neuropeptides, calcitonin gene-related peptide (CGRP) and substance P (SP), are responsible for the transfer of cardioprotection by remote limb ischemic postconditioning (RIPostC) from remote limb to heart during myocardial ischemia/reperfusion.

Methods Sprague–Dawley rats were subjected to 30 min of myocardial ischemia (ligation of left anterior descending coronary artery) followed by 3 h of reperfusion (release of the ligation). RIPostC was initiated by 3 cycles of 5 min of ischemia and 5 min of reperfusion on both hind limbs immediately at the onset of myocardial reperfusion using a natural rubber tourniquet in the presence or absence of deinnervation of $TRPV1^+$ sensory nerves, TRPV1 receptor antagonist capsazepine, CGRP receptor antagonist CGRP8-37 or SP receptor antagonist RP-67580. The levels of CGRP and SP in plasma and hearts, as well as the levels and mRNA expression of CGRP and SP in dorsal root ganglia (DRG) were measured immediately after RIPostC. In addition, infarct sise, serum creatine kinase (CK) and cardiac troponin I (TnI) levels, and cardiac function parameters including heart rate (HR), left ventricular end-diastolic pressure (LVEDP), left ventricular systolic pressure (LVSP) and maximum rise/fall rate of LV pressure (±dP/dtmax) were monitored at the end of 3 h of myocardial reperfusion. Results: RIPostC resulted in a significant increase in the levels of CGRP and SP in plasma and hearts, as well as the levels and mRNA expression of CGRP and SP in DRG. The increase in CGRP and SP levels in plasma and hearts were markedly inhibited in the presence of deinnervation of $TRPV1^+$ sensory nerves, TRPV1 receptor antagonist capsazepine, CGRP receptor antagonist CGRP8-37 or SP receptor antagonist RP-67580. In addition, RIPostC effectively protected hearts against ischemia/reperfusion injury by limiting infarct sise, lowering CK and cTnI release and improving cardiac function including decrease in LEVDP and increase in LVSP and ±dP/dtmax, which could be significantly abolished by deinnervation of $TRPV1^+$ sensory nerves or inhibition of TRPV1, CGRP or SP receptor.

Conclusions The beneficial effects of RIPostC against myocardial ischemia/reperfusion are associated with TRPV1 and its main neuropeptides, CGRP and SP. These findings indicate that TRPV1 contributes to the transfer of cardioprotection by RIPostC from remote limb to heart through increase in the release of CGRP and SP in circulation and hearts.

[gw22-e0771]

TRANSIENT RECEPTOR POTENTIAL VANILLOID 1 IS INVOLVED IN THE TRANSFER OF CARDIOPROTECTION BY REMOTE LIMB ISCHEMIC POSTCONDITIONING IN RATS

Song Jun-xian, Ren Jing-yi, Han Guan-ping, Chen Hong *Department of Cardiology, Peking University People´s Hospital, Beijing, China*

10.1136/heartjnl-2011-300867.237

Objective This study aimed at investigating whether transient receptor potential vanilloid 1 (TRPV1) and its main