METHODS HUVECs were treated with different concentrations of CdCl2 (0, 20, 40, and 80 μM) for 24 h. Cell viability was detected using Cell Counting Kit-8 (CCK-8). Pyroptosis in HUVECs was determined by LDH and Casp1 activation/STYX Green double staining. To determine the role of NLRP3 inflammatory cascade on cadmium (CdCl2) induced pyroptosis, the siRNA against NLRP3 transfections experiments was performed. The activity of caspase-1 was measured by the caspase-1 Activity Assay Kit. Expressions of caspase-1 and IL-1β were detected by western blot.

RESULTS Cell viability was significantly decreased after treated with CdCl2 in a concentration-dependent manner (control: 100 ± 4.11%, 20 μM: 99.21 ± 4.43%, 40 μM: 65.26 ± 9.75% and 80 μM: 25.22 ± 0.50% respectively). The cell death induced by CdCl2 appeared to involve pyroptosis based on our results from the increased of LDH release (1.97 ± 0.06 fold of change to control) and active Casp1/STYX double-positive (10.67 ± 0.71 fold of change to control) (Pyroptosis was defined by cytochrome c release and both active Casp1 and STYX positivity). As the makers of NLRP3 inflammatory cascade activation, the expression of caspase-1 (3.17 ± 0.19 fold of change to control) and IL-1β (1.60 ± 0.16 fold of change to control) are significantly up-regulated after incubated with CdCl2. Moreover, transfection of NLRP3 siRNA was highly efficient in inactivating the expression of caspase-1 and IL-1β, LDH release and active Casp1/STYX double-positive cells (P < 0.05).

CONCLUSIONS These results indicate that cadmium (CdCl2) could induce vascular endothelial cell pyroptosis and the NLRP3 inflammatory cascade activation is associated with HUVECs pyroptosis induced by cadmium.

GW26-e2934
The effect of Huoxue Qianyang recipe on the myocardial endoplasmic reticulum stress signaling pathway of obesity hypertensive rats
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OBJECTIVES To investigate whether and how the Ang II induced endoplasmic reticulum stress (ERS) and then the cardiomyocyte apoptosis involves in myocardial remodelling of obesity hypertensive rat models, then to investigate the huoxue qianyang(HXQY) recipe’s effects on these changes.

METHODS 72 five-week-old spontaneously hypertensive male rats (SHR) were selected as objects, and randomly divided into two groups according to the random number table(per ration of 2:8), that was control diet group (C group) and high fat diet group; 18 age-matched WKY rats were selected as the normal control group (W group). High fat diet group were fed 10 weeks, then 27 SHR were selected as obesity prone rats, while others were obesity resistant rats; obesity prone rats were randomly divided into three groups: obesity model group (M group), obesity model treatment group (MC group) and obesity control group (C group). The blood pressure of tail artery of 9 15-week-old C group, M group and W group rats were recorded, the blood and heart samples were obtained. The rest rats of C group, W group, MC group were gavaged with 15ml Kg-1 d-1 HXQY recipe. The blood pressure of tail artery were recorded twice a week. 8 weeks later, the blood and heart samples were obtained. All blood samples’ fasting glucose, insulin, lipids and angiotensin II were tested. These hearts’ Ang II levels were tested and myocardial cell apoptosis were tested with tunnel kit. Then the makers in myocardial cascade signaling pathway were tested via RT-PCR and Western Blot method.

RESULTS Before the treatment, the C, M groups’ HOMA-IR index, Ang II and lipids were higher than the W group (p<0.01), and the M groups were the highest(p<0.05). After treatment, MC Group’s those indicators increased (p<0.01), but MZ groups didn’t change significantly (P>0.05). We found no significantly difference between M and C groups’ blood pressure (P>0.05), but both of these two groups’ blood pressure higher than the W group (P<0.01). Rats treated with HXQY recipe experienced a reduction in blood pressure. Myocardiac Ang II levels in MC group was much higher than the C and W group of the same age (P<0.05), and HXQY recipe lowered the level of Ang II significantly(P<0.05). We found that cardiomyocyte apoptosis had enhanced in the MC group, much higher than the M, C, W groups (P<0.05). And HXQY recipe inhibited the progress of this(P<0.05). Apart from that, W group had the lowest GRP78, CHOP and Caspase12 mRNA and GRP78, CHOP and Caspase12 protein expression (P<0.01), however MC group had the highest ones (P<0.01). These genes’ mRNA and protein level notable decreased after HXQY recipe’treatment (P<0.01).

