Atherosclerosis by Targeting the Mitochondria-Inflammation Circle

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The mitochondrial redox equilibrium of endothelial cells (ECs) may become disturbed, which may result in persistent inflammation and atherosclerosis. Oxidative damage can cause endothelial dysfunction, and chronic sympathetic hyperactivity can make it worse. By reducing mitochondrial reactive oxygen species (ROS)-induced inflammation, we investigated whether renal denervation (RDN), a method for lowering sympathetic tone, could protect ECs from atherosclerosis.

ApoE-deficient (ApoE/-) mice underwent RDN or a sham procedure prior to consuming a high-fat diet for 20 weeks. The mitochondrial morphology, atherosclerosis, and EC phenotype were all found. Norepinephrine treatment of human artery ECs was used in vitro to investigate the underlying mechanisms of RDN-repressed endothelial inflammation. In EC mitochondria, RDN reduced oxidative stress, inflammation, and atherosclerosis. The amount of norepinephrine in the blood and the activity of the enzyme monoamine oxidase A (MAO-A) were both increased as a result of the persistent sympathetic hyperactivity impeded MAO-The development of atherogenic and proinflammatory particles was expanded in ECs because of ROS development and NF-B actuation brought about by the enactment of mitochondrial homeostasis. With the aid of NF-B and oxidative stress, it also inhibited PGC-1, a regulator of mitochondrial function. By disrupting the positive feedback regulation between mitochondrial dysfunction and inflammation brought on by RDN's inactivation of MAO-A, EC atheroprone phenotypic changes and atherosclerosis were prevented.

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