Cím: Wnt4 promotes tissue destruction during lung aging via inhibiting PPARγ expres

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The continuous increase of elderly population will put an enormous pressure on social and medical care in the near future. Therefore expanding the healthy lifespan or decreasing the occurrence of the age related diseases are the biggest challenges for developed countries.

While the aging of other organs are studied widely, the molecular background of lung senescence is hardly known. In the aging lung, the lung capacity decreasing and the formation of new alveoli slow down even in the absence of diseases.

For the lung regeneration ATII cells are one of the most important facultative progenitor cells. They are supported by the lipofibroblasts. The normal function is a PPARγ dependent mechanism. The loss of the number of lipofibroblast will cause destruction of ATII cell network, which will lead to different age related diseases such as COPD or IPF.

Recent studies suggest the role of Wnt molecules in aging. It was already proven that Wnt proteins have role in different fibrotic and inflammatory processes, like COPD and IPF. But we still don’t know, are there any connections between these two processes?

In our studies 1 month and 24 months Balb/C mice lungs were compared first with computed tomographic technique. On the recordings is clearly seen the enlargement of the alveoli, and it was also proven with microscopic sections with Hematoxylin-Eosin staining.

To investigate the molecular pattern of lung, first epithelial and non-epithelial cells were separated; by EpCAM1 positivity and gene expression analysis were performed. Because for the normal lung regeneration PPARγ and ADRP are essential, they expression were measured with quantitative real time PCR, beside Wnt molecules.

Our studies have shown that Wnt4 are increased in epithelial and non-epithelial cells, which based on literature, can decrease the PPARγ expression, which will lead to the loss of normal lung function and cause COPD in elderly.