**EFFECT OF ENDOTHELIAL CELL SENESCENCE ON THE HEAT SHOCK RESPONSE AND CELLULAR FUNCTION**

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Increasing evidence supports that replicative senescence, cessation of cell division, plays a role in the progression of the aging phenotype. With cellular senescence, cells undergo morphologic, physiologic and functional changes. We hypothesized that adult human coronary artery endothelial cells (HCAEC) would have detrimental changes with the onset of senescence including impairment of the protective heat shock response, which is critical for maintaining many cellular functions, and organelle dysfunction. Early passage (EP) and senescent (SEN) cells from the same donors were heat-shocked at 42 C in 5% CO2 for 1 hour, and then allowed to recover for 2 hours at 37 C. In EP HCAEC HSP90 and HSP60 levels did not change with heat shock, but the HSP72 protein was significantly elevated. This response was blunted in Sen EC. Furthermore, studies of overall cellular functions demonstrated impairment of key organelles, including the mitochondria. Aging associated cell senescence impairs endothelial cell function, contributing to vascular inflammation and dysfunction.