pathway increased, while AT2 level decreased. Interestingly, PLA2 and COX1/COX2 expressions in AA pathway increased in model group and their expressions were down-regulated by ACEI drug captopril, indicating that AA pathway was activated by up-regulation of RAAS. As important signal-transducing proteins, JAK1/STAT3, NFκb and Akt expressions all increased in model group remarkably. QSYQ treatment could attenuate myocardial fibrosis and this efficacy were further approved by decreasing levels of collagen I, collagen III, MMP2 and MMP9 in QSYQ group. RAAS pathway was inhibited by QSYQ, as indicated by decreased AT1 and increased AT2 expressions. PLA2, COX1 and COX2 expressions were also down-regulated in QSYQ group. In addition, “therapeutic” QSYQ administration seemed to down-regulate JAK1/STAT3, NFκb and Akt expressions which maybe play important roles in myocardial fibrosis.

CONCLUSIONS AA pathway in myocardial fibrosis may be mediated by activation of RAAS in HF model rats. QSYQ can exert anti-fibrosis effect by downregulating expressions of RAAS pathway, and subsequently inhibiting proteins in AA pathway. The mechanism of QSYQ cardioprotective efficacy may be through RAAS-JAK1/STAT3, Akt and PLA2-COXs-NFκb pathways. Our study provides new insights into the complicated mechanism of QSYQ in the clinical treatment of HF.

GW26-e1244 Effects of Lipopolysaccharide on the Growth and Proliferation of Human Coronary Artery Smooth Muscle Cells in Vitro
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OBJECTIVES To explore the safe range of lipopolysaccharide (LPS) density and provide the experimental data for the related research of human coronary artery smooth muscle cells (HACSMC) associated with inflammation, we investigated the effects of different ratios of LPS at different points on the growth of HACSMC cultivated in vitro.

METHODS The 3-5 generations of HACSMC was respectively seeded onto 96-well plates, then co-incubated with different concentration of LPS (0, 0.01, 0.1, 0.5, 1.5, 3, 10, 100 ng/ml) at different points (24, 48, and 72h). Then cell viability was determined via methylthiazolite-tetrazolium (MTT) assay. We determined the optimal concentration range and time of LPS for promoting the growth of HACSMC.

RESULTS The effect of LPS on the growth and proliferation of HACSMC was related with the density and action-time. Beyond certain density, the longer action-time of LPS (0, 24, 48h) did not inhibitory the growth of HACSMC but could increase the growth of HACSMC. The longer action-time of LPS (72h) with high dose (1000 ng/ml) could obviously inhibit the growth of HACSMC and the inhibitory rate increased with the increase in the concentration of LPS. Besides, with the longer action-time would decrease the cell viability of HACSMC more obviously.

CONCLUSIONS The low dose of LPS (<10ug/ml) cannot obviously produce cytotoxicity of HACSMC, and 0.10ug/ml LPS with action-time of 24h can maximize the proliferation of HACSMC, which can be used in the experimental study of HACSMC cultured in the inflammation condition.

GW26-e1257 Perivascular adipose tissue-derived adiponectin inhibits collar-induced carotid atherosclerosis by promoting macrophage autophagy
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OBJECTIVES Adiponectin (APN) secreted from perivascular adipose tissue (PVAT) is one of the important anti-inflammatory adipokines to inhibit development of atherosclerosis, but the underlying mechanism has not been clarified. In this study, we aimed to elucidate how APN regulates plaque formation in atherosclerosis.

METHODS To assess the role of APN secreted by PVAT in atherosclerosis progression, we performed PVAT transplantation experiments on carotid artery atherosclerosis model: ApoE knockout (ApoE−/−) mice with a perivascular collar placement around the left carotid artery in combination with a high-fat diet feeding.

RESULTS Our results showed that the ApoE−/− mice with PVAT derived from APN knockout (APN−/−) mice exhibited accelerated plaque volume formation compared to ApoE−/− mice transplanted with wild-type littersmate tissue. Conversely, autophagy in macrophages was significantly attenuated in ApoE−/− mice transplanted with APN−/− mouse-derived PVAT compared to controls. Furthermore, in vitro studies indicated APN induced autophagy in primary macrophages, as evidenced by increased LC3-I processing and Beclin expression, which was accompanied by down-regulation of p62. Moreover, our results showed that APN promotes macrophage autophagy by suppressing the Akt/FOXO3a signaling pathway.

