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miR-199a impairs autophagy and induces cardiac hypertrophy through mTOR activation

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Basal autophagy is tightly regulated by transcriptional and epigenetic factors to maintain cellular homeostasis. Dysregulation of cardiac autophagy is associated with heart diseases, including cardiac hypertrophy, but the mechanism governing cardiac autophagy is rarely identified. To analyze the *in vivo* function of miR-199a in cardiac autophagy and cardiac hypertrophy, we generated cardiac-specific miR-199a transgenic mice and showed that overexpression of miR-199a was sufficient to inhibit cardiomyocyte autophagy and induce cardiac hypertrophy in vivo. miR-199a impaired cardiomyocyte autophagy in a cellautonomous manner by targeting glycogen synthase kinase 3β (GSK3β)/mammalian target of rapamycin (mTOR) complex signaling. Overexpression of autophagy related gene 5 (Atg5) attenuated the hypertrophic effects of miR-199a overexpression on cardiomyocytes, and activation of autophagy using rapamycin was sufficient to restore cardiac autophagy and decrease cardiac hypertrophy in miR-199a transgenic mice. These results reveal a novel role of miR-199a as a key regulator of cardiac autophagy, suggesting that targeting miRNAs controlling autophagy as a potential therapeutic strategy for cardiac disease.

Biography

Zhenhua Li has completed his PhD from Beijing Institute of Biotechnology, China and is now the Research Assistant of Beijing Institute of Lifeomics. His work mainly focuses on exploring the regulatory mechanism of cardiac homeostasis maintenance and developing the potential therapeutic targets for cardiac diseases. He has published six papers in reputed journals with the total impact factor of 32.7.

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