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Mitochondrial dysfunction in macrophages: a key to defective bacterial phagocytosis in COPD

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Pulmonary macrophages are key to elimination of respiratory pathogens. Oxidative stress and mitochondrial dysfunction are associated with defective macrophage phagocytosis in COPD, with implications for aberrant cellular metabolism and early cell death. http://bit.ly/2m2uA0z

Cite this article as: Eapen MS, Sharma P, Sohal SS. Mitochondrial dysfunction in macrophages: a key to defective bacterial phagocytosis in COPD. *Eur Respir J* 2019; 54: 1901641 [https://doi.org/10.1183/13993003.01641-2019].

COPD is now well recognised as a complex, devastating disease which affects the lungs of tobacco smokers in developed nations, though the contribution to the disease by biomass and fossil fuel burning cannot be ruled out, especially in less advanced or third world nations [1, 2]. An estimated 250 million people are affected worldwide as per 2016 estimates, with 3.17 million deaths annually, which is 5% of total worldwide mortality [3]. COPD stands to be the third-largest cause of death worldwide, and is often associated with respiratory infections, comorbidities and lung cancer, which further increases the risk of mortality [4]. The disease is characterised as slowly progressive with partial reversibility at most, with some pathological changes evident even after smoking cessation [5]. COPD patients develop structural changes, which leads to small airway wall thickening, narrowing and, ultimately, obliteration [6, 7]. The large airway pathology includes squamous cell metaplasia, mucus hypersecretion, and smooth muscle hyperplasia accompanied by chronic bronchitis [8, 9].