE LIGHT) ON MITOCHONDRIA: A SYSTEMATIC REVIEW

S.T.Y Azeemi[1, 4], Maida Siddiqui[2, 4], Saadia Azeemi[3, 4]

[1] Govt. Postgraduate College For Women, Samanabad, Lahore.
[2] School of Biological Sciences, University of the Punjab.
[3] Postgraduate Resident, General Surgery, Punjab Rangers Teaching Hospital,Lahore.
[4] Khawaja Shamsuddin Azeemi Institute of Colour Therapy, Lahore.
Corresponding Author E-mail:saminatazayyen@yahoo.com

Background: Mitochondria play a central role in cellular energy production, and their function can be influenced by external stimuli such as light. While red and near-infrared (NIR) light are well-studied in photobiomodulation (PBM), the effects of other visible light wavelengths (blue and green) on mitochondrial function remain underexplored.

Methodology: This systematic review followed the PRISMA 2020 guidelines to synthesize experimental findings from provided research materials. Studies involving exposure to visible light (400–700 nm) and reporting outcomes related to mitochondrial function were included.

Results: Red light (~600–700 nm) predominantly enhanced mitochondrial activity, increasing ATP production, MMP, and O_2 consumption, with CCO identified as a primary photoacceptor. Blue light (~400–500 nm), in contrast, was associated with elevated ROS production, decreased MMP and ATP, and increased mitochondrial dysfunction, potentially through flavins, porphyrins, and opsins. Green light (~500–570 nm) showed mixed effects, with both inhibitory and stimulatory outcomes depending on the cell type and exposure parameters. The mechanisms underlying these effects involve a complex interplay of wavelength-specific photoacceptors and downstream signaling pathways.

Conclusion: Visible light modulates mitochondrial function in a wavelength-dependent manner.

Keywords: Mitochondria, Photobiomodulation, Visible Light, Red Light, Blue Light, Green Light.

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INTRODUCTION

Mitochondria are essential organelles within eukaryotic cells, renowned for their indispensable role as the primary sites of cellular energy production [1]. Through the process of oxidative phosphorylation, mitochondria efficiently convert energy stored in metabolic substrates into adenosine triphosphate (ATP), the universal energy currency of the cell [3]. This intricate process occurs via the electron transport chain (ETC), a series of protein complexes embedded within the highly folded inner mitochondrial membrane (IMM), which establishes a proton gradient subsequently utilized by ATP synthase [3].

Visible light constitutes a narrow band of the electromagnetic spectrum, typically defined as wavelengths between approximately 400 nanometers (nm) (violet/blue) and 700 nm (red) [14]. This range includes blue (400-500 nm), green (500-570 nm), yellow/orange (570-600 nm), and red (600-700 nm) light [7]. Historically, the biological impact of visible light was often underestimated compared to higher-energy ultraviolet (UV) radiation [16]. However, it is now evident that visible light photons possess sufficient energy to interact with biological tissues and elicit significant photochemical and photophysical effects [14], which forms the basis of their usage in Colour Therapy [11, 29, 33].This recognition gains further relevance in the modern era due to increased human exposure to artificial light sources, such as light-emitting diodes (LEDs), which often have distinct spectral profiles [17].

The foundational principle of photobiology states that light must first be absorbed by specific molecules, termed chromophores or photoacceptors, within the tissue to initiate a biological response [7]. Photobiomodulation (PBM), a therapeutic modality also widely known as Low-Level Light Therapy (LLLT), leverages this principle [7]. PBM typically utilizes non-ionizing light, often generated by lasers or LEDs, within the red (approx. 600-700 nm) and near-infrared (NIR, approx. 700-1100 nm) spectral regions, applied at low power densities to avoid thermal damage [5].

The therapeutic goals of PBM are diverse, including the stimulation of tissue repair and regeneration (e.g., wound closure, bone formation), reduction of pain, inflammation, and edema [5]. While red and NIR light

are the most studied wavelengths for PBM, research is increasingly exploring the potential biological and therapeutic effects of other visible wavelengths, including blue and green light [14]. These wavelengths may offer distinct mechanisms of action and applications, for instance in dermatology or antimicrobial therapies [14].

