Int J Immunopathol Pharmacol, 23 (4), 1167-76 Oct-Dec 2010

Inhibition of HSV-1 Replication by Laser Diode-Irradiation: Possible Mechanism of Action

G Donnarumma [1], V De Gregorio, A Fusco, E Farina, A Baroni, V Esposito, M Contaldo, M Petruzzi, G Pannone, R Serpico

Affiliations
PMID: 21244765 DOI: 10.1177/039463201002300420

Abstract

Herpes labialis are the most frequent clinical manifestations of HSV-1 infection. Epithelial cells are able to respond to HSV-1 presence inducing the expression of IL-6, IL-1, TNF-α and IL-8. These proinflammatory cytokines have a function in the acute-phase response mediation, chemotaxis, inflammatory cell activation and antigen-presenting cells. In the human epithelial cell models, it has been demonstrated that, after an early induction of proinflammatory host response, HSV-1 down-modulates the proinflammatory cytokine production through the accumulation of two viral proteins, ICP4 and ICP27, whose transcription is induced by tegument protein VP16. These viral proteins, through the decreasing of stabilizing the mRNAs of proinflammatory genes, delay cytokine production to an extent that allows the virus to replicate. Moreover, viral transactivating proteins, ICP-0 and VP-16 induce IL-10 expression. The conventional treatment of herpes labialis involves the topical and systemic use of antiviral drugs but it is necessary to find new therapies that can act in a selective and non-cytotoxic manner in viral infection. Laser diode therapy has been considered as a non-invasive alternative treatment to the conventional treatment of herpes labialis in pain therapy, in modulation of inflammation and in wound healing. This study aims to report a possible mechanism of action of laser diode irradiation in prevention and reduction of severity of labial manifestations of herpes labialis virus. We investigated, in an in vitro model of epithelial cells HaCat, the laser-effect on HSV-1 replication and we evaluated the modulation of expression of certain proinflammatory cytokines (TNF-α, IL-1β and IL-6), antimicrobial peptide HBD2, chemokine IL-8 and the immunosuppressive cytokine, IL-10. Our results lead us to hypothesize that LD-irradiation acts in the final stage of HSV-1 replication by limiting viral spread from cell to cell and that laser therapy acts also on the host immune response unblocking the suppression of proinflammatory mediators induced by accumulation of progeny virus in infected epithelial cells.

LinkOut - more resources

Full Text Sources

Atypon

Other Literature Sources

The Lens

Miscellaneous

NCI CPTC Antibody Characterization Program