

Elimination of senescent cells improves lung function in mice

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Most cells can divide only a limited number of times and eventually undergo permanent cell cycle arrest, a state known as cellular senescence. Cellular senescence is mediated by activation of specific cellular signaling pathways involving the proteins p19^{ARF} and p16^{INK4A}.

Precise control of cell cycle arrest and senescence are important for a number of biological processes, including embryonic development, wound healing, and tissue regeneration. Accumulating evidence also indicates that cellular senescence contributes to tissue aging.

In this issue of *JCI Insight*, Masataka Sugimoto and colleagues at the Juntendo University School of Medicine in Tokyo examined the role of <u>cellular senescence</u> in aging lungs, as there is a well-documented decrease in lung function with age.

Using transgenic mice in which they could selectively eliminate cells that express p19^{ARF}, Sugimoto and colleagues demonstrate that the loss of <u>senescent cells</u> improved lung function in mice. Further studies will be required to determine exactly how senescent cells impair <u>lung function</u>.

More information: Michihiro Hashimoto et al, Elimination of p19ARF-expressing cells enhances pulmonary function in mice, *JCI Insight* (2016). DOI: 10.1172/jci.insight.87732 Michihiro Hashimoto et al. Elimination of p19ARF-expressing cells enhances pulmonary function in mice, *JCI Insight* (2016). DOI: 10.1172/jci.insight.87732

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