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Effect of Near-Infrared Light Exposure on Mitochondrial Signaling in C_2C_{12} Muscle Cells

Linda M.-D. Nguyen, Angelina G. Malamo, Kelly A. Larkin-Kaiser, Paul A. Borsa, Peter J. Adhihetty

Department of Applied Physiology and Kinesiology, University of Florida, Gainesville, FL 32611, United States

Highlights:

- NIR light exposure activates mitochondrial signaling in C_2C_{12} muscle cells.
- Chronic NIR light exposure (4d) activates mitochondrial regulatory proteins.
- Mitochondrial responses to NIR light may involve ROS signaling.
- Mitochondrial adaptations may contribute to NIR light therapeutic benefits.

Abstract: Near-infrared (NIR) light is a complementary therapy used to treat musculoskeletal injuries but the underlying mechanisms are unclear. Acute NIR light treatment (~800–950 nm; 22.8 J/cm^2) induced a dose-dependent increase in mitochondrial signaling (AMPK, p38 MAPK) in differentiated muscle cells. Repeated NIR light exposure (4 days) appeared to elevate oxidative stress and increase the upstream mitochondrial regulatory proteins AMPK (3.1-fold), p38 (2.8-fold), PGC-1α (19.7%), Sirt1 (26.8%), and reduced RIP140 (23.2%), but downstream mitochondrial regulation/content (Tfam, NRF-1, Sirt3, cytochrome c, ETC subunits) was unaltered. Our data indicates that NIR light alters mitochondrial biogenesis signaling and may represent a mechanistic link to the clinical benefits.

Key Words: Mitochondrial biogenesis; Oxidative stress; NIR light; Reactive oxygen species; Photobiomodulation