Editorial
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Tobacco Use Accelerates Biological and Functional Lung Aging

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Editorial

The lung has two main functions. The first is respiratory function dependent on ventilatory mechanics and gas exchange, which is well correlated with the possibilities of using oxygen and producing carbon dioxide (the respiratory quotient) [1]. The second is non-respiratory functions represented by metabolic, immunological, and endocrine ones. Pulmonary aging will result in reduced efficiency of all these functions [1,2].

The pulmonary aging can be of intrinsic origin by touching cell division and causing chromosomal deletions [3,4] or of extrinsic origin (occupational exposure, atmospheric pollution, smoking...) [5,6]. L´opez-Ot´ın and colleagues [3] proposed nine hallmarks of aging: genomic instability, telomere attrition, cellular senescence, epigenetic alterations, loss of proteostasis, deregulated nutrient sensing, mitochondrial dysfunction, stem cell exhaustion, and altered cellular and intercellular communication [3].

There are several predictors of pulmonary function decline (ventilator flows and pulmonary volumes) in the elderly: male gender, ethnicity, some metabolic pathology, and smoking [7,8].

The analysis of 73 gene aging signature demonstrated that smoking significantly dysregulates 18 aging-related genes in the small airway epithelium. In an independent cohort of male subjects, smoking significantly reduced telomere length in the small airway epithelium of smokers by 14% compared to nonsmokers. These data provide biologic evidence that smoking accelerates aging of the small airway epithelium [9].

Lung function measurements can be divided into three categories: spirometry to assess the dynamic flow rates, the static lung volumes and the gas exchanges [10]. The fall in Forced Expiratory flow Volume at the first second (FEV1) as a function of age is variable from one subject to another. In normal subjects, FEV1 decreases from 8 to 20 ml/year in healthy and non-smoking subjects. FEV1 decreases from 30 to 60 ml/year in non-chronic obstructive pulmonary disease (COPD) smokers and from the moment when COPD is established, FEV1 decreases from 60 to 90 ml/year, and sometimes even more than 100 ml/year [6].

The losses in volumes and flow rates due to aging are in part due to changes in the elasticity of the thoraco-pulmonary tissues. As a result, an increase in the pulmonary relaxation volume (Functional Residual Capacity (FRC)) is notable with a decrease in the caliber of the small bronchi linked to an increase in the elastic forces of retraction of the pulmonary parenchyma [11]. The main effect of age in the lungs is an increase in alveolar size without any inflammation or alveolar wall destruction [1]. After the third decade of life, ageing leads to a slow but progressive degeneration of elastic fibers with an increase in their number and thickness [12]. Changes in muscle strength may also explain the drop in lung volumes and flows. The decrease in the strength of the respiratory muscles is related to changes in skeletal muscle structure including the diaphragm. Thus, maximal inspiratory and expiratory pressures decrease from the age of 50 years [11]. Indeed, with age, Chlif et al., [13] showed lower inspiratory muscle performance attested by a higher tension-time index (TT0.1) during exercise in the old endurance-trained athletes (OT) group than Young Athletes (YT) group. Note that this effect related to age appeared to be more marked in sedentary subjects and elderly smoking. Smoke can also induce skeletal muscle dysfunction. Indeed, muscles of non symptomatic smokers are

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weaker and less fatigue resistant than those of nonsmokers. Cigarette smoke constituents and systemic inflammatory mediators enhance proteolysis and inhibit protein synthesis, leading to loss of muscle mass. Reduced skeletal muscle contractile endurance in smokers may result from impaired oxygen delivery to the mitochondria and ability of the mitochondria to generate ATP due to interaction of carbon monoxide with hemoglobin, myoglobin, and components of the respiratory chain [14].

Besides stimulating protein degradation, smoke exposure may also inhibit anabolic pathways. Muscle protein synthesis is decreased in the quadriceps muscle of smokers, which was associated with an increased expression of myostatin. This molecule inhibits muscle growth by inactivation of protein kinase B (also known as Akt) and by hampering muscle cell renewal [14,15].

Gas exchange is most often dependent on an appropriate matching of ventilation with lung perfusion [16]. Using measures of ventilation and lung perfusion, Levin et al., work has demonstrated an age-related increase in ventilation-perfusion (VA/Q) inequality, characterized by heterogeneous distribution of lung units having high and low ratios [16]. The VA/Q inequality is associated with changes in the pulmonary circulation [17]. The age-related increase in VA/Q inequality may coexist with a decrease in the diffusion capacity of the lung for carbon monoxide (DLCO). The reduction in DLCO may be due to declines in the alveolar surface area and, possibly, in the density of lung capillaries [18,19]. Smoking accelerates the aging of the lung parenchyma. Indeed, smoking amount (pack-years) was negatively correlated with DLCO percent predicted. Therefore, Zhang et al., showed that non-smokers with COPD had less impairment in gas-exchange, and a lower prevalence of emphysema compared with their smoking counterparts [20].

The lungs perform several important non-respiratory functions that are vital for normal physiology: Vascular reservoir, filter for blood borne substances, defence against inhaled substances (Muco-ciliary escalator, Immune function), defence against inhaled chemicals, endocrine and metabolic functions (Isolated pulmonary neuroendocrine cells (PNECs) and innervated cell clusters). Nitric oxide (NO) is a metabolic mediator produced by pulmonary cells. As demonstrated by Rouatbi et al., the aging of the lung modified exhaled nitric oxide (FeNO) values [21].

Cigarette smoking decreased exhaled NO, suggesting that it may inhibit the enzyme NO synthase. Since endogenous NO is important in defending the respiratory tract against infection, in counteracting bronchoconstriction and vasoconstriction, and in inhibiting platelet aggregation, this effect may contribute to the increased risks of chronic respiratory and cardiovascular disease in cigarette smokers [22].

Lung aging can be explored by several methods: spirometry and measure of FEV1, Fletcher curve, and Lung age calculation. These tools are used in the smoking cessation counseling. Graphic (Fletcher Curve) and lung age value resonate more than FEV1 alone in smoking cessation results [23,24].

In conclusion, tobacco use accelerates lung aging and touch respiratory and non respiratory functions. Knowledge of these changes is very useful for better management of elderly tobacco addicts.

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