J Photochem Photobiol B. 2009 Mar 3;94(3):164-70. doi: 10.1016/j.jphotobiol.2008.11.012. Epub 2008 Dec 7.

Effects of linear polarized infrared light irradiation on the transcriptional regulation of IL-8 expression in IL-1beta-stimulated human rheumatoid synoviocytes involves phosphorylation of the NF-kappaB RelA subunit.

Shibata Y[1], Araki H, Oshitani T, Imaoka A, Matsui M, Miyazawa K, Abiko Y.

Author information

Abstract

Although recent clinical studies have shown that laser therapy acts as an anti-inflammatory effector in the treatment of some diseases, little is known about the mechanism by which it acts in rheumatoid arthritis (RA) patients. The purpose of our work was to examine how irradiation with linear polarized infrared light (LPIL) suppresses inflammatory responses in the MH7A rheumatoid fibroblast-like synoviocyte cell line. We initially confirmed the effects of two disease-modifying anti-rheumatic treatments, LPIL irradiation and dexamethasone (Dex) administration, under experimental inflammatory conditions using gene chip technology. We found that LPIL exerted a smaller effect on gene transcription than Dex; however, IL-1beta-inducible target genes such as the CXCL type chemokines IL-8, IL-1beta and IL-6 were all clearly suppressed by LPIL to the same degree as by Dex. We also found that IL-1beta-induced release of IL-8 from MH7A cells was completely blocked by pretreatment with the (IL-8) inhibitor Bay11-7085, indicating that activation of NF-kappaB signaling plays an important role in the secretion of IL-8. Although the levels of NFKB1 and RELA transcription were unaffected by IL-1beta stimulation, phosphorylation of RelA S276 was suppressed by both LPIL and Dex. Thus LPIL likely exerts its anti-inflammatory effects by inhibiting the release of the inflammatory chemokine IL-8. A fuller understanding of the anti-inflammatory mechanism of LPIL in rheumatoid synoviocytes could serve as the basis for improved treatment of RA patients in the future.

PMID: 19135383 DOI: 10.1016/j.jphotobiol.2008.11.012

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