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Title: IL32 expression in induced sputum of COPD subjects

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Body: IL32 is a cytokine produced by T lymphocytes, natural killers cells, lung epithelial cells, monocytes and macrophages, involved in some chronic inflammatory diseases characterized by an autoimmune pathogenesis. A recent study demonstrated increased expression of IL32 in peripheral lung of COPD subjects. The present research investigated whether induced sputum, being a non-invasive methods, could be useful to study IL32 expression levels in COPD subjects. Sputum was induced in 20 COPD patients, 20 smokers without airflow limitation, 20 non-smokers with normal respiratory function and 20 systemic sclerosis (SS) subjects, as disease control. IL32 expression was evaluated by immunocytochemistry. Immunoreactivity was quantified in alveolar macrophages as a score obtained multiplying the % of positive cells for the staining intensity (0-3). The mRNA for IL32 isoform α, β and ε was evaluated by RT-PCR. Adequate number of macrophages for immunocytochemistry was obtained in 50% COPD subjects only. Macrophages % was decreased significantly in COPD subjects (median 19, [interquartile range: 9.4-29.2]) compared to smokers (52.2, [23.3-66.7], $p<0.01$), non-smokers (50.8, [44.5-63.2], $p<0.01$) and SS subjects (49.9, [32.9-69.4], $p<0.01$). IL32 score was not different among the four groups: COPD (150, [15-300]), SS subjects (80, [30-160]), smokers (160, [6-292]) and non-smokers (160, [95-235]). Unlike lung tissue samples, IL32 α isoform was never detected in COPD patients. While the β isoform was observed in all sputum samples, the ε isoform was detected in 54% of COPD subjects, 27% of smokers and 50% of non-smokers. In conclusion, IL32 expression in induced sputum samples do not reflect that observed in peripheral lung tissue.