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MicroRNAs control mitochondrial network in the heart

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Mitochondria supply energy for physiological function and they participate in the regulation of other cellular events including apoptosis, calcium homeostasis and production of reactive oxygen species. Thus, mitochondria play a critical role in the cells. However, dysfunction of mitochondria is related to a variety of pathological processes and diseases. MicroRNAs (miRNAs) are a class of small noncoding RNAs about 22 nucleotides long, and they can bind to the 3' un-translated region (3'UTR) of mRNAs, thereby inhibiting mRNA translation or promoting mRNA degradation. Our data show that modulations of miR-499 levels can influence apoptosis, myocardial infarction and cardiac remodeling. Both the alpha-and beta-isoforms of calcineurin catalytic subunit are the targets of miR-499. Calcineurin dephosphorylates dynamin-related protein-1 (Drp1) leading to its accumulation in mitochondria, the activation of the mitochondrial fission program and the consequent apoptosis. miR-499 can regulate Drp1 phosphorylation status and mitochondrial fission through targeting calcineurin. Finally, p53 is shown to transcriptionally downregulate miR-499 expression. Our data provide novel evidence suggesting that miR-499 is a regulator of mitochondrial fission machinery, and the therapeutic approaches for myocardial infarction can be developed by modulating miR-499.

Biography

Peifeng Li has completed his MD and Ph.D. at the age of 30 years from Chinese Academy of Medical Science and postdoctoral studies from Max-Delbruck Center for Molecular Medicine, Germany. He is a Research Assistant Professor at University of Illinois at Chicago. He has published more than 62 papers in reputed journals and has been serving as an Editorial Board Member of Dataset Papers in Biology.

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