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-β (20 ng/ml) + mineralization media for 4 days, and the phenotype 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. Sphingosine-1-phosphate receptor 3 (S1PR3) can modulate the sphingosine-1-phosphate receptor 3 (S1PR3) to induce proliferation, 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 S1PR3 were detected by real-time polymerase chain reaction and fluorescent microscopy. 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 S1PR3 were detected by real-time polymerase chain reaction and fluorescent microscopy. 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 S1PR3 were detected by real-time polymerase chain reaction and fluorescent microscopy.

**RESULTS** At day 7, the reendothelialized area in the S1PR3-EPCs group was significantly larger than that in the GFP-EPCs transplanted arteries (50.21±5.66% vs 28.31±5.66%). The S1PR3-EPCs group showed significantly larger reendothelialization capacity and inhibiting neointima formation after vascular injury. Therefore, gene modified EPCs may be applied in clinical progenitor cell therapy to improve vascular repair after vascular injury.

**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-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 cells provide new insights into the mechanism and potential targets for the endothelial dysfunction.

**METHODS** HUVECs and RASMC were cultured in vitro with atorvastatin 0.02, 0.02, 0.2, 2 and 20 μmol/L for 6, 12 and 24 hours (h). The proliferation of HUVEC was promoted after co-incubation with atorvasta tin at 0.002, 0.02, 0.2, 2 and 20 μmol/L; the peak was at 20μmol/L (versus control, P<0.01). Adropin protein concentration in culture supernatant was increased after co-incubation with atorvastatin at 0.002, 0.02, 0.2, 2 and 20 μmol/L; the peak was at 20μmol/L (versus control, P<0.01).

**RESULTS** The proliferation of HUVEC was promoted after co-incubation with atorvastatin from 0.002 to 20 μmol/L concentration, and the peak was at 20μmol/L (versus control, P<0.05) and 24 h (versus 6 h, P<0.01). Adropin mRNA and adropin protein dose-dependently and time-dependently increased in response to atorvastatin co-incubation. Adropin mRNA and adropin protein concentration in culture supernatant 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 proliferation of RASMC was inhibited in the test concentration range of atorvastatin.