MAV(S)erick mitochondria: an unconventional role for mitochondrial antiviral signalling protein in pulmonary fibrosis

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Mitochondrial antiviral signalling protein (MAVS) mediates pulmonary fibrosis by regulating cGAS-STING and senescence programming https://bit.ly/35Tijjj

Cite this article as: Naikawadi RP, Wolters PJ. MAV(S)erick mitochondria: an unconventional role for mitochondrial antiviral signalling protein in pulmonary fibrosis. *Eur Respir J* 2021; 57: 2004500 [https://doi.org/10.1183/13993003.04500-2020].

Idiopathic pulmonary fibrosis (IPF) is a chronic, progressive, age-related interstitial lung disease. Incidence of IPF in the USA ranges between 14 and 63 per 100 000 people [1]. The average survival rate from the time of diagnosis is 3–5 years. IPF is associated with several genetic and environmental factors. Genes associated with a risk for developing IPF generally fall into two categories: epithelial cell genes (*e.g. MUC5B*, *DSP*, *SFTPA2*, *ABCA3*) or genes involved in telomere maintenance (*e.g. TERT*, *TERC*, *RTEL1*) [2, 3]. Environmental factors such as smoking, air pollution [4, 5] or accumulation of chitin polysaccharides [6] may also promote lung fibrosis. Consistent with the association of genes of the epithelium and telomere maintenance to IPF, short telomeres are a common finding in epithelial cells of IPF patients. An animal model of telomere dysfunction mediated by genetic deletion of TRF1 caused fibrosis that was progressive and chronic [7] indicating that telomere dysfunction is a molecular driver of fibrosis.