

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 [cellular senescence](#) 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 [senescent cells](#) improved lung function in mice. Further studies will be required to determine exactly how senescent cells impair [lung function](#).

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](https://doi.org/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](https://doi.org/10.1172/jci.insight.87732)

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