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Commentary

Enhanced Eploration of Mitochondria in Cardiovascular Health

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DESCRIPTION

Mitochondria are essential life and death regulators. Not unexpectedly, their position in the cardiovascular system is a difficult part of research, with numerous key breakthroughs lately published. Such articles have contributed to the assumption that mitochondrial dysfunction is a sine qua non of cardiovascular illness by increasing our understanding of the relevance of mitochondria in Cardiovascular Health (CVD).

Mitochondrial dynamics is also emerging as a key regulator of vascular health. The closure of the ductus arteriosus, which is essential for the transition from the foetal to the neonatal pattern of circulation, has been demonstrated to be dependent on mitochondrial fission (through Drp1) in smooth muscle. As a result, the structure of our mitochondria determines the physiological changes in circulation that occur when we take our first breath! In smooth muscle, however, mitochondrial fission may be a double-edged sword. It induces a highly proliferative phenotype, which could be harmful in illnesses like pulmonary artery hypertension. Diabetes and hyperglycemia also cause mitochondrial fragmentation by increasing the production of Fission-1 Protein (Fis1) and Drp1. Endothelial cells with mitochondrial fragmentation have a hyper proliferative phenotype, which generates greater levels of ROS and is prone to senesce, similar to smooth muscle cells. Silencing pro-fission proteins restores mitochondrial networks and reduces Reactive Oxygen Species (ROS) production while raising the activation state of nitric oxide synthase, which may play a direct role in adaptive mitochondrial dynamics. Regulators such Uncoupling Protein 2 (UCP2) appear to be integrated with pro-fission responses in healthy endothelium to shield endothelial cells from the detrimental ROS and p53 activation associated with the fragmented mitochondrial state.

Mitophagy, which appears to be crucial for removing damaged mitochondria and preserving bioenergetics function, is known to be intricately tied to such alterations in mitochondrial structure. Cell death by necrosis or apoptosis is a possible outcome if damaged mitochondria are not eliminated. Recent research has offered further information on how mitochondria govern cell death and which specific pathways mitochondrial signalling activates. Loss of Myeloid Cell Leukemia-1 (MCL-1), an anti-apoptotic BCL-2 protein, was found to decrease mitochondrial respiration and lead to heart failure in two separate experiments. The roles of Ca^{2+}/Calmodulin-Dependent Protein Kinase Ii (CaMKII), G protein-coupled Receptor Kinase 2 (GRK2), and mitochondrial Signal Transducer And Activator Of Transcription 3 (Stat3) in myocardial cell death and mitochondrial stress in the heart have also been revealed by findings from the past two years. New findings were also reported regarding the well-studied protein targets p53 and Cyclophilin D (CypD): p53 was found to bind to CypD, resulting in mPTP-dependent necrosis. While CypD is well recognized as a mPTP regulator, studies have shown that it also regulates branched chain amino acid, pyruvate, and fatty acid metabolism, which could be linked to its putative involvement in controlling the mitochondrial acetylome. Mitophagy has been linked to mitochondrial protein acetylation, which has been believed to have a role in the initiation of mitophagy.

Recent research has also revealed new details about the mechanisms underlying the beneficial benefits of common treatments like calorie restriction and exercise. Caloric restriction is well-known for reducing cardiovascular ageing symptoms and increasing longevity, its ability to modulate not just Sirt deacetylase activity, but also mitophagy and PGC1-mediated activities. Deacetylation of particular electron transport chain subunits (induced by caloric restriction) has been found to protect against ischemia stress during caloric restriction. Exercise has been proven to reduce doxorubicin-induced heart injury, prevent mtDNA depletion and mutations, boost oxidative capacity, promote healthy ageing, and prevent mtDNA depletion and mutations. Over time, mitochondrial function and antioxidant capacity become mismatched, resulting in mitochondria-mediated oxidative stress, bioenergetics failure, and cell death. This is supported by the fact that deleting the regulator of mitochondrial biogenetic programming PGC1 can increase mitochondrial ROS production, leading to vascular dysfunction and inflammation, and that mice with lower levels of the mitochondrial form of superoxide dismutase have aortic Stiffening and Cardiac Deterioration (SOD2).

In conclusion, contemporary research has not only addressed critical, long-standing issues in mitochondrial research, but it has also resulted in remarkable findings and novel insights into how

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mitochondria affect human cardiovascular health. Our efforts to determine the composition of mitochondria and the mPTP, to comprehend how metabolic enzymes regulate cardiovascular remodeling and function, and to confirm the identity of the mitoKATP channel will likely be active and contentious areas of mitochondrial research as a result of these discoveries.

CONFLICT OF INTEREST

Author has declared that he has no conflict of interest.