The increasing of left atrial ANP concentration was promoted by Ang-(1-7)- A-779, Ang II, and NME treatment. The blockage of the effects of Ang-(1-7) on electrophysiological parameters and ANP secretion. 

**Conclusions:** Ang-(1-7) prevented the acute electrical remodeling in rapid atrial pacing canine model via Ang-(1-7)/Mas/PEIki/Akt/NO signaling pathway. Furthermore, ANP was related to the anti-arrhythmic effects of Ang-(1-7) and may play a potential role in Ang-(1-7)/Mas/PEIki/Akt/NO signaling pathway.

**GW25e2152**

**Serum chemerin levels and risk of coronary atherosclerosis in early-onset coronary artery disease of Chinese population**

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**Objectives:** Increasing evidence indicates that adipokines may play an important role in the pathogenesis of coronary artery disease (CAD). However, it remains uncertain whether chemerin, a novel adipokine, is associated with coronary atherosclerosis and could predict for CAD. In the present study, we sought to determine: 1) the relationship between chemerin and the severity of coronary atherosclerosis, and 2) whether chemerin is an independent risk factor for CAD in Chinese population.

**Methods:** 382 early-onset CAD (EOCAD) patients and 305 matched controls undergoing coronary angiography were included into this study. Their serum levels of chemerin were measured by enzyme-linked immunosorbent assay (ELISA). The severity of coronary atherosclerosis was evaluated using the scaled score.

**Results:** Serum chemerin levels of EOCAD patients were significantly higher than that of control subjects (105.03±14.62 ng/ml vs. 85.16±20.37 ng/ml; P<0.001). Chemerin had a significant positive association with body mass index, LDL-c, triglyceride, and high-sensitivity CRP. Serum chemerin level was positively correlated with Gemisini score (r=-0.384, P<0.001) and negatively associated with adiponectin (r=-0.426, P<0.001). In multiple binary logistic regression analysis, chemerin was an independent risk factor for CAD (OR=1.218, 95% CI 1.87-1.340, P<0.05) after adjusting for conventional cardiovascular risk factors. ROC analysis indicated that chemerin had greater area than adiponectin with no significant difference (P=0.05).

**Conclusions:** Chemerin is negatively associated with the severity of coronary atherosclerosis, as well as an independent risk factor for CAD. It may be a promising target for further elucidating the role of metabolic factors in pathogenesis of CAD.

**GW25e3450**

**The Possibility of Rapid Assessment of Cardiac Contractility Using Ballistocardiography**

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**Objectives:** Myocardial contractility can describe the function of the heart and is an effective indicator to evaluate whether the heart is health or not. The most effective way to measure the contractility is using the catheter. There also exists a noninvasive method to evaluate the contractility. The ballistocardiogram (BCG) records the gravity variation caused by the reaction force during the ejection of blood in heart which highly represents the mechanical activity of heart. In this paper, we proved the hypothesis that the time interval between the J wave of BCG and the R wave of electrocardiogram (ECG) which is referred as RJ interval is correlated to PEP.

**Methods:** Four healthy male subjects and one male subject with premature ventricular beats participated in our study. The Doppler echocardiogram (ECHO), ECG and BCG were recorded synchronously. The PEP is the time interval between the Q wave of ECG and the beginning of ventricular depolarization. The RJ interval is the time interval between the J wave of BCG and the R wave of ECG. 

**Results:** In our study, across all the five subjects, 1974 beats were correlated. The correlation coefficient of PEP and RJ intervals was 0.79. As for the best linear fitting, the slope was 1.12, and the intercept was 99.56. The correlation coefficient r^2 between PEP and the corrected RJ interval was improved to 0.88. It is summarized the correlation coefficient r^2 of original data and corrected data. In consideration of all the RJ intervals and PEP intervals, the minimum of correlation coefficient was 0.68 and the maximum was 0.99, except the subject with premature ventricular beats. After correction, the minimum became 0.88, and the maximum became 0.99. As for the subject who had premature ventricular beat, the correlation coefficient of RJ interval and PEP was 0.63, which was much lower than the average level of other normal subjects. But after RJ interval correction, it was improved to 0.88.

**Conclusions:** The results support the hypothesis that the PEP is correlated to the RJ. Consequently, to some extent-at least for healthy people, the corrected RJ intervals can be suggested as a better indicator for assessment of myocardial contractility. However, the validity of this correction for those whose cardiac function is abnormal should be validated in the future work. In our study, BCG, obviously the corrected RJ interval, can be suggested as an effective method for assessment of myocardial contractility. Moreover, the BCG measurement system allows family health care and long-term monitoring of changes in cardiac contractility.

