



Effect and mechanism of miR-126 in myocardial ischemia reperfusion

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ABSTRACT. Multiple studies have shown microRNAs to play an important role in disease occurrence and development. The role of miRNAs in ischemia-reperfusion injury, however, requires further investigation and the aim of this study was therefore to assess miR-126 expression in myocardial ischemia reperfusion and the effects of miR-126 on myocardial ischemia-reperfusion injury. An *in vitro* model of ischemia-reperfusion injury was established using rat myocardial H9c2 cells and miR-126 expression in these cells was assessed by real-time PCR. The miR-126 mimic and inhibitor were transfected into H9c2 cells before the injury was induced. Flow cytometry and western blotting were used to assess myocardial cell apoptosis. The triphenyltetrazolium chloride method was used to assess the infarction area and a TUNEL assay was used to analyze myocardial cell apoptosis. The results of the western blot analyses indicate that the miR-126 mimic and inhibitor increase and decrease caspase 3 degradation in myocardial cells, respectively. The *in vivo* experiments, moreover, revealed that the miR-126 mimic and inhibitor increase and reduce the myocardial infarction area, respectively. The TUNEL assay results showed increases and decreases in apoptotic myocardial cell numbers after infusion with the miR-126 mimic or inhibitor, respectively. These findings indicate that miR-

126 is down-regulated in myocardial ischemia-reperfusion injury and that the inhibition of miR-126 may protect against myocardial cell apoptosis caused by ischemia-reperfusion.

Key words: miR-126; Myocardial ischemia-reperfusion; Apoptosis