VSMCs, accompanied by reduced expression of MCP-1 and IL-6 both in mRNA and protein. ChIP assay indicated that the underlying mechanisms were relevant to the restoration of H3K9me3 levels at the promoters of MCP-1 and IL-6, and then the suppressed expression of MCP-1 and IL-6.

CONCLUSIONS The JMJD2A inhibition significantly attenuated neointimal formation in balloon injured diabetic rats via the suppression of VSMCs proliferation, migration, and inflammation.

GW26-e1560

Sodium tanshinone A sulfonate alleviates cardiomyocytes injury induced by radiation in vitro

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OBJECTIVES The present study determines whether STS could provide cardioprotective effect on radiation-induced cardiomypathy and investigates the potential molecular mechanisms against radiation-induced cardiomyocytes apoptosis in vitro.

METHODS In vitro experiment, H9c2 cells were divided into seven groups, the control group, 4Gy X-rays group, 6Gy X-rays group, 8Gy X-rays group, STS + 4Gy X-rays group, STS + 6Gy X-rays group, STS + 8Gy X-rays group.H9c2 cells pretreated with or without STS (10 μg/mL) for 24 hour were exposed to X-ray radiation. The cardioprotective effects of STS were evaluated by applying MTT, flow-cytometric analysis, Hoechst 33258 nucleus staining, and western blot.

RESULTS Following mere 4Gy,6Gy and 8Gy exposure of H9c2 to irradiation, significant reductions were found in cell survival at 24h post-irradiation respectively(15.6%,34.9%,48.5%) and radiation-induced apoptosis was confirmed by Hoechst 33258 nuclear staining. While STS treatment resulted in a significant increase in cell survival, restoring cell survival to 95.6± 7.2%(4Gy),86.7±4.5%(6Gy) and 79.1±4.2%(8Gy). Hoechst 33258 nuclear staining observations demonstrated the same protective effect of STS on radiation-induced myocardial injury. After 24h of irradiation, the apoptosis index was markedly increased in the mere X-rays irradiation groups compared with that of control group (P=0.014), whereas the apoptosis index was significantly decreased by STS compared to that in the mere X-rays irradiation groups (P=0.032). Western blot analysis also showed that STS increased PAkt(P=0.023) and P-ERK1/2 (P=0.014) in H9c2 cells, and these changes were also accompanied by the increase of Bcl-2/Bax ratio (P=0.02) and the decrease of active caspase-3 expression(P=0.03).

CONCLUSIONS Radiation could aggravate H9c2 cells injury and STS may attenuate this damage by P13K/Akt and ERK1/2 signaling pathways. This effect may be related to its inhibition of apoptosis of H9c2 cells. These results suggest that STS shows a good prospect in clinical prevention and the treament of radiation-induced heart disease.

GW26-e1826

Effects of A prescription of Jia-Shen (PJS) on Angiotensin-II Induced Cardiac Fibroblast Proliferation

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OBJECTIVES This study was aimed to determine the mechanisms for PJS attenuating Ang II-induced cardiac fibroblast proliferation.

METHODS We induced cardiac fibroblast proliferation by the use of Ang II in an in vitro model cardiac fibroblast culture. Cardiac Fibroblasts were obtained from the hearts of neonatal (1-3 days old) Sprague-Dawley rats and were cultured in serum-free medium for 24 hr, then treated with the serum derived from the rats treated with PJS for 3 days with or without Ang II(10^{-6}mol/L) treatment. After a additional 24 hr of culture, cells were harvested for analysis of cell proliferation, collagen contents and p-Smad2 /3 mRNA and protein expression.

