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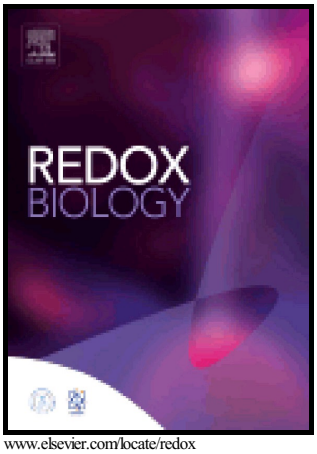

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) and cystic fibrosis (CF) are lethal pulmonary diseases. Cigarette consumption is the main cause for development of COPD, while CF is produced by mutations in the *CFTR* gene. Although these diseases have a different etiology, both share a CFTR activity impairment and proinflammatory state even under sterile conditions. The aim of this work was to study the extent of the protective effect of the antioxidant N-acetylcysteine (NAC) over the proinflammatory state (IL-6 and IL-8), oxidative stress (reactive oxygen species, ROS), and CFTR levels, caused by Cigarette Smoke Extract **(**CSE) in Calu-3 airway epithelial cells. CSE treatment (100 µg/ml during 24 h) decreased *CFTR* mRNA expression and activity, and increased the release of IL-6