

Design Growth-arrested AFs were incubated in serum-free medium with UII and some inhibitors of signal transduction pathways. Cell migration was determined by a transwell technique. The OPN mRNA expression and protein secretion induced by UII were evaluated by the reverse transcriptase PCR and ELISA method, respectively.

Results OPN antisense oligonucleotides inhibited UII-induced AFs migration significantly compared with UII (10^{-8} mol/l) group ($p < 0.05$). Moreover, UII promoted the OPN mRNA expression and protein secretion in a dose-dependent and time-dependent manner, with maximal effect at a concentration of 10^{-8} mol/l at 3 h for mRNA expression, or at 24 h for protein secretion, respectively ($p < 0.01$). The UII receptor antagonist SB710411 (10^{-6} mol/l), Ca^{2+} channel blocker nifedipine (10^{-5} mol/l), protein kinase C inhibitor H7 (10^{-5} mol/l), calcineurin inhibitor cyclosporine A (10^{-5} mol/l), Rho kinase inhibitor Y-27632 (10^{-5} mol/l) and mitogen activated protein kinase (MAPK) inhibitor PD98059 (10^{-5} mol/l) inhibited the UII effects significantly.

Conclusion This study indicated that UII may up-regulate OPN expression in AFs through the UII receptor, protein kinase C, MAPK, calcineurin, Rho kinase and Ca^{2+} signal transduction pathways, and OPN is involved in UII-induced AFs migration.

e0160 TRANSIENT PREHYPERTENSIVE TREATMENT IN SPONTANEOUSLY HYPERTENSIVE RATS: A COMPARISON OF LOSARTAN AND AMLODIPINE REGARDING LONG-TERM BLOOD PRESSURE AND RENAL PROTECTIVE EFFECT

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Aims To compare the effectiveness of transient prehypertensive treatment with losartan vs amlodipine in spontaneously hypertensive rats (SHR) on long-term blood pressure and cardiac protection. **Main methods** SHR were prehypertensively (weeks 4–10 of age) treated with losartan (SHR-Los: 20 mg/kg/day), amlodipine (SHR-Aml: 10 mg/kg/day) or saline ($n=24$ each group). Rats were followed up until week 46. Systolic blood pressure (SBP) was measured by tail-cuff method. Cardiac parameters including Left ventricular (LV) mass index (LVMI), collagen volume fraction (CVF) and LV function were assessed by histomorphometry and echocardiography. Plasma and myocardium Angiotensin II (Ang II) and aldosterone (Aldo) were measured by radioimmunoassay. Cardiac angiotensin II type 1 and type 2 receptor (AT1R and AT2R) protein were determined by immunoblotting and brain natriuretic peptide (BNP) mRNA was semi-quantified by reverse transcription-PCR (RT-PCR).

Key findings The SBP in SHR-Los was reduced until age 46 weeks, but returned to untreated SHR levels in SHR-Aml from 30 weeks onwards. Compared to untreated SHR, the LVMI and CVF in SHR-Los were markedly decreased until week 46, and the LV ejection fraction (LVEF) (SHR-Los vs SHR: $83.1 \pm 2.3\%$ vs $79.5 \pm 1.9\%$, $p < 0.05$) and cardiac BNP mRNA expression were improved, whereas comparable LVMI and elevated CVF were found in SHR-Aml, and the LVEF fell significantly below that of untreated SHR at week 46 (SHR-Aml vs SHR: $74.4 \pm 4.3\%$ vs $79.5 \pm 1.9\%$, $p < 0.05$), with cardiac BNP mRNA expression increasing slightly. Compared to untreated SHR, the plasma and myocardium AngII and Aldo levels in SHR-Los at week 46 were remarkably decreased (plasma AngII: 302 ± 32 vs 458 ± 32 pg/ml; plasma Aldo: 172 ± 20 vs 252 ± 41 pg/ml; cardiac Ang II: 126 ± 11 vs 199 ± 14 pg/100mg; cardiac Aldo: 497 ± 43 vs 766 ± 46 pg/100 mg, all $p < 0.05$), and the cardiac AT1R protein was down-regulated and AT2R protein was

up-regulated, no significant difference of these indices was found between SHR-Aml and untreated SHR. Significance Prehypertensive treatment with losartan was more effective than amlodipine on delaying long-term blood pressure rise and meliorating cardiac structure and function, which might be related to permanent attenuation of circulating and local renin-angiotensin (R-A) systems.

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Competing interests None.

e0161 ESTABLISHMENT OF MINIPIG MODEL OF ISCHAEMIC HEART FAILURE WITH ACUTE MYOCARDIAL INFARCTION BY CORONARY OCCLUSION WITH BALLOON OCCLUDING AND INTERMIXTURE INJECTING OF MICROTHROMBI AND PLASTIC MICROSPHERES

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Objective To evaluate the of method of minipig model of ischaemic heart failure (HF) with acute myocardial infarction (AMI) by coronary occlusion with balloon occluding and coadministration injecting of microthrombi and plastic microspheres.

Methods A total of 18 minipigs were selected. After coronary angiography, angioplasty balloons were placed in the mid-distal of left anterior descending (LAD). The balloon was inflated intermittently to occlude the LAD 3 times and then occlude continuously for 120 min. After the balloon was taken out, 4F judkins angiographic catheter was superselectively engaged in LAD and 3 ml intermixture of microthrombi and plastic microspheres were injected at 10 min interval until TIMI myocardial perfusion grade < 2 and left ventricular end-diastolic pressure maintaining from 15 to 18 mm Hg. Electrocardiography (ECG), haemodynamic parameters, ultrasonic cardiogram, cTnI and CK-MB were measured. Myocardial infarcted area was evaluated with pathologic examination.

Results 14 days later, 15 minipigs survived and fourteen satisfied the criteria (pulmonary capillary wedge pressure, PCWP > 18 mm Hg and cardio output, CO decreased beyond 30%). The change of ECG, haemodynamic parameters, CKMB, cTnI and cardiac pathologic examination were in accordance with AMI. Occlusion A stable experimental method of minipig model of ischaemic heart failure (HF) with acute myocardial infarction (AMI) by coronary occlusion with balloon occluding and coadministration injecting of microthrombi and plastic microspheres was established. The method had advantages of closed chest, higher succeed rate and stability to those of drug induced, tachycardia-pacing induced, coronary artery ligation induced or microsphere injection alone.

e0162 THE RELATIONSHIP BETWEEN THE G (+252)A POLYMORPHISM OF β_2 -ADRENERGIC RECEPTOR GENE AND ESSENTIAL HYPERTENSION IN KAZAKS OF XINJIANG

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Introduction Hypertension is a major risk factor for coronary artery disease, stroke, and renal disease. Recent research suggest genetic polymorphisms of β_2 -adrenergic receptor gene (ADRB2) have already been associated with obesity, diabetes mellitus, and essential