**THE CENTRAL ROLE OF SMOOTH MUSCLE CELL MITOCHONDRIA IN THE PREVENTION OF ABDOMINAL AORTIC ANEURYSM BY LOW LEVEL LASER THERAPY**

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We showed, by high frequency ultrasound, that phototherapy with low-level laser (LLL), a non-thermal, near-infrared radiation, used widely for reduction of pain and acceleration of wound healing, prevents *de novo* formation and progression of pre-existing abdominal aortic aneurysm (AAA) in the angiotensin-II-(Ang-II)-infused apolipoprotein e-deficient (Apo-E-/-)mouse model. Correlative histomorphometric and immunohistochemical studies have shown that the protective effect of LLL in this mouse model is associated with modification of the inflammatory response and enhancement of smooth muscle cell (SMC)-driven extracellular matrix reinforcement of transmedial defects in the aortic wall, that occur near side branches, and that subtend the aneurysmal expansion. In addition, LLL was found to stimulate mitochondrial membrane potential (mitMP), suppress the expression of the pro-inflammatory cytokine, IL-1-beta, and disperse subnuclear promyelocytic leukemia protein, a cell-cycle checkpoint protein, in HaCaT human keratinocytes. In recent studies by others, Ang-II was shown to cause decay in SMC mitMP followed by reduction in generation of ATP that is of critical importance for matrix synthesis. In this presentation we will review the nature and circumstances contributory to vulnerability of SMC mitochondria and the impact of such alterations on vascular wall integrity and function. We also will present the results of our studies designed to determine whether LLL prevents Ang-II-induced suppression of SMC mitMP, whether this mechanism underlies the inhibitory effect of LLL on progression of AAA in the Apo-E-/- mouse model, and the potential relevance of these findings for targeting mechanisms of aneurysm progression in the human interventional setting.