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Photobiomodulation directly benefits primary neurons functionally inactivated by toxins: role of cytochrome c oxidase.

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Abstract

Far red and near infrared (NIR) light promotes wound healing, but the mechanism is poorly understood. Our previous studies using 670 nm light-emitting diode (LED) arrays suggest that cytochrome c oxidase, a photoacceptor in the NIR range, plays an important role in therapeutic photobiomodulation. If this is true, then an irreversible inhibitor of cytochrome c oxidase, potassium cyanide (KCN), should compete with LED and reduce its beneficial effects. This hypothesis was tested on primary cultured neurons. LED treatment partially restored enzyme activity blocked by 10-100 microm KCN. It significantly reduced neuronal cell death induced by 300 microm KCN from 83.6 to 43.5%. However, at 1-100 mm KCN, the protective effects of LED decreased, and neuronal deaths increased. LED significantly restored neuronal ATP content only at 10 microm KCN but not at higher concentrations of KCN tested. Pretreatment with LED enhanced efficacy of LED during exposure to 10 or 100 microm KCN but did not restore enzyme activity to control levels. In contrast, LED was able to completely reverse the detrimental effect of tetrodotoxin, which only indirectly down-regulated enzyme levels. Among the wavelengths tested (670, 728, 770, 830, and 880 nm), the most effective ones (830 nm, 670 nm) paralleled the NIR absorption spectrum of oxidized cytochrome c oxidase, whereas the least effective wavelength, 728 nm, did not. The results are consistent with our hypothesis that the mechanism of photobiomodulation involves the up-regulation of cytochrome c oxidase, leading to increased energy metabolism in neurons functionally inactivated by toxins.

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