Despite significant research progress, particularly in the field of red/NIR PBM, considerable debate and uncertainty remain regarding the precise molecular mechanisms, the identity of primary photoacceptors across the full visible spectrum (beyond CCO for red/NIR), the reasons for observed wavelength-specific effects, and the optimal parameters (dose, irradiance, wavelength) for achieving desired biological outcomes [10]. Lack of standardization in research protocols and parameter reporting further complicates interpretation and comparison of studies [14]. Critiques have also been raised regarding the universality of the CCO-centric mechanism even for red/NIR light, suggesting other pathways may contribute significantly [10].

This systematic review aims to address these knowledge gaps by synthesizing the available evidence. The specific objectives are:

- To systematically review and synthesize the reported effects of visible spectrum light (focusing on blue, green, and red wavelengths) on mitochondrial structure, function, and associated signaling pathways, based on the provided research material.
- To identify and evaluate the key mitochondrial parameters modulated by visible light exposure, including ATP synthesis, ROS levels, mitochondrial membrane potential (MMP), respiratory chain activity (including CCO function), mitochondrial dynamics, and involvement in cell fate decisions (apoptosis/necrosis).
- To examine and compare the proposed photoacceptors (e.g., CCO, flavins, porphyrins, opsins, interfacial water) and downstream molecular mechanisms implicated in mediating the effects of different visible wavelengths.
- To summarize the current understanding derived from the synthesized evidence, highlighting areas of established understanding, points of controversy, limitations apparent from the source material, and identifying critical gaps for future research.

METHODS

This systematic review was performed and structured following the recommendations of the PRISMA 2020 statement [27]. The PRISMA 2020 checklist guided the reporting of methods and results to ensure transparency and completeness.

The selection of information for this review was based on the following criteria applied to the provided research snippets:

- **Study Selection**: Studies describing exposure to electromagnetic radiation within the visible light spectrum (~400-700 nm) were included. Specific focus was placed on data pertaining to blue (~400-500 nm), green (~500-570 nm), and red (~600-700 nm) light. Information on light source (LED, laser), wavelength, irradiance (mW/cm²), radiant exposure (dose, J/cm²), exposure duration, and pulsing parameters was extracted when provided. Contextual data on NIR light (>700 nm) was included when discussed in relation to red light PBM [3].

- **Outcomes**: The primary outcomes of interest were direct or indirect measures of mitochondrial function and structure, including ATP levels [2], ROS generation (including specific species like superoxide) [1], mitochondrial membrane potential (MMP) [6], oxygen consumption or respiratory chain activity [3], CCO activity or modulation [3], mitochondrial dynamics (fission/fusion) [13], and mitochondrial involvement in cell death pathways (apoptosis, necroptosis) [4]. Secondary outcomes included related cellular responses like proliferation, viability, migration, differentiation, and inflammation, where linked to mitochondrial effects.

RESULTS

The interventions described cover the visible light spectrum, with particular data available for blue (~400-500 nm), green (~500-570 nm), and red (~600-700 nm) wavelengths [7]. Near-infrared (NIR, ~700-1100 nm) light is frequently mentioned, primarily in the context of PBM studies that often investigate red and NIR light together [5]. Light sources include both LEDs and lasers.

The reported outcomes primarily focus on mitochondrial parameters such as ATP production, ROS levels, MMP, oxygen consumption/respiration, CCO activity, and mitochondrial dynamics. Downstream cellular consequences like viability, proliferation, apoptosis, inflammation, and signaling pathway activation are also frequently discussed.

The effects of visible light on mitochondria vary significantly depending on the wavelength, dose, and cellular context. The following sections synthesize the findings for red, blue, and green light, followed by comparative analyses and mechanistic discussions based on the included papers.

Effects of Red Light (~600-700 nm) on Mitochondria: Red light in the 630–670 nm range is the most intensively investigated portion of the photobiomodulation (PBM) spectrum and is repeatedly linked to modulation of mitochondrial activity.

