Background: Ischemic postconditioning (I-postC) is a newly discovered endogenous protective mechanism capable of protecting the myocardium from ischemia/reperfusion (I/R) injury. I-postC can be evoked by applying cycles of brief intermittent interruption of blood flow to the myocardium during the early reperfusion after a prolonged period of ischemia. This will protect the heart against myocardial infarction and reduce coronary endothelium dysfunction to an extent comparable to ischemic preconditioning (IPC). The fact that I-postC can be applied after a prolonged period of ischemia offers a novel approach to myocardial protection. It has been suggested that I/R injury causes endoplasmic reticulum (ER) stress-related apoptosis and I-postC attenuates apoptosis induced by I/R. The purpose of the present study was to determine whether I-postC attenuation of I/R injury involves reductions in ER stress mediated through signaling by mitogen-activated protein kinases (MAPKs).

Methods: A rat myocardial ischemia reperfusion model was used. Electron microscopy and flow cytometry were used to quantitate cardiomyocyte apoptosis. Myocardial expression of calreticulin, caspase-12 and activation of caspase-12, p38 MAPK, and JNK in myocardium or cardiomyocytes were measured by Western blots.

Results: It is found that I-postC protects the I/R heart against myocardial infarction and H-postC protects neonatal cardiomyocytes from hypoxia/reoxygenation-induced apoptosis to an extent comparable to ischemic preconditioning. I-postC suppressed I/R-induced ER stress, as shown by a decrease in expression of CRT and caspase-12 activation. H-postC upregulates p38 MAPK phosphorylation and downregulates JNK phosphorylation in cardiomyocytes subjected to H/R.

Conclusion: These results indicate that I-postC protects cardiomyocytes from I/R injury through suppressing ER stress by balancing the activation of p38 MAPK/JNK.