RESULTS Cardiac fibroblast proliferation measured by MTT method increased in Ang II treated group compared to the control group(0.17±0.02 vs 0.14±0.01, $P<0.05$), The levels of collagen were increased in Ang II treated group compared to the control group (1.18±0.01 vs 0.89±0.07ug/ml, $P<0.05$). In addition, treatment with Ang II increased the expression of α-SMA, p-Smad3 protein to 1.60±0.08 and 1.47±0.06- fold (P<0.05) compared to the control group, Moreover, Ang II treated group increased p-Smad2 and p-Smad3 mRNA expression(p-Smad2: 1.07±0.10 vs 1.37±0.20;p-Smad3:1.00±0.12 vs 1.70±0.44) ($P<0.05$) compared to the control group. The serum containing 10% of the serum derived from the PJS-treated rats inhibited the increased levels of collagen and cell proliferation, and the expression of p-Smad2 and p-Smad3 mRNA and p-Smad3 protein compared to Ang II treated group(P<0.05) although it had no effect on α-SMA protein expression ($P>0.05$) compared to Ang II treated group.

CONCLUSIONS Our data showed that PJS inhibited the increases in the cardiac fibroblast proliferation and the production of collagen induced by Ang II treatment. The results were associated with the decreased expression of p-Smad2/3 mRNA and protein. Our studies suggested that PJS may inhibit cardiac fibroblast proliferation possibly via attenuating TGF-β1/Smad signaling pathway.

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GW26-e0774

Influence of Gene Expression in Kidney of Dahl Rat under Sodium or Potassium Interventions

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OBJECTIVES A large number of evidences in population trials and animal experiments have confirmed that the renal dysfunctional metabolism of sodium or potassium play a key role in formation of salt sensitivity. Our previous studies have shown that potassium supplement could have a protective effect in both blood pressure and kidney, especially in persons with salt sensitivity, however the mechanisms remain unresolved. The aim was to investigate alteration of gene in kidney of Dahl rat and preliminarily explore the protective mechanism of potassium on blood pressure, which would shed some new light in future.

METHODS Male Dahl sensitive rats and 13BN-SS rats were randomly divided into nornal salt group, high salt group and high salt plus potassium supplement group respectively; Illumina RatRef-12 Expression BeadChip were used for screening the renal different genes between high salt group and high salt plus potassium supplement group, and the clusters, GO, Pathway analysis of functionally related genes were determined on the basis of their annotation term co-occurrence; qRT PCR was performed to validate the gene expression data obtained from microarray analysis; Western blotting was performed to identify the expression of SGK1 and Mcoln3 proteins.

RESULTS Microarray scanning had shown that 594 different gene were obtained between high salt group and high salt plus potassium supplement group in Dahl salt sensitive rats, including 292 upregulated genes and 302 downregulated genes; there were 429 upregulated genes and 479 downregulated genes between Dahl salt sensitive rats and 13BN-SS rat in high salt group, totally 908 different genes; GO functional analysis found that these genes mainly take part in the process of ion transport, energy metabolism, fatty acid metabolism, oxidative stress, apoptosis and so on. Five related to sodium or potassium metabolism genes Mcoln3, SGK1, Slc34a2, Atp1a4 and Trpv6 was validated to microarray data, and the results of qRT-PCR were in accordance with the pattern of gene microarray detection. Dietary high salt could increase the expression of SGK1 in Dahl salt sensitive rats, in contrast, potassium supplement could reverse this phenomenon; no difference was observed among nornal salt group, high salt group and high salt plus potassium supplement group in 13BN-SS rat; the expression of Mcoln3 was higher in Dahl salt sensitive rats than 13BN-SS rat, moreover, potassium supplement could upregulate the expression of Mcoln3 in Dahl salt sensitive rats and 13BN-SS rat.

CONCLUSIONS Dietary high salt could increase the expression of SGK1 in Dahl salt sensitive rats, in contrast, potassium supplement could reverse this phenomenon, which may be associated with the protective effect of potassium. Abnormality of the expression of Mcoln3 may be involved in salt-induced elevation of blood pressure in Dahl salt sensitive rats.

GW26-e2382

Cadmium induced NLRP3-mediated pyroptosis in Human umbilical vein endothelial cells

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OBJECTIVES The present study was designed to explore whether cadmium could induce vascular endothelial cell pyroptosis and the underlying mechanisms.