Low-level **laser** therapy inhibits bronchoconstriction, Th2 inflammation and airway remodeling in allergic asthma.

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Abstract
Low-level **laser** therapy (LLLT) controls bronchial hyperresponsiveness (BHR) associated with increased RhoA expression as well as pro-inflammatory mediators associated with NF-kB in acute lung inflammation. Herein, we explore if LLLT can reduce both BHR and Th2 cytokines in allergic asthma. Mice were studied for bronchial reactivity and lung inflammation after antigen challenge. BHR was measured through dose-response curves to acetylcholine. Some animals were pretreated with a RhoA inhibitor before the antigen. LLLT (660 nm, 30 mW and 5.4 J) was applied on the skin over the right upper bronchus and two irradiation protocols were used. Reduction of BHR post LLLT coincided with lower RhoA expression in bronchial muscle as well as reduction in eosinophils and eotaxin. LLLT also diminished ICAM expression and Th2 cytokines as well as signal transducer and activator of transduction 6 (STAT6) levels in lungs from challenged mice. Our results demonstrated that LLLT reduced BHR via RhoA and lessened allergic lung inflammation via STAT6.