



Low Level Laser Therapy & SPINAL CORD INJURY clinical research

LIGHT THERAPY (LLLT) ALTERS GENE EXPRESSION AFTER ACUTE SPINAL CORD INJURY

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Secondary injury in the spinal cord, which results in axonal degeneration, scar and cavity formation and cell death, occurs around the site of the initial trauma and is a primary cause for the lack of axonal regeneration observed after spinal cord injury (SCI). The immune response after SCI is under investigation as a potential mediator of secondary injury. Treatment of SCI with 810 nm light suppresses the immune response and improves axonal regeneration. We hypothesize that these beneficial effects observed in the injured spinal cord are accompanied by alterations in gene expression within the spinal cord, particularly of those genes involved in secondary injury and the immune response. To test this hypothesis, a dorsal hemisection at vertebral level T9 was performed. The injured spinal cord from rat was then exposed to laser light (810nm, 150mW, 2,997 seconds, 0.3cm² spot area, 1589 J/cm²) and spinal cord samples, including the injury site, were harvested at 6 and 48 hours and 4 days post-injury. Total RNA was extracted and purified from the lesioned spinal cord and cDNA copies were either labeled with [32P] for microarray analysis or amplified and analyzed with a polymerase chain reaction (PCR). Microarray results revealed a suppression of genes involved in the immune response and excitotoxic cell death at 6 hours post-injury, as well as cell proliferation and scar formation at 48 hours post-injury in the light treated group. Analysis of the PCR products revealed that light treatment resulted in a significant suppression of expression of genes that normally peak between 6 and 24 hours post-injury, including the pro-inflammatory cytokine interleukin 6 (IL6), the chemokine monocyte chemoattractant protein 1 (MCP- 1) and inducible nitric oxide synthase (iNOS; $p < 0.05$). Genes expressed earlier than 6 hours post-injury, such as IL1b, tumor necrosis factor α (TNF α) and macrophage inflammatory protein 1a (MIP-1a) were not affected by light treatment. Although the precise role some of these genes play in axonal regeneration after spinal cord injury is currently unclear, these data demonstrate that light therapy has an antiinflammatory effect on the injured spinal cord, and may reduce secondary injury, thus providing a possible mechanism by which light therapy may result in axonal regeneration.

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