trichrom staining 4 weeks after MI. Biochemical assays were conducted in the frozen ventricular tissues or in the cultured cardiomyocytes.

RESULTS SphK2 inhibition significantly increased mortality after MI (Vehicle 20.3% vs. ABC 50.1%). ABC administration obviously exacerbated cardiac dysfunction, interstitial fibrosis, and myocardial apoptosis following MI. SphK2 inhibition upregulated the expression of remodeling marker genes (ANP, BNP, and β-MHC) in the myocardium. Higher plasma BNP levels in ABC-treated mice indicated more severe heart failure progression. Compared with vehicle-treated mice, myocardial HDAC activities were elevated in ABC-treated group. In the cultured cardiomyocytes, β-adrenergic receptor agonist-induced apoptosis and remodeling gene expression were aggravated by SphK2 knockdown or ABC treatment in the cardiomyocytes, which was blocked by HDAC inhibitors.

CONCLUSIONS SphK2 and its product S1P exert protective roles in the remodeling processes and heart failure progression after MI potentially by inhibiting myocardial HDAC activation.

GW26-e0103
CD51 Positive Cardiac Cell Repair the Heart After Myocardial Infarction Through Transdifferentiation
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OBJECTIVES Acute myocardial infarction (AMI) is one of the most serious diseases that threaten human’s health. AMI may lead to myocardial dysfunction, reconstruction and even heart failure. Many experiments demonstrated the possibility of cardiac stem cells (CSCs) to treat AMI. They found evidence for enhanced revascularization of the infarct zone in stem cells-transplanted hearts, and even differentiate into functional myocytes. A variety of surface markers have been used to define CSCs. Until now, we do not virtually know the unified and specific surface markers of CSCs. Recently, the integrin αv (CD51) are found to express on testicle and myocardial cells. They can pro-liferate and differentiate into cardiomyocytes and endothelial cells. The aim of this study was to investigate the effects of CD51 positive cardiac cells on left ventricular dysfunction or heart failure after AMI.

METHODS CD51 positive cardiac cells were isolated from 7days old C57BL/6 mice. The cells were labeled by transfected with red fluorescent protein(RFP) before transplant. Wild- type C57BL/6 mice underwent myocardial infarction by ligating the left anterior descending coronary artery with injection of saline (n=7), or RFP-CD51+ cardiac cells (n=7). Cardiac function was monitored using echocardiography after 1 week and 4 weeks. The hearts were harvested and frozen at 4w after cardiac arrest. Myocardial tissue sections were stained with immunofluorescence to find cells which glowing red fluorescent and alpha sarcomeric actin positive under fluorescence microscope.

RESULTS Transplantation of CD51 positive cardiac cells into AMI mouse model improved cardiac function and remodeling, as determined by echocardiography. Compare with the control group, there were higher left ventricular ejection fraction (35.93±12.09% VS 16.59±1.88%, p<0.001), shorter heart period (17.30±6.355 VS 21.51±0.81%, p<0.001) and smaller left ventricular end-diastolic volume (59.73±30.841 VS 118.15±41.371, p<0.05) in the experimental group by 1w. The cell treatment group remained significantly improved relative to saline control animals at 4w. The immunofluorescence detection of cell treatment group showed RFP-CD51+ cells are alpha sarcomeric actin positive which indicate the transplanted cells can survive in myocardial infarction regions and differentiate into mature cardiomyocytes.

CONCLUSIONS The intramyocardial delivery of CD51 positive cardiac cells after AMI can improve cardiac function, and attenuate remodeling at all time points. The in vivo differentiated CD51 positive cells expressed cardiac markers, as determined by immunohistochemistry.

GW26-e0202
Expression of Matrix Metalloproteinase-2 and Mitogen-Activated Protein Kinases in Ascending Aortic Aneurysms and Aortic Valves of Patients with Bicuspid or Tricuspid Aortic Valves: A Comparative Study
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OBJECTIVES The objective of this study was to compare the expression of matrix metalloproteinase-2 (MMP-2) and mitogen-activated protein kinases (MAPKs) pathway molecules in ascending thoracic aortic aneurysms (AAAAs) of patients with bicuspid and tricuspid aortic valves.

