# Low-level laser therapy induces dose-dependent reduction of TNFalpha levels in acute inflammation.

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**Abstract**

**OBJECTIVE:**

The aim of this study was to investigate if low-level laser therapy (LLLT) can modulate acute inflammation and tumor necrosis factor (TNFalpha) levels.

**BACKGROUND DATA:**

Drug therapy with TNFalpha-inhibitors has become standard treatment for rheumatoid arthritis, but it is unknown if LLLT can reduce or modulate TNFalpha levels in inflammatory disorders.

**METHODS:**

Two controlled animal studies were undertaken, with 35 male Wistar rats randomly divided into five groups each. Rabbit antiserum to ovalbumin was instilled intrabronchially in one of the lobes, followed by the intravenous injection of 10 mg of ovalbumin in 0.5 mL to induce acute lung injury. The first study served to define the time profile of TNFalpha activity for the first 4 h, while the second study compared three different LLLT doses to a control group and a chlorpromazine group at a timepoint where TNFalpha activity was increased. The rats in LLLT groups were irradiated within 5 min at the site of injury by a 650-nm Ga-Al-As laser.

**RESULTS:**

There was a time-lag before TNFalpha activity increased after BSA injection. TNFalpha levels increased from < or =6.9 (95% confidence interval [CI], 5.6-8.2) units/mL in the first 3 h to 62.1 (95% CI, 60.8-63.4) units/mL ($p < 0.001$) at 4 h. An LLLT dose of 0.11 Joules administered with a power density of 31.3 mW/cm(2) in 42 sec significantly reduced TNFalpha level to 50.2 (95% CI, 49.4-51.0), $p < 0.01$ units/mL versus control. Chlorpromazine reduced TNFalpha level to 45.3 (95% CI, 44.0-46.6) units/mL, $p < 0.001$ versus control.

**CONCLUSION:**

LLLT can reduce TNFalpha expression after acute immunocomplex lung injury in rats, but LLLT dose appears to be critical for reducing TNFalpha release.