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miR-199-sponge transgenic mice develop physiological cardiac hypertrophy

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Overexpression of either member of the miR-199 family, miR-199a-5p, or miR-199b-5p (hereinafter referred to as miR-199a or miR-199b) promotes pathological cardiac hypertrophy, but little is known about the role of endogenous miR-199 in cardiac development and disease. Our study aimed to determine the physiological function of the endogenous miR-199 family in cardiac homeostasis maintenance. We generated a sponge transgenic mouse model with a specific disruption of miR-199 in the heart. To our surprise, we found that knockdown of endogenous miR-199 caused physiological cardiac hypertrophy characterized by an increased heart weight and cardiomyocyte size, but with normal cardiac morphology and function. Furthermore, we also identified PGC1a as the target gene of the miR-199 family and PGC1a was also increased in sponge transgenic mice. Inhibition of endogenous miR-199 led to physiological cardiac hypertrophy probably due to the up-regulation of PGC1a, uncovering a surprising role for endogenous miR-199 in the maintenance of cardiac homeostasis.

Biography

Ning Hou is specialized in transgenic and gene knockout/knock-in technologies and In-charge of the establishment and maintenance of transgenic and gene knockout/knock-in platform of the laboratory. She has contributed to the generation of >50 transgenic mouse lines including 7 tissue-specific Cre lines that were primarily reported, as well as 30 murine models of human disease. She has published 43 papers in scientific journals, including Cell Stem Cell, Journal of Clinical Investigation and Cell Research with the total impact factor of 167.

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