NRF2 targeting: a promising therapeutic strategy in chronic obstructive pulmonary disease.

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Résumé en anglais	Several convergent destructive mechanisms such as oxidative stress, alveolar cell apoptosis, extracellular matrix proteolysis and chronic inflammation contribute to chronic obstructive pulmonary disease (COPD) development. Evidence suggests that oxidative stress contributes to the pathophysiology of COPD, particularly during exacerbations. Nuclear factor erythroid-2-related factor 2 (NRF2), a transcription factor expressed predominantly in epithelium and alveolar macrophages, has an essential protective role in the lungs through the activation of antioxidant response element-regulated antioxidant and cytoprotective genes. Animal models and human studies have identified NRF2 and several NRF2 target genes as a protective system against inflammation and oxidative stress from cigarette smoke, a major causative factor in COPD development. Hence, NRF2 targeting might provide clinical benefit by reducing both oxidative stress and inflammation in COPD.
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