Deficient pulmonary IFN-? expression in COPD patients

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COPD patients are prone to acute infectious exacerbations that impair their quality of life and hamper prognosis. The purpose of the present study was to investigate the in situ IFN-? response in the lungs of stable COPD and non-COPD patients. Lung samples from 70 subjects (9 control never smokers, 19 control smokers without COPD, 21 patients with moderate COPD and 21 patients with very severe COPD) were studied for the expression of IFN-?, its main transcription factor, IRF-7, and two products of its autocrine function, namely RIG-I and MDA-5, by immunohistochemical techniques and quantitative real-time PCR. IFN-?, IRF-7, RIG-I and MDA-5 were widely detected in most lung cell types. In epithelial tissues and alveolar macrophages, IFN-? and IRF-7 labeling scores were decreased up to 65% and 74%, respectively, for COPD patients, paralleling an analogous reduction (43% and 65%, respectively) in the amount of their lung mRNA. Moreover, this decreased production of IFN-? in COPD patients correlated with a similar decrease in the expression of RIG-I and MDA-5, two essential members of the innate immune system. Our study indicates that most lung cells from stable COPD patients show a constitutive decreased expression of IFN-?, IRF-7, RIG-I and MDA-5, suggesting that this deficiency is the main cause of their acute viral exacerbations.