SHAREABLE PDF

Cellular senescence: friend or foe to respiratory viral infections?

William J. Kelley[1,2,3], Rachel L. Zemans[1,2,4] and Daniel R. Goldstein[1,2,3]

Affiliations: [1]Dept of Internal Medicine, University of Michigan, Ann Arbor, MI, USA. [2]Program in Immunology, University of Michigan, Ann Arbor, MI, USA. [3]Dept of Microbiology and Immunology, University of Michigan, Ann Arbor, MI USA. [4] Program in Cellular and Molecular Biology, University of Michigan, Ann Arbor, MI, USA.

Correspondence: NCRC B020-209W, 2800 Plymouth Road, Ann Arbor, MI 48104, USA. E-mail: drgoldst@umich.edu

@ERSpublications
Senescence associates with fibrotic lung diseases. Emerging therapies to reduce senescence may treat chronic lung diseases, but the impact of senescence during acute respiratory viral infections is unclear and requires future investigation. https://bit.ly/2SAPpx6

Cite this article as: Kelley WJ, Zemans RL, Goldstein DR. Cellular senescence: friend or foe to respiratory viral infections? *Eur Respir J* 2020; 56: 2002708 [https://doi.org/10.1183/13993003.02708-2020].

ABSTRACT Cellular senescence permanently arrests the replication of various cell types and contributes to age-associated diseases. In particular, cellular senescence may enhance chronic lung diseases including COPD and idiopathic pulmonary fibrosis. However, the role cellular senescence plays in the pathophysiology of acute inflammatory diseases, especially viral infections, is less well understood. There is evidence that cellular senescence prevents viral replication by increasing antiviral cytokines, but other evidence shows that senescence may enhance viral replication by downregulating antiviral signalling. Furthermore, cellular senescence leads to the secretion of inflammatory mediators, which may either promote host defence or exacerbate immune pathology during viral infections. In this Perspective article, we summarise how senescence contributes to physiology and disease, the role of senescence in chronic lung diseases, and how senescence impacts acute respiratory viral infections. Finally, we develop a potential framework for how senescence may contribute, both positively and negatively, to the pathophysiology of viral respiratory infections, including severe acute respiratory syndrome due to the coronavirus SARS-CoV-2.