Mitochondrial potassium channels protect senescent aortal smooth muscle cells from exceed ROS synthesis?

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Different factors may induce a stress response named cellular senescence. Senescent cells remain metabolically active and viable, however they do not proliferate. They accumulate during development of age-related diseases and in the tissue of aging organism. The presence of senescent vascular smooth muscle cells and endothelial cells was recognized in atherosclerotic plaque. Importantly, elimination of senescent cells significantly attenuates development of atherosclerosis progression. Mitochondria were shown to modulate proinflammatory senescence associated secretory phenotype, which support development of age-related diseases. Changes in mitochondrial functioning are important features of senescent cells.

Our project is focused on the role of mitochondrial potassium channels in smooth muscle cells senescence. Cellular stress can result in increased reactive oxygen species (ROS) synthesis. Among few pathways responsible for ROS synthesis mitochondria produced ROS (mROS) seems to have important role during cell functioning. Activation of mitochondrial potassium channels can inhibit mROS synthesis what lead to cytoprotection. Herein, we try to answer the question if those channels are present and active in human senescent aortal smooth muscle cells (SMC). Moreover, we describe differences in expression level of 84 genes encoding proteins involved in the biogenesis and functioning of the mitochondria. Overexpression of SOD2 gene and protein was also measured and distinguished from circSOD2 expression. Our studies suggest that senescence of human SMC cells is associated with significant changes in abundance and activity of mitochondrial large conductance calcium-activated channels what may preclude its influence on mROS synthesis and cytoprotective action.

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