METHODS The case-control study including a Han population (410 EH patients and 410 control subjects) and a Uygur population (371 EH patients and 463 control subjects). Individuals was conducted to identify the association of three SNPs in CYP19 with EH by using χ2 test or Fisher exact test. Differences in lipids and the parameters of echocardiography among individuals with different genotypes were assessed by using one way analysis of variance(ANOVA).

RESULTS For women in Han, the distribution of rs2289105 in CYP19 gene showed a significant difference between EH and controls(P=0.049) and the dominant model (CC vs CT+TT) has a significant lower risk than the homozygous wild-type CC(p=0.014), the dominant model of rs12050772 (GG vs GT+TT) has a significant lower risk in EH patients(p<0.001). For men in Uygur, the recessive model of rs4774585 (AA vs AG+GG) has a significant higher risk in EH patients (p=0.021). ANOVA indicated the left ventricular end-diastolic dimension is significant higher in the homozygous wild-type (GG) groups compared with other groups.

CONCLUSIONS The T allele of rs2289105 in CYP19 gene might be a protective genetic marker of EH for women in Han population. The T allele of rs12050772 in Han population and the A allele of rs4774585 in Uygur population could be a protective genetic marker, but further study is needed.

GW26-e2201
Recombinant adeno-associated virus serotype 9 transfection of atherosclerosis mice: determination of the optimal expression time in vivo
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OBJECTIVES To explore the optimal time point of recombinant adeno-associated virus serotype 9-enhanced green fluorescent protein (rAAV9-eGFP) expression in the aorta of atherosclerosis mice.

METHODS Atherosclerosis model was established with high-fat diet (HFD) for 16 weeks. Among them, 25 mice were injected with 1011 vg (virus genomes) rAAV9-eGFP through the tail vein, while the remaining 5 mice were injected with saline, serving as the control group. The virus-transfected mice were killed at 14, 21, 28, 35 and 60 days after transfection, and aortic tissue was harvested. The expression of enhanced green fluorescent protein was detected by using laser scanning confocal microscope. Western blot assays were used to detect the expression of enhanced green fluorescent protein in aorta. The expression of enhanced green fluorescent protein in vivo was observed and the optimal expression time point was determined.

RESULTS rAAV9-eGFP effectively transacted the aorta of atherosclerosis mice, enhanced green fluorescent protein was expressed in aortic tissue, and the expression intensity increased gradually with the increasing transfection time. The highest expression level was found at 35 days after transfection and then maintained stable at 60 days. There were significant differences at different time points after transfection (P<0.05).

CONCLUSIONS rAAV9-eGFP can be effectively expressed in the aorta of atherosclerosis mice and can be regarded as the optimal vector in the treatment of atherosclerosis.

GW26-e4649
The role of augmented late sodium current in atrial fibrillation
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OBJECTIVES To determine the role of increased late sodium current in atrial fibrillation (AF) by investigating the effect of a new anemone toxin-II (ATX-II) on atrial action potential duration (APD) and effective refractory period (ERP) and the incidence of AF.

METHODS Female New-Zealand rabbit hearts were isolated and perfused in Langendorff method. Hearts were paced at right atrial appendage at fixed rate and left atrial and ventricular endo- and epicardial APDs were recorded. Hearts were treated with ATX-II (3 nM) and paced at right atrium in a programmable mode of S1S2 to create AF. Ranolazine and TTX at different concentrations were administered to hearts with AF in order to observe their effectiveness on suppressing AF.

RESULTS When hearts were paced at 350 ms, ATX-II (3-15 nM) significantly prolonged atrial MAPD90 by 46 ± 5 ms (n=6, P<0.001). In the presence of ATX-II (10-15 nM), spontaneous AF were investigated in 64.3% (n=14) of hearts, but TTX (1 μM) and ranolazine (10 μM) terminated AF. In the presence of ATX-II, TTX (1 μM) and ranolazine (10 μM) significantly reduced AF window and AF burden in concentration dependent manners by 31 ± 11 s and 103 ± 8 ms (n=6, P<0.05), respectively. In the presence of ATX-II (3 nM), ranolazine (3-10 μM) and TTX (0.1-1 μM) significantly reduced AF window and AF burden in concentration dependent manners.

CONCLUSIONS When late sodium current was increased, AF was induced by prolonging atrial MAPD and ERP which was the new mechanism of AF. Both ranolazine and TTX shortened atrial MAPD and exerted the ability of reducing AF window and AF burden and preventing AF.