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The role of accelerated ageing in aberrant lung tissue repair and remodelling in COPD

Woldhuis, Roy

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Propositions / Stellingen

Related to this thesis

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1. Ageing is the effect of normal biological processes that occur when getting older, but when these processes are accelerated or altered it leads to disease (this thesis)
2. Lung fibroblasts from COPD patients show characteristics of accelerated ageing and this is already apparent in fibroblasts from the relatively young severe, early-onset COPD patients (this thesis)
3. Accumulation of senescent lung fibroblasts in COPD can contribute to disease pathology as this leads to altered ECM regulation (this thesis)
4. Senescent fibroblasts in COPD secrete a panel of pro-inflammatory proteins that are involved in COPD pathogenesis (this thesis)
5. Higher level of cellular senescence in airway smooth muscle cells is not likely to play a role in COPD pathogenesis (this thesis)
6. E-cigarette vape induces cellular senescence and reduces the tissue repair function of primary lung fibroblasts and therefore E-cigarettes are not a safe alternative for tobacco smoking (this thesis)
7. Induction of cellular senescence by environmental risk factors for COPD in primary lung fibroblasts results in an impaired tissue repair capacity (this thesis)
8. Research into the role of ageing in COPD brings back the famous old question about the chicken and the egg
9. Everyone desires to live long, but no one wishes to be old (Jonathan Swift)
10. There are no secrets to success. It is the result of preparation, hard work, and learning from failure (Colin Powell)

Roy R. Woldhuis