

EDITORIAL

Should Low-Dose Biophotonic Therapy Have a Role in the Treatment of Type 2 Diabetes (T2DM)

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Humans have always instinctively sought daylight and the sun's rays for many generations while seeking relief from many sorts of illnesses including infectious illnesses, wound healing and other maladies common to man and animals. Sunshine is deemed a reliable source of quanta of photons, a likely physiologic prerequisite for multiple aspects of mammalian health and vitality. Sunlight is known to deliver photonic energy, and while not exactly a reliable cure-all, it may contribute as a healthful adjunct to the healing process.¹⁻³ The oldest references to the benefits of sun therapy were reported on or before 1500 B.C. and as noted, references to multiple beneficial aspects of sun therapy continue to abound today. Although the molecular basis of photon-mediated therapies remain unclear or speculative, it is recognized that emerging findings of UV-derived light from sunlight or *in vivo*-derived sources now point to a nuclear disruptive element as a central factor in the ultimate wound healing process.^{4,5} Depending on the intensity and wave-length of the UV exposure, it appears to impede further local replication of an invasive infectious agent, combined with an enhancement of immunologic responses in the UV- or sunlight-exposed host. Oxygen availability is deemed essential for optimal wound healing, and the associated immune responses are also likely linked at least in part to enhanced oxygen delivery to peripheral tissues.⁵

According to the concepts of photobiology, light absorption requires the presence of a specific photo-acceptor molecule or complex that after photonic excitation could induce the downstream activation of biochemical or physiologic signaling pathways to bring about its desired healthful or other responses. Among the best documented photobiologic effects of sunlight is the dermal activation of vitamin D, and its link to immune related and other functions following activation.⁶ The blood protein Hemoglobin also contains additional photon energy absorbing capacity. The blood protein hemoglobin (Hb) is recognized to be an efficient light absorbing photochemical structure that is capable of absorbing energy from photons due to its unique electronic and atomic configurations, with an optimum wavelength at ~ 480 nm, in addition to the aromatic amino acids Tryptophan (Trp) and Tyrosine (Tyr) in the β -side chain that also absorb light at complimentary UV bandwidths that may serve to extend the energy absorbing spectral range.^{2,3} This oxygen-linked energy function also enables hemoglobin to undergo reversible taut vs relaxed states and thus accommodate the efficient transport and release of life-giving oxygen to peripheral tissues, in addition to its contributions to gas transport, buffering capacity, uptake of 2,3 deoxy diphosphoglycerate (2,3 DPG) and other critical biologic functions.^{7,8} In animal studies with type two diabetic (T2DM) mice, measures of insulin resistance, including GLUT4 activity, Oral Glucose Tolerance, Insulin response to a glucose challenge, the area under the OGT curve (AUC_{glc}), tissue ATP content, and glycogen synthesis in skeletal muscle were all found to be significantly improved following biophotonic treatments in the diabetic mice.⁹ The parameters of carbohydrate metabolism in rodents and humans are remarkably similar, and in human studies, Miley reported that blood oxygen saturation improved for several weeks following biophotonic exposure, a likely contribution to the improved wound healing and other wellness parameters reported in his studies.⁵ More recently it has been reported in a small clinical case study of prediabetic patients that hemoglobin A1c, a reliable clinical marker of glycemic status, was also improved proportionately to the improvements in blood oxygen saturation.^{7,10,15}

Glycated hemoglobin can exert pathophysiologic actions in vascular tissues and their supporting structures.¹⁶ Because Glycation results in movement of the hemoglobin saturation curve to the left, it impedes the release of oxygen from the hemoglobin moiety, and contributes to a state of relative hypoxia in erythrocytes and their supported tissues.¹¹ Hypoxia is a contributor to formation of inflammatory highly reactive oxygen species (iROS) and generation of damaging free radicals within the affected erythrocytes and in erythrocyte membrane damage.¹² The membrane damage contributes to an increased propensity for erythrocyte aggregation, an increase in the blood viscosity, and in impaired peripheral blood flow. Glycated hemoglobin also contributes to inflammation of endothelial cells, and to contributes to inflammation of endothelial cells

and to formation of atherosclerotic plaque. If left unchecked, the inflammatory responses can also bring alterations in the redox state of Fe^{2+} in hemoglobin to form Fe^{3+} - and Fe^{4+} - hemoglobin, that are capable of penetrating the subendothelium of veins and arteries, causing greater permeability and further tissue damage. Thus, clinical interventions that can reduce or reverse the formation of glycated hemoglobin *in vivo* are of considerable potential therapeutic potential in management of T2DM.

