RISK and SAFE Signaling Pathway Involvement in Apolipoprotein A-I-Induced Cardioprotection

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Résumé en anglais	Recent findings indicate that apolipoprotein A-I (ApoA-I) may be a protective humoral mediator involved in remote ischemic preconditioning (RIPC). This study sought to determine if ApoA-I mediates its protective effects via the RISK and SAFE signaling pathways implicated in RIPC. Wistar rats were allocated to one of the following groups. Control: rats were subjected to myocardial ischemia/reperfusion (I/R) without any further intervention; RIPC: four cycles of limb I/R were applied prior to myocardial ischemia; ApoA-I: 10 mg/Kg of ApoA-I were intravenously injected prior to myocardial ischemia; ApoA-I + inhibitor: pharmacological inhibitors of RISK/SAFE pro-survival kinase (Akt, ERK1/2 and STAT-3) were administered prior to ApoA-I injection. Infarct size was significantly reduced in the RIPC group compared to Control. Similarly, ApoA-I injection efficiently protected the heart, recapitulating RIPC-induced cardioprotection. The ApoA-I protective effect was associated with Akt and GSK-3β phosphorylation and substantially inhibited by pretreatment with Akt and ERK1/2 inhibitors. Pretreatment with ApoA-I in a rat model of I/R recapitulates RIPC-induced cardioprotection and shares some similar molecular mechanisms with those of RIPC-involved protection of the heart

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