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## **Mechanism of mitochondrial dysfunction in hypertrophic cardiomyocyte**

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The present study was undertaken to understand mitochondrial signaling mechanism in hypertrophic cardiomyocytes. Cardiomyocytes were treated with phenylephrine (PE, 100  $\mu$ M) for 24h to induce cardiac hypertrophy which was assessed by monitoring cell size and marker gene expression. Hypertrophy of cardiomyocytes and remodeling of the mitochondria were prevented by PPAR $\alpha$  agonist, fenofibrate. Fenofibrate also corrected deranged fatty acid oxidation genes in mitochondria in hypertrophic cardiomyocytes. Decrease in mitochondrial trans-membrane potential and dynamicity in PE-treated cardiomyocytes were also blocked by fenofibrate. Significant increase in reactive oxygen species (ROS) production and calcium level in cardiac hypertrophic condition was ameliorated by fenofibrate. Furthermore, PE induced impairment of mitochondrial activity and cellular ATP generation were partially checked when cells were co-treated with fenofibrate. Expression of some miRNAs which were found to be putative regulators of VDAC, were altered in hypertrophic cardiomyocytes which were restored when the cells were co-treated with PPAR $\alpha$  agonist, fenofibrate. Overall, the results demonstrate that PPAR $\alpha$  signaling is critically involved in mitochondrial dysfunction in hypertrophic cardiomyocytes in which miRNAs might play a significant role.

### **Biography**

Dipak Kar has completed his graduation & post graduation from Calcutta University, Kolkata, India. Currently he is doing PhD in Cardiovascular disease from CSIR-Indian Institute of Chemical Biology, Kolkata, India. His primary objective of research is to understand molecular mechanism of mitochondrial dysfunction in hypertrophic cardiomyocytes. Part of the work is already published in peer-reviewed journal and presented at various national and international meetings. He also have one patent in his name.

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