enhance these protective effects in the hearts of diabetic rats. Chronic Resveratrol treatment with or without the diabetic rats significantly reduced the infarct size in a dose-dependent manner, and the increase of LDH and CK levels were significantly inhibited by dobutamine (both \( P < 0.05 \) versus IR group). Meanwhile, dobutamine dose-dependently reduced oxidative stress by inhibiting the increase of the MDA level and the decrease of the SOD level (both \( P < 0.05 \) versus IR group). Otherwise, dobutamine produced a statistically significant reduction in the production of TNF-\( \alpha \) and IL-6 compared with the IR group (both \( P < 0.05 \) versus IR group). Furthermore, compared with IR group, dobutamine significantly and dose-dependently mediated HO-1 induction and NF-kB p65 and HMGB1 inhibition (all \( P < 0.05 \) versus IR group). However, all these effects were caused by dobutamine dosages significantly reversed by the presence of LY294002, SB203580, and ZnPPiX, respectively.

Conclusions: The present study demonstrated that dobutamine mediated the induction of HO-1 by selectively stimulating \( \beta \)-adrenergic receptor via PKA and p38 MAPK, which inhibited HMGB1 release and attenuated rat myocardial IR injury in vivo.

GW25-c3132

Single nucleotide Polymorphism of the CY2P22 Gene is Associated with Essential Hypertension in Uygur Population in China

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Objectives: Human Cytochrome P450 2J2 (CY2P22) is the major arachidonic acid epoxygenase, which can metabolizes arachidonic acid (AA) to bioactive epoxyeicosatrienoic acids (EETs). The EETs are potent endogenous vasodilators and inhibitors of vascular inflammation. Recently, much evidence from models and human studies has shown that variability in CY2P22 gene plays a mechanical role in the development of hypertension. The aim of the present study was to assess the association between the CY2P22 gene polymorphism and Essential Hypertension (EH) in a Han and Uygur population in China.

Methods: We used two independent case-control studies: a Han population (302 EH patients and 300 control subjects) and a Uygur population (567 EH patients and 215 control subjects). All EH patients and controls were genotyped for the same three single nucleotide polymorphisms (SNPs) (rs809293, rs11572223 and rs2280275) of CY2P22 gene by a Real-time PCR instrument.

Results: In the Uygur population, the distribution of SNP3 (rs2280275) genotypes, alleles and the dominant model (CC vs CT + TT) showed a significant difference between EH and control participants (for genotype: \( P = 0.007; \) for allele: \( P = 0.001; \) for dominant model: \( P = 0.002 \)). The significant difference in dominant model was retained after adjustment for covariates (OR: 3.500, 95% confidence interval [CI]: 1.680-7.300, \( P = 0.001 \)). However, all the above differences were not shown in the Han population.

Conclusions: The CC genotype of rs2280275 in CY2P22 gene could be a risk genetic marker of EH and T allele may be a protective genetic marker of EH in Uygur population in China.

GW25-c3140

Endothelial cells induced by inflammation release multiple angiogenesis-associated microRNAs into circulation through microparticles

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Objectives: Recent studies demonstrated that endothelial-derived microRNAs (miRNAs) can be detected in clinical plasma/serum samples, and there are also evidences indicated that inflammation disease states (e.g., atherosclerosis, cancer, etc.) may affect the levels of circulating miRNAs, but so far there is no direct proof about whether inflammation could induce endothelial cells to release miRNAs into circulation. This study aimed to explore whether inflammation could induce endothelial cells to release miRNAs into circulation and to investigate whether these released miRNAs derived from endothelial cells were transported in microparticles.

Methods: Microparticles were isolated from human atherosclerotic plaques with a single nucleotide polymorphism of the CYP2J2 Gene in a Han and Uygur population in China.

GW25-c3129

\( \beta \)-Adrenergic receptor-mediated HO-1/HMGB1 axis via PI3K and p38 MAPK attenuates rat myocardial ischemia/reperfusion injury in vivo

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Objectives: \( \beta \)-adrenergic receptor-mediated HO-1/HMGB1 axis via PI3K and p38 MAPK attenuates rat myocardial ischemia/reperfusion injury in vivo.

Methods: Anesthetized male rats were pretreated with dobutamine (5 or 10 mg/kg, i.v.) for 24 hours, before ischemia in the absence and/or presence of LY294002 (20 ng/ml, i.v.) or TNF-\( \alpha \). After 4 h reperfusion, the hearts were harvested, and myocardial infarction size was assessed.

Results: After 4 h reperfusion, compared with IR group, the pretreatment of \( \beta \)-adrenergic receptor-mediated HO-1/HMGB1 axis via PI3K and p38 MAPK significantly reduced infarct size in a dose-dependent manner, and the increase of LDH and CK levels were significantly inhibited by dobutamine (both \( P < 0.05 \) versus IR group). Meanwhile, dobutamine dose-dependently attenuated HO-1 induction and NF-kB p65 and HMGB1 inhibition (all \( P < 0.05 \) versus IR group). However, all these effects were caused by dobutamine dosages significantly reversed by the presence of LY294002, SB203580, and ZnPPiX, respectively.

Conclusions: The present study demonstrated that dobutamine mediated the induction of HO-1 by selectively stimulating \( \beta \)-adrenergic receptor via PI3K and p38 MAPK, which inhibited HMGB1 release and attenuated rat myocardial IR injury in vivo.