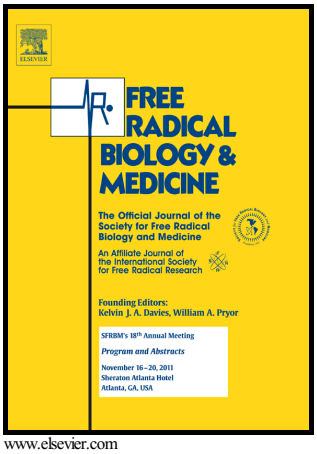

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RelB attenuates cigarette smoke extract-induced apoptosis in association with transcriptional regulation of the aryl hydrocarbon receptor

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ABSTRACT

Chronic obstructive pulmonary disease (COPD) is a chronic and prevalent respiratory disease caused primarily by long term inhalation of cigarette smoke. A major hallmark of COPD is elevated apoptosis of structural lung cells including fibroblasts. The NF-κB member RelB may suppress apoptosis in response to cigarette smoke, but its role in lung cell survival is not known. RelB may act as a pro-survival factor by controlling the expression of superoxide dismutase 2 (SOD2). SOD2 is also regulated by the aryl hydrocarbon receptor (AhR), a ligand-activated transcription factor that suppresses cigarette smoke-induced apoptosis. As the AhR is also a