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miR-96 inhibits cardiac hypertrophy by targeting growth factor receptor-bound 2

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ABSTRACT. Increasing evidence has indicated that microRNAs are involved in the pathogenesis of cardiac hypertrophy. However, whether miR-96 is involved in heart diseases, particularly cardiac hypertrophy, remains unclear. In this study, we found that miR-96 is a negative regulator of cardiac hypertrophy. In primary cardiomyocytes, overexpression of miR-96 inhibited phenylephrine-induced cardiomyocyte hypertrophy and decreased the mRNA expression of cardiac hypertrophy markers such as atrial natriuretic factor and β-myosin heavy chain. Interestingly, we found that growth factor receptor-bound 2 is a direct target of miR-96, which is a negative regulator of cardiac hypertrophy. Overexpression of miR-96 in cardiomyocytes led to reduced growth factor receptor-bound 2 expression. More importantly, miR-96 repressed the extracellular-regulated protein kinase signaling pathway by targeting growth factor receptor-bound 2 in cardiomyocytes. Our data demonstrate that miR-96 is a negative regulator of cardiac hypertrophy and extracellular-regulated protein kinase signaling, thus offering a new therapeutic strategy for cardiac hypertrophy.

Key words: Cardiac hypertrophy; Growth factor receptor-bound 2; miR-96