Cytochrome c oxidase (CCO; Complex IV of the electron-transport chain) is generally regarded as the primary photoacceptor for these wavelengths, because PBM action spectra closely parallel CCO absorption spectra [5] and red light can reverse the effects of CCO inhibitors [8]. The oxidized CuA and CuB centers appear especially photosensitive [8]. A leading mechanistic model proposes that photons displace inhibitory nitric oxide (NO) from CCO, thereby restoring electron flow, raising oxygen consumption, and increasing proton pumping, effects that are particularly pronounced in metabolically stressed cells [5, 7], although the centrality of CCO to all PBM outcomes remains contested [10].

Downstream mitochondrial responses are well documented. Red light commonly elevates the mitochondrial membrane potential ($\Delta\Psi m$); for example, 660 nm irradiation at 3 J cm^{-2} produces an 8–15 % rise in human adipose-derived stem cells (hASCs) [21], peaks 3–6 h after exposure in C2C12 myotubes [28], and remains sustained in human fibroblasts treated with a 645 nm LED [26]. A concomitant increase in ATP synthesis is a hallmark outcome, with the same 660 nm dose boosting ATP in hASCs by 15–20 % [21], 670 nm light enhancing ATP-synthase flux in the aging human brain [2], and 660 nm producing a more durable ATP rise than 980 nm [20]; the response shows a biphasic dose profile [21] and depends on both ATP-synthase integrity [20] and substrate availability [19]. Red-light exposure also provokes a transient, modest rise in mitochondrial reactive-oxygen species (ROS) in healthy cells, initiating NF-κB-dependent adaptive signaling that can be blocked by antioxidants [5, 7]; in stressed cells, however, red or near-infrared irradiation often lowers net ROS by improving ETC efficiency or up-regulating antioxidant defenses, as observed in fibroblasts treated with a 636 nm laser [22]. In line with CCO activation, most studies report elevated oxygen consumption, though exceptions exist: osteoblast spheroids showed reduced basal OCR after 660 nm PBM yet an increase in maximal respiration with 808 nm irradiation, underscoring wavelength-specific metabolic nuances [31].

Collectively, these mitochondrial effects translate into improved cellular performance. Red-light PBM promotes proliferation [21], migration [32], and survival under diverse stressors [9]; guides stem-cell differentiation patterns [18]; attenuates inflammation [5]; accelerates wound closure [5]; and confers neuroprotection [9]. Thus, despite ongoing debate about precise primary targets, red-light PBM reproducibly enhances mitochondrial function and triggers a cascade of bioenergetic and signaling events with broad therapeutic potential.

Effects of Blue Light (~400-500 nm) on Mitochondria: Blue light in the 390–500 nm window engages mitochondria through mechanisms that diverge markedly from those of red or near-infrared irradiation and can yield both adverse and context-dependent therapeutic outcomes. Several chromophores absorb within this range. Porphyrin-containing heme centers, most prominently the Soret band of cytochromes such as CCO (~400–420 nm), may modulate electron transport but generate functional effects distinct from those elicited by red light-mediated CCO activation [7, 14]. Flavins associated with Complex I (FMN) and Complex II (FAD) absorb at ~450–460 nm; their excitation is tightly coupled to the formation of reactive oxygen species (ROS), including singlet oxygen and superoxide [14]. Non-visual opsins (e.g., melanopsin OPN4 at ~479 nm and OPN3) provide a parallel sensing route, acting via Ca^{2+} or ROS-dependent second-messenger pathways that ultimately influence mitochondrial function [7]. Additional photoacceptor candidates include nitrosated proteins [16] and tryptophan photo-oxidation products [7], underscoring the chemical diversity of blue-light targets.

