Apolipoprotein a-I is a potential mediator of remote ischemic preconditioning.

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Apolipoprotein a-I is a potential mediator of remote ischemic preconditioning.

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BACKGROUND: Remote ischemic preconditioning (RIPC) has emerged as an attractive strategy in clinical settings. Despite convincing evidence of the critical role played by circulating humoral mediators, their actual identities remain unknown. In this study, we aimed to identify RIPC-induced humoral mediators using a proteomic approach.

METHODS: and Results Rats were exposed to 10-min limb ischemia followed by 5- (RIPC 5') or 10-min (RIPC 10') reperfusion prior to blood sampling. The control group only underwent blood sampling. Plasma samples were analyzed using surface-enhanced laser desorption and ionization - time of flight - mass spectrometry (SELDI-TOF-MS). Three protein peaks were selected for their significant increase in RIPC 10'. They were identified and confirmed as apolipoprotein A-I (ApoA-I). Additional rats were exposed to myocardial ischemia-reperfusion (I/R) and assigned to one of the following groups RIPC+myocardial infarction (MI) (10-min limb ischemia followed by 10-min reperfusion initiated 20 minutes prior to myocardial I/R), ApoA-I+MI (10 mg/kg ApoA-I injection 10 minutes before myocardial I/R), and MI (no further intervention). In comparison with untreated MI rats, RIPC reduced infarct size (52.2±3.7% in RIPC+MI vs. 64.9±2.6% in MI; p<0.05). Similarly, ApoA-I injection decreased infarct size (50.9±3.8%; p<0.05 vs. MI).

CONCLUSIONS: RIPC was associated with a plasmatic increase in ApoA-I. Furthermore, ApoA-I injection before myocardial I/R recapitulated the cardioprotection offered by RIPC in rats. This data suggests that ApoA-I may be a protective blood-borne factor involved in the RIPC mechanism.

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