Objectives: The main aim of this study was to systematically evaluate the expression patterns of the Nestin in the developing or damaged adult heart issue, and probe into whether Nestin can be as a marker of cardiac stem cell.

Methods: Nestin expression was assessed in the embryonic 13.5 d and postnatal 1d, 7d, 1M, 3M old Nestin-GFP transgenic mouse heart tissue by fluorescence microscopy, real-time quantitative PCR and RT-PCR. Myocardial infarction model was established by ligation of left anterior descending coronary in adult Nestin-GFP mice and the Nestin expression was observed in the myocardium at 7d after injury. Then, the correlation between Nestin and other stem cell markers’ expression in mouse heart tissue were determined by immunofluorescent assay.

Results: In embryonic 13.5 d, the Nestin mainly expressed in the brain, spinal cord and the retina, and also can be observed in the heart tissue. After the mouse was born. Nestin expression is gradually reduced with growth, and that was also confirmed by the RT-PCR, Q-PCR analysis. Nestin-positive cells increased significantly in myocardial infarction compared to the normal tissue. Sca-1, -kit, Isl-1 and Nestin are widely expressed in heart tissue, which suggest that such cells play an important role in the growth and maintenance of the cardiogenesis and regeneration.

GW25-0610
A Novel Model of Intimal Hyperplasia in the Bama Miniature Pig
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Objectives: To develop a bama miniature pig intimal hyperplasia model in superficial femoral artery.

Methods: Following 1 month of a 3% cholesterol diet, 4 pigs underwent surgical femoral artery.

Results: In experimental group, the vessels were analyzed.

Conclusions: This novel intimal hyperplasia model may be a useful tool for evaluating drugs and therapeutic devices.

GW25-0741
Calreticulin is localized in the mitochondria of rat cardiomyocytes and affected by furazolidone
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Objectives: Calreticulin is a calcium-buffering protein which is predominately located in endoplasmic reticulum. We have previously shown calreticulin is also localized in the myocardial mitochondria and up-regulated in a rat model of furazolidone-induced dilated cardiomyopathy. The aim of this study was to determine whether calreticulin is localized in the mitochondria of rat cardiomyocytes and whether mitochondrial calreticulin is affected by furazolidone.

Methods: The mitochondrial preparations were isolated from primary cultured neonatal rat cardiomyocytes and purified by differential centrifugation. The immunoreactivities of calreticulin and markers for cytosol, nucleus, endoplasmic reticulum and plasma membrane were detected by western blot. The distribution of calreticulin to mitochondria was further confirmed by immuno-electron microscopy, flow cytometry and laser scanning confocal microscopy (double staining with MitoTracker Red and calreticulin).

Results: To study the content of mitochondrial calreticulin was affected by furazolidone, the rat cardiomyocytes were exposed to 100 μmol/L furazolidone for 48 h and then the mitochondrial calreticulin expression was analyzed using western blot.

Conclusions: In summary, the present results suggest that calreticulin is localized in the mitochondria of rat cardiomyocytes and such localization is affected by furazolidone.

GW25-0767
Bisphenol A can injure the heart via DNA damage
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Objectives: Bisphenol A (BPA) is a man-made high volume production chemical and human is widely-spread exposure to BPA. Previous studies have shown that the BPA exposure is associated with heart disease, but the mechanisms of BPA on the heart are still unclear. The purpose of this research is to investigate the relationship between the concentrations of BPA and severity of the lesions in the heart and analyze the molecular mechanism of BPA harmful effect.

Methods: Mice were subcutaneously injected with normal saline or 0.1, 1 and 10 mg/kg/day BPA for 1 month, and then were detected by Vevo 770 ultrasonic diagnostic apparatus, respective the heart was excised and the hearts were treated by PBS or 0.1, 1 and 10μM BPA. The protein of γH2AX and P52 were detected by western blot. The mRNA level and the protein level of P52 were tested by real-time PCR and western blot. The protein maps of the cardiomyocytes stimulated by PBS or BPA were measured by two-dimensional gel electrophoresis and the differential protein spots were identified by mass spectrometry.

Results: EF value and FS value were significantly decreased in 1 and 10mg/kg/day BPA groups compared with normal saline group, and BPA produced a dose-dependent reduction in EF and FS value. The expression of γH2AX and P52 were obviously increased with the concentration of BPA in a dose-dependent manner. Some differentially expressed proteins were determined to be the signal transduction associated proteins of DNA damage.

Conclusions: This study mainly reveals that BPA is harmful to the heart and cardiomyocytes. Its mechanism may be that BPA causes DNA damage in cardiac muscle cell.

GW25-0775
Hepatocyte Growth Factor Suppresses Hypoxia/Reoxygenation-induced XO Activation in Cardiac Microvascular Endothelial Cells
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Objectives: To detect the effect of hepatocyte growth factor (HGF) on xanthine oxidase (XO) under hypoxia/reoxygenation (H/R) conditions in rat cardiac microvascular endothelial cells (CMECs).

Methods: Primary cultured rat cardiac microvascular endothelia cells (CMECs) were exposed to 4h of hypoxia and followed by 1h of reoxygenation. Generation of ROS and cytosolic Ca2+ concentration was measured by flow cytometry after HGF treatment. Generation of ROS and cytosolic Ca2+ concentration was measured by flow cytometry and XDH mRNA was quantified by RT-PCR analysis.

Conclusions: The present results suggest that calreticulin is localized in the mitochondria of rat cardiomyocytes and such localization is affected by furazolidone.

GW25-0784
Knock-down of metallothionein exacerbates intermittent hypoxia induced oxidative and inflammatory injury in aorta
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Objectives: Obstructive sleep apnea (OSA) is an independent risk factor for cardiovascular diseases possibly via intermittent hypoxia (IH) - elicited oxidative stress and inflammation, while metallothionein (MT) has been recognized as an inducible anti-oxidant which may protect against damages of a variety of oxidant stimuli. The present study was to explore the effect of MT on IH-induced aortic pathogenic changes.

Methods: To mimic hypoxia/reoxygenation events that occur in adult OSA patients, mice were exposed to IH for up to 8 weeks. The IH parameters consisted of alternating cycles of 20.92% O2 /8% O2 /F2O2 (30 episodes per hour) with 20 seconds at the nadir F2O2 for 12 hours a day during the light phase. Markers of oxidative damages, inflammation, and vascular remodeling were observed by immunohistochemical staining and Western blotting. After 3 days, 1, 3 and 5 days after IH exposure, the mice were sacrificed. The mRNA and protein levels of metallothionein were measured by real-time PCR and western blot.

Conclusions: This novel intimal hyperplasia model may be a useful tool for evaluating drugs and therapeutic devices.