group, TMP reduced apoptosis index (15.0±2.9% vs 25.2±3.3% in the IR group, $p<0.05$) and caspase-3 activity (4.03±1.14 nmol/mgpro vs 9.54±2.69 nmol/mgpro in the IR group, $p<0.05$). TMP resulted in a marked increase in NO_x (0.40±0.04 μmol/g vs 0.30 ±0.03 μmol/g in IR group, $p<0.05$). The expression of eNOS mRNA and protein in the myocardium of rats in the TMP group increased significantly compared to that in the IR group. However, these effects could be significantly reversed by L-NAME which abolished the increase of NO production and eNOS mRNA and protein expression brought by TMP.

Conclusions Ligustrazine offers antiapoptotic and cardioprotection effects against myocardial IR injury possibly through increasing the expression of eNOS and the level of NO.

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PROTECTIVE EFFECT OF LIGUSTRAZINE IN ATTENUATING MYOCARDIAL APOPTOSIS ON RATS WITH MYOCARDIAL ISCHAEMIA REPERFUSION AND ITS MECHANISM

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Objectives To investigate the effect of ligustrazine (tetramethylpyrazine, TMP) in attenuating myocardial apoptosis on rats with myocardial ischaemia reperfusion (IR) injury and to explore its mechanism.

Methods The animal model was built by ligation of the left anterior descending artery of rats (35 min of regional ischaemia followed by 120 min of reperfusion). Male Sprague-Dawley rats were randomly divided into sham, IR, TMP pretreated (TMP 10 mg/kg intravenous injection, 5 min before ligation), and TMP+L-NAME group (TMP 10 mg/kg intravenous injection, 5 min before ligation and L-NAME 30 mg/kg intravenous injection, 15 min before reperfusion). Caspase-3 activity and TUNEL staining were used to detect myocardial apoptosis. Nitric oxide production (NO_x) of myocardial tissue was examined using nitric oxide detection kit and expression of eNOS were observed by RT-PCR and western blot.

Results There were some TUNEL-positive staining myocardial cells in experimental groups except the sham group. Compared with IR