Near infrared light protects cardiomyocytes from hypoxia and reoxygenation injury by a nitric oxide dependent mechanism

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Abstract

Photobiomodulation with near infrared light (NIR) provides cellular protection in various disease models. Previously, infrared light emitted by a low-energy laser has been shown to significantly improve recovery from ischemic injury of the canine heart. The goal of this investigation was to test the hypothesis that NIR (670 nm) from light emitting diodes produces cellular protection against hypoxia and reoxygenation-induced cardiomyocyte injury. Additionally, nitric oxide (NO) was investigated as a potential cellular mediator of NIR.
Our results demonstrate that exposure to NIR at the time of reoxygenation protects neonatal rat cardiomyocytes and HL-1 cells from injury, as assessed by lactate dehydrogenase release and MTT assay. Similarly, indices of apoptosis, including caspase 3 activity, annexin binding and the release of cytochrome c from mitochondria into the cytosol, were decreased after NIR treatment. NIR increased NO in cardiomyocytes, and the protective effect of NIR was completely reversed by the NO scavengers carboxy-PTIO and oxyhemoglobin, but only partially blocked by the NO synthase (NOS) inhibitor L-NMMA. Mitochondrial metabolism, measured by ATP synthase activity, was increased by NIR, and NO-induced inhibition of oxygen consumption with substrates for complex I or complex IV was reversed by exposure to NIR. Taken together these data provide evidence for protection against hypoxia and reoxygenation injury in cardiomyocytes by NIR in a manner that is dependent upon NO derived from NOS and non-NOS sources.