GW23-e0181 **ANTI-INFLAMMATORY EFFECT OF GINSENOSIDE RG1 ON CARDIOMYOCYTE INJURY INDUCED BY ADRIAMYCIN**

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Objectives Chronic inflammation have played an important role in heart failure (HF). Ginsenoside Rg1 (Gs-Rg1), stemming from Ginseng, is a kind of major pharmacologically active components. However, the effect of Gs-Rg1 in HF remains to being explored, let alone its mechanisms. Thus, the present study aimed at examining the effect of Gs-Rg1 in HF, and then further elucidates its effects on proinflammatory factor, such as tumour necrosis factor alpha (TNFα), and nuclear transcription factor kappa B (NF-κB) basing on rat HF model induced by adriamycin, in vivo.

Methods Rat with adriamycin-induced HF were randomly divided into control group (age-matched health rat, n=15), HF group (ie, adriamycin group, n=15), Gs-Rg1 group (Gs-Rg1 intervention basing on HF, n=15), Gs-Rg1 was intraperitoneally administered according to body weight (4 mg/100 g) once a day for 14 days. Left ventricular ejection fraction (LVEF) was estimated through echocardiographic examination, the above gene and the above protein was estimated through ELISA, real-time RT-PCR, western blot and Electrophoretic mobility shift assay.

Results

1. Gs-Rg1 significantly improved LVEF (p=0.005).
2. Both protein and mRNA of TNFα and TNFR-1 in the HF group were higher than that in the control group (all p<0.001), which were markedly reduced by Gs-Rg1 (all p<0.001).
3. Gs-Rg1 augmented protein and mRNA of TNFR-2, which was lower in HF group than in control group.
4. Compared to HF group, Gs-Rg1 markedly inhibited the protein level of total-IKKα, phospho-IKKα, total-IKKβ and phospho-IKKβ, including their IKKα phosporylation (ie, the ratio of phosphorylated to total protein) and IKKβ phosporylation (all p<0.001).
5. Treatment with Gs-Rb1 caused a significant increase in total-IκB and IκB, and a significant decrease in IκB phosporylation compared with HF group (all p<0.01).
6. Gs-Rb1 markedly decreased total-NF-κB protein, phospho-NF-κB protein and NF-κB mRNA than that in HF group (all p<0.01).

Conclusions Gs-Rg1 may improve HF, which was mediated by proinflammatory factors, including a decrease in TNFα, NF-κB and an increase in both TNFR-2 and IκB.