Targeting accelerated pulmonary ageing to treat COPD induced neuropathological comorbidities.

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## Abstract

Chronic Obstructive Pulmonary Disease (COPD) is a major incurable health burden, ranking as the 3rd leading cause of death worldwide, mainly driven by cigarette smoking. COPD is characterised by persistent airway inflammation, lung function decline, and premature aging with the presence of pulmonary senescent cells. This review proposes that cellular senescence, a state of stable cell cycle arrest linked to ageing; induced by inflammation and oxidative stress in COPD, extends beyond the lungs and impacts the systemic circulation. This "spill over" of senescent cells contributes to brain inflammation and damage, increasing the risk of neurological comorbidities, such as stroke, cerebral small vessel disease, and Alzheimer's disease. The review explores the role of cellular senescence in COPD-associated brain conditions and investigates the relationship between cellular senescence and circadian rhythm in COPD. Additionally, it discusses potential therapies, including senomorphic and senolytic treatments, as novel strategies to halt or improve COPD progression.

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Figure 1

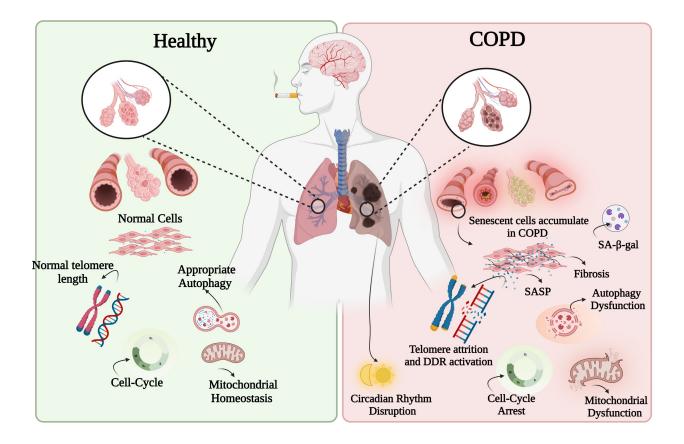


Figure 2

