of Luofengning-0 complexes on the growth of human coronary artery smooth muscle cells (HCASMC) and endothelial cells (HCAEC) cultivated in vitro.

METHODS The 3-5 generation of HCASMC and HCAEC were respectively seeded on 96-well plates, then co-incubated with different concentration of hirudin (0.25, 0.5, 1, 2.5, 5, 10 mg/mL), methyelhistamotetrazolium (MTT) assay for cell viability, and then determined the optimal concentration range of hirudin to inhibit the growth of HCAEC, while promote the growth of HCAEC. On this basis, choosing hirudin concentrations as optimal different ratio of Luofengning-0 complexes. Co-incubated with different ratio of Luofengning-0 complexes in 96-well culture plate, every doses were added into 6 wells. The first 5 wells added culture medium for blank contrast group and the remaining 1 well for zero-adjustment, the second 6 wells only added single paclitaxel. After co-incubated 48 hours, detected the change of cells growth activity by MTT assay and observed the state of cells, then determined the appropriate ratio of paclitaxel and hirudin to maximize the inhibition of HCAEC and minimum the inhibition of HCAEC.

RESULTS Medium dose of hirudin (0.2-3.13mg/mL) could obviously inhibited the growth of HCAEC(P<0.05) while not apparently the growth of HCAEC when hirudin concentration was equal to blank contrast group. This range of hirudin complexes with tumol/L paclitaxel(Luofengning-0 complexes) could obviously inhibited the growth of HCAEC(P<0.05). Besides, low dose of LFN-0 complexes tumol/L paclitaxel(0.39mg/mL hirudin) group were apparently increased the growth of HCAEC(P<0.05), and decreased the inhibition ratio of the growth of HCAEC compared with single paclitaxel group(P<0.05).

CONCLUSIONS For low dose of LFN-0 complexes can maximize the inhibition of HCAEC and minimum the inhibition of HCAEC, we choose tumol/L paclitaxel matches 0.39mg/mL hirudin as our final ratio of LFN-0 complexes. All these proved the rationality of TCM compatibility to reduce the poison theory and provided an objective basis for the new theory of “endogenous collaterals wind".

GW26-e1239 Ethanol promoted the development of atherogenic-diet-induced atherosclerosis in murine abdominal aortas
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OBJECTIVES The mechanism through which alcohol promotes the development of atherosclerosis remains elusive. The first detectable stage of atherosclerosis is the fatty streak of the arterial wall, which can be noninvasively evaluated by measuring intima media thickness (IMT) using the Ultrasound. Monocyte chemotactic protein-1 (MCP-1) plays an important role in the recruitment and activation of monocytes and thus in the development of atherosclerosis. The aim of this study was to evaluate the effects of chronic high ethanol consumption (RTOH) and/or atherogenic diet (AD) on the development of atherosclerosis via up-regulating MCP-1 in murine abdominal aorta (AoA).

METHODS C57BL/6j male mice (13 weeks of age) were fed by control diet (CD), AD, and AD + 10 g/dL ethanol (AD + ETOH) for 16 weeks. AoA IMT, the gene expression of MCP-1 in the murine abdominal aorta, and total cholesterol level in blood were assessed.

RESULTS Sixteen weeks AD treatment increased the AoA IMT and the total blood cholesterol level. While, 16 weeks AD + ETOH treatment deteriorated the increases in AoA IMT and blood total cholesterol level accompanied the up-regulation of the MCP-1 gene expressions, which were not shown in the AD group mice.

CONCLUSIONS Chronic high ethanol consumption may promote the development of the AD-induced atherosclerosis via up-regulated MCP-1.

GW26-e2434 Protection of XUEBIJING in CLP-induced Pulmonary Function Injury in rats by Inactivation of NFkB
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OBJECTIVES We aimed to investigate the effect of XUEBIJING on lung function and to search for possible mechanism in septic rats.

