Mitochondrial fission and autophagy in the normal and diseased heart

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Sustained hypertension promotes structural, functional and metabolic remodeling of cardiomyocyte mitochondria. As long-lived, postmitotic cells, cardiomyocytes turn over mitochondria continuously to compensate for changes in energy demands and to remove damaged organelles. This process involves fusion and fission of existing mitochondria to generate new organelles and separate old ones for degradation via autophagy. Autophagy is a lysosome-dependent proteolytic pathway capable of processing cellular components, including organelles and protein aggregates. Autophagy can be either nonselective or selective and contributes to remodeling of the myocardium under stress. Fission of mitochondria, loss of membrane potential, and ubiquitination are emerging as critical steps that direct selective autophagic degradation of mitochondria. This review discusses the molecular mechanisms controlling mitochondrial dynamics, including fission, fusion, transport, and degradation. Furthermore, it examine