Because flavin and porphyrin photochemistry efficiently yields ROS, blue light almost invariably elevates mitochondrial and cytosolic ROS to levels far exceeding those induced by red/NIR PBM [7, 14]. When antioxidant defenses are overwhelmed, this oxidative load precipitates mitochondrial depolarization: retinal pigment epithelial cells, for example, lose 55–60 % of their membrane potential after 30 min [24], and human adipose-derived stem cells (hASCs) exposed to 415 nm at 3 J cm^{-2} show similar declines [21]. Depolarization, together with putative opening of the mitochondrial permeability-transition pore, suppresses ATP synthesis, hASCs exhibit roughly a 10 % ATP drop under the same 415 nm parameters, with larger deficits at higher doses [21], and can inhibit respiration outright [25]. Yet blue light is not uniformly inhibitory: under certain conditions it can partially reverse nitric-oxide-mediated respiratory block, illustrating a nuanced, wavelength- and redox-state-dependent bioenergetic profile [14].

At the structural level, 450 nm illumination triggers fission events, elevating Drp1 and reducing Mfn2 in R28 neuronal-precursor cells [13], hallmarks of mitochondrial stress. The cellular consequences, including damage, cytotoxicity, apoptosis, and necroptosis, are well documented in retinal tissues [12]. Notably, blue light has been shown to inhibit proliferation in human adipose-derived stem cells (hASCs) [21] and fails to stimulate proliferation in HUVECs [32], whereas red and green light enhance it.Nevertheless, carefully tuned blue-light regimens (typically 415 nm or 470 nm) confer therapeutic advantages where shallow penetration suffices: they exhibit potent bactericidal action, aid wound healing, reduce superficial inflammation, and are widely used for acne management [14]. Indeed, a systematic review found that 72 % of blue-light–focused studies reported beneficial effects [14], highlighting how dose, exposure

time, and tissue optics dictate whether blue light becomes a cytotoxic threat or a valuable clinical tool.

Effects of Green Light (~500-570 nm) on Mitochondria: Green light in the 500–570 nm band is comparatively under-studied and its mitochondrial actions appear more heterogeneous than those of red or blue light. Action-spectra peaks near 540 nm point to cytochrome-based photoacceptors, possibly CCO or other respiratory cytochromes, while weak flavin absorption tails into the green could still generate reactive-oxygen species (ROS) [14, 35]. Additional photoreceptors have been proposed, including green-sensitive opsins or light-gated ion channels such as TRPV family members [23], and even dietary chlorophyll metabolites (e.g., pyropheophorbide-a) that might harvest green photons to support mitochondrial ATP production, though in-vivo relevance remains uncertain [34]. Functionally, green light often elicits a moderate ROS burst, intermediate between the low levels typical of red light and the high spikes induced by blue light, as seen in human adipose-derived stem cells (hASCs) exposed to 540 nm at 3 J cm^{-2} [21], and has been implicated in photo-aging through MMP-1 up-regulation in fibroblasts [15]. Concomitantly, the same 540 nm dose reduces mitochondrial membrane potential and lowers ATP content by about 10 % in hASCs, with greater declines at higher fluences [21]. Cellular outcomes are correspondingly mixed: hASCs exhibit reduced proliferation under green light [21], yet a systematic review reported beneficial effects in 75 % of studies, citing improvements in cellulite and edema, stimulation of endothelial cell growth and migration, promotion of myogenesis, acceleration of wound repair, osteogenic differentiation, and angiogenesis [14]. Indeed, a 532 nm laser enhanced proliferation and produced anti-inflammatory effects in human adipose-derived mesenchymal stem cells (hADMSCs) [30]. Such discrepancies underscore the strong influence of wavelength, dose, exposure time, and cell type, exacerbated by inconsistent reporting, while limited tissue penetration further constrains in vivo applications [5].

Conclusion: Visible light effects on mitochondria result from a complex interplay of multiple photoacceptors (CCO, flavins, opsins, etc.) and pathways (NO, ROS, Ca^{2+}). The specific mechanism depends heavily on wavelength, dose, cell type, and metabolic state. The CCO-centric view is important for red/NIR but insufficient for the full spectrum, especially blue/green light where flavins, opsins, and possibly ion channels play major roles.

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