**Results:** HF rats had higher levels of corticosterone-releasing hormone (CRH), monoamine oxidase (NE), and glutamate (Glu). Lower levels of gamma-aminobutyric acid (GABA), and more positive fra-like activity in PVN when compared to Sham rats. HF rats also had higher level of NE, epinephrine (EPI) and IL-1β in plasma. Plasma infusion of L-NAME attenuated the decreases in GABA and the increases in NE and Glu in the heart of HF rats. Plasma infusions of IL-1β could further increase expression of CRH, NE, Glu, IL-1β and eNOS and decrease GABA expression. Treatment with IL-1β together with losartan could eliminate the effects of IL-1β.

**Conclusions:** Interaction between AT1-R and IL-1β in the PVN contributes to deterioration of heart failure.

**GW25-e1659**

**Nicotine induce mast cells degranulation to promoteate the atherogenesis and reduce the atherosclerotic plaque stability**

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**Objectives:** Nicotine has been identified to promote atherosclerosis. But the mechanism of nicotine induced atherogenesis has not been well elucidated. Mast cell plays an important role in high-fat diet induced atherogenesis. This study focus on the role of mast cell in nicotine induced atherogenesis and plaque instability.

**Methods:** Peritoneal administration of 100nm disodium cromoglicate (DSCG) was injected, mast cell degranulation and apoptosis were divided into 3 groups: high-fat diet, high-fat diet + nicotine, and high-fat diet + nicotine + DSCG (n = 15 each). After 12 weeks of treatments, atherosclerotic lesion size of the aortas were quantified. Toluidine blue and tryptase staining identified mast cell counts and distribution at the aorta. Immuno-staining of CD68, CD45 were used to evaluate the inflammatory filtration.SMA, Ki-67 and sinus red staining were used to study smooth muscle cell proliferation and collagen content in the lesion. In vitro, bone marrow-derived mast cells (BMMCs) were harvested and divided into 5 groups, with PBS, a negative control, compound 48/80 as a positive control, 100μg/ml nicotine, 100μg/ml nicotine with 100nm DSCG pretreatment and nicotine 100μg/ml with 10μg/ml mecamylamine pretreatment. At 0.5hr, 1hr, 2hrs, supernatants were harvested to analyze the mast cell degranulation. Furthermore, conditioned medium were also used to induce the macrophage migration and foam cell formation.

**Results:** Nicotine increases plaque size, and macrophage infiltration, decreases smooth muscle collagen content along with the increases in mast cells count and activation ratio at the lesion, which could be inhibited by DSCG. Nicotine induced mast cell degranulation at 2 hours comparing to PBS (43.60% vs 2.36%), which could be inhibited by mast cell stabilizer DSCG (23.78%) and nACHR blocker mecamylamine (20.35%). Macrophage migration ability in the compound 48/80 and nicotine condition medium group were significantly higher comparing to PBS, DSCG and mecamylamine group. Foam cell formation ratio in the compound 48/80 and nicotine condition medium group were significantly higher comparing to PBS, DSCG and mecamylamine group.

**Conclusions:** Nicotine might induce mast cell degranulation through nACHR and then activate mast cell to release a range of proinflammatory mediators to increase the neointimal hyperplasia and as well as late and function of endometrium stem cells (EnSCs) in the atherosclerotic plaque induced by the administration of nicotine. Administration of mast cell stabilizer revealed the potential of applying mast cell stabilizer in preventing nicotine induced atherogenesis.

**GW25-e1582**

**Impact of SOD mimetic tempol and exercise training on NOS in spontaneously hypertensive rats**

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**Objectives:** The exercise training (Ex) and superoxide dismutase (SOD) mimetic tempol have antihypertensive effects in spontaneously hypertensive rats (SHR). However, the effects of the combination of Ex and tempol on NOS expression in SHR remain to be elucidated. To clarify the mechanism of antihypertensive and renal-protective effects of tempol, we investigated the influence of tempol on NOS expression and NO production in SHR. H2O2 may mediate these effects of the Ex and tempol in SHR.

**GW25-e1654**

**Comparison of Transplantation of bone marrow-derived stem cells, adipose-derived stem cells and endometrium- derived stem cells in the Infarcted Heart**

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**Objectives:** A variety of adult stem cells have been used to transplant into the infarcted heart to cure myocardial infarction (MI), however, compared studies are lacking to show more suitable source of cells for transplantation. Mesenchymal stem cells hold promise for myocardial regeneration therapy. Derivation of these cells from the endometrium tissue might be easier compared to bone marrow and adipose tissue. However, the effects of the combination with Ex and tempol on NOS expression in SHR remain to be elucidated. To clarify the mechanism of antihypertensive and renal-protective effects through the upregulation of NOS expression and NO production in SHR. H2O2 may mediate these effects of the Ex and tempol in SHR.

**GW25-e3339**

**Exhaustive Swimming Induces Cardiac Lesion in Rats**

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**Objectives:** The purpose of present study was to investigate the heart injury caused by exhaustive swimming in detail in rats.

**Methods:** Adult male Sprague-Dawley rats randomly were divided into two groups: the control group and acute exhaustive group. The acute exhaustive group rats were exposed to exhaustive swimming. Referring to Thomas exhaustive standards, single bout of exhaustive swimming were trained to exhaustion. The control animals lived in the same environment as the exhaustive animals with free access to food and water excepting exhaustive swimming in detail in rats.

**Results:** The content in serum of myocardial damage markers, troponin I (TnI) troponin T (TnT) and Brain natriuretic peptide (BNP) were markedly decreased (P<0.05) compared with the levels of E, NE in the control group (137.80±19.26 pg/mL, 217.37±37.84 pg/mL, respectively, P<0.05). The left ventricular end-systolic pressure (LVESP) obviously decreased (P<0.05) with decrease GABA expression. Treatment with IL-1β significantly increased Ccr (by 81% and 37%) in SHR. Ex and tempol also upregulated the eNOS and nNOS expressions in the kidney cortex (eNOS:25% and 31%, nNOS:24% and 35%), the outer medulla (eNOS:24% and 40%, nNOS:23% and 33%), the inner medulla (eNOS:21% and 43%, nNOS:22% and 25%) and thoracic aorta (eNOS:20% and 37%, nNOS:18% and 32%) of SHR with the increased plasma and urinary H2O2 (plasma: by 22% and 23%; urinary: by 26% and 22%) and NOX (plasma: by 14% and 14%; urinary: by 23% and 24%) significantly. Furthermore, the effects of the combination therapy with Ex and tempol on these factors were cumulate in SHR.

**Conclusions:** These results indicate that tempol enhances the Ex-induced anti-hypertensive and renal-protective effects through the upregulation of NOS expression and NO production in SHR. H2O2 may mediate these effects of the Ex and tempol in SHR.