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Title: Inflammasome pathway activation in patients with non-small cell lung cancer (NSCLC): A bronchoalveolar lavage fluid study

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Body: Introduction: Inflammasome activation is mediated by NLR proteins. Among NLRs, NLRP3-inflammasome is a multiprotein molecular platform activated by infection or host-derived danger signals that trigger an innate immune response via maturation of pro-inflammatory cytokines such as interleukin-1 β (IL-1 β), in a caspase-1-dependent way. Aim of the study: Our aim was to investigate the NLRP3 pathway activation in human BALF and peripheral blood samples from NSCLC patients and healthy controls. Methods: We prospectively studied BALF and peripheral blood leukocyte samples from 19 NSCLC and 12 healthy controls. All samples were treated with LPS (250 pg/ml, 2hrs) to induce TLR4 stimulation, followed by NLRP3-inflammasome activation with pulse ATP (5mM, 20min). Secreted TNFa, IL-1β and IL-6 were measured using commercial ELISA kits. Results: The main result is that LPS treatment resulted in increased levels of IL-1b production in NSCLC patients. Moreover, LPS treatment, pulse ATP and inhibition of the NLRP3-inflammasome activation using caspase-1 inhibitor resulted in increased IL-6 levels in NSCLC in comparison with controls. On the contrary, the same treatments resulted in a significant decrease in TNF-a secretion in NSCLC in comparison with controls. Conclusion: NLRP3-inflammasome is activated in NSCLC patients in the presence of infectious stimuli thus exhibiting a possible role as a proinflammatory "danger" receptor.