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.d; 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 (t-C18:1 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/∑TFA 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 DYSFUNCTION OF CARDIAC MICROVASCULAR 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 researches mostly focused on the relationship between TLR-4 and global heart dysfunction or cadiocyte apoptosis. The effect of TLR-4 on CMECs which are the most important component in MIRI is not clear. To explore the change of TLR-4 signal pathoway during hypoxia−reoxygenation (H-R) of cardiac microvascular endothelial cells (CMECs) injury.
Methods The CMECs were isolated from the hearts of adult rats. The obtained CMECs were exposed to hypoxia (940 ml/l N_2, 50 ml/l CO_2 and 10 ml/l O_2) for 6 h, following by reoxygenation (950 ml/l air, 50 ml/l CO_2) for 2 h, 12 h or 24 h. The proliferation of CMECs was assessed by MTT colourimetry. TLR-4 and NF-κB expressions were analysed by Western blot. The levels of IL-6 and TNF-α were detected by ELISA.
Results The proliferation ability of CMECs was significantly inhibited by H-R injury ($p<0.01$). H-R injury increased TLR-4 expression after 2 h or 12 h reoxygenation ($p<0.05$). The level of NF-κB increased after 2 h and 24 h reoxygenation ($p<0.05$). H-R injury enhanced IL-6 and TNF-α secretion as compared with the control group ($p<0.05$).
Conclusion H-R injury increases TLR-4 and NF-κB expressions in CMECs and enhances the secretions of IL-6 and TNF-α. The activation of TLR-4 signal pathway on CMECs may participate in the H-R induced of CMECs injury.

e0003 CARDIAC-SPECIFIC EXPRESSION OF E3 LIGASE NRDP1 INCREASES ISCHAEMIA AND REPERFUSION-INDUCED CARDIAC INJURY IN TRANSGENIC MICE

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Objective Neuregulin receptor degradation protein-1 (Nrdp1) is an E3 ubiquitin ligase that regulates the proteasomal degradation and activity of proteins involved in cell growth, inflammation and apoptosis, including ErbB3, BRUCE, MyD88 and TBK1. However, the effect of Nrdp1 on cardiac ischaemia/reperfusion (I/R) injury in vivo has not yet been investigated.
Methods and results 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, 33.53±3.17%; WT mice, EF, 67.52±11.07%; FS, 37.64±8.64%). 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 TG6 mice (28.6%; 17.0%) compared with that of WT mice (18.4%; 11.4%, $p<0.05$). Furthermore, the survival rate after I/R in Nrdp1 TG6 mice (75.9%, 22/29) was significantly lower than that of WT mice (84.6%, 22/26). Moreover, the numbers of TUNEL-positive nuclei (22.83%; 15.78%) neutrophil and macrophage infiltration after I/R were significantly higher 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.

e0004 HYDROGEN SULFIDE INHABITS NEURONS APOPTOSIS IN RATS AFTER CARDIOPULMONARY RESUSCITATION

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Objective To investigate the effects of hydrogen sulfide (H_2S) on brain injury after cardiopulmonary resuscitation (CPR) in rats by examining neurons apoptosis.
Methods The 40 male SD rats were randomly divided into experimental and control groups equally. In control group, CPR was performed with Utstein mode at 6 min after CA. On this basis, sodium hydrosulfide was administrated to the rats after restoration