METHODS Fifty male Sprague-Dawley (SD) rats were randomized into Sham-operation group (Sham group, n=10), saline group (NS group, n=20) and XUEBIJING group (XBJ group, n=20). Polymicrobial sepsis model was induced by cecal ligation and puncture (CLP) in NS and XBJ group. Rats in NS group were injected with saline (10ml/Kg) 2h after CLP and then twice a day for 7 days. Rats in XBJ group were injected XBJ (4mg/Kg) instead of saline at the same time points. Dad time of rats was recorded every day and lung function was tested by Buxco pulmonary analysis computer/software. Weight changes, acute pulmonary edema (lung wet-to-dry ratio) and lung histopathology were measured to evaluate their efficacy. Serum levels of TNF-a, IL-6 and IL-18 were measured by ELISA. Meanwhile, the mRNA expression of IKKz and NF-κB p65 were measured by RT-PCR. The protein expression of IKKz, phospho-IKKz, NF-κB p65 and phospho-NF-κB p65 were measured by western blot.

RESULTS On day 7 after CLP, 4 out of 20 XBJ treated rats survived, but all rats in NS group died. Compared with the NS group, XBJ group could obviously increase Dynamic lung compliance (Cdyn) and decrease pulmonary resistance (Rl) (P<0.05). The acute lung edema calculated by wet-to-dry ratio was eased compared with that in model group (P<0.05). Correspondingly, the expression of TNF-a, IL-6 and IL-18 in lung in XBJ treated rats were obviously reduced (P<0.05). Also, histomorphology indicated that XBJ significantly reduced the pathological injuries of lung (P<0.05). Compared with CLP group, XBJ reduced expression of IKKz and NF-κB p65 mRNA (P<0.05). Moreover, the protein expression of IKKz, phospho-IKKz, NF-κB p65 and phospho-NF-κB p65 in XBJ treated rats was obviously decreased (P<0.05).

CONCLUSIONS XBJ could effectively extend the survival time, significantly assuage the lung function and decrease the expression of inflammation related protein in septic rats induced by CLP. The protection of XBJ is induces more likely by means of IKKz and NF-κB p65 inhibition.

GW26-e2939 Research on pharmacological mechanisms of Qishen Granule using Methodology 1H-NMR Metabolomics in mini pigs with cardiac functional insufficiency and qi-deficiency and blood stasis syndrome (QDBS) induced by Ameorid constricting ring
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OBJECTIVES To explore the pharmacological mechanisms of Qishen Granule, a compound Chinese herbal medicine, using modern technique 1H-NMR metabolomics in a mini pig model of cardiac functional insufficiency and qi-deficiency and blood stasis syndrome (QDBS) induced by Ameorid constricting ring. Moreover, drug targets and effect mechanisms pathways which may exist were also studied.

METHODS Establishment and assessment of a qi-deficiency and blood stasis syndrome model in mini pigs with cardiac functional insufficiency experi- mental mini pigs (20±5 kg) were instrumented with a size-matched Ameorid constrictor on the anterior descending branch under general anesthesia in sterile conditions. Sham group didn't place the Ameorid constrictor, the other were the same to the model group. And the total 40 piglets of animal models were randomly divided into 4 groups (P<0.05). Also, histomorphology indicated that XBJ significantly reduced the pathological injuries of lung (P<0.05). Compared with CLP group, XBJ reduced expression of IKKz and NF-κB p65 mRNA (P<0.05). Moreover, the protein expression of IKKz, phospho-IKKz, NF-κB p65 and phospho-NF-κB p65 in XBJ treated rats was obviously decreased (P<0.05).

RESULTS Model group, treatment group and sham group using ‘H-NMR metabolomics found total 25 differences metabolism material, including histidine, acacetacetate, acetate, ethanol, valine, aspartate, glycine, proline, citrate, urea, histidine, leucine, isoleucine, myo-inositol, succinate, glycero, dimethylglycine, dimethylamine, methionine, choline, phenylalanine, b-Hydroxybutyrate, b-Glucose, 1-Methyhistidine. After 8 weeks administration, compared