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Extended Abstract
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COPD 2016: Is COPD a disease of accelerated ageing?_William MacNee_University of Edinburgh, UK

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Aging is associated with a progressive degeneration of tissues, which has a negative impact on the structure and function of vital organs and is one of the most important known risk factors for most chronic diseases. It is increasingly evident that many chronic inflammatory diseases represent an acceleration of the aging process. Chronic lung disease is an important component of the multiple and increasingly common debilitating diseases that are a major cause of morbidity and mortality, particularly in the elderly. The lungs are aging and it has been suggested that chronic obstructive pulmonary disease (COPD) is a condition of accelerated pulmonary aging and that aging can provide a mechanical link between COPD and many of its extrapulmonary effects and comorbidities. In this presentation, the physiological changes and mechanisms of aging will be described with particular emphasis on the pulmonary effects of aging and how they can be relevant for the development of COPD and its main extrapulmonary manifestations.

With age, there is a progressive deterioration in lung function, leading to an increased risk of shortness of breath and an increased prevalence of chronic lung disease in the elderly. Aging is associated with a gradual reduction in FEV1 of approximately 20 ml / year, as well as a reduction in the FEV1 / FVC ratio and an increase in residual volume with total lung capacity preserved. These changes in lung function cause lower oxygen levels and a decreased ability to remove carbon dioxide due to decreased compliance of the chest wall, elastic recoil of the lungs, and muscle strength. respiratory. These changes in lung function with age are similar to those that develop in COPD. Changes in pulmonary physiology with age are associated with structural changes in the lung involving alveolar enlargement, resulting in a decrease in the area available for gas exchange. However, this alveolar enlargement is different from that which occurs in COPD, since there is no destruction of the alveolar walls in the aging lung, as occurs in emphysema in COPD. Since the classic epidemiological studies of Fletcher and Peto, it has been considered that, in smokers of sensitive cigarettes who develop COPD, there is an accelerated decrease in lung function with the age of 50 to 100 ml of FEV1 / year. However, it is clear from recent studies that there is marked individual variability in the decline in FEV1 in subjects with COPD and that the development of the persistent airflow limitation characteristic of COPD is not always the result. result of an accelerated decrease in FEV1 but may be due, for example, to suboptimal lung growth during childhood. Thus, accelerated pulmonary aging may not be a pathogenic mechanism in all people with COPD. The incidence of COPD increases dramatically with age. The rate of newly diagnosed COPD in patients has increased from approximately 200 cases per 10,000 patients under the age of 45 to 1,200 cases per 10,000 patients aged 45 years or older. The greatest increase occurs in patients aged 65 to 74 years.

Aging has been thought to result from the accumulation of genetic damage and DNA repair defects. Oxidative stress is recognized as a mechanism for DNA damage from premature aging in the free radical theory of aging. Increased oxidative stress is known to occur in the lungs of COPD patients, as shown by the increased presence of oxidative stress biomarkers. Smokers and COPD patients also have increased evidence of oxidative DNA damage in the lungs, as shown by an increase in the concentration of 8-hydroxy-2-deoxyguanosine in the peripheral lung and type II pneumocytes. In addition, there is an increase in the number of double stranded DNA breaks, as shown by an increase in foci of phosphorylated histone 2AX in pulmonary endothelial cells, which also express an increase in p16 (a marker of cellular senescence). There is also evidence of a failed repair of DNA strand rupture in COPD. This imbalance between oxygen-induced DNA damage and COPD repair can lead to increased cellular senescence, although the pathogenic link to DNA damage in COPD may be more linked to the increased risk of lung cancer.

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