Upstream stimulatory factor 2 protects cardiomyocytes by regulating mitochondrial homeostasis

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Abstract

Myocardial ischemia and hypoxia are one of the main causes of heart failure, and cardiomyocyte apoptosis induced by mitochondrial injury is the basis of poor heart remodeling and heart failure. Upstream stimulatory factor 2 (USF2), a transcription factor involved in multiple cellular processes, has recently been identified as having an active role in mitochondrial function and energy homeostasis; however, the role of USF2 in cardiovascular disease has not been reported. In this study, we demonstrated that the expression of USF2 protein can be degraded by the ubiquitin-proteasome pathway when cardiomyocytes are hypoxic, and the loss of USF2 can lead to mitochondrial dysfunction in cardiomyocytes, aggravating mitochondrial damage and further promoting apoptosis.Mechanistically, we also demonstrate that USF2 deficiency induces mitochondrial autophagy by [regulating](javascript:;) the AMPK/mTOR signaling pathway.Altogether, this study provides new insights into the protective role of USF2 in hypoxic cardiomyocyte injury. USF2 may serve as a potential therapeutic target for myocardial hypoxia.

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