METHODS Ascending aortic aneurysmal wall specimens and valve samples were taken from tricuspid aortic valves (TAV) patients (n=20, 51.5±7.6 years) and tricuspid aortic valves(BAV)patients (n=20, 47.4±6.3 years) when surgery repair. Normal ascending aortic specimens (n=20) were obtained from 20 patients with coronary heart diseases (CHD) undergoing CABG without aortopathy. MMP-2 expression was examined by qPCR, Western blot in aortic and valve tissues. MAPKs were measured by Western blot in aortic and valvular specimens. Some aneurysmal walls were stained with HE and EVG.

RESULTS By histological examination all aortic aneurysm walls showed severe remodeling gene expression patients exhibited severe elastin fragmentation and markedly decreased elastin and much collagen deposition, compared with BAV aneurysms. Aortas from the BAV group displayed intact and regular arrayed elastic fibers in the intima and media and very few collagens. MMP-2 expression was greater in valves and aortic aneurysm tissues from AAAAs associated with bicuspid valves when compared with those from tricuspid valves, irrespective of the level of mRNA and protein (p<0.05). In the aneurysmal wall specimens, t-p38 MAPK was higher in TAV patients than that in BAV patients and CHD patients (p<0.05). The p-p38 level was also increased in TAV aneurysmal aortic walls compared with BAV group. However, there is no significant difference in t-p38 MAPK between BAV patients and CHD patients (p>0.05). In addition, t-p38 and p-p38 MAPK levels had also no significant differences between BAV and CHD patients. Additional, the statistical differences were found in p-p38 and t-p38 between these three groups. Compared with normal aortic group, p-JNK, t-JNK and p-JNK/t-JNK were increased in two aneurysmal groups associated with BAV and TAV (p<0.05). Additionally, the BAV patients have increased levels of t-p-S41/JNK and p-p41/JNK compared with TAV patients. In the valvular tissues, p-p38, t-p38 and p-p38/t-p38 MAPK were no significant differences between BAV and TAV patients. Additionally, t-ERK1/2 level was higher in TAV patient than in BAV patient while p-ERK1/2 and p-ERK1/2/t-ERK1/2 levels had no statistical differences between TAV and BAV patients. Compared with BAV patients, the levels of p-JNK, t-JNK and p-JNK/t-JNK were increased in TAV patients (p<0.05).

CONCLUSIONS The up-regulation of MMP-2 in aneurysms associated with BAV may partly elucidate the predilection to aneurysm formation in these patients compared with TAV. Furthermore, in the BAV associated with ascending aortic aneurysms, increased p-p41/JNK level was found, which may contribute to elucidate the elevated level of MMP-2 compared with TAV associated with ascending aortic aneurysms.

GW26-e0203
Effects of Nerve Growth Factor on Late Reperfusion after Myocardial Infarction
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OBJECTIVES Nerve growth factor (NGF) is one of the most important bioactive molecules in nervous system, which involved in neuronal survival, embryonic development, learning and memory, neurite outgrowth and synaptic plasticity, and regulation of axonal growth and survival. NGF plays a protective role in myocardial infarction and early reperfusion by reducing the myocardial cell apoptosis and improving ventricular remodeling. In the study, we investigated the role of nerve growth factor on late reperfusion by detecting cardiac structure and function with echocardiography.

METHODS Rats were randomly divided into four groups: Adenoviral vector group (Adv group, intramyocardial injection, adenoviral vector 10μl in total), NGF overexpression group (NGF group, constructing the adenovirus vector Ad-NGF containing nerve growth factor gene, 10μl Ad-NGF as previously described). Sham group and LR group (10μl normal saline as previously described). The models of late reperfusion were performed as previously described. Sham group and LR group (10μl normal saline as previously described, respectively). The models of late reperfusion (LR group, Adv group, NGF group) were established by ligating the anterior descending branch of coronary artery in anesthetized rats, then administrating the ligature after 4 hours, and starting to reperfusion, the Sham group underwent thoracotomy without coronary ligation. On the 3rd, 5th, 7th, 14th and 28th day after operation, 5 rats in each group were sacrificed and their hearts were harvested. The expression of NGF protein was examined with immunohistochemistry technology, cardiac structure and function were measured with echocardiography.