Conclusion Short-term use of CT-1C-terminal peptide early in reperfusion can reduce myocardial tissue injury and oxidative damage, as well as the extent of cardiomyocyte apoptosis, so that the extension of animal survival time; but the intraperitoneal injection of CT-1C-terminal peptide after a longer period of time reduced the tolerance of SD rats on ischaemia reperfusion injury, the tissue injury and the extent of oxidative damage increased significantly, and cardiac myocyte apoptosis have occurred in the surrounding area of infarction, and the animals have a shorter survival time.

Methods
Ischaemia-reperfusion (A) were made. But to B and C, rat hearts of globe ischaemia and 60 min of reperfusion, then the models of ischaemia postconditioning in normal rats (A group), ischaemia postconditioning in diabetic rats (C group). Rats were used for Langendorff isolated heart perfusion with 30 min of globe ischaemia and 60 min of reperfusion, then the models of ischaemia-reperfusion (A) were made. But to B and C, rat hearts were subjected to six cycles of 10 s of globe ischaemia and 10 s of reperfusion as ischaemia postconditioning during the early minutes of reperfusion. The levels of lactate dehydrogenase (LDH) in the coronary effluent and infarction size was determined by TTC staining. Phosphorylation of akt and gsk-3β were analysed by western blotting and immunohistochemical staining.

Results
Ischemic postconditioning reduced LDH, CK and improved the haemodynamic parameters and reduced myocardial infarction size (29.50±3.4% vs 45.65±4.8%), phospho-Akt and phospho-GSK-3β expression increased markedly in B group. But compared A group there were no parently difference in C group. The level of LDH, CK didn’t decline and the myocardial infarction size were not reduced. phospho-Akt and phospho-GSK-3β expression in C group is more than in B group.

Conclusion Ischemic postconditioning may significantly protect myocardium from reperfusion injury in isolated normal rat hearts. But in diabetic rats, the protection of Ischaemic postconditioning has no effect, the mechanism of this phenomenon maybe connected with lower expression of Phosphorylation of Akt and GSK-3β in the condition of diabetic and impaired Reperfusion Injury Salvage Kinase (RISK) signalling pathway (RISK pathway).

MMP-9 GENE POLYMORPHISMS CONTRIBUTE TO CORONARY ARTERY DISEASE RISK IN THE UIGHUR POPULATION OF CHINA

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Background Matrix metalloproteinase-9 (MMP-9) plays a pivotal role in early atherosclerosis, vascular remodelling and development of atherosclerotic lesion. The potentially functional MMP-9 polymorphisms may contribute to the susceptibility of coronary artery disease (CAD). We aimed to investigate the association between three SNPs (−1562C>T, R279Q, R668G) of the MMP-9 gene with CAD in the Uighur population of China.

Materials and methods 375 angiographic allied proven patients with coronary artery disease and 417 sex-matched and ethnically matched controls were genotyped for MMP-9 polymorphisms by the PCR-restriction fragment length polymorphism (PCR-RFLP) technique. Genotype/allele frequencies were compared in patients and controls using the χ² test. The relationship between the polymorphism of the MMP-9 gene and the severity of coronary arterial stenosis was analysed also.

Results At MMP-9 -1562 locus, there were significant differences between patients and controls (p<0.05), leading to significant OR for TT genotype (OR=2.93, CI 1.03 to 8.72) and R allele (OR=1.85,