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Endothelial cell senescence with aging in healthy humans: Prevention by habitual exercise and relation to vascular endothelial function

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Cellular senescence is emerging as a key mechanism of age-related vascular endothelial dysfunction, but evidence in healthy humans is lacking. Moreover, the influence of lifestyle factors such as habitual exercise on endothelial cell (EC) senescence is unknown. We tested the hypothesis that EC senescence increases with sedentary, but not physically active, aging and is associated with vascular endothelial dysfunction. Protein expression (quantitative immunofluorescence) of p53, a transcription factor related to increased cellular senescence, and the cyclin dependent kinase inhibitors p21 and p16 were 116%, 119%, and 128% greater (all $P<0.05$), respectively, in ECs obtained from antecubital veins of older sedentary (n=12, 60±1 yrs) vs. young sedentary (n=9, 22±1 yrs) adults. These age-related differences were not present (all $P>0.05$) in venous ECs from older exercising adults (n=13, 57±1 yrs). Furthermore, venous EC protein levels of p53 (r=-0.49, P=0.003), p21 (r=-0.38, P=0.03), and p16 (r=-0.58, P=0.002) were inversely associated with vascular endothelial function (brachial artery flow-mediated dilation). Similarly, p53 and p21 protein expression were 26% and 23% higher (both $P<0.05$), respectively, in ECs sampled from brachial arteries of healthy older sedentary (n=18, 63±1 yrs) vs. young sedentary (n=9, 25±1 yrs) adults; age-related changes in arterial EC p53 and p21 expression were not observed ($P>0.05$) in older habitually exercising adults (n=14, 59±1 yrs). These data indicate that EC senescence is associated with sedentary aging and is linked to endothelial dysfunction. Moreover, these data suggest that prevention of EC senescence may be one mechanism by which aerobic exercise protects against endothelial dysfunction with age.

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