**GW25e3455**

**Prenatal nicotine exposure induces gender-related left ventricular arterial uncoupling in adult offspring**

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**Objectives:** Changes in vascular and myocardial structure and function have been demonstrated in prenatal nicotine exposure offspring, but limited data are available on how the changes interact in adulthood. We studied testis tissue sections from Ang-(1-7) and chemerin function. Due to Ang-(1-7) and chemerin exposure induces gender-specific alternations of left ventricular-arterial coupling indices in adult offspring.

**Methods:** Female Sprague-Dawley rats were exposed to either nicotine (8 mg/kg/day) or saline via subcutaneous osmotic mini-pumps throughout the gestation. After 8 months, noninvasive echocardiography and invasive left ventricular cannulation were performed. Left ventricular arterial coupling was assessed as the ratio of ventricular end-systolic elastance (Ees) to effective arterial elastance (Ea). Left ventricular myocardium and aorta were stained with Hematoxylin and eosin and myocardial cell cross-sectional area were calculated. Anatomically, average wall thickness and the ratio of medium thickness to internal diameter were determined. Fibrosis component of left ventricle myocardium was analysed by Sirius-red staining and further confirmed by hydroxyproline determination. While the elastic properties of the aortic wall were analysed by van Gieson staining.

**Results:** PNE caused significant increases in pulse pressure (56.7±50.4; P<0.05) and left ventricular meridional wall stress (78.25±9.12 vs. 69.64±7.58; P<0.05) in male offspring with respect to control. Conversely, no identical effect was found in female offspring. Elevated augmentation in left ventricular Ea/Ees was marked in male offspring, whereas it was attenuated in females, compared to their respective control. Simultaneously, increased degeneration and fragmentation of elastic network was noted, as was the ratio of medial thickness to internal diameter in mesenteric artery.

**Conclusions:** PNE caused combined ventricular-arterial mismatching in both male and female offspring, with lower Ea/Ees in male while preserved Ea/Ees in female. Enhanced collagen cross-linking in myocardium, underdeveloped elastic fibers in aorta, and remodeled resistance vessels were associated to the pathological ventricular arterial mismatching. These effects present a gender dichotomy with males being more susceptible than females.

**GW25e3558**

**Prevention of Angiotensin II-induced Cardiomyopathy by Sulforaphane-activated Nrf2 Partially Via AKT/GSK-3β/Fyn Pathway**

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**Objectives:** Ang II (Ang II) is an important causative of diabetic cardiomyopathy. In the diabetes, hyperglycemia induces the generation of Ang II in the heart. Mechanistically the pathogenesis of cardiac Ang II in the development of diabetic cardiomyopathy was considered its induction of nitrative and oxidative stress via Ang II interaction with its receptors and its mitogenic effects on Nrf2-regulated growth factors of superoxide and peroxynitrite. Nuclear factor-related factor 2 (Nrf2) is a master regulator of cellular detoxification responses and redox status. Sulforaphane (SFN) is anti-oxidative supplement via up-regulating Nrf2-mediated multiple endogenous antioxidant enzymes. Therefore, the present study examined whether SFN could protect from Ang II-induced cardiomyopathy and the underlying mechanism.

**Methods:** FVB mice were given subcutaneous injection of Ang II (0.5 mg/kg) for 2 months with or without SFN treatment (0.5 mg/kg) for 3 months and then kept until 6 months. The aortic pressure and cardiac function were measured. Cardiac fibrosis, inflammation, and oxidative damage were detected by Western blotting, real-time PCR, and immunohistochemical staining. Furthermore, cardiac- overexpressing Nrf2 gene (Nrf2-TG) and wild-type (WT) mice were treated with Ang II (0.5 mg/kg) for 2 months to confirm the critical roles of cardiac Nrf2 on prevention of Ang II-induced cardiomyopathy. In vitro, H9c2 cells were treated with SFN and Ang II with or without Nrf2 siRNA or Akt inhibitor (LY294002) to dissect the mechanism of SFN’s protective effect.

**Results:** SFN significantly prevented Ang II-induced high blood pressure at 6 months and cardiac dysfunction at both 3 and 6 months. Ang II caused remarkable pathological changes, including myocardial hypertrophy and collagen accumulation, along with increases in cardiac oxidative damage (3-NIT and 4-HNE), inflammation (TNF-α and PAI-1), and fibrotic response (TGF-β1 and CTGF). Those damages were almost eliminated by SFN treatment that up-regulated Nrf2 function. Ang II action was reflected by increased Nrf2 phosphorylation and downstream antioxidants. To define the direct role of SFN-activated Nrf2 in preventing Ang II-induced cardiomyopathy, in vitro H9c2 cells were treated with Ang II in the absence or presence of Nrf2 siRNA to