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Title: The effect of smoking on the activity of inflammatory markers in patients with COPD

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Body: At the heart of the onset and progression of COPD is a chronic non-allergic airway inflammation. The most important factor in the development of COPD is expected cytokine involved in inflammation, particularly interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF-α). Study of the effect of smoking on the humoral mechanisms of inflammation is important for understanding the pathogenesis of COPD. Objectives: To investigate the activity of the inflammatory process in dependence on smoking. 112 patients, 96 smokers with the experience of smoking 25,9 ± 1,8 a pack of / years. Markers of systemic inflammation are elevated in smokers. Fibrinogen was significantly higher in smokers than in nonsmokers 4,91 ± 0,89 and 3,25 ± 0,56 g / l ($p < 0,005$). CRP (11,1 ± 1,65 mg/l) in smokers was 3 times higher than in non-smokers(3, 45 ± 0,98 mg / l) $p < 0,005$. Opposite results were obtained for IL-10. Activity of IL-10 in serum is higher in non-smokers (64,75 ± 6,50 pg / ml) than smokers (4,35 ± 0,13 pg / ml) $p < 0,005$. TNF-α levels in the sputum of smokers (12, 9 ± 1,32 pg / ml) was significantly higher than non-smokers(4,35 ± 0,13 pg / ml) $p < 0,005$. Conclusion: Smoking affects the cytokine imbalance in COPD increases the activity of local and systemic inflammation, inhibits the synthesis of anti-inflammatory cytokine IL-10.