Methods Quantitative real time-PCR data showed that the expression of miR-15a and miR-15b was upregulated in C57BL/6 mice heart subjected to I/R (n=7) while miR-16 without any change, especially miR-15b showed an increase in expression >3-fold. And the data was consistent with cardiomyocytes exposed to hypoxia/reoxygenation (H/R) (n=3). Recombinant adenoviral vectors were constructed to explore the functional role of miR-15b in cultured cardiomyocytes exposed to H/R. Overexpression of miR-15b enhanced cell apoptosis and the loss of mitochondrial membrane potential ($\Delta\psi_m$) determined by flow cytometry analysis. Conversely, downexpression was cytoprotective. Furthermore, the inhibition of miR-15b increased the expression of Bcl-2 protein, suppressed the release of mitochondrial cytochrome c (Cyt-c) to cytosol and decreased the activity of caspase-3 and caspase-9.

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Conclusions MiR-15b may be the upstream regulator of mitochondrial signalling pathway of I/R induced apoptosis by targeting Bcl-2.

MiRNAs expression was analysed in I/R and sham group (or H/R and control group) by using quantitative real-time PCR. Furthermore, gain of function and loss of function methods were employed to investigate the functional roles of miR-15b.

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MICRORNA-15B IS IMPLICATED IN REGULATING MYOCARDIAL REPERFUSION INJURY BY PROMOTING APOPTOSIS

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Objectives Myocardial ischaemia reperfusion (I/R) could induce altered expression of microRNAs (miRNAs), which served as powerful regulators of gene expression. Cancer studies have indicated that miR-15a, miR-15b and miR-16 have a potential relationship with apoptosis. The present study was aimed to reveal the roles of above miRNAs for the first time in myocardial I/R injury induced apoptosis.