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*Lasers Surg Med.* 2007 Aug;39(7):614-21.

## The anti-inflammatory mechanism of 635 nm light-emitting-diode irradiation compared with existing COX inhibitors.

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### Abstract

**BACKGROUND AND OBJECTIVES:** Inhibition of cyclooxygenase (COX) and prostaglandin E(2) (PGE(2)) protects cells against cell injury in specific pathophysiological situations: inflammation and oxidative stress. Although the anti-inflammatory effects have been reported in clinical fields for specific wavelength irradiation during wound healing, the physiological mechanism has not been clarified yet. The aim of the present study is to investigate the anti-inflammatory mechanism of 635 nm light-emitting-diode (LED) irradiation compared with existing COX inhibitors.

**STUDY DESIGN/MATERIALS AND METHODS:** The present study investigated anti-inflammatory effects of 635 nm irradiation on PGE(2) release, COX and phospholipase A(2) (PLA(2)) expression, and reactive oxygen species (ROS) dissociation in arachidonic acid (AA)-treated human gingival fibroblast (hGF). These results were compared with their existing COX inhibitors: indomethacin and ibuprofen. The PGE(2) release was measured by enzyme immunoassay, the COX expression was measured by western blot and reverse transcriptase polymerase chain reaction (RT-PCR), and ROS level was measured by flow cytometry, laser scanning confocal microscope and RT-PCR.

**RESULTS:** Results showed that 635 nm irradiation and existing COX inhibitors inhibit expression of COX and PGE(2) release. Unlike indomethacin and ibuprofen, 635 nm irradiation leads to a decrease of ROS levels and mRNA expression of cytosolic phospholipase A(2) (cPLA(2)) and secretory phospholipase A(2) (sPLA(2)).

**CONCLUSION:** Taken together, 635 nm irradiation, unlike indomethacin and ibuprofen, can directly dissociate the ROS. This inhibits cPLA(2), sPLA(2), and COX expression, and results in the inhibition of PGE(2) release. Thus, we suggest that 635 nm irradiation inhibits PGE(2) synthesis like COX inhibitor and appears to be useful as an anti-inflammatory tool.

PMID: 17868110 [PubMed - indexed for MEDLINE]

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