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Phospho-p38 MAPK expression in COPD bronchi and in oxidative and inflammatory challenged bronchial epithelium

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The role of MAPK kinases in inducing the inflammatory response in the airways of chronic obstructive pulmonary diseases (COPD) patients is incompletely studied. Objectives: To investigate the expression of activated MAPK kinases in bronchial biopsies of COPD patients and the MAPK kinase bronchial epithelial response to oxidative and inflammatory stimuli related to COPD. Expression of phospho(p)-p38, p-JNK1 and p-ERK1/2 was measured in the bronchial mucosa using immunohistochemistry in patients with mild/moderate (n=17), severe/very severe (n=16) stable COPD, control smokers (n=16), control non smokers (n=9) and in a group with mild asthma (n=9). 16HBE cells, challenged with oxidative and inflammatory stimuli, were also studied for IL-8 and MAPK kinases mRNA production. P-p38 was the most expressed MAPK kinase in the bronchial mucosa of all subjects. No significant differences were observed for immune-expression of p-p38, p-JNK and p-ERK1/2 between COPD and control subjects. 16HBE cells treated with H_2O_2, cytomix (TNFα+IL-1β+IFNγ) and Lipopolysaccharide (LPS) up-regulated IL-8 mRNA production at 1h or 2h after treatments. P38α mRNA was significantly increased after H_2O_2 and LPS. JNK1 and ERK1 mRNA were not significantly increased after H_2O_2, cytomix or LPS treatments. Blocking p38α activity IL-8 mRNA production was not changed at 1h, 2h and 4h after H_2O_2 or LPS challenge. P-p38 immune-positivity is prevalent in the bronchial mucosa of COPD and asthmatic patients and p38 mRNA is increased after bronchial epithelial challenges suggesting a relevant role for this MAPK kinase in the induction of bronchial inflammation in COPD and asthma.

Keywords

Bronchial mucosa, MAPK kinases, COPD, Phospho-p38, Cytomix, Lipopolysaccharide.