CONCLUSIONS Our studies demonstrated that PVAT-secreted APN suppresses plaque formation by inducing macrophage autophagy.

GW26-e1351 Effect of Rosuvastatin on Blood Pressure, Mesenteric Arteries Structure and Vasodilatation Function in Spontaneously Hypertensive Rats
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OBJECTIVES To study the effect of Rosuvastatin (Rsv) on blood pressure, vascular structure and vasodilatation function of 3rd grade branch mesenteric in spontaneously hypertensive rats (SHRs).

METHODS Thirty-two male SHRs at 12 wks old were randomly divided into 2 groups: Rosuvastatin (SHR-R, n = 16, 10mg kg−1 d−1) and untreated controls (SHR, n = 16). Age- and weight-matched WKY rats served as control (WKY, n = 16). The control rats were administrated equivalent distilled water. Systolic blood pressure (SBP) was determined by tail-cuff method before treatment, 4 and 8 wks after treatment. The wall-to-lumen area ratios (W/L), the ratios of wall thickness (WT) to lumen radius (LR) of 3rd grade branch mesenteric arteries were assessed morphometrically. Endothelium-dependent relaxation (EDdR), endothelium-independent relaxation (EDiR) were measured by PowerLab biological signal analytical system. Serum 25-Hydroxvitamin D2(25(OH)D2) was determined by ELISA.

RESULTS SBP in Rsv-treated rats was significantly lower than that in untreated SHR rats after 4, 8 wks of treatment. SBP in SHR-R at 4wks was 189.88 ± 18.90 mmHg, p < 0.05 vs SHR 200.72 ± 13.94 mmHg, 8wks: SHR-R 180.01 ± 14.56 vs SHR 200.27 ± 13.94 mmHg, p < 0.05 vs SHR. Compared with untreated SHR, W/L and WT/LR in SHR-R showed a descending tendency while exited no statistical difference after 4wks treatment (p > 0.05). Compared with untreated SHR, W/L [SHR-R 0.31 ± 0.21 vs SHR 1.82 ± 0.06, P < 0.01] and WT/LR [SHR-R 0.23 ± 0.04 vs SHR 0.33 ± 0.29, P < 0.01] of 3rd grade branch mesenteric arteries in Rsv-treated rats were markedly lower than that of untreated SHR after 8wks treatment. WT/LR in Rsv-treated rats was similar to the level as that of WKY (p > 0.05). EDdR and EDiR of 3rd grade branch mesenteric arteries were improved in SHR. After 8 wks treatment, EDdR of 3rd grade branch mesenteric arteries was increased [Emax/Emax%: SHR 29.10 ± 7.35; 47.41 ± 10.74, WKY 88.85 ± 5.17; P < 0.001, SHR 6.39 ± 0.90, WKY 8.34 ± 0.21, both P < 0.01]. EDiR was also enhanced [Emax/Emax%: SHR 46.13 ± 11.45, SHR-R 75.23 ± 20.10, WKY 96.28 ± 1.68, P < 0.001]. EDiR of 3rd grade branch mesenteric arteries was inhibited by Rosuvastatin (p < 0.01). After 8 wks treatment, its continuing enhancement was not statistically significant for EDdR and EDiR. Compared with SHR, serum 25(OH)D is higher after 8 wks of R treated. [SHR-R 5.93 ± 1.70 vs SHR 3.50 ± 0.91 ng/ml, P < 0.01], but didn’t reach the level of that in WKY (15.23 ± 1.55 ng/ml).

CONCLUSIONS The treatment of Rosuvastatin may mildly lower blood pressure, increase the plasma level of 25(OH)D, attenuate remodeling of 3rd grade branch mesenteric arteries, and ameliorate vasodilatation function in SHRs. The increase of vasodilatation function was prior to improvement of vascular function.

GW26-e2113 Polymorphisms in the CELSR2-PSRC1-SORT1 are associated with serum lipid traits, the risk of coronary artery disease and ischemic stroke
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OBJECTIVES Recent genome-wide association studies (GWAS) have identified CELSR2-PSRC1-SORT1 variants on chromosome 1p13.1 associated with CAD and plasma lipoproteins based on populations of European, eastern Asia, southern Asia, Middle-Eastern Asia, and African populations. Some loci associations were consistent across different populations. However, there were no data in the southern Chinese populations. Our study was to assess the association of CELSR2-PSRC1-SORT1