Basic Science: Cardiovascular Disease Basic Research

**e0001** THE EFFECTS OF TRANS FATTY ACIDS ON FATTY ACID
CONSTITUTION RATIOS OF ERYTHROCYTE MEMBRANE IN
RABBITS

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**Objective** To investigate the influences of high trans fatty acids (TFA) intake on fatty-acid constitution ratios of erythrocyte membrane in rabbits.

**Method** 32 New Zealand white rabbits were randomly divided into four groups: control group with common feed; high TFA group with additional TFA 5.0 g/kg/day; high-fat (HF) group with high cholesterol feed and TFA+HF group. The erythrocyte membranes were prepared at 0, 4, 8, 12 weeks. Two kinds of saturated FA (C18:0 and C16:0), four kinds of unsaturated FA (C18:1, C18:2, C20:4, C20:5 and total ω–3) and two kinds of TFA (c-tC18:1 and t-C16:1) in erythrocyte membrane were determined with gas chroma. Four constitution ratios of C18:1/C18:0, C18:1/t-C18:1, ω–3/C18:0 and ω–3/C18:0 were calculated.

**Results** Compared with the control group, TFA group showed not only obviously higher constitution ratios of TFA, but obviously higher ratios of saturated FA and lower ratios of polyunsaturated FA, especially ω–3 FA (2.38±0.35 vs 3.28±0.48, p<0.05), in erythrocyte membrane. The abnormality of constitution ratios of unsaturated FA and polyunsaturated FA in TFA were similar to that in HF group. More abnormal changes of erythrocyte membrane FA constitution ratios were showed in TFA+HF group.

**Conclusions** High TFA intake could increase the constitution ratios of TFA and saturated fatty acids, but decrease polyunsaturated fatty acids, especially ω–3 fatty acids, in erythrocyte membrane. These effects were equivalent with the effects of high cholesterol intake. Combined with TFA and high cholesterol intake had obviously synergistic effects.

**e0002** EFFECT OF TOLL-LIKE RECEPTOR-4 SIGNAL PATHWAY ON THE DISFUNCTION OF CARDIOVASCULAR ENDOTHELIAL CELLS CAUSED BY HYPOXIA/
REOXYGENATION INJURY

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**Aim** TLR-4 has been proved to take part in MIRI of heart. But the mechanism is not clear. To explore the change of TLR-4 signal pathway during hypoxia–reoxygenation injury (H-R) of cardiac microvascular endothelial cells (CMECs). In vivo has not yet been investigated.

**Methods** We generated transgenic mice with cardiac-specific overexpression of Nrdp1 using α-myosin heavy chain promoter. Echocardiography demonstrated that cardiac-specific Nrdp1 expression resulted in depression of cardiac contractile function under basal condition (TG6 mice, EF, 62.74±4.40%, FS, 33.55±5.17%, WT mice, EF, 67.52±11.07%, FS, 37.64±6.64%). When subjected to 30 min of left coronary artery ischaemia and 24 h of reperfusion, the infarct size, expressed as the ratio of infarct/AAR and infarct/LV, was significantly increased in Nrdp1 transgenic mice compared with WT mice (22.83%; 15.78%) neutrophil and macrophage infiltration after I/R in Nrdp1 transgenic mice than in WT mice (p<0.05). Additionally, the activation of ErbB3, AKT, ERK1/2 and STAT3 after I/R were markedly suppressed in Nrdp1 transgenic mice compared with WT mice (p<0.01).

**Conclusion** These data provide the first in vivo evidence that overexpression of Nrdp1 enhances cardiac I/R injury, this effect is mediated by inhibition of ErbB3-dependent signalling pathways.