developed pressure (LVDP), the maximum change rate of left ventricular pressure rise and fall (±dp/dtmax) were recorded. The activity of creatine kinase (CK) in coronary outflow, the activity of malonyldialdehyde (MDA) and superoxide dismutase (SOD) in myocardium were detected. The percentage of necrotic area were observed.

**Results** In adult rats, the content of CK (89.48±18.72 U/l vs 115.76±16.72U/l, p<0.01) and MDA (9.53±3.44 nmol/mg vs 16.84±2.29 nmol/mg, p<0.01) were significantly less in IPC group than those in I/R group. In IPC group, the activity of SOD (584.7±122.62 U/mg vs 429.46±55.24 U/mg, the recovery rate of the left ventricular function, including CO, LVDP and±dp/dtmax, were much higher than those in I/R group (78.69±9.68% vs 65.10±8.63%, 83.61±8.46% vs 67.23±8.68%, 81.66±8.6% vs 67.89±8.89%, 89.79±7.78% vs 66.79±8.46%, p<0.01). And the percentage of necrotic area were lower in adult IPC group than in I/R group (5.25±4.33 vs 14.75±8.02, p<0.01). But there were no significant changes between IPC group and I/R group in elderly rats (p>0.05).

However, there were great significant changes between enhanced IPC group and IR group in elderly rats, the content of CK (58.60±28.52 U/l vs 105.76±9.64 U/l, p<0.01) and MDA (3.38±3.56 nmol/mg vs 16.80±3.06 nmol/mg, p<0.05), the activity of SOD (559.57±78.66 U/mg vs 453.75±56.65 U/mg, p<0.01), the recovery rate of the left ventricular function, such as CO, LVDP and±dp/dtmax, were much higher than those in I/R group (77.99±10.02% vs 66.26±9.78%, 85.59±6.67% vs 73.90±6.66%, 83.87±9.92% vs 68.90±8.68%, 86.01±7.66% vs 70.39±7.98%, p<0.01). The percentage of necrotic area were lower in elderly IPC group than in I/R group (7.95±6.32% vs 15.62±10.56%, p<0.01).

**Conclusion** The effect of IPC on ischemic reperfused myocardial of elderly rats was weaken. Prolonged ischemia was able to resume the protective effect of IPC on elderly rat hearts.

**e0016 TONCXINLUO REDUCES MYOCARDIAL ISCHAEMIA-REFUSION INJURY AND NO-REFLOW BY STIMULATING THE EXPRESSION AND PHOSPHORYLATION OF ENOS VIA PKA PATHWAY**

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**Objective** To investigate whether oral administration of Tонксинлуо (TXL), a traditional Chinese medicine, at a single low loading dose 1 h before myocardial ischaemia can attenuate ischaemia-reperfusion injury by upregulating the expression of eNOS and p-eNOS (Ser 1179 and Ser 635), and this effect is partially mediated by PKA pathway.

**Methods and results** In 30-min ischaemia and 3-h reperfusion model, Minipigs were randomly assigned to four groups (n=8 in each group): (1) Sham; (2) Control; (3) TXL: 0.05 μg·kg⁻¹ of TXL was gavaged 1 h prior myocardial ischaemia; (4) TXL: H89 (1.0 μg·kg⁻¹·min⁻¹, an inhibitor of PKA). TXL significantly decreased creatine kinase (CK) activity, reduced the infarct size from 78.5% to 59.2% and no-reflow area from 48.6% to 9.5% (p<0.05), while H-89 completely abolished the reduction of CK activity and necrosis size, and partially diminished the reduction of no-reflow size. TXL enhanced the PKA activity in ischaemic myocardium, increased the expression of Pka, Thr 198 p-PKA and Ser 635 p-eNOS in no-reflow area, and upregulated the expression of eNOS and Ser 1179 p-eNOS in reflow area. H-89 repressed the enhancement of PKA activity and the upregulation of eNOS and Ser 635 p-eNOS, but without great inhibition on the expression of PKA and Thr 198 p-PKA in no-reflow area, and even stimulated the expression of Ser 635 p-eNOS in reflow area.

**Conclusion** Pretreatment with single low loading dose of TXL 1 h before myocardial ischaemia reduces myocardial no-reflow and ischaemia-reperfusion injury by upregulating the expression of eNOS and p-eNOS (Ser 1179 and Ser 635), and this effect is partially mediated by PKA pathway.