CONCLUSIONS We propose that obesity hypertension regulate structural remodeling of the heart. HXQY recipe can improve metabolic disturbance, lower the blood pressure and prevent the progression of obesity hypertensive heart remodeling by depression of the Ang II induced ERS and cardiac apoptosis.

GW26-e4468
Effect of Aconine on the expression of Caveolin-1 and eNOS in EAhby926 cell injured by Homocysteine
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OBJECTIVES To detect the effect of aconine on the expression of Caveolin-1 and eNOS in EAhby926 cell injured by homocysteine.

METHODS Model of EAhby926 cell injured by homocysteine was made, the protection on the EAhby926 cell of aconine with different dosages and different durations were observed. The effect of aconine on the expression of protein of Caveolin-1 and eNOS in EAhby926 cell were observed by Western-blot, and effect of aconine on the expression of mRNA of Caveolin-1 and eNOS in EAhby926 cell were observed by fluorescent quantitation PCR.

RESULTS After Model of EAhby926 cell injured by homocysteine was made, we found that cultured with 0.5, 1.0, 2.0, 4.0, 8.0 μmol/L homocysteine, cells grew less than cultured with normal culture medium, with the increase of homocysteine concentration, the number of attached cell grew downwards obviously, as culturing with homocysteine 4.0 μmol/L for 24h did lower damage to cells and could induce effective cell injury, it was made to be the model of injury. To detect the effect of aconine on EAhby926 cell injured by homocysteine, well growing EAhby926 cells were cultured in culture plate. 24h later, cells were cultured with DMEM medium containing 2% fetal calf serum for 8 hours to make cells hungry, then cultured with medium containing aconine 0, 0.05, 0.10, 0.20 mg/ml respectively for 30 minutes, then cultured with normal culture medium for 18 hours. To detect the effect of aconine on EAhby926 cell injured by homocysteine, well growing EAhby926 cells were cultured in culture plate. 24h later, cells were cultured with DMEM medium containing 2% fetal calf serum for 8 hours to make cells hungry, then cultured with medium containing aconine 0, 0.05, 0.10, 0.20 mg/ml respectively for 30 minutes, then cultured with normal culture medium for 18 hours. To detect the effect of aconine on EAhby926 cell injured by homocysteine, well growing EAhby926 cells were cultured in culture plate. 24h later, cells were cultured with DMEM medium containing 2% fetal calf serum for 8 hours to make cells hungry, then cultured with medium containing aconine 0, 0.05, 0.10, 0.20 mg/ml respectively for 30 minutes, then cultured with normal culture medium for 18 hours. To detect the effect of aconine on EAhby926 cell injured by homocysteine, well growing EAhby926 cells were cultured in culture plate. 24h later, cells were cultured with DMEM medium containing 2% fetal calf serum for 8 hours to make cells hungry, then cultured with medium containing aconine 0, 0.05, 0.10, 0.20 mg/ml respectively for 30 minutes, then cultured with normal culture medium for 18 hours. To detect the effect of aconine on EAhby926 cell injured by homocysteine, well growing EAhby926 cells were cultured in culture plate. 24h later, cells were cultured with DMEM medium containing 2% fetal calf serum for 8 hours to make cells hungry, then cultured with medium containing aconine 0, 0.05, 0.10, 0.20 mg/ml respectively for 30 minutes, then cultured with normal culture medium for 18 hours. To detect the effect of aconine on EAhby926 cell injured by homocysteine, well growing EAhby926 cells were cultured in culture plate. 24h later, cells were cultured with DMEM medium containing 2% fetal calf serum for 8 hours to make cells hungry, then cultured with medium containing aconine 0, 0.05, 0.10, 0.20 mg/ml respectively for 30 minutes, then cultured with normal culture medium for 18 hours. To detect the effect of aconine on EAhby926 cell injured by homocysteine, well growing EAhby926 cells were cultured in culture plate. 24h later, cells were cultured with DMEM medium containing 2% fetal calf serum for 8 hours to make cells hungry, then cultured with medium containing aconine 0, 0.05, 0.10, 0.20 mg/ml respectively for 30 minutes, then cultured with normal culture medium for 18 hours.

CONCLUSIONS Homocysteine may injure EAhby926 cell by enhancing the expression of caveolin-1 then suppressing the expression of eNOS, while aconine may protect EAhby926 cell by suppressing the expression of caveolin-1 then enhancing the expression of eNOS.