





Blood mitochondrial DNA as a biomarker of clinical outcomes in idiopathic pulmonary fibrosis

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Plasma mtDNA level could serve as a potential marker for disease progression and acute exacerbation in idiopathic pulmonary fibrosis https://bit.ly/2XoXsAv

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To the Editor:

Idiopathic pulmonary fibrosis (IPF) is a chronic, progressive fibrosing interstitial pneumonia of unknown aetiology [1] with ageing being one of the major risk factors [2]. Several ageing-related changes, such as the overproduction of mitochondrial reactive oxygen species, low adenosine triphosphate production, reduced mitochondrial biogenesis and inadequate mitochondrial (mt)DNA repair [3] have also been reported in the IPF lungs [4, 5] and in a bleomycin-induced mouse model [6]. Plasma cell-free DNA, including mtDNA, is released from dying cells into the circulatory system in response to injury [7]. Since circulating mtDNA contains CpG-rich sequences similar to bacterial DNA, disengaged mtDNA can activate the innate immune system by functioning as a damage-associated molecular pattern [8] and contribute to the stimulation of the profibrotic pathway [9].

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