Collagen III, connective tissue growth factor (CTGF) in neonatal rat cardiac fibroblasts induced by TGF-β. DIM attenuated the increased phosphorylation of AKT and GSK-3β induced by TGF-β.

**CONCLUSIONS** Our results showed that DIM was a potential drug to attenuate myocardial differentiation and modulate the excessive ECM production induced by TGF-β, through down-regulated AKT/GSK-3β signaling pathways.

**GW26-e4573**

Anti-inflammatory effect of 3,3′-Diindolylmethane on LPS-induced inflammatory injury in neonatal rat cardiac myocytes via suppressing TLR-4/MAPKs signaling pathways

Jin Li, Qingsong Wu, Wei Deng, Qizhu Tang
Department of cardiology, Renmin Hospital of Wuhan university

**OBJECTIVES** 3,3′-Diindolylmethane (DIM), extracted from cruciferous plants, has been shown to possess anti-inflammatory properties in various models of inflammatory diseases. However, whether DIM has an anti-inflammatory effect on lipopolysaccharide (LPS)-induced neonatal rat cardiac myocytes injury is poorly understood. Therefore, this study aimed to evaluate the protective effect of DIM on LPS-induced inflammatory injury in neonatal rat cardiomyocytes and explore the anti-inflammatory molecular mechanisms.

**METHODS** The cultured neonatal rat cardiomyocytes in vitro were stimulated with LPS (10 mg/L) for 12 h or 24 h to induce inflammatory injury, and DIM was incubated with these cells in the presence and absence of LPS. Cell viability was measured by Trypan blue. ROS production was detected by fluorescence staining. The mRNA expression levels of inflammatory mediators were measured with Real-time PCR and the protein expression levels were examined with Western Blotting analysis.

**RESULTS** Our data showed that DIM could obviously attenuate the increased mRNA expression levels of pro-inflammatory cytokines including interleukin (IL)-6, tumor necrosis factor (TNF)-α and high mobility group box 1 (HMGB1) induced by LPS. Moreover, DIM could also remarkably inhibit the elevated protein expression levels of Toll like receptor-4 (TLR-4), phosphorylated extracellular signal-regulated kinases 1/2 (ERK1/2), phosphorylated P38 and phosphorylated c-Jun NH2-terminal kinase (JNK) induced by LPS.

**CONCLUSIONS** Our results suggested that DIM had a protective effect on LPS-induced inflammatory injury in neonatal rat cardiomyocytes, and modulated the excessive production of pro-inflammatory cytokines IL-6, TNF-α and HMGB1 by suppressing the TLR-4/MAPKs signaling pathways.

**GW26-e4587**

IL-24 gene protects against H2O2-mediated injury of human umbilical vein endothelial cells and may be useful as a treatment for cardiovascular disease

Zhaoxia Wang,1 Yang Wang,2 Juyuan Lv1
1First Hospital of Shanxi Medical University, Taiyuan, Shanxi, P.R. China; 2Bank of China Shanxi Branch, Taiyuan, Shanxi, P.R. China

**OBJECTIVES** To investigate the protective effect of IL-24 on H2O2-mediated vascular endothelial injury and to examine the association between IL-24 and cardiovascular disease.

**METHODS** Human umbilical vein endothelial cells (HUVECs) were treated with increasing concentrations of H2O2 in the presence or absence of IL-24, which was introduced via Lipofectamine 2000-mediated transfection. Successful uptake of IL-24 plasmid was confirmed by Western blotting. Cell viability was determined by CCK-8. Apoptosis and IL-24 on HUVEC proliferation and migration was determined by the MTT assay, whereas the production of Type I collagen was determined by ELISA and reverse transcription-polymerase chain reaction. Nuclear Type I procollagen protein levels were evaluated using immunocytochemistry.

**RESULTS** CTRP 9 treatment effectively inhibited HG (25 mmol/L)-induced increases in the proliferation of CFs and collagen synthesis, concomitant with suppression of HG-induced upregulation of cAMP levels. Blockade of cAMP-PKA pathway reversed the inhibitory effect of CTRP9 on ERK phosphorylation in response to HG.

**CONCLUSIONS** The present data indicate that CTRP9 functions to inhibit HG (25 mmol/L D-glucose)-induced increases in the proliferation of CFs and collagen synthesis via cAMP-dependent mechanism, suggesting that the therapeutic approaches to enhance CTRP9 production could be valuable for prevention of diabetes cardiac fibrosis.

**GW26-e4642**

The effects of AGEs and RAGE on the accelerated progression of age-related intrarenal small arterial stiffness in hypertensive rats

Yajing Bai,1 Huashan Hong1
1Department of Intensive Care Unit, Fujian Medical University Union Hospital, Fuzhou, Fujian, China; 2Department of Geriatrics, Fujian Medical University Union Hospital, Fuzhou, Fujian, China

**OBJECTIVES** To dynamically investigate the morphological change of age-related intrarenal small arterial(IRS) in Spontaneously hypertensive rat (SHR) and Wistar-Kyoto (WKY) rats respectively and to explore the effects of Advanced glycation end-products (AGEs) and Receptor for AGEs (RAGE) on the progression of age-related IRS stiffness accelerated by hypertension.

**METHODS** SHR and WKY rats were respectively randomized into 4, 12, 24, 48 and 72-week-old group (n = 16). Minimal renal vascular resistance (minRVR) was detected at each group. Renal arcuate arteries (RAA) and interlobular arteries (RILA) were analyzed by EVG, and minimal renal vascular resistance (minRVR) was measured. The expression of AGEs and RAGE in RILA media at 12w SHR had begun to be much higher than that in 4w SHR group (p < 0.05).

**CONCLUSIONS** Hypertension accelerates the progression of age-related intrarenal small arterial stiffness and the hypertension to