RISK and SAFE signaling pathway interactions in remote limb ischemic perconditioning in combination with local ischemic postconditioning

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Résumé en anglais	Local ischemic postconditioning (IPost) and remote ischemic perconditioning (RIPer) are promising methods to decrease ischemia–reperfusion (I/R) injury. We tested whether the use of the two procedures in combination led to an improvement in cardioprotection through a higher activation of survival signaling pathways. Rats exposed to myocardial I/R were allocated to one of the following four groups: Control, no intervention at myocardial reperfusion; IPost, three cycles of 10-s coronary artery occlusion followed by 10-s reperfusion applied at the onset of myocardial reperfusion; RIPer, 10-min limb ischemia followed by 10-min reperfusion initiated 20 min after coronary artery occlusion; IPost+RIPer, IPost and RIPer in combination. Infarct size was significantly reduced in both IPost and RIPer (34.25 ± 3.36 and 24.69 ± 6.02%, respectively) groups compared to Control (54.93 ± 6.46%, both $p < 0.05$). IPost+RIPer (infarct size = 18.04 ± 4.86%) was significantly more cardioprotective than IPost alone ($p < 0.05$). RISK pathway (Akt, ERK1/2, and GSK-3β) activation was enhanced in IPost, RIPer, and IPost+RIPer groups compared to Control. IPost+RIPer did not enhance RISK pathway activation as compared to IPost alone, but instead increased phospho-STAT-3 levels, highlighting the crucial role of the SAFE pathway. In IPost+RIPer, a SAFE inhibitor (AG490) abolished cardioprotection and blocked both Akt and GSK-3β phosphorylations, whereas RISK inhibitors (wortmannin or U0126) abolished cardioprotection and blocked STAT-3 phosphorylation. In our experimental model, the combination of IPost and RIPer improved cardioprotection through the recruitment of the SAFE pathway. Our findings also indicate that cross talk exists between the RISK and SAFE pathways.

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