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Title: Low-level laser therapy restores the oxidative stress balance in acute lung injury induced by gut ischemia and reperfusion

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Body: It is unknown if the oxidative stress can be regulated by low- level laser therapy (LLLT) in acute lung inflammation induced by intestinal ischemia and reperfusion (i-I/R). A study was developed in which rats were irradiated (660 nm, 30 mW, 5.4 J) on the skin over the bronchus 1 h post- mesenteric artery occlusion and euthanized 2 h later. Lung edema and BALF neutrophils was measured by the Evans blue extravasation and myeloperoxidase (MPO) activity, respectively. Lung histology was used for analyzing the injury score. Reactive oxygen species (ROS) was measured by fluorescence. Both expression intercellular adhesion molecule-1 (ICAM-1) and peroxisome-proliferator-activated receptor-γ (PPARγ) were measured by Real Time-PCR. The lung immunohistochemical localization of ICAM-1 was visualized as a brown stain. Both lung HSP 70 and glutathione (GSH) protein were evaluated by ELISA. LLLT reduced neatly the edema, neutrophils influx, MPO activity and ICAM-1 mRNA expression. LLLT also reduced the ROS formation and oppositely increased GSH concentration in lung from i-I/R groups. Both HSP 70 and PPARγ expression also were elevated after laser. The results indicate that laser effect in attenuating the acute lung inflammation is driven to restore the balance between the pro- and anti-oxidants mediators rising of PPARγ expression and consequently the HSP 70 production.