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Title: Chronic obstructive pulmonary disease is characterized with suppressed lipoxin A4 and increased lipoxin receptor expression in lungs

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Body: Persistent inflammation of COPD could be influenced by disorders of arachidonic acid metabolism when synthesis of leukotrienes does not switch to lipoxin generation, and thereby promotes further inflammation process. The aim: to estimate concentration of lipoxin A4 (LXA4), leukotriene B4 (LtB4), as well as expression of LXA4 receptor (FPRL-1) in induced sputum (IS) of COPD patients and healthy controls. Materials and methods: 17 COPD patients and 7 healthy controls. LXA4 and LtB4 concentration in IS was assessed with ELISA. FPRL-1 expression was detected immunocytochemically. Results: Concentration of LXA4 in COPD patient's IS was decreased compared to healthy controls (0,909+/-0,43 ng/ml vs 2,198+/-1,189 ng/ml; p=0,009). Ratio LtB4/LXA4 in COPD patients was three times greater compared to healthy persons (8,884+/-2,789 vs 3,328+/-2,94; p=0,0071). In COPD patient's IS FPRL-1 positive polinuclear cells (PPC) were in greater amount compared to healthy controls (8,802+/-5,758 cells/mm2 vs 2,123+/-2,232 cells/mm2; p=0,0109). Also, in COPD patients a count of FPRL-1 positive mononuclear cells (PMC) in IS was increased compared to healthy controls (2,563+/-1,711 cells/mm2 vs 0,655+/-0,522 cells/mm2; p=0,0311). Correlation between FPRL-1 PPC and LtB4 concentration in IS (r=0,628; p=0,0013). Conclusions: Increased LtB4/LXA4 indicate a disbalance of inflammatory mediators in COPD patients that could be one of the causes of inflammation persistence. In turn, increased LXA4 receptor expression and its correlation with LtB4 concentration in COPD patients could denote mechanism of inflammation adaption that is initiated by LtB4.