MOLECULAR MODIFICATIONS OCCURRING IN RAT LUNG DURING AGING

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Background: An adequate oxygen supply is essential to the life of all aerobic organism. In mammals, at tissue and cell level, oxygen transport is optimized, since it serves as the terminal electron acceptor in mitochondrial oxidative phosphorylation and several enzymatic processes require molecular oxygen as substrate. During development and aging, redundant cells and exhausted cells are eliminated, respectively, whereas others can adapt to the stressful environment and survive.

Objective: The aim of this study was to investigate the expression of p53, p66Shc, putative cysteine protease (CPP32), vascular endothelial growth factor (VEGF), kinaseB-alpha phosphorilation (pIkB?) and molecular mechanism activated during aging by the lung, which can be able to create a life-support system, adapting to stressful situations caused by reduced oxygen supply to the tissues.

Methods: Twelve animals from two groups, each consisting of 6 male Wistar rats, 3 and 24 months old, were kept under physiological conditions; 3 young and 3 old rats, were kept in room air (21% O2) and used as control. The rats were anaesthetized with Nembutal (40 mg/kg, ip), and lung, excised from each rat, was processed for TUNEL and Western Blotting analyses.

Results: The expression of p53, p66Shc and CPP32 is significantly increased in old rats, when compared to the young ones. In parallel, the increased expression of VEGF and pIkB? are increased in old rats rather than young.

Conclusions: Aging leads to an increased expression of p53, p66Shc and CPP32. This data suggess that apoptosis is in progress. In the same time, the lung defends own survival through the production of VEGF and pIkB? in order to counteract to the low oxygen availability at tissue level, and this suggests that the lung tries to counteract this physiological process, through the stimulation of the growth of new blood vessels, growth factor-mediated cell survival and inhibition of apoptosis respectively.