Propionibacterium Acnes Susceptibility To Low-Level 449 nm Blue Light Photobiomodulation

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Background and Objective: Recent advances in low-level light devices have opened new treatment options for mild to moderate acne patients. Light therapies have been used to treat a variety of skin conditions over the years but were typically only available as treatments provided by professional clinicians. Clinical application of blue light has proven to be effective for a broader spectral range and at lower fluences than previously utilized. Herein, we tested the hypothesis that sub-milliwatt/cm^2 levels of long-wave blue light (449 nm) effectively kills *Propionibacterium acnes*, a causative agent of acne vulgaris, *in vitro*.

Materials and Methods: Two types of LED light boards were designed to facilitate *in vitro* blue light irradiation to either six-well plates containing fluid culture or a petri plate containing solid medium. *P. acnes*. Survival was determined by counting colony forming units (CFU) following irradiation. *P. acnes* was exposed in the presence and absence of oxygen. Coproporphyrin III (CPIII) photoexcitation was spectrophotometrically evaluated at 415 and 440 nm to compare the relative photochemical activities of these wavelengths.

Results: 422 and 449 nm blue light killed *P. acnes* in planktonic culture. Irradiation with 449 nm light also effectively killed *P. acnes* on a solid agar surface. Variation of time or intensity of light exposure resulted in a fluence-dependent improvement of antimicrobial activity. The presence of oxygen was necessary for killing of *P. acnes* with 449 nm light. CPIII displayed clear photoexcitation at both 415 and 440 nm, indicating that both wavelengths are capable of initiating CPIII photoexcitation at low incident light intensities (50 uW/cm^2).

Conclusion: Herein we demonstrate that sub-milliwatt/cm^2 levels of long-wave blue light (449 nm) effectively kill *P. acnes*. The methods and results presented allow for deeper exploration and design of light therapy treatments. Results from these studies are expanding our understanding of the mode of action and functionality of blue light, allowing for improved options for acne patients. Lasers Surg. Med. © 2019 Wiley Periodicals, Inc.

Key words: blue light; *P. acnes*; low dose; *C. acnes* low-level light therapy; porphyrin; photobiomodulation

BACKGROUND

Acne vulgaris is a chronic skin disorder of the pilosebaceous unit [1,2]. There are multiple pathogenic factors for acne, including increased sebum production, obstructed pilosebaceous units by abnormal follicular keratinization, follicular overgrowth of *Propionibacterium acnes* (*P. acnes*), and inflammation [3]. *P. acnes* contributes to inflammatory acne by activating Toll-like receptors (TLRs) [4,5] and stimulating skin cells to produce interleukin (IL)-1α, tumor-necrosis factor (TNF)-α, and chemokines (e.g., IL-8) [6–12]. *P. acnes* can also induce tissue injury directly [12–17]. For example, *P. acnes* isolated from acne lesions has been shown to produce chondroitin sulfatase and hyaluronidase which may cause extracellular matrix degradation leading to tissue injury [13]. As such, acne vulgaris treatment regimens overwhelmingly include strategies to reduce the level of *P. acnes* on the skin.

Acne treatments may be administered either topically or systemically. Over-the-counter (OTC) topical products for acne including salicylic acid, benzoyl peroxide, alpha hydroxy acids and sulfur, have been used to mitigate mild to moderate acne. Systemic treatments for severe acne include isotretinoin, oral antibiotics, and hormonal agents [18]. Combination treatments targeting multiple underlying factors of acne pathogenesis yield more successful results; however, *P. acnes*, a gram-positive bacterium, can develop resistance to commonly used topical and systemic anti-acne treatments. The use of

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