Lessons learned from computer models on blue light therapy for psoriasis vulgaris

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Blue light irradiation has been clinically proven to reduce the symptoms of psoriasis vulgaris, a common chronic inflammatory skin disease that affects 2% - 3% of the world's population. This dermatological condition is characterized by hyperproliferation and disturbed differentiation of keratinocytes, which is evident in lesional areas as thick flaky skin. The lesional areas also exhibit sustained inflammation, induced by immune cells, such as T cells and dendritic cells, infiltrating the affected skin. Blue light reduces the proliferation of keratinocytes and increases their differentiation in a wavelength and fluence dependent manner. Also, it induces apoptosis in T cells and suppresses the activation of dendritic cells. These effects can explain the symptom reduction after treatment. But, the efficacy shown in the clinical studies could be further improved by having a deeper understanding on the underlying mechanism of this therapeutic approach and optimizing the treatment regimens currently used. Diverse findings have been published in different studies of blue light therapy for psoriasis describing large, little or no therapeutic effect. These results may be due to variances in the main treatment parameters of the implemented protocols, i.e. fluence, intensity, and length of treatment. Computational methods can provide a suitable platform to investigate the complex interactions leading to the management of psoriasis by blue light therapy and optimize the treatment protocols. Here, we explore *in silico* the underlying mechanism of blue light irradiation of psoriatic skin and predict the outcome of a wide range of therapeutic regimens with varying fluence, intensity, and length of treatment. The computational model is defined by a set of ordinary differential equations describing the time evolution of keratinocytes as they move vertically through the layers of the epidermis. The results of our simulations suggest that the temporary decrease in the severity of psoriasis can be explained by the transient decline in the proliferative capacity of keratinocytes. However, it is still unclear how the effects of blue light on the immune system contribute to the reduction of psoriasis symptoms. Simulations implemented for several combinations of treatment parameters predict that high efficacy is achieved by protocols with long duration and high fluence levels, regardless of the chosen intensity. These predictions provide general guidelines for treatment. Our *in silico* approach constitutes a framework for testing diverse hypotheses on the underlying mechanism of blue light therapy and designing effective strategies for the treatment of psoriasis vulgaris.