The Common primary UV band widths of Sunlight include three major bands: UVA (320–400 nm), UVB (280–320 nm), and UVC (200–280 nm).^{2,3,14} The UVA band is less energetic than that of UVB or UVC but up to 20 times more intense. Over exposure to the UVB band can lead to the formation of cyclobutene pyrimidine dimers (CPDs) and other damaged photoproducts including pyrimidine (6–4) and pyrimidones (64PPs) that are also inhibitory to *in vivo* genome replication in host cells and tissues. The greater intensity of the UVC band can induce the formation of (CPDs) in addition to a variety of oxidatively generated lesions including single strand RNA breaks and oxidized bases that also effectively impede further replication of the viral or microbial genome by the receptive host cells. Exposure to the UVC band can also cause photochemical structural damage to both DNA and RNA polymers, disrupts cross-linking events, and generates oxidative damage to the nucleotide bases, also inhibiting further genome replication of the infectious agent.¹⁴ Thus the stronger nature of the UVC band has been determined to contain the most effective antiviral and antimicrobial properties.¹⁵ As noted, exposure to Red light in the wavelength of 660 nm may further augment the virucidal responses, as phototherapy with red light further enhances the biophotonic responses, in that the red light irradiation enables the participating entities to absorb additional energy, which is then believed to be transferred to the oxygen molecules contained in the virus, thereby making the oxygenation more highly oxidized, and causing further irreversible damage to the membranes and genomic material of the viral particles and microbial entities.^{13,14} In contrast, favorable ROS are able to activate redox sensitive signal transduction pathways such as Nrf-2, NF- κ B, ERK and cytokines which collectively act as key redox checkpoints in cell cycle survival processes and replication mechanisms. Thus, the more healthful biostimulation process that occurs following controlled, low level intensity UV photonic emission is proposed to promotes cell survival and proliferation in both *in vitro* and *in vivo* applications. Thus, for UV exposure, the old adage of ‘the dose makes the poison’ clearly applies, as biologic exposure to UV irradiation may induce either potentially harmful or beneficial effects on key physiologic parameters, depending on the intensity, duration, and wavelength of the UV source applied. Collectively these observations suggest that the

application of low dose, biophotonic therapy at controlled wavelengths may extend beyond its more commonly applied applications for the treatment and control of infectious illnesses and anti-aging therapeutics and now may also include important potential beneficial effects in insulin resistant conditions typical of type 2 diabetes mellitus.

Excess UV irradiation may be harmful to both mammalian species and invasive, infectious agents, however. Typical UV light exposure consists of A, B and C bands.^{2,3,13} The high-energy UV-C, X-rays, and gamma rays can cause single and double strand DNA breakage, deemed essential in the inactivation of infectious microbial, viral, and parasitic agents. Once the genomic material is inactivated, further replication of the infectious agent becomes impaired. In addition, the higher dose UV exposure may bring about the denaturation of microbial or viral proteins, which may initiate an immunogenic response to the now foreign protein.¹³ In contrast, exposure to non-ionizing radiation can induce the formation of dimers between two adjacent pyrimidine bases of RNA, and in both cases of ionizing or nonionizing exposure the usual consequence is also the prevention of further *in vivo* replication of an infectious agent.¹⁴ The consensus is that that the denaturation of the viral or microbial genetic material occurs is likely to be the primary and more efficacious target for irradiation-induced viral and microbial inactivation. In contrast, chemical inactivation of virions and microbial agents as occurs with disinfectant agents, imparts chemical damage to the protein-lipid envelopes, also precluding continued viability of the infectious agent.

In conclusion, the observations of biophotonic-linked decreases in HbA1c concentrations represent a new observation with important applications to clinical management of diabetic status. The molecular mechanism of lowering HbA1c concentrations remains unclear in the studies cited.^{4,5,7} However, biophonic or biophysical processes that can bring about a lowering the percent of HbA1c improves oxygen release from oxyhemoglobin and likely decreases the magnitude of potential pathophysiologic effects of HbA1c. The biostimulation process also improves peripheral oxygen delivery to tissues via what is consistent with a laser-induced photodissociation of oxygen from oxyhemoglobin in cutaneous blood vessels, that results in increasing blood pO₂ saturation concentrations in peripheral circulation. The improved O₂ delivery further contributes to its essential roles in the biomedical processes of tissue regeneration and healing in humans and animals.

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Conflicts of Interest

None

USE of Artificial Intelligence

The author reports that no applications of AI were utilized in the generation of this manuscript.

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