induced for calcification. Culture of smooth muscle cells (SMCs), which comprise only a small percentage of all cells in the adventitia, in calcifying medium resulted in calcification.

RESULTS Following 48 weeks, calcified lesions were observed in the aorta adventitia and coronary artery adventitia of ApoE-/- mice. Von Kossa staining showed calcification in the human aorta adventitia. Explant culture of fibroblasts, was successfully induced for calcification after incubation with TGF-β1 (20 ng/ml) + mineralization medium for 4 days, and the conversion of vascular adventitial fibroblasts into myofibroblasts was identified. Culture of smooth muscle cells (SMCs), which comprise only a small percentage of all cells in the adventitia, in calcifying medium for 14 days resulted in significant calcification.

CONCLUSIONS Vascular calcification can occur in the adventitia. The conversion of fibroblasts into myofibroblasts may contribute to the calcification process. Although SMCs comprise only a small percentage of the entire adventitia cell population, they may also contribute.

GW26-e1388 Transplantation of EPCs Overexpressing S1PR3 Promotes Vascular Repair in the Early Phase After Vascular Injury
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OBJECTIVES Endothelial progenitor cells (EPCs) play important roles in the process of reendothelialization and prevent neointimal formation after vascular injury. Sphingo sine-1-phosphate (SIP) can via the sphingosine-1-phosphate receptor 3 (S1PR3) induce pro-liferation, migration and angiogenesis of EPCs. This study aims to investigate the effects of transplantation of EPCs overexpressing S1PR3 on reendothelialization and neointimal formation in response to vascular injury in mice.

METHODS Spleen-derived EPCs were cultured and expanded in endothelial basal medium. EPCs were infected with lentivirus vectors expressing mouse S1PR3 (S1PR3- EPCs) or green fluorescent protein (GFP- EPCs). Three days after gene transfection, the mRNA level and protein expression of EPCs were detected and the cytokines concentration in the culture medium was determined. The proliferation of HUVECs and RASMC were investigated.

RESULTS At day 7, the reendothelialized area in the S1PR3- EPCs transplanted group was significantly larger (56.32 ± 6.33%) than that in the GFP- EPCs transplanted arteries (42.47 ± 4.69%, p < 0.05) and that in the saline control group (28.31 ± 7.32%, p < 0.01). At day 14, the reendothelialized area in the S1PR3- EPCs transplanted arteries (72.28 ± 8.37%) was significantly larger than that in the GFP- EPCs transplanted arteries (50.21 ± 5.66%, p < 0.01) and that in the saline control group (29.98 ± 7.58%, p < 0.01). At day 14, a significant decrease (P < 0.05) in neo-intimal/media (NI/M) ratio was noted in the S1PR3- EPCs group (0.31 ± 0.02) as compared with that in the GFP- EPCs group (0.45 ± 0.10) and in the saline control group (0.77 ± 0.21). Our results showed that EPCs overexpressing S1PR3 significantly accelerates reendothelialization and mitigates neointimal formation in the early phase after carotid artery injury in mice.

CONCLUSIONS Our data suggest that transplantation of EPCs overexpressing S1PR3 can have a combined effect of both amplifying the reendothelialization capacity of EPCs and inhibiting neointima formation so as to facilitate better inhibition of adverse remodeling after vascular injury. Therefore, gene modified EPCs may be applied in clinical progenitor cell therapy to improve vascular repair after vascular injury.

GW26-e1483 Aberrant Expression of Circular RNAs in Endothelial Dysfunction
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OBJECTIVES The aim of this study was to determine the expression profile of circular RNAs (circRNAs) in oxidized low-density lipoprotein (ox-LDL) treated human umbilical vein endothelial cells (HUVECs) compared with normal HUVECs through human circRNA microarray and predict their miRNA binding sites.

METHODS The global circRNAs expression profiles in ox-LDL treated HUVECs compared with normal HUVECs were measured by the Arraystar Human circRNA Array (8x15K, Arraystar). Agilent Feature Extraction software (version 11.0.1.1) was used to analyze acquired microarray images. The circRNA/microRNA interaction was predicted with Arraystar’s home-made miRNA target prediction software.

RESULTS Compared with normal HUVECs, 24 circRNAs were different expression (fold change >1.5, p-value cut-off is 0.05) in ox-LDL treated HUVECs. 15 circRNA were up-regulated and 9 circRNAs were down-regulated. The most up-regulated circRNA was circRNA-104137 and the most down-regulated circRNA was circRNA-100188. The miRNA binding sites of circRNA-104137 were miR-455-3p, miR-218-1-3p, miR-423-5p, miR-503-5p and miR-223-3p. The miRNA binding sites of circRNA-100188 were miR-637, miR-608, miR-328-3p, miR-877-3p and miR-189-3p.

CONCLUSIONS The aberrantly expression profile of circRNAs in ox-LDL treated HUVECs compared with normal HUVECs indicates the potential roles of circRNAs in endothelial dysfunction. This study may provide new insights into the mechanism and potential targets for the endothelial dysfunction.

GW26-e1596 Effects of atorvastatin on mRNA and protein expression of adropin in cultured human umbilical endothelial cells and rat artery smooth muscle cells
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OBJECTIVES Adropin is a newly-identified secretory protein that participates in the regulation of energy homeostasis and insulin response. Growing published evidence presented the beneficial association of adropin with coronary artery disease. A laboratory test showed the beneficial effects of adropin on endothelial cells proliferation and migration. The endothelial cell dysfunction and proliferation of vascular smooth muscle cell form the core mechanism of atherosclerosis. Therefore, to increase the expression of adropin by some therapy may play a role in the prevention and treatment of atherosclerosis. In the present study, the effects of atorvastatin on mRNA and protein expression of adropin in cultured human umbilical vein endothelial cells (HUVEC) and rat artery smooth muscle cell (RASMC) were investigated.

METHODS HUVEC and RASMC were cultured in vitro with atorvastatin of 0.002, 0.02, 0.2, 2 and 20 umol/L for 6, 12 and 24 hours (h). The proliferation of HUVEC and RASMC were detected by MTT chromatin of 0.002, 0.02, 0.2, 2 and 20 umol/L for 6, 12 and 24 hours (h). The proliferation of HUVEC was promoted after co-incubation with atorvastatin from 0.002 to 20 umol/L concentration, and the peak was at 20umol/L (versus control, P < 0.01). Atorvastatin upregulated the expression of adropin mRNA and adropin protein dose-dependently and time-dependently in HUVEC with a peak at 20umol/L (versus control, P < 0.01) and 24 h (versus 6 h, P < 0.01). Adropin protein concentration in culture medium was positively correlated with the OD value indicating proliferation at 12 h (P < 0.001) and 24 h (P < 0.001) after co-incubation.

The proliferation of RASMC was decreased after co-incubation with atorvastatin from 0.002 to 20umol/L concentration, and the peak was at 20umol/L (versus control, P < 0.05) and 24 h (versus 6 h, P < 0.05). Atorvastatin upregulated the expression of adropin mRNA and adropin protein dose-dependently and time-dependently in HUVEC with a peak at 20umol/L (versus control, P < 0.01) and 24 h (versus 6 h, P < 0.01). Adropin protein concentration in culture medium was positively correlated with the OD value indicating proliferation at 12 h (P < 0.001) and 24 h (P < 0.001) after co-incubation.

CONCLUSIONS Atorvastatin promoted the expression of adropin in HUVEC and RASMC at a appropriate range of concentration, with the corresponding results that proliferation of HUVEC was promoted but RASMC proliferation was inhibited in the test concentration range of atorvastatin.